Министерство здравоохранения Республики Беларусь

УО «ГРОДНЕНСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

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ДИАГНОСТИКА И ЛЕЧЕНИЕ ТЕРАПЕВТИЧЕСКИХ ЗАБОЛЕВАНИЙ НА АМБУЛАТОРНОМ ЭТАПЕ

Учебно-методическое пособие
для студентов 5 и 6 курсов лечебного факультета

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DIAGNOSIS AND TREATMENT OF INTERNAL DISEASES IN POLYCLINIC

Handbook
for 5th and 6thyear foreign students

Гродно
ГрГМУ
2018

УДК
ББК
К

Рекомендовано Центральным научно-методическим советом

УО «ГрГМУ» (протокол № от 2018 г.).

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Рецензент:

Кежун, Е.Н.
Диагностика и лечение терапевтических заболеваний на амбулаторном этапе: учебно-методическое пособие для студентов 5 и 6 курсов инстранного факультета/ Е.Н. Кежун, Н.С. Слободская, Д.Ю. Пицко, К.В. Гончар – Гродно, 2018. - с. 137.
ISBN

Учебно-методическое пособие содержит материал по диагностике, лечению в амбулаторных условиях бронхообструктивного, диспептического, анемического, мочевого и суставного синдромов, сахарного диабета 2 типа, некоронарогенных заболеваний миокарда и гепатобилиарной системы, диагностику и лечебную тактику при некоторых неотложных состояниях (приступ бронхиальной астмы, почечная и печеночная колика, диабетические и недифференцированная комы), а так же планы и содержание всех тем занятий, предусмотренных программой для студентов 5 курса и отдельных тем занятий для студентов 6 курса ФИУ. В пособии изложены планы и содержание всех тем занятий, предусмотренных программой для студентов 5 курса ФИУ с английским языком обучения.

Ответственный за выпуск – первый проректор В.В.Воробьев

CONTENTS

REDUCTION

ACD – anemia of chronic disease
ACS - acute coronary syndrome
ARI - acute respiratory infection
CCPA - cyclic citrullinated peptide antibody
CGN - chronic glomerulonephritis
CKD - chronic kidney disease
COPD - chronic obstructive pulmonary disease
CP - chronic pyelonephritis
CPK - creatine phosphokinase
CRF - chronic renal failure
CT - computed tomography
ERF –examinarion of respiratory function
FDA – folic deficiency anemia
FEV_1 - forced expiratory volume in 1^{st} second
FEVC - forced expiratory vital capacity
GEBT - genetically engineered biological therapy
G-6-PD – glucose-6-phosphate dehydrogenase
IBS - irritable bowel syndrome
IC - immune complexes
IDA – iron deficiency anemia
IGCC - inhaled glucocorticosteroids
LDH - lactate dehydrogenase
MI - myocardial infarction
MOH – ministry of healthcare
MRI - magnetic resonance imaging
NCD - neurocirculatory dystonia
NSAIDs - non-steroidal anti-inflammatory drugs
OA – osteoarthritis
PEF –peak expiratory flow
RA - rheumatoid arthritis
ReA - reactive arthritis
RDW - red blood cell distribution width
RR – respiratory rate
TIBCS – total iron binding capacity of serum
UA - unstable angina
UICS – unsaturated iron binding capacity of serum
VLC - vital lung capacity
WWTP - peak expiratory flow

INTRODUCTION

Students of 5th and 6thyears work in the clinic as an assistant practitioner at the reception of patients. 6th year students visit patients at homeunder the supervision of a teacher and a doctor. When time is limited admission student should be able to interview, perform physical examination of the patient, to carry out differentiated diagnosis in identifying the syndrome, plan survey, examine disability, and determine the tactics of treatment. Particularly important is the diagnosis of emergency conditions, students' knowledge of the principles of providing emergency aid in an outpatient setting. For this work, the student must be prepared. Algorithms for diagnosis and emergency care are brought according to clinical protocols of MOH.

The manual used common medical terminology and wording. These algorithms and schemes for diagnosis and treatment correspond with stated material.

Class 1 Bronchial obstructive syndrome: differential diagnosis. Diagnosis and treatment of asthma and chronic obstructive pulmonary disease (COPD) in an outpatient setting, medical tactics, medical and social assessment, clinical examination, primary prevention. Emergency care in asthma attack and developing status asthmaticus.

Session Purpose: To teach students the diagnosis, treatment, clinical examination, and examination of the prevention of disability in patients with bronchial asthma and COPD in an outpatient setting and principles of emergency care at an attack of asthma patients in a clinic.

Study Questions

1. Concept of bronchial obstructive syndrome, major diseases, accompanied by the syndrome. Diagnostic search algorithm in bronchial obstructive syndrome.

2. Classification of asthma and COPD. Plan examination of the patient with asthma and COPD in an outpatient setting.

3. General principles of treatment of asthma and COPD in an outpatient setting, indications for hospitalization.

4. Medical and social assessment (the period of temporary disability, the indications for rational employment of patients sent for MREB), clinical examination.

5. Prevention of asthma and COPD.

6. Emergency outpatient care in asthma attack and developing status asthmaticus.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.

3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

4. Documentations forms for lesson topic registered in Belarus (in the department).

Bronchoobstructive syndrome - a condition in which the bronchial obstruction observed multicomponent create obstructions to the air flow (more on the exhale). It is composed of components virtually irreversible (epithelial hyperplasia, hypertrophy of smooth muscle and peribronchial fibrosis), determining prognosis, and reversible (cell infiltration with inflammatory bronchial wall edema, smooth muscle spasm, and hypersecretion) determining the severity of the patient and are a target for therapeutic interventions.

Bronchial obstruction in the practice of general practitioner is common and the vast majority of patients - are patients with bronchial asthma and chronic obstructive pulmonary disease (COPD). Clinically it is manifested by cough, shortness of breath, and sometimes bouts of breathlessness. In diseases of the larynx, including cancer, trachea and mediastinum, accompanied by a narrowing of the upper airway can also be paroxysmal or persistent shortness of breath, and sometimes statusasthmaticus.

Bronchospastic syndrome can develop as a result of serotonin crises in carcinoma of the bronchus or intestine, seen as a variant debut periarteritis nodosa. The clinical picture resembling bronchial asthma attack can occur when exogenous allergic alveolitis after inhalation of dust of organic raw materials, usually agricultural, affected by mold, chronic heart failure with cardiac asthma attacks.

Bronchial obstruction syndrome can be observed as well in tracheobronchial dyskinesia, gastroesophageal reflux disease, bronchial foreign body, obstructive sleep apnea syndrome, etc.

Diagnostic search algorithm in outpatient.

Detailed analysis of:

• patient's complaints (character of cough, sputum, shortness of breath, asthma attacks, the dynamics of weight, etc.);

• history of disease, triggers breathlessness, asthma attacks, duration of disease, the results of previous tests and treatments;

• history of life (risk factors - smoking, occupational exposures, heredity, allergic history);

• an objective examination of the patient (not only the respiratory system but also other systems);

• X-ray of lungs;

• sputum general analysis, atypical cells, Koch bacillus, Cushman spiral, eosinophilia;

• total blood count;

• study of respiratory function, including with the use of bronchodilators.

Further screening program will depend on the results obtained.

Chronic obstructive pulmonary disease (COPD) (ICD-10 J44) is one of common diseases and is an important medical and social problem throughout the world, is among the leading causes of temporary disability and invalidity, took 4th place in the world of causes of death. The incidence of COPD is increasing worldwide, due to increased tobacco consumption, environmental degradation.

In Belarus, COPD was diagnosed in about 1 % of the population - registered consist of about 60.000 patients.

According to the latest version of GOLD («Global Initiative for Chronic ObstructiveLung Disease», 2017,http://goldcopd.org/) COPD is defined as a "disease characterized partially irreversible airflow limitation. Restriction of air flow tends to be steadily and due to the progressive nature of the abnormal inflammatory response of the lung tissue of the tracheobronchial tree, the lung parenchyma and vascular irritation by various pathogenic particles and gases".

The term "COPD" now includes chronic obstructive bronchitis, emphysema (secondary that has arisen as a morphological change in the lungs as a result of prolonged bronchial obstruction), pulmonary fibrosis, secondary pulmonary hypertension, chronic pulmonary heart.

Airflow obstruction in patients with COPD is formed by reversible and irreversible components. Reversible component formed as a result of spasm of smooth muscle, edema of the bronchial mucosa, and mucus hypersecretion, arising under the influence of a large selection range of inflammatory mediators (IL-6, tumor necrosis factor, neutrophil proteases, and free radicals). Irreversible component of airflow obstruction determined by developing emphysema, epithelial hyperplasia, hypertrophy of smooth muscle cells and peribronchial fibrosis. Due to the elastic properties of the lung disorders varies mechanics of breathing and formed expiratory collapse is an important cause of irreversible airflow obstruction. Peribronchial fibrosis - a consequence of chronic inflammation affects the formation of irreversible component less than emphysema. The development of emphysema causes reduction in the vasculature of the lung tissue sections is not capable to gas exchange. As a result, blood flow is redistributed to the remaining sections of the lung tissue, there are ventilation perfusion expressed violations. Unevennessof ventilation-perfusion relationships is one of the important elements of the pathogenesis of COPD. Perfusion of poorly ventilated areas leads to a reduction of arterial oxygenation, ventilation is insufficient excess perfused areas leads to an increase in dead space ventilation and delay CO_2 emissions. Chronic hypoxia leads to compensatory polycythemia - secondary polycythemia with a corresponding increase in blood viscosity and impaired microcirculation, which aggravate the ventilation-perfusion mismatch. An important component of the pathogenesis of COPD is a respiratory muscle fatigue, which in turn reduces the work of breathing and ventilation exacerbates violations.

Thus, due to the unevenness of ventilation and ventilation-perfusion disorders relationship develops arterial hypoxia. COPD outcome is the development of precapillary pulmonary hypertension caused by vasoconstriction of small pulmonary arterioles and alveolar vessels as a

result of alveolar hypoxia. Gradually developed hypertrophy of the right ventricle of the heart. Forms of chronic pulmonary heart syndrome, decompensated it appears at first transient, and then constantrightventricule failure.

Risk factors of COPD:

- Internal (endogenous, genetically determined):

• α-antitrypsin deficiency;
• bronchial hyperreactivity;
• a high level of IgE;
• congenital anomalies of the bronchi and lungs;
• perinatal pathology and childhood illness (low birth weight, malnutrition, childhood bronchiolitis, rickets and other childhood diseases).

- External (exogenous disease-causing)

• tobacco smoking (active and passive), the main risk factor for COPD in 80-90% of patients.
• the impact of occupational hazards (dust, chemical pollutants, pairs of acids and alkalis, etc.);

Classification .

The basis of stratification based on two criteria: clinical, including cough, phlegm and wheezing production, and functional, taking into account the degree of irreversibility of airway obstruction. Also previously discriminated risk of developing COPD as stage 0 of disease, but COPD occurs and progresses flows long before significant functional disorders defined instrumentally. During this time, the inflammation in the bronchi leads to gross morphological changes irreversible, so this stratification does not solve the issue of early diagnosis and the timing of initiation of treatment.

In recent texts GOLD abandoned this category because of insufficient evidence that patients with "At Risk" (chronic cough, sputum formation under normal respiratory function), carefully developed stage I COPD.

COPD stages of disturbances of respiratory function (GOLD, 2017).

Cited are post-bronchodilator FEV_1, i.e. severity assessed by airflow obstruction after inhalation of a bronchodilator:

Stage I. Light

• FEV_1/FEVC less than 70 % of predicted level
• FEV_1 80 % of predictedlevel
• With or without chronic symptoms (cough, phlegm)

Usually, at this stage, to the doctor do not address rare exacerbation, cough and sputum production are perceived as the norm, especially smokers.

Stage II. Moderate

• FEV_1/FEVC less than 70 % of predicted level
• FEV_1 less than 80 % of predicted level
• With or without chronic symptoms (cough, usually in the morning, expectoration scanty, dyspnea)

This stage, in which patients seek medical care due to dyspnea or aggravation of the disease, characterized by an increase of obstructive disorders (FEV_1 50-80 % of predicted level). Manifesting dyspnea appears on exertion.

Stage III. Severe

• FEV_1/FEVC less than 70 % predicted level

• FEV_1 less than 50 % predicted level

• With or without chronic symptoms (cough, phlegm, shortness of breath)

Characterized by a further increase in airflow limitation (FEV_1 30-50 % predicted level), buildup of breathlessness, frequent exacerbations.

Stage IV. Very severe

• FEV_1/FEVC less than 70 % predicted level

• FEV_1 less than 30 % predicted level or less than 50 % in combination with chronic respiratory failure

At this stage, quality of life deteriorates significantly, and exacerbation can be life-threatening. The disease becomes debilitating. Characterized by extremely severe airflow obstruction (FEV_1< 30 % predicted level or <50 % in the presence of respiratory failure).

Diagnostics

Diagnosis of COPD is based on anamnestic data, clinical manifestations and results of a study of pulmonary ventilation function. The disease usually develops in middle age and progresses slowly. Risk factors, as indicated earlier, is the habit of smoking, occupational hazards, atmospheric pollution, smoke from domestic heating appliances, kitchen fumes, chemical irritants. The main clinical manifestations are cough and breathlessness. Cough and sputum scant separation may occur only in the morning. Cough usually celebrated throughout the day, sometimes at night only. Phlegm usually small, it is exacerbations mucous, phlegm often occurs after prolonged cough. Dyspnea usually progresses over time. It is enhanced during exercise in wet weather, during exacerbations. On examination, the patient listened scattered dry rales different timbre. Sometimes auscultatory phenomena in the lungs are not defined and their identification should be offered to the patient to make a forced expiration. In the later stages of COPD are present clinical signs of emphysema (increased anteroposterior chest size, extended intercostal spaces, box sound with percussion). With the development of chronic respiratory failure and pulmonary hypertension observed "warm" acrocyanosis, swollen neck veins. Patients with moderate to severe disease identified two clinical forms of COPD - emphysemic (panacinous emphysema, "pink puffers") and bronchitic (centroacinous emphysema, "blue puffy"), although this division is rather arbitrary and in practice are observed mixed with options predominance of one of the forms.

Progressive exertional dyspnea, weight loss predominates in the clinical pictureof emphysematous form. Cough and sputum production are insignificant or absent, hypoxemia, pulmonary hypertension, pulmonary heart decompensation developed in the late stages of the disease.

In the clinical picture of bronchitic form dominates productive cough, develop early severe hypoxia, pulmonary hypertension, cor pulmonale and right heart failure. A marked cyanosis, acrocyanosis, edema could appears. Dyspnea expressed relatively weak.

Also distinguish two main phases of COPD: a stable and aggravation. Considered stable state when the symptoms did not significantly change during the weeks and even months, and

the progression of the disease can be detected only after prolonged dynamic observation of the patient (6-12 months).

Exacerbation of COPD - a relatively long period (at least 24 h) deterioration of the patient's condition by its severity is outside the normal daily variability of symptoms characterized by acute onset and requires changes to conventional therapy schema. The frequency of exacerbations of COPD increases progressively with increasing severity of the disease. Distinguish acute infectious and noninfectious nature.

Non-infectious causes of COPD exacerbations (massive exposure of aerosolvents, cardiac decompensation, pulmonary thromboembolism, cardiac arrhythmias) occur in approximately 30 % of cases. Almost in every third patient the cause of aggravation can not be established.

Respiratory exacerbation criteria:

• Increased shortness of breath;
• Increased volume and purulent character of the sputum;
• Increased cough;
• Shallow rapid breathing.

Systemic symptoms: fever, increased heart rate, impaired consciousness.

In blood count usually marked leukocytosis with stab shift and increased ESRduring exacerbation.

X-ray examination of the chest is carried out in the period of acute illness to avoid pneumonia, spontaneous pneumothorax, pleural effusion, as well as in the differential diagnosis with other diseases (lung cancer, tuberculosis, etc.).

Study of respiratory function (ERF) - a key step in the diagnosis of COPD. The main criterion of existing patient bronchial obstruction is to reduce the FEV_1/FEVC ratio less than 70 % of predicted level, even with the FEV_1 more than 80% of the predicted value and this change has been observed in the 1st stage of the disease. Obstruction is considered chronic if it registers at least 3 times in one year, in spite of therapy.

Minimum diagnostic evaluation of patients with COPD also includes ECG, echocardiography, according to testimony bronchoscopy, computed tomography of the chest and other research. Diagnostic standard is to identify the partially irreversible airflow obstruction in the study of pulmonary ventilation. Forced expiratory volume in the first second (FEV_1) was reduced, and decreases as the disease progresses. To assess the reversibility of obstructive ventilation disorders conducted pharmacological trial. Initial FEV_1 compared with the same parameter 30-45 minutes after inhalation of sympathomimetic (salbutamol) or anticholinergic, or a combination of bronchodilators of different mechanism of action. FEV growth by more than 15 % or 200 ml, and more evidence of reversibility of bronchial obstruction. 12 % or less –proof of irreversible or partially reversible obstruction. Reversible obstruction does not exclude the presence of COPD.

Treatment and prevention. The main goals of treatment are: warning of disease progression, exacerbations and complications, reducing the severity of clinical symptoms and improving quality of life, reduced mortality.

Non-pharmacologicaltreatment includes the following groups of activities:

• patient education in educational programs;
• smoking cessation, which significantly improves the prognosis;
• elimination of the effects of other exogenous pollutants;
• modifying the diet, as for patients with COPD in the early stages of its development is characterized by the hypersthenicconstitution, overweight, while in severe and very severe stages –need weight loss, because overweight is an independent risk factor for mortality of patients;
• physical training - the "ideal" duration of training programs uncertain. The optimal period of training is considered to 8 weeks. The duration of one physical exercise (depending on the patient) ranges from 10 to 45 min, multiple classes - from 1 to 5 times per week. Commonly used dosed walking, exercise equipment, hand ergometer,lifting dumbbells 0.2 - 1.4 kg, physiotherapy.

Drug therapy in stable COPD involves the use of basic bronchodilators (β_2- agonists, anticholinergics, theophylline, and combinations thereof) that increase exercise tolerance even in the absence of changes in FEV_1). Inhalation therapy is preferred. Patients with mild COPD used short-acting drugs as needed; with moderate, severe and very severe recommend long-term regular treatment with long-acting bronchodilators or combination therapy.

Dosages most common bronchodilators: salbutamol (Ventolin) - 100-200 mg up to 4 times a day; fenoterol (berotek) - 100-200 mg up to 4 times a day; salmeterol - 25 mg 2 times a day; formoterol "Autohaler" - 12 mg 2 times a day; formoterol "turbuhaler" - 4.5 - 9.0 mg 2 times a day. Contraindications to the use of β_2-agonists in COPD are increased sensitivity to any component of the drug, tachyarrhythmia, heart defects, aortic stenosis, hypertrophic cardiomyopathy, decompensated diabetes mellitus, hyperthyroidism, glaucoma, threatened abortion. Should be used with caution especially this group of drugs in elderly patients with concomitant diseases of the heart, arterial hypertension.

Traditionally, basic bronchodilators for the treatment of COPD, especially in older age groups are considered anticholinergics. Of this group received a wide application ipratropium bromide (Atrovent 40 mg 4 times per day). After a single application of 40 mg (2 inhalations) ipratropium action starts after 20-40 minutes, reaching a maximum after 60 minutes and lasts for 5-6 hours. At doses providing bronchodilator action does not penetrate the central nervous system, to a lesser extent inhibits the secretion of the salivary glands, has no effect on the locomotor activity of the ciliated epithelium of the trachea and does not alter blood pressure and heart rate.

Effective combinations of bronchodilators are in particular ipratropium with fenoterol (Flomax), formoterol and budesonide (Symbicort), salmeterol and fluticasone propionate (Seretide) 1-2 times a day. If these groups are available drugs, are ineffective or inefficient, they may be administered sustained release theophyllines (teopek, teotard, teostat, eufilong etc) 1 - 2 times a day. Theophylline may be added to the regular inhalation of bronchodilator therapy in more severe COPD.

Inhaled steroids (beclomethasone, budesonide, fluticasone, etc.) shall be appointed in addition to bronchodilator therapy in patients with COPD and FEV less than 50 % predicted (severe and very severe COPD) and frequent exacerbations (more than 3 times in the last 3

years). The most effective combination of inhaled glucocorticosteroids (IGCS) with long-acting β-adrenergic agonists: salmeterol + fluticasone propionate (Seretide) and budesonide + formoterol (sumbikort).

Treatment of patients with COPD exacerbation can be performed depending on the severity of exacerbation as an outpatient (mild to moderate exacerbation) and in hospital.

Indications for hospitalization: initially severe COPD , the appearance of new symptoms that characterize the severity of respiratory and right heart failure (shortness of breath at rest, cyanosis, peripheral edema), lack of positive dynamics of outpatient treatment or worsening of the patient during treatment, severe comorbidities (ischemic heart disease, congestive heart failure).

Exacerbation of COPD usually associated with activation of microbial inflammation in the bronchial tree and should be considered as a factor in disease progression. For relief of acute bronchodilator therapy along with antibiotics are used for 7-14 days, glucocorticosteroids (including system up to 10 days) and in the hospital - additional oxygen therapy.

The choice of antibiotic is carried out empirically. With mild to moderate uncomplicated exacerbation is assigned one of the following drugs inside: amoxicillin 0.5-1.0 g every 8 hours, amoxicillin/clavulanic acid (Augmentin, amoxiclav), 0.625 g before meals every 8 hours or 1.0 g 2 times a day, macrolides (clarithromycin 0.5 g 2 times a day, azithromycin 0.5-1.0 g once daily for 3 days), respiratory fluoroquinolones (levofloxacin at 0.5-1.0 g ormoxifloxacin day or 0.4 g 1 time per day).

Antibiotic effectiveness criteria: positive clinical dynamics in 3-5 days (decrease exacerbation symptoms - shortness of breath, cough, sputum quantity or purulent component body temperature, etc.), normalization of ESR and leukocytosis, slimy character of sputum. Clinical exacerbation of COPD typically resolved within 10 days, but almost 75% of patients FEV1 indicators (longer than 1 month) do not return to baseline.

Medical and social assessment. COPD patients with compensated pulmonary heart are mainly employable. They are contraindicated in heavy physical labor, especially in unfavorable sanitary conditions and associated occupational hazards. If possible, profession could be changed.

Temporary disability occurs during exacerbation and continued depending on the severity continues 9-16 days. There is often respiratory and pulmonary heart disease. Such patients are directed to the MREB. The issue of disability, the disability group setting.

Dispanserization. Observation frequency depends on the severity. In mild - 1 times per year, in moderate - 2 times per year, in severe stage - 2 times per year.

Examinations by medical specialists: pathologist, pulmonologist, cardiologist - for medical reasons.

Laboratory and instrumental investigations:

• total blood count, sputum analisis, spirography - 1-2 times per year;

• ECG , chest X-ray - 1 time per year, in severe ECG - 2 times per year;

• bronchoscopic study - for medical reasons;

• monitoring of respiratory function in the progression of respiratory failure.

Basic medical and preventive measures: basic treatment according to clinical protocols (ipratropium bromide, prolonged methylxanthines, inhaled corticosteroids, systemic corticosteroids). Symptomatic therapy (mucolytics, antibiotics) - indicated. Herbal medicine, physiotherapy, breathing exercises also could make some effect. During exacerbation - hospitalization.

Observation periods and deregistration criteria: observation for life.

Clinical examination performance criteria: reduction in the frequency and duration of exacerbations, improved clinical performance.

Bronchial asthma (ICD-10 - J45)

Bronchial asthma (BA) - a chronic inflammatory disease of the airways, which is based on pathogenetic chronic allergic inflammation and bronchial hyperreactivity characterized by repeated episodes of bronchial obstruction, reversible, either spontaneously or under the influence of the treatment, manifested attacks of breathlessness, wheezing lungs, often heard at a distance, cough, chest tightness, especially at night or early morning.

Risk factors.

- Internal factors :

• genetic predisposition;
• atopy;
• airway hyperresponsiveness (acquired);
• the female sex;
• race/ethnicity.

- External factors:

• household allergens;
• external allergens (pollen, fungi);
• professional allergens;
• smoking (active and passive);
• air pollutants (indoor and external);
• respiratory infections;
• parasitic infections;
• foods and medicines;
• neuropsychiatric effects.

- Factors that trigger an exacerbation of asthma and / or supporting symptoms (triggers):

• domestic and external allergens (see above);
• respiratory tract infections (primarily ARI);
• pollutants premises and external air pollutants (sulfur oxides, nitrogen, etc.);
• exercise and hyperventilation;

• adverse weather conditions (extreme changes in temperature, humidity, significant fluctuations of atmospheric pressure);

• taking medicines (antibiotics, β- blockers, NSAIDs, etc.);

• the impact of non-specific stimuli (odors, perfumes, paints, varnishes, etc.);

• excessive psycho-emotional stress;

• smoking (active and passive);

• nutritional supplements.

Classification. Greatest practical importance is the classification of asthma etiology (clinical form), severity and level of control.

- Clinical :

• allergic (atopic, extrinsic, IgE- mediated) - there is a clear positive association between allergen and aggravation after contact with him;

• non-allergic (non-atopic, endogenous, cryptogenic) - There is no clear relationship with allergic manifestations;

• Mixed - includes symptoms as allergic and non-allergic forms.

Table 1. 1 Classification of asthma severity of the disease

Severity of disease	Diagnostic criteria
Stage 1 Mild intermittent asthma	Easy periodical exposure asthma symptoms at least 1 time per week. Short exacerbation Nocturnal symptoms not more than 2 times per month. FEV_1 or peak expiratory flow (daily activities) greater than or equal to 80 % predicted. Daily variability indices daily activities or FEV_1 less than 20%
Stage 2 Mild persistent asthma	symptoms more than 1 time per week, but less than 1 time per day Exacerbations may affect physical activity and sleep Nocturnal symptoms more than 2 times per month Daily activities or FEV_1 greater than or equal to 80 % predicted Daily variability indices daily activities or FEV_1 between 20-30 %
Stage 3 Moderate persistent asthma	Daily symptoms Exacerbations may affect physical activity and sleep Nocturnal symptoms more than 1 time per week Daily intake of $ß_2$ - agonist short-acting FEV_1 or daily activities from 60 to 80 % of the predicted value Daily variability indices daily activities or FEV_1 more than 30%
Stage 4 Severe persistent asthma	Daily symptoms frequent exacerbations Frequent nocturnal symptoms Limitation of physical activity FEV_1 or daily activities less or equal to 60 % predicted Daily variability indices daily activities or FEV_1 more than

	30%

Table 1.2 Classification of asthma by level of control ("Global Strategy for the prevention and treatment of asthma", GINA, 2018)

Clinical Evaluation of the current control (preferably within 4 weeks)			
Features	ControlledBA(all of the above)	Partly controlled BA (presence of any manifestation within 1 week)	Uncontrolled BA
1	2	3	4
Dayly symptoms	None or less 2 episodes per week	More than 2 episodes per week	3 or more signs partially controlled asthma during any week
Activity limitation	None	Any	
Nocturnal symptoms/awakening	None	Any	
The need for emergency drugs	No or less than 2 times per week	More than 2 times per week	
Lung function (FEV_1or PFM)	Normal	Lessthan 80 % ofpredictedlevelorless fromthebestindicatorforthepatient (ifknown)	

Risk assessment in the future (risk of exacerbations, not stable course, the rapid deterioration of lung function, side effects)

Patient with any of the following signs has the risk of undesirable effects in the future: uncontrolled disease, frequent exacerbations in the last year, hospitalization in the intensive care unit, low level FEV_1 exposure to tobacco smoke (cigarette smoking), high-dose drug therapy to maintain control.

Table 1.3 Diagnosis of asthma

Obligatory	Additional (by prescription)
Analysis of complaints, anamnesis of disease, life (family, professional), allergic history	Immunogram the definition of general and specific IgE
Chest percussion	Coagulation: clotting time Lee-White, activated partial thromboplastin time, prothrombin time, ethanol test, fibrinogen A blood clot retraction, spontaneous fibrinolysis
Auscultation of the lungs	Definitionbloodgases (PaO_2, $PaCO_2$, $SatO_2$) Determination of parameters of acid-base status
Measuring the frequency of breathing	High resolution computed tomography of the chest
Peak expiratory flow	Bronchoscopy Ultrasound investigation of the heart Consultation with psychotherapist
Radiography of the paranasal sinuses	Level of nitric oxide (NO) in exhaled air
Spirography Pneumotachograph	
Study of respiratory function when exposed to bronchodilators	

Study of respiratory function in drug provocation (perfomed by allergist)	
Study of respiratory function during exercise provocation	
Total blood count + platelets	
Cytological study of mucus from the nose	
Sputum smear microscopy (eosinophils, Cushmann spiral, Charcot-Leyden crystals)	
Fecal test on helminths and giardia	
ECG	
Consultation with allergologist-immunologist	
Consultation with an otolaryngologist	
Consultation withpulmonologist	

In this regard, any patient with complaints of cough, wheezing , or shortness of breath that occurs more than 3 times a year, is regarded as a potential patient with bronchial asthma, requiring a thorough investigation to confirm or exclude the diagnosis.

Clinical manifestations of asthma.

The diagnosis of asthma is based on the identification of symptoms such as episodic expiratory dyspnea, wheezing, feeling of chest tightness, coughing.

Expiratory dyspnea is nature involving auxiliary muscles. Cough is paroxysmal, sometimes with expectoration of thick, viscous mucus. Position of the patient at the time of an asthma attack forced - "sitting" with fixing the upper body. There is marked swelling of the wings of the nose, swelling of the neck veins. Skin - pale, cyanosis pronounced nasolabial triangle, acrocyanosis. When percussion of lungsis defined box sound (acute emphysema). Auscultation on the background of vesicular breathing c elongated exhalation listened diffuse dry wheezing, they heard at a distance (distant wheezing).

Symptoms of the disease often occur or are worse at night and in the morning. Due to the fact that the clinical symptoms of asthma varie during the day, it is detected for examination of a patient must be carried out at occurrence of symptoms and before receiving bronchodilator drugs. Significant clinical marker of bronchial asthma is disappearance of the symptoms either spontaneously or with the application of anti-inflammatory and bronchodilator medicament.

At moderately fit the patient can not lie in the bed. Stimulated vertical position (orthopnea) during an attack so typical that its absence indicates either a light attack or error diagnostics. At the same time it is necessary to know what the night can cause coughing received angiotensin converting enzyme inhibitors, gastroesophageal reflux and chronic sinusitis.

Evaluation of RF in bronchial asthma. RF study is required in the diagnosis of asthma, including a survey of patients in the early stages of the alleged disease (high-risk), dynamic assessment of the patient during treatment, it is as for asthma is characterized by reversible airway obstruction, high speed characteristics of respiratory variability (indirect hyperactivity score). High sensitivity of patient to bronchoconstrictors. Every patient with suspected

bronchospasm in any of the available instruments should register FEVC and FEV_1 and calculate the ratio of FEV_1/FEVC, and daily activities. FEV_1 normal rate is not less than 80 % of the due value. Considered moderately reduced level from 79 % to 60%, significantly reduced - less than 60%.

Bronchodilator tests reveal bronchial lability (hidden bronchospasm), allow to evaluate the reversibility of obstruction, which is of great importance for the diagnosis of asthma. Bronhodilatation response is considered to be positive with an increase in FEV_1 of more than 15 % of the predicted value after 15-20 minutes after inhalation of β_2-agonist short action.

Complete study of RF in the interictal period includes samples to identify hyperactivity disorder (hypersensitive) bronchi - an exercise tolerance test. Methods for determination of nonspecific bronchial hyperactivity: Exercise stress tests, tests with hyperventilation dry cool air, etc. Standard exercise test is 6-10 min. bicycle ergometry or 6-10 min. running on a treadmill (constant load rate of 1.5 W per 1 kg of body weight). The criterion is to reduce bronchospasm poslenagruzochnogo indicators daily activities, FEVC,FEV_1 more than 15 % after 3-5 minutes of rest.

Principles of treatment of asthma in an outpatient setting.

Treatment of patients with bronchial asthma is carried out in an outpatient and inpatient health care organizations of the Republic of Belarus at all levels of care. The majority of patients treated on an outpatient basis.

The main goals and objectives of the treatment of asthma:

• elimination or mitigation of clinical manifestations (achieving disease control);

• reducing the frequency and severity of exacerbations;

• prevent the development of life-threatening conditions and deaths;

• normalization or improvement of respiratory function;

• restoration and maintenance of vital activity (age-appropriate), including exercise tolerance;

• lack of or reduced need for bronchodilator therapy;

• prevention of disability.

Overseeing the development and course of asthma is achieved using a set of non-drug and drug activities in cooperation of the patient and the doctor.

Non-pharmacological interventions include six main components:

• training in patients “asthma school”;

• Conduct monitoring and evaluation of the severity of asthma by recording patient symptoms (diary) and, if possible, peak flow in the morning, immediately after rising from bed, then after 12 hours with the definition of the deviation PSV;

• elimination of risk factors;

• development of individual plans for long-term drug therapy management of the patient (taking into account the severity of the disease and the availability of asthma medication);

• development of individual plans cupping exacerbations;

• ensuring regular dynamic monitoring;

• social and medical significance of these events in the Republic of Belarus unified "agreed national guidance on the diagnosis, treatment, prevention and rehabilitation of asthma" (2006).

Drug therapy involves the use of two main groups of drugs:

• drugs for alleviating symptoms (emergency drugs): β_2- agonists, short steps, short-acting theophylline and systemic glucocorticosteroids;

• drugs for long-term preventive therapy (baseline, control) anti-inflammatory (inhaled and systemic glucocorticosteroids cromones), β_2 agonist long-acting drugs, antileukotriene and long-acting theophylline.

Preparations basic therapy is long, taken regularly, every day. The most effective of these are glucocorticosteroids multiple units acting on the inflammatory process.

Inhaled steroids have a preference (IGCS) – beclomethasone, dipropionate, budesonide, fluticasone propionate, flunisolide, providing mainly local anti-inflammatory effect and cause virtually no systemic side effects.

Systemic glucocorticosteroids prednisolone group (prednisolone, prednisone, metipred) and triamcinolone group (triamcinolone, kenokort, berlikort, polkartolon) indicated in patients with severe asthma with the ineffectiveness of high doses of inhaled corticosteroids in combination with a regular intake of bronchodilators.

Mast cell stabilizers (cromones) - sodium cromolyn (Intal) and nedocromil sodium (tayled) applied by inhalation of 1-2 (or 1 at the dose of 20 mg and 2 mg) for treating patients with mild persistent asthma as well as prophylactically to prevent bronchospasm during exercise, inhalation of cold air, possibly contact with the allergen.

Leukotriene receptor antagonists (zafirlukast, montelukast) - a new group of anti-inflammatory and anti-asthmatic drugs, reducing the need for β_2- adrenomimetic short-acting , effective in preventing attacks of bronchospasm.

β_2- agonists, long-acting (salmeterol, formoterol) and their combination with inhaled corticosteroids: salmeterol + fluticasone propionate (Seretide) and formoterol + budesonide (Symbicort) and the theophylline sustained release (teopek, teotard, teoetat, eufilong etc.) are applied simultaneously with anti-inflammatory drugs increase the efficacy of treatment using lower doses of inhaled corticosteroids, reduce the frequency of nocturnal attacks etc.

Formed a 5-speed (steps) with the increasing volume of therapies aimed at achieving asthma control.

1st stage - a drug to relieve symptoms as needed. Intended only for patients who do not receive support (controlling) therapy and occasionally experiencing short-term (up to several hours), asthma symptoms.

2nd stage - a drug to relieve symptoms (Reliever medication) plus one drug for maintenance (controlling) therapy. As an initial maintenance therapy is recommended at this stage at low doses of inhaled corticosteroids. Alternative means of maintenance therapy is considered antileukotriene drugs.

3rd stage - a drug to relieve symptoms plus one or two drugs for maintenance therapy. Recommended low-dose inhaled corticosteroids with inhaled long-acting β_2- agonists in a fixed combination or in different inhalers. Increasing the dose of inhaled corticosteroids is indicated in patients whose asthma control is not achieved after 3-4 months of such treatment. Combination of formoterol and budesonide (Symbicort) can be used as maintenance therapy, as well as for emergency treatment. This approach provides a reduction in the frequency of exacerbations and improved asthma control.

The second treatment option - appointment to medium doses of inhaled corticosteroids.

Alternatively, the possible combinations of low-dose inhaled corticosteroids with antileukotriene drug, theophylline of delayed action (the latter combination is less effective).

4th stage - a drug to relieve symptoms plus two or more drugs for maintenance therapy. Preferred inhaled corticosteroids in medium or high doses of β_2- agonists with long-acting . A combination of medium-dose inhaled corticosteroids with antileukotriene drugs or sustained-release theophylline, which, however, is less effective than the combination with β_2-agonists. However, high doses of inhaled corticosteroids with β_2- agonists long-acting only recommended if there is no effect for 3-6 months from the appointment of moderate doses in combination with β_2-agonists and/or third drug for maintenance therapy (antileukotriene or sustained-release theophylline).

5th stage - a drug to relieve symptoms of asthma plus additional maintenance therapy options. Treatment at level 5 involves administering tablets IGCS . The lack of effectiveness of combination therapy including high-dose inhaled corticosteroids or oral systemic glucocorticosteroids in patients with allergic asthma can use antibodies to IgE (omalizumab subcutaneously at a dose of 150-375 mg 1 time every 2-4 weeks).

In the interictal period used physiotherapists physiotherapy techniques, sanatorium treatment.

Indications for hospitalization of patients with bronchial asthma:

- severe exacerbation of asthma;
- lack of response to bronchodilativetherapy for 1-2 hours;
- a risk of death from asthma;
- threatening respiratory arrest;
- unfavorable living conditions.

In the group of patients with increased risk of death from asthma include:

• patients taking or have recently stopped (less than 6 months ago) intakeof glucocortikoids (severe stage of disease);

• patients hospitalised in the ICU during the last year;

• patients with mental illness or psychological problems;

• teenagers, young adults (15-25 years) and elderly patients (over 55 years), especially with panic and fear during an attack;

• patients who do not fulfill the prescribing physician;

• patients who are constantly taking more than three medications for asthma (severe);

• patients admitting error in receiving glucocorticoids;

• patients with a combination of asthma and diabetes, epilepsy;

• patients with a sudden and violent character attacks (attacks of type II);

• patients often uncontrolled intakeβ_2-agonists (more than 1 package per month), especially without parallel receiving inhaled glucocorticoids.

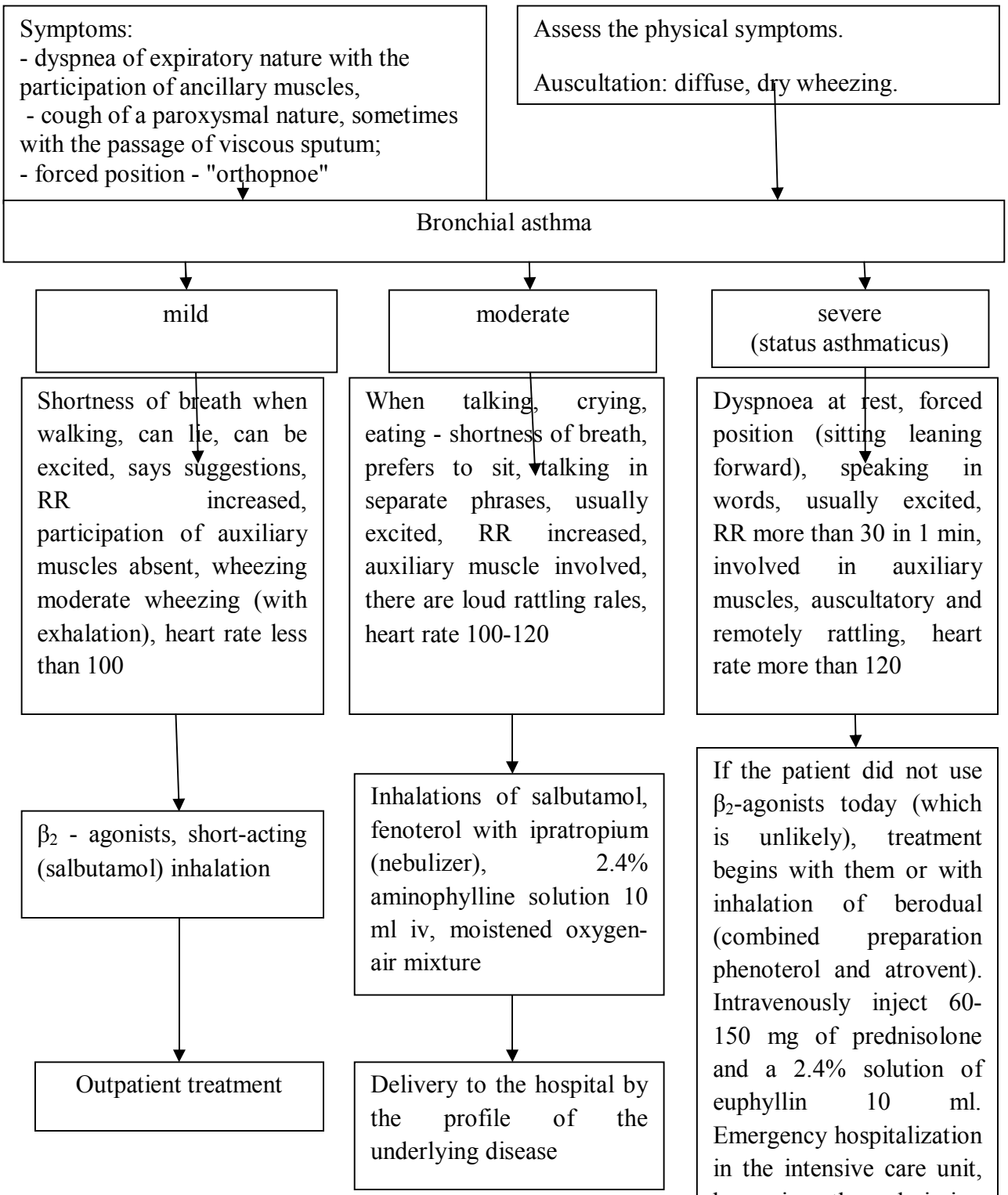


Fig 1.1 Emergency aid for attack of asthma, and developing status asthmaticus.

Medical and social assessment. With full control of the disease as a result of adequate therapy, patients with asthma for a long time remain disabled. The main condition of its preservation is to limit contact with allergen-triggers.

Contraindicated in patients: the hard physical labor, work associated with severe mental stress, the influence of a number of professional allergens and risk factors that may cause exacerbation (dust, gas pollution, chemicals, etc.), as well as some professions (e.g., hairdresser, cutter, furrier, a worker in the milling industry, etc.) and types of work, which is a sudden interruption in connection with an attack of breathlessness might harm the patient or others (air traffic controllers, drivers of vehicles, work at height, the moving mechanisms etc.).

In the acute phase of the disease, patients are temporarily unable. Duration of disability depends on the clinical form and severity of asthma, severity of complications and treatment. Tentative dates of temporary disability: in mild exacerbation - 5-7 days, with moderate - 7-10 days, with moderate to severe respiratory failure - with a possible 18-21 days to rule on – MREB to establish disability group (III, II). In severe bronchial asthma, patients with presence of pulmonary heart failure are directed to the MREB for determination of invalidity (as a rule, II group). When the direction of the patient on speleotherapy in Soligorsk medical certificate issued for the entire duration of treatment and the patient travel to the resort and back.

Dispanserization. Depending on the type and severity of asthma frequency of inspections district physician and medical specialists, the volume of research and therapeutic measures are somewhat different.

In mild intermittent and persistent course observation frequency - 1 time per year, in moderate– 3-4 times.

Examinations by medical specialists: pulmonologist, allergist - indicated.

Laboratory and instrumental investigations:

• total blood count, a general analysis of sputum, spirography - 1 times per year;

• pikfloumetric monitoring;

• chest X-ray;

• cells - 1 time per year.

Basic medical and preventive measures: identifying and avoiding contact with allergens, irritants, hypoallergenic diet, smoking cessation. Education in "asthma-school." Specific

immunotherapy - indicated. ARI and influenza prevention (vaccination), readjustment of foci of infection. Observation periods and deregistration criteria: observation for life.

Prevention. Primary prevention also aims to eliminate from the environment potentially dangerous allergens, np ritantov and other factors contributing to the possible implementation of existing clinical patient biological defects and development of asthma.

Patients suffering from bronchial asthma and even with risk factors, you should choose the appropriate profession and occupation, exclusion – foreclosing or limiting exposure to airway irritants and allergens production. It is particularly important to encourage them to avoid contact with allergens that are the cause of disease.

Sanatorium treatment of patients with bronchial asthma and COPD.

Showing most climatic resorts (the southern coast of Crimea, the Baltic resorts) resorts located in the low mountain areas, local sanatoriums of RB located in forest areas, protected from the winds. Patients referred for treatment in the warm season. Good results are obtained after speleotherapy, carring out in Soligorsk whole year round.

Class 2. Differential diagnosis of chest pain. Noncoronary heart disease: patient diagnosis, treatment guidelines, medical tactics, medical and social assessment, clinical examination, prevention

Session Purpose: To teach students outpatient diagnosis noncoronary heart disease, treatment strategies, clinical examination, and examination methods of prevention of disability of patients with this pathology in a clinic.

Study Questions

1. Pain in acute coronary syndrome (ACS), myocardial infarction (MI) and unstable angina (UA). Differential diagnosis, treatment of MI, outpatient rehabilitation.

2. Features of pain in noncoronary heart disease (dry pericarditis, myocardial, myocarditis, dilated and hypertrophic cardiomyopathy), with somatoform dysfunction of the autonomic nervous system (neurodystonia), mitral valve prolapse.

3. Features of pain in the chest caused by diseases of the abdominal organs, lungs and pleura, peripheral nervous system and muscles of the shoulder girdle (intercostal neuralgia, osteochondrosis of the cervical-thoracic segment of spine, shingles). Differential diagnosis with heart disease.

4. Diagnostic search algorithm. Differential diagnosis.

5. General principles of treatment and examination of patients with noncoronary heart disease in an outpatient setting. Indications for hospitalization.

6. Medical and social assessment in MI and noncoronary heart disease (justification and timing of temporary disability, the indications for rational employment of patients sent for MREB), dispanserization.

7. Primary and secondary prevention of noncoronary heart disease.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.
3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

The term **acute coronary syndrome** was proposed in New Zealand by clinician Nasbiz, 1996-1997. In clinical practice, the term ACS began to accept in the end of XX century.

The term ACS include new group of clinical symptoms or terms, allowing suspected acute myocardial infarction, or UA. ACS includes MI with ST elevation and without lifting ST; infarction diagnosed to modify the content of enzymes, biomarkers, late electrocardiografic signs and UA.

Unstable angina - is when angina or discomfort behind the breastbone, like angina occurs at rest or with minimal exertion and lasts longer than 10 minutes, and along with that the patient has expressed pain behind the breastbone with a tendency to increased as the duration, and intensity. In this case the boundary between the UA and MI without ST segment elevation are erased. MI without ST elevation differs from NA increased levels of markers of myocardial necrosis, which are absent in UA, since UA acute myocardial ischemia insufficient for the development of necrosis. For quick distinction without ST elevation MI and UA requires determination of cardiac troponin, but it does not apply in all hospitals, so the terms of MI without ST elevation and UA are used interchangeably.

Unstable angina has some features of pain: a sudden and persistent decrease in physical activity, in which there is pain, an increase in frequency, severity and duration of pain, the occurrence of angina at rest or nocturnal angina, a new localization of the pain radiating to the appearance of new symptoms, such as sweating, nausea, palpitations and shortness of breath. Nitroglycerin, limitation of physical activity is effective enough, there is a decrease in nitrate tolerance.

The diagnosis of ACS based on the data history if the patient uninterrupted chest discomfort lasting more than 20 minutes, not cropped nitroglycerin. In young adults (25-40 years) and elderly (over 75 years) ACS can occur atypically. The pain may be localized in the epigastrium, hands, and wrists, radiate to the shoulders, neck and jaw. In many patients, there are signs of activation of the autonomic nervous system - pallor, cold sweat. Can shortness of breath, unexplained weakness, dizziness, sweating, fainting. Often there is painless options ACS. Differential diagnosis of pain attack, accompanied by typical ECG changes is straightforward. Hardship cases are initially with treason ECG (bundle-branch block, myocardial infarction, aneurysm). Diseases, which require differential diagnosis, include pericarditis, an inflammatory disease of the lungs and pleura, pneumothorax, pulmonary embolism, musculoskeletal chest disease, aortic aneurysm, esophageal disease, sometimes psychopathic state. The purpose of the physical examination is to exclude extracardiac causes pain, heart disease, non-ischemic origin.

Thus, acute coronary syndrome implies a period of illness in which there is a high risk of damage or infarction. Introduction of the term acute coronary syndrome is necessary, as these patients require not only more careful observation, but also the rapid determination of treatment.

Disease course and prognosis largely depend on several factors: the volume of lesions, presence of aggravating factors such as diabetes, hypertension, heart failure, advanced age, and to a large extent on the speed and completeness of care. Therefore, in cases of suspected ACS, treatment should begin in the prehospital.

Medical Assistance in outpatient:

• to ensure compliance with bed rest;

• give nitroglycerin 0.5 mg sublingually, nitroglycerin spray or, if necessary - 2-3 times with an interval of 3-5 min.;

• if there are opportunities for the i/v Nitroglycerin 10 mg/min.;

• no effect and remains severe pain: morphine i/v 1ml 1% solution diluted in 10 ml physiological solution.First inject 5ml. Further optionally at least 2 ml of 5 min. until complete elimination of pain or until side effects appears (not recommended in older age groups, especially chronic diseases of the respiratory system);

• control of heart rate, blood pressure, ECG;

• acetylsalicylic acid 325 mg to chew;

• subject to availability propranolol 80 mg or 50 mg metoprolol (if the patient is not taking beta-blockers now), captopril 25-50 mg (with arterial hypertension), atorvastatin 40 mg.;

• provide emergency hospitalization in the cardiologicaldepartment, or ICU, bypassing the emergency room.

Cardiomyopathy (ICD-10 - I42) - a group of cardiac muscle injury of unknown etiology in which important features are cardiomyopathy and heart failure.

Depending on the pathologic mechanism leading expert working group of WHO and the International Society of Cardiology (1996) proposed the following classification of cardiomyopathies:

1. Dilated;

2. Hypertrophic;

3. Restrictive;

4. Specific (with known etiology and pathogenesis, which are part of systemic disease);

5. Arrhythmogenic right ventricular cardiomyopathy;

6. Unclassified cardiomyopathy.

Dilated congestive cardiomyopathy (ICD-10 -I42.0) - primary heart muscle disease of unknown etiology, characterized by dilation of the left or both ventricles with a decrease in myocardial contractility. Dilated cardiomyopathy - the most common form of cardiomyopathy. Equally common among men and women at any age, but is more common in persons 30-40 years. Is a severe disease with a high rate of disability among people of working age.

In recent years, dilated cardiomyopathy is treated with a genetic determinism positions and autoimmune effect, as well as chronic viral infection. There is evidence of viral myocarditis and the relationship of dilated cardiomyopathy. Advances in molecular genetics have allowed to identify the genes responsible for the development of this disease. Third of idiopathic cardiomyopathy are familial. The role of autoimmune mechanisms supported by the presence in some patients with cardiac organ specific auto-antibodies.

The clinical picture. The first clinical manifestation of dilated cardiomyopathy is dyspnea on slight exertion and at rest. Shortness of breath may be isolated, but more often simultaneously with the general weakness, palpitations, heaviness in the right upper quadrant, swelling of the feet. These symptoms may appear suddenly and may gradually grow and grow. In some cases, the disease can be manifested by pain in the heart, resembling picture angina or acute myocardial infarction. Cause of the pain is to increase the size of the heart, leading to relative coronary insufficiency, and narrowing of a large number of small arteries, leading to insufficient blood circulation infarction. To the fore in 75-85 % of patients are the clinical manifestations of heart failure, with 90% - 3 and 4 dysfunctional classes. Patients Liver enlargement, swelling of the legs, stagnant rales in the lungs, swelling of the neck veins. Percussion borders define the extension of relative cardiac dullness. Cardiomyopathy caused moderate hypertrophy and significantly more pronounced dilatation of the myocardium. Auscultation of heart sounds characteristic of deafness and gallop rhythm, which is best determined with the patient on the left side after exercise. Not being pathognomic sign gallop indicates severe heart muscle injury. At the top or on the bottom edge of the sternum almost always auscultated systolic murmur, diastolic sometimes on top. Most patients have tachycardia, cardiac arrhythmias. In rare cases, it may be due to transient bradycardia or persistent cross-blockade.

Diagnosis of dilated cardiomyopathy based on anamnestic data, clinical presentation, functional and instrumental examination of patients.

Electrocardiography often reveals abnormal heart rhythm and intraventricular conduction and atrioventricularconduction. In this disease can occur virtually all types of arrhythmias and conduction disturbances. In addition, to detect signs of atrial overload (expansion, splitting P wave), and the defeat of the ventricular myocardium, or to create the impression of extensive myocardial infarction (offset ST, T wave change, pathological tooth Q, deformation of QRS). Cause of these changes are dystrophic, but more focal or diffuse myocardial fibrosis.

Great diagnostic importance of echocardiography. With its help reveal dilatation of the heart chambers of varying severity. Violation contractile with a pronounced decrease in left ventricular ejection fraction phenomenon must sign echocardiography dilated cardiomyopathy. Fluoroscopic study of the chest reveals cardiomegaly, while its shape can be varied. Changes in laboratory and biochemical studies are not specific to this disease.

Course and outcome of dilated cardiomyopathy characterized by significant variability, but most unfavorable. After the onset of clinical symptoms, irrespective of the type of flow and medical therapy patients die at different times (from 2-4 months to 2-8 years). The most frequent causes of death are congestive circulatory failure, and thromboembolic complications in the lungs, brain and terminal arrhythmias.

Treatment is aimed at eliminating or reducing clinical symptoms, improves quality of life and its duration. ACE inhibitors are required in patients with dilated cardiomyopathy. Their application improves functional classes of heart failure, reducing the need for hospitalization, enhance quality of life.

Diuretics during phenomena in small and large circulation are first-line drugs. Appointed are thiazide and loop diuretics. β-blockers are shown in stable heart failure. Cardiac glycosides prescribed for atrial fibrillation, especially tachiform. Amiodarone is the drug of choice of antiarrhythmic drugs. With prophylactic and therapeutic purposes prescribed anticoagulants and antiplatelet drugs.

Examination of disability. Patients with dilated cardiomyopathy is contraindicated in heavy physical work, work in night shifts, travel, etc. in the progression of disease in patients with heart failure 2 and 3 functional classes. Referral to MREB to establish the degree of disability. Observed patients with dilated cardiomyopathy in the districtphysician, if necessary consulting cardiologist, with a multiplicity of observations 4 times a year, ECG monitoring 3-4 times a year, Echo - 1 time per year. Performance criteria of clinical supervision is to maintain a relatively compensated hemodynamics, prevention of complications, reduce the need for hospitalization.

Hypertrophic cardiomyopathy (ICD-10 -I42.1) - myocardial disease characterized by symmetric or asymmetric hypertrophy of the left ventricular myocardium with mandatory involvement of the interventricular septum. The true prevalence is difficult to judge, with the advent of echocardiography detection increased significantly. Occurs at any age, usually 30-40 years, men are affected twice as often as women. Found that when examining patients with hypertrophic cardiomyopathy relatives, 17-60 % has also revealed this pathology.

In the occurrence of hypertrophic cardiomyopathy proved the role of genetic factors - a violation of myocardial contractile elements of differentiation in the embryonic period.

The clinical picture is diverse and nonspecific. Often patients complain of pain in the chest cardialgiaor stenokardia like, dyspnea on exertion or at rest, irregular heartbeat, seizures, dizziness, fainting. In some cases, the disease is asymptomatic. Pain in the heart usually stabbing, aching prolonged nature, not related to physical activity, as a right of passage on their own. The occurrence of this pain associated with myocardial hypoxia caused by diastolic dysfunction and left ventricular hypertrophy, which leads to increased intramyocardial tension, compression of the coronary arteries and reduced coronary blood flow. Fainting and dizziness during exercise more frequently observed in subaortic obstruction. Exertional dyspnea due to venous congestion in the lungs as a consequence of diastolic dysfunction of the hypertrophied left ventricle.

Diagnosis is based on clinical data and the objective and instrumental studies.

ECG recorded at different changes, but strictly no specific signs. More frequent changes in ST interval in the form of depression, negative T waves, and signs of left ventricular hypertrophy, deep Q waves and signs of hypertrophy of the left atrial arrhythmias. The nature and extent of ECG changes did not correlate with the severity of clinical manifestations.

Differential diagnosis must be done with CHD, heart diseases, hypertensive heart.

Course and outcome of hypertrophic cardiomyopathy can be divided into five options:

1. stable benign

2. progressive course

3. sudden death (observed in 20-50 % of patients)

4. atrial fibrillation and heart complications

5. progression of congestive heart failure due to remodeling and left ventricular systolic dysfunction

Treatment of patients with hypertrophic cardiomyopathy is aimed at reducing the severity of symptoms, hemodynamic improvement, slowing the progression of the disease, prevention of complications and prolong life in these patients. The choice of drugs depends on the presence or absence of obstruction in a patient output of the left ventricle and the prevalence of symptoms.

Of medicinal drugs used mainly β-blockers without intrinsic symptomatic activity (propranolol, metoprolol, bisoprolol at maximum tolerated doses).

Second-line drugs used to treat hypertrophic cardiomyopathy are non-dihydropyridine calcium antagonists that have negative inotropic and chronotropic action of the group and verapamil diltiazem.

Celebrating sustained improvement - reduction of anginal pain, shortness of breath on exertion. But these drugs do not prevent sudden death and do not improve the prognosis.

To prevent ventricular and supraventricular arrhythmias is a highly effective means of amiodarone. Thanks to the antiarrhythmic activity it is able to improve the prognosis of the disease and prevent sudden death.

The combination of amiodarone and verapamil is contraindicated because of the risk of bradycardia, conduction abnormalities and pronounced negative effect.

When a permanent form of atrial fibrillation adequate ventricular rate can be achieved with the appointment of propranolol or verapamil with digoxin. Because atrial fibrillation are at high risk trombembaly necessary appointment these patients antiplatelet (aspirin) and anticoagulant (warfarin) therapy.

With the development of heart failure are assigned β-blockers or calcium channel blockers in combination with low doses of diuretics. In the advanced stage of the disease in the development of cardiac decompensation patients received therapy with diuretics and ACE-inhibitors, cardiac glycosides, along with β-blockers or verapamil.

Surgical treatment is indicated in patients with severe obstruction of the left ventricular outflow tract.

Examination of disability is solved individually for each patient, the period of temporary disability from 7 to 35 days. In the early period of hypertrophic cardiomyopathy shown employment patients. Labor associated with increased risk (working at height, drivers, etc.) is contraindicated. In case of severe complications the patient is directed to the MREB to determine the degree of disability.

Dispanserization - provides local therapist with mandatory inspection cardiologist. Complete clinical and instrumental examination is held twice a year. Observed for life.

Prevention includes conducting effective clinical examination, identification and examination of persons with a history of sudden death in the immediate family.

Disease of the lungs and pleura. Discomfort and chest pain can be caused by diseases of the lung and pleura. Bronchopulmonary pathology pain caused defeat of the parietal pleura or bronchial mucosa and trachea. Stabbing pain usually, sometimes aching, dull, strongly associated with coughing, breathing. In this case the patient may experience shortness of breath. Sputum, hemoptysis. In the differential diagnosis are important medical history, especially of percussion, auscultation, laboratory and radiological data.

Pneumonia is characterized by acute onset, accompanied by fever or hypothermia after suffering ARI. Chest pain is aggravated by breathing and coughing. Marked expectoration. When percussion shortening of percussion sounds on the affected side, auscultation - depending on the stage of the disease respiratory depression, rales, pleural friction rub. In blood leukocytosis, accelerated ESR, radiological - infiltration of the lung tissue.

Dry pleurisy is characterized by pain in the chest that is ameliorated by a healthy side, pleural friction noise, fever, leukocytosis with a left shift, increased ESR.

Spontaneous pneumothorax is characterized by sudden intense pain in the chest, cyanosis, tachycardia, usually after paroxysmal cough, possible subcutaneous emphysema around the neck. Lesion over the area determined thympanitis percussion, auscultation - breathing weakened or does not listen. X-ray examination shows extensive enlightenment in the affected side, mediastinal shift to the opposite side.

When lung cancer is of great importance bronchoscopy and biopsy and sputum cytology or pleural fluid.

Diseases of the gastrointestinal tract. Cause of chest pain may be abdominal disease: esophagitis, esophageal spasm, reflux esophagitis, gastric ulcer and duodenal ulcer, cholecystitis, pancreatitis, cholelithiasis. Pain in diseases of the esophagus is similar to anginal on its localization and paroxysmal character. There are strong, are burning in nature, sometimes irradiate to the neck, interscapular region, often cropped nitroglycerin. But they are not related to physical activity, usually associated with eating, combined with dysphagia, heartburn, belching, nausea. Pain at gastroduodenal ulcers, cholecystitis, and pancreatitis, also combined with dyspeptic symptoms, more often localized in the lower third of the sternum and epigastric pain,

associated with eating. On the ECG signs of myocardial ischemia are absent. Endoscopic and radiological methods of investigation can detect signs of disease of the gastrointestinal tract.

Diseases of the nervous system and muscles of the shoulder girdle. Chest pain in the heart and often causes osteochondrosis of cervical-thoracic spine. Compression of the nerve roots at the level of the cervical and thoracic vertebrae leads to radicular syndrome, manifested by pain in the chest, arms, shoulders. Neck pain may be accompanied by dizziness, autonomic, auditory, vestibular symptoms. Matters identify pain with pressure on the spinous processes of the cervical vertebrae.

Intercostal neuralgia may be due to various reasons - injuries, broken ribs, intercostal nerve compression. At the same time the pain increases with movement, torso in a sick way, there is a corresponding hyposthenia intercostal space. The defeat of the intercostal nerves at shingles, accompanied by subsequent blistered rash on the skin, it may begin with the appearance of burning and stabbing pain. Often proceeded by itching and fever.

If the patient has trouble or pain in the chest wall termed "false angina" and should call a doctor alertness. These sensations may be perceived as a feeling of tightness, pressure, burning, fullness, or other causes discomfort to the patient. Pain in the heart characterized by many, including non-cardiac diseases. In this regard, regular and timely diagnosis will help determine the proper management of patients.

Affections of the pericardium (ICD-10 -I30) also may be accompanied by pain, since its parietal surface has the nerve fibers within the system of the phrenic nerve. Pain dull, aching, sharp, cutting may irradirovat in the shoulder, neck, trapezius muscle, sometimes in the right upper quadrant. Often it depends on the act of breathing, body position, increased by coughing, deep breathing, decreases in the sitting position, bending forward.

Clinical manifestations of pericarditis usually is noise, pericardial friction. On ECG characteristic concordant ST-segment elevation and T wave in standard leads, reinforced limb and in most infants. When pericardial effusion, pain is less pronounced, there is heaviness in the heart, shortness of breath. Auscultation determined voiceless heart tones, the ECG - reducing the voltage of all teeth. Echocardiography confirms the diagnosis at which revealed extra fluid in the pericardial cavity.

Myocarditis (ICD-10 -I40) - an inflammation of the heart muscle, in which violated his basic functions: excitability, conduction and contractility. Myocarditis may result from exposure to infections, physical and chemical agents in allergic and autoimmune diseases. The first symptoms of myocarditis begin to appear within 7-10 days from the onset. Complaints varied and nonspecific. Initially appears asthenic syndrome: emotional lability, vegetative disorders, sleep disorders. Then there is the pain in the hearts of different duration and intensity is not related to physical activity. Pain syndrome is often preceded by shortness of breath. The survey can be observed muted tones of the heart, systolic murmur at the apex, is not associated with one voice, the intensity of which does not change with a change in body position. Important role in the diagnosis takes ECG study. Often, changes are detected in the final part of the ventricular complex: segment elevation of RS-T, asymmetrical T waves with reduced amplitude. Also on the ECG changes are possible accompanying arrhythmias (tachycardia, extrasystoles, atrial fibrillation) and conduction (bundle-branch block). Changes in echocardiography revealed only

in severe cases of the disease. Laboratory and biochemical studies show an increase in ESR, C-reactive protein, LDH, CPK.

Myocardiodisrtophy (ICD-10 -I43) - noninflammatory myocardial damage, characterized by degeneration of contractile cardiac muscle cells, cardiac conduction system structures and violations of the basic symptoms of heart function (automatism, excitability, conduction, contractility). Myocardiodystrophy reasons varied: endocrine disorders, and disorders of all kinds of metabolic poisoning by industrial and household poisons, chronic alcohol poisoning, cardiovascular diseases (hypertension, ischemic heart disease, etc.). Myocardiodystrophy manifest themselves in different ways. The pathological clinical symptoms myocardiodistrophy exists. Often the disease progresses over several years, gradually progressing. Patients complain of pain in the heart, which increases with stress and exertion, palpitations, shortness of breath, fatigue, decreased performance. On the ECG signs of myocardial dystrophy may occur in slowing intraatrial conduction lengthening the interval QT, reducing the duration and amplitude of the T wave, reducing the voltage of all teeth. Sometimes identified violations intaventrical conductivity arrythmia. Laboratory parameters: total blood count, C-reactive protein, sialic acid.

Mitral valve prolapse - a very common pathology, the essence of which is that the mitral valve during systole ventricular bend toward the atrium and allow blood to flow out of the ventricle to the atrium. The reason for this is not clear until the end. Suggest that it is based on hereditary deficiency of collagen type 3. Patients complain of pain in the heart, palpitations, arrhythmias. Prolonged pain, aching or pressing, usually localized in the 3-4 intercostal space left of the sternum, sometimes behind the breastbone, irradiate in the left arm and shoulder blade, is not stoppedby nitroglycerin. Patients are fainting, headaches, asthenia, and emotional lability. On echocardiography revealed sagging of one or both leaflets into the left atrium during systole.

Neurocirculatory dystonia (cardiopsychoneurosis, NCD) or dysfunction of the autonomic nervous system. By definition, of Professor VI Makolkin NCD is polyethiologic disease, the main features of which are the lability of the pulse and blood pressure, false angina, respiratory discomfort, vegetative and psycho-emotional disorders, vascular and muscle tone, low tolerance to stressful situations in benign course and good prognosis for life. Patients with this disease are 25-30 % of visitors to the district physician. Pain when NCD is a different character from the intensity, duration, and sensations. Pain may be aching squeezing , burning , sometimes accompanied by shortness of breath, palpitations, sweating, anxiety, do not go away after taking nitroglycerin, but stoped sedatives in combination with α-blockers.

Primary prevention noncoronary heart disease:

• the risk factors;
• if there is family history of a rational approach to the choice of profession and employment;
• adequate treatment of acute and chronic infections.

Secondary prevention includes timely, adequate treatment of diseases, aimed at improving prognosis.

Class 3 Articular syndrome: differential diagnosis. Diagnosis and treatment of inflammatory diseases (rheumatoid arthritis, reactive arthritis) and degenerative (primary

osteoarthritis) joint disease at outpatient setting, medical tactic, medical and social assessment, dispanserization, primary prevention.

Session Purpose: To teach students the diagnosis, principles of rational therapy, disability examination, dispanserization and prevention of patients with inflammatory diseases (rheumatoid arthritis, reactive arthritis) and degenerative (primary osteoarthritis) joint diseases in the outpatient setting.

Study Questions

1. Articular syndrome: the notion of arthritis, arthrosis, arthropathy. Methods of general and special physical examination of the joints and spine.

2. Major diseases accompanied articular syndrome. Diagnostic search algorithm with articular syndrome.

3. Rheumatoid arthritis: survey design, diagnostic criteria. Medical tactic.

4. General principles of outpatient treatment, indications for hospitalization.

5. Medical and social assessment (justification and timing of temporary disability, the indications for rational employment of patients sent for MREB), dispanserization.

6. Reactive arthritis: a definition, survey, differential diagnosis. General principles of treatment on an outpatient basis, medical and social assessment, dispanserization.

7. Primary osteoarthritis: diagnostic criteria, survey the patient, outpatient treatment, medical and social assessment (direction to MREB), dispanserization.

8. Prevention of inflammatory joint disease and primary osteoarthrosis.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.
3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

Articular syndrome (AS) - a combination of several signs and symptoms of major lesions (articular surface, cavity, bag, cartilage) and auxiliary (ligaments, discs or menisci) formations joints and periarticular tissues (bursa, tendon, ligaments, muscles, fascia, etc.), manifested by pain and soreness, stiffness in the joints, change in shape and impaired joint function.

By the nature of the pathological process the following types of joint damage:

- arthritis - inflammation in all tissues of the joint (subchondral bone department, articular cartilage and synovium) or at least one of them. Only inflammation of the synovial membrane (its thickening and/or effusion in the joint cavity) is referred to as synovitis;

- osteoarthritis (arthrosis) - primary or secondary degenerative-dystrophic lesions of the articular cartilage and underlying bone. Secondary inflammation of the synovium (synovitis jet) on the background of osteoarthritis called osteartritom;
- periarthritis - inflammatory and degenerative diseases of the periarticular tissues (tendons, serous bags, capsules);
- arthralgia - the presence of joint pain not associated with structural changes in the tissues of this joint;
- arthropathy - a pathological process in the joints due to non-rheumatic diseases of other organs and systems, can be both inflammatory (arthritis) and degenerative-dystrophic;
- osteochondropathy (osteochondritis, osteohondrolisis, epifizonekrosis) - subchondral aseptic necrosis of the edge portion of the cancellous bone in the field carrying an increased static and functional load, leading to a change in secondary cartilage and bone deformation.

According to the number of joints involved in the three types of lesions:

♣ monoarthritis - the defeat of one of the joint;

♣ oligoarthritis - lost two or three joints;

♣ polyarthritis - defeat four or more joints.

In most cases, the data obtained allow to determine the nature of the pathological process: arthritis, osteoarthritis, loss of periarticular tissues.

Indicate the presence of arthritis:

• "inflammatory" type of pain: intense, permanent (day and night, at rest and during movement), more severe pain in the beginning of the movement;

• morning stiffness in the joints (more than 1 h), provoked by prolonged stay;

• universal stress pain (during movement in almost all planes);

• antalgic position of the joint (flexion moderate) to the exclusion of contracture;

• local signs of inflammation (hyperthermia, swelling and uniform smoothness contours of the joint);

• diffuse pain and soreness, tenderness along the joint space;

• effusion in the joint cavity (bulging symptoms, fluctuations, floating patella when the knee joint pathology);

• palpation of thickened synovium;

• a decrease of the same active and passive movements of the joint;

• limitation of all or most of the possible in this joint movements in all planes;

• uneven swelling (defiguration) of joints in chronic arthritis,in the later stages of the violation of their shape (deformation) due to destruction of epiphyses, subluxations and dislocations, the total lack of movement (ankylosis);

• clinically in most cases, fever, tiring, deterioration of general condition, increased ESR, leukocytosis and other positive acute phase reactions in the blood;

• radiographic changes typical for the particular form of arthritis (see below).

Typical indicators of osteoarthritis are:

• variable nature of pain:

• mechanical nature of pain - the same intensity throughout the movements subsided and alone at night;

• blunt continuous nocturnal pain;

• «start» pain - short-term, lasting 15-20 min pain that occurs after a period of rest and gradually decreasing to background movements, characterized by joint "blockade" ("jamming" of the joint);
• persistent pain caused by muscle spasm, as well as the development of reactive synovitis (in this case it is possible swelling of the joint, sometimes local hyperemia/hyperthermia, osteoarthritis);
• crunch in joints with active and passive movements;
• defiguration of joint by bone growths, including Heberden's and Bouchard'snodes;
• limitation of all possible joint active and passive movements;
• the absence of fever, worsening of general condition and changes in laboratory parameters of inflammation or weak expression in synovitis;
• radiographic changes: joint space narrowing, subchondral osteosclerosis, osteophytes, deformation of the articular surfaces.

Indicators of periarticular tissues affection:

• Spot or local pain and tenderness (corresponding to the anatomic location of periarticular structures);
• pain when active, not passive movements;
• selective stress pain (in one plane);
• pain in the resistive active movement in the corresponding direction;
• violation of joint mobility in a single plane;
• lack of local signs of inflammation or swelling in a limited area of the projection of the affected periarticular structures;
• reduction of active movements at the retained amount of passive movements;
• the absence of fever, worsening of general condition, normal laboratory parameters of blood;
• absence of radiological changes in the lesion.

In case of arthritis should determine its etiology and nosology. To a certain extent this is possible, given the rate of development of arthritis, involve certain joints (large or small), their number (mono-, oligo-arthritis), the availability of symmetric lesions severity and persistence of inflammatory changes characteristic of laboratory and radiological data.

The main symptom, forcing the patient to see a doctor is a pain in the joints (arthralgia). Most often it is associated with the pathological process in the joint or periarticular tissues, but may also be reflected in internal diseases. Angina pectoris, myocardial infarction pain can be localized in the shoulder, elbow, wrist, the pathology of the pelvic organs - in the sacrum, etc. Arthralgia occur in many diseases, including malignant tumors (paraneoplastic syndrome). In this connection should be carried out a detailed examination not only of the musculoskeletal system, but also the patient's general condition.

Careful analysis of pain is of great importance in the diagnosis of diseases of the joints. distinguish:

- pain movement - appear or aggravated by movement in the joint, weakening alone (mechanical overload damage to the joint, synovial injury osteophytes, stretching tendon and ligaments , etc.)

- pain at rest - worse after rest, weakened during movement (inflammation, degenerative changes in the joint, inappropriate microcirculation in patient with meteosensitivity, venous stasis)

- night or bone "pain" - occur mostly at night, with traffic not normally associated (metabolic abnormalities in bone in osteoporosis).

Stiffness - feeling the impossibility of free movement in the joint after a period of rest (morning stiffness or page). The duration and severity of her supposedly allows estimating the intensity of the inflammatory process.

Is important information about the patient: sex, age, occupation, lifestyle, previous diseases (cystitis, prostatitis, diarrhea and abdominal pain, skin rash, conjunctivitis, etc.), joint injuries, physical overload, obesity, medications, foci of chronic infection (chronic sinusitis, tonsillitis), obesity, medications, heredity (gout, osteoarthritis, systemic diseases with relatives).

Detailed physical examination of the musculoskeletal system include an external examination (at rest and during movement in the joints), palpation and assessment of joint movement.

Examination at rest reveals:

• changes in the skin;
• swelling/increase in volume;
• muscle atrophy;
• forced position;
• deformation.

Evaluation of joint movement reveals the following changes.

➢ Mobility limitation:

• in one plane;
• periarticular lesion;
• virtually all movements in the joint;
• failure of the joint.

➢ Increased range of motion :

• in the joint hypermobility, instability of the joint.

➢ Pain on movement:

• stress pain, pain at movement in the joint, aggravated as deviations from the median physiological status;
• universal stress pain (due to the motion of the joint in all or almost all directions)
• synovitis;
• electoral stress pain (arising from the movement in the same plane) - the defeat of the periarticular tissues.

Palpation together with an assessment of joint movement reveals the following changes.

➢ Soreness:

• in the projection of the joint - the defeat of individual or joint intraarticular structures entirely;
• periarticular pain - the defeat of the periarticular tissues.

➢ Increased temperature of the skin over the joint:

• inflammation (e.g., synovitis, bursitis).

➢ Edema/Increment:

• accumulation of fluid in the joint cavity (the point fluctuation);
• swelling of the periarticular tissues (soft consistency, nonfluctuating);
• an increase in bone volume ("swelling" tight to the touch)

➢ Crepitus:

• rough, easily felt by palpation, sometimes even determined by ear - damage to the joint;

• tender, localized, only listens with a stethoscope - the defeat of the synovial tendon sheath or periarticular bags.

➢ Restrictions during active movements:

• due to pain in the muscles, tendons, muscle attachment sites at the bones.

➢ Stress tests:

• provoke pain in the pathology of the ligaments and synovial sheaths of tendons.

Nonspecific signs of systemic disease may be weight loss, loss of appetite, chills and sweating, especially at night, fatigue, a general feeling of a painful condition, weakness, irritability.

In most cases, the pathology of the musculoskeletal survey and clinical examination by themselves provide sufficient information to make a diagnosis and develop a plan of treatment of the patient. Additional studies may help when in doubt in the diagnosis (differential diagnosis with other diseases involving articular syndrome), as well as in assessing the activity and the rate of disease progression. Laboratory and instrumental studies always complements the clinical examination of the patient, but never replace it.

The next step is to assess the functional status of the organs. Upon detection of pathology conduct further examination of the patient, clarify the possible connection of the identified disease articular syndrome in this patient.

In outpatient settings often conducted radiography joints. With the help of X-rays reveal changes characteristic of severe pathological processes:

• swelling of soft tissues - is visible on x-ray because of the changes the contours of the skin layer that will blend layer of adipose tissue and fat accumulation in the synovial folds (adipose tissue on x-ray looks like enlightenment);

• reduction of bone density (osteopenia) or increase bone density (osteosclerosis), which may be localized or generalised;

• increase and deformation of the bones;

• erosion of joints (nonproliferative or proliferative marginal erosion, centrally-located erosion);

• narrowing of the joint space (in a limited area of the joint space - osteoarthritis, throughout the joint space - inflammatory arthritis);

• new bone formation (osteophyts, entezofits, sindesmofits) and changes in the periosteum;

• calcification of cartilage - chondrocalcinosis, synovium, joint capsule, ligaments, tendons, muscles, adipose tissue, blood vessels, skin and intraarticular osteochondral inclusion.

Although virtually all of the pathological signs themselves have low specificity, and their various combinations, along with specific localization of the lesion can be folded into a characteristic pathological picture which has a high specificity.

Computed tomography (CT) and magnetic resonance imaging (MRI).

These studies yield three-dimensional images of complex anatomical structures, such as the spinal canal and intervertebral facet joints, which can not be adequately visualized with the aid of conventional radiography. Disadvantages of CT - limited resolution when imaging soft tissues, as well as a high dose of radiation, so in most cases nowadays prefer MRI. This research method is particularly informative when:

• early osteonecrosis;

• pathology of intervertebral discs, and compression of spinal nerve roots and spinal cord compression;

• infectious arthritis and osteomyelitis, cellulitis of soft tissue;

• malignant tumors of bones, joints and soft tissues;

• internal structural violations, such as the knee joint;

• pathology of soft tissue and periarticular tissues (e.g., early synovitis, muscle-tendon ruptures of rotary cuff of shoulder, bursitis, tendosynovitis).

Ultrasound examination. It is safe and common research helps reveal pathology of soft tissues, such as effusion of the hip joint, popliteal cysts and thickening of the Achilles tendon. Nevertheless, the limited resolution of the method puts it below the CT or MRI in the evaluation of informative anatomical changes.

Laboratory Methods: total blood count, urinalysis, defined acute phase reactant (C-reactive protein), total protein fraction of autoantibodies (rheumatoid factor, Antibody to Cyclic Citrullinated Peptide - ACCP)**,** uric acid, urea, creatinine. If you suspect a microcrystalline (gout, pyrophosphate arthropathy) and infectious arthritis, bleeding into the joint cavity - is performed arthrocentesis and synovial fluid study.

In the hospital at the moment with the aim of visual assessment of intra-articular cartilage and synovial membrane (usually knee) for the differential diagnosis of diseases of the joints arthroscopy is performed, and if necessary, biopsy.

Differential diagnosis with arthralgia. Arthralgia as a form of joint syndrome characterized by the absence of any external signs of joint damage (swelling, deformity, flushing, hyperthermia), palpation pain, and changes detected by X-ray and instrumental studies. In such cases, only a thorough questioning of the patient about the nature and type of pain, palpation of the joints, check active and passive movements allow you to make sure that the pain is coming from the joint or surrounding soft tissues, bone diaphysis, passing near the peripheral nerves and blood vessels or even distant sources (referred pain). Contact your local (zonal) joint pain only with certain movements typical for the defeat of the periarticular tissue, muscles, fascia, tendons, peripheral nerves or the central nervous system. Persistent pain fuzzy localization (neuropathic pain), accompanied by paresthesias or sinestopathy are characteristic of damage to the peripheral nervous system.

Transient and persistent polyarthralgia most often the result does not actually defeat the joints, as one of the symptoms of other diseases. An example can be polyarthralgia with acute infectious diseases (angina, influenza, SARS, hepatitis, etc.), foci of chronic infection (tonsillitis, sinusitis, adnexitis, cholecystitis, etc.), endocrinopathy (diabetes mellitus, hyperthyroidism, etc.) and as a consequence toxic- allergic reactions in the body.

Polyarthralgia combined with morning stiffness less than 30 minutes, flexion contractures mostly joints of the hands, swelling of the skin tight fingers (scleroderma) and acroscleroderma symptom friction tendons, Raynaud's phenomenon, digital ulcers and acroosteolysis, erosive arthropathy in the absence of arthritis characterized by systemic sclerosis.

Polyarthralgia in Lyme borreliosishas characteristic combination with pain in the tendons and periarticular tissues ossalgiya and myalgia.

Long-existing and persistent monoarthralgia predetermines thorough visualization of various anatomical structures in the area of pain, especially bone (the effects of trauma, manifestations of paraneoplastic process, etc.).

In a patient with arthralgias ambiguous nature in the knee should include the possibility of irradiation of pain from the affected hip (mostly due to ischemic necrosis of the femoral head).

Pain in the elbow and knee joints is typical of diffuse fasciitis and pseudoartralgia shoulder and hip joints - for polymyalgia rheumatica.

Radiating pain in the shoulder joint can be caused by osteochondrosis of the cervical spine, pleural affection, coronary artery disease (angina, myocardial infarction), gallbladder disease, lung apex tumor (Pancoast tumor).

Arthralgia is frequently observed in violation of statics.

Osteoarthritis (ICD-10 M15-M19)

Osteoarthritis (OA) - a heterogeneous group of diseases of the joints with different etiology, but with similar clinical morfobiologicalsigns and outcomes, leading to the loss of articular cartilage and involvement in the process of subchondral bone, synovium, ligaments, capsules and periarticular tissues.

The problem of rheumatic diseases is relevant to the world's population, due to the prevalence and diversity of this group of diseases. The relevance of the problem suggests the WHO declaration of the first decade of the XXI century Bone and Joint Decade.

10-12 % of the global population has OA. Disease correlates with age often develops between the ages of 30-35 years and those aged 60 years and older found in 97 % of patients. In women, the disease is more common than in men.

Etiology. OA - multifactorial chronic, progressive disease of the joints, the etiology of which is unknown to date. Allocate the following risk factors for this disease.

1. Genetic: predominantly female, inherited disorders of collagen type II (Stickler syndrome), gene mutation of type II collagen (COL 2 A1), congenital malformations (hypermobility, dysplasia of bones and joints), varus/valgus knee, flatfoot,ethnic affiliation. It is known that if the mother is nodular (menopausal) form of OA, in which lesions are localized in the distal (Heberden's nodes) and proximal (Bouchard nodules) interphalangeal joints, the risk of such changes her daughter is doubled. This is explained by an autosomal dominant inheritance of this disease in women and recessive in men.

2. Nongenetic: age over 45 years, overweight, obesity, low levels of female sex hormones, postmenopause, vitamin D_3, surgery on the joints, diseases of the joints (arthritis), endocrine diseases (diabetes, hyperparathyroidism, etc.), atherosclerotic changes in the vascular bed of the joint and the like;

3. Environmental factors: physical stress on joints, professional load (work standing, exercise, etc.), joint injuries, torn ligaments or meniscus, etc.

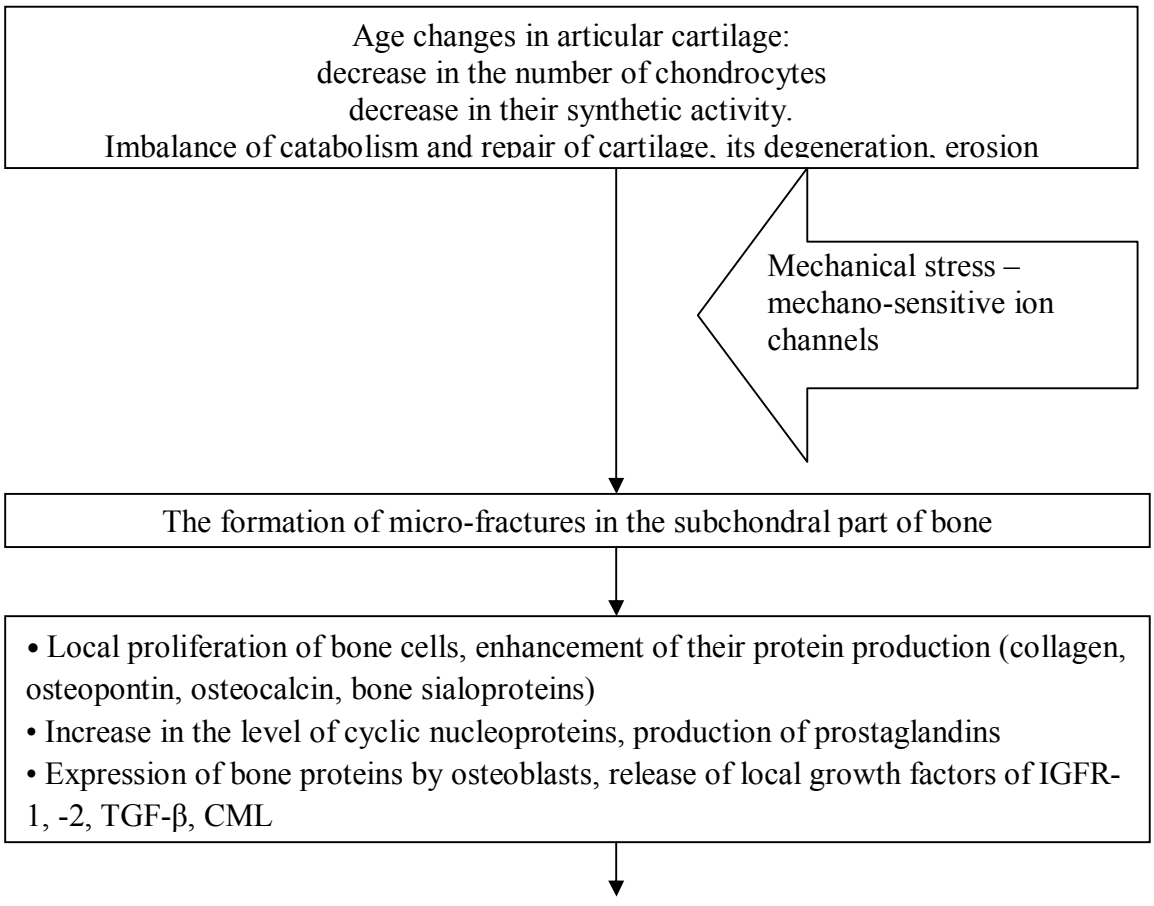
The pathogenesis of OA. The basis of the pathogenesis of OA is a metabolic disorder in the cartilage tissue with a predominance of catabolic processes over anabolic. Underlying mechanisms causing loss of cartilage are considered violations in exchange proteoglycans that with chondrocytes form cartilage. OA occurs when the loss of proteoglycan matrix

componentparts - the surface of the glycosaminoglycans, of the intermediate and deep zones of cartilage, leading to excess hydration, pulping and splitting of the matrix, followed by dehydration, alteration of diffusion of metabolites and disorganized collagen fiber rupture. Violated the basic function of cartilage, providing regulation of the synthesis and degradation of cartilage matrix components and amortization joint function. Chondrocyte has considerable sensitivity to the content of proteoglycans in the surrounding matrix and reacts quickly to their quantitative and qualitative changes. In the development of OA is not the last role to play and persistent inflammation in the tissues of the joint.

Damaged chondrocyte secretescatabolic stress enzymes that destroy the collagen (elastase, collagenase, peptidase, etc.) and proteoglycans (metalloproteinase, stromelysin, cathepsin, interleukin-1, tumor necrosis factor, etc.). Important in this case is an increase in the production of cytokines that stimulate the synthesis of proteases, plasminogen activator prostaglandin E2 and contribute , on the one hand, proliferation of synovial cells, on the other - inhibit collagen synthesis by chondrocytes and proteoglycans.

Among other possible factors in the pathogenesis of OA immune mechanisms are discussed, as cartilage has antigenic determinants, poor circulation in the joint tissues, genetic predisposition, when possible devolution of congenital skeletal anomalies, weakness of tendon and ligaments and other disturbances, leading to changes in the articular congruence surfaces and development of OA. Hereditary transmission of imperfect synthesis of intermediate substance of cartilage is nit excluded. One possible cause of OA is considered insufficient synthesis of hyaluronic acid, which are fixed proteoglycans.

Play important role age-related changes of the articular cartilage.



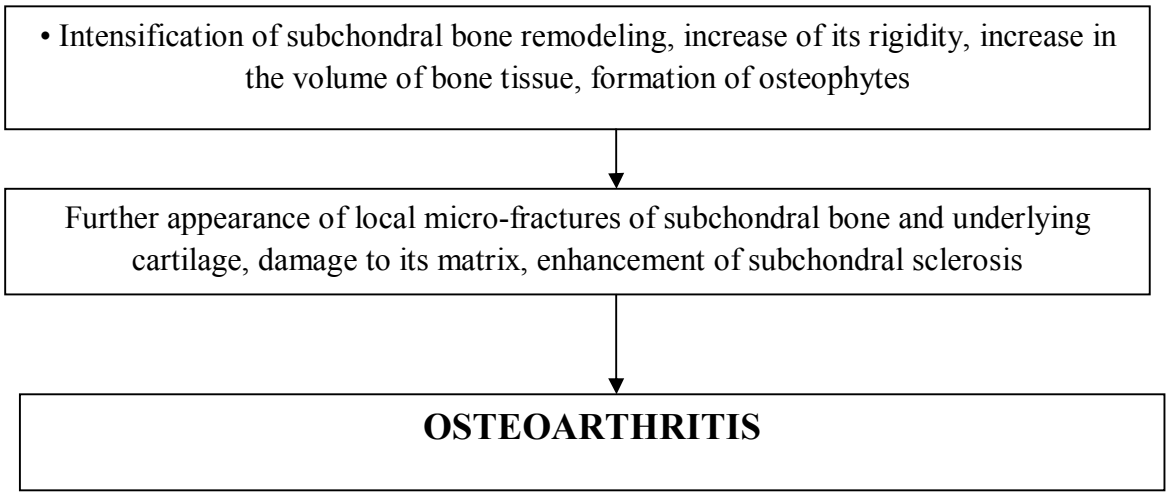


Fig. 3.1The pathogenesis of OA.

Clinical classification(Altman R. et al.,1986)

Primary (idiopathic) OA:

• Localized: joints of hands, joints, feet, knees, hips, spine and other joints;

• Generalized: the defeat of three or more different joints.

Secondary OA.OA develops because of number of reasons.

• Post-traumatic;

• Congenital, acquired or endemic disease (Perthes disease, hypermobility syndrome, etc.);

• Metabolic disease: ochronosis, hemochromatosis, Wilson's disease, Gaucher's disease;

• endocrinopathies: diabetes, acromegaly, hyperparathyroidism, hypothyroidism;

• Disease calcium deposits (calcium phosphate, calcium hydroxyapatite);

• Neuropathy (Lou Gehrig's disease).

Other diseases: avascular necrosis, RA, Paget's disease.

To determine the radiographic OA stage is classification system Kellgren JH, Lawrence JS. This classification is based on an assessment of the severity of symptoms such as joint space narrowing and the presence of osteophytes.

Radiographic stage of OA (system Kellgren JH, Lawrence JS, 1957)

0 - absence of radiographic abnormalities (normal joint space, no osteophytes).

I - doubtful radiological signs (single osteophytes, joint space is not changed).

II. - Minimal radiographic signs (single osteophytes, slight narrowing of the joint space).

III - moderate radiographic changes (multiple osteophytes, moderate joint space narrowing).

IV - pronounced OA (joint space can not be traced, subchondral bone sclerosis, osteophytes rough).

Clinic and diagnostics

The main clinical manifestations of OA are joint pain and limit their mobility. When the diagnosis of OA should be considered the main symptoms associated with joint damage.

The main symptoms associated with OA:

1. Gradual onset of pain.
2. Increased pain in a standing position or a decrease in load and at rest.
3. Morning stiffness lasting less than 30 minutes.
4. Deformation (Heberden’s, Bouchard’s nodules)
5. Restriction of active and passive movements of the joint.
6. Crepitus with active motion of the joint.
7. Atrophy of surrounding muscles.

OA of the shoulder and elbow, as well as the first carpometacarpal, acromioclavicular, are not as common, and a limited number of patients, whereas the first metatarsophalangeal-phalangeal joint is actively involved in the process.

Generalized OA is also called Kellgren'ssyndrome. It is diagnosed when a patient reveals typical for this form of joint disease related to at least four different groups, and is defined as more severe downstream.

It is believed that in this case there is a defect in patients amino acid composition of collagen type II and IX, which defines a more rapid destruction of cartilage.

Currently, special emphasis nodular generalized OA (menopausal), in which the lesions are localized in the distal (Heberden's nodules) and proximal (Bouchard nodules) interphalangeal joints. The reason for this separation is genetically determined predisposition, inherited through the female line. And if the mother has nodular form of OA, the risk of such changes in her daughter increased two times. The inflammatory-erosive OA affects the distal and proximal interphalangeal joints, I carpometacarpal joints of hands and I metatarsus-phalangeal joints of the feet. Inflammatory changes dominate degenerative. Exacerbation of articular syndrome is not accompanied by general symptoms, not marked acceleration of ESR changes and other indicators of inflammation. This form of primary OA mostly occurs in women older than 50 years. In the process, as a rule, are not involved metacarpophalangeal, wrist and II-V metatarsus-phalangeal joints.

Clinically significant and the most disabling forms of arthritis are loading OA joints: the knee (gonarthrosis) and hip (coxarthrosis), which can occur in adults older people, representing a partial manifestation of the general phenomenon of aging, accelerated by various factors.

Gonarthrosis (ICD-10 -M17) - is the most common form of osteoarthritis of the limbs (75%). It affects women more often, since preklimakticheskogo period (after 40-50 years), and the process is bilateral. Symptoms of the disease were observed in 50 % of those over 45 years and 100% in 70 years.

Gonarthrosis proceeds more favorably than coxarthrosis, long and often asymptomatic. Stifle often injured (with the possible development of chondromalacia patellae).

Symptoms characteristic for gonarthrosis. Report pain of mechanical nature, its severity increases when descending the stairs to the end of the day. The pain may be localized around the joint, irradirovat shin and hamstring. After prolonged sitting patients stand with difficulty. Characterized by a sudden sharp pain that causes joint blockade "loose body".

Symptoms associated with knee OA revealed by physical examination:

• limitation of flexion and extension;

• crepitus on motion;

• moderate or small effusion, is not accompanied by an increase in local body temperature (determined by ultrasound examination of the joints);

• joint tenderness and/or periarticular regions;

• joint deformity: varus (less valgus) and/or a fixed flexion in the joint;

• overgrowth of bone tissue at the edges of the articular surfaces.

Coxarthrosis (ICD-10 -M16)

Osteoarthritis of the hip is more common in men, and in the initial stage of the disease, lesions often develop on one side of the body. The joint on the opposite side will eventually involved in the process 20% of patients. It is estimated that 80 % of this disease are secondary to congenital disorders or hidden congenital malformations joint. Progressive course of the disease, often leading to disability of the patient.

Symptoms associated with hip OA.

Features pain in coxarthrosis:

• pain is most pronounced in the groin , may radiate to the buttocks, on the front and side of the thigh, knee, shin;

• reflected from hip pain may be localized only in the knee, but unlike the pain of knee OA, it is diffuse, decreases after rubbing, it does not provoke movement in the knee and hip

joint;

• pain on the lateral surface of the hip joint, aggravated by lying on the side, accompanied by tenderness to palpation of the greater trochanter indicates secondary trochanter bursitis.

Symptoms associated with OA of the hip joints, physical examination revealed:

• limitation of internal rotation in the flexed joint (early and most sensitive sign of hip OA), other users may also be limited and painful;

• tenderness groin lateral femoral artery pulsation;

• fixed flexion and/or external rotation of the joint, shortening the legs.

Osteoarthritis of the interphalangeal joints of the fingers (nodular generalized, menopausal OA). This is the most frequent manifestation polyosteoarthrosis, develops mainly in women in menopause and post menopause can be observed in COPD patients , the disease can sometimes make his debut on the 3rd decade of life.

Diagnosis of osteoarthritis:

Diagnostic process begins with asking: Is amazed joints, bones, nerves, muscles or blood vessels. If amazed joints should clarify osteoarthritis or arthritis it. For this it is necessary to study the history, the nature of the complaints, the clinic, the results of objective laboratory and instrumental examination. In favor of osteoarthritis evidenced by such distinctive features:

• Intermediate and advanced age (presence of risk factors for OA);

• slow progression of the disease;

• absence of a disease with focal infection, allergy;

• liaison with the physical overload;

• involvement in most of the knee, hipand spine;

• lack of rheumatoid nodules, rheumatoid factor;

• Morphology: the presence of cartilage degeneration and fibrosis of the synovial membrane;

• the presence of characteristic X-ray signs for osteoarthritis.

Laboratory data are not essential in the diagnosis of OA, as often studied parameters within normal limits.

In the presence of reactive synovitis may increase ESR 20-25 mm/h, CRP, α-globulin, fibrinogen, seromucoid, sialic acids. However, it should be remembered that in elderly patients, who constitute the majority of patients with osteoarthritis, a slight increase in ESR and rheumatoid factor titers may be a feature of age and does not exclude the diagnosis of osteoarthritis.

Synovial fluid - a transparent, high and medium viscosity, good mucin clot is formed. Reliable marker of cartilage metabolism in osteoarthritis, which would reflect the progression of the disease and have prognostic significance, are not currently available. Of instrumental methods in outpatient conditions often use X-rays of the joints. X-rays are usually performed symmetrical joints, not only the affected, which allows to identify deviations. If you suspect a spinal osteoarthritis joints must necessarily spend radiography of the spine in the lateral projection. However, it should be noted that in the early stages of OA X-ray signs may be absent. At the same time it should be remembered that the radiographic manifestations of osteoarthritis found a very high frequency in older age groups in the absence of clinical signs of osteoarthritis. Arthroscopy is also used, which allows you to visually detect changes of articular cartilage, synovial membrane. In recent years, more and more methods are used ultrasound joints that allow to characterize the articular surfaces , to determine the thickness of the cartilage , its uniformity , the echogenicity of the subchondral plate boundary identify bony growths, determine the amount of joint fluid.

Differential diagnosis. On another diagnosis to think if there are joint syndrome followingsymptoms:

• the patient's age to 45 years;

• fever and general weakness (typical for rheumatoid arthritis, gout, septic arthritis, viral arthropathy);

• there are clear signs of inflammation in the joints and duration of morning stiffness (typical for RA, gout);

• amazed wrist, elbow, shoulder or ankle joints.

If the patient is over the age of 40 years complained of pain in the knee, you must first focus on the diagnosis of OA.

If the clinical picture of the patient has some features recommended differential diagnosis of OA with the following diseases:

• ankylosing spondylitis;
• reactive arthritis;
• gout;
• polymyalgia rheumatica;
• psoriatic arthritis;
• rheumatoid arthritis;
• paraneoplastic arthropathy and others;
• systemic connective tissue diseases.

Treatment of osteoarthritis.

Trends in the treatment of OA include:

• a decrease of pain;
• reducing the severity of inflammation (reactive synovitis);
• reduce the environmental factor contributing to the pain and progression of the disease;
• prevent the development of joint deformity and disability;
• slowing the progression of the disease;
• improving the quality of life of patients.

Pharmacological treatment.

All of drugs used in the treatment of OA, is divided into two main groups: symptom-modifying and structure-modifying.

By modeling the symptom medications include NSAIDs, analgesics and opioid simple, muscle relaxants, and glucocorticosteroids (GCS). Actions of drugs in this group aimed at eliminating joint pain or a significant reduction in its intensity, as well as restoration of function of the musculoskeletal system.

The main groups of drugs used for the relief of pain associated with RA (LI Butler, 2005)

• simple analgesics (acetaminophen)
• Centrally acting analgesics (tramadol)
• Non-selective inhibitors COG1andCOG_2 (standard NSAIDs)
• COG_2 selective inhibitors (meloxicam, nimesulide)
• a combination of drugs (NSAID + misoprostol)

However, developing tactics patients OA, every doctor should imagine that one analgesic therapy is not enough to achieve the stabilization process in the joint. Only long-term treatment aimed at improving the function of the main components of the joint, can lead to a stable analgesic effect and improve the structure of cartilage.

By structurally modifying drugs (symptomatic slow-acting drugs, chondroprotectors) include chondroitin sulfate, avocado unsaponifiable compounds/soybean diaceriin and hyaluronic acid, which have a symptomatic effect and slow the pace disease progression, to normalize the structure of hyaline cartilage.

At moderate intermittent pain with no signs of inflammation of the joints is preferable to appoint paracetamol, non-narcotic analgesic central action that does not have anti-inflammatory effect in the daily dose of 4 g (older - 2 g/day). Proved that its use in OA dose of 2 g/day is safe for 2 years or more. The drug should be administered with caution to patients with liver disease and is not recommended for patients who abuse alcohol.

Group of symptom-modifying drugs widest practical application found NSAIDs. They are shown in OA in case of failure of paracetamol, as well as the presence of signs of inflammation. Efficiency advantage over any other NSAIDs is not revealed. The choice of

drug is determined primarily by its safety for specific clinical conditions.

Pronounced analgesic and anti-inflammatory NSAIDs exert holding in doses equivalent to approximately the same analgesic activity. The optimal considered ibuprofen (at a dose of 1200-2400 mg/day), ketoprofen (at doses up to 300 mg/day), diclofenac (at a dose of 75-100 mg/day). Piroxicam and indomethacin is not recommended because of possible severe side effects (stomach ulcers, stomach bleeding) particularly in elderly patients and drug interactions (e.g., antihypertensives, β- blockers, diuretics, etc.). Additionally, indomethacin hasadversely impact – consequence metabolism of articular cartilage, it may contribute to the progression of degeneration, particularly in coxarthrosis. Priority is given to selective NSAIDs that block COX-2: meloxicam (movalis) at a dose of 7.5 mg/day, nimesulide (nimesil, nise) at a dose of 200 mg/day, celecoxib (Celebrex) in a dose of 100-200 mg/day, which inhibit the synthesis of enzymes that destroy cartilage (metallo-elastase) that leads not only to reduce damage to the cartilage matrix, but also normalization of cartilage synthesis.

Given the possibility of complications, including the negative impact of a number of drugs in this group on cartilage metabolism, recommended to prescribe NSAIDs only for the period of exacerbation. In addition, the decrease in joint pain when taking NSAIDs may impair patient compliance and increase his physical activity. To enhance the effect, it is advisable to combine NSAIDs with muscle relaxants (Mydocalmum, sirdalud) and sedatives.

Local therapy in OA.

Of particular importance if there are clear contraindications to the use of systemic NSAIDs, especially in elderly patients is local therapy.

For topical cutaneous application of NSAID (gels, creams, ointments containing, diclofenac, ketoprofen, ibuprofen, indomethacin, etc.) in the soft tissues directly under the place of application are therapeutic drug concentrations. In general circulation enters a small amount of it, which allows almost avoid systemic adverse effects of NSAIDs (gastropathy, increased blood pressure, fluid retention, etc.), which are frequently observed in patients with these drugs.

In the outpatient setting can be prescribed compresses on painful joints 50 % solution with anti-inflammatory ointments Dimexidum (gels). Dimexidum significantly increasepermeabilityof skin for drugs used as part of compresses.

With reactive synovitis good anti-inflammatory and analgesic effect can be obtained by intraarticular injection of corticosteroids (betamethasone, triamcinolone, methylprednisolone 0.5-1 ml in large joints no more than three injections per year in the knee joints) in conjunction with anesthetics (procaine, lidocaine) for potentiation of their action. In coxarthrosis intraarticular introduction of GCS is not recommended.

Slow-acting (disease-modifying) symptomatic drugs have chondroprotektive properties and pronounced analgesic effect, which occurs within 2-8 weeks of starting treatment and lasts for 3-6 months after cessation of therapy.

The main components of slow-acting drugs (chondroprotektors)

• chondroitin sulfate (struktum, arthritis, alflutop, teraflex, chondrazamine, chondro, chondroxid, stopartrozforte);

• glucosamine (DONA, glucosamine sulfate, arthritis, teraflex, chondrazamine, chondro, stopartroz, stopartrozforte);

• keratan sulfate (alflutop);

• hyaluronate, hyaluronic acid (Synvisc, hylan, Austen fermatron, alflutop);

Structurally modifying drugs for OA are able to control, slow down, stabilize or even cause regression of existing changes in the tissues of the joint. They have almost no side effects, and the effect is comparable to simptomatic NSAIDs (which allows you to reduce the dose of NSAIDs), the effect persists after treatment. Reception structurally modifying drugs can be combined with analgesics (paracetamol) and NSAIDs.

Chondroprotektiv action of this group of drugs associated with the ability to provide a stimulating effect on chondroblasts, which leads to increased synthesis of intermediate

macromolecular substance of hyaline cartilage, in particular of proteoglycans and collagen fibers. Furthermore, they reduce the activity of metalloproteinases (phospholipase A2, collagenase) and proinflammatory cytokines, thereby reducing the inflammatory changes in the tissues of the joint.

Chondroitin sulfate is a major component of connective tissue extracellular matrix, including cartilage, bone, skin. The drug stimulates matrix synthesis by chondrocytes and inhibits the enzymes that are involved in the destruction of cartilage and reduces the synthesis of inflammatory mediators. The drug is used by 750 mg 2 times a day the first 2 weeks, followed by 500 mg orally 2 times a day, the duration of the course of 6 months.

Glucosamine sulfate. Glucosamine is aminomonosacharid, it is used in the body chondrocytes as starting material for the synthesis of proteoglycans, glycosaminoglycans, and hyaluronic acid. The drug is prescribed inside of 1500 mg per day (single dose) or intramuscularly 2-3 times a week, the general course of 4-12 weeks, the course is repeated 2-3 times a year.

Increasing the use of drugs are combined in the composition of which, along with chondroitin sulfate, glucosamine include sulfate or hydrochloride. The positive effect of the combined use of these drugs in patients with OA is not in doubt. While taking glucosamine and chondroitin sulfate occurs synergies of their actions. To combined chondroprotektors include teraflex, arthritis, chondro new, stopartroz forte.

Conventional method of treatment of osteoarthritis of the knee is the intra-articular hyaluronic acid preparations. It is proved that they not only improve the mechanical function of affected joints, but they retard the progression of the pathological process. Depending on the size of the joint is introduced of from 0.5 to 2.0 ml of a 1 % solution of sodium hyaluronate into the joint once a week 3-5. Effect lasts from 6 months to a year.

Non-pharmacological treatments

• Education of the patient with OA. The clinic is organized by "School for patients with OA". In the classroom the physician should explain to patients in an accessible form the essence of the disease, focusing on avoidable risk factors, its forecast, which is important from a psychological point of view. Should be taught how to behave in everyday life (avoid prolonged fixed postures, unloading joints, comfortable shoes);

• Therapeutic exercises. Regular exercise for 20-30 minutes twice a day to strengthen the muscles. They perform better lying down or sitting at the maximum load reduction weight on the joints;

• Physical therapy methods: heat or cold procedures. Therapeutic massage joints and muscles held outside regional acute synovitis;

• Orthopedic correction. In gonarthrosis recommended wearing kneepad, which captures the joint. Knee OA is often accompanied by flatfeet, in connection with which patients should be wearing supinators.

Patients with severe hip and knee surgical treatment (joint replacement, arthroscopic surgery). In some cases, operate and other joints: ankle, elbow, shoulder.

Sanatorium treatment.

Patients with OA stage I or II without signs of synovitis or its activity 1 degree, unsharp exacerbation treatment is indicated in local sanatoriums or with hydrogen sulfide, radon, chloride, sodium or other mineral waters ozokerit-use, paraffin and mud. In Belarus, specialized sanatoriums for treatment of such patients are categories sanatorium "Radon", "Dnieper", "Letsi", "Lakeside", "Crane" (resort "Naroch"), "Borovoe".

Medical and social assessment.

With the defeat of the small joints, even long flowing, disabled patients persist long enough. Temporary disability is determined when expressed pain syndrome or development of synovitis in individuals whose work is associated with a significant or moderate physical exertion, severe mechanical stress on the affected joints, heavy lifting and long monotonous forced posture, prolonged stay on his feet. Tentative dates of temporary disability depend on

localization, clinical course and treatment efficacy of unilateral primary coxarthrosis, gonarthrosis, and generalized OA and is 10-25 days.

Direction on the MREB to establish disability groups subject patients with contraindications views and working conditions for the recognition of their disability for a period of rational employment or retraining, patients with severe OA of the hip and knee joints in violation of statics and contraindications for surgical correction, as well as patients with OA with the presence of complications (recurrent bursitis, reactive synovitis, radicular syndrome), difficult to treat.

Dispanserization

Working-age patients with primary OA of large joints subject to medical check-ups.

Rheumatologist observation frequency: 4 times a year in the I^{th} year, then - 2 times a year; the development of remission - 1 time per year.

Examinations by medical specialists: orthopedist - annually, other professionals –when indicated.

Laboratory and instrumental investigations:

• total blood count, urinalysis - 2 times a year;

• ECG, X-ray of the chest, arthrography -1 times a year;

• CT or MRI of joints - indicated. Observation periods and deregistration criteria: observation for life.

Prophylaxis

Osteoarthritis prevention should begin in childhood. Parents should follow the correct posture of the child, the situation at the desk to avoid the development of scoliosis. To strengthen the musculo-ligamentous apparatus are useful systematic children gymnastics.

If there is congenital or acquired disorders of joint-sceletal system (curvature of the spine, hip hypoplasia, flatfeet) require an earlier consultation orthopedist for possible correction of these violations, the detection of flat feet-wearing insoles for prevention of further lowering the arch. Young people need to consider a family history of osteoarthritis in choosing a profession, sports.

Adults with pain in the joints need to monitor body weight, do not overload the joints to avoid fixed postures at work. Useful: water aerobics, swimming with obligatory followed by rest.

Rheumatoid arthritis (ICD-10- MO5, MO6)

Rheumatoid arthritis (RA) - a chronic systemic autoimmune inflammatory disease of the connective tissue of unknown etiology, characterized by progressive symmetrical lesion predominantly peripheral (synovial) joint type of erosive and destructive polyarthritis and systemic damage of visceral organs.

The etiology of RA is unknown. Established family genetic predisposition. Examines the impact of infectious agents (viruses, bacteria). Contributing factors to the development of RA may be the weakness of the regulatory systems of the body (one or more), dysfunction of the endocrine glands, central nervous system, metabolism. Predisposing factors like the stage for the onset of illness. The immediate impetus for the emergence of RA can be a sore throat, flu, and other respiratory diseases, hypothermia, childbirth, trauma, stressful situations.

Pathogenesis: unknown etiological factor causes the development of the immune response, resulting in the formation of antibody immunoglobulin IgG, M - 80% of the Russian Federation , to DNA, the nuclei of cells, immune complexes (IC), which damage the tissue of the joints, increases the synthesis of proinflammatory cytokines. Damage begins with synovitis, which then acquires the proliferative character with damage cartilage and bone.

Formation of IC promotes platelet aggregation and formation of microthromb, disruption in the microcirculation system. Joint tissue damage by IC leads to further production of antibodies thereto, and chronic inflammation. Development of IC vasculitis leads to the defeat of the connective tissue, and other organs and systems.

Diagnosis of RA

RA is more common in women. Prevalence increases with age. Onset of the disease is often associated with physiological changes in the body (puberty, menopause), stay in the raw climate conditions. Development of RA can be triggered by a previous infection, hypothermia, stressful situation.

Articular syndrome - a leading clinical symptom of RA. RA is a typical two-way symmetrical lesion joints of the hands, particularly arthritis intraphalangial proximal joints, giving the spindle-shaped fingers and inflammation of carpometacarpal joints. Often marked by inflammation of the wrist, carpometacarpal and intercarpal joints. Along with arthritis of the small joints should be noted defeat muscle tendons in the hand.

One of the early symptoms of RA (in cases of joint damage brushes) is to reduce the weight of interosseous muscles of the hand. The defeat of the tendons and muscles change by playing a major role in the formation of resistant strains of brushes : lateral deviation of the fingers, toe deformities such as "Button-loop" (flexion contracture of the proximal and distal interphalangeal joint hyperextension) or type "swan neck" (flexion contracture of the distal and hyperextension of the proximal interphalangeal joints). These deformations are found almost exclusively in RA, define the term "rheumatoid hand" and have a diagnostic value.

In the region of the elbow joints can be found, such as diagnostic signs rheumatoid subcutaneous or supraperiostal nodules.

Pain, feeling of stiffness in the cervical spine - often complain of RA patients. The defeat of the thoracic and lumbar spine is not typical for RA.

Violation of mobility and hip pain in patients with RA requires the exclusion of avascular necrosis of the femoral head, usually develops with prolonged corticosteroid therapy.

Arthritis metatarsophalangeal joints feet - almost a permanent phenomenon in RA. Consequence of persistent arthritis of these joints are hammer deformity of the fingers, the metatarsal heads subluxations towards soles deviation fingers outwards. As a result of these changes is formed flat, there are painful "corns." The complex of these changes is called "rheumatoid foot". Inflammation of the ankle joints also often noted.

Systemic manifestations of RA.

Systemic disease occurs in almost all cases of RA on clinical expression are relatively infrequent, so they require a targeted search.

More likely to occur subcutaneous nodules in the joints (usually the elbow), and polyneuropathy.

Polyneuropathy - a manifestation of vasculitis vessels feeding the peripheral nerves. Patients concerned about numbness, burning, coldness in the distal extremities, marked tenderness, not only the affected joints, but also tissues located at a distance from them, decreased or increased sensitivity of the nerves in the affected area.

Rheumatoid lung disease include: diffuse fibrosing alveolitis, nodular interstitial lung disease, pulmonary vasculitis, adhesive pleurisy.

The heart, in addition to pericarditis can be observed changes caused by myocarditis and endocarditis, rarely formed heart defects (mitral or aortic valve).

One of the most severe complications of RA is amyloidosis. It occurs in 10-15% of patients, usually after many years of onset.

Glomerulonephritis is rarer than amyloidosis and drug nephropathy.

Of all the rheumatoid lesions of the internal organs are the most serious damage to the kidneys, often worsens the prognosis of RA.

Laboratory and instrumental methods of diagnosis of RA in a clinic.

Laboratory changes in RA are nonspecific and should be interpreted only on the basis of clinical manifestations:

- total blood count - increased erythrocyte sedimentation rate, and anemia;
- increase of acute phase parameters of inflammation (CRP);
- CCPA - cyclic citrullinated peptide antibody;
- rheumatoid factor (revealed in seropositive RA synovial fluid often);

• X-rays of joints (namely joints of the hands and feet with the capture of the wrist and ankle joints and meticulously counting erosion);

• Ultrasound of joints.

Table 3.2 Diagnostic criteria of rheumatoid arthritis (ARA, 1987)

Criteria	**Definition**
1. Morning stiffness	Morning stiffness in the joints or around it, continuing for at least 1 hour before maximal improvement
2. Arthritis of three or more joints	at least three joint areas simultaneously, swelling (not bony growths) soft tissue or effusion seen a doctor; account for 14 regions: the proximal interphalangeal, carpophalangeal, wrist, elbow, knee, ankle and metatarsophalangeal joints (7 in right and left)
3. Arthritis hand joints	Swelling by at least one region (as defined above) at the wrist, metacarpophalangeal or proximal interphalangeal joint
4. Symmetric arthritis	Simultaneous defeat of identical joint areas (as defined in paragraph 2) on both sides (symmetrical, incomplete lesion of the proximal interphalangeal,metacarpophalangeal and metatarsophalangeal joints)
5. Rheumatoid nodules	Subcutaneous nodules over bony prominences or extensor surfaces or periarticular areas observed doctor
6. Serum RF	Determining of elevated titers of RF in the serum by any method yielding less than 5 % positive results in a control.
7. Radiographic changes	Radiographicchangestypicalofrheumatoidarthritisontheanteroposteriorradiographofbrusheswiththewristjoint, whichshouldincludeerosionorunevendecalcificationlocalizedinordirectlyoutsidetheaffectedjoints (changescharacteristiconlyforosteoarthritis, ignore)

A joint working group of the American College of Rheumatology (ACR) and European League Against Rheumatism (EURAR) offered in 2010. point scoring system (1 to 10 points) early RA criteria.

Table 3.3 RA classification criteria: domains, categories and point scores(ACR/EULAR, 2010)

Domain	**Category**	**Point score**
A	Joint involvement (0–5 points)[a]	
	1 large joint	0
	2–10 large joints	1
	1–3 small joints (large joints not counted)	2
	4–10 small joints (large joints not counted)	3
	>10 joints including at least one small joint	5
B	Serology (at least one test needed for classification; 0–3 points)[b]	

Domain	Category	Point score
	Negative RF and negative ACPA	0
	Low positive RF or low positive ACPA	2
	High positive RF or high positive ACPA	3
C	Acute-phase reactants (at least one test needed for classification; 0–1 point)[c]	
	Normal CRP and normal ESR	0
	Abnormal CRP or abnormal ESR	1
D	Duration of symptoms[d]	
	<6 weeks	0
	≥6 weeks	1

The points from each of domains A through D are added and the sum is considered to be the total score. A total score of ≥6 is needed to classify a patient as having definite RA.

[a]Joint involvement refers to any swollen or tender joint on examination, which may be confirmed by imaging evidence of synovitis. DIP joints, first CMC joints and first MTP joints are excluded from assessment. Large joints refers to shoulders, elbows, hips, knees and ankles. Small joints refers to MCP joints, PIP joints, second through fifth MTP joints, thumb IP joints and wrists.

[b]Negative means less than or equal to the upper limit of normal (ULN); low positive means >ULN; high positive means >3× ULN.

[c]Normal and abnormal are determined by local laboratory standards.

[d]Duration of symptoms as per patient's self-report.

Table 3.4 Classification of Rheumatoid Arthritis (Minsk, 2003)

Clinical and immunological characteristics	Level of activity	Radiological stage	Functional class
Seropositive rheumatoid arthritis • Polyarthritis • Rheumatoid vasculitis (digital arteritis, chronic leg ulcers, Raynaud's syndrome, etc.) • Rheumatoid nodes • polyneuropathy • Rheumatoid lung disease • Felty Syndrome Rheumatoid arthritis, seronegative • Polyarthritis • Still's syndrome in adults	-Remission 1 –Low 2 –Moderate 3 - High	I - periarticular osteoporozis II - osteoporosis + narrowing of the joint slits, can be isolated uzurs III - the same + multiple uzurs IV - the same + bone ankylosis	I II III IV

Table 3.5Degree ofRA activity

Parameter	Degree of activity			
	0	1	2	3
Pain (VAS), cm	0	≤3	from 3 to 6	>6

Morning stiffness, min.	None	Up to 60 min.	To 12 hours	during the day
Arthritis, the severity	None	Minor	Moderate	Significant
ESR, mm/h	≤15	16-30	31-45	>45
CRP	N	≤2N	≤3N	>3N

Note: N-norm

Examples of clinical diagnosis formulations:

1. Rheumatoid arthritis: seropositive, 2 st. of activity, III st., FC III.
2. Rheumatoid arthritis: seropositive, digital arthritis, rheumatoid nodules, 3 st. of activity, II st., FC II.
3. Rheumatoid arthritis: seronegative, 2 st. of activity, I st., FC II.
4. Rheumatoid arthritis: remission, II ct. FC I.

Feature of functional classes:

I - A fully preserved, B, C;

II - saved A, B; (limitation of certain types of professional activity)

III - A preserved; B, C are limited;

IV - A, B, Care limited.

A – self-: dressing, eating, grooming, etc.

B - unprofessional activity: elements of leisure, recreation, sports, taking into account gender and age;

In - professional activities: work, study, housework (domestic workers), gender and age.

Physician needs disease dlassification to clarify the nature of a particular patient flow activity and the stage of the pathological process that allows you to define individual treatment policy, as there are no patients with RA identical currents, examine disability.

Differential diagnosis is made with arthritis and other diseases accompanied by articular syndrome.

Characteristic of rheumatic polyarthritis:

• migratory symmetrical polyarthritis of large joints , quick passing , especially against the background of NSAIDs;
• development of polyarthritis 2-3 weeks after the sore throat, pharyngitis;
• combination of fever with cardiac damage (carditis);
• the possibility of extra-articular lesions (erythema annulare on the skin of the trunk, legs, subcutaneous rheumatoid nodules, chorea in childhood and adolescence);
• no changes on radiographs of the affected joints;
• specific positive (in relation to β-hemolytic group streptococcus A) serological response.

Characteristics of joint damage in systemic lupus erythematosus (SLE):

• unstable symmetric (rarely asymmetric) oligo-or migratory polyarthritis any localization, most small joints of the hands, wrist and knee joints. In rare cases, "rheumatoidlike" arthritis of joints of the hands, mainly affecting the tendons and deformation fingers without erosive and destructive changes syndrome (Jacques syndrome);
• moderately expressed inflammatory changes in the affected joints , sometimes erythema over the affected joints ;
• fever;
• typical systemic manifestations: skin lesions ("lupus butterfly"), erythema in the zone"cleavage", photosensitivity, alopecia, renal and other internal organs, the nervous system;
• absence of bone changes on radiographs;
• characteristic changes in the blood count (leukopenia, thrombocytopenia, anemia) and immunological parameters: LE-cells in the diagnostic titre, antinuclear factor, antibodies to DNA and other markers confirming SLE.

Characteristics of gouty arthritis:

• typical character attack arthritis (monoartritis) and localization in the metatarsophalangeal joint of the big toe;

• inflammatory changes in the joint with hyperemia and cyanotic hue of the skin over the affected joint;
• the absence of symptoms between attacks until the development of chronic gouty arthritis;
• hyperuricemia;
• presence of tophi in chronic gouty arthritis;
• symptom of " swelling of the bone edge " over separately or cystoid modified portions of bone, as well as a symptom of "punch" in the X-ray of bone;
• presence of uric acid crystals in tophi and synovial fluid;
• urolithiasis and other variants of renal pathology.

Characteristic of psoriatic arthritis:

• asymmetric mono-or oligoarthritis with localization in the joints "exceptions" for rheumatoid arthritis: the distal interphalangeal, metacarpophalangeal joint of the thumb and the proximal interphalangeal fifth thumb;
• «sausagelike" form with finger purple-bluish color of the skin of the finger joints;
• synchronicity occurs in arthritis and degeneration of the nail plate in the form of a "thimble";
• pathology of spine (sacroiliitis, spondylitis);
• the presence or appearance for dynamic monitoring of psoriatic plaques on the skin (psoriasis without psoriatic arthritis does not happen);
• «anarchic" type of deformation of the joints on the radiograph, possible osteolysis of the terminal phalanges and metacarpals brush heads, rarely stop;
• the presence of HLA-B 27 antigen in the serum.

Characteristic of ankylosing spondylitis (Bechterew's disease):

• mono-or oligoarthritis, mainly large and medium-sized joints of the lower extremities, sacroiliac joint, spine, joints, sternum, sternocostal, sternoclavicular, costovertebral, temporomandibular joint;
• tightness and stiffness of the spine in the morning;
• limitation of movement in the lumbar spine in the sagittal and frontal planes;
• sustained and progressive spine involvement with the outcome in ankylosis "bottom-up";
• early bilateral sacroiliitis followed ankilosis;
• pelvic exostosis and calcanea;
• the presence of HLA-B 27 antigen in the serum.

Characterization of paraneoplastic arthritis:

• asymmetric mono-or oligoarthritis (rarely polyarthritis) large joints;
• no or little inflammatory reaction of the joints;
• seal periarticular tissues;
• Lack of joint deformities;
• Parallel for arthritis and cancer process (established or search);
• lack of pathological changes on radiographs of joints;
• ineffectiveness of anti-inflammatory drugs NSAIDs;
• the effectiveness of anticancer therapy.

Significant difficulties in the diagnosis of RA is experiencing outpatient physician in the diagnosis of early stages of RA, when there are no radiological signs of erosive arthritis and rheumatoid factor in the blood serum, as well as in older groups, when RA is attached to their existing osteoarthritis.

Approach to the Patient.

All patients with a presumptive diagnosis of RA should consult a rheumatologist and rheumatology hospitalized in the hospital for further diagnosis, especially in the presence of systemic manifestations, for the selection of the basic anti-inflammatory drugs and determine tactics further outpatient management of patients.

Be hospitalized patients with acute exacerbation of the disease, suspected development interkurent infection, septic arthritis, and other serious complications of the disease leading to a possible pulse therapy, extracorporeal therapies, as well as surgical treatment.

Drug therapy. Used for the treatment of RA following groups of drugs:

• basic drugs (disease-modifying). These drugs affect the essence of the pathological process, suppress the immune system aggression against its own tissues. This group of drugs that Propafenone slow the progression of the disease and, as a consequence, prevent the development of joint deformities;

• symptom-modifying drugs that reduce the symptoms (manifestation of disease) - pain, inflammation. These include non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticosteroids (GCS). The physician should be aware that NSAIDs do not slow radiographic progression of the disease, so treatment NSAIDs should only be done in conjunction with active treatment basic anti-inflammatory drugs. And yet, to establish the diagnosis of RA patients typically assigned NSAIDs, alone (no more than 6 weeks) with performance evaluation after 2 weeks. (NSAID used to treat diseases of the joints - see "Osteoarthritis").

Glucocorticosteroids.

In rheumatoid arthritis, systemic treatment with corticosteroids to proceed only on the strict condition. In recent years, proved that low-dose glucocorticosteroids (4-6 mg in terms of methylprednisolone) eliminate the deficit in patients with endogenous cortisol, enhance anti-inflammatory effects of NSAIDs, mainly due to the effect on cytokine levels significantly improve quality of life of patients. Low-dose of glucocorticoids, even at long years of admission, rarely causes side effects. Currently reading for appointment of corticosteroids in patients with rheumatoid arthritis inside widened. Corticosteroids for long-term use can be assigned:

• in severe forms of rheumatoid arthritis with systemic manifestations, visceritis, high fever, the maximum activity of the process;

• the ineffectiveness of other methods of treatment of active rheumatoid inflammation even without systemic manifestations;

• rheumatoid arthritis in the elderly, when the patient's body is not very sensitive to NSAIDs;

• for social in small doses in the morning when the patient - the only breadwinner in the family, forced to continue to work and look decent among colleagues.

In some cases, glucocorticoids inside administered in combination with a slow-acting drugs for the time required to achieve a therapeutic effect (2-4 months). During treatment (so-called bridge-therapy). In any case, glucocorticoids in patients with rheumatoid arthritis should not be the first drug, appointed immediately after diagnosis.

Treatment of patients with rheumatoid arthritis corticosteroids can start with a daily dose of 6-12-24 mg of methylprednisolone, or 7.5-15-30mg of prednisolone, in the most severe cases - 32-48 mg of methylprednisolone or carried pulse therapy. Such doses are administered before a therapeutic effect, then every 5 - 7 days, the dose was gradually reduced by 1/4 - 1/8 of the tablets until the supporting (usually 2-4-6 mg of methylprednisolone, or 2.5-5-7.5 mg of prednisolone). This dose is taken before a persistent improvement or, where necessary, kept. The duration of treatment depends on the nature of glucocorticoid flow and activity of rheumatoid arthritis.

After the establishment of maintenance doses can try to go to the procedure alternating therapy, i.e. destination doubling the maintenance dose every other day. Alternating therapy can reduce the likelihood of adrenal suppression, as in the day when not taking glucocorticoids, there is an increased release of corticotropin by the pituitary gland that stimulates the adrenal cortex-stream.

Transfer the patient to the alternating method of glucocorticoid therapy should be carried out under the supervision of his condition. In strengthening joint pain and stiffness on the background alternating method return to daily treatment with glucocorticoids.

It is preferable to take full daily dose glucocorticoid 8 a.m. to 2 hours or overnight. If this method does not provide reception glucocorticoids reduce stiffness and joint pain for a long

period of time, you can assign 2/3 doses at 8 am and 1/3 dose of 10-12 h, i.e., the entire dose of the drug taken in the first half of the day.

In some patients, there is a need three or even four times the reception during the day (morning, afternoon and 2 times at night). However, in this case, most of the dose taken in the morning.

With prolonged use of systemic glucocorticoid therapy is not recommended to use depot intramuscular preparations, since in this case it is impossible to take into account the circadian rhythm of pain, drug adsorbed uneven blood levels varies.

Intramuscular depot preparations can be used in patients with rheumatoid arthritis as a method of treatment used periodically to enhance the therapeutic effect of oral glucocorticoids or the lack of effectiveness of NSAIDs. In such situations, more efficient single intramuscular depot.

It should be remembered that patients with rheumatoid arthritis develops easily corticosteroid dependence, especially in the appointment of high doses of hormone therapy and the duration of its use. It is therefore important to try to avoid lower doses and short duration courses, and use alternating method of treatment.

Glucocorticoids in patients with rheumatoid arthritis do not have to be the only drug, they may be only part of an integrated drug therapy. Typically, they are combined with NSAIDs and with slow- drugs.

Basic therapy. Under basal (pathogenic) understand therapy aimed at slowing the progression of the pathological process in RA - is primarily slow the progression of bone-destructive changes in the joints. Pathogenetic therapy includes standard basic therapy drugs that are most commonly used in clinical practice, and preparations "genetically engineered biological therapy" (GEBT), created on the basis of genetic engineering technologies (see below). GEBT drugs are expensive and are assigned strictly on the evidence. The effect of the application of the standard drugs 'underlying' therapy develops in 1.5-3 months. Since these drugs do not directly influence the symptoms of the disease, their use is not initially accompanied by a decrease in pain, inflammation. When applying preparty GEBT clinical effect certain proportion of patients may develop after the first administration of the drug and grow in subsequent infusions; others - he can wear deferred nature or clinical effect may be unexpressed.

Preparations pathogenetic therapy requires a long, sometimes lifelong reception. Assigned to all patients with RA only when the diagnosis is prooved.

The choice of drug therapy base, its dosage determines in each case rheumatologist.

Problem of district physician or GP - monitoring of patient drug regimens, treatment efficacy, side effects and the development of warning them, business consulting a doctor rheumatologist, since he is receiving, not all clinics. Increasing the dose of drugs, as well as reducing its rate of decline should be discussed with the rheumatologist.

I Preparations standard basic therapy (according to recommendations of the European League Against Rheumatism against 2010)

- methotrexate;
- sulfasalazine;
- leflunomide (Arava);
- gold salts (Tauredon);
- hydroxychloroquine (Plaquenil);

II. Preparations of GEBT:

- infliximab, adalimumab;
- rituximab;
- tocilizumab;

Basic drugs:

- slow radiographic progression of RA;
- reduce the need for NSAIDs and glucocorticoids;

• improve quality of life;

• increase lifespan, reduce the risk of cardiovascular complications (methotrexate)

Methotrexate is considered the "gold standard" in the treatment of RA, it is the most effective drug, which begin with the treatment of disease.

Methotrexate is taken 1 time a week to 12 hours apart, preferably in two steps (and possible 3-fold the drug). e.g. first Monday methotrexate carried out in 8.00; second - after 12 hours, 20.00 ; third - Tuesday, 8.00 . initial dose of methotrexate - 7.5 mg/week, during normal tolerability dose methotrexate increased by 2.5-5 mg per week, leading up to 12.5-25 mg/week. Efficacy was dose-dependent, but the reception over 25-30 mg weekly impractical because efficiency does not increase, but increases the frequency of side effects. Perhaps parenteral (subcutaneous or intramuscular) administration of methotrexate. Methotrexate for parenteral (subcutaneous) administration (metoject) is better tolerated than tablet form of the drug, and is used in cases of toxicity in the gastrointestinal tract or in the absence of effect when orally reception, which can be associated with low absorbability of the drug in the gastrointestinal tract.

Drug efficacy was assessed after 3-6 months.

Side effects occur infrequently. Possible liver dysfunction with the development of nonalcoholic steatohepatitis, fibrosis. Clinical manifestations are usually minimal (right upper quadrant heaviness, bloating, etc). When recommendations for receiving methotrexateare fulfilled that significantly reduced the risk of complications.

With a deficit of folic acid in patients may develop ulcerative stomatitis (sores in the mouth). Methotrexate may be accompanied by worsening of chronic foci of infection (chronic pyelonephritis, chronic tonsillitis, chronic adnexitis etc.), slowing the healing of erosions, ulcers.

Rare complications include dysfunction of the bone marrow with a decrease in peripheral blood formed elements, with the development of lung interstitial pneumonitis.

To reduce the risk of complications need to change eating habits and lifestyle. It is strictly forbidden to drink alcohol, the reception of which greatly increases the risk of liver damage. Necessary to limit the use of products containing caffeine (reduces the efficacy of methotrexate), provide control over laboratory parameters: complete blood count, biochemical parameters of liver function (AST, ALT) - 1 times a week until a stable dose, then 1 time a month; urea, creatinine – 1 time every 6-12 months.

Need to take folic acid at a dose of 5-10 mg/week of methotrexate (folic acid reduces the risk of stomatitis, liver complications, and violations of the hematopoietic system).

On the day of methotrexate undesirable nonsteroidal anti-inflammatory drugs because they may lead to an undesirable increase in its concentration.

With the development of diseases of infectious nature, exacerbation of chronic foci of infection (chronic pyelonephritis, tonsillitis, etc.) should work together to resolve the issue with the rheumatologist on temporary discontinuation.

In patients receiving methotrexate should refrain from carrying out vaccinations.

Leflunamid (Arava) - appointed by the lack of effectiveness of methotrexate, the development of side effects or contraindications to its use. Outpatient patients taking 10-20 mg/day. The effect is 4-12 weeks. Is as effective as methotrexate. When receiving leflunomide characterized by the development of fewer side effects than other drugs while taking basic therapy. May cause diarrhea, elevated liver enzymes. A number of patients may increase blood pressure, decrease the number of leukocytes, erythrocytes, platelets, develop an allergic reaction. Necessary to carry out monitoring of blood pressure, control laboratory parameters as in the treatment with methotrexate.

Sulfasalazine prescribed for moderate RA activity without clinical extra-articular manifestations. The drug can be used with contraindications to treatment with methotrexate, and in combination therapy with methotrexate and corticosteroids. Prescribed by the scheme: the first week - 1 tablet once a day; second week - 1 tablet 2 times a day; third week - 1 tablet

in the morning and 2 tablets in the evening; fourth week - 2 tablets 2 times a day.

Side effects from the gastrointestinal tract development of nausea, vomiting, loose stool; CNS - headache, dizziness; development of increased sensitivity to sunlight; changes in the blood in the form of anemia, reduced leukocyte count, platelet count. In women of childbearing may develop infertility, in men - reducing the number of sperm.

Very rarely indicated the development of "sulfasalasine" syndrome, which manifests with fever, rash, impaired liver function.

For the diagnosis of complications monitor blood count: every 2 weeks to achieve a stable dose, then every 6 weeks.

Indicators of biochemical analysis of blood (liver enzymes - ALT, AST) control every 1.5 months; urea, creatinine - every 6 months.

Gold salts. Mostly used Tauredon. Begin treatment in the hospital with a dose of 10 mg intramuscularly once a week. With good endurance and good effect of outpatient Tauredon inserted deep into the muscle 1 every 2 weeks for years.

Side effects: allergic rash, stomatitis, hepatitis, nephropathy ("golden jade"), pancytopenia, diarrhea, bone fractures.

Necessary to monitor health of the patient, total blood count, urinalysis, body temperature measurement, a study of liver enzymes. In connection with the common side effects currently used infrequently gold salts.

For treating the most severe forms of RA are currently using a new group of drugs, known as the "biological agents", anti-cytokine or biological immune response modifiers. These include infliximab (Remicade) - receptor blocker TNF-α, monoklonal recombinant antibodies to human receptor interleykine-6 (tocilizumab - Actemra), monoclonal antibodies against SD-20 receptors of β-lymphocytes (rituximab - MabThera). Treatment with these drugs is carried out only in rheumatology centers.

Patients with RA recommended systematic physical therapy classes, massage courses limb muscles (no joints) and back.

The purpose of massage - a regional improve blood and lymph circulation, redox processes in the muscles, joints and surrounding tissues.

Sanatorium treatment in patients with RA is shown in remission. It is possible and with a minimum degree of inflammatory activity with functional impairment of the joints is not more than II degre (contraindicatedif there are visceral lesions).

Basic sanatorium for the treatment of patients with RA in the Republic of Belarus: "Radon", "named after Lenin", "Dnieper","Letsi","Lakeside","Crane" (resort "Naroch"), "Borovoe".

Medical and social assessment. Contraindicated in patients with RA work with considerable physical and mental stress, adverse weather conditions, in a damp, cold room, in drafts, significant variations of the ambient temperature. Mild stress on the joints functional contrary, is useful.

With a slight variant of RA flow (activity grade 1), with occasional (1-2 times per year) unexpressed exacerbations temporary disability is 14-16 days. Movement and self- limited patients slightly.

Average period of temporary disability in the primary manifestation of RA is 25-30 days, with a high degree of activity - 1.5-2 months.

Medium heavy RA with severe exacerbations more than 2 times per year with a degree of activity II , II-III X-ray stage of the pathological process significantly restricts the movement , self-service patients and professional activity that allows you to send them to the MREB to establish the degree of disability . The average duration of temporary disability at the same time is approximately 25-30 days.

Severe RA, characterized by persistently high activity of the process, severe exacerbations more than 3 times a year, III-IV stage on the X-ray background combined - bath therapy accompanied by a pronounced decrease in patient vital signs, indicates a poor prognosis, and

the apparent disability of patients (II group I or disability) . Temporary disability in the degree of activity of rheumatoid process is on average 40-60 days.

Dispanserization . Patients with RA are subject to medical check-ups rheumatology clinics or district physician.

Observation frequency - 4 times a year.

Examinations by medical specialists: orthopedist, dentist, urologist, gynecologist, otolaryngologist, neurologist - indicated.

Laboratory and instrumental investigations:

• total blood count, urinalysis - 4 times a year (in the treatment of cytostatic - monthly);

• biochemical analysis (bilirubin, AST, ALT, urea, total protein, glucose), rheumatoid factor - 2 times a year;

• X-rays of joints, ECG, X-ray WGC - 1 times a year (on the testimony - often);

• Echo - indicated.

Basic medical and preventive measures:

• continued medical therapy;

• Rehabilitation (mechanic wearing longuet, prosthetics);

• rehabilitation of chronic foci of infection;

• spa treatment (annually if no contraindications);

• in-patient treatment at exacerbation.

Observation periods - for life.

Prevention. The main tasks of prevention are to prevent relapse and progression, restoring functional joint status and disability of the patient. For this purpose, be sure to perform therapeutic measures defined for dispanserization.

Advised to avoid drafts, hypothermia, wear warm clothes, knee pads, warm gloves. For colds, sudden weather changes, during stressful situations renew short-term NSAID. Annually sanatorium treatment.

Reactive arthritis (ICD-10 -M02)

Reactive arthritis (ReA) - immuno-inflammatory joint disease that develops after certain types of infections (most of urogenital and intestinal) and is associated with antigen HLA-B27 (80% of patients).

Etiology of ReA

Infectious agents are:

• Chlamidia trachomatis

• Jersinia enterocolitica and preudotuberculosis

• Salmonella enteritidis

• Schigella flexneri

• Campylobacter jejuni

They do not penetrate into the joint, but launch an immune response resulting in the immune synovitis.

Classification of ReA

Isolated urogenital and postenterocoliticform.

Variants of the course:

• acute (up to 3 months)

• subacute (3-6 months)

• prolonged (6 months - 1 year)

• chronic (over 1 year)

• relapsing

Clinic of ReA

• previous (1 - 3 days to 1.5 months). Or simultaneously developing urethritis, diarrhea

• migratory asymmetric mono or oligoarthritis of the lower extremities (knee, ankle).

Arthritis of the big toe, sausage-shaped defiguration of toes, frequent affection of Achilles

tendon, plantar aponeurosis with severe pain, subcalcaneal bursitis.

• asymmetrical sacroiliitis

Extra-articular manifestations of ReA

• Skin - psoriasiform rashes, keratoderma

• mucous - urethritis, cervicitis, erosion, ulcers on the mucous membranes of the mouth, the foreskin in men

• eye - conjunctivitis (in 70-75 % of patients), iritis, iridocyclitis

• intestines – Proctosigmoiditis

• myocarditis

• Kidney - mild proteinuria, microscopic hematuria,aseptic pyuria

Conjunctivitis is one of the earliest signs of ReA and in combination with urethritis and arthritis is the classic triad of the disease (uretrookulosinovial syndrome - syndrome or Reiter's disease). Reason Reiter's disease - urogenital chlamydial infection, and Reiter's syndrome develops after acute intestinal infection (history, lack of primary lesions of the mucous membranes of the urinary organs, the absence of chlamydial infection in the patient and the sexual partner, the presence persinioza, salmonellosis, dysentery, etc.)

Laboratory diagnostics

• Detection Chlamidia trachomatis in scrapings from the cervical canal and the urethra, enterobacteria in feces;

• PCR (polymerase chain reaction) for verification of chlamydial infection;

• Pyuria, especially in the 1st portions of 3-cuptest;

• Radiography of sacroiliac joints and feet.

Diagnostic criteria for classification of ReA (IV International Workshop on the diagnosis of ReA, 1999).

- Large criteria:

• asymmetry of articular lesions involving 1-4 localization arthritis joints in the lower extremities (requires two of three such signs);

• symptomatic infection of the intestinal or genitourinary tracts (enteritis or urethritis one-third of the day - 6 weeks prior to the development of the disease).

- Minor criteria:

• laboratory confirmation of urogenital or intestinal infection (detection of Chlamydia trachomatis in scrapings of the urethra and cervical canal or detection of Enterobacteriaceae in feces);

• identification of the infectious agent by polymerase chain reaction.

Certain ReA diagnosed with two large and relevant criteria of small and ReA possible when there are two big criteria without corresponding small or one large and one of the minor criteria.

When evaluating urinalysis certain diagnostic value has even a slight leykocyturia, especially in the 1st portions portions of 3-cup test.

Treatment of ReA

In identifying hlamidynoy infection antibiotics should be continued for 3-4 weeks. Course of antibiotic therapy for 10-14 days and is carried out sexual partners even when negative performance having had chlamydia.

Antibiotic therapy (macrolides, tetracyclines, fluoroquinolones)

• 1 g of azithromycin on day, followed by 0.5 g/day 10 days, and then 1 g/day 1 every 5 days during 20 days, or doxycycline 0.1 2 times a day 14 days, then 0.1 night if necessary to 2-3 months;

• rovamycin 3 million 3 times day 10 days, then ciprofloxacin 0.5 to 3 times a day, 10 days, then sumamed 1g/day 10 days (in the absence of effects of treatment with azithromycin or doxycycline);

When enterocolitic ReA antimicrobial therapy is indicated if not seeded fecal pathogens from enterocolitis.

• NSAIDs. Assigned to all patients regardless of age and the nature of the disease in the

doses used to treat other arthritis;

• glucocorticosteroids (high activity);

• sulfasalazine 1 g/day 6-12-18 months (in severe, recurrent or chronic course);

• methotrexate (with sulfasalazine treatment failure).

Medical and social assessment.

The period of temporary disability depends on the severity of the disease (loclisation arthritis treatment efficacy) and the nature of work of the patient. Tentative dates from 25 to 60 days. In severe patients are directed to the MREB to establish the degree of disability.

Dispanserization.

Observation frequency rheumatologist in the first 3 months after hospital - monthly, then - 1 every 3 months during the year after - 2 times a year.

Examinations by medical specialists: urologist, gynecologist, ophthalmologist, gastroenterologist - indicated.

Laboratory and instrumental investigations:

• total blood count, urinalysis, 3-cup test of urine, to nechyporenko test of urine- 1 every 3 months;

• X-rays of the sacroiliac joint, the distal portions of the stop (direct projection) and the heel bone (lateral view) in case of defeat -1 times a year;

• control study of infection that caused articular syndrome - 1,5-2 months after completion of the active antibacterial therapy.

Basic medical and preventive measures: if the signs of urogenital inflammation - tetracycline antibiotics, macrolides, fluoroquinolones, anticandidose drugs, NSAIDs - the liquidation of the joint syndrome, or intra-articular corticosteroids inside - indicated. Physiotherapy - indicated. Sanatorium treatment. Mandatory sanitation urogenital inflammatory focus of sexual partners. Remediation of intestinal infection.

Observation periods and criteria deregistration: until recovery (absence for years of clinical and laboratory signs of the disease).

Class 4. Dyspeptic syndrome and abdominal pain: differential diagnosis and tactics of GP in outpatient conditions. Irritable bowel syndrome: patient diagnosis, treatment and rehabilitation of patients. Acute abdomen, medical tactic.

Session Purpose: To teach students the 5th year of the differential diagnosis, choosing the right tactics and emergency care to patients with abdominal pain .

Study Questions

1. Differential diagnosis of abdominal pain; "Acute abdomen", tactics therapist outpatient conditions.

2. Clinic, diagnostics and tactics practitioner with pancreatitis. Treatment in outpatient settings.

3. Concept chronic colitis. The clinic, diagnosis and tactics practitioner in chronic colitis. Treatment in outpatient settings.

4. Functional gastrointestinal diseases. Dyspeptic syndrome various digestive tract: stomach, intestines, biliary tract, survey the patient in an outpatient setting.

5. IBS: clinical, diagnostic criteria, treatment in outpatient settings.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.

2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.

3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

The appearance of pain in the abdomen, regardless of their intensity should always be considered as a medical emergency requiring immediate on all diagnostic measures, treatment, and in some cases emergency hospitalization. Some clinicians have known that the concept of "acute abdomen" includes not only the disease, leading to peritonitis, but also those that may be complicated by its development.

Examination of the patient with abdominal pain.

Complaints of abdominal pain is necessary to clarify and detail, for this patient asks about pain following features:

• localization and irradiation - ask the patient where it hurts and pain distribution area , show a hand (or hands);

• time of pain, whether such happened before;

• qualitative and quantitative characteristics of pain: persistent or cramping, pain or tolerable if the patient screams , moans, tossing in bed;

• the cause of the pain from the perspective of the patient - eating, physical stress , emotional experience and other factors , as well as to find out what enhances or soothes the pain - eating, vomiting, carminative , antispasmodic reception or analgesics, forced position ;

• establishment of other pathological manifestations accompanying pain - general weakness , fever, diarrheal symptoms (nausea, vomiting, diarrhea, constipation, etc.), cold sweat, perspiration, etc.

Carefully collect the history of this disease. It is important to find out whether the patient was in good health before the pain attack, were there any signs of any disease, previous pain, if the patient does not suffer from any chronic disease (gastric ulcer, duodenal ulcer, gallbladder disease, kidney disease, diabetes) which was a complication of abdominal pain.

Physical examination of the patient should be carried out in the classical terms, not focusing on the abdominal organs, studying in a conventional sequence of all state organs and systems. Thorough examination provides important information for assessing the condition of the patient, the nature of abdominal pain, reveals the symptoms of other diseases, which will greatly help in determining the tactics of the patient. Diagnostically significant appearance of the patient, face, skin and mucous membranes (rashes, color, skin turgor), posture patient behavior in bed or

doctor's office. Identification of certain changes or respiratory and cardiovascular systems can give a rational direction diagnostic search. On examination of the abdomen is necessary to pay attention to its symmetry, participation in breathing, swelling, or on the contrary, is retracted. Thus, when intestinal obstruction is visible hyperprochoresis, peritoneal irritation sign is the absence of respiratory movements of the abdomen.

Abdominal palpation study begins with superficial palpation, allowing reveal diffuse or local tenderness, and abdominal straining, which is based on reflexive tension of the abdominal muscles . There are two types of high tone of the abdominal wall : its resistance and muscle tension or Defense. Each of them has a specific diagnostic value.

Abdominal wall resistance arises partly because of the pain that exists in the affected organs of the abdominal cavity, but mainly because of the pain that causes the doctor's hand, performing superficial and deep palpation especially. At the same time the muscles of the abdominal wall will never become "desklike" or "stone" in the density. Reduce the resistance of the muscles of the abdomen stroking doctor may or distraction patient conversation. The degree of resistance is not constant and changes in the dynamic monitoring of the patients.

"Muscle defance" - in contrast to the resistance of the abdominal wall - always a sign of peritoneal irritation. Pain impulse from the inflamed peritoneum department enters the rear (sensitive) horn of the corresponding segment of the spinal cord passes to the motor neurons of the same segment, forming a stable protective tonic contraction of certain areas of the anterior abdominal wall muscles. Because sensory and motor innervation of parietal peritoneum is carried somatic nervous system, muscular defense arises in that part of the abdomen, which corresponds to the location of the affected abdominal cover. Defanse emerged in response to the irritation of the peritoneum, there constantly. Revealed by superficial palpation, touching her stomach does not affect its expression. Tension of the abdominal muscles can not be overcome and is not reduced by distraction of the patient. A classic example is the "desklike" stomach ulcer perforation in the stomach or duodenum. Muscle defance accompanied by other signs of peritoneal irritation - a positive symptom Shchetkina - Blumberg .

Deep palpation with acute abdominal pain should be performed carefully, starting with the Department of the abdomen, where there is no pain or minimally expressed. When diffuse peritonitis produce deep palpation virtually impossible.

Auscultation of the abdomen was performed to assess intestinal motility. In a healthy person peristaltic waves are heard as a kind of noise every 4-5 seconds. In the pathology of peristalsis can be strengthened (in the wee hours of bowel obstruction), weakened if empty (peritonitis). Sometimes it is possible to listen to the noise of the vascular aortic aneurysm or friction noise peritoneum myocardial spleen.

The analysis of anamnesis and physical examination by a doctor must give the impression to any type of abdominal pain include pain in this patient. Most experts identify the following types of:

- peritoneal (somatic, cerebrospinal) pain;
- visceral pain ;
- reflected pain

Peritoneal pain occur during stimulation of the parietal peritoneum sheet. Due to its metameric somatic innervation peritoneal pain differs precise localization in the place where there was irritation of the peritoneum, which distinguishes it from visceral pain. By the nature of continuous peritoneal pain, severe, painful and there is no cramping. Any concussion peritoneum when moving, touching, breathing intensifies the pain. A distinctive feature of the behavior of the patient - immobility, it takes a forced position on the side with knees bent, on the back, trying not to move or breathe deeply. Often the patient intentionally hamper the doctor palpate the abdomen. Palpation can detect muscle tension anterior abdominal wall and Blumbergsymptom. Auscultation reveals weak peristalsis or complete lack thereof. Typically, the pain is accompanied by peritoneal tachycardia, decreased blood pressure, increased body temperature, increase of intoxication. Peritonitis may be due to inflammatory diseases of the abdominal cavity: appendicitis, cholecystitis, perforation of hollow organs, inflammatory and necrotic changes in bowel obstruction or mesenteric artery embolism. Identification of peritonitis is an indication for immediate hospitalization.

Visceral pain arises in hollow and parenchymatous abdominal organs during stimulation of nerve endings related to the sympathetic and parasympathetic divisions of the autonomic nervous system. Unlike somatic autonomic nervous system does not have receptors specifically irritable. She perceived the changes occurring in the internal organs, which give rise to the feeling of pain. These changes, in the sense of transforming pain include:

- spasm of smooth muscles of hollow organs;
- tensile hollow and parenchymal organs , ligaments tension;
- disturbance of blood supply (ischemia) body .

With various diseases of the pain involved in the formation of one or more units or pathophysiological clearly prevalent one. There are three types of pain - spastic , distenzionnye and vascular . Spastic visceral pain differ in that they have no clear localization . So pain arising from spastic contractions of the small intestine , are felt in the umbilical or epigastric region, regardless of the affected part of the intestine . It was there localized pain with food poisoning , intestinal infections , and even appendicitis , while inflammation of the appendix is not spread on its front cover. More precise localization is characterized by pain that occurred in patients with lesions of the colon, biliary and renal colic . This type of pain is usually accompanied by an increase in vagal tone , which is manifested by vomiting , nausea, cold sweats , slow pulse . Patients in pain behave restlessly tossing in bed , take a knee- elbow position . An objective examination is determined by the ability to breathe belly move indefinitely . Defined muscular resistance and not defance abdominal muscles . Distenzionnye pain observed in lesions as parenchymal and hollow organs . Their cause is a rapid increase in body , leading to a stretching of his capsule . Characterized by constant pain and the accuracy of its localization in the location of the patient's body . Vascular pain are present in chronic ischemic bowel disease (abdominal toad) , thrombosis and embolism of the mesenteric vessels .

Reflected pain called reflex . Irritation resulting in internal thoracic or abdominal cavity, retroperitoneal space , and even the brain and meninges. Reflected the pain does not possess any of the features , so they are difficult to differentiate .

As a result of the first phase of the survey in determining acute peritoneal abdominal pain, shock or mesenteric obstruction patient is subject to immediate hospitalization with outpatient appointment or home.

Those patients who have formulated a preliminary diagnosis , the surgeon should be inspected and, if necessary , and other experts - an infectious diseases , urology, gynecology . While the diagnosis is unclear , it is not recommended to probe patients , put them enemas, stomach wash , prescribe painkillers , laxatives and antibiotics . All patients should be carried out clinical analysis of blood, urine, biochemical determination of bilirubin , creatinine, glucose , transaminases , blood amylase , as well as acetone and urine amylase . Instrumental studies are required fluoroscopy of the chest and abdomen, and electrocardiography . According to the testimony may be performed on an outpatient basis ultrasound of the heart and abdominal organs , fluoroscopic study of the esophagus , stomach, intestine , endoscopy , angiography , CT . At impossibility of rapid outpatient examination of a patient with acute abdominal pain , after inspecting his surgeon , you need to be hospitalized. If the doctor during the examination of the patient at home suspected acute surgical pathology , immediately forward it to the surgical ward , a preliminary diagnosis .

Diseases of " surgical " profile is acute abdominal pain are most common acute appendicitis, perforated gastric ulcer and 12 duodenal ulcer , acute cholecystitis , acute pancreatitis , acute intestinal obstruction , abdominal aortic bundle and ectopic pregnancy . Delayed hospitalization of patients with these diseases can result in peritonitis .

For diseases of "therapeutic " profile , which may manifest acute abdominal pain include: pneumonia, acute myocardial infarction, acute right ventricular failure , acute adrenal insufficiency, hemorrhagic vasculitis , intestinal angina .

Chronic pancreatitis (ICD-10 -K86) - progressive inflammatory- dystrophic lesions of the pancreas, which leads to the progression of fibrosis and organ failure of his exo- and endocrine function.

Chronic pancreatitis prevalence , increased morbidity , incapacity and disability is an important socio- economic problem . In the structure of the gastrointestinal tract it is 5-9 %. Over the past 30 years there has been a global trend to increased incidence of acute and chronic pancreatitis is more than two times.

The main causes of chronic pancreatitis are :

- alcohol abuse ;
- smoking ;
- abuse of fatty foods ;
- diseases of the gastrointestinal tract, primarily the hepatobiliary system (cholelithiasis);
- genetic predisposition ;
- pankreotoksicheskie medicines;
- psycho-emotional stress .

In the development of chronic pancreatitis are two pathogenetic mechanism: inappropriate secretion of pancreatic juice to form in small ducts protein precipitates , which are then calcined and occlusive intrapancreatic ducts and intrapancreatic activation of enzymes of pancreatic juice (trypsin, chymotrypsin, oxidase proelastase, phospholipase A, and lysosomal enzymes). This leads to the necrotic inflammatory process and the subsequent development of fibrosis and fatty degeneration of pancreatic tissue. Chronic pancreatitis is quite often the outcome of acute pancreatitis as a result of violations of ductal patency and fibrosing process in pancreas.

According to the classification of Marseilles - Rome (1989), adopted in European countries, there are the following clinical forms of chronic pancreatitis : obstructive , and parenchymal calcification (inflammation).

Chronic obstructive pancreatitis is caused obtturatsii main (Wirsung) pancreatic duct. Defeat occurs above the obstruction , it is uniform and is not accompanied by formation of stones within the ducts. The clinical picture is the leading constant pain .

Chronic calcific pancreatitis is characterized by irregular lobular lesions of the pancreas, differing in intensity of adjacent lobes. In ducts defined protein precipitates or calcifications , cysts and pseudocysts , atresia and stenosis , as well as atrophy of acinar tissue. Often has alcohol etiology. Characterized by relapsing .

Chronic pancreatitis is characterized by the development of parenchymal foci of inflammation with a predominance of mononuclear cell infiltrates in the areas of fibrosis and replacing pancreatic parenchyma . In this form there are no calcifications and defeat ducts. The clinical picture are leading the progression of endocrine insufficiency and lack of pain .

Usually the first 1-2 attack regarded as acute pancreatitis , all subsequent - as chronic pancreatitis .

Severity of chronic pancreatitis :

• mild course - rare exacerbation (1-2) times a year , short-lived , quickly stoped , moderate pain, pancreatic function is not broken, coprogram OK ;

• average severity - exacerbation 3-4 times a year , lasting pain, pancreatic hyperenzymemia determined , a moderate reduction of exocrine pancreatic function , weight loss, and steatorrhea , and kreatoreya amylorrhea ;

• severe course - frequent and prolonged exacerbation with severe pain and dyspeptic syndromes are marked pancreatic diarrhea , weight loss until exhaustion expressed human exocrine pancreatic function .

The diagnosis of chronic pancreatitis is based on the data history, assessment of risk factors, clinical manifestations of disease, structural changes of the pancreas. The characteristic symptoms of chronic pancreatitis are epigastric pain and left upper quadrant , dyspepsia and weight loss . Pain may radiate to the back, left side of the chest , sometimes wearing shingles vary in nature and intensity. Triggered by eating fatty and fried foods , as well as raw fruits and vegetables . In the pathogenesis of pain is increasing intraductal pressure in violation of the outflow of pancreatic secretion , activation of the kallikrein- kinin system and the influence of kinins on pain receptors , impaired microcirculation in the gland , swelling and stretching of the capsule body . Besides pain patients concerned about bloating , flatulence, nausea and vomiting, not bringing relief . Because of the pain patients limit the diet , leading to weight loss . Furthermore, exocrine pancreatic insufficiency leads to violations of the processes of digestion and absorption in the small intestine . There may be a transient and mild jaundice due to mechanical compression of the common bile duct edematous head of the pancreas. Progression of the process leads to an increase in exocrine insufficiency , which manifests persistent diarrhea. Liquid stool , large, gray with a fetid odor , occurs almost immediately after eating. Along with

exocrine and endocrine insufficiency develops . Arises spontaneously hyperglycemic condition caused by deficiency of glucagon . The emergence of insulin deficiency leads the development of diabetes . Exacerbation of chronic pancreatitis may be accompanied by fever, leukocytosis in blood marked shift to the left formula , increased erythrocyte sedimentation rate . Exocrine insufficiency confirmed by studies coprological : polifekaliya , steatorrhea , kreatoreya , amylorrhea . In the early hours of aggravation increased amylase levels, reaching a maximum by the end of the day , and normalizing to 4 days . Amylase (diastase) urine rises 6-8 hours after the onset of exacerbation and remains elevated up to 2-3 days. Changes in the structure of the pancreas detected by imaging studies . Ultrasound Increased size , uneven contours , structure inhomogeneity , low echogenicity , calcifications , cysts of the pancreas. Endoscopic retrograde cholangiopancreatography (performed only in a hospital) is the "gold standard" of morphological diagnosis of chronic pancreatitis. To confirm the diagnosis and the differential diagnosis can be performed computed tomography, magnetic resonance imaging , biopsy of the pancreas.

The differential diagnosis is carried out : with peptic ulcer disease , gastroduodenitom , cholecystitis , cholelithiasis , irritable bowel syndrome , pancreatic tumors , myocardial infarction. Patients with severe acute exacerbation of chronic pancreatitis (pain , high levels of amylase in the blood , accelerated erythrocyte sedimentation rate, leukocytosis) hospitalized in the surgical ward .

Treatment. In mild acute treatment can be performed on an outpatient basis . In early acute recommended bed rest, cold on the attachment region of the pancreas , the hunger for 1-3 days . Assign to drink 1.5 liters (alkaline mineral water , weak tea without sugar , broth hips) . During the hunger for pain prescribed Creon (0.5) or pancreatin (0.25) 2 capsules 4 times a day . Meals begin with a low-calorie diet with reduced fat and carbohydrates and high in protein. Food should be thoroughly crushed , broth or steamed . Excluded dishes containing extractives, stimulating the secretion of gastric and pancreatic juice , bile secretion. Ingestion of small doses 5-6 times a day . Before each meal should be taken multienzyme preparation (Creon, pancitrat, pancreatin, Mesim forte)

Drug treatment is aimed at suppressing the secretion fermentootdeleniya pancreas, pain relief and normalization of homeostasis disorders . Appointed by the proton pump inhibitors (omeprazole 20 mg 2 times a day , pantoprazole , rabeprazole) or blockers of histamine H_2-receptor (famotidine 20 mg 3 times daily) . Can also be used in the early days of antacids and enveloping means (Almagelum 1 teaspoonful 4 times a day , Maalox 1 tablet 3-4 times a day , Aluminium phosphate gel 1 sachet every 4 hours , etc.) Antacids quickly connect hydrochloric acid in therefore the observed analgesic effect. With a mild pain syndrome appointed antispasmodics : drotaverin 40-80 mg mebeverine hydrochloride (dyuskatalin) 0.2 g, 2 times a day for 2 weeks . And can be administered drugs from the group M- cholinergic receptor blockers : 0.1% solution of atropine sulfate 1 ml or 0.2% solution platifillina gidrotartrata 1 ml subcutaneously. When expressed pain syndrome nonselective used analgesic tramadol (Tramal) 50-100 mg parenteral or oral capsules or rectal suppositories , application of 1% solution of Promedol po1ml intramuscularly. In the early days of acute intravenous shown 5-10% glucose solution , 500 ml, 400 ml reopoliglyukina , gemodeza 300 ml. In the presence of infection in the biliary system the first day shown exacerbation antibiotics (cephalosporins , semisynthetic penicillins , tetracyclines , macrolides) for 7 days.

With the disappearance of symptoms of acute long recommended diet and pancreatic enzyme replacement therapy (ie: Creon , pantsitrat , mezim pancreatin rate of 10,000-30,000 units . Lipase with each meal . Appointment festal, digestal , panzinorma forte such patients is not recommended because as attached in the bile acid composition of the extracts of the gastric mucosa , vegetable cholagogue enhance pancreatic secretion and may contribute to pain. Tselesoobroazno usages vitamins (B6 - amino acids to improve digestion) and vitamin E (£-tocopheryl acetate) , which is the active antioxidant enhances synthesis of endogenous proteinase inhibitors , increases the body's nonspecific defense .

During the period of stable remission can be applied physiotherapy : UHF , ultrasound, inductothermy , paraffin baths projection zone of the pancreas ; water treatment - bath (coniferous , radon) , circular shower .

Medical and social assessment . Average period of temporary disability during exacerbation of chronic pancreatitis depending on the severity ranged from 10-14 to 30-40 days , given the duration of hospitalization .

In severe chronic pancreatitis with severe pancreatic insufficiency , persistent diarrhea , significant weight loss, patients are directed to the MEDC to determine the degree of disability .

Clinical examination is carried out in mild therapist 2 times a year, with moderate and severe 3-4 times a year . At the dispensary estimated patient's general condition , and held a general blood chemistry (total protein , urea, creatinine , glucose, ALT, AST, alkaline phosphatase, GGT , bilirubin) , urinalysis , coprogram, ultrasound of the abdomen , EGD , EKG .

Treatment and preventive measures include dieting , maintaining a healthy lifestyle (avoiding stressful situations , avoiding alcohol , smoking) , enzyme replacement therapy continuously or courses , herbal medicine , physiotherapy , spa treatment with drinking mineral water at the resorts ' Narač " " Letsy" , "Zhdanovichy ", " Bobruisk ," sanatorium " Riverlands ".

Primary prevention is aimed at the exclusion of risk factors for chronic pancreatitis , secondary - aims to conduct activities that prevent exacerbation.

Chronic colitis (ICD-10 -K51) - polietiological disease characterized by inflammatory and degenerative changes in the mucosa and impaired function of the colon. Chronic colitis may result from many causes exo-and endogenous nature, the main of which are carried in the past intestinal infections . The inflammatory process may be supported by helminths and protozoa. To colitis can cause nutritional factors: monotonous, poor dietary fiber and vitamins diet, alcohol abuse , low-calorie diet , haphazard meal. Chronic colitis can develop as a result of poisoning by mercury compounds, arsenic, lead , as well as endogenous intoxication in hepatic failure , hyperthyroidism , gout, uremia , food and drug allergies. Chronic colitis may be accompanied by other diseases of the gastrointestinal tract .

Classification of chronic colitis.

Etiology :

• post-infection ;

• parasite ;

• alimentary ;

• medical ;

• in diseases of other organs of the digestive system ;

• mixed etiology .

By localization :

• total ;

• segmental (rightsided, cecitis, transversitis , sigmoiditis , Proctosigmoiditis) .

The morphological features :

• catarrhal ;

• atrophic ;

• mixed .

Phases of the disease:

• acute phase ;

• phase of partial remission ;

• phase of complete remission .

Severity :

• mild ;

• moderate ;

• severe .

By the nature of functional disorders :

• with impaired motor function of the colon ;

• without dysfunction ;

• with symptoms of intestinal dyspepsia ;

• without the effects of intestinal dyspepsia .

The clinical picture of chronic colitis is dependent on the etiology , localization process , the degree of morphological and functional changes in the intestine. Leading symptoms are abdominal pain and violation of the chair. The pain is aching, pressing, sometimes paroxysmal appearing or increasing after 30-90 minutes after eating, before stool . Localized in different departments , but can be spilled , often radiates to the back , the area of the anus , decreases after

a bowel movement or flatus . When distributing the pathological process in the bowel serosa (pericolitis) or lymph nodes (mesadenitis) pain is constant monotonous character , not associated with food intake , but can be aggravated by motion, after defecation, from warmer .

The most characteristic feature of chronic colitis - a violation of stool. At one and the same patient may has unstable stool , constipation alternating with diarrhea. Urging to stool may disturb immediately after meals , at bedtime. Often patients complain of incomplete emptying of the bowel . There are so-called " locking diarrhea " when constipation is replaced once or twice a profuse diarrhea. Frequent symptom of the disease is flatulence . Bloating celebrated mainly in the lower and lateral parts , accompanied by rumbling , a feeling of heaviness . In some patients the dyspeptic symptoms (nausea, burping air , bitter taste in the mouth , loss of appetite) . Patients concerned about poor sleep , weakness, irritability, emotional lability .

In the absence of total lesions of the colon clinical manifestations associated localization of pathological process . In right-sided colitis or diarrhea cecitis often observed up to 10 times a day, pain in the right part of the abdomen , especially in the iliac region , radiating to the leg, loin . On palpation revealed spasm or extension of the cecum . Transversitis - the defeat of the transverse colon - is characterized by pain , rumbling , feeling of fullness in the middle of the abdomen, appearing immediately after a meal , alternating constipation and diarrhea . With the defeat of the splenic flexure of the colon (Angulitis) patient concerned severe pain in the left upper quadrant, radiating to the back and left side of the chest , unstable stool , a loud rumbling in the right upper quadrant . When percussion in the left upper quadrant determined thympanitis , palpation - soreness. Left-sided colitis (proctitis, sigmoiditis , Proctosigmoiditis) are the most common . This form is characterized by pain in the left iliac region , rectum , tearing in mesogaster worse after a bowel movement. Possible false desires with flatus and mucus. On palpation the sigmoid colon cut , sore , swollen gases. It can be defined dense stool , giving it a lumpy appearance.
The **diagnosis** of chronic colitis based on medical history , clinical data , results coprological and bacteriological examination of feces, confirmed by barium enema , sigmoidoscopy , colonoscopy with biopsy mandatory and morphological study of mucosal biopsies , ultrasound of the colon.

Treatment , given the state of the patient and the phase of the disease , can be performed on an outpatient basis or in the hospital. First of all need gentle mechanical and chemical diet , split meals 4-6 times a day . Supplied food must be in a shabby , cooked by steaming or broth. Should be excluded from the diet, alcohol , milk , salted, smoked and spicy dishes . Patients with severe constipation shown products containing adequate amounts of fiber , dietary fiber , as well as vegetable and fruit juices . Appoint astringent for diarrhea , enveloping and adsorbing agents : De-nol , smectite (1 pack . 3 times a day) , bismuth nitrate (1 g three times a day) . Normalization of motor-evacuation function diarrhea contributes metaclopramide (0.001 3 times a day) . Have a similar effect and decoctions of plants containing tannins - fruits dried blueberries, oak bark , sage leaf , stems alder. Flatulence in fees herbs suitable to add the cumin seeds , fennel , chamomile . In the presence of inflammatory changes of the mucous shown antibacterial drugs . Increasingly used sulfa - ftalazol (0.5 to 4 times a day) , nitrofuranovye drugs - furagin , furadonin (0.1 , 3 times a day) . Treatment 7-14 days. When expressed pain syndrome prescribe antispasmodics (drotaverin , Nospanum) platifillin . With the purpose of bracing used vitamins, ascorbic acid. With the defeat of the rectum have a good effect microclysters - oil (sea buckthorn, rose) , decoction of chamomile. Is effective in the treatment

of physiotherapy - electrophoresis with novocaine platifillina , calcium chloride , UHF , ultrasound, Amplipuls , diathermy, warm baths, hot compress . If necessary, in the range of therapeutic interventions include sedatives , psychotherapy , acupuncture . Sanatorium treatment indicated only in remission (sanatorium " Krynica ", " Riverlands " resorts "Naroch ", " Bobruisk ," " Druskeninkay ", " Essentuki").

Medical and social assessment. During an exacerbation of chronic colitis patients need relief from work. In chronic colitis patient contraindicated moderate trips, daily and night shifts . Limitation of employment may necessitate the establishment of their three disability groups .

Prevention is to prevent acute intestinal infections , elimination of chronic foci of infection , compliance culture and nutrition , healthy lifestyle , Restriction of the use of alcohol, constant physical training and sports , strengthening the nervous system.

Functional dyspepsia (ICD-10 -K30) - a complex of dyspeptic symptoms and complaints in which a careful examination fails to identify the organic changes of the gastrointestinal tract .

Diarrheal disorders are among the most common gastrointestinal complaints. In Europe, they are found in 30-40 % of the population , while 35-40 % are accounted for organic dyspepsia , and 60-65 % - the share of functional dyspepsia . The high prevalence of dyspepsia syndrome defines enormous health care costs for screening and treatment of these patients.

The etiology and pathogenesis of functional dyspepsia remains understudied . Among the causes and mechanisms of the following factors are considered : nutritional errors , alcohol, smoking , neuro- psychological factors , infection Helikobacter pylori, medication, hypersecretion of hydrochloric acid , impaired motility of stomach and duodenum . Dysmotility is the only proven pathogenetic factor of functional dyspepsia . Traumatic and stressful situations identified in most patients with functional dyspepsia . They contribute to disruption of gastric motility and 12 duodenal ulcer . These include disorders adaptability , gastric accommodation to food intake (the ability to relax the proximal stomach after eating under the influence of the growing pressure of food) ; dysrhythmia gastric motility ; antral motility weakening with its subsequent expansion and gastroparesis , impaired antroduodenal coordination. If evacuation function of the stomach is not broken , the cause of dyspepsia may be increased sensitivity reciprocal apparatus of the stomach wall to stretch. In accordance with the decision of the international medical community from 2016, which was called the " Rome criteria 4 "(https://theromefoundation.org/).Diagnosis of functional dyspepsia can be delivered if there are three prerequisites :

1. Patient should feel persistent or recurrent symptoms of dyspepsia (pain or discomfort in the epigastric region, having connection with meals) , exceeding in duration of 12 weeks during the year.

2. Examination of the patient should not be identified organic disease to explain the presenting symptoms.

3. There should be instructions on the disappearance of dyspeptic symptoms after defecation or their relationship with the frequency or nature of the chair , ie there should be no signs of irritable bowel.

Depending on the prevalence of symptoms of dyspepsia are the following options for clinical syndrome of functional dyspepsia:

1. Ulcer -like - is dominated by complaints of pain in the epigastric region.

2. Dyskinetic - dominated by a feeling of fullness and heaviness in the epigastric region , early satiety , nausea .

3. Nonspecific - existing complaints is difficult to attribute to the first or second group.

Clinical manifestations of gastric dyspepsia:

1. Pain in the epigastric region.

2. Discomfort in the central part of the epigastric region - unpleasant subjective sensation that the patient does not mention pain. Discomfort may include the following symptoms :

• early satiety - the feeling of fullness soon after the start of the meal ;

• feeling of fullness - a feeling of heaviness , fullness in the epigastrium ;

• swelling in the epigastric region - a subjective feeling of fullness .

3. Heartburn - a burning sensation in the upper part of the esophagus or throat due to regurgitation of acidic stomach contents.

4. Nausea - the emergence of the urge to empty the stomach, may be accompanied by dizziness, pale skin, sweating, palpitations sometimes.

5. Vomiting - sudden emptying of the stomach associated with stimulation of the vomiting center .

Because the symptoms of dyspepsia may occur in various diseases and not only of the digestive system , the diagnosis of functional dyspepsia can be delivered only when excluded organic nature of dyspepsia , which is found in the following diseases :

• - peptic ulcer and 12 duodenal ulcer;

•- symptomatic ulcers with Zollinger-Ellison syndrome , hyperparathyroidism , heart failure;

• - erosive gastoduodenitis;

• - gastritis .

Hiatal hernia;

- Gastroesophageal reflux disease;

- Medication gastropathy ;

- Cancer of the stomach ;

- Cholecystitis and cholelithiasis ;

- disease of the pancreas ;

- Liver tumors .

Doctor must carefully gather history and physical examination. For a correct diagnosis requires careful laboratory and instrumental studies . "Gold standard" for diagnosis of gastric dyspepsia is endoscopy , which allows you to detect abnormalities of the gastric mucosa biopsy , bacteriological examination to identify Helikobacter pylori. The program includes ultrasound examination of internal organs, clinical and biochemical blood tests , coprogram, fecal occult blood. According to the testimony conduct X-ray examination of the esophagus and stomach.

Treatment of patients with functional dyspepsia syndrome complicated task . Treatment should be comprehensive and include not only prescription drugs , but also includes the normalization of lifestyle and diet regime , if necessary - psychotherapy . Drug therapy is chosen based on the clinical variant of functional dyspepsia . When ulcer -like functional dyspepsia embodiment appointed antacids and antisecretory drugs (H_2 blockers - ranitidine, famotidine and proton pump blockers, omeprazole, lansoprazole, pantoprazole, rabeprazole). When treating patients with functional dyspepsia dyskinetic embodiment designate prokinetics primarily dopamine receptor blockers, and metoclopramide Motilium. Preference is given Motilium as metoclopramide penetrates the blood-brain barrier and causes a large number of patients the side effects (drowsiness , fatigue , anxiety , extrapyramidal disorders) . It conducted the baseline treatment after consulting a therapist if necessary prescribe tranquilizers or antidepressants, as well as other therapeutic treatments. Effective physical therapy and physiotherapy .

Prevention of functional dyspepsia includes mode and quality of food, avoiding harmful habits , sanitation foci of infection , increased physical activity , tempering .

Dispanserization conducted therapist , frequency of observation 2 times per year, with no signs of disease - 1 time per year.

Medical and social assessment . Disabled patients with functional dyspepsia , are generally not disrupted. Temporary disability is usually not more than 3-5 days and is associated with the survey or sudden worsening of symptoms.

Irritable Bowel Syndrome (IBS) (ICD-10 -K58) - functional bowel disorder in which abdominal pain or discomfort is associated with defecation or a change in the behavior and characteristics of bowel disorders bowel movements, without any structural and biochemical changes.

IBS (Rome Consensus IV, 2016):

C. Bowel Disorders

- C1. Irritable bowel syndrome (IBS)
    - IBS with predominant constipation (IBS-C)
    - IBS with predominant diarrhea (IBS-D)
    - IBS with mixed bowel habits (IBS-M)

- IBS unclassified (IBS-U)

- C2. Functional constipation
- C3. Functional diarrhea
- C4. Functional abdominal bloating/distension
- C5. Unspecified functional bowel disorder
- C6. Opioid-induced constipation

Diagnostic criteria for IBS:

• recurrent abdominal pain or discomfort at least 3 days per month in the last 3 months associated with two or more of the following symptoms :

• improvement after defecation ;

• start, which is associated with a change in frequency of stool ;

• start, which is associated with a change in form (appearance) of the chair.

Functional intestinal disorder - a disorder that arose in terms of 6 months or more before contacting the patient and there are 3 or more days per month during the last 3 months.

Supporting criteria IBS are not part of the diagnostic criteria include:

1. Changing the frequency of bowel movements:

• < 3 bowel movements per week;

•> 3 bowel movements per day ;

2 . Changing the shape of the stool:

• lumpy / hard stools ;

• relaxed / watery stools ;

• straining during bowel movements ;

• urgency of defecation or feeling of incomplete evacuation , mucus and bloating .

ICD-10 Classification:

K58 • Irritable Bowel Syndrome ;

• K58.0 Irritable bowel syndrome with diarrhea ;

• K58.1 Irritable bowel syndrome without diarrhea.

Classification of IBS severity :

• mild ;

• Moderate;

• severe.

The wording of the diagnosis:

• Irritable bowel syndrome with constipation-predominant ;

• Irritable Bowel Syndrome diarrhea-predominant .

IBS subtypes

• IBS with constipation - hard or lumpy stools > 25% and relaxed (soft) or watery stools <25% of the number of bowel movements ;

• IBS with diarrhea - relaxed (soft) or watery stools > 25% and hard or lumpy stools <25% of the number of bowel movements ;

• mixed IBS - hard or lumpy stools > 25% and relaxed (soft) or watery stools > 25% of the number of bowel movements ;

• unclassified IBS - insufficient severity of abnormalities in stool consistency for other subtypes ;

• alternating IBS - picture of the behavior of the intestine may vary.

The clinical picture of symptoms. Abdominal pain in IBS is associated with increased visceral sensitivity. Abdominal pain is a required component of the clinical picture of IBS.

It has a wide range of intensity - from mild discomfort , tolerable aching pain to intense constant cramping and even intolerable acute pain that mimics the clinical picture of intestinal colic .

The pain is localized in the lower abdomen , usually in the left iliac region , but can be observed in almost every department up to the epigastric region . Pain is continuously recurrent nature , with periods of exacerbation are most commonly associated with impaired diet , stress factors , fatigue , etc. For patients with IBS characterized by the appearance of pain immediately after eating.

Amid the pain noted bloating , flatulence , increased intestinal motility , diarrhea or slowing chair. Pain subsides , usually after defecation and flatus and not disturb at night. Characteristically, in IBS pain rarely leads to a significant loss of body weight and the development of malnutrition.

Flatulence . One of the most common complaints is heavy and bloated feeling of abdominal distention , increasing its size , the appearance of an audible rumbling in the distance and high gas liberation . These symptoms also occur immediately after a meal together with the appearance of pain.

The auxiliary symptoms , helping diagnose IBS and to determine its clinical variant flow , symptoms of the disorder include transit of intestinal contents and the act of defecation .

Diarrhea . The most reliable sign of diarrhea is considered a form of the chair , not the frequency . Speeded defecation and urgent desires with dense chair are not diarrhea. Typical occurrence of diarrhea in the morning (" morning rush syndrome ") and in the first half of the day , and the absence of diarrhea at night. Stool consistency may gradually change from executed at the first bowel movement until pasty and liquid at the next .

Further, in the chair during the day is usually absent . Stool weight is small - say in IBS problem is not diarrhea as such, and frequent trips to the bathroom . Urging to stool may be mandatory , particularly in relation to emotional stress.

Constipation :

• may consist in a feeling of incomplete emptying of the need to tense . Stool frequency , many patients can not go beyond the norm ;

• according to the Rome criteria adopted for the inclusion of patients in research protocols , is considered pathological stool frequency to three times a day (diarrhea) and less than three times per week (constipation) ;

• constipation patient may complain of a single fecal excretion , which at the beginning of defecation decorated and then mushy and watery even so-called " plug-like " chair. Mucus in stool - quite a common complaint of patients.

Diagnosis of IBS :

- Builds on clinical evaluation of the patient's complaints and their compliance with the criteria of the Rome III, as well as the exclusion of anxiety symptoms , which include :

• weight loss ;

• fever ;

• intestinal bleeding ;

• onset of the disease in the elderly ;

• The emergence of symptoms during the night ;

• constant intense pain in the abdomen, as the only leading symptom ;

• presence of relatives of colon cancer ;

• pathological changes in objective examination (hepatomegaly , splenomegaly , etc.);

• deviations in laboratory studies (anemia , leukocytosis, leukopenia, increased erythrocyte sedimentation rate , changes in LHC) .

- However, be aware that symptoms of anxiety can sometimes be combined with IBS , such as the presence of blood in the stool with hemorrhoids .
- Discharge of blood in the feces , night diarrhea, malabsorption syndrome , or loss of body weight require persistent search for organic disease .

Diagnosis of IBS :

- The diagnosis of IBS is a diagnosis of exclusion. Patients with IBS complain that are not strictly specific. Similar complaints can lay sick with prognostically unfavorable organic pathology : inflammatory diseases of the colon, polyposis , colorectal cancer , chronic pancreatitis , tuberculosis, colon cancer and many others, excluding doctor may stay at the functional diagnosis of the disease ;
- Based on the characteristic clinical picture of the disease . In 80% of cases in young patients under 40 years of IBS with confidence can only be diagnosed on the basis of properly conducted by questioning the patient, detailed collection of complaints and anamnesis ;
- Conduct sophisticated diagnostic studies such as barium enema , colonoscopy with biopsy , computed tomography shows people with symptoms of anxiety or over 50-60 years with newly developed symptoms of IBS , as well as family history of bowel cancer .

Algorithm for the diagnosis of IBS. Patient history → Inspection → Laboratory tests (blood, feces) → → Other Colonoscopy studies necessary for the detection of organic disease → IBS diagnosis (to the exclusion of all possible organic diseases)

The etiology of IBS. According to modern concepts , IBS is a biopsychosocial disorder in which development is based on the interaction of two major pathological mechanisms : psychosocial intervention and sensory- motor dysfunction , ie violations of visceral sensitivity and motor activity of the intestine . The third factor is now attracting the attention of researchers , are persistent neuroimmune injuries that occur after infectious intestinal diseases and considered as a possible cause for the formation of sensory- motor dysfunction .

The pathogenesis of IBS

Aggression factors in IBS :

• psychosomatic disorders that are realized on an axis : brain-gut - brain ;

• increase visceral sensitivity receptors in the intestine to neurotransmitters , gastrointestinal hormones , metabolites, and others;

• violation of the intestinal contents ;

• changes in the quantitative and qualitative composition of the intestinal microflora .

Protective factors in IBS :

• changes in motor function of the intestine (or spastic dyskinesia hypermotoric or alternating);

• Excessive production of mucus.

For patients with IBS revealed several features of the perception of pain and cerebroenteralnyh interactions. Whitehead using balloon dilatation test discovered the phenomenon of visceral hypersensitivity, which does not apply to the perception of somatic pain. Moreover, in his view , hypersensitivity underlies the entire functional gastrointestinal pathology. It was found that the measurement is influenced not only inflating the balloon

material, but an increase in its volume mode is more important. Continuous increase in the accommodation causes rectum while intermittent stretching it does not matter.

The study revealed two kinds of visceral hyperalgesia: to lower the threshold of pain, and with a more intense sensation of pain in the normal threshold of perception , called allodynia .

Intensity syndrome visceral hyperalgesia correlated well with IBS symptoms and balloon dilatation test proved highly reproducible and highly specific for IBS. In this regard, visceral hyperalgesia now considered to be a biological marker of IBS , and balloon dilatation test - specific (95%) and sensitive (70%) method for diagnosing the disease and evaluate the effect of drugs in their trials patients.

Condition for the formation of visceral hypersensitivity is the impact of the so-called sensitizing factors, among which are considered an intestinal infection , in particular, transferred dysentery, psychosocial stress , physical trauma , somehow associated with abdominal pain .

A life filled with stress, plays an important role in the development of symptoms of functional disorders of the digestive system !

Thus, IBS is now regarded as the interaction of important biological and psychosocial factors . Depending on the person in the etiopathogenesis of IBS in varying degrees involved altered motility , visceral hyperalgesia disorder interaction in the " gut brain ", ie abnormal central processing , autonomic and hormonal changes , genetic factors and environmental factors , and psychosocial consequences of post-infectious disorders .

Treatment of IBS. Treatment of patients with IBS is a very difficult task. Start with the individual assessment , the explanations and assurances. It is important to have information about the features of everyday life of the patient , the recent stress (divorce , bereavement or job loss) , - the type and severity of symptoms and their nature may be associated with psychosocial problems . Explanation to the patient the nature of his disease , reassurance that the symptoms do not occur from a life-threatening disease (primarily cancer) - is of paramount importance . For example, the patient should be reassured that the absence of the chair for 2-3 days safely , especially with full bowel movement and no discomfort.

Of lifestyle modifications can expect more benefits than from drugs. Thus, treatment of constipation involves increasing physical activity , the volume of fluid consumed (1.5-2 liters per day)

Food. Patients should avoid restrictive diets . It should identify the product , cause or exacerbate symptoms to limit it or abandon it. Thus, an excessive consumption of fructose , artificial sweeteners such as sorbitol , xylitol , milk consumption may be the cause of diarrhea , pain or flatulence. It will be appreciated that the consumption of fructose in the last 30 years has increased dramatically - it is added to foods, especially beverages .

Dietary recommendations for constipation are to increase intake of dietary fiber - indigestible (ballast) components of plant foods . They increase the volume of feces , increase the content of the liquid and the amount of microflora in the stool, making it easier bowel movements. However, in many patients due to increased fiber gassing increases the symptoms of IBS.

IBS treatment program consists of two phases :

•- primary course ;

•- follow basic therapy .

Execution of the program takes a long time : the duration of the initial course of treatment is at least 6-8 weeks of basic therapy - 1-3 months.

Program selection is determined by the interaction of several factors and depends on the leading symptom (pain / bloating, diarrhea, constipation) , its severity and impact on quality of life of the patient and the nature and behavior of the patient 's mental state.

An important element of therapy is to solve the problem of psychosocial adaptation of patients with the mandatory involvement of the patient in the process of diagnosis and treatment.

Competence, authority and power of persuasion doctor determine contact with the patient , the degree of trust in the doctor and treatment success .

Table 4.1 Pharmacotherapy

Symptoms	group of medications / drugs
Diarrhea	Loperamide
	Acting on the opiate receptors : trimebutine maleate (Tribuxum)
	Antispasmodics: otilonium bromide (Spazmomen), pinaverium bromide (Dicetel)
Constipation	Laxatives swelling action: psyllium seeds (Pinch)
	Osmotic laxative action: lactulose, macrogol, polyethylene glycol, magnesium salts, sorbidol phosphates
	Softening laxatives : liquid paraffin
	Contact laxatives: diphenylmethane derivatives (bisacodyl, sodium picosulphate); ricinoleic acid; anthraquinones (senna, aloe , rhubarb)
	Prokinetics : domperidone, metoclopramide
Pain	Cholinoblockers: hyoscine butylbromide (Buscopan)
	Antispasmodics: drotaverine, otiloniumbromide (40-80 spazmomenmg 3 timesaday 1-2 months). Mebeverine, papaverine, pinaveriumbromide
	Trimebutine maleate (Tribuxum 100 mg 3 times daily), talmetal
	Surfactants
Bloating	Simethicone (Espumizan) diosmektin
The combination of symptoms	Alverin + simethicone
Antidepressants	Paroxetine

The purpose of these drugs reduce pain due to decreased visceral sensitivity , slow motility ; reduces intestinal secretion and increases the amount of mucus , reducing stool frequency and normalizes its consistency.

Life expectancy in IBS. Forecast favorable life in IBS - deaths from the disease has not been described . In 30% of patients show high efficiency of treatment and the total long-term clinical remission observed in 10% of patients .

Class 5 . Differential diagnosis of jaundice and hepatosplenomegaly. Diagnosis and treatment of diseases of the liver, gallbladder and biliary tract outpatient, medical tactics , medical and social assessment , clinical examination , primary prevention . Emergency care in hepatic colic

Session Purpose: To teach students the diagnosis, treatment, dispanserization, examination of disability of diseases of liver and biliary tract in patients on an outpatient basis.

Study Questions

1. Jaundice: concept, classification.

2. Hepatomegaly , splenomegaly, hypersplenism : concept, causes ; diseases accompanied by hepato-and / or splenomegaly , differential diagnosis.

3. Laboratory and clinical syndromes of liver damage (cytolysis mesenchymal inflammation, cholestasis, hepatocellular insufficiency), the clinical significance.

4. Chronic hepatitis and cirrhosis: differential diagnosis of cirrhosis, chronic hepatitis and primary liver cancer. Survey plan of the patient.

5. General principles of treatment on an outpatient basis, medical tactics, medical and social assessment, dispanserization.

6. Chronic cholecystitis , gallbladder dysfunction and dysfunction of the sphincter of Oddi : a plan of patient examination , differential diagnosis .

7. Outpatient treatment, indications for patient consultation the surgeon, medical and social examination, dispanserization. Prevention of diseases of the hepatobiliary system.

8. Emergency care in hepatic colic, medical tactic.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.
3. Harrison’s Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

Jaundice is yellow coloration of the skin. True jaundice appears jaundiced staining of the skin, mucous membranes, and sclera. It develops as a result of the accumulation in the blood of excess amounts of bile pigments - bilirubin and products of its metabolism. If true jaundice icteric coloration detected primarily on the sclera, the sky and the bottom surface of the tongue.

Misleading jaundice due to deposition in the skin yellow pigments nebilirubinovogo origin such as excessive consumption of fruits and vegetables containing carotene - carrots , oranges . When pseudoicterus sclera never painted.

Distinguish different shades of jaundice coloring:

• an orange- yellow color characteristic of intestinal and hepatic lesions ;

• greenish- yellow color, observed with occlusion of the extrahepatic biliary tract and accumulated in the skin due to biliverdin ; with prolonged cholestasis skin gets dark olive color ;

• a light yellow color is observed at gemolitichekih jaundice .

The true cause of jaundice is an imbalance between the formation and release of bilirubin from the body through the liver , which leads to excessive amounts of blood .

Bilirubin is the final breakdown product of hemoglobin. The main part of bilirubin (80-85%) is formed from hemoglobin of erythrocytes. A small part of bilirubin (15%) is formed from heme-containing proteins. In the reticuloendothelial system (RES) heme under the action of hemoxygenase andoxygen is converted into biliverdin, then with a biliverdinreduktase is converted into bilirubin, which enters into the blood, binds to albumin and via the portal vein came to the liver . This is called free bilirubin , it is insoluble in water. In the liver, unconjugated bilirubin is coupled with glucuronic acid and becomes bound bilirubin glucuronide soluble in water. In this form, bilirubin enters the duodenum .

In the intestine under the influence of bilirubin related intestinal microflora is hydrolyzed to form urobilinogenov which are oxidized and converted to urobilin stercobilin . Approximately 20 % of urobilinogen is absorbed into the blood in the ileum and in the form of urobilin the portal vein to the liver and excreted by it again into the bile . Very small part of urobilin excreted by the kidneys in the urine. The remainder of urobilinogen enters the large intestine and excreted in the feces as sterkobeline , giving it a brown color .

In human blood, the majority of bilirubin is insoluble in water free form. It is not filtered by the kidneys and therefore absent in the urine. The bound bilirubin may be filtered by the kidneys , but because of its small amount of blood in the urine given. This bilirubin is still referred to as a straight line.

Modern classification divides all of jaundice on the mechanism of their occurrence in 3 groups.

Suprahepatic jaundice associated with increased formation and accumulation of bilirubin in the blood .

This type of jaundice develops during hemolysis , which is accompanied by increased formation of bilirubin. Accompanied by more or less severe anemia. For hemolytic jaundice

characteristic yellowness of sclera and fair skin with lemon - yellow tinge , no itching , pallor . Jaundice appears or worsens due to hemolytic crisis . For this characteristic of jaundice due to indirect hyperbilirubinemia fraction. In a large number of urine urobilin , bilirubin is absent. In many sterkobelina feces , he dark . In the overall analysis of the blood revealed normochromic anemia and reticulocytosis .

An objective examination can determine an enlarged spleen .

Cause hemolytic jaundice are hemolytic anemia :

1. Hereditary anemia :

• microspherocytosis hereditary disease (Minkowski-Chauffard syndrome);

• hereditary hemolytic anemia associated with deficiency of glucose -6- phosphate dehydrogenase ;

• hemoglobinopathies (sickle cell anemia , thalassemia)

2 . Acquired anemia :

• immune hemolytic anemia;

• hemolytic anemia with constant Paroxysmal nocturnal hemoglobinuria(Marchiafava-Micheli Syndrome)

Hepatic (parenchymal) jaundice, which is based on the damage of hepatocytes and bile capillaries. Divided into hepatocellular, cholestatic and enzimopaticheskuyu

Jaundice is caused by isolated or combined seizure disorders , binding and excretion of bilirubin . Hepatocellular jaundice due to impaired permeability or hepatocyte membrane integrity with the release of bilirubin in the blood, which is observed in acute and chronic liver diseases. This type of jaundice develops quickly and the skin has a yellow-orange hue. In the blood revealed elevated bilirubin , mainly associated , as well as free , elevated transaminases . Appears in the urine bilirubin , high urobilin . Stercobilin in feces may be somewhat reduced.

Cholestatic liver jaundice in violation of the flow of bile into the duodenum , due to destruction of the intrahepatic bile ducts . They are characteristic of primary biliary cirrhosis, primary sclerosing cholangitis , cholangiocarcinoma . Cholestatic jaundice may also occur during pregnancy , secondary cholestatic hepatitis, benign recurrent intrahepatic cholestasis , acute cholangitis , syndromes Ano Rössle and Mc - Macon Tanhauzera . For this jaundice characterized by complaints of itching . In marked increase in blood bilirubin , alkaline phosphatase , GGT and increased concentration of bile acids. Simultaneously appears hypercholesterolemia and increase in the concentration of bile acids.

Enzymatic hepatic jaundice - linked to shortages of enzymes responsible for the capture, conjugation or excretion of bilirubin hepatocytes . This is mainly due to hereditary disease. They fall into the disease with increased indirect bilirubin (Zhiliber syndrome , syndrome Tracing syndrome Crisler - Najjar) and with increased direct bilirubin (syndrome Dubin -Jones , Rotor syndrome)

Subhepatic (obstructive) jaundice.

At the heart of its development is a mechanical barrier drain bile from the liver due to obstruction , compression or stricture of the extrahepatic biliary tract . The mechanism of its occurrence is in violation of direct bilirubin excretion related to the duodenum . Increased pressure in the overlying biliary bilirubin leads to diffusion through the walls of dilated bile capillaries. Obstructive jaundice develops gradually , the skin becomes greenish , itchy . In the blood accumulate all components of bile, bilirubin, cholesterol, bile kislomy , increased alkaline phosphatase activity . Urine gets the color of dark beer , feces discolored . In the blood of hyperbilirubinemia due to direct fraction. Level of alkaline phosphatase is the most informative marker for differential diagnosis of cholestatic jaundice.

Causes of obstructive jaundice :

• neoplastic lesions - cancer of the common bile duct , gall bladder cancer , head cancer , pancreatic cancer, major duodenal papilla , liver tumors ;

• non-tumorous lesions - holeliteaz , cholecystitis, common bile duct stricture , pseudotumor chronic pancreatitis, papillary stenosis .

Hepatomegaly - enlarged liver , which occurs as a consequence of any disease affecting the function of the body , which can be determined by percussion, palpation or instrumental methods .

In case of injury occurs tissue swelling , development of tumor formation , accumulation of metabolic products . As a result, the liver is increased in size.

Causes of hepatomegaly can be divided into three groups depending on what caused the increase of liver disease : liver disease , metabolic disorders , cardiovascular disease . The last stage of any chronic disease causing hepatomegaly , is cirrhosis. Upon detection of a patient hepatomegaly assigned a number of laboratory and instrumental investigations. These include : clinical and biochemical analysis of blood , blood clotting parameters , ultrasound, CT or MRI of the abdominal cavity . In some cases, a diagnostic laparoscopy , liver biopsy .

Differential diagnosis. Hepatomegaly may be caused by venous congestion in the liver (congestive heart failure, hepatic vein obstruction); infectious diseases (viral hepatitis, cirrhosis, leptospirosis, hydatid cyst , liver abscess) , alcoholic hepatitis and cirrhosis , drug autoimmune etiology ; obstruction of bile ducts (cholelithiasis, pancreatic tumor , sclerosing cholangitis , etc); tumors - hepatocellular carcinoma , metastases , leukemia .

Splenomegaly - enlargement of the spleen .

Hypersplenism - combination of increased spleen cell with increasing amounts of elements in the marrow and a decrease in the formed elements in the peripheral blood.

Causes of splenomegaly :

• venous stasis - cirrhosis, extrahepatic form of portal hypertension ;

• immune response in infective endocarditis , infectious mononucleosis , and others;

• increased destruction of red blood cells in congenital spherocytosis or thalassemia ;

• storage disease ;

• tumor ;

• undiagnosed injury.

Diagnosis of liver disease requires a detailed survey and overhaul patients. Need a chronological sequence of development of clinical and functional syndromes , representing a combination of medical history , physical and laboratory - instrumental signs .

Cytolysis or cytolytic syndrome - a violation of the integrity of the membrane of hepatocytes and their organelles due to exposure to viruses, toxins , drugs , immune effects or cancer. Report of membrane integrity leads to composite parts exit the cell into the extracellular space , thereby the entering water and sodium into the cells. They swell , violated their structure and function , developed hepatocellular failure. Biochemical methods allow to detect early pregnancy cytolysis.

In the blood and increases the concentration of unbound bilirubin , increases the activity of organ-specific enzymes - lactate dehydrogenase (LDH) , alaninaminotransferase (ALT) , aspartate aminotransferase (AST) , aldolase , alkaline phosphatase .

Mesenchymal inflammation - sensitization processes expression and proliferation of T and B lymphocytes with production of antibodies, as well as leukocytes, and macrophages. Part of sensitized T and B cells into a "memory cells" persist for a long time, and capable of rapid antitelobrazovaniyu at the second meeting with the antigen. The most important diagnostic criteria mesenchymal inflammation are fever, increased erythrocyte sedimentation rate, increase in serum £ 2 and γ- globulins. To clarify the nature and severity of the syndrome applied immunological and morphological methods.

Cholestasis - a violation of the outflow of bile from the hepatocytes to the accumulation of its components in the liver and blood. Often intra-and extrahepatic cholestasis. Intrahepatic cholestasis can occur at both the hepatocyte and bile duct level. At the heart of extrahepatic cholestasis is a violation of the outflow of bile by the mechanical factor. In the blood, and therefore in the urine comes conjugated bilirubin. Reduction or elimination of revenue sterkobilina the intestine leads to discoloration of feces , and the disappearance of urobilin in the urine. Receipt of bile acids in the blood (cholehemia) accompanied by pruritus , insomnia , emotional lability, hypotonia. Violation of excretion of cholesterol leads to hypercholesterolemia, to its subcutaneous and intradermal deposition (xanthomas and xanthelasma) . Malabsorption of vitamin A - leads to a reduction of twilight ; Vitamin D - to arthralgia, osteoporosis , tooth decay , ; Vitamin K - reduce vit. K dependent coagulation factors and hemorrhagic syndrome.

Hepatocellular insufficiency - a consequence of the progressive reduction of functioning liver parenchyma by necrosis and degeneration of hepatocytes. This leads to the accumulation of toxic products in the blood , biologically active substances , hormones . What matters most is tserebrotoksichkeskie products (ammonia , mercaptans , phenols) , hypoglycemia (subject to its depletion of glycogen in the liver) , respiratory alkalosis (hyperventilation due to toxic irritation of the respiratory center) , hypokalemia , and hypomagnesemia causing apathy and lethargy ,

increased activity of vasodilating substances leads to a drop in blood pressure and collapse. Clinical signs of liver - cell deficiency often grow slowly , sometimes catastrophically progress , which leads to hepatic coma .

Chronic hepatitis (ICD-10 -K73) - polietiological liver disease characterized by varying degrees of severity of hepatocellular cirrhosis and inflammation, lasting for 6 months or more.

The most common cause of chronic hepatitis - is transferred acute hepatitis B, C, D. One of the most frequent hepatic lesions observed in patients with autoimmune hepatitis. In addition, the causes of chronic hepatitis may be drugs, alcohol and toxic substances.

According to the International Classification of chronic hepatitis (Los Angeles, 1994) was isolated :

• autoimmune hepatitis ;

• chronic hepatitis B , C, D ;

• chronic viral hepatitis, characterized by not otherwise;

• Chronic hepatitis , not classifiable as a viral or autoimmune ;

• chronic hepatitis drug.

In a separate category stands for alcoholic liver disease.

The degree of activity is determined on the basis of clinical and biochemical data, primarily ALT activity:

• Minimal - ALT activity increased by no more than 5 times;

• moderate - ALT activity, increased by 5-10 times;

• high - ALT activity more than 10 times higher than normal .

For semi-quantitative evaluation of the degree of fibrosis in the morphological study are the following steps:

0 - no fibrosis ;

1 - mild portal and periportal fibrosis

2 - moderate fibrosis with the presence of proto- portal septa

3 - fibrosis with porto- central septa

4 - cirrhosis

The clinical picture of chronic hepatitis B depends on the degree of liver dysfunction . In the initial stages can vary only laboratory parameters .

Cirrhosis of the liver (ICD-10 -K74) - a chronic, progressive disease polietiologicheskoe characterized by the development of fibrosis and a significant decrease in the number of

hepatocytes , reconstruction of normal liver structure and development in subsequent liver failure and portal hypertension.

Cirrhotic patients complain of fatigue, loss of appetite , impaired consciousness and behavior, bloating, dull or aching pain in the abdomen, jaundice , swelling of the legs, bruising , nosebleeds , decreased sexual desire .

At survey characteristic of cirrhosis " hepatic signs" spider telangiectasia on the skin , body, palmar erythema . Characteristic syndrome is a " head of Medusa " - overflow veins of the anterior abdominal wall , ascites , bruising on the skin.

Differential diagnosis. In chronic hepatitis B , in contrast to cirrhosis no signs of portal hypertension. Also helps biopsy method : in chronic hepatitis preserved lobular structure of the liver . It is often difficult to carry out differential . diagnosis between cirrhosis and primary liver cancer , as both of these diseases have similar symptoms . Differential diagnosis distinguish instrumental studies (ehogepatogramma , angiogepatogramma) , allowing to establish the basic character of liver damage . In addition to liver tumors is not characterized by a significant increase in the spleen and the presence of hepatic sigmatov (especially telangiectasia) , so often observed in cirrhosis .

Plan evaluation of patients suspected of having chronic hepatitis or cirrhosis of the liver include:

1 - assessment of the data history of the disease , family history .

2 - data evaluation functional inspection

3 - data laboratory and instrumental examinations:

• total blood count;

• urinalysis ;

• biochemical blood test (total protein , creatinine , bilirubin, cholesterol, ALT, AST, GGT);

• Ultrasound of the hepatobiliary system ;

• EGD .

Treatment of patients with chronic hepatitis , compensated cirrhosis is preferably carried out on an outpatient basis . During exacerbation of chronic hepatitis with a high degree of activity shown hospitalization. Normal mode , but with limited functional activity. Prerequisite therapy is to normalize lifestyle: dieting (diet 4-5 times a day) , with the exception of alcohol , smoking, stress , limiting exposure to the sun , with the exception of vaccinations .

To improve the digestive processes are assigned bacterial preparations (lactulose, bactisubtil, biofllor, Linex, etc.), enzyme preparations, not containing bile acids (pancreatin mezim forte, Creon, etc). Widely used hepatoprotectors: lipoic acid, essentiale, hepatil or Geptor, tavamine, ursosan, thiotriazoline. For the prevention of bleeding from esophageal varices and gastric portal hypertension in patients with liver cirrhosis effective drugs are non-selective β-blockers (propranolole, nadolole) to the maximum tolerated dose (320 mg and 240 mg / day,

respectively) and nitrates prolonged action (isosorbide 5-mononitrate, isosorbide dinitrate). Application of β-blockers, patients with cirrhosis should be appointed for life and immediately after the verification of the diagnosis.

Treatment edematous ascitic syndrome begins in the hospital and continues on an outpatient basis . Recommended salt-free diet , fluid restriction to 1 liter , regular weighing of the patient , monitoring daily diuresis . Please appoint aldosterone antagonists (veroshpiron) and then added if necessary saluretics (furosemide, hydrochlorothiazide and others). Effective and a combination thereof.

When one diuretic treatment once a month is necessary to monitor blood levels of potassium, sodium , creatinine, liver enzyme levels , ECG, neuro- psychological status of the patient.

Vitamin (B_1, B_2 , B_6 , vitamin E) are necessary.

Medical and social assessment . Patients with chronic hepatitis and liver cirrhosis is contraindicated in heavy physical labor, work with toxic substances , nervous tension . Sustainable employment through the MAC. If employment involves decreasing the qualifications patients are referred by MREB to establish three disability groups . Temporary incapacity occurs during exacerbation and depending on the degree of activity is 10-30 days.

Dispanserization carried therapist 2 times a year, one gastroenterologist once a year. Laboratory control two times a year: complete blood count, blood chemistry (bilirubin, AST, ALT, GGT, alkaline phosphatase , albumin, urea, creatinine , prothrombin index) . Ultrasound, ECG 1 time per year; EGD - 1 every 2 years . Patients with liver cirrhosis EGD performed depending on the esophageal varices .

Chronic cholecystitis (ICD-10 -K81) - polietiological inflammation of the gallbladder .

More common in women . May be due to acute cholecystitis , but usually develops gradually as a primary chronic disease. Chronic inflammation of cholecystitis often represents the initial stage of calculous cholecystitis .

Plays a leading role infection that enters the gallbladder from other organs . Infection often occurs by ascending bile from the intestine. This contributes to a loss or reduction in the tone Oddi's sphincter. Otherpredisposing factors are diet, family history , overweight , diabetes, gipodininamiya stress .

Classification:

• chronic inflammatory cholecystitis
• calculous cholecystitis .
Severity :
• mild (exacerbation 1-2 times a year);
• moderate (aggravation 5-6 times a year);
• severe (exacerbation 1-2 times a month) .
On the stage of disease :
• aggravation ;

• remission

By the nature of the flow :

• latent ;

• relapsing .

For complications :

• uncomplicated ;

• complicated .

The clinical picture of chronic cholecystitis manifestation depends on the severity of the inflammatory process in the gallbladder. During an exacerbation common symptom is pain in the right upper quadrant, sometimes prradiruyuschaya in the right shoulder blade . Pain worse after taking spicy fatty foods, exercise. Sometimes there is an increase in body temperature. Abdominal palpation , tenderness at the point of projection of the gallbladder. Pain occurs at the surface and deep palpation (symptom Murphy) palpation, new or worsening pain in percussion on costal arch. In some cases, pain in the right upper quadrant accompanied by irradiation to the heart that can cause typical angina, called cholecystocardial syndrome, first described by S.P. Botkin, which requires an assessment of ECG dynamics. Characteristic symptoms of chronic cholecystitis is bitterness in the mouth , loss of appetite , heartburn, vomiting may occur and other diarrheal symptoms.

Diagnosis of chronic cholecystitis is based on a comprehensive examination of the patient .

In the clinical history of blood in acute noted leukocytosis, eosinophilia, increased erythrocyte sedimentation rate. Biochemical study reveals increased ALT, AST and GGT, bilirubin, acute-phase proteins presence. Definite value in the diagnosis of a fractional sensing and study of bile. In bile revealed a large number of flakes of mucus , columnar epithelium . Biochemical study of bile to determine disruption of normal relations ingredients bile characteristic predkamennyh states (reduction of bilirubin and cholic acid) . Bacteriological study of bile allows you to set the bacterial flora and its sensitivity to antibiotics. Confirming the diagnosis of chronic cholecystitis is ultrasound, in which there are thickened gallbladder wall with irregular contours , with different contents of inhomogeneous inclusions.

The differential diagnosis spend with functional disorders of the biliary tract , peptic ulcer disease , bowel disease , gallbladder cancer .

Plan of patient’s examination

1. Laboratory Methods

a) total blood count ;

b) biochemical blood test : bilirubin and its fractions, ALT, AST, GGT , sugar , amylase ;

c) urinalysis ;

g) coprogram, fecal helminth eggs, Giardia cysts.

2 . Instrumental methods:

a) ultrasound investigation;

b) EGD

c) X-ray examination (oral cholecystography in / vennaya cholecystocholangiography)

3. Consultation gastroenterologist , surgeon, gynecologist.

If the patient cholecystitis complications such as jaundice , empyema , perforation of the gallbladder is necessary to consult a surgeon , as patients in need of immediate hospitalization in the surgical department of the hospital .

Treatment of chronic cholecystitis , usually on an outpatient basis . Leading therapeutic measure is diet therapy, which aims to ensure the normal byle excretion. Food should be chemically and mechanically gentle , welcome to frequent , 4-6 times a day , with the inclusion of cereals, vegetable soups , cooked meat and fish, vegetables and sweet fruits.

When expressed using peripheral pain syndrome cholinolimimetics M (0.1 % solution of atropine sulphate 1 ml, 0.2 % solution for platyphylline 1.2 ml, 2 % solution buscopan subcutaneously 1 ml) myotropic antispasmodics (2% solution of papaverine hydrochloride 2 -4 ml, 2 % solution of no-spa 2-4 ml).

Pronounced antispasmodic and cholagogue action has mebeverin hydrochloride (Duspatalin), 2 times a day for 30 minutes . before eating. Widely used drugs in mixed allocholum , cholosas , cholenzim , festal, digestal and sedatives .

In cases when the inflammatory process in the gallbladder basis of medical treatment is antibiotic therapy. Are the most effective drugs of fluoroquinolones norfloxacin 0.4 2 times a day , levofloxacin 0.5 2 times a day , ciprofloxacin, 0.5 , 2 times a day macrolides : azithromycin 0.5 1 times a day , clarithromycin 0.5 2 times a day. Semisynthetic penicillins , ampicillin, oxacillin less effective , so it's best to use a secure form of Augmentin , amoxiclav .

Medical and social assessment . The average duration of disability in exacerbation of the disease is 10-14 days.

Dispanserization. Patients with chronic cholecystitis are registered with a local therapist. Multiplicity of observations depends on the severity of the disease . Mild 1 times per year, with frequent exacerbations 2-3 times a year . Conducted clinical and biochemical studies , ultrasound of the liver and biliary tract 1 per year EGD 1 every 2 years .

Prevention . Primary prevention involves a healthy lifestyle, good nutrition, sufficient physical activity , smoking cessation and alcohol abuse , strengthening the nervous system and avoiding stressful situations. An important condition for the prevention of chronic cholecystitis is sanitation foci of infection.

Secondary prevention is carried out implementation of measures to clinical examination of patients.

Dysfunction of the gallbladder and sphincter of Oddi (ICD-10 -K83) - is inconsistent , inadequate or excessive contraction of the gallbladder and sphincter of Oddi , and manifest violation of the outflow of bile from the gallbladder and common bile duct into the duodenum.

According to the Rome criteria IV (2016) Functional disorders are divided into:

E. Gallbladder and Sphincter of Oddi disorders

- E1. Biliary pain
    - E1a. Functional gallbladder disorder

- E1b. Functional biliary sphincter of Oddi disorder

- E2. Functional pancreatic sphincter of Oddi disorder.

Dysfunction of the biliary tract is divided :

etiology :

• primary ;

• secondary .

functional state :

• hypertension (hypermotoric);

• hypotension (hypomotoric) .

Primary dysfunction develops due to disorder neurohumoral regulatory mechanisms. Secondary - as a result of liver, intestine , pancreas, systemic diseases , as well as in gormalnyh disorders. Most suffer from women aged 20-40 years.

Clinic . Hyperkinetic form gall bladder dysfunction manifested acute kolikopodobnoy pain in the right upper quadrant , radiating to the right shoulder , sometimes in the left half of the thorax.

Pain occurs after errors in diet , physical activity, mental and emotional stress . During an attack of pain may be nausea, vomiting, headache, sweating . Pain syndrome is caused by increased pressure in the gallbladder , which is reduced when hypertension occurred sfinklera Oddi . On palpation may experience soreness in the projection of the gallbladder. Changes in clinical and biochemical blood tests available. Ultrasound study determined size reduction and accelerated gallbladder emptying .

Hypokinetic function of the gallbladder causes pain in the right upper quadrant without a clear irradiation . There may be a feeling of heaviness and fullness in the right upper quadrant . Patients complain of loss of appetite, bitter taste in the mouth, bloating , nausea , belching . Palpation revealed a small pain in the projection of the gallbladder. Development of hypokinetic gall bladder dysfunction associated with decreased formation of cholecystokinin in gut wall 12perstnoy that slows the motor function of the gallbladder. Gallbladder ultrasonography increased, pear-shaped , slowly and poorly drained . Changes in clinical and biochemical blood tests not detected .

Sphincter of Oddi dysfunction - functional impairment of its contractile power, preventing the normal flow of bile and pancreatic secretions into the duodenum. Observed after cholecystectomy , recurrent idiopathic pancreatitis , very rarely in patients with biliary tract intact . Develops spasm, less muscle dilatation of sphincter of Oddi or its components - sphincters gall and pancreatic ducts, and disrupted the flow of bile pancreatic secretion in 12perstnuyu intestine , increases the pressure in the bile duct or pancreatic duct . Depending on the violation of an outflow of isolated biliary , pancreatic and mixed violation sfinkera Oddi . The clinical picture depends on the place of infringement of outflow of bile and pancreatic

secretions. When biliary dysfunction pain similar pain in gall bladder dysfunction . When pancreatic - pain in the left upper quadrant radiating to the back . When mixed variant of the pain is localized in the epigastrium and left upper quadrant, is the nature of shingles .

The **differential diagnosis** is carried out with cholecystitis , pancreatitis , peptic ulcer and 12PK , myocardial infarction.

Treatment in outpatient setting. The goal of treatment is to restore normal bile flow and pancreatic secretion . Important role in this is played by diet therapy . When hyperfunction recommended split meals with the exception of products that cause contraction of the gallbladder (fat, meat and fish products , alcohol) . When hypofunction recommended diet with enough cholagogue substances (black rye bread , weak broth, vegetables, fruits) . Drug therapy is performed differently according to the biliary tract function . When hyperfunction gallbladder and Oddi sfinkera appointed myotropic antispasmodic drugs mebeverine hydrochloride (0.2 g, 2 times a day before meals) , Odeston (0.2-0.4 g for 30 minutes before a meal), and 0.0005 g of nitroglycerin under the tongue) , which quickly relieves spasm of the sphincter of Oddi and is indicated for patients with concomitant cardiovascular disease. For pain relief used a combination of drugs baralgin , spazgan etc. In case of disfunction of pancreatic sphincter of Oddi for each type of food at appointed enzyme preparations - Creon , pancreatin , mezim forte . When hypofunction appointed cholagogue preparations - chofitol, holosas , solutions of xylitol , sorbitol , olive oil, teas , yarrow , parsley . Shows the assignment of sedative and tranquilizing drugs , rational psychotherapy and physiotherapy treatments . Treatment was carried out for 2-4 weeks.

Recommended the use of methods of physiotherapy and exercise therapy.

Employability patients with dysfunction of the biliary tract usually is not broken. The prognosis is favorable .

Dyskinesia in patients with cholecystitis and nekalkulunym shown sanatorium treatment no less than 2 months after the exacerbation. Such patients are shown balneomud resorts "Naroch ", " Krynica ", " Druskininkai ", " Essentuki" , "Truskavets" etc.

Prevention of diseases of the hepatobiliary system

Primary prevention is aimed at compliance with the principles of a healthy lifestyle , prevention of alcohol intake and exposure to hepatotoxic substances. Of great importance is the definition of screening healthy segment of the population of asymptomatic carriers of hepatitis B virus health workers and persons in contact with patients with viral hepatitis , immunizations against hepatitis B.

Secondary prevention aims to achieve long-term remission of the disease , the struggle with relapses and complications. Important work and rest , diet , abstinence from alcohol .

Liver colic

Colic can be caused by cholelithiasis , which for a long time may be asymptomatic . Pain occur most often after errors in diet during exercise , psycho-emotional stress , bumpy ride. The pains are intense constant cutting, stitching , tearing character. The cause of the pain attack is a

denial of the stone in the neck of the gallbladder or common bile duct . Pain localized in the right upper quadrant and epigastric region (due to irradiation in celiac plexus) , give to the lumbar region right shoulder , right shoulder girdle (irritation of the right phrenic nerve branches), rarely occurs irradiation of pain in the heart area , simulating angina (cholecysto - cardiac syndrome) . Frequent attacks of biliary colic accompanied by nausea and repeated vomiting with bile , not bringing relief to the patient . Duration of colic from several minutes to several hours. Patients with restless, frequently change posture , trying to find a comfortable position , which reduces the intensity of pain. Body temperature during the attack remains normal , there is a moderate tachycardia up to 100 per minute Language moist, coated with a whitish bloom. On examination, attention is drawn to some bloating, the right half of the abdominal wall behind in the act of breathing . On palpation of the abdomen there is a sharp pain in the right upper quadrant , especially in the projection of the gallbladder. Protective muscle tension is absent or expressed slightly positive symptoms Ortner - Grekova (effleurage sharp pain on the right costal arch) Musso - St. George (pain with pressure between the legs of sternocleidomastoid muscle). No symptoms of peritoneal irritation . In the analysis of blood leukocyte count is normal or slightly elevated .

Emergency aid during the attack includes antispastic and analgesic subcutaneously 1 ml of 0.1 % solution of atropine sulfate , 2 ml of a 2 % solution of papaverine hydrochloride , 2 ml of 2% drotaverine solution, intramuscular or intravenous aminophylline solution , intramuscularly 2 ml of 50 % dipyrone solution , nitroglycerin per os. In severe cases, the injected 1 ml of 1 % solution of morphine in combination with hydrochloride atropine to reduce the effect of morphine on the sphincter of Oddi. Patients with hepatic colic should be hospitalized in the surgical department to monitor the surgeon and determine the indications for operative treatment.

Class 6. Urinary syndrome : differential diagnosis . Methods of diagnosis of kidney disease in an outpatient setting . Treatment of chronic pyelonephritis , glomerulonephritis, and chronic renal failure in an outpatient setting , medical tactics , medical and social assessment , clinical examination , primary prevention . Emergency care in renal colic

Session Purpose: To teach students diagnosis, differential diagnosis, treatment strategy, examination of disability and prevention of kidney disease in patients on an outpatient basis .

Study Questions

1. Notion of urinary syndrome, its features in glomerulonephritis, pyelonephritis , urolithiasis, urethritis , cystitis , bladder cancer , renal disease .

2. Diagnostic search algorithm with urinary syndrome. Plan examination of the patient with chronic pyelonephritis , chronic glomerulonephritis , chronic renal failure in an outpatient setting. Methods of diagnosis of kidney disease in an outpatient setting . Indications for hospitalization of patients.

3. Approach to the Patient with chronic renal failure .

4. General principles of treatment of patients with chronic pyelonephritis , chronic glomerulonephritis , and chronic renal failure in an outpatient setting .

5. Medico-social examination (justification and timing of temporary disability, the indications for rational employment of patients sent for MREB), dispanserization.

6. Prevention of chronic pyelonephritis, chronic glomerulonephritis, and chronic renal failure.

7. Renal colic: clinical, emergency outpatient care, medical tactic.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
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3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

Urinary syndrome - clinical and laboratory symptoms, including proteinuria, pyuria , red blood cell , cylindruria . These symptoms may be present in full strength (all 4 symptoms), and separately , except cylindruria (not occur in isolation) , or in combination (proteinuria and leycocyturia ; leycocyturia and erithrocyturii) . Due to the fact that the components are not part of the syndrome common pathogenesis , differential diagnosis is carried out within each symptom.

Proteinuria - urinary protein excretion . We practically healthy person negligible amount of protein in the urine (10-100 mg / day) , but in the uroanalysis given quantity of urine is not defined . Most experts considered acceptable in a healthy person pro- proteinuria in an amount of 60 mg of protein per 1 m^2 of body surface . It should be noted, however, that the discovery of a single morning urine sample minimum amount of protein (0.033 g / l) and even traces of it , especially if it is confirmed by repeated analyzes of drive requires investigations to rule out renal disease .

Allocate the following criteria for classifying proteinuria.

- In terms of urinary protein excretion :

• Minimal proteinuria (up to 1.0 g / d or protein concentration in the urine from 0.033 to 1.0 g / l);

• moderate proteinuria (from 1.0 to 3.0 g / d or protein concentration in the urine of from 1.0 to 3.0 g / l);

• severe or massive proteinuria (greater than 3.0 g / d or protein concentration in urine is greater than 3.0 g / l).

- For the duration of urinary protein excretion :

• constant;

• transient .

- According to the qualitative composition of urinary protein excretion :

• low selective or nonselective (emphasis of macromolecularprotein - globulins) shows a deep damaged kidney glomerular filter ;

• highly selective or selective proteinuria (separation of low molecular weight proteins - albumin , pre-and postalbumine) .

- As the structural changes of the nephron :

• physiological (no structural changes nephron);

• pathological (found structural changes) .

Physiological proteinuria is minimal (up to 1 g/l), transient , associated with the action of the causal factors and Pathogenesis refers to renal proteinuria , as it is believed that it is fundamental to its origin is a violation of renal blood flow and increased permeability of the glomerular filter On proteins blood plasma .

The following types of physiological proteinuria

• alimentary - after consuming large amounts of protein foods ;
• «march», or working - after considerable physical ¬ tion load in athletes , soldiers;
• emotional - in stressful situations ;
• centrogenic - after a concussion or an epileptic seizure ;
• feverish - for diseases that occur with high temperature ;
• palpation - as a result of deep palpation of the kidneys;
• orthostatic - occurs in young persons with asthenic constitution due to increased lumbar lordosis in the standing position. Feature of this type of proteinuria is the lack of protein in the morning urine (appears only in the daytime as it is connected with the stay of the patient in an upright position) and the presence of moderate or severe proteinuria (1 g/l to 6.3 g/l). Some authors exclude orthostatic proteinuria physiological nature as using biopsies revealed morphological changes (focal changes in the glomerular capillary basement membranes) in the kidney.

Pathological proteinuria (detected structural changes) observed in multiple myeloma , various diseases of the kidneys and urinary tract, is permanent, but may differ in the amount of protein excreted in the urine (minimal, moderate and emphasized) and the level of engagement (pre-renal, renal and postrenal).

- The level of destruction :

• prerenal - caused diseases and pathological conditions (multiple myeloma , Waldenstrom's macroglobulinemia , expressed hemolysis , etc.) that lead to a change in plasma concentration ¬ tration of protein quantity and quality of protein fractions of abnormal proteins;

• Renal : glomerular (glomerulonephritis , renal damage in diffuse connective tissue diseases , renal amyloidosis , hypertension , congestive kidney, diabetic glomerulosclerosis) , tubular (interstitial nephritis , pyelonephritis, Fanconi syndrome) and physiological (see above) ;

• Postrenal - caused urinary excretion of mucus and protein exudate in inflammation of the urinary tract (urolithiasis, cystitis, prostatitis , urethritis) .

Pre-and Post-renal proteinuria in the number of excreted protein are usually minimal.

Pyuria - urinary excretion of leukocytes above the norm: in urinalysis - 5-6 in sight, in the analysis of urine Nechyporenko - more than 2.5 x 10^6 / L . Pyuria may indicate a pathology of kidneys, urinary tract, prostate.

Erithrocyturia or hematuria - urinary excretion of red blood cells above normal: a general analysis of urine red blood cells are absent in the analysis of urine to Nechyporenko - more than 1.0 x 10^6/L. Erithrocyturia is a hallmark of acute and chronic glomerulonephritis , interstitial nephritis caused by taking drugs (aspirin, Analginum , sulfonamides , streptomycin , kanamycin , gentamicin and others). Can occur in acute and chronic calculous pyelonephritis , hemorrhagic fever , tuberculosis, tumors of kidney, bladder and prostate cancer , hemorrhagic cystitis, urolithiasis, renal injury , with secondary renal disease (systemic vasculitis , systemic lupus erythematosus) and in case of overdose anticoagulants.

Hematuria is differentiated as follows.

- By number, excreted in the urine red blood cells :

• microscopic hematuria (red blood cell count - from the unit up to 100 ¬ tion in the field of view does not change color of urine);

• hematuria (erythrocytes to quantify , one gu ¬ pokrshayut entire field of view , is the color of urine (“meat slops”).

The presence of hematuria regardless of the degree of its severity in any patient requires the exclusion of first tumor and tuberculosis of the urinary system, urolithiasis, and men over 50 years old - benign prostatic hyperplasia, and prostate cancer.

Cylindruria - urinary excretion of cylinders representing casts formed in the tubular lumen of protein or cellular elements. This symptom , indicating only the defeat of the nephron , practically does not occur in isolation . Cylinders are well identified and long remain only in acidic urine. Alkaline urine when they are not formed or are rapidly destroyed. Distinguish hyaline , granular , waxy , erythrocyte and leukocyte cylinders.

Hyaline casts - is coagulated whey protein , filtered in the glomerulus and reabsorbed in the proximal tubules. Isolated hyaline casts are sometimes found in the urine of healthy people, especially after a big exercise.

Granular casts - degenerated (dystrophic changes) epithelial cells of the proximal tubules. These cylinders have a grainy surface due to buildup of debris (in the form of grains) disintegrated epithelial cells on the coagulated protein and darker than hyaline.

Waxy cylinders consist of dystrophic and atrophic changes in the epithelium of the distal portions of the tubules . These cylinders have a yellowish color, they are shorter and wider than hyaline casts and appearance resemble wax. Waxy appearance cylinder is a poor prognostic sign, because it indicates severe acute kidney failure .

Erythrocyte and leukocyte cylinders are identified respectively with severe hematuria and pyuria of different origin.

Dynamic monitoring of patients that have been identified urinary syndrome is mandatory.

Pyelonephritis (ICD-10 - N10-N12)

Pyelonephritis - not specific infectious-inflammatory renal disease with predominant localization of the pathological process in the pro ¬ interstitial tissue and must defeat pyelocaliceal system.

The incidence of pyelonephritis has 3 age peak , sex-linked : the first peak is in early childhood (up to 3 years) , the girls are sick more often than boys (8:1); second peak - to the most active reproductive age (18-35 years) , retained predominance of women (7:1) ; third peak - to elderly age and ¬ is characterized by progressive increase in the incidence of men.

Etiology and pathogenesis. The most common pathogens are pyelonephritis Escherichia coli (about 80 %), Proteus mirabilis, P. aerugiposa , Klebsiella spp, Epterobacter spp., Staphylococcus aureus, Epterococcus . The main pathways of infection in the kidney - ascending or urinogenous (through the urethra , bladder and ureter through the lumen or wall of the latter) and hematogenous (at a dose of acute and chronic infection , appendicitis , osteomyelitis , postpartum and chronic infection , etc.). During acute and chronic intestinal infections and lymphogenous possible route of infection .

Risk factors for pyelonephritis (most important):

• female - female anatomical features may cheispuskatelnogo - channel (wide and short) not obstruct the path of the rising infection;

• reflux at different levels (vesicoureteral , ureterovaginal junction);

• anomalies of the kidneys and the upper urinary tract , polycystic , nephroptosis , hypermobile kidney doubling of the kidneys , ureters , and others;

• bladder dysfunction (" neurogenic bladder ");

• factors of urinary tract occlusion - kidney stones , tumors , prostatic hyperplasia ;

• defloration cystitis, pregnancy;

• immunodeficiency disorders (diabetes, neutropenia, AIDS);

• metabolic disorders (calcium oxalate , uric acid , phosphate crystalluria);

• radiation, chemical and toxic effects (including drugs : sulfonamides, tsistostatiki , etc.);

• physical factors (cooling, trauma);

• operations, instrumental intervention urinary tract, their external drainage (permanent or temporary).

Classification . Pyelonephritis are classified according to several criteria.

- Pathogenesis : primary, secondary ;
- According to the course of the disease: acute, chronic ;
- Localization : single-sided , double-sided .
- By place of occurrence:

• extramural (outpatient) - except for outpatients or 48 hours after discharge from the hospital ;

• in-hospital (nosocomial) - developed in the hospital or within 48 h of admission .

- In form : obstructive , obstructive .
- According to the presence of complications : uncomplicated , complicated (renal abscess, renal carbuncle, paranephritis , sepsis, necrotic papillitis).
- Special forms :

• pyelonephritis newborn and childhood ;

• pyelonephritis elderly;

• gestational pyelonephritis pregnant (generic, postpartum);

• calculous pyelonephritis;

• pyelonephritis in patients with diabetes mellitus;

• pyelonephritis in patients with spinal cord injury;

• other forms.

Chronic pyelonephritis (ICD-10 - N11)

Chronic pyelonephritis (chronic tubulointerstitial nephritis) - indolent, periodically increasing bacterial kidney interstitial tissue inflammation, leading to irreversible changes in the renal collecting system , followed by sclerosis of the parenchyma and renal scarring .

Prevalence of chronic pyelonephritis - 18 per 1,000. Among the cases women predominate over men (6:1).

Etiology and pathogenesis. Chronic pyelonephritis is usually the outcome of unhealed or undiagnosed acute pyelonephritis arising after 6 months from the onset of the disease, as well as two or more of its recurrence within 3-5 months from the onset of acute pyelonephritis. Does not exclude primary chronic pyelonephritis option.

Chronic pyelonephritis develops, as a rule, on the background urodynamics which provides ingress of urine into the kidney tissue containing immune complexes (but not microbe itself). Immune complexes are located at the same foci, which explains the almost complete absence of common immunological reactions in laboratory studies in patients.

Clinical presentation and diagnosis. Severity of clinical and laboratory signs of chronic pyelonephritis depends on the phase of inflammation. The clinical picture identifies a number of syndromes.

Intoxication syndrome - often low-grade temperature, preferably in the evening, but can reach 38-39 °. In 80 % of patients this syndrome is absent.

Pain syndrome is characteristic of active inflammation phase. The pain is localized in the lumbar region and the lateral flanks of the abdomen, is not related to the position of the body, radiating down the abdomen, groin and anterior thigh. In primary pyelonephritis pain in the lumbar region is bilateral, and in the secondary - sided.

Compartment syndrome arrhythmias urine appears pollakiuria (increased frequency of urination) and nocturia (the predominance of nocturnal diuresis) . Nocturia reflects a decrease in the concentration of kidney function and is a sign of differential diagnostic differentiation of pyelonephritis and glomerulonephritis in the absence of chronic renal failure or heart failure.

Hypertension syndrome occurs in 50-75 % of patients , 10% of patients formed its malignant form .

Anemia syndrome due to inhibition of renal erythropoietic factor production . Normochromic anemia, microcytic with reticulocytosis.

Exacerbation of chronic pyelonephritis is accompanied by deterioration of general condition , loss of appetite , headache, nausea, vomiting and other symptoms similar to those in acute pyelonephritis , but less intense . In 50-60 % of cases of chronic pyelonephritis has no clinical symptoms, or may be recurrent episodes of acute pyelonephritis or manifestations of chronic nonspecific signs of infection, including fever, anemia, azotemia .

The diagnosis of chronic pyelonephritis is installed on the basis of history (transferred urethritis, acute pyelonephritis, renal colic, kidney malformations and urinary tract), the clinical picture and the results of laboratory and instrumental investigations. It is characterized by:

• similarity of clinical manifestations in acute with acute pyelonephritis, without exacerbation usually little or asymptomatic;

• dysuria as pollakiuria , strangury , nocturia , poly- , oliguria ;

• a positive sign of a percussion (Pasternacky symptom) on the affected side during the exacerbation of the disease;

• typical urinary syndrome: alkaline reaction of urine, reducing the relative density (less than 1015), expressed leycocyturia , true bacteriuria , mild proteinuria (less than 1 g/l), microscopic hematuria, cylindruria possible. Abjection of pathogens of the disease in the bacteriological examination of urine;

• sample to Nechyporenko - domination over leycocyturia erithrocyturia (especially indicated for the latent form of the disease), as well as test and holding prednisolone 30 mg prednisolone in 10 ml 0.9 % sodium chloride solution administered intravenously for 5 min with counting the number of leukocytes in a one-hour urine sample (more than 400,000 leukocytes - test is positive);

• sample on Zimnitski - reduction in the relative density of urine , the prevalence of nocturnal urine ;

• total blood count: anemia, leukocytosis , leukocyte shift to the left, elevated erythrocyte sedimentation rate ;

• blood chemistry : increase of seromucoid , sialic acid , fibrinogen , α2- , and γ- globulin , the presence of C-reactive protein , increased levels of creatinine and urea in chronic renal failure ;

• renal ultrasonography : roughness and contour deformation kidneys , reducing its size and thickness of the parenchyma , increased echogenicity , coarsening contour cups , chashechnolohanochnoy extension system , as well as the presence of cysts , stones , kidney malformations .

Where necessary, and conducted other research methods (X-ray, radioisotope, CT, MRI, angiography of the kidneys).

Percutaneous kidney biopsy is indicated for certain difficulties in diagnosis , but due to the localized nature of pathological changes in the kidney tissue , this method , depending on the accuracy hit puncture needle is not always possible to confirm or exclude the diagnosis of pyelonephritis. Diagnostic value have only positive biopsy results , ie confirming the diagnosis of pyelonephritis .

Chronic pyelonephritis should be differentiated primarily with chronic glomerulonephritis , primary arterial hypertension , diabetic glomerulosclerosis and renal amyloidosis.

Treatment. In most cases, treatment of usual outpatient . The main goal of treatment - to achieve clinical and laboratory remission, prevention and correction of complications.

Indications for hospitalization: marked exacerbation, complications, lack of effect of outpatient treatment, the progression of chronic renal failure secondary pyelonephritis on the background of urolithiasis.

Bed mode for the period of fever.

Diet food diet requires the exclusion of the rich broth, hot and spicy dishes, alcoholic beverages. Diet should contain a physiologically necessary amount of proteins, fats and carbohydrates, and make daily kalorazh 2000-2500 kcal. Recommended milk and dairy products, eggs , meat , boiled fish , a dish of vegetables (potatoes, carrots, cabbage, beets) and fruit (apples, plums, apricots , raisins , figs) , rich in potassium and vitamin C, P, group V.

When hypertension requires strict limitation of salt (up to 4-6 g / day) .

In all forms and at any stage of pyelonephritis are encouraged to include in the diet watermelons, melons , pumpkins , a diuretic and helps cleanse the urinary tract of bacteria , mucus , small stones . The patient may need to increase your fluid intake to 2-2.5 liters / day in the form of juices, fruit drinks (cranberry , lingonberry) , compotes , jellies , in the absence of contraindications (urinary tract obstruction , nephrotic syndrome, uncontrolled hypertension , chronic heart failure since II A stage preeclampsia 2nd half of pregnancy , chronic renal failure terminal) .

Antibiotic therapy is the basis of the treatment of chronic pyelonephritis. Antimicrobial 1st line I include fluoroquinolones generation antimicrobial 2nd row - II generation fluoroquinolones (respiratory), aminopenicillins, cephalosporins II-III generation, macrolides .

Course of antibiotic therapy is 14 days. In the case of successful selection of antibiotic reduction in temperature and sterility of urine occur within 1-3 days of starting treatment , leykotsituriya disappears after 5-10 days. Elevated erythrocyte sedimentation rate can be maintained up to 2-3 weeks . Long-term treatment of chronic pyelonephritis (from a few months to 1.5 years) lost their positions for extended benefits have been identified regimens compared with the two-week course . Next on the 2 -4th week - need must assign fitotepapiyu - uroseptic decoctions of herbs (bearberry , horsetail , birch leaves , cranberries , cranberry, juniper berries , rose hips , etc.).

The criteria for effectiveness of the treatment is to normalize the temperature , the disappearance dizuricheskih events return to normal peripheral blood (white blood cell count , erythrocyte sedimentation rate) , a persistent lack of , or at least a significant reduction in proteinuria , leukocyturia and bacteriuria .

For the treatment of chronic pyelonephritis is not currently recommended for use :

• ampicillin and Biseptolum due to the high resistance of E. coli to them ;

• I generation of cephalosporins due to low activity against gram-negative flora ;

• nitrofurans , nalidixic acid, due to toxicity , low therapeutic blood concentration and a brief action;

• gentamicin due to potential toxicity.

The drug of choice for the treatment of chronic pyelonephritis in pregnant women is amoksutsulluna clavulanate (Augmentin, amoxyklav) .

To improve microcirculation appointed disaggregants (pentoxifylline by 0.1-0.2 g 2-3 times a day , chimes , etc.).

If there are indications provided symptomatic pain therapy (NSAIDs, antispasmodics) , secondary hypertension (antihypertensives) , anemia (iron supplements , vitamin B12, folic acid) .

Physiotherapy (inductothermy, UHF and MWSS - therapy, paraffin- ozocerite baths) is assigned individually tailored contraindications latent phase of inflammation (partial remission) . Showing physiotherapy under the supervision of an instructor , walking .

For surgical treatment resorted to eliminate the causes of stasis of urine in patients with secondary pyelonephritis .

Sanatorium treatment of chronic pyelonephritis held in resorts - Zheleznovodsk (Caucasian Mineral Waters), Truskavets (Ukraine), Krainka (Tula region), the Sary-Agach (Kazakhstan), Istisu (Azerbaijan) klimatobalneological resorts - Yangantau (Ufa , Bashkortostan), Jermuk (Armenia), Sairme (Georgia), as well as in the sanatorium "Rassvet" (Belarus). Systematic

drinking mineral water promotes elimination of the inflammatory process in the kidneys and urinary tract.

Contraindications to the sanatorium treatment : high arterial hypertension (180/100 mm Hg), Anemia, moderate and severe , signs of chronic renal failure, urolithiasis (requiring surgical correction) , prostatic hyperplasia grade II and III .

Medical and social assessment. Temporary disability of patients is an average of 15-20 days at easy to exacerbations occurring 50-65 days - during severe. Frequent relapses, the appearance and progression of chronic renal failure is grounds for referral to the MREB to establish the degree of disability .

Dispanserization . Observation frequency nephrologist or divisional therapist - 2 times a year .

Examinations by medical specialists : otolaryngologist, ophthalmologist - 1 times per year; urologist, gynecologist - indicated.

Laboratory and instrumental investigations : total blood count, OAM , urine culture on the microflora, urinalysis for Zimnitskiy, urinalysis to Nechyporenko, BAC (electrolytes, urea, creatinine, total protein, glucose), creatinine clearance and reabsorption of water, renal ultrasonography, radioisotope radiography (RWG) - 2 times a year ; cytology urine sediment , urine for the presence of acid-fast bacilli , ECG, chest X-ray - 1 time per year.

Physiotherapy - 2 times a year . Remediation of foci of chronic infection ¬ tion . Spa treatment (in the absence of contraindications) . During exacerbation process inefficiencies preventive treatment - treatment in a hospital .

Time of observation - for life .

Prevention . Primary prevention of chronic pyelonephritis is prompt and adequate treatment of acute pyelonephritis , acute diseases of the bladder and urinary tract , as well as in addressing the causes that impede the normal flow of urine .

Secondary prevention provides for dynamic dispensary observation of patients with an established diagnosis of chronic pyelonephritis . Patients can perform work that is not related with great physical and nervous tension , with the possibility of hypothermia , prolonged stay on his feet, on the night shift , in sorrow sneeze shops. Patients should be under medical supervision urologist or nephrologist (therapist) .

In the prevention of recurrence of pyelonephritis and its further progression to ESRD are important early detection and thorough treatment of hidden or obvious foci of infection and disease interkurrent , suitable herbal medicine .

Chronic glomerulonephritis (ICD-10 -N03)

Chronic glomerulonephritis (chronic nephritic syndrome) - a chronic immunoinflammatory disease of kidneys with long- persistent or recurrent urinary syndrome (proteinuria and / or hematuria) , progressive deterioration of renal function and outcome in chronic renal failure .

Incidence of 13-50 cases per 10,000 population per year . Occurs 2-4 times more acute glomerulonephritis with equal frequency in both men and women .

Chronic glomerulonephritis (CGN) is a consequence of uncured or timely undiagnosed AGN. About 10-15 % of cases of CGN defined as pervichnohronichesky as a history of patients there is no indication transferred acute glomerulonephritis .

Etiology and pathogenesis . In the development and maintenance of immune inflammation in CGN involving the same mechanisms as in acute glomerulonephritis . However, in his progression participate and non-immune factors leading place among them belongs to a breach in the kidney hemodynamics (intraglomerular hypertension and hyperfiltration of macromolecules of different blood plasma into the tissues of the nephron) and nephrotoxicity of proteinuria (primarily albumin and transferrin) , which play a major role in the development of glomerulonephritis and tubulointerstitial sclerosis ¬ fibrosis , constituting the basis of renal histopathology .

Classification . Currently there are two classifications of CGN : clinical and morphological .

Clinical classification divides CGN for different criteria.

- Pathogenesis : primary (idiopathic) and secondary CGN associated with another disease .
- According to clinical forms :

• latent (with isolated urinary syndrome);

• hematuric ;

• hypertension ;

• nephrotic ;

• Mixed (nephrotic form in conjunction with hyper ¬ tonic) .

- According phases : exacerbation (active phase , relapse) and Remis ¬ And this (inactive phase) .
- on the course of the disease:

• slow-progressive (CKD after 10 years from the onset of the disease);

• fastly progressing (CKD after 2-5 years from the onset of the disease);

• rapidly progressive (CKD after 6-8 months of onset);

• intermittent flow (characterized by the complete disappearance of the urinary syndrome within a certain period of time, observed in a latent form and hematuric).

Chronickidney disease (CKD) latent , compensated , intermittent , terminal .

According to the morphological classification , there are the following forms :

• mesangioproliferative glomerulonephritis ;

• membranoproliferative (mezangiocapillar) glomerulonephritis ;

• membranous glomerulonephritis ;

• glomerulonephritis with minimal changes ;

• focal segmental glomerulosclerosis ;

• Fibrillar-immunotactoidglomerulonephritis ;

• fibroplastic glomerulonephritis .

During a transition from CGN one clinical or morphological form to another. However, to establish the morphological form CGN need a needle biopsy of the kidneys , unfortunately not always possible . Therefore, in our conditions is increasingly used clinical classification of CGN.

Clinical presentation and diagnosis . The clinical picture of CGN varies considerably depending on the clinical and morphological variant . However, it depends on the shape of renal function and phase of the disease . In the onset of the disease and during the exacerbation of CGN usually resembles acute glomerulonephritis , has an undulating but steadily progressive nature of the flow , and in clinical remission corresponds to the form of CGN.

Latent form (isolated urinary syndrome) is characterized by slow-progressive course , the presence of moderate bladder syndrome : minimal proteinuria (up to 1 g / l) , low red blood cell (5-10 in sight) and cylindruria in the absence of extra- renal disease signs (edema and arterial hypertension) . This form is up to 50 % of all cases of CGN.

Hematuric form is manifested persistent hematuria (from micro - to gross hematuria) snevyrazhennoy proteinuria (1.5 g / l) in the absence of extrarenal symptoms. There are about 5%.

Hypertensive heart shape is determined by hypertension , minor urinary syndrome : minimal proteinuria (up to 1 g / l) , red blood cell (erythrocyte 5-10 in the field of view) , single hyaline casts and lack of edema. Found changes in the retinal vessels (narrowing of the retinal vessels , symptoms chiasm , " copper " and " silver wire ", sometimes blood ¬ voizliyaniya , in severe cases - retinal detachment neyroretinopatiya) , left ventricular hypertrophy . Krizovoe not typical . Hypertension with hypertonic ¬ tion form CGN appears early in the disease that differs from her ¬ is symptomatic hypertension developing in all forms of CGN with signs and CRF. Observed in 20% of cases of all forms of CGN.

Nephrotic form (nephrotic syndrome) involves massive proteinuria (more than 3.5 g / d) , a violation of protein, lipid , water and electrolyte exchanges with hypoproteinemia , dysproteinemia , hyperlipidemia lipiduriey and significant swelling . Meets up to 20 % of cases.

Mixed form diagnosed with nephrotic syndrome and hypertensive . This form is the most prognostically unfavorable and up to 7% .

CGN expressed aggravation may be accompanied by the development of acute nephritic syndrome , which is characterized by severe general condition , the rapid emergence or increase of edema , oliguria, a significant increase in proteinuria , hematuria , cylindruria , a sharp

increase in blood pressure . Clinical manifestations and course of acute nephritic syndrome resembles the classic version of AGN .

The clinical picture of morphological forms of CGN has some features of the course and prognosis.

Mesangioproliferative CGN (IgA-nephropathy, Berger's disease) is characterized by deposition of immune complexes containing IgA, in the mesangium and glomerular capillaries beneath the endothelium and mesangial reaction to these deposits. Marked expansion of mesangial vascular bundle glomerular mesangial cells , mesangial matrix accumulation . This is a fairly frequent (50-60%) morphological type of glomerulonephritis , which meets all the criteria of immunological diseases .

Develops mainly in young men. The clinical picture is dominated by proteinuria (from persistent moderate to nephrotic) , hematuria , hypertension . Typical episodes of gross hematuria with pain in the lumbar region associated with nasopharyngeal or gastrointestinal infections. Unlike acute glo ¬ merulonefrita postinfection time of occurrence of renal symptoms coincides with the impact of precipitating factors .

A relatively favorable : a ten-year survival rate is about 80%.

Membranoproliferative (mesangiocapillary) glomerulonephritis characterized by marked proliferation mezangiotsitov volume expansion and mesangial ma ¬ Trix with diffuse increase in vascular loops , creating a picture of lobular glomerulus (" lobular " jade) as well as thickening of the capillary walls or dvukonturnostyu caused penetration (interpozitsin) and location of mesangial cells . In this regard, there are three (sometimes four) type Membranoproliferative jade : I type - immune deposits are located beneath the endothelium and in the mesangial area of glomeruli (subendothelial or classical type), II type - deposits are located within the glomerular basement membrane (" dense deposits disease ") , III type - subepithelial deposits are located and form characteristic "peaks" , noted patchy damage ¬ denie membrane. Clinically, all types are identical but differ ¬ prepared on laboratory data and results of transplant kidney ¬ tion . Occurs with equal frequency in men and women. Often begins as acute glomerulonephritis (acute nephritic syndrome) , accounting for 15% of cases in children and 30% of cases in adults. Approximately 50% of patients develop nephrotic syndrome . Chance of isolated urinary syndrome with hematuria . Characterized by severe hypertension, gipokomplementemiya and anemia , cryoglobulinemia can sometimes massive proteinuria . For progressing steadily . The prognosis is poor . CRF develops within 3-5 years; 10 -year survival rate - 50%.

Membranous glomerulonephritis characterized by diffuse thickening of the glomerular basement membrane with the formation ¬ vaniem subepithelial protrusions surrounding the deposition of immune complexes. Signs of inflammatory cell reaction in the glomeruli are absent.

The disease usually occurs between the ages of 30-50 years , twice as often in men than in women. In 80% of cases of nephrotic syndrome manifested . Microscopic hematuria occurs in 54 % of cases , hypertension - 20-40 %. When membranous glomerulonephritis more frequently than in other embodiments , developing venous thrombosis , including thrombosis of the renal

veins . A relatively favorable 10 - year survival rate is 60-75 % , in the treatment of complete remission is achieved in 33% of patients.

Glomerulonephritis with minimal changes can be diagnosed only by electron microscopy - is found a merger (smoothing) of small legs of podocytes throughout the glomerular capillaries , resulting in the loss of the negative charge of the membrane and is usually a "big" proteinuria . Immune deposits not detected. Light microscopy and immunofluorescence biopsies no pathological changes were detected.

The debut of the disease are concentrated in the age of 6-8 years , develops acutely , usually after undergoing upper respiratory tract infection . The clinical picture is dominated by nephrotic syndrome . Massive proteinuria , mainly due to albumin , but in small amounts detected IgG and α2- macroglobulin . In 20-30% of cases there is gross hematuria . Hypertension and renal failure are rare , the process tends to spontaneously resolve even without treatment . Favorable prognosis : 5 -year survival rate of more than 95 %.

Focal segmental glomerulosclerosis - the only state in which glomerulosclerosis begins only in juxtamedullary nephrons. In a process involving ¬ are separate segments remaining intact glomeruli. Focal segmental glomerulosclerosis - the cause of 10-15 cases of nephrotic syndrome in children and 15-25 % of cases in adults. In the urinary sediment revealed erythrocytes, leukocytes . Nonselective proteinuria , most patients combined with hematuria and arterial hypertension ¬ sion , which is considered a characteristic clinical picture . Natural development of chronic renal failure, 20% of patients , even at the onset of the disease. For progressive disease . Typically the lack of response to treatment with corticosteroids . Complete remissions are extremely rare , and 5 - year survival rate is 50-70%. Heavy progressiruyushim over collapsing different form (the collapse of capillary loops of the glomeruli in the affected segments) in HIV-infected patients and heroin users .

Fibrillar immunotaktoid glomerulonephritis characterized by varying changes of mesangial expansion and thickening of the glomerular basement membrane to proliferative glomerulonephritis and extracapillary “crescents”. Typical changes detected by electron microscopy, extracellular amyloid fibrillar inclusion in mesangial or capillary wall. Distinguishes them from amyloid larger diameter; moreover, they are not stained with Congo red.

There is less than 1 % of all cases of glomerulonephritis in adults. Clinically severe proteinuria in 50% of cases - nephrotic syndrome. Most patients have hematuria, hypertension and kidney dysfunction. The course is progressive.

Fibroplastic glomerulonefrum characterized by significant expression of fibrotic processes : glomerular capillary loops sclerosis , multiple sclerosis and thickening of the capsule , the capsule seam with vascular loops. Overall fibroplastic changes are the outcome of any form of glomerulonephritis.

Plan Survey in outpatient :

 clinical examination (complaints, anamnesis, objective data), proteinuria;

 urinalysis and urine analysis for Necheporenko (in dynamics);

 complete blood count;

 blood chemistry: urea, creatinine, uric acid, potassium, C-reactive protein, sugar, bilirubin, transamilase ;

 ECG ;

 R- scopy of the chest ;

 BP profile ;

 fundus examination ;

 glomerular filtration (sample Rehberg);

 renal ultrasonography

CGN treated as outpatients.

Indications for hospitalization :

• exacerbation of CGN (erythrocytes and increase the appearance of gross hematuria , nephrotic syndrome or appearance nekorregiruemoy hypertension, progressive deterioration of renal function azotvydelitelnoy);

• the development of acute nephrotic syndrome ;

• for stationary survey to clarify the diagnosis (CT, MRI , punctional kidney biopsy);

• to select a treatment strategy based on the individual patient and clinical data punktsionalnoy biopsy.

Latent within CGN subclinical without apparent exacerbations and remissions , diagnosed by changes in urinalysis , identified by chance when routine inspection .

Relapsing course characterized by various forms of CGN exacerbations that develop after 3-5 days (usually 2-3 days) after exposure to cold, stress or without visible reasons.

Acute nephritic syndrome (a combination of hematuria , proteinuria , hypertension and renal function often decline) was observed at the onset and exacerbation of idiopathic forms , quickly progressing , mesangioproliferative , Membranoproliferative (mesangiocapillary) glomerulonephritis , exacerbation of lupus nephritis .

Arterial hypertension in combination with proteinuria and urinary sediment minimal changes characteristic of diabetic nephropathy, renal involvement in essential hypertension. In the latter case, hypertension, usually preceded by the appearance of changes in urine.

Nephrotic syndrome most frequently (almost constantly) occurs in nephrotic and mixed forms, with minimal change glomerulonephritis, membranous, focal segmental glomerulosclerosis, less membraneproliferative glomerulonephritis, diabetic glomerulosclerosis, renal amyloidosis.

Possible combination of the nephritic and nephrotic syndromes suggests subacute and quickly progressing glomerulonephritis.

However, diagnosis of any morphological form CGN possible only after nefrobiopsy and pathologic study of renal biopsy .

Treatment of chronic glomerulonephritis (CGN)

In most cases, CGN etiological treatment not essential or impossible. In rare cases, alcohol CGN discontinuation of intake of alcohol leads to a large improvement and relief of disease. Recommended sanitation foci of chronic infection, bed rest during SARS, angina patient .

Pathogenetic treatment of CGN.

Given the seriousness of modern pathological therapy jade , the possibility of its various complications before treatment is recommended :

1. Rate severity of renal disease and the degree of activity of the process

2 . Substantiate the diagnosis of acute nephritis

3 . Complete kidney puncture biopsy to clarify morphological variant of nephritis, as currently medikament CGN therapy (a combination of drugs, treatment regimen and its duration) depend on the clinical and morphological form of the latter.

All these issues are addressed in terms of nephrology department of the hospital or specialized center where the patient directs the family doctor or a general practitioner.

Pathogenetic drug therapy for CGN includes :

• methods , preemptive action is determined by immune depression (glucocorticosteroids cytostatics , plasmapheresis) ;

• anticoagulants and antiplatelet drugs (heparin , dipiridamol);

• combined schemes

Glucocorticosteroids: prednisolone, methylprednisolone (metipred, solumedrol)

Indications:

• nephrotic form CGN;

• latent form (Form with isolated urinary syndrome) in the acute stage with severe proteinuria and a tendency to develop nephrotic syndrome.

Glucocorticosteroids most effective when the following variants of CGN: "minimal" change, membranous glomerulonephritis, and mezangioproliferative mezangiomembranous glomerulonephritis. Prednisolone is administered in a dose of 1 mg / kg per day for a month: then gradually reduce the dose. The treatment course of 1-2 years. High activity CGN sharply expressed phenomena of nephrotic syndrome, rapidly progressive course (rapid increase in renal

failure) is shown with methylprednisolone pulse therapy - daily intravenous infusion of 1000 mg methylprednisolone for 3 days followed by prednisone .

Glucocorticosteroids are ineffective at mesangiocapillary , proliferative glomerulonephritis and Fibroplastic focal-segmental glomerulosclerosis .

Glucocorticosteroidsare contraindicated in hypertensive and mixed forms of chronic glomerulonephritis , their effectiveness is questionable in its latent form CGN flowing with asymptomatic proteinuria or isolated hematuria .

Cytostatics : cyclophosphamide, azathioprine, 6- mercaptopurine.

Indications for cytotoxic drugs: high activity of CGN , hypertensive syndrome, initial signs of renal failure (in terminal chronic kudney disease cytostatics arecontraindicated), and with membranous glomerulonephritis mesangiocapillary morphological variants , the ineffectiveness of hormone therapy or some of its complications (pronounced " kushingoid " ulcerative gastric lesions , aseptic bone necrosis) .

Azathioprine and merkaptopurin administered 2-3 mg / kg body weight per day , cyclophosphamide - 1.5-2 mg / kg cyclosporine (Sandimun) - 7.5 mg / day , 4-10 weeks. Then gradually reduce the dose. Supportive therapy (1/ 2 or 1/ 3 of daily dose) on an outpatient basis for 6-12 months.

Anticoagulants and antiplatelet agents: heparin and dipiridamol (Curantyle). Often used in conjunction with glucocorticosteroids and cytostatics. Curantyle a daily dose of 200-400 mg can be assigned to virtually all forms of CGN, except hematuric.

Combination Therapy

In severe forms of CGN (membranous, proliferative membranous) resistant to treatment resistant nephrotic syndrome, glomerulonephritis rapidly progressing recommended a four-combination therapy, consisting of cytostatic glucocorticosteroids, anticoagulant and antiocoagulant. Method of combined therapyleads to a significant improvement in mesangioproliferative glomerulonephritis , as well as Fibroplastic changesin the glomeruli. This method is effective inhypertonic and impractical in latent nephritis.

Plasmapheresis (3-5 sessions) used in the process high activity , proliferative extracapillary glomerulonephritis , ineffectiveness or contraindication to demonstrate how glyukokosteroidov , tsitostatinov .

In the treatment with cytostatics, severe complications:

• gematocytopenic effects (anemia , leukopenia, thrombocytopenia);

• hepatotoxicity ;

• development of infectious complications;

• chromosome aberration, teratogenic , carcinogenic effect .

NSAIDs for the treatment of CGN currently not practically used because of their ability to reduce the synthesis of prostaglandins and improve hypertension , reduced glomerular filtration rate .

From the foregoing it follows that the pathogenetic drug therapy CGN very unsafe for the patient and the problem of the treatment of nephritis one of the sharpest in nephrology . Some authors (B.I. Shulotko) expressed the view that untreated patients with nephritis live as much and treated , and the last to die from treatment. With this we can not agree, but there is a grain of truth . This is especially true morphological variants of nephritis in which CGN developed through decades. Therefore, the patient should be sent to the nephrology department, or even a specialized center for further diagnosis, including morphological variant of nephritis, determining subsequent treatment of the patient.

Outpatient treatment.

After discharge from the hospital in the case of a patient appointment pathogenetic therapy with glucocorticoids and (or) cytostatics local doctor is obliged to implement the recommendations of the nephrologist and treat the patient , in consultation with him (to adjust the dose of medication) .

Particular attention of GP should be directed at identifying the pathogenetic therapy side effects and their treatment. For prevention of steroid ulcers omeprozol administered 20 ml/day, rabeprazole - 10 mg/day, pantoprozole, steroidal osteoporosis - complex preparations of calcium and vitamin D in the 1500 mg ionized calcium and vitamin D is 400-800 mg/day, and/or bisphosphonates: alendronate 35-70 mg 1 time a week or ibandronic acid (Bonviva) 150 mg 1 time a month.

Syndromic treatment. Hypertension .

Correction BP slows ESRD onset and reduces the risk of cardiovascular disease. The aim of antihypertensive therapy is to maintain a systolic blood pressure 120-139 mm Hg level, diastolic blood pressure <90 mm Hg When long-term decline in glomerular filtration rate target blood pressure can be up to 150 mm Hg (systolic) . Hypotension should be avoided , because systolic blood pressure < 110 mm Hg may accelerate the progression of chronic renal failure .

Drugs of choice (usually in combination) in this sequence are: ACE inhibitors , sartans, diuretics , calcium antagonists, selective ß- blockers. Thus the preference of calcium blockers given Normodipine, lercanidipine, bisoprolol of ß- blockers - nebivolol , carvedilol , bisoprolol and metoprolol succinate . ACE inhibitors for today recognition starting preparations in the treatment of hypertension in chronic kidney disease, as proved that they possess renoprotektornymi properties reduce intraglomerular hypertension and hyperfiltration , efferent arterioles extending glomeruli , reduce albuminuria , slow the rate of decline in GFR .

Drugs with renal route of elimination (enalapril , lisinopril , perindopril) in these patients have a more pronounced antiproteinuric antihypertensive effect. In contrast, drugs with hepatic excretion by (quinapril , moexipril , Sartana) safer with a significant reduction in GFR . Thus , the choice of ACE inhibitor / ARB determined by the activity of kidney and renal function of the process .

Need to titrate ACE inhibitors and ARBs to moderate and high doses . On -stage renal failure , usually monotherapy leads to stabilization of blood pressure , so it is recommended combination therapy with antihypertensive drugs belonging to different groups .

Table 6.1 Dose angiotensin converting enzyme inhibitors and angiotensin receptor blockers with two kidney dysfunction (K/DOQ, 2004 ; ESC, 2004)

Drug	***Renal excretion, %***	***Dose (mg / day / multiple)***	
		GFR > 60 ml/min/1 73 m^2	***GFR > 10-30 ml/min/1 73 m^2***
Angiotensin -converting enzyme inhibitors			
Kaptorpril	95	25-150/2-3	6,25-12,5/2-3
Zofenopril	60	7,5-30/2	7,5-30/2
Lisinopril (Diroton)	70	20-40/1-2	2,5-5/1
Perindopril	75	4-8/1-2	2-4/1
Ramipril	85	2,5-20/1-2	1,25-5/1
Spirapril	50	3-6/1	3-6/1
Fosinopril	50	10-40/1-2	10-40/1
Enalapril	88	10-40/1-2	2,5-20/1
Angiotensin receptor 2 blockers			
Valsartan	30	80-320/1	80-320/1
Irbesartan	20	150-300/1	150-300/1
Candesartan	33	4-32/1	4/1
Losartan	43	50-100/1-2	50-100/1-2
Eprosartan	37	400-800/1-2	400-800/1-2

Combination therapy :

1. ACEI + ARB with normal renal function in patients under the age of 55 years.

2. ACEI + diuretic (indapamide 12.5 - 25 mg or 25 mg or veroshpiron, eplerenone)

3 . ACEi + CCB (normodipin) or BB (nebilet)

When dyslipidemia with increased levels of cholesterol , low-density lipoprotein shown statin (atorvastatin 20-40 mg / day).

Anemia

Partial cause of anemia can be iron deficiency associated with shortening the life of erythrocytes, malabsorption , blood loss . Prescribers iron rate of 200 mg of elemental iron per day (ferro- gradument , tardiferon 1-2 1 pill once a day) .

Severe anemia is associated with reduced renal synthesis of erythropoietin - a hormone providing erythropoiesis. In appointing erythropoietin (Recormon 20 mg / kg 3 times per week) need to weigh the risk (hypertension, hyperkalemia, trombosis) and benefits (improved quality of life, the rejection of blood transfusions). When severe uncontrolled hypertension and coronary heart disease treated with erythropoietin contraindicated.

Erythropoietin treatment may be imposed only when the level of hemoglobin below 100 g / l and above this threshold the drug pekomenduetsya stop.

During treatment with erythropoietin enhanced serum iron deficiency, so for a more effective treatment with erythropoietin therapy is advisable to combine with iron-containing drugs.

Chronic renal failure (CRF) (ICD-10 -N18)

Chronic renal failure (CRF) - pathological simptomakompleks due to a sharp decrease in the number and function of nephrons , which leads to disruption of the endocrine and excretory renal function , homeostasis , rsstoroystvu all types of metabolism , acid-base balance , of all organs and systems. Cause of chronic renal failure are chronic kidney disease (CKD) of various etiologies .

Table 6.2 Classification of chronic kidney disease

Pathological characteristics	***causing disease***	***Among all patients with CKD***
Diabetic glomerulosclerosis	Diabetes mellitus type 1 and 2	33%
Glomerular lesions	autoimmune disease , systemic infections, toxic substances, and the action of drugs, tumors	19%
vascular lesions	Pathology of of large arteries , hypertension, microangiopathy	21%
Tubulointerstitial pathology	Urinary tract infections , ICD , urinary tract obstruction , the effect of toxic substances and drugs , PMR	4%
Cystic lesion	Autosomedominantandautosomerecessive polycystickidney	6%
The defeat of the transplanted	rejection reaction , the action of toxic	

kidney	substances and drugs (cyclosporine, tacrolimus) , transplant glomerulopathy	

The most common of all diseases of the kidneys , leading to chronic renal failure, chronic kidney disease are particularly CGN and chronic pyelonephritis .

No generally accepted classification of CKD . The most common classification of NA Lopatkina and IN Kuczynski (1973), according to which distinguish four stages of clinical treatment of chronic renal failure: a latent, compensated, periodical exposure and terminal.

Latent stage of chronic renal failure is characterized by the absence of clinical manifestations of chronic renal failure. GFR decreased to 45-60 ml / min , may be a slight increase in blood creatinine and urea . These manifestations of chronic renal failure patient and do not bother discovered incidentally. Latent stage of the disease is the most favorable , its timely detection, elimination of the causes of CRF may suspend a process to delay for many years the development of subsequent stages of CKD .

Compensated stage CRF appears easy dyspepsia, dry mouth, skin discoloration, fatigue. Glomerular filtration is reduced to 30-40 ml/min , marked polyuria, thirst.

Periodical exposure stage CRF is clinically characterized by the fact that a satisfactory state of patients often replaced sharp deterioration when amplified thirst, nausea, weakness in unaffected diuresis, glomerular filtration is reduced to 20-30 ml/min.

Worsening of chronic renal failure usually develops as a result of acute kidney disease due to intercurrent illness (flu, sore throat, pneumonia), either due to occlusion of the urinary tract konkrementnom, tumor. This stage is characterized by reversibility of clinical and biochemical manifestations of chronic renal failure, but the period of exacerbation may take several weeks or months.

Terminal stage of CRF is characterized by the progressive development of uremia due to loss clinics and fibrous replacement of the majority (85-90 %) of nephrons.

Treatment

For right choice of adequate treatment is extremely important to consider the classification of CKD .

1. Conservative stage with falling glomerular filtration to 40-15 ml / min.

With more possibilities of conservative treatment .

2 . Terminal stage with a glomerular filtration rate of about 15 ml/min., When should be discussed the issue of extrarenal purification (hemodialysis, peritoneal dialysis) or a kidney transplant .

1.1. Treatment of the underlying disease that led to the development of chronic renal failure in the conservative stage can still have a positive impact and reduce the severity of CRF. This

applies particularly to chronic pyelonephritis with initial or moderately severe CRF phenomena . Relief of acute inflammation in the kidneys reduces the severity of renal failure phenomena .

1.2. Mode .

The patient should avoid hypothermia , great physical and emotional stress . Patient needs in optimal conditions of work and life. It should be surrounded with attention and care .

1.3. Clinical nutrition .

The diet is based on the following principles :

- Limiting dietary intake of protein to 60-40-20 g / day depending on the severity of CKD ;

provide sufficient caloric intake, appropriate energy needs of the body, from fat, carbohydrates, providing full body with vitamins and microelements;

- Limiting employment phosphate intake;

- Control the flow of sodium chloride, water, potassium . -1

1.4. Correction of disorders of water balance

If the level of creatinine in blood plasma is 0.35-1.3 mmol / l, which corresponds to the glomerular filtration rate of 10-40 ml / min , and no signs of heart failure , the patient should receive a sufficient amount of fluid to maintain diuresis within 2-2.5 liters day.

Correction of electrolyte imbalance

Reception salt CRF patients without edema syndrome and hypertension should not be limited . Sharp and prolonged salt restriction leads to dehydration patients , hypo- volemii and deterioration of renal function , rise of weakness, loss of appetite. The recommended amount of salt in the conservative phase of chronic renal failure in the absence of edema and hypertension is 10-15 grams per day , with the development of edema syndrome and severe hypertension salt intake - 3-5 g / day. It is desirable to distribute the amount of sodium excreted in urine per day , to calculate the necessary amount of salt in the diet.

In moderate hyperkalemia (6-6.5 mmol / l) should be limited in the diet foods rich in potassium , potassium-sparing diuretics avoided , if they contain potassium in the blood 7 mmol / l and above shows hemodialysis.

When hyperphosphatemia limit foods rich in phosphorus (fish , cheese, buckwheat) , injected drugs binding phosphorus in the intestine (calcium carbonate) for the treatment and prevention of hypocalcemia hyperparathyroidism prescribed calcium carbonate 0.5-1 g orally 3 times a day with meals and the ineffectiveness - active metabolites of vitamin D (calcitriol) .

Renal colic

Renal or ureteric colic - one of the most severe types of pain that a person can experience . Colic occurs only in case of a sudden patency of the ureter , which leads to a sharp increase vnutrilohanochnogo and then intrarenal pressure . Stretching the walls of the pelvis causes

irritation of nerve endings, which results in a feeling of severe pain in the lumbar region . Cause of acute ureteral patency is often a blockage of his small calculi in urolithiasis . However , renal colic may be in gross hematuria (blockage of a blood clot) , pyuria (Haaretz purulent plug) .

Clinic . Renal colic comes on suddenly , severe pain along the ureter is given in the lower abdomen , groin area , genitals . Often there is vomiting, increased blood pressure , impaired urination, can raise the body temperature . Positive sign of a beating with a sore hand . Pain syndrome in older age groups is less pronounced . Characteristic feature is hematuria .

Approach to the Patient in outpatient settings. Attack first arose . Intravenously 2-4 ml of 2% drotaverine , admission to hospital. In the absence of a history of appendectomy and right-sided , with the symptoms of peritoneal irritation delivery to the surgical department to exclude acute surgical pathology. If you have a history of appendectomy , no peritoneal signs - hospitalization in the urology department for further diagnosis (ambulance) .

Attack re-emerged with a history of urolithiasis .

Baralgin 5 ml i/mor drotaverine 2% 2 ml i/v, in the absence of the effect atropine 0.1% - 0.5 subcutaneously. In the absence of the effect of medical care , the presence of complications (anuria, hematuria , hyperthermia with symptoms of intoxication) , bilateral renal colic , kidney stones , kidney only - urgent hospitalization in urological hospital.

Class 7 Definition of anemia syndrome. Classification of anemia in pathogenesis (posthemorrhagic, dyseritropoietic, hemolytic), color index, size and volume of erythrocytes, saturation with hemoglobin, regenerative capacity of the bone marrow.

Differential diagnosis of anemic syndrome. Outpatient aspects of diagnosis and treatment of anemias caused by iron deficiency, B_{12}-vitamins and folic acid deficiency

Differential diagnosis of conditions accompanied by loss of consciousness (syncope, coma). Diabetic (hyperglycemic and hypoglycemic) coma. Therapeutic aspects of management of patients with type 2 diabetes mellitus in outpatient settings, prevention of its complications.

Session Purpose: To teach students the diagnosis, treatment, dispanserization, examination of disability, first aid in patients with anemia, diabetes mellitus and coma on an outpatient basis.

Study Questions

1 Definition of anemia syndrome. Classification of anemia in pathogenesis (posthemorrhagic, dyseritropoietic, hemolytic), color index, size and volume of erythrocytes, saturation with hemoglobin, regenerative capacity of the bone marrow.

2. Algorithm of diagnostic search in anemic syndrome.
3. Differential diagnosis of hypochromic anemia (with iron deficiency and normal iron saturation).
4. Differential diagnosis of hyperchromic anemia (B_{12}-vitamin and folic deficiency).
5. Differential diagnosis of normochromic anemia (hypo- and aplastic, hemolytic).
6. Iron deficiency anemia: causes, features of clinical manifestations, blood picture, medical tactics, patient examination plan, outpatient treatment, rehabilitation, examination of temporary disability, prevention.
7. B_{12}-vitamin and folic acid deficiency anemia: causes, clinical manifestations and blood picture, differential diagnosis, the patient's plan of inspection, medical tactics, outpatient treatment, rehabilitation, examination of temporary disability, prevention.

8. Conditions accompanied by loss of consciousness: fainting, coma.
9. Pathogenesis and objective signs of coma, degree of severity.
10. Systematization of coma in terms of speed and duration.
11. Causes of sudden short-term loss of consciousness, sudden and prolonged loss of consciousness, prolonged loss of consciousness with a gradual onset, loss of consciousness with unknown origin and unknown duration.
12. The sequence and characteristics of a physical examination of a patient who is unconscious, a questioning of eyewitnesses. Emergency medical care for syncope, coma of unclear etiology, medical tactics.
13. Causes and pathogenesis of coma in diabetes mellitus. Hyperglycemic (ketoacidotic, hyperosmolar, lactacidemic) and hypoglycemic coma: diagnosis, emergency medical care, prevention.
14. Diabetes mellitus type 2: principles of diagnosis and treatment in outpatient settings, examination of temporary disability, indications for referral to MREB, dispanserization, prevention of complications.

Main literature:

1. Diagnosis and treatment of internal diseases in polyclinic / E.N. Kezhun – 1st ed. – Grodno. GrSMU, 2018.
2. Oxford handbook ofgeneral practice [Text] / Simon Chantal [et al.]. - 4th ed. - Oxford : Oxford university press, reprinted 2015.
3. Harrison's Manual ofMedicine [Text] / editors: Dan L. Longo [et al.]. - 18th ed. - New York [etc.] : McGraw-Hill, Medical, 2013.

Anemia is a common condition, particularly in young women and in the geriatric population, and is a significant public health problem in developing countries. Anemia is defined by the World Health Organization as haemoglobin (Hb) level < 120g/L in women and Hb< 130 g/L in men. This definition also includes the so-called pseudo anaemic states (pregnancy, cardiac heart failure and hyperproteinaemia) where Hb concentration falls as the result of an increase of the plasma volume. Incontrast, a decreased red blood cell mass can be masked by haemoconcentration resulting from a decrease in plasma volume.

Pathogenetic classification

I. Anemia due to blood loss (posthemorrhagic).

1. Acute hemorrhagic anemia.
2. Chronic hemorrhagic anemia.

II. Anemia due to violations of the formation of red blood cells and hemoglobin.

3. Iron deficiency anemia.
4. Iron redistribution anemia (iron reutilization violation).
5. Iron-saturated (sideroahrestical) anemia associated with impaired heme synthesis.
6. Megaloblastic anemia associated with impaired DNA synthesis.
6.1. B_{12}-vitamin and folic acid deficiency anemia.
6.2. Megaloblastic anemia caused by a hereditary deficiency of enzymes involved in the synthesis of purine and pyrimidine bases.
6.3. B_{12}-vitamin achrestic anemia.
7. Hypoproliferative anemia.
8. Anemia associated with bone marrow failure.
8.1. Hypoplastic (aplastic) anemia.
8.2. Refractory anemia with myelodysplastic syndrome.
9. Metaplastic anemia.
9.1 Anemia in hematological malignancies.

9.2. Anemia with cancer metastases in the bone marrow.

10. Diserythropoietic anemia.

III. Anemia due to increased blood loss (hemolytic)

11. Hereditary.

11.1. Associated with the violation of the structure of the membrane of erythrocytes (microspherocytic anemia or Minkowsky-Shauffard disease, ovalocytosis, acanthocytosis).

11.2. Associated with deficiency of enzymes in erythrocytes.

11.3. Associated with a violation of hemoglobin synthesis (sickle cell anemia, hemoglobinosis, and thalassemia).

12. Purchased.

12.1. Autoimmune diseases.

12.2. Paroxysmal nocturnal hemoglobinuria.

12.3. Medicinal.

12.4. Traumatic and microangiopathic.

12.5. Due to poisoning with hemolytic poisons and bacterial toxins.

IV. Anemia mixed

Along with the pathogenetic classification, there is a morphological classification of anemia (Wintrobe, Lukens, Lee, 1993), in which the main feature is the size of the erythrocyte:

I. Macrocytic anemia (MCV *> 100 μm3 (FL), erythrocyte diameter> 8 μm)

1. Megaloblastic: deficiency of vitamin B_{12}, folic acid

2. NonMegaloblastic

II. Microcytic anemia (MCV <80 μm^3 (FL**), erythrocyte diameter <6.5 μm): iron deficiency

III. Normocytic anemia (MCV 81-99 μm^3 (FL), erythrocyte diameter 7.2-7.5 μm):

recent hemorrhage, hemolysis of erythrocytes

* MCV (mean corpuscular volume) - mean erythrocyte volume

** FL - femtolitre.

Classification of anemia by color index (CI) allows the doctor to narrow down to a certain extent circle of diagnostic search and simplify differential diagnosis:

Normochromic anemia (CI-0.85-1.05):

- Hemolytic (except thalassemia).
- Aplastic.
- Anemia of chronic diseases

Hypochromic anemia (CI less than 0.85):

- Iron deficiency.
- Sidero-acridical.
- Thalassemia.
- Anemia of chronic diseases.

Hyperchromic anemia (CI greater than 1.05):

- B_{12}-vitamin-deficiency anemia.
- Folic deficiency anemia.

In terms of severity, anemia is distinguished by mild (hemoglobin level of blood above 90 g/l), moderate (hemoglobin - 70-89 g/l) and severe (hemoglobin less than 70 g/l).

At the same time, the International Classification of Diseases of the World Health Organization (ICD-10) provides for the main sections dealing with anemia, with an appropriate code for individual nosological forms, which are given below.

1. D50-D53 - Anemias associated with diet:

D50 - iron deficiency;

D51 - B_{12}-vitamin -deficient;

D52 - folic deficiency;

D53 - other anemias associated with diet.

2. D55-D59 - hemolytic anemia:
D55 - associated with enzymatic disorders;
D56 - Thalassemia;
D57 - sickle cell disease;
D58 - other hereditary hemolytic anemia;
D59 - acute (acquired) hemolytic.
3. D60-D64 - aplastic and other anemia:
D60 - Acquired red cell aplasia (erythroblastopenia);
D61 - other aplastic anemia;
D62 - acute posthemorrhagic anemia;
D63 - anemia of chronic diseases;
D64 - other anemia

Algorithm of diagnostic search in anemic syndrome

I. Syndrome diagnosis: the determination of the pathogenetic variant of anemia.

II. Nosological diagnosis: the detection of a disease or pathological process, which is the basis of this anemia.

Laboratory data are the basis for the diagnosis of anemia. Mosta clinically important blood test is a diagnostic and important laboratorytest.Correct interpretation of blood test results allows not only establishing the presence of anemia, but also carrying initial differential diagnosis. In addition, the main laboratory tests used to diagnose anemia include the definition ofthe content of iron, ferritin and transferrin in the serum, as well as a studybone marrow using a sternal puncture or trepanobiopsy.

Laboratory research necessary to determine the pathogenetic variant of anemia:

•**Hematologic**: Hb, Er, gemotocrit (Htc), color index (CI), average Er volume (MCV), mean erythrocyte Hb concentration (MCHC), mean Hb content in erythrocyte(MCH), reticulocytes (Rtc), leukocytes (Le) + formula, platelets (Tr), ESR, smear review blood to determine abnormal cell forms, determination of blood groups and Rh factor.

• **Serum and plasma**: iron concentration, total iron binding capacity serum, ferritin, transaminase, alkaline phosphatase, lactate dehydrogenase, bilirubin, blood proteins, urea, creatinine.

• **Urinalysis**: color, pH, transparency, specific gravity, protein, urobilin, erythrocytes, microscopic examination of the precipitate.

• **Chair:** color, consistency, occult bleeding, research on eggs of worms.

Bone marrow examination:

- sternal puncture (cytosis, morphological study of cells, correlation erythroid and myeloid cells);
- trepanobiopsy (bone marrow cellularity, morphological study of cells).

Additional laboratory studies are used in the diagnosis of anemia. Anemia caused by impaired production of red blood cells:

- Vitamin B_{12} content in blood serum (vitamin B_{12} deficiency);
- folate content in serum (deficiency of folic acid);
- Electrophoresis of hemoglobin (thalassemia);
- Sternal puncture - cytochemistry, cytogenetics (acute leukemia);
- trepanobiopsy (myelofibrosis).

Hemolytic anemia:

- bilirubin in the blood serum (hemolysis);
- Lactate dehydrogenase in the blood serum (hemolysis);
- Hemoglobin in the blood plasma (intravascular hemolysis);
- Haptoglobin in the blood serum (intravascular hemolysis);
- methemalbumin in the blood serum (intravascular hemolysis);
- Hemoglobin in the urine (intravascular hemolysis);

- hemosiderin in the urine (intravascular hemolysis);
- Coombs probe (immuno-hemolytic anemia);
-title of cold agglutinins (immuno-hemolytic anemia);
-osmotic resistance of erythrocytes (hereditary microspherocytosis);
- Electrophoresis of hemoglobin (hemoglobinopathy);
-G-6-FD in erythrocytes (deficiency of G-6-PD);
-a sucrose test and Ham's test (paroxysmal nocturnal hemoglobinuria).

Additional studies are performed in cases where previous tests with a high degree of probability indicate the presence of a particular anemia. Complex of laboratory and instrumental surveys for identifing the disease or pathological process underlying this anemia, for example, the determination of the cause of iron deficiency (anamnesis, examination of feces for latent blood, colonoscopy, ultrasoundof internal organs, esophagogastroduodenoscopy, urinalysis by Nechiporenko and others).

Differential diagnosis of hypochromic anemia (iron deficiency and iron saturation).

It is known that all iron deficiency anemias are hypochromic, but not every hypochromic anemia is iron deficient. Therefore, differential diagnosis is primarily carried out with other hypochromic anemia, which, in particular, include sidero-acridical ("iron-unsaturated") anemia (with a violation of heme synthesis due to inhibition of enzyme activity, including iron in heme composition). This group includes hereditary sidero-achrestic and acquired (medicamentous) anemia, as well as anemia with lead intoxication. This group of anemia is fundamentally different from IDA in that there is not a flaw in the body, but an excess of iron, which can be determined by appropriate tests. When viewing a blood smear in case of lead poisoning basophilic puncture in erythrocytes appears coarser, whereas with IDA a gentler granularity is noted. In this case, a screening test may be the determination of free erythrocyte protoporphyrin (norma 2.7-9.0 μmol/l), the level of which is usually increased in IDA and, conversely, with lead intoxication - with impaired porphyrin metabolism. To hypochromic anemia is thalassemia - hereditary hemolytic anemia with a violation of hemoglobin synthesis. In differential diagnosis, thalassemia is associated with a family history, the presence of signs of hemolysis and the definition of fractions of hemoglobin. In addition, in cases of detection of microcytosis with moderate severity anemia, it is very important to determine the indicator RDW (red blood cell distribution width) (an indicator of anisocytosis of erythrocytes) - elevated for IDA and normal for thalassemia, where there is a more homogeneous population of red blood cells.

There may be a need for differential diagnosis with the so-called anemia of chronic diseases (ACD) - iron-redistributive anemia with a complex pathogenetic mechanism, in which, as in the case of IDA, hypochromic anemia is combined with a low level of reticulocytes. With ACD as well as with other above conditions (thalassemia, sideroblastic anemia), serum ferritin is normal or elevated. However, if the level of serum ferritin at the lower limit of the norm can be difficult distinguish anemia in chronic diseases from iron deficiency. Moreover, some patients combine these two conditions. An early differential diagnostic criterion for IDA and ACD is the ratio of the concentration of soluble serum transferrin receptors and ferritin levels. Serum receptor levelTransferrin is normal in cases of anemia in chronic diseases. Simultaneous detection of serum and ferritin TfR levels provides very high sensitivity and specificity in detecting depletion of iron stores and in the future can replace such currently accepted iron exchange rates as determined separately serum iron, transferrin and ferritin.

We should not forget about the likelihood of polydeficiency anemia, especially in elderly people, often depending on a number of geographic and socio-demographic factors. The likelihood of the combined nature of anemia in diseases or resection of the small intestine is high. Normal MCV at high RDW indicates the possibility of mixed the etiology of anemia, in particular, the combination of iron and B_{12} and folio deficiencies. It is extremely important to determine the cause of iron deficiency (anamnesis, examination of feces for latent blood, colonoscopy, ultrasound of internal organs, esophagogastroduodenoscopy, and urinalysis according to Nechiporenko et al.).

Table - Indicators of iron metabolism in true IDA and IDA of redistributive genesis

Indicators	Norm	True IDA	Iron-redistributive anemia
Blood serum iron, μM/l	12,0–26,0	<12,0 ↑	↓
Total iron binding capacity of serum, μM/l	42,3-66,7	>67,0 ↑	N ↓ or
UICS, μM /l	22,2-49,6	>50,0 ↑	N ↓
transferrin saturation factor, %	20,1–49,4	↓ <16,0	↓
Serum Ferritin	Wom.17,0-20,0 Men33,0–65,0	↓ <12,0	↑ N

Differential diagnosis of hyperchromic anemia (B_{12} and folic deficiency).

To clarify the diagnosis and differentiation of FDA and B_{12}-DA, it is advisable to determine the level of serum and erythrocyte folates. However, serum folate levels change in wide limits and can temporarily normalize with admission with food, and therefore does not always accurately reflect the degree of their deficiency in the body. A more reliable indicator of folate stocks is the level of folate red blood cells. However, as a standard study, the definition of folate in erythrocytes has not been established in clinical practice due to the complexity and lack of informativity. Differential diagnosis of FDA is based on the same principles as B_{12}-DA. At the same time, it must be borne in mind that often associated anemia, especially in pregnant women.

To distinguish between B_{12}-DA and FDA, it is necessary to identify the characteristic etiological factors, to determine signs of funicular myelosis inherent in B_{12}-DA. Most biochemical indicators in this case are of little informative, except for the direct determination of the concentration of cobalamin and folate in the patient's blood. Thus, the increase in the level of methylmalonic acid and total homocysteine are available both with a deficiency of cobalamin, and with a deficiency of folate. However, the level of homocysteine increased due to cobalamin deficiency does not change under the action of folic acid treatment, and vice versa. Therefore, repeated studies during specific therapy sometimes make it possible to distinguish between the two diseases.

The same applies to the development of reticulocyte crisis in the trial of B_{12} or folic acid. There is also a possible combined deficit of folate and iron deficiency, which is characterized by

normocytic anemia with a heterogeneous population of erythrocytes. In this case, the study shows the parameters of iron metabolism and the level of folic acid.

Differential diagnosis of normochromic anemia (hypo- and aplastic, hemolytic).

Accelerated red blood cell (RBC) destruction is called hemolysis. When bone marrow compensation is adequate, hemoglobin levels remain unchanged; however, if RBC destruction surpasses production, anemia will result. Hemolytic anemiais traditionally categorized as either congenital or acquired.The term "acquired hemolytic anemia" was first coined in the early 1900s and it is now commonly used to describe hemolytic anemia caused by antibodies (with or without complement), drugs or mechanical trauma to RBCs. Acquired hemolytic anemia can be classified as immune (autoimmune, alloimmune or drug-induced) and non-immune (infection-induced, mechanical trauma and paroxysmal nocturnal hemoglobinuria) and different causes of hemolytic anemia can overlap; for example, drug-induced hemolysis may be caused by immune mechanisms or by direct damage to the RBC membrane. In this chapter, the pathogenesis, clinical presentation and treatment of acquired hemolytic anemia will be reviewed. The symptoms of acquired hemolytic anemia relate to the severity of the anemia and the rate of RBC destruction. Anemia may lead to cardiovascular symptoms such as dyspnea, angina and tachycardia; or nonspecific complaints of generalized malaise and dizziness. Rapid destruction of RBCs can be associated with fever, abdominal pain, backpain or limb pain, whereas patients with hemolytic anemia that develops gradually are often asymptomatic. Signs of anemia include dyspnea, pallor, jaundice and brown discolored urine and in massive acute hemolysis, shock andrenal failure can occur.The laboratory tests useful for the diagnosis of acquired hemolytic anemia include peripheral blood examination, reticulocyte count, direct antiglobulin test (Coombs'test), lactate dehydrogenase (LDH), bilirubin, aspartateaminotransferase (AST), haptoglobin, hemoglobinemia, methemalbumin and hemopexin, hemoglobinuria and hemosiderinuria. Bone marrow examination may be usefulto uncover an underlying cause. The determination of RBC life span with radioactive isotope-labeled RBCs is rarely indicated. Laboratory tests can help determine whether the hemolytic anemia is occurring predominantly in the intravascular or extravascular space (table 2).

Table 2 -Laboratory features of intravascular and extravascular hemolysis

Indicators	Intravascular	Extravascular
Serum bilirubin	Elevated	Elevated
Serum lactate dehydrogenase	Elevated	Elevated
Serum gaptoglobin	Reduced or absent	Reduced or absent
Hemoglobinemia	Present	Absent (may be present with severe extravascular hemolysis)
Hemoglobinuria	Present	Absent
Urine hemosiderin	Present	Absent
Hemopexin-heme	Present	Absent (may be present with severe extravascular hemolysis)
Methemalbumin	Present	Absent

General clinical and laboratory manifestations of all hemolytic anemia:

- jaundice with a lemon-yellow hue (increased indirect bilirubin);
- Urine of dark, sometimes black color (urobilinuria or hemoglobinuria);
- feces of normal color or saturated color (excess of sterocilin in the feces);
- enlargement of the liver and spleen;
- hemosiderosis of organs and tissues (increase of serum iron);
- Anemia of normochromic nature (except for thalassemia, it is hypochromic);

• decreased osmotic resistance of red blood cells (except thalassemia, there it is increased);
• reticulocytosis, often leukocytosis, aniso- and poikilocytosis;
• Disturbance of microcirculation due to vascular obstruction.

The absolute criterion of hemolysis is a decrease in the lifespan of erythrocytes, determined by the radioisotope method. In a wide clinical practice, this study is usually not available, so it is necessary to focus on the indirect criteria of hemolysis.

Indirect criteria for hemolysis are:

1. The fact of the presence of anemia, i.e. decrease hemoglobin.
2. Reticulocytosis of blood more than 2%.
3. Erythroid bone marrow hyperplasia (an increase in erythrocaryocytes in the bone marrow is greater than 20-24%).
4. Increased concentration of free or "indirect" bilirubin in blood serum.
5. Reduction of haptoglobin concentration in blood serum due to its binding to free hemoglobin.

The following two indirect criteria of hemolysis in the number of previous ones allow one to suspectintravascular hemolysis:

6. Hemosiderinuria. The appearance of hemosiderin in urine, determined by thecolor of its sediment,indicates that the filtration threshold through the glomerulus of the freehemoglobin plasma.
7. Hemoglobinuria.

Differential diagnosis of aplastic anemia is carried out with leukemia, occurring with cytopenia, with agranulocytosis, autoimmune thrombocytopenia, and hypersplenism. With leukemia, spleen is usually enlarged; in the punctate bone marrow increased cellularity, increased the number of blast elements, which is not characteristic for aplastic anemia. With agranulocytosis there is no anemia and thrombocytopenia; in the myelogram the number of granulocyte germ cells is reduced, but megakaryocytosis is preserved. Hypersplenism occurs in liver cirrhosis, thrombophlebitis spleen, accumulation diseases (Gauchers disease, etc.). With liver diseases, yellowness (more often expressed) of the skin is observed due to damage to its tissue; syndromes of cytolysis, cholestasis, and liver-cell insufficiency are revealed, which is not typical for aplastic anemia. In the postthrombophlebitic spleen, it is enlarged; at palpation it is sharply morbid; when auscultation can sometimes be determined above it, the friction noise of the peritoneum; punctate bone marrow - cellular with delayed maturation cells granulocyte germ; the content of megakaryocytes remains normal, but platelet loosening is impaired. Gaucher's disease can be accompanied by cytopenia, with objective research - pronounced splenomegaly; rarely there is hemorrhagic syndrome; in the punctate of the bone marrow and spleen, specific cellular elements (Gaucher cells) are found with the preserved cellularity of the bone marrow.

Iron deficiency anemia: causes, features of clinical manifestations, blood picture, medical tactics, patient examination plan, outpatient treatment, rehabilitation, examination of temporary disability, prevention.

Iron deficiency anemia (IDA) - pathological condition, which are based on deficiency of iron in the body, accompanied by a violation of the synthesis of iron-containing pigment heme in the hemoglobin molecule and, as a consequence, the development of anemia. Deficiency of iron in the body develops due to a violation of its intake, assimilation or increased losses (imbalance between needs and the intake of iron in the body). WHO iron deficiency anemia is recognized as one of the important social problems, taking into account the wide spread of this pathology, including among women of childbearing age and children, and the fact that social factors (standard of living, education, health) affect the frequency morbidity. Iron deficiency anemia is a widespread pathological condition and accounts for more than 80% of all anemia. The most common disease occurs in developing countries, and in different population groups a high predisposition to iron deficiency and iron deficiency anemia was noted in children of the first years of life, adolescents and women of childbearing age. According to various authors, IDA on average suffers from 5-10% of the world's population. In addition, latent iron deficiency is

detected in 12-15% and more of the examined and among women of reproductive age - in 50%.

Classification. There is no common general classification of IDA, but usually the following options:

1) chronic posthemorrhagic iron deficiency anemia (constitutes the main part IDA);

2) juvenile anemia (associated with imbalance of iron metabolism against a background of intensive growth and the beginning of the menstrual period in girls);

3) in terms of severity (see above);

4) stages - prelatent iron deficiency, stage of latent iron deficiency and actually IDA.

Certain stages of development of iron deficiency in the body correspond to the following laboratory indicators.

1. Prelatent iron deficiency - decrease in serum ferritin level below 12 mcg/l (whendetermined by radioimmunoassay); in the desferal test, a decrease in the selection iron with urine less than 0.4-0.2 mg; decrease in the number of sideroblasts in the sternal pointup to 15% or less.

2. Latent iron deficiency (with the appearance of clinical signs of sideropenia) -serum iron less than 14 μM/l; Total iron binding capacity of serum (TIBCS) exceed the normal parameters;decrease in transferrin saturation.

3. Iron deficiency anemia - the appearance of hypochromic anemia.

The reasons for the development of IDA.

1. Chronic blood loss

Gastrointestinal bleeding.

Uterine bleeding.

Tumors:

- cancer of the rectum and large intestine;
- cancer of the esophagus, stomach, small intestine;
-hypernephroma;
- bladder cancer;

Other reasons:

Paroxysmal nocturnal hemoglobinuria;

- hemosiderosis of the lung;
- Hemorrhagic diathesis.

2. Increased need for iron:

- pregnancy and lactation;
- Infancy;
- adolescence (juvenile chlorosis).

3. Insufficient iron supply:

- gastroduodenitis;
- gastrectomy;
- Alimentary iron deficiency, vegetarianism, starvation;
- infectious lesion of the intestine.

4. Violation of iron transport:- with insufficient amount or low functional activity of transferrin.

Clinical and laboratory diagnostic criteria for IDA.

1. Anemic syndrome. Anemic syndrome is caused by a decrease in hemoglobin and a decrease in the number of erythrocytes, insufficient supply of tissue with oxygen and is represented by nonspecific symptoms. Patients complain of general weakness, increased fatigue, decreased performance, dizziness, tinnitus, flies before the eyes, palpitations, shortness of breath during physical exertion, the appearance of syncope. There may be a decrease in mental performance, memory impairment, drowsiness. Subjective manifestations of anemic syndrome initially disturb patients with physical exertion, and then in rest (as anemia develops).

2. Sideropenic syndrome: dystrophic changes in the skin and its appendages dry skin, fragility and stratification of nails, coilonihia, hair loss), angular cheilitis, stomatitis,perversion

of taste and smell, muscle pain and muscle hypotension (dysuria and incontinence urine when coughing, laughing).

Peripheral blood: hypochromic anemia (decrease: CI, MCH-average content hemoglobin in erythrocyte, MCHC-average hemoglobin concentration in erythrocyte),microcytic (decrease in MCV-average volume of erythrocytes), in the smear -anisocytosis Er, hypochromic ovalocytosis, the presence of target cells.

Metabolic parameters of iron: decreased serum levels, increased TIBCS, decreased percentage iron saturation of the transferrin, a decrease in serum ferritin in the blood, an increase in free erythrocyte protoporphyrin (normal - 2.7-9.0 μM/l) – veryspecific for diagnosis of IDA.

Bone marrow: expansion of the erythroid sprout, decrease in number and index maturation of erythroblasts.

Principles of IDA treatment.

1. It is impossible to compensate for iron deficiency without iron preparations.
2. Therapy should be carried out mainly with iron preparations for oral administration.
3. Therapy should not cease after the normalization of Hb.
4. Conduct blood transfusion strictly according to indications.
5. Treatment of the cause of anemia (therapy of peptic ulcer, prompt removal of the tumor, adequate nutrition, etc.), treatment of "background" diseases. Simultaneously, the correction of iron deficiency with appropriate medications is performed.

The therapeutic daily dose of the preparation of iron is from 100 to 300 mg according to the "elementary" (divalent) iron. The duration of taking the therapeutic dose is before the normalization of Hb, which on average takes 4 to 8weeks (normalization of hemoglobin,the disappearance of hypochromia usually occurs by the end of the first month of treatment with adequate doses of drugs). However, in order to saturate the depot, it is recommended that a half dose of iron-containing preparations be used for another 4-8 weeks. After normalization of Hb, a maintenance dose (50% of the therapeutic dose) lasts 1-2 months, depending on the severity of the anemia. Saturation of the depot is determined by means of a complex biochemical study. In the absence of these methods, treatment is carried out empirically. So in IDA therapy, two stages are identified: the stage of normalization of the hemoglobin level and the stage of restoration of the reserve iron reserves. Control of the effectiveness of therapy is an obligatory component of the rational use of iron-containing drugs. First the days of treatment are subjective assessment of subjective sensations, on the 5th-8th day, the definition of a reticulocytic crisis is mandatory (a 2-10-fold increase in the number of reticulocytes in comparison with the initial value). At the third week, the increase in hemoglobin and the number of erythrocytes is estimated. The absence of a reticulocytic crisis indicates either an erroneous prescription of the drug or an inadequately low dose. Indication for parenteral administration of iron preparations - impaired absorption pathology of the intestine (enteritis, a syndrome of insufficiency of absorption, resection of the small intestine, etc.).

Prevention of the development of IDA:

1. Identification of latent IDA in the risk group.
2. Elimination of risk factors.
3. Anti-relapse treatment with iron preparations several times a year in people with unavoidable causes of IDA development.
4. Use of dietary supplements in the diet.

Primary prevention:

1) pregnant women and breastfeeding (iron intake is prescribed in a dose of 30-40 mg by elemental gland from the 31st week of pregnancy for 8 weeks.) A ferroplex is prescribed 1 tablet 3-4 times a day;

2) adolescent girls, especially with heavy menstruation (2 courses of therapy with a duration of 6 weeks with a daily iron dose of 30-40 mg or after menstruation for 7-10 days every month for a year);

3) donors and children of sports schools (1-2 courses of preventive treatment for 6 weeks in

combination with an antioxidant complex); women with profuse and prolonged menstruation (2 courses of therapy with a duration of 6 weeks with a daily iron dose of 30-40 mg or after menstruation for 7-10 days every month for a year.

The forecast is favorable. Therapy may be ineffective when:

a) the continued loss of iron in the body, when blood loss exceeds the capacity to increased absorption of iron with an additional appointment of iron-containing preparation;

b) increased consumption of products that reduce iron absorption;

c) the presence of inflammatory and malignant diseases or an incorrectly established diagnosis of IDA.Thus, with proper diagnosis and adequate therapy, the disease is completely curable.

B_{12} and folic acid deficiency anemia: causes, clinical manifestations and blood picture, differential diagnosis, the patient's plan of inspection, medical tactics, outpatient treatment, rehabilitation, examination of temporary disability prevention.

B_{12}-deficiency anemia (B_{12}-DA) is included in the group of megaloblastic anemia associated with a violation of DNA and RNA synthesis; develops in conditions of deficiency of vitamin B_{12} (cobalamin) in the body and is characterized by a megaloblastic type of hematopoiesis. Megaloblastosis refers to pathological processes characterized by a delay in the maturation of the nuclei of hematopoietic progenitor cells with ongoing development and normal hemoglobinization of the cytoplasm. The result of such nuclear-cytoplasmic dissociation is the production of cells larger than normal cells.

Etiology. The etiology of the disease can be exogenous (alimentary) and endogenous. Alimentary insufficiency is rare among people whose diet includes food of animal origin, since B_{12} is found in all types of animal food and is contained in muscles, parenchymal tissues, and daily requirements for cobalamin are small (about 1 μg). In this case, the body has certain reserves of B_{12}, so that the deficit accumulates over the years, usually among patients with impaired absorption and strict vegetarians. Endogenous causes of vitamin B_{12} deficiency include small intestine resection, total gastrectomy, partial gastrectomy and the creation of jejunal or other intestinal detours. Insufficiency of the pancreas and excessive development of the microflora of the small intestine, which destroys the vitamin, can also cause malabsorption of cobalamin. To endogenous B_{12}-DA carry the anemia of Addison-Birmer caused by atrophy of the mucosa of the fundus of the stomach with the cessation of the secretion of the internal factor of the Castle and the violation of absorption of B_{12}. There is rarely a congenital absence of B_{12}-binding components (the Immerslund-Grasbeck syndrome) or the disruption of the structure of their molecules. A certain proportion of patients with severe cobalamin deficiency suffers from an autoimmune disease - pernicious anemia. The term was proposed by A. Birmer in 1872 when the pathogenesis of this type of anemia was still unclear and all B_{12}-DA patients were referred to this category. Currently under pernicious anemia is meant a disease of an autoimmune nature, characterized by a violation of the secretion of the WF (internal factor or factor of the Castle's protein, which is secreted by the parietal cells of the stomach) of the gastric mucosa. Patients with pernicious anemia have gastric achlorhydria and atrophy of parietal cells of the stomach, which produce an internal factor necessary for normal absorption vitamin of B_{12}. Pernicious anemia is usually detected in middle-aged or elderly people, but it may also develop in young patients.

The main reasons for the development of B_{12}-DA include:

1) gastrectomy, resection of the small intestine;

2) atrophic gastritis with no internal factor and impaired absorption of B_{12};

3) cancer of the fundus of the stomach;

4) enteritis with impaired absorption;

5) helminthiases (invasion by a broad tapeworm) and excessive development of the intestinal microflora (in conditions of multiple diverticulosis, etc.), in which there is a competitiveconsumption of vitamin parasites or microflora;

6) Inadequate intake of B_{12} with food - strict vegetarianism.

Classification. B_{12}-DA, like other types of anemia, is divided into groupsdepending on the severity and etiologic factor.Below is the main options for B_{12}-DA.

I. Exogenous B_{12}-DA.

1. AlmentaryB_{12}-DA.

II. Endogenous B_{12}-DA

1. B_{12}-DA, caused by resection of the stomach, small intestine.

2. B_{12}-DA, caused by enteritis with impaired absorption, helminthic invasion.

3. Addison-Birmer anemia due to atrophy of the mucosa of the fundus of the stomach.

4. Pernicious anemia - autoimmune atrophy of parietal cells.

5. Congenital absence or structural and functional anomalies of B_{12}-binding components (Immerslund-Grasbek syndrome, hereditary transcobalamin deficiency, etc.).

Clinical picture. The disease develops gradually, and often patients seek medical help even with a significant decrease in hemoglobin. The clinical picture of the disease is determined by violations from the gastrointestinal tract (GIT), as well as the hematopoietic and nervous system. Patients complain of dyspepsia, increased weakness, increased fatigue, dizziness and other manifestations, associated with progressive anemia. In a number of patients taste sensations are lost; there is aversion to meat and other types of food. At various stages of the disease, pain and burning sensation can occur in the tongue, especially with the use of acidic products, and the glossitis characteristic of B_{12}-DA is often determined during examination. Initially, the areas of inflammation along the edges and on the tip of the tongue are determined, and then the process extends to the entire tongue and other oral mucosa. Language can take the form of "crimson". Later, the papillae of the tongue atrophy, and it takes the form of "lacquered tongue". At the same time, patients often note violations of sleep, paresthesia in the form of "crawling" numbness of the distal parts of the limbs, the feeling of "cotton feet", sometimes there are pains of radicular nature. Neurological disorders with B_{12}-DA are known as funicular myelosis. The most frequent symptoms are painful paresthesias and atactic gait. The pathological process can involve the eye nerves and the autonomic nervous system. Sometimes the patient has neuropsychiatric disorders in the form of depression, emotional lability, memory impairment or organic psychosis.

In the absence of adequate therapy and the progression of the pathological process, severe trophic disorders, paresis and paralysis of the lower extremities, dysfunction of the pelvic organs, mental disorders with the appearance of delirium, hallucinations and epileptic seizures can be observed in patients. And there may be significant differences between the severity of neurological manifestations and megaloblastic anemia. Thus, in 25% of patients with cobalamin deficiency, mainly neurologic disorders occur with normal or almost normal haematological parameters. For the appearance of patients B_{12}-DA pale skin with a lemon-yellow hue, subicteric sclera, and the face is often puffy. There may be diffuse or local hyperpigmentation of the skin. Often determined by an enlarged painless liver with soft consistency. Slightly splenomegaly may be less common. At physical examination, disturbances of vibrational and proprioceptive sensitivity can be determined.

Diagnosis.

Diagnostic criteria of B_{12}-deficiency anemia:

I Basic diagnostic criteria.

1. Hyperchromic character of anemia comparatively rarely the color index is normal).

2. Characteristic changes in erythrocytes of peripheral blood: an increase in diameter (macrocytosis), volume, preservation of the remains of the core (Jolly's body, Cabot's ring), reticulocytopenia.

3. Characteristic changes from peripheral blood leukocytes: leukopenia, hypersegmentation of neutrophils.

4. Thrombocytopenia.

5. Characteristic changes in the myelogram: the appearance of megaloblasts in the bone marrow, hyperplasia of red hematopoietic shoot, hyper-segmentation of neutrophils (sternal

puncture should be produce before treatment with vitamin B_{12}, since even 1-2 injections of vitamin B_{12} lead to the disappearance of megaloblasts).

6. Development of a clinical picture of funicular myelosis (usually in severe and prolonged course of the disease).

7. Low content of vitamin B_{12} in the blood (the definition of vitamin B_{12} in the blood is most often produced by radioimmunoassay, the deficiency of vitamin B_{12} is reliably confirmed by a value below 150 pg/ml).

II. Additional diagnostic criteria.

1. Atrophic gastritis, absence of hydrochloric acid, pepsin and gastromucoprotein in gastric juice (in 80-90% of patients is classical pernicious anemia, caused by a deficiency of gastromucoprotein).

2. Detection of antibodies to the parietal cells of the stomach, gastromucoprotein or "vitamin B_{12} + gastromucoprotein" in the blood.

3. Positive result of Schelling's test.

4. Increased excretion in the urine of methylmalonic acid. Normally, with urine, 0-3.5 mg of methylmalonic acid is released per day. With a deficiency of vitamin B_{12}, its excretion can increase tens of times.

5. Positive result of trial therapy with vitamin B_{12}. On the 5^{th}-7^{th} day of treatment, the number of reticulocytes in peripheral blood sharply increases (reticulocyte crisis). In the diagnosis of B_{12}-deficiency anemia, the greatest importance is attached to the basic diagnostic criteria, primarily detection in thesternal point of megaloblasts. It is also advisable to determine the content of vitamin B_{12} in the blood. After the diagnosis of B_{12}-deficiency anemia, it is necessary to find out the cause of vitamin B_{12} deficiency. In most patients, it is a violation of the synthesis of the "internal factor of Castle" (gastromucoprotein) due to thedevelopment of diffuse atrophic gastritis (asrule, autoimmune genesis). The diagnosis of diffuse atrophic gastritis is easily established with the help of fibrogastroscopy and biopsy of the gastric mucosa. Other causes of vitamin B_{12} deficiency are established by careful analysis of the clinical picture of the disease and the identification of characteristic symptoms of the disease. A practitioner should always remember that megaloblastic anemia can be combined with stomach cancer. Therefore, all patients with megaloblastic anemia must necessarily perform fibrogastroscopy and a biopsy of the gastric mucosa to exclude stomach cancer. In all patients with B_{12}-deficiency anemia, a stool test (repeated!) must be performed to exclude invasion by a broad tapeworm (can be found in the stool of the egg, ribbon, scraps of strobila). It should also be clarified whether the patient was operated on the stomach and its volume. B_{12}-deficiency anemia develops 3-5 years after gastrectomy in association with no secretion of gastromucoprotein. When resecting 2/3 of the stomach, B_{12}-deficiency anemia in most patients do not develop. B_{12}-deficiency anemia should always be assumed when anemia is detected in an elderly person, especially if it is combined with leukopenia and thrombocytopenia, mild hemolysis syndrome, pain and burning in the tongue, paresthesia. If there is a suspicion of B_{12}-deficiency anemia, it is always necessary to make a sternal puncture beforethe first introduction of vitamin B_{12}.

Survey plan.

1. Total blood count: the determination of the number of erythrocytes, the diameter and volume of erythrocytes, level of hemoglobin, color index, the number of reticulocytes, white blood cells, platelets, leukocyte formula, ESR.

2. General analysis of urine.

3. Biochemical blood test: determination of bilirubin, aminotransferase, total protein and protein fractions.

4. Determination of the blood content of vitamin B_{12} and folic acid.

5. Ultrasound of the liver, spleen, pancreas, gallbladder, kidney.

6. Fibrogastroduodenoscopy.

7. Fibrocolonoscopy.

8. Sternal puncture.

Differential diagnosis.B_{12}-deficiency anemia must be differentiated from diseases in which there is a megaloblastic type of hematopoiesis, as well as with anemia, in which the syndromes of pancytopenia and hemolysis are detected.

Treatment. Therapy of patients with B_{12}-DA is aimed at eliminating the cause of the disease, correcting the available anemic and neurological manifestations and replenishing the cyanocobalamin depot in the body.The main method of treatment of B_{12}-deficient anemia is parenteral (intramuscular) administration of cyanocobalamin in two stages: the stage of saturation of the body with vitamin B_{12} and the stage of maintenance therapy. In the presence of a neurologic syndrome (funicular myelosis), cyanocobalamin is given in a dose of 1000 μg daily intramuscularly for 3-10 days, after which they switch to a standard dose of 400-500 μg/day. In the absence of neurologic syndrome, cyanocobalamin is prescribed in a dose of 400-500 mcg/day. The usual initial daily dose of the drug for severe anemia is a dose of 400-500 μg, some authors have suggested that one-step administration of 1000 μg or more of cobalamin is inadvisable, since in such concentrations the preparation is not fully bound by blood proteins. With an increase in hemoglobin, the dose can be reduced to 200-400 μg per day. In cases accompanied by manifestations of funicular myelosis, large doses of cyanocobalamin are used for a longer time.The indicator of the adequacy and effectiveness of therapy is the reticulocyte crisis, which means a significant increase in the level of reticulocytes on the 3-5th day of vitamin B_{12} therapy, with a maximum rise on the 4-10th day. During the treatment, potassium levels in the blood are monitored, sincehypokalemia may develop. When determining the duration of the course of therapy, it should be borne in mind that the goal of treatment is not only the normalization of clinical and hematological parameters, but also the replenishment of vitamin B_{12} stores in the body, which average 2-5 mg. The ability of drugs to linger in the body is also taken into account. So, oxycobalamine has the ability to better bind to plasma proteins compared to cyanocobalamin. Accordingly, the average course of therapy with cyanocobalamin usually lasts up to 4-6 weeks, oxycobalamine - 2-3 weeks. In the future, supportive therapy, regimens, intensity and duration of which (from several months to several years) depends on the etiology and the initial severity of anemia.

It is necessary to pay attention to the fact that with the probability of a combination of B_{12}-DA and folic deficiency anemia, as well as the impossibility of differential diagnosis between these two conditions, the correct tactic of therapy will be the appointment of cyanocobalamin followed by the addition of folic acid. Otherwise, if patients with B_{12}-DA are treated with folic acid, then hematologic changes may be partially normalized, however neurological disorders will progress, as the deficiency of cobalamin is not compensated. With a combination of vitamin B12 deficiency and iron deficiency therapy is carried out by a combination of preparations of vitamin and iron-containing preparations in appropriate dosages. Moreover, the appointment of the latter can be recommended to patients without clinical signs of IDA in the case of severe B_{12}-DA during the activation of hematopoiesis with cobalamin therapy.

The cause of megaloblastic anemia, which does not regress with specific vitamin therapy, is not a deficiency of vitamins. In this case, a diagnosis is required. Forecast. Prior to the explanation of the pathogenesis of B_{12}-DA and the introduction of cobalamin preparations into practice, the disease was characterized by poor prognosis, up to a lethal outcome, especially with pernicious anemia. Currently, the outlook is favorable. Most patients are cured. In cases where the cause of the disease can not be completely eliminated (total, subtotal gastrectomy andothers), patients need regular, often lifelong maintenance therapy, which will ensure they have no anemia and a good quality of life.

Folic deficiency anemia

Folic deficiency anemia (FDA) is one of the types of megaloblastic anemiacaused by a deficiency of folate in the body.Etiology. This form of anemia often affects people of young and middle age. Deficiency of folate is often the result of in adequate folate in food. The main sources of folic acid are offal, green leafy vegetables and fruits. Rapidly growing children, pregnant women, as well as patients with hemolytic anemia requires more folate and they may

develop deficiency even with adequate diet. Diseases of the upper gastrointestinal tract, gluten enteropathy and Crohn's disease can lead to a violation of folate absorption and its deficiency.

There are a number of medications that, interfering with the metabolism of folate, lead to megaloblastic anemia. Alcohol abuse often leads to megaloblastic anemia, because alcohol interferes with folate metabolism, and sufficient the number of folate with food chronic alcoholics rarely. Thus, the main reasons for the development of the FDA are:

1) alimentary deficiency (lack of fresh vegetables, fruits);

2) enteritis with impaired absorption;

3) taking medications that interfere with absorption and inhibit the synthesis of folic acid (anticonvulsants, oral contraceptives, barbiturates, methotrexate, etc.);

4) chronic alcohol intoxication;

5) increased need for folic acid (pregnancy, hemodialysis, neoplasms, hemolysis, exfoliative dermatitis).

The risk group for folic acid deficiency anemia:

• Persons who do not eat fresh vegetables and fruits or eat poorly

• Pregnant women

• Persons with cirrhosis of the liver

• Patients with hemolytic anemia, myeloproliferative processes, exfoliative dermatitis (psoriasis)

• Persons taking anticonvulsant and tuberculostatic drugs for a long time, oral contraceptives, etc.

• Persons who take antifolia drugs for a long time (Methotrexate, Trimethoprim, Triamteren).

• Patients with chronic enteritis

• Patients with tropical sprue

• Patients with celiac disease

• Patients with chronic hepatitis.

Because folic acid and vitamin B_{12} are involved in various biochemical processes, their deficiency or metabolic disorders lead to the defeat of many organs and systems of the body. Consequences of deficiency of vitamin B_{12} and folic acid:

- megaloblastic anemia;
- Violation of proliferation and macrocytosis of epithelial cells;
- Neuropathy (only with vitamin B_{12} deficiency);
- Increased likelihood of thrombosis;
- impaired spermatogenesis;
- reversible skin pigmentation with melanin (rarely);
- decreased osteoblast activity.

Deficiency of folic acid is usually manifested by anemic syndrome and gastrointestinal lesions, which can be more pronounced than with a deficiency of vitamin B_{12}. Neurological disorders with a deficiency of folic acid is not observed.

Laboratory signs of megaloblastic anemia:

- Macrocytic hyperchromic anemia;
- poikilocytosis (ovalocytes, degenerative forms of erythrocytes), Jolly's body, Kebot's rings; In severe cases, megalocytes and megaloblasts are determined;
- the number of reticulocytes is reduced, there is no reticulocyte reaction to anemia;
- Leukopenia, thrombocytopenia. Characterized by hypersegmentation (6 or more segments) of the nucleus of neutrophils, sometimes there are giant stab neutrophils and metamyelocytes;
- in the bone marrow the pronounced hyperplasia of the erythroid sprout and megaloblastic type of hematopoiesis (increase in the size of erythroid cells, "tender" structure of the nucleus, lag of development of the nucleus from the well hemoglobinized cytoplasm). There are giant cells of the neutrophilic row of anomalous form;
- Increased serum levels of unbound bilirubin, lactate dehydrogenase; the serum iron and

ferritin content is normal.

Diagnostics. The diagnosis of folic acid deficiency anemia is based on the characteristic changes in the hemo- and myelogram with the corresponding clinical picture of the disease. To distinguish between B_{12}-DA and FDA, it is necessary to identify the characteristic etiological factors, to determine signs of funicular myelosis inherent in B_{12}-DA. Most biochemical indicators in this case are of little informative, except for the direct determination of the concentration of cobalamin and folate in the patient's blood.

Treatment. In most patients, folic deficiency anemia can be cured by administering 1 mg of folic acid per day. However, the therapeutic daily dose of folic acid in patients with impaired absorption in the pathology of the gastrointestinal tract is 5-15 mg per day. Duration of treatment is, as a rule, about a month. The therapy is performed under the control of the hemogram parameters (hemoglobin and erythrocyte level, erythrocyte parameters, appearance of reticulocytic crisis) to the normalization of red blood indices. If it is not possible to completely eliminate the factors contributing to the development of folate deficiency, further preventive courses of therapy are conducted.

Conditions accompanied by loss of consciousness: syncope, coma.

Pathogenesis and objective signs of coma, degree of severity.

Systematization of coma in terms of speed and duration.

TERMINOLOGY AND PATHOGENESIS

SYNCOPE is characterized by generalized muscle weakness, decreased postural tone, inability to stand upright and loss of consciousness.

COMA (from the Greek koma - deep sleep) - a complete shutdown of consciousness with a total loss of perception of the environment and itself and with more or less pronounced neurological and vegetative disorders. The degree of coma severity depends on the duration of the neurological and autonomic disorders. The coma of any etiology (ketoacidotic, uremic, hepatic, etc.) have a common symptomatology and are manifested by loss of consciousness, a decrease or disappearance of sensitivity, reflexes, tone of skeletal muscles, and a disorder in the vegetative functions of the body (VFO). Along with this, there are symptoms characteristic of the underlying disease (focal neurological symptoms, jaundice, azotemia, etc.).

The main clinical characteristics of assessing the state of consciousness (AI Konovalov et al, 1982).

Clear consciousness is its complete safety, adequate reaction to the environment, full orientation, wakefulness.

Moderate stunning - moderate drowsiness, partial disorientation, delay in answering questions (often requiring repetition), slow execution of commands.

Deep stunning - deep drowsiness, disorientation, almost complete sleep state, restriction and difficulty of speech contact, monosyllabic answers to repeated questions, performance of only simple commands.

Sopor (unconsciousness, strong sleep) is almost complete absence of consciousness, preservation of purposeful, coordinated defensive movements, opening eyes to pain and sound stimuli, episodically monosyllabic answers to repeated repetitions of the question, immobility or automated stereotyped movements, loss of control over pelvic functions.

Moderate coma (I) - undetectability, chaotic uncoordinated defensive movements to painful stimuli, absence of eye opening to irritants and control of pelvic functions, slight violations of respiration and cardiovascular activity are possible.

Deep coma (II) - undetectability, lack of protective movements, violation of muscle tone, suppression of tendon reflexes, gross violation of breathing, cardiovascular decompensation.

Terminal coma (III) - agonal state, atony, areflexia, vital functions are maintained by respiratory apparatus and cardiovascular drugs.

QUANTITATIVE SYNDROME OF VIOLATION OF CONSCIOUSNESS

Assessment of the depth of impairment of consciousness in emergency situations in an adult without resorting to special methods of investigation can be carried out on the Glasgow scale, where each answer corresponds to a certain score (see Table 1), and in newborns - according to the Apgar scale.

Table 1. GlasgowComa Scale

	1	2	3	4	5	6
Eye	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters incoherent words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes to painful stimuli	Obeys commands

Note that a motor response in any limb is acceptable. The scale is composed of three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person).

Individual elements as well as the sum of the score are important. Hence, the score is expressed in the form "GCS 9 = E2 V4 M3 at 07:35".

The assessment of the state of consciousness is made by summing up the scores from each subgroup. 15 points correspond to a state of clear consciousness, 13-14 to stunning, 9-12 to a match, 4-8 to a coma, 3 points to a brain death.

Note. The correlation between the Glasgow scale and mortality in coma is highly reliable. The number of points from 3 to 8 corresponds to the mortality rate of 60%, from 9 to 12 - 2%, from 13 to 15 about 0%.

INTERPRETATION

Tracheal intubation and severe facial/eye swelling or damage make it impossible to test the verbal and eye responses. In these circumstances, the score is given as 1 with a modifier attached (e.g. "E1c", where "c" = closed, or "V1t" where t = tube). Often the 1 is left out, so the scale reads Ec or Vt. A composite might be "GCS 5tc". This would mean, for example, eyes closed because of swelling = 1, intubated = 1, leaving a motor score of 3 for "abnormal flexion".

The GCS has limited applicability to children, especially below the age of 36 months (where the verbal performance of even a healthy child would be expected to be poor). Consequently, the Pediatric Glasgow Coma Scale was developed for assessing younger children.

In addition to assessing the violation of consciousness and clarifying the etiological factor, it is important to assess the general condition of the patient.

In the clinic, there are 5 degrees of severity in the general condition of the patient: satisfactory, of medium severity, severe, extremely severe and terminal.

Satisfactory state-the consciousness is clear. Vital functions are not violated.

A condition of average gravity-consciousness clear or there is a moderate stunning. Vital functions are not significantly affected.

Severe state-consciousness is broken to a deep stun or sopor. There are pronounced disorders of the respiratory or cardiovascular systems.

The condition is extremely severe-a moderate or deep coma, grossly expressed symptoms of the defeat of the respiratory and / or cardiovascular systems.

The terminal state is an exorbitant coma with coarse signs of trunk damage and impairment of vital functions.

The overwhelming majority of coma, depending on the etiologic factor, can be reduced to the following three groups:

1. Diseases not accompanied by focal neurological signs. The cellular composition of the cerebrospinal fluid is normal. Computer tomography (CT) and magnetic resonance imaging (MRI) are normal. To this group belong:

• intoxication (alcohol, barbiturates, opiates, anticonvulsants, benzodiazepines, tricyclic antidepressants, phenothiazines, ethylene glycol, etc.);

• metabolic disorders (hypoxia, diabetic acidosis, uremia, hepatic coma, hypoglycaemia, adrenal insufficiency);

• severe common infections (pneumonia, typhoid, malaria, sepsis);

• Vascular collapse (shock) of any etiology and cardiac decompensation in old age;

• epilepsy;

• hypertensive encephalopathy and eclampsia;

• Hyperthermia and hypothermia.

2. Diseases that cause irritation of the meninges with an admixture of blood or cytosis in the cerebrospinal fluid, usually without focal cerebral and stem marks. CT and MRI can be normal or altered. The diseases of this group include:

• subarachnoid hemorrhage with aneurysm rupture;

• acute bacterial meningitis;

• some forms of viral encephalitis.

3. Diseases accompanied by focal stem or lateral cerebral signs with or without changes in the cerebrospinal fluid. CT and MRI reveal pathological changes. This group includes:

• cerebral hemorrhages;

• cerebral infarctions due to thrombosis or embolism;

• Brain abscesses and subdural empyema;

• epidural and subdural hematomas;

• brain contusion;

• brain tumors.

According to the simplified classification, a coma is divided into a destructive (anatomical) and a metabolic (dysmetabolic) one.

Systematization of types of loss of consciousness

For a systematized approach to diagnosis and emergency care, all accidents with loss of consciousness are most conveniently considered by the following types:

1. Sudden and short-term loss of consciousness.

2. Sudden and prolonged loss of consciousness.

3. Continuous loss of consciousness with a gradual onset.

4. Loss of consciousness with unknown beginning and duration.

The term "sudden and short-term" implies a duration of loss of consciousness from a few seconds to several minutes, and the term "gradual and continuous" means hours or days.

GENERAL RECOMMENDATIONS FOR EMERGENCY CARE

The issues of rendering emergency care to victims who are in an unconscious state have their own specifics: the limited time in a life-threatening condition, the absence of an anamnesis and the history of the disease makes the doctor to be extremely collected and follow the general recommendations below.

1. If possible, an eyewitness should be interviewed according to the scheme given below. Correct interpretation of the obtained data can be a good help in setting a clinical diagnosis.

Scheme of an eyewitness interview

• Time of day

• A place

The provoking factor, heat, excitement, pain, change in body position, physical activity, etc.

• The initial position of the body: standing, sitting, lying

• Scream

• Skin color: pallor, hyperemia, cyanosis

• Pulse: frequency, rhythm, filling

• Movement, convulsive or involuntary; local or shared

• Injury due to fall, involuntary urination

• Seizure duration

• Symptoms of recovery, headache, confusion, speech disorders, paresis, etc.

2. Any kind of loss of consciousness can be either a consequence or a cause of craniocerebral trauma (CCT), so at the initial stages of diagnosis and treatment it must be excluded or confirmed. Do not forget that with a sudden loss of consciousness, you can hit your head on solid objects, which in itself can cause TBT.

3. Quite often the cause of a coma is alcohol intoxication, but even with its very characteristic signs, alcohol can not be considered the root cause of coma, until the "drunken" trauma is eliminated and a laboratory confirmation of a high concentration of alcohol in the blood is not obtained.

4. When examining a patient who has lost consciousness, you need to determine the degree of impairment of consciousness, its etiology and assess the overall condition of the patient.

SUDDEN AND SHORT-TERM LOSS OF CONSCIOUSNESS

The most common cause of sudden and short-term loss of consciousness can be:

1. A simple syncope.

2. Transient constriction or occlusion of arteries supplying the brain.

A simple syncope

The diagnosis of SIMPLE SYNCOPE (postural syncope) can be exposed to the victim only if the loss of consciousness occurs in an upright position, and its recovery occurred after several tens of seconds (up to 5 minutes) after the body is in a horizontal position.

Etiology.

The provoking factors of simple fainting can be:

1. Sharply rising or prolonged standing, especially on heat (an orthostatic type of fainting).

2. Factors that activate vasovagal reflexes - pain, blood type, fear, psycho-emotional overload, urination, defecation, cough (vaso-depressor (vasovagal) type of fainting).

3. Depression of the area of the carotid sinus (syncope in the syndrome of carotid sinus hypersensitivity).

4. Vegetative neuropathy.

5. Uncontrolled administration of hypotensive, sedative, antihistamines and other drugs.

Pathogenesis.

Simple syncope is associated with a short-term decrease in the venous tone of the vessels of the lower extremities and the abdominal cavity, ie, the volume of circulating blood (VBC) becomes relatively small for the vascular bed and blood is deposited at the periphery. This causes a decrease in venous return and a drop in cardiac output, and as a result, there is a disruption in the blood supply to the brain. The basis of the vasodepressor type of syncope (with defecation, urination) is a sharp increase in intrathoracic pressure during straining, which causes a decrease in venous influx and a drop in cardiac output.

Clinic.

Fainting can occur suddenly or with precursors. Harbinger of the development of a simple fainting is the appearance in the victim of a feeling of weakness, dizziness, nausea, darkening in the eyes. Objectively at this time you can note the pallor of the skin, drops of sweat on the face, bradycardia and hypotension. With a loss of consciousness, there is a decreased tone of the musculature and weakening of the tendon reflexes. A characteristic feature of simple syncope is the appearance of a sinus bradycardia. Rapid restoration of consciousness in the horizontal position confirms the correctness of the diagnosis of fainting. With deep fainting, urinary incontinence is possible, but this syndrome is more common in epilepsy.

Differential diagnostics

1. Internal bleeding. If it is present, especially in a slow flow with no pain syndrome and visible discharge of blood, the patient may have a fainting with a fairly rapid recovery of consciousness in the horizontal position of the body, but the preservation of tachycardia, instead of the typical bradycardia, dyspnea and pallor of the skin, will be indirect signs Existing anemia The decisive role in this situation is the study of indicators of red blood.

2. The painless forms of acute myocardial infarction or thromboembolism of the pulmonary artery may be accompanied by a brief loss of consciousness.

With the horizontal position of the victim's body after restoration of consciousness, there are signs of respiratory and circulatory insufficiency with signs of overloading of the small circle of blood circulation, disturbances in the rhythm of cardiac activity, etc.

In typical cases, short-term loss of consciousness for the above reasons occurs when the body is upright (standing or sitting). If the loss of consciousness occurred in the victim lying down, you should think about a violation of the rhythm of the heart activity (first of all - the attack of Morgagni-Edessa-Stokes, or the violation of cerebral circulation.) Sudden and short-term loss of consciousness against the background of narrowing or occlusion of the arteries supplying the brain.

This variant of pathology is mainly found in elderly people against the background of atherosclerotic lesions of arteries, blood supplying the brain. At the heart of pathogenesis can be:

1. Spasm.

2. Embolism of individual parts of the brain by small emboli formed at the site of narrowing of the arteries.

3. Mechanical enhancement of existing occlusion.

4. "Syndrome of subclavian stealing".

5. Aortic stenosis.

1. Spasm of the cerebral arteries, as a cause of cerebral circulatory disturbances, can be assumed if syncope has occurred against the background of a migraine attack or hypertensive crisis.

2. The place of stenosis of vertebral or carotid arteries, blood supplying the brain, can be a source of micro-embolism. When the patient leaves the syncope of this etiology, a characteristic feature is the appearance of a specific neurological symptomatology.

3. Loss of vision in one eye (transient amaurosis) or immediately after fainting hemiparesis indicate acute impairment of blood circulation in the carotid artery system, the appearance of dizziness, hemianopsia, diplopia and imbalance indicates an acute impairment of blood circulation in the vertebrobasilar artery system.

4. An unconscious condition arising on the background of a mechanical enhancement of the existing stenosis of the vertebral arteries is called the syndrome of the Sistine Chapel. This condition was first described in the elderly tourists in Rome, when examining the frescoes of Michelangelo on the dome of the Sistine Chapel. Loss of consciousness is associated with prolonged overdistension of the neck and compression or kinking of the vertebral arteries.

5. "Syndrome of subclavian stealing" arises against the background of the initial stenosis of the subclavian arteries proximal to the site of the deviation of the shielded trunk. With intensive hands, the blood flow in the vertebral arteries becomes retrograde and acute cerebral ischemia occurs.

6. Short-term loss of consciousness is possible against aortic stenosis, with rapid exercise; a harbinger of syncope may be the appearance of ischemic pain in the heart.

In children, less often in adults, one of the reasons for a short-term loss of consciousness may be a "small epileptic seizure" (absent). During such an attack, it is sometimes possible to notice instant movements of the muscles of the face, eyes or extremities. The duration of these seizures is so short that the victim does not have time to fall and can only drop what was in his hands.

If within a few minutes of the patient, despite the emergency assistance. Emergency care in case of unconsciousness), consciousness is not restored, one should think about the development of a COMATOSE STATE.

Comatose states can be manifested by a sudden and prolonged loss of consciousness and a gradual and prolonged loss of consciousness.

SUDDEN AND PROLONGED LOSS OF CONSCIOUSNESS

Sudden and prolonged loss of consciousness can be a manifestation of acute disturbance of cerebral circulation, hypoglycaemia, epilepsy, and hysteria. Clinical signs and methods of treatment of these conditions are given in the chapters mentioned in the book. Examination of any patient in an unconscious state should be started, if possible, according to the scheme given in Table. 1. If, on the background of emergency treatment, the patient does not regain consciousness within a few minutes, the contents of the victim's pockets and wallet can serve as additional information: prescriptions for specific medications or medications themselves can prompt the correct way of diagnosis and treatment. The presence of a home telephone number will allow you to quickly contact your relatives and get information on the issues of interest; a diabetic or epileptic card will indicate the likely cause of a coma. To prevent possible undesirable legal complications, check the contents of the pockets should be carried out in the presence of

witnesses, with the subsequent compilation of an inventory of everything found. After this, it is necessary to start a clinical examination in accordance with Scheme of examination of a patient in a coma.

Scheme of examination of a patient in a coma

1. Skin: moist, dry, hyperemic, cyanotic, icteric
2. Head and face: the presence of injuries
3. Eyes: conjunctiva (hemorrhage, jaundice); pupils' reaction to light; the ocular fundus (edema of the disc, hypertensive or diabetic retinopathy)
4. The nose and ears: the excretion of pus, blood; liquorrhea; acrocyanosis
5. Language: dryness; traces of biting or scars
6. Breathing: the smell of urine, acetone, alcohol
7. Neck: stiff neck, rippling of carotid arteries
8. Chest: frequency, depth, rhythm of breathing
9. Heart: rhythm disturbance (bradycardia); sources of cerebral vascular embolism (mitral stenosis)
10. Stomach: enlargement of the liver, spleen, or kidney
11. Hands: blood pressure, hemiplegia, traces of injections
12. Brushes: frequency, rhythm and pulse filling, tremor
13. Legs: hemiplegia, plantar reflexes
14. Urine, urinary incontinence or delay, protein, sugar, acetone

First of all, when examining a patient, CCT should be excluded. At the slightest suspicion, an X-ray examination of the skull in two projections should be made and the specialist should be consulted.

Focal neurological symptoms suggest the existence of an acute disorder of the cerebral circulation.

Fresh bites of the tongue or old scars on it are very likely to indicate epilepsy.

The diagnosis of a hysterical coma will only be exhibited after the complete elimination of organic pathology. It should be emphasized that this complication of hysteria, despite the prevailing opinion, is relatively rare.

The presence of multiple traces of subcutaneous injections in typical places will talk about diabetes, and multiple traces of intravenous injections, often in the most unexpected places, suggest drug addiction.

At the slightest suspicion of a hypoglycemic condition, without waiting for laboratory confirmation, it is urgent to inject iv 40-60 ml of 40% glucose solution. If the patient subsequently has even a ketoacidotic coma, his condition will not deteriorate, and with hypoglycemia this simple method of treatment will save the life of the victim.

LONG-TERM LOSS OF CONSCIOUSNESS WITH GRADUAL ONSET

Comatose conditions that develop gradually in a hospital environment, as a rule, do not present difficulties in diagnosis. So, if the patient has acute liver failure, which is not amenable to treatment, then in the future he may develop a hepatic coma. The main causes of the occurrence of a gradual and prolonged loss of consciousness are given in Table 4.

The diagnosis and treatment of comatose conditions in this table are discussed in the relevant chapters of the textbook.

The most common causes and diagnostic signs of coma with gradual onset and prolonged loss of consciousness

I. Brain pathology

1. Injury Damage to the outer covers or bones of the skull, bleeding or liquorrhea from the nose or ears

2. Vascular disorders of hemiplegia (hemiparesis), hypertension, rigidity of cervical muscles (with subarachnoid hemorrhage)

3 Tumor Focal symptomatology of the central nervous system, edema of the disc of the optic nerve papilla on the affected side

4. Infection The expiration of pus from the nose or ears, rigidity of the neck muscles, fever

5. Epilepsy Seizures on examination or a history, scars or fresh bite marks in the tongue

II. Metabolic pathology

1. Uremia Uremic smell from the mouth, dehydration, muscle twitching, retinopathy, proteinuria

2. Diabetes The odor of acetone from the mouth, dehydration, retinopathy (microaneurysms), sugar and ketone bodies in the urine

3. Hypoglycemia Sweating, trembling, there may be a symptom of Babinsky

4. Hepatic coma Jaundice, splenomegaly, bloody vomiting, "clapping" tremor

III. Intoxication

1. Alcohol The smell of alcohol from the mouth, flushing of the face (you should carefully look for CCT)

2. Psychotropic drugs Disturbance of breathing, moderate hypersalivation

3. Carbon monoxide Disturbance of breathing, characteristic hyperemia

LOSS OF CONSCIOUSNESS WITH UNKNOWN BEGINNING AND DURATION

The greatest difficulties in the work of resuscitation physicians arise when entering the intensive care units of patients with an unknown beginning and duration of coma. In these situations, additional information is provided by a conversation with relatives or neighbors who brought the victim to the hospital. It is necessary to find out the reasons that caused sudden and prolonged loss of consciousness or the appearance of a gradual and prolonged coma. Clinical examination of patients is recommended to be carried out according to the above schemes. A close examination of the victim sometimes gives much more information than a lot of laboratory and functional research methods: low body temperature in hot summer, reduced skin turgor and the presence of bedsores can indicate the duration of coma, stable anisocoria in combination with alcohol intoxication should, in the first place, to suggest the existence of an CCT, and so on.

Emergency care for unconsciousness

If the doctor is a witness of a sudden loss of consciousness, you should remember, and in the future and describe this state according to Table. 2.

The order of rendering of the first aid:

1 Eliminate potentially harmful to the life of the victim external factors: electric current, gas, flame, etc.

2. If the above external factors do not threaten the life of the victim and the activities of his cardiovascular and respiratory systems are adequate, the patient should be given or maintained a horizontal position with an elevated leg end and do not move it until the following additional measures are taken:

• ensure free breathing: unbutton collar, belt;

• Spray the face with cold water, pat on the cheeks;

• the inhalation of stimulants (ammonia, vinegar) helps a lot;

• with prolonged fainting, rub the body, cover with warm heaters; is shown in / m the introduction of 1 ml of a 1% solution of mezaton or s / to 1 ml of 10% caffeine solution; with severe hypotension and bradycardia sc, 0.5-1 ml of a 0.1% solution of atropine sulfate.

Note. The listed activities are adapted to the specific situation.

IF THE LOSS OF CONSCIOUSNESS CONTINUES MORE THAN SEVERAL MINUTES, SHOULD THINK ABOUT THE DEVELOPMENT OF THE COMBAT STATE AND ACCEPT THE FOLLOWING ADDITIONAL MEASURES:

1. Make sure that there is breathing, a pulse on the carotid arteries; if they are not available, begin resuscitation, as in cardiac arrest.

2. In the presence of cramps, to avoid biting the tongue, insert a suitable object (but not metal!) Between the teeth of the patient; to stop the convulsive syndrome.

3. In case of injury, if there is external bleeding, stop it.

4. Look for a medical card (epileptics, diabetics, etc.) in the pockets or wallet, or medications that can cause loss of consciousness, to examine the patient according to the scheme given in Table. 3.

5. Protect the patient from overheating or hypothermia.

6. If the cause of coma remains unclear, non-specific symptomatic treatment, laboratory and instrumental express diagnostics should be performed.

Note. The listed activities are adapted to the specific situation.

Nonspecific symptomatic treatment.

1. Introduce iv in 40-60 ml of 40% glucose solution. If the cause of a coma is a hypoglycemic coma, the patient will come to consciousness. In all other cases, glucose will be assimilated as an energy product. If the patient subsequently reveals even a ketoacidotic coma, the injected glucose to the patient will not cause harm.

2. Normalize intracranial pressure with signs of its increase.

3. Prepare convulsive seizures (diazepam) if they are present.

4. Start fighting infection with signs of bacterial meningitis or purulent otitis media.

5. Restore the KHS and electrolyte balance.

6. Enter vitamin B_1 (thiamine has a cardiotrophic and neuroprotective effect).

7. If there are signs of acute poisoning, select a specific antidote, start antidote therapy.

8. Protect eyes. In patients who are in a coma, the eyelids are elevated, there is no blinking, as a result of which the cornea dries up. To prevent this complication it is necessary periodically, as the cornea dries up, to instill a saline solution or an albucid solution. It is unacceptable to apply moist wipes to the cornea, because if the medical staff does not re-wet them in time, they may dry out.

Note: The listed events are adapted to the specific situation

COMATOSE CONDITIONS IN DIABETES MELLITUS, TYPES, FEATURES OF PATHOGENESIS.

Diabetic coma is a critical dehydration of body tissues with damage to the functions of the brain. It develops at a blood glucose concentration of 19.4-33.3 mmol/l. Under these conditions, as a result of ketoacidosis, K enters the extracellular space, which underlies the violation of the contractile function of the myocardium, as well as of the respiratory musculature. Types of diabetic coma: 1. *Hyperglycemic ketoacidotic coma*. It develops with CD1 due to hyperglycemia, hyperketonemia, metabolic acidosis. Glu and ketone bodies are excreted in the urine, which contributes to an increase in osmotic pressure in the primary urine, loss of sodium and is accompanied by polyuria. There is dehydration, insufficiency of

peripheral circulation and tissue hypoxia. Acidosis causes the breathing of Kusmaul, in which CO_2 is lost and, as a consequence, the disturbances in the water-electrolyte balance, acid-base balance are aggravated, there is a sharp disruption of the metabolism and functions of the CNS cells. In the blood glu 22 mmol/l, ketone bodies 17 mmol/l, increased content of residual nitrogen, urea, cholesterol, LC, sodium level, potassium normal. 2. *Hyperglycemic hyperosmolar coma in DM 2 type*with additional effects of dehydrating factors (diarrhea, vomiting, diuretic intake). Dehydration of the body and development of hyperosmolarity of the plasma develop, glycemia up to 55 mmol/l. Increased blood glucose leads to increased diuresis. Dehydration leads to hypovolemia, stimulation of aldosterone secretion and retention of sodium and chlorine. The osmolarity of the plasma is 500 mosmol/l, which leads to an intracellular dehydration, an abnormal water and electrolyte equilibrium in the brain cells, a hypoxia of the central nervous system with severe neurological symptoms and a loss of consciousness. 3. *Hyperglycemic coma with lactic acidosis*. In the mechanism of its development, a decrease in the activity of the enzymatic pyruvate dehydrogenase complex (with an insulin deficiency) that converts pyruvate to acetyl-CoA, pyruvate in a reversible reaction catalyzed by lactate is converted to lactic acid; use lek drugs stimulating anaerobic glycolysis, thereby increasing the lactate and pyruvate content in the body; a hypoxic condition (in which glycolysis is stimulated) caused by fatigue, the heart and the breath are not available. Lactic acid accumulates to the blood, collapses, the heart and the functions of the dyspnoea center are dysfunctional (depression of the Kusmaul), depression of consciousness, a violation of sensitivity, dysfunction of the gastrointestinal tract, pronounced dehydration of tissues. 4. *Hypoglycemic coma* is associated with the overdose of insulin, prepulfonylureas, the development of secondary hypopituitarism (a consequence of angiopathic vessels of the pituitary gland), weakening the response to hypoglycemia. The cause may be insulinoma, lack of counterinsulin hormones, hepatic forms of glycogenoses, starvation, liver disease, a violation of the digestion and absorption of carbohydrates in the digestive tract. Mechanism: the delivery of gluc to the nerve cells is reduced, which leads to their energy depletion and disruption of the functions of the central nervous system. Less than 3mM/l of gland there is tremor, sweating, a sense of anxiety, hunger, weakness, then a condition resembling alcoholic intoxication, accompanying hallucinations, aggressiveness. At 2.5 mmole, clonic convulsions, loss of consciousness, edema and necrosis of individual parts of the brain.

DIABETES MELLITUS (DM)

According to WHO, there are currently about 100 million patients with diabetes, and 200-300 million suffer from hidden diabetes.

DM is a syndrome of chronic hyperglycemia, which develops as a result of relative or absolute insulin insufficiency, characterized by violation of all kinds of metabolism and, first of all, carbohydrate, as well as development of vascular complications.

Diabetes mellitus develops as a result of many causes, but all of them are pathogenetically expressed in any of the three variants:

1. Defect in the insular apparatus
2. Defect in peripheral tissues of glucose utilization
3. Defect of enzymes

All these three factors lead to a deficiency in the amount of insulin, or to a deficit in its effect.

The etiology of DM is divided into:

1. DM type 1 is characterized by absolute insulin deficiency. It often develops in young people and is usually associated with the destruction of beta cells of the pancreas under the influence of a viral infection (the virus is nonspecific: it can be influenza virus, Coxsackie, paratyphoid, rubella, etc.)

2. DM type 2. If the absolute type of insulin is absent at type 1, then at DM2 the level of insulin is normal or even increased and the provoking factor of development of DM2 is the situation requiring an increased level of insulin. For example, obesity creates conditions for increased insulin consumption by tissues. And while the body has reserves, the clinic does not have diabetes, but if the reserve is exhausted, the relative insufficiency of insulin becomes absolute, which leads to the appearance of the clinic DM. At DM2, the failure of antiviral immunity caused by the HLA system is of less importance, as is the viral infection.

Pathogenetic links of DM develop in any type of diabetes similarly and in the basis of insulin deficiency, which causes a violation in all types of metabolism: protein, water-salt, carbohydrate, fat.

Violation of carbohydrate metabolism:

1. Violation of the transport of glucose through cell membranes leads to inhibition of glucose utilization along the pathway of oxidative phosphorylation.

2. Since insulin is a powerful factor contributing to the synthesis of glycogen in the liver and muscles, then its deficit does not lead to the deposition of glucose in the tissues, which leads to a lack of energy substrates in the tissues (reduced energy capabilities)

3. Insulin suppresses gluconeogenesis (this is the formation of carbohydrates from non-carbohydrate components, often from amino acids). In conditions of deficiency in the violation of the intake of glucose in the insulin cell, activation of gluconeogenesis takes place, with increased protein breakdown and the formation of carbohydrates. This leads to a violation of protein metabolism.

The consequence of these 3 processes is the excessive accumulation of glucose in the blood - the hyperglycemia of which is the basis for diagnosis DM.

If there is a violation of the intake of glucose into the cell, the basic energy mechanisms are violated, while the central nervous system suffers more. Against the backdrop of insulin deficiency, low-insulin glucose metabolism increases (for aerobic glycolysis). This path is less energy-efficient and leads to a gradual accumulation of lactic acid in the tissues, thus lactoacidosis develops. In addition, glucose is utilized in the polyol cycle, the products of which are highly hydrophilic, therefore accumulating cause swelling of the tissue, which is of great importance in the development of complications of diabetes and the development of tissue hypoxia. Due to the activation of bezinsular pathways, energy expenditure can be maintained for some time at the required level. In addition, another compensatory pathway is the enhancement of lipolysis.

Disturbance of fat metabolism: insulin increases the formation of fats. With a lack of glycolysis, lipolysis is stimulated. The fatty acids formed in lipolysis in large quantities can be used as an energy source. Lipolysis is also enhanced by counterinsulant hormones (TSH, cortisol, glucocorticoids). This compensatory reaction at the beginning is positive for the body, but then it also has a negative character, as free fatty acids accumulating in the body can promote fatty liver disease, in addition, underoxylated lipolysis products accumulating in the body (acetoacetic acid, beta-hydroxybutyric acid, acetone) contribute to the development of acidosis. Because enzymes function within certain pH limits, they decrease their activity when shifted toward acidosis.

The body tries to get rid of the ketone bodies and starts to excrete them in the urine (which is determined with the help of special tests). These ketone bodies accumulate in avalanche and acidosis with a lethal outcome occurs. If you do not enter insulin, then acidosis leads to a fatal outcome.

Protein metabolism: there is an increased disintegration, an intensive conversion of it into carbohydrates. Accumulated decay products of the protein - amino acids, urea, nitrogen. There is a hyperaemia, which is also one of the manifestations of decompensation DM.

Water-salt exchange: because of the high osmotic properties of glucose, there is a redistribution of water, i.e. the fluid leaves the cells in the blood, which leads to tissue dehydration (the mechanism of polyuria). The concentration of glucose in primary urine increases, which acquires high-osmotic properties, is stimulated by osmodyurez, which aggravates the dehydration of the body. In this case, the hemodynamics of blood pressure decrease arises, and the death of the patient is a model of hypovolemic shock, in which there are still accompanying complications: hypoxia, which still occurs due to intoxication with products of lactic fermentation, redistribution of electrolytes (potassium, sodium, chlorine). At the same time, potassium is lost. With a lack of potassium, excitability, contractility of the myocardium is disturbed, automaticity and conduction are violated, which leads to violations of the heart rhythm. But with SD all types of metabolism are violated, and with an insulin deficiency, the organism copes for a while, but in the end, decompensation occurs. The patient can not leave this state.

TREATMENT OF DM TYPE 2:

1. Diet. The diet is prescribed for:

- Limits of the insular apparatus from excessive stimulation (decrease in the number of long-term and rapidly digesting carbohydrates)
- normalization of body weight due to reduced caloric content of diet Diet is the main method of treatment (the amount of cereal, bread and potatoes in the diet is needed significantlyreduce, sugar must be excluded from the diet for any type of diabetes)

2-3 weeks try to compensate DM if blood sugar is not normalized, then tableted antidiabetic drugs are prescribed.

DM type2 is usually first treated by increasing physical activity, and eliminating saturated fat and reducing sugar and carbohydrate intake with a goal of losing weight. These can restore insulin sensitivity even when the weight loss is modest, for example around 5 kg (10 to 15lb), most especially when it is in abdominal fat deposits. Diets that are very low in saturated fats have been claimed to reverse insulin resistance.

Cognitive Behavioural Therapy is an effective intervention for improving adherence to medication, depression and glycaemic control, with enduring and clinically meaningful benefits for diabetes self-management and glycaemic control in adults with type 2 diabetes mellitus and comorbid depression.

Testosterone replacement therapy may improve glucose tolerance and insulin sensitivity in diabetic hypogonadal men. The mechanisms by which testosterone decreases insulin resistance is under study. Moreover, testosterone may have a protective effect on pancreatic beta cells, which is possibly exerted by androgen-receptor-mediated mechanisms and influence of inflammatory cytokines.

Recently it has been suggested that a type of gastric bypass surgery may normalize blood glucose levels in 80–100% of severely obese patients with diabetes. The precise causal mechanisms are being intensively researched; its results may not simply be attributable to weight loss, as the improvement in blood sugars seems to precede any change in body mass. This approach may become a treatment for some people with type 2 diabetes mellitus, but has not yet been studied in prospective clinical trials.This surgery may have the additional benefit of reducing the death rate from all causes by up to 40% in severely obese people. A small number of normal to moderately obese patients with type2 diabetes mellitus have successfully undergone similar operations.