LECTURE COMPLEX

Discipline:	Basics of general medical practice
Code:	BGMP 5301
Speciality:	6B10101 «General Medicine»
Amount of hours/credits:	20/8
Cours:	5
Term:	IX

Compiled by Abdraimova S.E.

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Record of meeting № 1

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I. Theme № 1: Basics of general medical practice.

II. Learning goals: Basics of medical practice and organization of work of general practitioner.

III. Lecture thesis:

Primary health care (PHC) is the main and most important link in the organization of public health protection. It is provided mainly on a territorial basis, that is, a medical worker serves a certain number of people living in the territory, and is assigned to him.
The World Health Organization (WHO) gives the following definition of PHC: "Primary health care includes basic curative care - simple diagnosis and treatment of the main common diseases, injuries and poisoning; referral in difficult cases for consultation and treatment to a higher level (directions for the provision of specialized and highly qualified care), preventive care and basic sanitary and educational activities " In developed countries, the leading primary health care provider is the family doctor, or general practitioner. Long-term historical experience and practical implementation of the idea of family medicine in many countries of the world have shown its advantages.
First, the family doctor personified all responsibility for the health of every family. He concentrated in his hands up to 90% of all necessary medical care. Secondly, the institution of a family doctor turned out to be the most successful organizational form, which ensured the economic efficiency of the entire health care system. The need for a further increase in the number of doctors has disappeared, and auxiliary services have been significantly reduced.
The proportion of family doctors ranges from 15% in the US to 50% in Canada. It is a common medical profession everywhere. The number of residents served by a family doctor averages 1,300 in America, 1,500 in Canada, and 2,000 in England.
The general principles of the functioning of family medicine in the developed countries of the world are:
1. A family doctor, freely elected by his patients, he provides care around the clock.
2. An outpatient clinic for a family doctor, as a rule, consists of 3 offices - an admission office, an examination room and an office for auxiliary examinations.
3. The main section of his activities is preventive work. Observing all family members, the doctor determines the risk factor for chronic diseases, conducts preventive examinations for the early detection of arterial hypertension, diabetes mellitus, glaucoma, ischemic heart disease, peptic ulcer disease, neoplasms. The family doctor gives each patient individual advice on the regimen and nature of nutrition, physical education and other types of recreation, recognition of early symptoms of illness.
4. Treatment and diagnostic work. The family doctor diagnoses and treats the vast majority of diseases. When there is a need for assistance from a narrow specialist, or treatment in a hospital, he actively intervenes in these processes, giving his advice and consultations.
5. Sanitary and educational work consists in conducting conversations on the observance of a healthy lifestyle by their wards, the harmful effects on the body of smoking, alcohol abuse, environmental protection, and the care of each patient for their own health.
6. The assistant to the family doctor is, as a rule, a nurse who actively cooperates with him in all his activities.
7. In the vast majority of countries, family medicine has an autonomous status, i.e. removed from the general structure of medical institutions and is directly subordinate to local authorities. The administrative apparatus does NOT control the actions of the family doctor, but before the patient, he is freely chosen or by the local authority that finances him. However, in all cases (directly or through an insurance company) funds are allocated by the state.

Features and content of the work of a family doctor.

A general practitioner / family doctor is a specialist with a higher medical education who has the legal right to provide primary health care to assigned families and other assigned categories of the population, regardless of age, gender and the nature of the pathology.

The position of a family doctor is a doctor who has been trained in internship in the specialty "general practice - family medicine" or doctors of education "general practice" and "pediatrics" who have been trained in the cycle of specialization in family medicine in higher medical institutions of improvement in various forms of training of doctors and received a certificate of a specialist in the specialty "general practice (family medicine)".

The family doctor carries out his activities:

- In a state medical institution (polyclinic, medical unit, city or rural medical outpatient clinic);
- In a non-state medical institution (private, joint-stock, public, etc.).

A family doctor can work individually or in a group of family doctors (group practice). Depending on the structure of the population, a team of doctors serves, it is advisable to involve other specialists, including social workers.

A family doctor works under a contract (agreement) with state or municipal authorities, medical insurance companies, medical institutions.

The contingents of the population, their number are formed taking into account the right to freely choose a doctor and are fixed by the terms of the contract.

The family doctor carries out outpatient visits and home visits, a complex of preventive, diagnostic, therapeutic and rehabilitation measures in all age groups, for all types of diseases and injuries in accordance with the qualification characteristics of a specialist in the specialty "general practice (family medicine)", provides urgent medical care in case of emergency conditions and traumatic injuries, assists in solving medical and social problems of the family.

By agreement, the family doctor can be allocated hospital beds in the hospital for the management of patients. The family doctor organizes home hospitals, day hospitals in outpatient clinics.

The family doctor is subordinate to nursing staff with special training, as well as, in agreement with the social protection authorities, social workers.

The main functions of a family doctor

Provision of primary health care to the population in accordance with the requirements of the qualification characteristics and the received certificate.

Conducting sanitary and educational work to educate the population in the formation, preservation and strengthening of the health of family members, self-and mutual assistance. Providing advice to families on family planning, ethics, psychology, hygiene, social aspects of family life, running a "school for parents".

Implementation of preventive work aimed at assessing the role of environmental factors, identifying early and latent forms of the disease and risk factors.

Implementation of dynamic monitoring of the health of family members with the necessary examination and recovery with an individual complex of medical and recreational activities.

Providing emergency medical care to sick and injured people in emergency conditions, regardless of where they live.

Timely and fully diagnostics and treatment of diseases in outpatient settings, day and home hospitals and at home within the competence of a general practitioner.

Timely targeting of patients to receive medical care in cases beyond the competence of the family doctor; hospitalization of planned and urgent patients in accordance with the established procedure.

Carrying out rehabilitation measures.

Conducting an examination of the temporary disability of patients in accordance with the current legislation and referral to VTEK.

Carrying out timely diagnostics, early detection and appropriate treatment of infectious diseases, implementation of anti-epidemic measures.

Immunoprophylaxis of diseases.

Organization of medical, social and household assistance in conjunction with social protection bodies and mercy services for the lonely, the elderly, the disabled, the chronically ill.

Participation in the implementation of state and regional targeted medical programs.

Analysis of the health status of the population served by a family doctor.

IV. Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Definition of specialty and general practitioner.
Philosophy and principles of family medicine.
The main regulatory legal acts on PHC.
Organization of the work of a family medical outpatient clinic.
General principles of patient-centered counseling.
Identification of risk factors, principles of early diagnosis and prevention of socially significant diseases.
Criteria for evaluating the effectiveness of GPs.

I. Theme № 2: Pneumonia in GMP and "COVID-19".
II. Learning goals: Diagnosis and treatment of pneumonia and COVID-19 on ambulatory level.
III. Lecture thesis:
Pneumonia is an acute infectious disease that arose in a community setting (i.e. outside the hospital or later 4 weeks after discharge from it, or diagnosed in the first 48 hours from the moment of hospitalization, or developed in a patient who was not in nursing homes) nursing/long-term medical observation for ≥14 days accompanied by symptoms of lower respiratory tract infection (fever, cough, sputum, possibly purulent, chest pain, shortness of breath) and radiological evidence of "fresh" focal-infiltrative changes in the lungs in the absence of an obvious diagnostic alternative.

Classification

Community-acquiredpneumonia	**Nosocomialpneumonia**	**Healthcare associated pneumonia**
I. Typical (in patients with lack of immunity disorders): a. bacterial; b. viral; c. fungal; d. mycobacterial; e. parasitic. **II.In patients with severe immunity disorders:** **a.** Acquired Immune Deficiency Syndrome(AIDS); **b.** other diseases / pathological conditions. **II.In patients with severe immunity disorders:** **a.** Acquired Immune Deficiency Syndrome(AIDS); **b.** other diseases / pathological conditions.	I. **Actually nosocomial pneumonia.** **II. Ventilator-associated pneumonia.** **III. Nosocomial pneumonia in patients with severe immunity disorders:** a. in recipients of donor organs; b. in patients receiving cytostatic therapy.	Pneumonia in the inhabitants of houses for the elderly. II. Other categories of patients: a. antibiotic therapy in the previous 3 months; b. hospitalization (for any reason) for ≥ 2 days in previous 90 days; c. stay in other long-term care facilities; d. chronic dialysis for ≥ 30 days; e. treatment of the wound surface at home; f. Immunodeficiency conditions / diseases.

Criteria for the severity of pneumonia

Mild course –
unexpressed symptoms of intoxication,
subfebrile body temperature,
no respiratory failure and hemodynamic disturbances,
pulmonary infiltration within 1 segment,
white blood cells 9.0-10.0x109/l,
no concomitant diseases
Moderate severity –
Moderate intoxicationsymptoms,
fever up to 38 ° C,
pulmonary infiltrate within 1-2 segments,
respiratory rate up to 22 / min, heart rate up to 100 beats / min,
no complications.
Severe:
severe intoxication symptoms,
body temperature <35.5 °C or > 38 °C;
II-III respiratory failure,
hemodynamic disorders (blood pressure <90/60 mm Hg, heart rate> 100 beats / min),
infectious toxic shock,
leukopenia < 4.0x109/l or leukocytosis 20.0x109 / l;
infiltration in more than one lobe of the lung;
the presence of a decay cavity;
pleural effusion, sepsis, insufficiency of other organs and systems, impaired consciousness, exacerbation of concomitant diseases.
Etiology
Streptococcuspneumoniae - 30-50%
Haemophilus influenza
Respiratorysyncytial virus
Staphylococcusaureus
Diagnostics
Complaints:
fever
cough,
dyspnea,
sputum
chest pain
unmotivated weakness;
fatigue, malaise;
heavy sweating at night.
Physical examination:
Classic objective signs:
increased voice trembling;
shortening (dullness) of percussion sound over the affected area of the lung;
locally listened to bronchial or weakened vesicular breath;
sonorous, small bubbling rales or crackling.
Laboratory researchmethods
general blood test (leukocytosis or leukopenia, neutrophilic shift, acceleration of ESR);
biochemical blood test (there may be an increase in the level of urea and creatinine);
C-reactive protein (CRP) quantification;
general sputum analysis (increase in leukocytes mainly due to neutrophils and lymphocytes);
sputum testing for flora and sensitivity to antibiotics (according to indications);
sputum examination for Koch Bacilli (according to indications);

coagulogram;
determination of the gas composition of arterial blood (with SpO2 <92%);
Instrumental research methods
Radiography of the chest cavity in the anterior direct and lateral projections - the main sign of pneumonia is shading, infiltration (focal, segmental, lobar and more) of lung tissue. The diagnosis is certain in the presence of radiologically confirmed focal lung tissue infiltration.
Computed tomography of the thoracic segment is performed in the presence of clinical signs of pneumonia and an X-ray of the negative picture, severe pneumonia, the absence of the effect of starting antibiotic therapy, and the deterioration of the patient's condition;
ECG;
Ultrasound of the heart in severe pneumonia for differential diagnosis with congestion in the pulmonary circulation and thromboembolism.
Indications for consultation of specialists
TB consultation - to rule out pulmonary tuberculosis;
Oncologist consultation - if a neoplasm is suspected;
Cardiologist consultation - to exclude cardiovascular pathology;
Consultation of a **thoracic surgeon** - in the presence of exudative pleurisy and other complications (abscesses, destructive processes, etc.);
consultation of the **endocrinologist** - in the presence of endocrinological diseases.
Tactics of treatment
Non-drug treatment
to reduce intoxication syndrome and facilitate the release of sputum - maintaining an adequate water balance (adequate fluid intake);
smoking cessation;
elimination of the effect on the patient of environmental factors causing a cough (smoke, dust, pungent odors, cold air).
Drug treatment
The main drugs for the treatment of community-acquired pneumonia are **antibacterial drugs**.**Empirical antibiotic therapy** is usually performed.

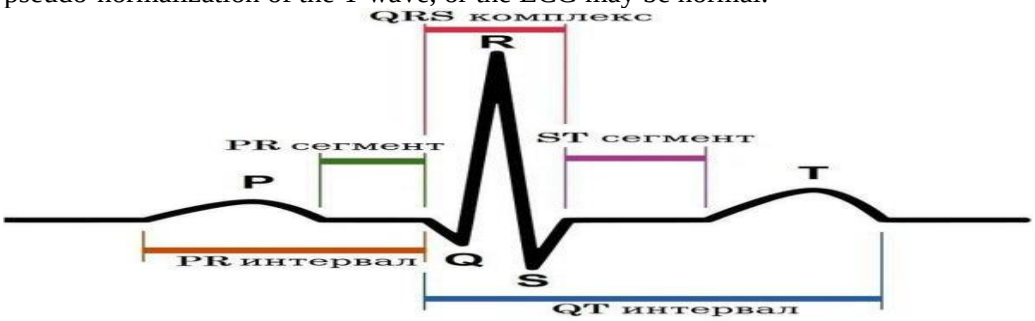
Patients	**First row drugs**	**Second row drugs / alternative**
Group 1 patients: Patients without concomitant diseases who did not take the last 3 months antibiotics, or age less than 60 years	Amoxicillin orally 0.5 g 3 times a day or 1.0 g 2 times a day	Macrolides inside: Spiramycin 3 million units 2 times a day Azithromycin 250-500 mg 1 time per day Clarithromycin 500 mg 2 times a day Josamycin 500 mg 3 times a day
Group 2 patients: Patients at high risk of infection with antibiotic-resistant strains, with concomitant diseases or age ≥60 years	Amoxicillin orally 1.0 g 3 times a day or Amoxicillin / clavulanate orally 0.625 g 3 times a day or 1.0 g 2 times a day or (Amoxicillin / sulbactam orally 0.5 g 3 times a day or 1.0 g 2 times a day) Macrolides orally : Spiramycin 3 million 2 times a day	Cephalosporins of III generation (orally, i/m *): Cefixime orally 400 mg 1 time per day Ceftriaxone 1.0 1 time per day IM or Respiratory fluoroquinolones (orally) Levofloxacin 500-750 mg 1 time per day

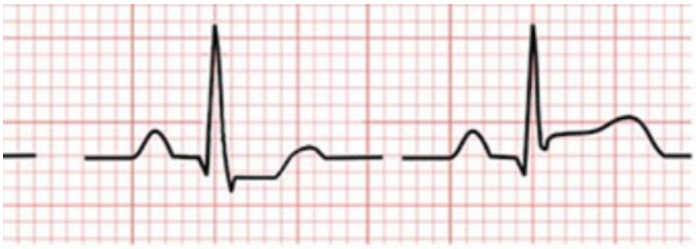
	Azithromycin 250-500 mg 1 time per day Clarithromycin 500 mg 2 times a day Josamycin 500 mg 3 times a day	

Criteria for the effectiveness of antibiotic therapy:
body temperature <37.5ºC;
lack of intoxication;
absence of respiratory failure (respiratory rate <20 per min);
absence of purulent sputum;
the number of leukocytes <10x109 /l, neutrophils <80%, “young” forms <6%;
the absence of negative dynamics on the chest x-ray (when it is performed on time).
Further management of the patient
The doctor must re-examine the patient:
no later than the 3rd day of treatment;
and after the end of the course of antibacterial therapy.
Repeated x-ray examination is carried out on the 7-10th day of treatment. It is necessary to explain to patients about the features of the state change during treatment, in most patients:
By the end of the 1st week: fever should resolve;
By the end of the 4th week: chest pain and sputum separation are significantly reduced;
By the end of the 6th week: cough and shortness of breath are significantly reduced;
after 3 months: slight fatigue may persist;
after 3-6 months: the condition is completely normal.
IV. Visual material: are in the appendix
V. Bibliography: at the end
VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Preventio

I. Theme № 3: Acute coronary syndrome in GMP.
II. Learning goals: Diagnosis and treatment of Acute coronary syndrome on ambulatory level.
III. Lecture thesis:
Acute coronary syndrome
Acute coronary syndrome (ACS) is a clinical condition reflecting the period of exacerbation of Ischemic heart disease (IHD), the leading symptom of which is **chest pain**
Acute coronary syndrome (ACS) is a clinical condition reflecting the period of exacerbation of Ischemic heart disease (IHD), the leading symptom of which is **chest pain**
Classification
1. Patients with acute persistent chest pain and stable **STsegment elevation** on ECG (> 20 min) belong to the ACS group with ST-segment elevation, which reflects the presence of acute total occlusion of the coronary artery. The mainstay of treatment for such patients is immediate pharmacological reperfusion or primary angioplasty / stenting (ST-segment elevation myocardial infarction is considered in a separate protocol).
2. Patients with acute chest pain but **no ST-segment elevation** on the ECG belong to the ACS group without ST-segmentelevation, which reflects the presence of transient partial coronary artery occlusion or

distal embolization by fragments of a thrombus or damaged plaque. ECG changes may be transient ST-segment elevation (<20 min), persistent or transient ST-segment depression, inversion, leveling, or pseudo-normalization of the T wave, or the ECG may be normal.





Clinical forms
Unstable angina - acute myocardial ischemia, the severity and duration of which is insufficient for the development of myocardial necrosis (there are no diagnostically significant increases in troponin levels).

Myocardial infarction without ST segment elevation - acute myocardial ischemia, the severity and duration of which leads to myocardial necrosis.

Classification of unstable angina
Newly-onset angina pectoris (newly-onset angina pectoris II-III functional class according to the classification of the Canadian Cardiovascular Society, lasting no more than 2 months)
Progressive (progression of previously stable angina pectoris, at least up to functional class III, which occurred in the last 2 months)
Early postinfarction angina pectoris (up to 2 weeks from the development of MI)
Prinzmetal's vasospastic angina.

Classification of types of myocardial infarction
By the depth of the lesion: myocardial infarction with ST-elevation; myocardial infarction without ST-elevation.
By localization: anterio-septal, anterio-apical, anteiro-lateral, anterio-widespread, inferior, inferior-lateral, posterior-basal, high anterior and their combinations.
By stage: acute, subacute, recovery.
Classification of acute heart failure by Killip

Class	**Feature**
I	No heart failure.
II	Moist wheezing <50% of pulmonary fields, III tone, pulmonary hypertension.
III	Moist wheezing> 50% of pulmonary fields. Pulmonary edema

IV	Cardiogenic shock

DIAGNOSTICS AND TREATMENT AT THE AMBULATORY LEVEL

The main goal is diagnosis and immediate transportation to a specialized hospital.

Diagnostic criteria

Complaints and anamnesis:

Prolonged (> 20 min) anginal chest pain at rest: typical heart pain is characterized by discomfort or heaviness behind the breastbone (angina pectoris) radiating to the left arm, neck or jaw, which may be transient (usually lasting several minutes) or longer. The pain may be accompanied by sweating, nausea, abdominal pain, shortness of breath, and fainting. Atypical manifestations such as epigastric pain, dyspepsia, or isolated dyspnea are common. Atypical symptoms are more common in elderly patients, in women with diabetes mellitus, chronic renal failure or dementia.

Newly onset angina pectoris (II or III FC) with a history of the disease for 1-2 months with a tendency to progression of clinical symptoms. Attacks can occur during physical exertion and remain relatively stereotyped at first, in other cases, angina attacks rapidly increase in frequency and intensity, up to spontaneous attacks lasting from 5 to 15 minutes or more.

Progressive angina pectoris, at least up to FC III: an increase in the severity of angina attacks with a progressive decrease in exercise tolerance, expansion of the pain zone and their irradiation, prolongation of the duration of attacks, decrease in the effectiveness of
nitroglycerin, the appearance of new concomitant symptoms (shortness of breath, interruptions in the heart , weakness, fear, etc.).

Early postinfarction angina pectoris developed within 2 weeks after myocardial infarction.

Diagnostic criteria

Physical examination: not very informative.

Laboratory tests: (including determination of troponin level) at the outpatient level is possible, but not advisable.

Laboratory tests: (including determination of troponin level) at the outpatient stage is possible, but not advisable.

Instrumental studies: 12-lead resting ECG is the first diagnostic method

Treatment tactics

The tactics of treatment at the prehospital stage is aimed at timely diagnosis of ACS, provision of emergency care, prevention of complications, transportation to a specialized clinic.

Treatment

Primary therapeutic measures:

Oxygen therapy with oxygen saturation less than 90% or severe shortness of breath (I A).

β-blockers. Early use of β-blockers is recommended in patients with ischemic symptoms unless contraindicated. β-blockers are prescribed as early as possible for symptoms of ischemia in patients without contraindications. β-blockers reduce myocardial oxygen consumption by reducing heart rate, blood pressure and myocardial contractility (I B).

Nitrates are used only in the presence of pain and SBP> 90 mm Hg. Intravenous nitrate administration is more effective than sublingual administration in reducing symptoms of anginal pain and regression of ST segment depression. In the absence of nitrates for intravenous administration, nitroglycerin forms are used in tablets of 0.5 mg or in an aerosol of 0.4 mg (1 dose), with repeated use after 3-5 minutes if ineffective and in the absence of contraindications (SBP <90 mm Hg)

Narcotic analgesics. With intense long-term pain in the chest, morphine (IV or SC) may be used (IA).

Blockers of calcium channels. In patients with suspected / confirmed vasospastic angina, calcium channel blockers and nitrates should be given and βblockers should be avoided (IIa B).

Acetylsalicylic acid. During the initial examination of a patient with suspected ACS, ASA is prescribed in a loading dose of 150–300 mg, not with an "enteric" coating (IA).

Inhibitors of platelet P2Y12 receptors. Appointment of a second antiplatelet agent in addition to ASA with a high probability of ACS:

Ticagrelor 180 mg loading dose is recommended in the absence of contraindications (history of intracranial hemorrhage or ongoing bleeding) in all moderate to high-risk NSTE-ACS patients (with elevated troponin levels) (IA)or**Clopidogrel** at a loading dose of 300 mg (if a conservative strategy is expected) or 600 mg (if an invasive strategy is intended) is recommended for patients who cannot take ticagrelor or need additional prescription of indirect anticoagulants (atrial fibrillation) (IB).
• **Anticoagulant therapy** is prescribed for all patients upon diagnosis of ACS. As anticoagulants in the acute phase of NSTE-ACS, one of the following drugs can be used:
fondaparinux;
low molecular weight heparin (enoxaparin);
UFH (UNFRACTED HEPARIN)
Indications for consultation with specialists: not provided, except for possibly emergency - a cardiologist.
Preventive measures - screening for early detection of dyslipidemia, hypertension, diabetes mellitus and coronary artery disease with an assessment of the pre-test probability and carrying out stress testing, if necessary, with imaging (stress ECG, stress echocardiography, MRI, etc.) in identifying a high ischemic risk, timely referral to specialized centers for diagnostic CAG (see also the protocols for diagnosis and treatment of stable exertional angina, arterial hypertension, diabetes mellitus).
Patient monitoring: (patient observation card, individual patient observation card, individual action plan) - not provided.
Treatment effectiveness indicators:
1. Compliance with time intervals in the diagnosis of ACS (registration and interpretation of ECG for <10 min).
2. Conducting primary therapeutic measures (point 4).
3. Immediate transportation to a specialized hospital <30 minutes
4. Raising awareness of the population about the signs of an acute heart attack and the need for treatment "103".
INDICATIONS FOR HOSPITALIZATION: ACS without ST-segment elevationdiagnostics is an indication for emergency hospitalization in a specialized hospital, regardless of the presence or absence of ECG changes.
Preventive measures
- screening for the early detection of dyslipidemia, hypertension, diabetes mellitus and ischemic heart disease with an assessment of the pretest probability and carrying out stress testing, if necessary with visualization (stress ECG, stress echocardiography, MRI, etc.) when a high ischemic risk is detected, timely referral to specialized centers for diagnostic CAG.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention.

I. Theme № 4: Arterial hypertension in GMP.

II. Learning goals: Diagnosis and treatment of arterial hypertension on ambulatory level.

III. Lecture thesis:

Arterial hypertension is a chronic stable increase in blood pressure, in which the level of systolic blood pressure equal to or more than 140 mmHg and / or the level of diastolic blood pressure equal to or more than 90 mmHg in people who do not receive antihypertensive drugs.

Classification of blood pressure levels

Categories of BP	Systolic blood pressure		Diastolic blood pressure
Optimal	< 120	and	<80
Normal	120 - 129	and/or	80-84
High normal	130 - 139	and/or	85-89
• 1st degree hypertension	140 - 159	and/or	90-99
• 2 degree hypertension	160 - 179	and/or	100-109
• 3 degree hypertension	≥ 180	and/or	≥110
Isolated systolic hypertension *	≥ 140	and	<90

Risk stratification criteria (factors influencing the prognosis)

Risk factors

■ Importance of systolic blood pressure and diastolic blood pressure
■ The level of pulse blood pressure (in the elderly).
■ Age (men> 55 years old, women> 65 years old)
■ smoking
■ Dyslipidemia: cholesterol> 5.0 mmol / L (> 190 mg / dl), or low density lipoproteins> 3.0 mmol / L (> 115 mg / dl), or high density lipoproteins in men <1.0 mmol / l (40 mg / dl), in women <1.2 mmol / l (4 mg / dl), or triglycerides> 1.7 mmol / l (> 150 mg / dl)
■ Plasma glycemia 5.6-6.9 mmol / L (102-125 mg / dL)
■ Impaired glucose tolerance
■ Abdominal obesity: waist circumference in men ≥102 cm, in women ≥88 cm
■ Family history of early cardiovascular disease (in women under 65 years old, in men under 55 years old).
A combination of 3 of the following 5 criteria indicates the presence of a metabolic syndrome: abdominal obesity, fasting glycemia, blood pressure> 130/85 mm Hg, low levels of high density lipoproteins, high triglycerides.

Asymptomatic target organ damage

■ ECG signs of LVH (Sokolov-Lyon index> 3 8 mm, Cornell index> 2440 mm x ms) or:
■ Echo cardiographic signs of LVH * (LV myocardial mass index> 125 g / m2 in men and> 110 g / m2 in women)
■ Thickening of the wall of the carotid artery (intima-media complex> 0.9 mm) or the presence of an atherosclerotic plaque
■ A slight increase in serum creatinine: up to 115-133 μmol / L in men 107-124 μmol / L in women
■ Low creatinine clearance ** (<60 ml / min)
■ Microalbuminuria 30-300 mg / day or albumin / creatinine ratio> 22 mg / g in men or women> 31 mg / g

Diabetes

■ Plasma glucose on an empty stomach> 7.0 mmol / L (126 mg / dl) with repeated measurements
■ Plasma glucose after test with glucose> 11.0 mmol / L (198 mg / dl).

Associated Clinical Conditions

■ Cerebrovascular disease: ischemic stroke, cerebral hemorrhage, transient ischemic attack;
■ Heart diseases: myocardial infarction, angina pectoris, revascularization, heart failure;
■ Kidney damage: diabetic nephropathy, impaired renal function (serum creatinine in men> 133 μmol (> 1.5 mg / dl), in women> 124 μmol / l (> 1.4 mg / dl); proteinuria> 300 mg / day
■ Peripheral artery disease
■ Severe retinopathy: hemorrhages or exudates, swelling of the optic nerve papilla

Depending on the degree of increase in blood pressure, the presence of risk factors, damage to target organs and associated clinical conditions, all hypertensive patients can be assigned to one of 4 risk levels:
low,
medium
high
very high

Stratification of hypertensive patients by the risk of developing cardiovascular complications

Risk factors, damage to target organs and associated clinical conditions	BP, mmHg				
	Normal BP: SBP 20-129 or DBP 80-84	High normal BP: SBP 130-139 or DBP 85-89	1st degree hypertension SBP 140-159 DBP 90-99	2 degree hypertension SBP 160-179 DBP 100-109	3 degree hypertension SBP ≥ 180 DBP ≥ 110
No other risk factors.	Medium risk	Medium risk	Low additional risk	Moderate additional risk	Very high additional risk
1-2 risk factors	Low additional risk	Low additional risk	Moderate additional risk	Moderate additional risk	Very high additional risk
≥3 risk factors, metabolic syndrome, damage to target organs or diabetes	Moderate additional risk	High additional risk	High additional risk	High additional risk	Very high additional risk
Identified cardiovascular or kidney disease	Very high additional risk	Very high additional risk	Very high additional risk	Very high additional risk	Very high additional risk

The prognosis of hypertension patients and the choice of treatment tactics depend on the level of blood pressure and the presence of concomitant risk factors, involvement of target organs in the pathological process, and the presence of associated diseases.

Risk groups (of patients)

• **Low risk (risk 1)** - AH of the 1st degree, there are no risk factors, damage to target organs and associated diseases. The risk of developing cardiovascular diseases and complications in the next 10 years is 15%.
• **Medium risk (risk 2)** - AH of the 2nd to 3rd degree, there are no risk factors, damage to target organs and associated diseases.
Or 1-3 degree of hypertension, there are 1 or more risk factors, there is no damage to target organs (POM) and associated diseases. The risk of developing cardiovascular complications in the next 10 years is 15-20%.
• **High risk (risk 3)** - AH 1-3 degrees, there is damage to target organs and other risk factors, there are no associated diseases. The risk of developing cardiovascular complications in the next 10 years is more than 20%.
• **Very high risk (risk 4)** - hypertension 1-3 degrees, there are risk factors, damage to target organs, associated diseases. The risk of developing cardiovascular complications in the next 10 years exceeds 30%.

Complaints:
headache, dizziness, noise in ears, palpitations, chest pain, shortness of breath, swelling
Anamnesis:
1. The duration of the existence of hypertension, the level of increase in blood pressure, the presence of HA;
2. Diagnosis of secondary forms of hypertension:
■ family history of renal disease (polycystic kidney disease);
■ a history of renal diseases, bladder infections, hematuria, abuse of analgesics (parenchymal kidney disease);
■ use of various drugs or substances: oral contraceptives, nasal drops, steroidal and non-steroidal anti-inflammatory drugs, cocaine, erythropoietin, cyclosporins;
■ episodes of paroxysmal sweating, headaches, anxiety, palpitations

Physical examination:
Physical examination of a patient with hypertension is aimed at blood pressure measurement, determining RF, signs of the secondary nature of hypertension, and organ lesions. Height and weight are measured with the calculation of body mass index (BMI) in kg / m2, and waist circumference (OT).

Laboratory research:
Mandatory studies that should be carried out prior to treatment in order to detect damage to target organs and risk factors:
- general analysis of blood and urine;
- biochemical analysis of blood (potassium, sodium, glucose, creatinine, uric acid, lipid spectrum).

Instrumental research:
- ECG in 12 leads
- Echocardiography to assess left ventricular hypertrophy, systolic and diastolic functions
- chest x-ray
fundus examination
- ultrasound examination of arteries
- Kidney ultrasound
- daily monitoring of blood pressure.

Indications for consultation:
Neurologist:
1. acute cerebrovascular accident
- stroke (ischemic, hemorrhagic)

transient cerebrovascular accident
2. Chronic forms of vascular pathology of the brain
- The initial manifestations of insufficient blood supply to the brain
- encephalopathy
Oculist:
- hypertensive angioretinopathy
- retinal hemorrhages
- swelling of the optic nerve
- retinal disinsertion
- progressive loss of vision
Nephrologist:
- exclusion of symptomatic hypertension

Tactics of treatment:

Non-drug treatment

• reduced consumption of alcoholic beverages <30 g of alcohol per day for men and 20 g / day. for women;
• increase in physical activity - regular aerobic (dynamic) physical activity for 30-40 minutes at least 4 times a week;
• reduced consumption of salt to 5 g / day;
• a change in diet with an increase in the consumption of plant foods, an increase in the diet of potassium, calcium (found in vegetables, fruits, grains) and magnesium (found in dairy products), as well as a decrease in the consumption of animal fats;
• to give up smoking;
• normalization of body weight (BMI <25 kg / m2);

Drug treatment

List of essential drugs
ACE inhibitors
Enalapril 5 mg, 10 mg, 20 mg
Perindopril 5 mg, 10 mg
Ramipril 2.5 mg, 5 mg, 10 mg
Lisinopril 10 mg, 20 mg
Fosinopril 10 mg, 20 mg
Zofenopril 7.5 mg, 30 mg

Angiotensin Receptor Blockers
Valsartan 80 mg, 160 mg
Losartan 50 5mg. 100 mg
Candesartan 8 mg, 16 mg

Calcium antagonists
Amlodipine 2.5 mg 5 mg, 10 mg

Lercanidipine 10 mg
Nifedipine 10 mg, 20 mg, 40 mg

Beta blockers
Metoprolol 50 mg, 100 mg
Bisoprolol 2.5 mg, 5 mg., 10 mg
Carvedilol 6.5 mg, 12.5 mg, 25 mg
Nebivolol 5 mg

Diuretics
Hydrochlorothiazide 25 mg
Indapamide 1.5 mg, 2.5 mg
Torasemide 2.5 mg, 5 mg
Furosemide 40 mg
Spironolactone 25 mg, 50 mg

Alpha blockers
Urapidil 30 mg, 60 mg, 90 mg

Antiplatelet agents
Acetylsalicylic acid 75 mg, 100 mg.

Statins
Atorvastatin 10 mg, 20 mg
Simvastatin 10 mg, 20 mg, 40 mg
Rosuvastatin 10 mg, 20 mg, 40 mg

Further management:
Objectives of dynamic observation: control and correction of risk factors, achievement of target levels of blood pressure, LDL cholesterol, control of glycemic profile indicators, dynamic assessment of the state of target organs.
According to the "Rules for the provision of primary health care and the Rules for attachment to primary health care organizations" (order No. 281 dated April 28, 2015), the frequency of examinations of patients with hypertension is:
1) examination of the SMR - once every 3 months - for patients with medium and low risk, once a month - for patients with high and very high risk, and for those with low adherence to treatment:
2) examination by a PHC doctor - once every 6 months - for patients with medium and low risk, once every 3 months - for patients with high and very high risk, and for people with low adherence to treatment
3) examination by a cardiologist - once a year

Mandatory minimum of diagnostic tests:
1) general urine analysis (quantitative determination of urine protein, and / or the ratio of albumin / creatinine), determination of LDL cholesterol, glycated hemoglobin, ECG - once a year.
2) ABPM and echocardiography - once every 2 years.

Treatment effectiveness indicators:
• Achievement of target blood pressure levels depending on age and associated clinical conditions (table 21).
• Achievement of target LDL levels:

<1.8 mmol / L (70 mg / dL) or a decrease of ≥50% from baseline 1.8-3.5 mmol / L (70-135 mg / dL) in patients with very high CVC risk (IB),
<2.6 mmol / L (100 mg / dL) or a decrease of ≥50% from baseline 2.6-5.2 mmol / L (100-200 mg / dL) in high-risk CVD patients (IB),
<3.0 mmol / L (115 mg / dL) in patients with low / moderate CVC risk (IIa C)
• Disappearance or decrease in the degree of albuminuria, normalization of the albumin / creatinine ratio.
• Elimination of hyperuricemia in hypertensive patients with high and very high cardiovascular risk before reaching the target values (in high-risk patients with CVC <360 mmol / L, in patients with very high risk of CVC <300 mmol / L).
• Reduction of LVH.
• Reducing the frequency of ambulance calls and emergency hospitalizations.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 5: Acute rheumatic fever and chronic rheumatic heart disease in GMP.
II. Learning goals: Diagnosis and treatment of acute rheumatic fever and chronic rheumatic heart disease on ambulatory level.
III. Lecture thesis:
Acute rheumatic fever (ARF) -systemic inflammatory disease of the connective tissuewith predominant localization in the:
- cardiovascular system (rheumatic heart disease),
- joints (migratory polyarthritis),
- brain (chorea)
- skin (annularerythema, rheumatic nodules)
which develops in predisposed individuals (mainly young people, from 7-15 years old) due to the body's autoimmuneresponse to streptococcus antigen of beta-hemolytic streptococcus of group A.
Acute rheumatic fever (ARF) - post-infectious complication of A-streptococcal tonsillitis or pharyngitis
Chronic rheumatic heart disease (CRHD) is a disease characterized by damage to the heart valves in the form of post-inflammatory marginal fibrosis of the valve leaflets or heart disease (insufficiency and / or stenosis), formed after an acute rheumatic fever.
Classification:
Clinical forms:
Acute rheumatic fever;
Recurrent rheumatic fever.
Clinical manifestations:
Main: carditis, arthritis, chorea, erythema annulus, rheumatic nodules;
Additional: fever, arthralgia, abdominal syndrome, serositis.
Outcomes:

A. Convalescence;
B. Chronic rheumatic heart disease:
 without a heart defect;
 with heart defect.

Circulatory failure:
 According to the classification of Strazhesko –Vasilenko: (stages 0, I, IIA, IIB, III);
 According to the classification of the New York Heart Association – NYHA (functional classes 0, I, II, III, IV).

Special cases:
Isolated ("pure") chorea in the absence of other causes;
"Late" carditis - extended in time (> 2 months) development of clinical and instrumental symptoms of valvulitis (in the absence of other causes);
Recurrent ARF with chronic rheumatic heart disease (or withouther).

DIAGNOSTICS AND TREATMENT AT THE AMBULATORY LEVEL:

Diagnostic criteria:
are applied to diagnose ARF Kissel-Jones, revised by the American Heart Association.

Big criteria:
 Carditis;
 Polyarthritis;
Chorea;
 Ring-shaped erythema;
Subcutaneous rheumatic nodules.

Small criteria:
 Clinical: arthralgia, fever;
 Laboratory: increased ESR, increased concentration of CRP;
 Prolongation of the P-R interval on the ECG, signs of mitral and / or aortic echocardiographic regurgitation;

Data confirming the previous GABHS infection:
1. Positive GABHS culture isolated from the pharynx, or positive test quick determination of group BGSA-Ag.
2. Elevated or increasing titers of anti-streptococcal Ab.
The presence of two large criteria or one large and two small criteria in combined with data confirming the previous GABHS infection, indicates a high likelihood of ARF.

Complaints:
increase in body temperature more often to subfebrile numbers;
migrating pains of a symmetrical nature in large joints (more often total knee);
pericardial pain;
Shortness of breath during normal physical activity;
rapid heartbeat;
fatigue, general weakness;
signs of chorea (hyperkinesis - multiple violent movements muscles of the face, trunk and limbs, emotional lability, change behavior).

Anamnesis:
The onset of ARF begins on average 2-4 weeks after an episode of acute streptococcal nasopharyngeal infection. The temperature suddenly rises to febrile, symmetrical migrating pains appear in large joints and signs of carditis (pericardial pain, shortness of breath, palpitations and etc.).
More often in children, there is a monosyndromic course with a predominance signs of arthritis or carditis, or - rarely - chorea. Equally sharp - by type "Outbreaks" of ARF develops in middle-aged schoolchildren and soldiers recruits who have had an epidemic GABHS-sore throat.
For teens and young people are characterized by a gradual beginning - after the clinical manifestations of angina appear subfebrile temperature, arthralgia in large joints or only mild signs of carditis.

A repeated attack (relapse) of ARF is provoked by GABHS infection and manifests itself mainly by the development of carditis.

Physical examination:

Temperature reaction varies from subfebrile to febrile.

Skin syndrome:

Ring-shaped erythema (pale pink ring-shaped rash on trunk and proximal extremities, but not on the face; not accompanied by itching, not rising above the surface of the skin, not leaving traces behind) - characteristic, but rare (4-17% of all cases ARF) sign.

 Subcutaneous rheumatic nodules (small nodules located in places attachment of tendons in the knee, elbow or occipital area bones) - a characteristic, but extremely rare (1-3% of all cases of ARF) sign.

Joint damage:

the predominant form of damage - oligoarthritis, less often - monoarthritis.

The pathological process involves knee, ankle, wrist, elbow joints. Characterized by: good quality, volatility of inflammatory lesions with variable, often symmetrical involvement of the joints. At 10- 15% of cases revealed polyarthralgia, not accompanied by limitation movements, pain on palpation and other symptoms of inflammation.

The articular syndrome is quickly resolved against the background of NSAIDs, the deformity is not develops.

Heart damage:

A **systolic murmur** reflecting mitral regurgitation has the following characteristics: long-lasting in nature, blowing; has different intensity, especially in the early stages of the disease; not substantially changes with a change in body position and breathing phase; associated with I tone and takes up most of the systole, and is heard at the apex heart and is carried out into the left axillary region.

Mesodiastolic murmur (low frequency) that develops in acute carditis with mitral regurgitation, has the following characteristics: often follows the III tone or drowns it out, is heard at the apex of the heart in position of the patient on the left side while holding the breath while exhaling.

Protodiastolic murmur, reflecting aortic regurgitation, has the following characteristics: begins immediately after the II tone, has high-frequency blowing decaying character, best listened to along the left edge of the sternum after a deep exhalation when the patient bends forward, usually combined with systolic murmur.

The outcome of carditis is the formation of rheumatic heart disease. Rheumatic heart disease after the first attack of ARF in children develops in 20 - 25% of cases. Dominated isolated rheumatic heart defects, more often mitral insufficiency. Failure is less common aortic valve, mitral stenosis and mitral-aortic disease. Approximately 7-10% of children after suffering rheumatic heart disease mitral valve prolapse develops.

In adolescents who have had the first attack of ARF, heart defects are diagnosed in 1/3 of cases. In adult patients, this indicator accounts for 39-45% of cases. Maximum frequency of formation of rheumatic heart defects (75%) observed within 3 years from the onset of the disease. ARF repeated attacks aggravate the severity of valvular heart disease.

Damage to the nervous system:

in 6-30% of cases, signs of a small chorea (hyperkinesis, muscle hypotonia, stato-coordination disorders, vascular dystonia, psychoemotional disorders). In 5-7% of patients with chorea acts as the only sign of ARF. Chorea are more susceptible to girls 10-15 years. Chorea duration is 3-6 months. Chorea usually ends recovery.

Laboratory research:

General blood analysis: increased ESR, possibly leukocytosis with by shifting the leukoformula to the left;

Biochemical blood analysis: (ALT, AST, total protein and fractions, glucose, creatinine, urea, cholesterol);

• **immunological blood analysis:** C reactive protein (CRP) (positive), Rheumatoid factor (RF) negative, Antistreptolysin-O (ASL-O) increased or, more importantly, increased in dynamics of titles;

Bacteriological examination: throat swab for determination of B-hemolytic streptococcus group A (GABHS) - detection in a smear from the throat GABHS, can be both with active infection and with carriage.

Instrumental research:

ECG: clarification of the nature of heart rhythm and conduction disorders (with concomitant myocarditis);

Chest X-ray: for diagnostic purposes. (Signs of rheumatic pneumonitis are possible)

Echocardiography: necessary for the diagnosis of valvular heart disease and detection of pericarditis. In the absence of valvulitis, rheumatic the nature of myocarditis or pericarditis should be interpreted with a large caution.

X-ray of joints for differential diagnosis with others arthritis.

Computed tomography in special cases, to identify signs of rheumatic pneumonitis, thromboembolism in small branches of the pulmonary artery.

Diagnostic algorithm

The diagnosis of ARF is confirmed if there are two large criteria or one large and two small in combination with data confirming previous BSHA infection of the nasopharynx.

Treatment

Outpatient treatment is indicated:

- patients with chronic rheumatic heart disease to carry out anti-relapse therapy;
- with chronic, including congestive heart failure on background of chronic rheumatic heart disease
- to continue treatment after discharge from the hospital with ARF and repeated rheumatic fever.

Drug treatment:

Treatment for ARF includes **etiotropic antibiotic therapy**.

The purpose of etiotropic therapy is to influence streptococcal infection.

Pathogenetic therapy is aimed at suppressing the inflammatory process.

Glucocorticoids are used in ARF with severe carditis and / or polyserositis. Prednisone is prescribed for adults and adolescents at a dose of 20 mg / day, children - 0.7-0.8 mg / kg in 1 dose in the morning after meals before achieving a therapeutic effect (on average, within 2 weeks). Then the dose is gradually reduced (by 2.5 mg every 5-7 days) until complete cancellation. The total duration of the course is 1.5-2 months.

NSAIDs are prescribed for mild valvulitis, rheumatic arthritis without valvulitis, minimal process activity (ESR <30 mm / h), after the high activity subsided and the blood glucose was canceled, with repeated ARF on background of RPS. NSAIDs are prescribed for adults and adolescents up to 3 times a day until normalization of indicators of inflammatory activity (on average during 1.5-2 months). If necessary, the course of NSAID treatment can be extended to 3-5 months.

Symptomatic therapy consists of correcting congestive heart failure, which can develop as a result of active valvulitis or in patients with rheumatic heart defects.

List of essential medicines:

benzylpenicillin sodium salt;
cefuroxime;
azithromycin;
Benzathine benzylpenicillin;
bicillin-5;
diclofenac;
aceclofenac;
etoricoxib;
prednisolone;
methylprednisolone.

List of additional medicines:

acetylsalicylic acid;
warfarin;
enalapril;

fosinopril;
verapamil;
metoprolol;
carvedilol;
bisoprolol;
diltiazem;
verapamil;
amlodipine;
valsartan;
Digoxin;
furosemide;
Hydrochlorothiazide;
torasemide;
spirinolactone.

Indications for specialist consultation:

Consultation of a neurologist is indicated in the case of the development of neurological symptoms to clarify the nature and degree of damage to the nervous system and selection of symptomatic therapy;
Consultation with an otolaryngologist - to clarify the pathology organs, choice of treatment tactics;
Consultation with a cardiologist - in case of symptoms of acute / chronic cardiac insufficiency, complex disturbances of rhythm and conduction for correction therapy;
Consultation with a cardiac surgeon - in case of suspicion of involvement in pathological process of heart valves with the formation of defect (s) os the heart (stenosis / insufficiency), infective endocarditis with low the effectiveness of the therapy.

Preventive actions:

Primary prevention: Primary prevention for the ambulatory level timely adequate antimicrobial therapy for acute and chronic recurrent GABHS - upper respiratory tract infections (tone-illitis and pharyngitis). Antimicrobial therapy for acute GABHS –tonsillitis.

Secondary prevention:

The goal is to prevent repeated attacks and progression of the disease in persons with ARF. Secondary prophylaxis begins in the hospital immediately after the end of etiotropic antistreptococcal therapy.
Benzathine benzylpenicillin is the main drug used for secondary prevention of ARF - intramuscularly 1 time in 3 weeks adults and adolescents 2.4 million units.
The duration of secondary prophylaxis for each patient is set individually. As a rule, it should be for patients who have had ARF without carditis (arthritis, chorea) - at least 5 years after the attack or before the age of 18
for patients with healed carditis without heart defect - at least 10 years after the attack or up to 25 years of age (according to the principle "which is longer").
for patients with a formed heart defect (including operated ones) - for life.
The most effective dosage form of benzathine benzylpenicillin is **extencillin**. Or **Bicillin-1** is recommended, which is prescribed in the above doses 1 time in 7 days.

Patients with rheumatic heart disease:

According to the experts of the American Heart Association, all patients with RPD are at moderate risk of developing infective endocarditis. These patients, when performing various medical procedures accompanied by bacteremia (tooth extraction, tonsillectomy, adenotomy, biliary tract surgery or intestines, interventions on the prostate gland, etc.), prophylactic antibiotics are needed.

Patient monitoring:

- Recommended medical examination 4 times a year.
- Observation by a general practitioner, rheumatologist, cardiac surgeon.

Hospitalization indications:

ARF with and without involvement of the valvular apparatus of the heart;
repeated attacks of rheumatic heart disease;

congestive heart failure against the background of rheumatic heart disease, subacute left ventricular failure.

IV.Visual material: are in theappendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 6: Peptic ulcer disease in GMP.
II. Learning goals: Diagnosis and treatment of Peptic ulcer disease on ambulatory level.
III. Lecture thesis:
Peptic ulcer disease is a chronic recurrent disease caused by Helicobacter pylori infection and occurring with alternating periods of exacerbation and remission, the leading manifestation of which is the formation of a defect (ulcer) in the wall of the stomach and duodenum.
Helicobacter pylori infection causes progressive damage to the gastric mucosa and plays an important role in the development of gastric ulcer and duodenal ulcer.
Classification:
Of nosological independence, there are:
Peptic ulcer disease and symptomatic gastroduodenal ulcers;
Peptic ulcer disease associated and not associated with H. pylori.
I.**General characteristics of the disease:**
1. peptic ulcer;
2. duodenal ulcer;
3. peptic ulcer of unspecified etiology;
4. peptic gastrojejunal ulcer after gastric resection.
II. Clinical form:
1. acute or newly diagnosed;
2. chronic.
III. Flow:
1. latent;
2. mild or rarely recurrent;
3. moderate or recurrent (1-2 relapses during the year);
4.severe (three or more relapses within a year) or continuously relapsing; the development of complications.
IV. Phase:
1.exacerbation (relapse);
2.fading.
V. Characterization of the morphological substrate of the disease.
Types of ulcers:
1.Acute ulcer;
2. chronic ulcer.
Ulcer dimensions:
1.small (less than 0.5 cm);

2. medium (0.5-1 cm);
3. large (1.1-3 cm);
4. giant (more than 3 cm).

Stages of ulcer development:

1. active;
2. scarring;
3. stage of the red scar;
4. stage of the white scar;
5.Long-term scarring.

Ulcer localization:

1.stomach (cardia, subcardial section, stomach body, antrum section, pyloric canal, anterior wall, posterior wall, lesser curvature, greater curvature);
2.duodenum (bulb, postbulbar part, anterior wall, posterior wall, lesser curvature, greater curvature).

VI. Characteristics of the functions of the gastroduodenal system (only pronounced violations of the secretory, motor and evacuation functions are indicated).

VII. Complications:

1.bleeding (mild, moderate, severe, extremely severe);
2.perforation;
3. penetration;
4. stenosis (compensated, subcompensated, decompensated);
5. malignancy.

Diagnostic criteria:

Complaints.

The leading symptom of exacerbation of ulcer is pain in the epigastric region, which can radiate to the left half of the chest and left scapula, thoracic or lumbar spine.
Pain is noted in 92-96% of patients. In intensity, they depend on the depth of the ulcer.

Characteristics of pain syndrome in ulcer.

Localization of ulcers	**Time of onset of pain**
Ulcers of the cardiac and subcardial parts of the stomach	Immediately after eating
Stomachulcers	0.5-1 hours after eating
Ulcers of the pyloric canal and duodenal bulb	Late pains (2-3 hours after eating), "hungry" pains that occur on an empty stomach and pass after eating, as well as night pains

The pains are dull, cutting, burning in nature.
Pain in ulcer is characterized by periodicity, seasonality and rhythm.
The frequency of pain is manifested by the change of painless intervals by periods of pain.
Pain occurs at different times depending on the meal:
- early,
- late,
- nocturnal
- hungry pains.

Early pains appear within the first hour after eating and are usually characteristic of ulcers. Late, nocturnal and hungry pains occur 1.5-4 hours after eating. These pains usually appear in patients with duodenal ulcer and are caused by motor disorders, hypersecretion of gastric juice and inflammatory changes in the duodenal mucosa.
With exacerbation of ulcer, heartburn, sour belching, nausea, and constipation are also common. Heartburn is observed in 50% of patients, which occurs as a result of a violation of the secretory and motor activity of the stomach.

Belching in ulcer is sour, empty, or food. Sour eructation occurs with hypersecretion of gastric juice. Nausea in ulcer is rare and may precede vomiting.
Vomiting usually occurs at the height of ulcer pain and can be early and late, it is caused by reflex irritation of the inflamed cutting fluid with gastric juice. Often, after vomiting, the intensity of pain decreases or they stop altogether, which improves the patient's condition.

Anamnesis:

In uncomplicated cases, ulcer usually proceeds with alternating periods of exacerbation (lasting from 3-4 to 6-8 weeks) and remission (lasting from several weeks to many years). Under the influence of unfavorable factors (physical stress, taking NSAIDs and / or drugs, alcohol abuse, etc.), complications may develop.
Typical for ulcer are seasonal (spring and autumn) periods of increased pain and dyspeptic symptoms. With an exacerbation, weight loss is often noted, because, despite the preserved, and sometimes even increased appetite, patients limit themselves to food, fearing increased pain.
In the exacerbation phase, regardless of the severity of the clinical picture of the disease, an ulcer with active inflammatory changes in the mucous membrane of the stomach and duodenal mucosa is found.
The phase of exacerbation subsiding (phase of incomplete remission) is characterized by the disappearance of clinical signs of the disease and the appearance of fresh cicatricial changes at the site of the former ulcer while maintaining signs of inflammation.
In the remission phase, there are no clinical, endoscopic, histological manifestations of exacerbation of the disease and colonization of mucous membrane by H. pylori infection.

Physical examination:

During an exacerbation of ulcer, an objective examination often reveals tenderness in the epigastrium on palpation, combined with moderate resistance of the muscles of the anterior abdominal wall. Local percussion pain in the same area (Mendel's symptom) can also be found.
However, these signs are not strictly specific for exacerbation of ulcer:
 forced position of the patient: bending over and pressing his hands to the epigastrium - the zone of greatest pain;
 pain on palpation in the epigastric or pylorobulbar region, combined with moderate resistance of the muscles of the anterior abdominal wall (70% of cases);
 a positive Mendel's symptom (local percussion pain in the epigastrium).
The data obtained during physical examination does not always reflect the presence of ulcer, since the same symptoms may be observed with other diseases.
The main diagnostic information is obtained using instrumental research methods, which are combined with biochemical, histochemical and other methods.

List of basic diagnostic measures

Laboratory research:

General blood analysis - possible anemia (with obvious or latent ulcer bleeding), leukocytosis and increased ESR in complicated forms of ulcer (with ulcer penetration, pronounced perivisceritis);
• occult blood feces analysis - a positive reaction indicates gastroduodenal bleeding.

Instrumental research:

• esophagogastroduodenoscopy - endoscopic examination confirms the presence of an ulcer defect, specifies its localization, depth, shape, size, condition of the bottom and edges of the ulcer (with biopsy and histological examination to exclude the malignant nature of ulcerative lesions in case of localization of an ulcer in the stomach and detection of H. pylori).
With the help of gastroduodenoscopy, it is possible to differentiate between acute and chronic ulcers. The first is characterized by pronounced inflammatory changes on the part of the mucous membrane surrounding the ulcer. The shape of the acute ulcer is round or oval. The bottom of the ulcer is usually pale yellow to brown. The edges of acute ulcers are sharply defined. The endoscopic picture of chronic gastric ulcer changes significantly depending on the stage of its development (exacerbation, attenuation of the process, healing), which is a criterion for assessing the quality of treatment. Gastroduodenoscopy in combination with biopsy is of great importance for the differential diagnosis of benign and malignant processes in the stomach.

 Histological examination of biopsy specimen reveals signs of an inflammatory process - neutrophilic infiltration. Histological examination is especially important in the presence of an ulcer, since an ulcerative form of gastric cancer is often observed.

Diagnosis of H. pylori infection

To determine further treatment tactics, the results of a study of the presence of an ulcer infection in a patient are extremely important. Determination of H. pylori in histological preparations taken with EFGDS;

 X-ray examination for the diagnosis of ulcer is currently not used. It is used in the following situations:
 the impossibility for some reason (for example, the presence of contraindications) to conduct an endoscopic examination;
 to assess the peristalsis of the stomach wall;
 to assess the nature of evacuation from the stomach;
 to assess the degree of pyloric stenosis (with complicated course).

X-ray examination reveals a direct signs of ulcer:

 local circular spasm of muscle fibers on the stomach wall opposite to the ulcer in the form of a “pointing finger”;
 ulcerative deformity of the stomach and duodenal bulb;
 fasting hypersecretion;
 disorders of gastroduodenal motility.

List of additional diagnostic measures:

determination of serum iron in the blood and analysis of feces for occult blood - in case of anemia;
Ultrasound of the liver, biliary tract and pancreas - with concomitant pathology of the hepatobiliary system;
biochemical blood tests (total bilirubin and its fractions, total protein, albumin, cholesterol, ALT, AST, glucose, amylase) - with concomitant pathology of the hepatobiliary system;

Indications for specialist consultation:

Consultation with a surgeon if a **complication** of peptic ulcer is suspected:
perforation,
penetration,
cicatricial-ulcerative stenosis of the pylorus,
malignant ulcer.

Ulcerative bleeding is observed in 15-20% of patients with ulcer, more often with localization of ulcers in the stomach.

Risk factors for its occurrence are:
intake of acetylsalicylic acid and NSAIDs;
H. pylori infection;
size of ulcers> 1 cm.

Ulcerative bleeding is manifested by vomiting of contents such as "coffee grounds" (hematemesis) or black tarry stools (melena). With massive bleeding and low secretion of hydrochloric acid, as well as localization of an ulcer in the cardiac part of the stomach, an admixture of unchanged blood may be noted in the vomit. Sometimes in the first place in the clinical picture of ulcerative bleeding are general complaints (weakness, loss of consciousness, decreased blood pressure, tachycardia).

Perforation of the ulcer occurs in 5-15% of patients with ulcer, more often in men.

It is predisposed to its development:
physical stress;
drinking alcohol;
overeating.

Sometimes perforation occurs suddenly, against the background of an asymptomatic ("silent") course of ulcer.

Ulcer perforation is clinically manifested by acute ("dagger") pain in the epigastric region, the development of a collaptoid state. Examination of the patient reveals a "board-like" tension in the muscles of the anterior abdominal wall and a sharp pain on palpation of the abdomen, a positive symptom of

Shchetkin-Blumberg. Later, sometimes after a period of apparent improvement, the picture of diffuse peritonitis progresses.

Ulcer penetration - the penetration of a stomach or duodenal ulcer into the surrounding tissues - the pancreas, the lesser omentum, the gallbladder and the common bile duct. With the penetration of the ulcer, persistent pains occur, which lose their previous connection with food intake, the body temperature rises, and an increase in ESR is detected in blood tests. The presence of ulcer penetration is confirmed by X-ray and endoscopic examination.

Pyloric stenosis is usually formed after scarring of ulcers located in the pyloric canal or the initial part of the duodenum. The most typical clinical symptoms of pylorus stenosis are vomiting of food eaten the day before, as well as belching with the smell of "rotten" eggs.

DIAGNOSTICS AND TREATMENT AT THE AMBULATORY LEVEL

Treatment goals:

quick elimination of symptoms of the disease, painful for the patient;
Ulcer healing;
elimination of the infectious agent H. pylori to prevent recurrence of the disease and prevention of its exacerbations;
in case of a complicated course of the disease - treatment of complications and elimination of the danger to the patient's life. Treatment of ulcer is complex and includes:
diet food;
-cessation of smoking and alcohol consumption;
- refusal to take drugs with ulcerogenic action;
- normalization of work and rest regime, sanatorium treatment.

Non-drug treatment:

Treatment is complex and includes:
- diet food,
- smoking cessation and alcohol consumption,
- rejection of ulcerogenic drugs (primarily NSAIDs).

Diet meals should be frequent, fractional, mechanically and chemically sparing.
Diet: Recommended frequent (5-6 times a day), fractional meals, corresponding to the rule "6 small meals are better than 3 large", mechanical, thermal and chemical sparing of the coolant.
The diet should be rich in proteins, since the therapeutic effect in this case occurs faster, the healing of ulcers is accelerated and the inflammatory process subsides

Drug treatment

Principles of pharmacotherapy:

the same approach to the treatment of gastric and duodenal ulcers;
obligatory basic antisecretory therapy;
selection of an antisecretory drug that maintains an intragastric pH> 3 for about 18 hours a day;
the appointment of an antisecretory drug in a strictly defined dose;
endoscopic control at 2-week intervals;
the duration of antisecretory therapy, depending on the timing of the healing of the ulcer;
eradication anti-Helicobacter pylori therapy in HP-positive patients;
obligatory control of the effectiveness of anti-Helicobacter therapy after 4-6 weeks;
repeated courses of anti-Helicobacter pylori therapy if it is ineffective;
maintenance anti-relapse therapy with an antisecretory drug in HP-negative patients;

Proton pump inhibitors are a basic therapy for ulcer. They are prescribed for the purpose of relieving pain and dyspeptic disorders, as well as to achieve scarring of the ulcer as soon as possible.

Antacids are able to maintain the level of intragastric pH> 3 for 4-6 hours during the day, which determines their insufficiently high efficiency when used as monotherapy. Nevertheless, patients with ulcer take antacids to relieve pain and dyspeptic complaints, which is largely due to the speed of their action and over-the-counter leave

Recommended eradication scheme (Maastricht-V, 2015)

First line therapy (10-14 days):	**Second line therapy (10-14 days):**

3-component scheme: PPI + amoxicillin + clarithromycin	3-component scheme: PPI + amoxicillin + fluoroquinolone
quadrotherapy without bismuth: PPI + amoxicillin + clarithromycin + nitroimidazole	quadrotherapy without bismuth: PPI + amoxicillin + clarithromycin + nitroimidazole
	quadrotherapy with bismuth: PPI + amoxicillin + clarithromycin + bismuth tripotassium dicitrate

The effectiveness of treatment is increased when an increased dose of PPI (twice the standard) is prescribed twice a day. With 14-day therapy, the increase in the frequency of eradication is more significant than with 10-day therapy.
H. pylori eradication therapy can lead to the development of antibiotic-associated diarrhea. The addition of the probiotic Saccharomyces boulardii to the standard triple therapy increases the rate of H. pylori eradication

List of essential medicines

Proton pump inhibitors

omeprazole
lansoprazole
pantoprazole
Rabeprazole
Esomeprazole

H2 histamine receptor blockers

Famotidine
Ranitidine

Antimicrobial drugs are used for H. pylori-associated ulcer

Amoxicillin
Clarithromycin
Metronidazole
Levofloxacin
Tetracycline
Bismuth tripotassium dicitrate

Errors during eradication therapy:

• lack of testing for H. pylori infection in patients with ulcer, as a result of which patients receive only antisecretory therapy;
• improper treatment of H. pylori-negative ulcer;
• use of drugs in eradication regimens that are not included in approved regimens (for example, H2 blockers);
use of drugs in insufficient doses;
the appointment of 7-day courses of eradication instead of 10-14 days leads to a decrease in the effectiveness of eradication;
• refusal (for various reasons) to carry out control of eradication in a timely manner;
before making a conclusion about the ineffectiveness of conservative treatment of ulcer in a patient, it is necessary to make sure that the treatment and examination were carried out in strict accordance with the existing protocol.

Preventive actions:

• simultaneous prescription of PPIs when it is necessary to take NSAIDs and anticoagulants (especially for elderly patients, patients with a history of ulcer disease, patients with concomitant diseases of the cardiovascular system and liver cirrhosis);
• elimination of factors contributing to ulcer formation: elimination of bad habits (smoking and alcohol abuse);
 normalization of the work and rest regime;

Organization of the correct regimen and nature of nutrition,
Conducting a urea breath test for the presence of H. pylori and the use of eradication anti-Helicobacter pylori therapy in case of H. pylori detection.
Further management:
Dynamic observation of the patient, 1 time per year to carry out FEGDS, diagnosis of H. pylori infection. In case of exacerbation - to carry out eradication therapy (see above). With a continuously recurrent course - consultation with a surgeon (especially with peptic ulcer)
Indicators of the effectiveness of treatment and the safety of methods:
healing of an ulcer in the mucous membrane;
effective eradication of H. pylori;
ensuring stable remission of ulcer;
absence of complications.
Indications for planned hospitalization: ineffectiveness of outpatient drug therapy.
Indications for emergency hospitalization:
• complicated forms of the disease;
• severe pain syndrome;
• ineffectiveness of outpatient drug therapy;
• concomitant diseases.
IV.Visual material: are in the appendix
V. Bibliography: at the end
VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention
I. Theme № 7: Hepatitis.
II. Learning goals: Diagnosis and treatment of hepatitis on ambulatory level.
III. Lecture thesis:
Acute viral hepatitis B (HBV) is a viral anthroponotic infectious disease with predominant parenteral and vertical mechanisms of pathogen transmission; characterized by cyclically occurring parenchymal hepatitis with possible chronicity.
Acute viral hepatitis D (delta hepatitis, hepatitis B with a delta agent) is a viral hepatitis with a parenteral mechanism of pathogen transmission, caused by a defective virus, the replication of which is possible only in the presence of HBsAg in the body, occurring in the form of co- and superinfection.
Acute viral hepatitis C (HCV) is an anthroponotic infectious disease with a parenteral mechanism of pathogen transmission, characterized by a mild or subclinical course of the acute period of the disease, the frequent formation of chronic hepatitis C, the possible development of liver cirrhosis and hepatocellular carcinoma.

Clinical classification:
According to the severity of clinical manifestations Clinical variants:
• icteric;
• anicteric;
• erased;
• subclinical (inapparent) variant.

According to the duration and cyclicity of the flow:
• acute (up to 3 months);

• protracted (more than 3 months);
• with relapses, exacerbations (clinical, enzymatic).

Gravity forms:
• light;
• moderate;
• heavy;
• fulminant (lightning).

Complications: acute and subacute liver dystrophy with the development of hepatic encephalopathy and hepatic coma.
Outcomes:
• recovery;
• chronic hepatitis, liver cirrhosis, hepatocellular carcinoma;
• lethal outcome in the development of acute liver dystrophy.

Forms of acute viral hepatitis:

• Innaparant - no clinic, normal ALT values, specific markers of viral hepatitis in the blood.
• Subclinical - no clinic, specific markers of viral hepatitis in the blood, in combination with changes in biochemical liver tests.
• Typical icteric cyclic - jaundice with cytolytic syndrome with a clear distinction between 3 periods of the disease, specific markers of viral hepatitis.
• Typical icteric with cholestatic component -
• jaundice is more intense, high bilirubinemia, slight transaminasemia, there is a tendency to increase alkaline phosphatase, specific markers of viral hepatitis in the blood. The icteric period is longer.
• Atypical cholestatic - rarely observed in elderly patients. Jaundice intense with severe itching of the skin. Hyperbilirubinemia, hypercholesterolemia, increased alkaline phosphatase and GGTP. The tendency to accelerate ESR and subfebrile condition in the icteric period, specific markers of viral hepatitis in the blood.
• Atypical anicteric - clinical symptoms identical to the preicteric period, complete absence of jaundice, hepatomegaly. Specific markers of viral hepatitis in combination with elevated ALT levels.
• Acute cyclic - within 1-1.5 months, the virus replication stops, it is eliminated from the body and complete sanitation occurs (for hepatitis A, hepatitis E - a typical form; for hepatitis B, C and D - one of the options).
• Acute progredient - the phase of active virus replication lasts 1.5-3 months. Completion of the infection: either sanitation or transformation into a chronic course.

Diagnostic criteria:

Complaints:
weakness
• fatigue
• weakness
• loss of appetite
• decreased taste sensations
• nausea,
• vomit
• bitterness in the mouth
• heaviness and dull pain in the right hypochondrium and in the epigastrium
• headache
• sleep disorders

• 20–30% of patients experience pain in large joints
• urticarial rash
• increase in body temperature
• less often pruritus.

Anamnesis: The cyclical course of the disease with the presence of a preicteric period in dyspeptic, asthenovegetative, arthralgic, influenza-like, mixed variants in terms of 1 to 14 days (maximum - up to 30 days), followed by the appearance of jaundice. In the icteric period, the severity of symptoms of hepatic intoxication may increase, up to the development of acute liver failure.

Physical examination:
In the icteric period - icterus of the sclera, mucous membranes of the oropharynx, skin. The intensity of jaundice increases rapidly and in most cases reaches its maximum in the next week. Arterial pressure is normal or slightly reduced. The first heart sound at the top is weakened. On palpation of the abdominal organs, there is an increase, thickening and increased sensitivity of the liver, and rarely an increase in the spleen. The color of the urine becomes dark, the stools are acholic.

The severity of the course of Acute VG:
Mild course - no intoxication or mild. Jaundice is mild. The content of total bilirubin is not higher than 80-85 μmol/l. PI (PO) within normal limits.
Moderate course - moderately expressed symptoms of hepatic intoxication. Jaundice is moderate. PI (PO) is reduced to 60%. The content of total bilirubin is in the range of 100-180 μmol/l.
Severe course - severe intoxication. PI (PO) below 55%. The content of total bilirubin exceeds 180 μmol/l, reaching 400 μmol/l, 1/8 of the total bilirubin is the free fraction. Serum albumin is reduced to 47-45% and below.
Fulminant course (malignant) with massive and submassive liver necrosis:
• **massive (acute) variant** - hepatic coma develops within 0-14 days from jaundice to encephalopathy;
• **submassive (subacute)** development of liver necrosis is preceded by a period of normal course of OH in terms of 15 days - 12 weeks before encephalopathy.

Complications: edema-swelling of brain cells, DIC syndrome, acute renal failure, acute respiratory failure, cardiovascular failure, generalized secondary infection, multiple organ failure syndrome.

Laboratory research.

GBA: leukopenia, neutropenia, relative lymphocytosis and monocytosis, normal or delayed ESR.
Biochemical analysis of blood: the content of total bilirubin is increased, due to direct (bound), the activity of aminotransferases, especially ALT, increases sharply, the prothrombin index is reduced.
Serological diagnosis:
• with HBV - detection of serological markers of acute HBV infection (HBsAg, anti-HBcore IgM) in blood serum;
• with HCV - detection of serological markers of acute HCV infection (anti-HCV) in blood serum;
• with HBD- coinfection - the presence of markers of hepatitis B and D in the blood serum (HBsAg, anti-HBc IgM in combination with anti-HDV IgM, anti-HDV IgG) in the blood serum.
Virological diagnosis of HBV, HCV, HDV infections (in order to determine virus replication) is carried out on the basis of polymerase chain reaction (qualitative test) using closed-type automated systems in real time with a lower detection limit of 6-10 IU / ml (detection of virus DNA hepatitis B, HDV RNA of hepatitis D virus, HCV RNA of hepatitis C virus).

Instrumental research.

Ultrasound can assess the structure of the liver, splenomegaly and other signs of portal hypertension, as well as the presence of space-occupying formations.
Other imaging modalities (CT or MRI with intravenous contrast enhancement) are used to verify mass lesions and thrombosis.

Treatment:

Treatment goals:
• relief of clinical manifestations;
• normalization of biochemical parameters (resolution of cytolysis, cholestasis);
• improvement of metabolism in the liver;
• ensuring daily bowel movements;
• relief of cholestasis.

Treatment tactics:
Therapy of patients with acute viral hepatitis B, C, D is complex and includes the following types:
• etiotropic;
• pathogenetic;
• symptomatic.

The basis of treatment tactics is the protective mode of the liver: regimen, diet, restriction of prescriptions for drugs.

Non-drug treatment:
• diet № 5, 5a;
• bed rest (with moderate and severe course);
• plentiful drink (1.5-2.0 l/day).

Medical treatment:
Antiviral drugs for the treatment of patients with acute HBV, HDV are not used.
With OVHS, the indication for antiviral therapy is the presence of viremia. Antiviral therapy may be delayed for 8-12 weeks from the onset of the disease (due to the possibility of spontaneous recovery).

Detoxification therapy - the volume depends on the severity of the patient. Mild severity - oral detoxification in the amount of 2-3 liters of liquid per day (weakly brewed tea with milk, honey, jam, as well as rosehip broth, freshly prepared fruit and berry juices, compotes, alkaline mineral waters).
Moderate severity - infusion-detoxification therapy.
Preventive actions:
Active immunization with recombinant HBV vaccine against HBV. Vaccinations are subject to:
• newborns to prevent perinatal transmission in the first twelve hours of life;
• contact persons in HBV foci for the prevention of sexual and domestic transmission;
• medical workers (doctors, middle and junior medical personnel) of medical organizations;
• persons studying in institutions of secondary and higher medical education;
• recipients of blood, its components and preparations, regardless of the frequency of transfusion;
• newly diagnosed HIV-infected;
• newly identified persons subject to hemodialysis and transplantation of tissues and organs;
• oncohematological patients, as well as patients receiving immunosuppressive drugs, who, due to a weak immune response a double dose of the vaccine is administered and an additional revaccination is carried out six months after the completed vaccination;
• the use of barrier contraceptives when HBsAg-positive persons have contacts with sexual partners who do not have HBV infection or post-vaccination immunity;

• vaccination of sexual partners and family members in the absence of HBV infection or post-vaccination immunity;
• use of individual hygiene products (toothbrushes, razors, scissors, washcloths, etc.);
• vaccination of patients on hemodialysis.

A vaccine against HCV has not been developed. The risk of HCV infection can be reduced by avoiding:
• drug use;
• carrying out invasive medical and non-medical manipulations;
• promiscuous unprotected sex;
• sharing personal hygiene items;
• non-compliance with safety regulations by medical workers.

Further management:
Rules for discharge from the hospital: with complete clinical recovery, the disappearance of symptoms of hepatic intoxication, jaundice, normalization of bilirubin; ALT within the normal range or exceeding the norm by no more than 2 times.

Examination of those who have had viral HBV is carried out after 1, 3, 6 months, and then, depending on the doctor's conclusion. Dispensary monitoring of patients with HCV is carried out constantly.

Treatment effectiveness indicators:
• relief of signs of intoxication;
• normalization of the size of the liver and indicators of biochemical samples of the liver;
• seroconversion of HBeAg and HBsAg in HBV;
• relief of complications (if any).
• 800-1200 ml of 5% dextrose solution intravenously;
• in patients with elevated blood glucose levels - Ringer's solution in a daily dose - 5-20 ml / kg, if necessary, up to 30-50 ml / kg;
• duration of the course - 5 days.

Severe severity - increased detoxification therapy, 10% albumin solution, blood plasma.
Metabolic therapy - inosine, intravenous solution 20 mg / ml per 200 mg.
Patients with cholestatic syndrome, regardless of the severity of the disease, are prescribed ademetionine.
Scheme for the use of ademetionine: the first two weeks intravenously by bolus at a dose of 800-1600 mg daily, followed by the transition to tablets - 2-4 tablets per day.
Prevention of constipation. Ensuring daily bowel movements: lactulose, the dose of which is selected individually (30–60 ml / day) so that the stool is daily, formed or mushy;
Anticholestatic therapy. With prolonged hyperbilirubinemia, symptoms of cholestasis, it is necessary to prescribe bile acid preparations (not in the acute period of viral hepatitis) in combination with ademetionine.

IV. Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.

Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 8: Chronic pyelonephritis in GMP.
II. Learning goals: Diagnosis and treatment of chronic pyelonephritis on ambulatory level.
III. Lecture thesis:
Chronic tubulointerstitial nephritis is a primary lesion of the renal tubules and interstitium associated with a variety of diseases or the use of certain medications and herbal infusions for several years and is characterized by the progressive development of sclerosis of the tubules and renal interstitium.
Clinical classification:
Forms: analgesic nephropathy, nephropathy with lithium therapy, nephropathy due to heavy metals and radiation.
International Classification of Chronic Kidney Disease, 2002:
- Stage I, GFR (glomerular filtration rate) - ≥ 90 ml / min;
- stage II, GFR - 89-60 ml / min;
- stage III, GFR - 59-30 ml / min;
- IV stage, GFR - 29-15 ml / min;
- Stage V, GFR - less than 15 ml / min (terminal chronic renal failure).

Diagnostic criteria:
Complaints and anamnesis: long-term use of mixed analgesics or NSAIDs (about 3000 tablets for 5 years), colicky pain in the lumbar region (with papillary necrosis), long-term treatment with lithium drugs, polyuria or decreased urine output, weakness, malaise.
Physical examination: tenderness to palpation in the area of the projection of the kidneys, there may be arterial hypertension.
List of main diagnostic measures:
1. Complete blood count
2. General urine analysis
3. Electrophoresis of urine proteins (tubular proteinuria)
4. Ultrasound of the kidneys
5. Determination of creatinine, urea, uric acid, potassium, sodium, calcium, phosphorus, parathyroid hormone.
6. Determination of phenacetin metabolites and heavy metals in urine
7. Zimnitsky's test
8. Bacterial culture of urine
9. Calculating GFR
10. KShchS
11. Excreted sodium fraction (in urine)

List of additional diagnostic measures:
1. FGDS
2. Serum ferritin
3. Methemoglobin, sulfhemoglobin
4. ECG
5. CT of the kidneys without contrast in case of suspected necrosis of the papillae of the kidneys

Laboratory tests: possible anemia, hematuria, sterile leukocyturia, tubular proteinuria, progressive decrease in renal functions (concentration and filtration functions), signs of tubular acidosis, hyperkalemia, nephrogenic diabetes insipidus (when treated with lithium preparations) and other electrolyte disturbances.
Instrumental research:
- **Ultrasound of the kidneys:** a decrease in the size of the kidneys, the absence of calculi in the presence of renal colic (papillary necrosis), cysts.
- **Kidney biopsy:** interstitial fibrosis, tubular atrophy, papillary necrosis.

Differential diagnosis

Sign	Chronic pyelonephritis	Chronic glomerulonephritis
The onset of the	From birth in the presence of congenital kidney disease	disease is gradual, more often after intercurrent diseases
Swelling	Rarely in the presence of chronic renal failure	more often чаще
Age	From birth	Children of different ages, more often boys
Blood pressure	Not typical	Depends on the degree of renal dysfunction
General symptoms	With the addition of chronic renal failure	Moderate
Lag in physical development	Not typical	Not typical
Local symptoms	dysuria, hyperthermia, lower back pain	Pain in the lower back, in the area of the projection of the kidneys, edema
Dysuria	With neurogenic bladder dysfunction	With urinary tract infection
Leukocyturia	Not typical	Not typical
Hematuria	Not typical	typical
Pasternatsky's syndrome	More often permanent	More often negative
Decreased concentration function of the kidneys	Characteristic with the addition of chronic renal failure	Expressed with the presence of edema
Ultrasound of the kidneys	Signs of pyelonephritis, uneven contours and signs of increased echogenicity, uneven contours, deformation of the PCS	Increase in size due to edema of the parenchyma
Intravenous urography	Signs of pyelonephritis, renal function is reduced to varying degrees, the presence of congenital renal pathology	Signs of impaired urodynamics of varying degrees
Cystography	Signs of an increase in the size of the bladder, irregularity of the shape	Without pathologies
Cystoscopy	Signs of cystitis	No pathology

Treatment tactics:
Drug treatment:
Antibacterial therapy (cephalosporins, aminoglycosides, uroseptics): at the beginning the choice of the drug is empirical, then according to the sensitivity of the microflora. Administration of antibiotics parenteral or parenteral + oral.
Supportive treatment is carried out after complete normalization of urine tests for 2 months or more.

Other treatments:
in the presence of anemia - erythropoietin preparations (epoetin-alpha, beta, theta, darbepoetin), iron preparations for parenteral administration (iron sugar, iron dextran); in the presence of mineral and bone disorders - phosphorus-binding agents (Sevelamera carbonate), the active form of vitamin D3 (alfacalcidol);
in the presence of complications from the gastrointestinal tract - antiulcer therapy (proton pump inhibitors, H2-histamine receptor blockers, antibiotics, aminocaproic acid);

Antibacterial therapy for IMS attachment: -Gram-negative flora: Fluoroquins, (Ciprofloxacin, Ofloxacin, Levofloxacin, Moxifloxacin);
- Reserve antibiotics: Amoxicillin-clavulanate, Cephalosporins, Imipenems.

List of essential and additional medicines:
Basic medicines:
1. Gentamicin, brulamycin, 80 mg
2. Furagin tab., Nitroxalin tab.
3. Cephalosporins 2 - 3 - 4 generations
4. Nystatin, linex
5. Thiamine, pyridoxine
6. Cyanocobalmin
7. Infusion device
8. Novocaine, lidocaine

Additional medicines
1. Aktiferrin - tab., Syrup
2. Heparin, 25000ME, vial.
3. Meroperan, cefaclor

Preventive actions:
- prevention of viral, bacterial, fungal infections
- prevention of electrolyte imbalance
- slowing down the progression of CKD

Further management:
control of filtration, concentration functions of the kidneys, urine tests, blood pressure, ultrasound of the kidneys. (Conducted at outpatient and inpatient levels).
IV. Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
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Outpatient management tactics.
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Prevention

I. Theme № 9: Iron-deficiency anemia in GMP.
II. Learning goals: Diagnosis and treatment of Iron-deficiency anemia on ambulatory level.
III. Lecture thesis:
Iron deficiency anemia - clinical and hematologicala syndrome characterized by anemia (decrease in hemoglobin level less than 130 g / l in men and 120 g / l in women) and iron deficiency (decrease in TSat- Transferrin Saturation<16% and ferritin concentration less than 30 mg /L or ng / ml).
TSat - calculated value, depending on the level of serum iron, therefore, the criterion for IDA is also a low level of serum iron.
IDA classification by severity:
I. Light (Hb content 90-120 g / l)
II. Medium (Hb content 70-89 g / l)

III. Heavy (Hb content below 70 g / l)

Etiological classification of IDA (according to Camaschella C., 2015, modified)

Cause	**Example**
Increased iron intake	Rapid growth in adolescence, menstrual periodsblood loss, pregnancy in the second and third trimesters, blood donation
Insufficient alimentary iron intake	Insufficient nutritional intake of iron due to social malnutrition, vegetarianism, etc.
Violation iron absorption	Gastrectomy, duodenal shunt, bariatric surgery, celiac disease, inflammatory diseases intestines, atrophic gastritis, helminthic invasion
Chronic blood loss	From the gastrointestinal tract: esophagitis, gastritis, ulcer stomach, duodenal ulcer, diverticulosis, tumors of the gastrointestinal tract, inflammatory bowel disease, angiodysplasia, hemorrhoids, parasitosis, occult bleeding From the genital and urinary tract: abundant and / or prolonged periods, intravascular hemolysis (including with paroxysmal nocturnal hemoglobinuria, autoimmune hemolytic anemia with cold antibodies, march hemoglobinuria, microangiopathic hemolysis, damage to erythrocytes prosthetic valves) Systemic bleeding, including hemorrhagic telangiectasia, chronic schistosomiasis,
Related medicinal drugs	Glucocorticosteroids, salicylates, non-steroidal anti-inflammatory, proton pump inhibitors
Hereditary	IRIDA (mutation in the TMPRSS6 gene) and rarer causes
Erythropoiesis, limited iron	Treatment with erythropoietins for anemia chronic diseases, chronic kidney disease

Diagnostic criteria:

General anemic syndrome: weakness, increased fatigue, dizziness, headaches (more often in the evening), shortness of breath on exertion, palpitations, syncope, flashing "flies"before the eyes with a low level of blood pressure, there is often a moderate increase in temperature, often daytime sleepiness and poor sleepat night, irritability, nervousness, conflict, tearfulness, loss of memory and attention, loss of appetite. The severity of complaints depends onadaptation to anemia. The best adaptation is facilitated by a slow rate of anemization.

Sideropenic syndrome:

changes in the skin and its derivatives (dryness, peeling, easy formationcracks, pallor). Hair is dull, brittle, "splits", turns gray early, intensefall out, nail changes: thinning, brittleness, transverse striation,sometimes spoon-shaped concavity (koilonychia).

Changes in mucous membranes (glossitis with papillary atrophy, cracks in the corners mouth, angular stomatitis).

Changes in the gastrointestinal tract (atrophic gastritis,atrophy of the esophageal mucosa, dysphagia). Difficulty swallowing dry and hardfood.

Muscular system. Myasthenia gravis (due to weakening of the sphincters appearimperative urge to urinate, inability to hold urine whenlaughter, coughing, sometimes bedwetting in girls). The consequence of myasthenia gravisthere may be miscarriage, complications during pregnancyand childbirth (decreased contractility of the myometrium).

• addiction to unusual smells.
• perversion of taste. It is expressed in the desire to eat something slightly edible.
• tendency to tachycardia, hypotension.

Laboratory research:

Laboratory tests for suspected IDA may include, in addition togeneral blood analysis with reticulocytes and indicators of iron metabolism is also a study of the levelvitamin B12, folic acid, biochemical parameters (total protein,creatinine, urea, blood glucose, total bilirubin, direct bilirubin,transaminases) and other studies depending on the characteristics of the clinicalpictures and breadth of differential diagnostic search. Planresearch can also be expanded to clarify the causes of iron deficiency andexclusion of latent blood loss and cancer search.

№	Laboratory value	Reference interval (may change depending on laboratories)	Changes in IDA
1	Morphological changes in red blood cells	normocytes - 68% microcytes - 15.2% macrocytes - 16.8%	Microcytosis is combined with anisocytosis, poikilocytosis, in the presence of anulocytes, plantocytes
2	Color index	0.86 -1.05	Hypochromia index less than 0.86
3	Hemoglobin content	Women – not less than 120 g/L Men – not less than 130 g /L	Reduced
4	MCH	27-31 pg	Less than 27 pg
5	MCHC	33-37%	Less than 33 %
6	MCV	2-10:1000	Not changed
7	Serum iron	Women - 12-25 micromole / L Men -13-30 micromole / L	Reduced
8	Total iron-binding capacity of blood serum	30-85 micromole / L	Increased
9	Latent iron binding capacity of serum	Less than 47 micromole / L	Above 47 micromole / L
10	Transferrin iron saturation coefficient (TSat)	≥16%	Decreased
11	Ferritin level *	15-150 micrograms / L	Decreased

Instrumental research:

In order to identify sources of blood loss, pathology of other organs and systems, including solid tumors:
fibrogastroduodenoscopy according to indications;
X-ray examination of the gastrointestinal tract according to indications;
X-ray examination of the chest organs according to indications;

fibrocolonoscopy according to indications;
sigmoidoscopy according to indications;
Ultrasound of the pelvic organs according to indications;
ultrasound of the abdominal organs according to indications;

Indications for specialist consultation:

• consultation with a surgeon - to exclude bleeding from the gastrointestinal tract;
• consultation with a gastroenterologist - in case of suspicion of malabsorption or chronic blood loss in pathology of the gastrointestinal tract;
• dentist consultation - dental problems leading to anemia;
 consultation with an otorhinolaryngologist - nosebleeds;
• consultation with an oncologist - a malignant lesion that causes bleeding;
• consultation of a nephrologist - exclusion of kidney diseases in the differential diagnosis of chronic diseases with anemia;
• consultation with a phthisiatrician - bleeding or secondary anemia against the background of tuberculosis;
• consultation with a pulmonologist - blood loss or secondary anemia against the background of diseases of the bronchopulmonary system;
• consultation with a gynecologist - bleeding from the genital tract (juvenile bleeding, dysmenorrhea, etc.);
• consultation of an endocrinologist - decreased thyroid function, the presence of diabetic nephropathy;
• consultation with a hematologist - to exclude diseases of the blood system and in case of ineffectiveness of ferrotherapy in patients with verified iron deficiency;
• consultation with a proctologist - rectal bleeding;
• consultation with an infectious disease specialist - if there are signs of helminthiasis or other parasitosis leading to anemia.

Treatment

The treatment program for IDA includes:

• elimination of etiological factors (treatment of the underlying disease);
medical nutrition (diet number 11);
• treatment with iron-containing drugs;
• replenishment of iron stores (satiety therapy).

Non-drug treatment:

Diet. With iron deficiency anemia, the patient is shown a diet rich in iron. Iron from animal products is absorbed in the intestines in much greater amounts than from plant foods.

Drug treatment:

IDA cannot be successfully cured if its cause is not eliminated.
Iron supplements are the basis of pathogenetic therapy for IDA. Transfusions are not a substitute for ferrotherapy.
Ferrotherapy can be performed with iron preparations for **oral** administration and **parenteral** drugs.
Of the parenteral drugs, the use of intravenous drugs is preferable, because intramuscular administration is painful, has variable absorption and can lead to the formation of infiltrates.
The end result of therapy with iron preparations, regardless of the route of administration, is the same - an increase in the level of hemoglobin. Differences between various
drugs with different routes of administration are in the tolerability of treatment and the rate of increase in the level of hemoglobin.

Iron preparations for oral administration.

The main principles of treatment of the Iron preparations for oral administration are as follows:
appointment of Iron preparations with a sufficient content of elemental iron;
inexpediency of the simultaneous administration of B vitamins (including B12), folic acid without special indications due to the lack of evidence of advantages in efficacy and safety over monocomponent drugs, inability to track pharmacokinetics the drug in the presence of 3 or more components in 1 tablet;

avoiding the appointment of iron preparations by mouth in the presence of signs of impaired absorption in the intestine;
sufficient duration of the saturating course of therapy (at least 3 months, may increase up to 5-6 months); the need for maintenance therapy of the pancreas after the normalization of hemoglobin parameters in appropriate situations.
Recommended daily intake of elemental iron for most adults is 150-200 mg. The use of higher doses does not make sense, since iron absorption does not increase. For example, a 325 mg iron sulfate tablet contains 65 elemental iron, three tablets - 195 mg of iron of which only 25 mg can be adsorbed and utilized.
Therapy with iron preparations for oral administration should be carried out within 3 months to replenish the reserves in the depot.
With oral ferrotherapy, gastrointestinal side effects are most common. These include metallic taste, nausea, diarrhea, constipation, darkening of the stool.
To reduce the severity of side effects, it is possible to reduce the dose (transfer to a single dose, for example) or increase the interval of administration, switching to another iron preparation with a lower content of elemental iron, switching from tablets to liquid dosage forms that make it easier to select the tolerated dose, discontinuation of oral medications and administration of intravenous medications.
Reasons for the ineffectiveness of Iron preparations therapy for oral administration:
• absence of iron deficiency (incorrect interpretation of the nature of hypochromic anemia and erroneous prescription of Iron preparations);
• insufficient dosage of Iron preparations (underestimation of the amount of ferric iron in the preparation);
• insufficient duration of Iron preparations treatment;
• impaired absorption of Iron preparations, administered orally to patients with the corresponding pathology;
• simultaneous administration of drugs that interfere with iron absorption;
• the presence of chronic (occult) blood loss, most often from the digestive tract;
• combination of IDA with other anemic syndromes (B12-deficiency, folic acid deficiency).
Parenteral iron preparations.
Indications for prescribing parenteral iron preparations:
Malabsorption in intestinal pathology (enteritis, syndrome insufficiency of absorption, resection of the small intestine, resection of the stomach according to the Billroth II method with switching off the duodenum);
• pronounced gastrointestinal side effects of oral therapy, which cannot be eliminated by other means;
Persistent blood loss, in which the need for iron exceeds the physiological capacity for iron absorption (for example, severe uterine bleeding, hereditary hemorrhagic telangiectasia with damage to the mucous membranes);
• the patient's wish for a quick (within 1-2 visits) replenishment of iron deficiency and refusal from long-term ferrotherapy for many months.

Combined therapy with parenteral iron preparations and oral iron preparations.
There is no evidence that combination ferrotherapy is more effective. At the same time, when the two drugs are combined, side effects of each of the drugs may develop.

List of essential medicines:
Monocomponent iron medicines:
Ferrous gluconate * 300 mg - Oral administration 2 tab. x 2-3 times/day
Ferrous sulfate 256.3 mg (80 mg iron), Ferrous sulfate 325 mg (105 mg iron (II) ion - Oral administration 1 tab. X 2 times
Iron fumarate suspension 3 g) for children, Iron fumarate 200 - tab. 200 mg (65 mg of iron), Iron fumarate - caps. 300 mg (100 mg of iron el.) - Oral administration: iron fumarate by 1tab. x 3 times a day, 1 caps. x 2-4 times a day

Combined drugs:
Ferrous sulfate tab. 320 mg + ascorbic acid 60 mg - Oral intake, 1 tab. x 1-2 times a day
Iron fumarate 163.56 mg (50 mg iron) and folic acid 540 mcg - Orally 1 caps. x 2 times a day
Iron preparations III – valence:
Iron (III) hydroxide polymaltose 400 mg (100 mg elemental iron) Tab. 375 mg (100 mg e iron) - Oral administration, 1 chewable. tab. x 2-3 r per day
Iron carboxymaltose 156-208 mg (50 mg iron) - intravenous stream, in a maximum single dose of up to 4 ml (200 mg iron) per day, but not more than 3 times a week. intravenous (intravenous) drip (infusion) 1000 mg of iron once a week.
Further management:
For patients receiving oral ferrotherapy, control studies of the GBA should be performed 1-2 weeks after the start of treatment.
• When treating with parenteral drugs, especially with a single dose, control studies of the CBC can be performed 4-8 weeks after drug administration.
• Patients with ongoing bleeding (eg, hereditary hemorrhagic telangiectasia) need more frequent monitoring.
NB! The reasons for the recurrence of IDA may be insufficient duration of oral medications, ongoing blood loss, incorrect diagnosis IDA, the presence of additional reasons for the development of anemia.
INDICATIONS FOR HOSPITALIZATION:
Hospitalization in a 24-hour hospital can be carried out only in exceptional cases if there are signs of hemodynamic instability due only to confirmed IDA with the exclusion of all other causes of hemodynamic instability.
Indications for planned hospitalization: absent.
Indications for emergency hospitalization:
Emergency hospitalization of a patient with IDA is indicated only if there are signs of hemodynamic instability with the exclusion of other causes of hemodynamic instability:
• if the source of bleeding is identified, depending on the source of blood loss, to the department of the surgical profile (surgery, gynecology, proctology, etc.);
• in the absence of established bleeding - admission to the therapeutic department.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 10: Diabetes mellitus in GMP.
II. Learning goals: Diagnosis and treatment of Diabetes mellitus on ambulatory level.
III. Lecture thesis:
Diabetes mellitus (DM) is a group of metabolic (metabolic) diseases characterized by chronic hyperglycemia, which is the result of impaired insulin secretion, the action of insulin, or both.
Chronic hyperglycemia in diabetes is associated with damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels.

Classification:
Clinical classification

Type 1 diabetes	Destruction of β-cells of the pancreas, usually leading to absolute insulin deficiency
DM type 2	Progressive violation of insulin secretion in the presence of insulin resistance
Other specific types of diabetes	- genetic defects in β-cell function; - genetic defects in insulin action; - diseases of the exocrine pancreas; - induced by drugs or chemicals (in the treatment of HIV / AIDS or after organ transplantation); - endocrinopathy; - infections; - other genetic syndromes associated with diabetes
Gestational diabetes	occurs during pregnancy

DIAGNOSTIC METHODS, APPROACHES AND PROCEDURES
Diagnostic criteria
Complaints:
• weakness;
• malaise;
• decreased performance;
• skin and vaginal itching
• polyuria;
• polydipsia;
• complaints of periodic blurred vision;
• feeling of heat in the feet;
• cramps in the lower extremities and paresthesia at night;
• dystrophic changes in the skin and nails on the feet.
Anamnesis:
The disease usually manifests itself at the age of over 40 years, it is preceded by the presence of metabolic syndrome components (obesity, arterial hypertension, etc.).

Physical examination:
Patients with type 2 diabetes have:
• signs of visceral obesity, AH, acanthosis nigricans;
• an increase in the size of the liver;
• signs of dehydration (dry mucous membranes, skin, decreased skin turgor, hypotension);
• signs of hypokalemia (extrasystoles, muscle weakness, intestinal atony);
• signs of neuropathy (paresthesias, dystrophic changes in the skin and nails, ulcerative defects of the feet).

Laboratory tests:
• Determination of blood glucose;
• Determination of HbA1c - glycosylated hemoglobin.

Diagnostic criteria for diabetes mellitus and other glycemic disorders

Determination time	**Glucose concentration, mmol/L**	
	Capillary blood	**Venous plasma**

NORM		
On an empty stomach	< 5,6	< 6,1
2 hours after oral glucose tolerance test	< 7,8	< 7,8
Diabetes		
On an empty stomach	≥ 6,1	≥ 7,0
or 2 hours after oral glucose tolerance test	≥ 11,1	≥ 11,1
or random definition	≥ 11,1	≥ 11,1

HbA1c (glycosylated hemoglobin) as a diagnostic criterion for diabetes:
The HbAlc level ≥ 6.5% (48 mmol / mol) was chosen as a diagnostic criterion for diabetes.
An HbAlc level of up to 5.7% is considered normal, provided that it is determined by the National Glicohemoglobin Standardization Program (NGSP) method, according to the standardized Diabetes Control and Complications Trial (DCCT).
In the absence of symptoms of acute metabolic decompensation, the diagnosis should be made on the basis of two digits that are in the diabetic range, for example, a double HbAlc test or a single HbAlc + test at least one glucose test.

Diagnostic criteria

HbAlc≥ 6,5%	**Glycemia on an empty stomach ≥ 7,0**
Glycemia 2 hours after oral glucose tolerance test ≥ 11,1	**A patient with classic symptoms of hyperglycemia has glycemia at any time ≥11,1**

Instrumental research methods:
• ECG - to detect rhythm disturbances, myocardial ischemia, signs of left ventricular myocardial hypertrophy, systolic overload;
• EchoCG - to detect signs of dystrophy of certain areas of the myocardium, ischemic zones, decrease in the expulsion fraction, dilatation of the heart cavities, myocardial hypertrophy;
• ultrasound of the abdominal organs - to identify concomitant pathology;
• Doppler ultrasonography of the vessels of the lower extremities - to detect changes in the speed indicators of blood flow in the main arteries and arteries of the feet;
• Holter monitoring - to detect hidden increases in blood pressure, arrhythmias;
• daily continuous monitoring of glycemia in order to select and correct antihyperglycemic therapy, educate patients and involve them in the treatment process;
• X-ray of the feet - to assess the severity and depth of tissue damage in diabetic foot syndrome;
• microbiological examination of wound discharge in case of trophic lesions of the feet - for rational antibiotic therapy;
• electromyography of the lower extremities - for early diagnosis of diabetic polyneuropathy.

Indications for specialist consultations

Specialist	**Objectives of the consultation**

Ophthalmologist's consultation	For the diagnosis and treatment of diabetic eye damage - according to indications
Neurologist's consultation	For the diagnosis and treatment of complications of diabetes - according to indications
Nephrologist's consultation	For the diagnosis and treatment of complications of diabetes - according to indications
Cardiologist consultation	For the diagnosis and treatment of complications of diabetes - according to indications
Angiosurgeon's consultation	For the diagnosis and treatment of complications of diabetes - according to indications

Algorithm for screening and diagnosis of type 2 diabetes mellitus.
Screening methods for type 2 diabetes
Screening is done to identify patients who may have type 2 diabetes. Screening for type 2 diabetes begins with the determination of fasting blood glucose. In case of detection of normoglycemia or impaired fasting glucose (FGN) - more than 5.5 mmol / L, but less than 6.1 mmol / L for capillary blood and more than 6.1 mmol / L, but less than 7.0 mmol / L for venous blood plasma is prescribed an oral glucose tolerance test (OGTT).
Oral glucose tolerance test is not performed:
• against the background of an acute illness;
• against the background of short-term use of drugs that increase the level of glycemia (glucocorticoids, thyroid hormones, thiazides, beta-blockers, etc.)

Oral glucose tolerance test should be done in the morning with at least 3 days of unrestricted nutrition (more than 150 g of carbohydrates per day). The test should be preceded by an overnight fast for at least 8-14 hours (you can drink water). After taking blood on an empty stomach, the subject should drink 75 g of anhydrous glucose or 82.5 g of glucose monohydrate dissolved in 250-300 ml of water in no more than 5 minutes. For children, the load is 1.75 g of anhydrous glucose per kg of body weight, but not more than 75 g. After 2 hours, a second blood sample is taken.
Indications for Screening for Asymptomatic Diabetes
All individuals with a BMI ≥25 kg / m2 and the following risk factors are eligible for screening:
• sedentary lifestyle;
• relatives of the 1st line of kinship suffering from diabetes mellitus;
• ethnic populations with a high risk of diabetes mellitus;
• women with a history of large fetal delivery or established gestational diabetes;
• hypertension (≥140 / 90 mm Hg or on antihypertensive therapy);
• High-density lipoprotein level 0.9 mmol / L (or 35 mg / dL) and / or triglyceride level 2.82 mmol / L (250 mg / dL);
• presence of HbAlc ≥ 5.7%, preceding impaired glucose tolerance or impaired fasting glucose;
• history of cardiovascular disease;
• other clinical conditions associated with insulin resistance (including severe obesity, acanthosis nigricans);
• polycystic ovary syndrome.

If the test is normal, it should be repeated every 3 years. If there are no risk factors, screening is done for all persons over 45 years of age. If the test is normal, it should be repeated every 3 years.
Differential diagnosis
Criteria for the differential diagnosis of type 1 diabetes and type 2 diabetes

type 1 diabetes	type 2 diabetes
Young age, acute onset (thirst, polyuria, weight loss, presence of acetone in urine)	Obesity, hypertension, sedentary lifestyle, the presence of diabetes in relatives
Autoimmune destruction of β-cells of the islets of the pancreas	Insulin resistance combined with β-cell secretory dysfunction
Low level of immunoreactive insulin, C-peptide, high titer of specific antibodiesto islet cells	Increased level of IRI, C-peptide in the blood, lack of specific antibodiesto islet cells

TACTICS OF TREATMENT AT THE AMBULATORY LEVEL
Patients with type 2 diabetes without acute complications are subject to outpatient treatment.

Treatment goals:
• achievement of individual target levels of glycemia and HbA1c;
• normalization of blood pressure;
• normalization of lipid metabolism;
• prevention of diabetes complications.

These HbAlc target levels must meet the following pre / post-prandial plasma glucose targets

HbAlc	Plasma glucose on an empty stomach / before meals, mmol / l	Plasma glucose 2 hours after eating, mmol / l
< 6,5	< 6,5	< 8,0
< 7,0	< 7,0	< 9,0
< 7,5	< 7,5	<10,0
< 8,0	< 8,0	< 11,0

Target indicators of lipid metabolism in patients with diabetes

Risk groups	Target values of low density lipoproteins, mmol / l
With moderate cardiovascular risk	<2,6
High cardiovascular risk	<1,8
Very high cardiovascular risk	<1,4 or decrease by 50%

Target blood pressure in patients with diabetes

Age	Systolic blood pressure, mm Hg Art.	Diastolic blood pressure mm Hg. Art.
18-65	> 120-<130	> 70-<80

Treatment tactics for type 2 diabetes:
• Diet therapy;
• Physical activity;
• Training and self-control;
• Sugar-lowering drugs.

Non-drug treatment:
• Diet number 9
• Physical activity - taking into account the state of the cardiovascular system • Education at the School of Diabetes
• Self-control and daily continuous glucose monitoring

Drug treatment:
General principles of initiation and intensification of hypoglycemic therapy
• stratification of treatment tactics depending on the baseline level of HbA1c detected during the diagnosis of type 2 diabetes and the patient's condition;
• monitoring of the effectiveness of **hypoglycemic therapy** by the level of HbA1c is carried out every 3 months;
• change (intensification) of **hypoglycemic therapy** in case of its ineffectiveness (in the absence of achievement of individual goals HbA1c) is carried out no later than in 3-6 months.
List of essential drugs (100% likely to be used):
Sulfonylureapreparations:
-Gliclazide
- Glimepiride
- Glibenclamide
Glinides:
Repaglinide
Biguanides:
Metformin
Further management:
List of laboratory parameters requiring dynamic monitoring in patients with type 2 diabetes:

Laboratory indicator	**Examination frequency**
Self-control of glycemia	At the onset of the disease and during decompensation - several times a day every day. In the future, depending on the type of antihyperglycemic therapy: - on intensified insulin therapy: at least 4 times daily; - on ready-made mixtures of insulin: at least 2 times a day at different times + 1 glycemic profile (at least 4 times a day) per week;
HbA1c	Once every 3 months

Biochemical blood test (total protein, cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, bilirubin, AST, ALT, creatinine, GFR calculation, K, Na,)	Once a year
General urine analysis	Once a year
Determination of the ratio of albumin and creatinine in urine	Once a year
Determination of ketone bodies in urine and blood	According to indications

List of instrumental examinations required for dynamic control in patients with type 2 diabetes

Instrumental examination method	**Examination frequency**
Daily continuous glucose monitoring	According to indications, at least 4 times a year
Blood pressure control	At every visit to the doctor. In the presence of hypertension, self-control of blood pressure
Examining the feet and assessing the sensitivity of the feet	At each visit to the doctor
Electroneuromyography of the legs	Once a year
ECG	Once a year
ECG (with stress tests)	Once a year
Chest X-ray	Once a year
Doppler ultrasonography of the vessels of the lower extremities and kidneys	Once a year
Abdominal ultrasound	Once a year

Treatment effectiveness indicators:
• achievement of individual goals of HbA1c and glycemia;
• achievement of target indicators of lipid metabolism;
• achievement of target blood pressure levels;
• development of motivation for self-control.

INDICATIONS FOR HOSPITALIZATION WITH INDICATION OF THE TYPE OF HOSPITALIZATION:

Indications for planned hospitalization:
• the state of decompensation of carbohydrate metabolism, uncorrected in outpatient setting;
• frequently recurring hypoglycemia for a month or more;
• progression of neurological and vascular (retinopathy, nephropathy) complications of type 2 diabetes, diabetic foot syndrome;
• pregnant women with type 2 diabetes detected during pregnancy.

Indications for emergency hospitalization:
• coma - hyperosmolar, hypoglycemic, ketoacidotic, lactic acid.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
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Prevention

I. Theme № 11: Hyperthyroidism in GMP.
II. Learning goals: Diagnosis and treatment of hyperthyroidism on ambulatory level.
III. Lecture thesis:
Hyperthyroidism is a clinical syndrome caused by an excess of thyroid hormones (TG) in the blood and their toxic effects on various organs and tissues.

Classification:
1) Thyrotoxicosis due to increased production of thyroid hormones:
• Graves' disease (GD);
• toxic adenoma (TA);
• iodine-induced hyperthyroidism;
• hyperthyroid phase of autoimmune thyroiditis (AIT);
• TSH - due to hyperthyroidism.
− TSH-producing pituitary adenoma;
- syndrome of inadequate secretion of TSH (resistance of thyrotrophs to thyroid hormones).
• trophoblastic hyperthyroidism.
2) Hyperthyroidism due to the production of thyroid hormones outside the thyroid gland:
• metastases of thyroid cancer producing thyroid hormones;
• chorinoepithelioma.
3) Thyrotoxicosis, not associated with hyperproduction of thyroid hormones:
• drug-induced thyrotoxicosis (overdose of thyroid hormone preparations);
• thyrotoxicosis, as a stage of subacute de Quervain's thyroiditis, postpartum thyroiditis.

Grade of goiter	**Characteristic**
0	There is no goiter. On palpation, the size of each lobe does not exceed the size distal phalanx of the thumb of the examined
I	The size of the goiter is larger than the distal phalanx of the thumb examined, the goiter is palpable, but not visible
II	Goiter is palpable and visible to the eye

Diagnostic criteria:
Complaints and anamnesis:
Complaints:
• nervousness;
• sweating;
• heartbeat;

• increased fatigue;
• increased appetite and, despite this, weight loss;
• general weakness;
• emotional lability;
• shortness of breath;
• sleep disturbance, sometimes insomnia;
• poor tolerance of elevated ambient temperature;
• diarrhea;
• discomfort from the eyes - discomfort in the area of the eyeballs, trembling of the eyelids;
• violations of the menstrual cycle.

History:
• the presence of relatives suffering from diseases of the thyroid gland;
• frequent acute respiratory diseases;
• local infectious processes (chronic tonsillitis).
Physical examination:
• increase in the size of the thyroid gland;
• violations of cardiac activity (tachycardia, loud heart sounds, sometimes systolic murmur at the apex, increased systolic and decreased diastolic blood pressure, attacks of atrial fibrillation);
• violations of the central and sympathetic nervous system (tremor of the fingers, tongue, entire body, sweating, irritability, anxiety and fear, hyperreflexia);
• metabolic disorders (heat intolerance, weight loss, increased appetite, thirst, accelerated growth);
• violations of the gastrointestinal tract (loose stools, abdominal pain, increased peristalsis);
• eye symptoms (wide opening of the palpebral fissures, exophthalmos, frightened or wary look, blurred vision, doubling, lagging of the upper eyelid when looking down and the lower eyelid when looking up). Approximately 40-50% of patients develop endocrine ophthalmopathy, which is characterized by damage to the soft tissues of the orbit: retrobulbar tissue, oculomotor muscles; with involvement of the optic nerve and auxiliary apparatus of the eye (eyelids, cornea, conjunctiva, lacrimal gland). Patients develop spontaneous retrobulbar pain, pain with eye movements, erythema of the eyelids, edema or swelling of the eyelids, conjunctival hyperemia, chemosis, proptosis, limitation of the mobility of the oculomotor muscles. The most severe complications of endocrine ophthalmopathy are: optic neuropathy, keratopathy with the formation of a walleye, corneal perforation, ophthalmoplegia, diplopia, from the side of the muscular system (muscle weakness, atrophy, myasthenia gravis, periodic paralysis)).
Laboratory research:

Test	**Indications**
TSH (thyroid stimulating hormone)	Decreased less than 0.1 mIU/l
Free T4	Increased
Free T3	Increased
antibodies to thyroperoxidase, antibodies to thyroglobulin	Increased
antibodies to the TSH receptor	Increased
ESR	Increased in subacute de Quervain's thyroiditis
Chorionic gonadotropin	Increased in choriocarcinoma

Instrumental research:

Test	**Indications**
Ultrasound	The volume and echostructure of the thyroid gland is determined. In hyperthyroidism: diffuse increase in thyroid volume, thyroid

	echogenicity is evenly reduced, echo structure is homogeneous, blood supply is increased. In AIT: heterogeneity of echogenicity. In multinodular toxic goiter: formations in the thyroid gland. In thyroid cancer: hypoechoic formations with uneven contours of the node, growth of the node behind the capsule and calcification.
Scintigraphy of the thyroid gland. Used technetium isotope 99mTc, I^{123}, I^{131}	In hyperthyroidism, there is an increase and a uniform isotope distribution. In functional autonomy, the isotope accumulates actively functioning node, while surrounding thyroid tissue is in a state suppression. In destructive thyroiditis (subacute, postpartum) radiopharmaceutical uptake is reduced. Thyrotoxic adenoma and multinodular toxic goiter are characterized by "hot nodes", in cancer - "cold nodes". In iodine-deficient regions, thyroid scintigraphy in multinodular toxic goiter is indicated even if the TSH level is in the region of the lower limit of normal
CT scan Magnetic resonance imaging X-ray examination with contrasting of the esophagus with barium	These methods help diagnose retrosternal goiter, clarify the location of the goiter in relation to the surrounding tissue, determine the displacement or compression of the trachea and esophagus
Fine-angle aspiration biopsy of the thyroid gland and cytological examination	They are carried out in the presence of nodes in the thyroid gland. Needle biopsy is indicated for all palpable nodules; the risk of cancer is the same in solitary nodular formation and multinodular goiter. In neoplasms of the thyroid gland, cancer cells are detected. With AIT - lymphocytic infiltration.

TACTICS OF TREATMENT AT OUTPATIENT LEVEL:

patients with previously diagnosed hyperthyroidism without decompensation of the disease, not requiring radioiodine therapy, surgical treatment, without thyrotoxic crisis are subject to outpatient treatment.

Non-drug treatment:

• Mode: depends on the severity of the condition and the presence of complications. Exclude physical activity, tk. with thyrotoxicosis, muscle weakness and fatigue increase, thermoregulation is disturbed, and the load on the heart increases.

• Diet: before the establishment of euthyroidism, it is necessary to limit the intake of iodine with contrast agents, because. iodine in most cases contributes to the development of thyrotoxicosis. Caffeine should be excluded, because. caffeine may exacerbate symptoms of thyrotoxicosis

Drug treatment:

Conservative thyreostatic therapy:

To suppress the production of thyroid hormones of the thyroid gland, it is necessary to use thiamazole. Apply thiamazole in a daily dose of 20-40 mg. In case of severe clinical and biochemical hyperthyroidism, the doses can be increased by 50-100%.

Side effects of thyreostatic therapy are possible: allergic reactions, liver pathology (1.3%), agranulocytosis (0.2 - 0.4%). With the development of fever, arthralgia, ulcers on the tongue, pharyngitis or severe malaise, the use of thyreostatics should be immediately stopped and an extended leukogram determined.

The duration of conservative treatment with thyreostatics is 12-18 months.

Further management:

• Monitoring of patients receiving antithyroid therapy is carried out for early detection of side effects such as rash, liver pathology, agranulocytosis. It is necessary to study the levels of free T4 and TSH every 4 weeks for early detection of hypothyroidism and the appointment of replacement therapy. Within a year after reaching euthyroidism, a laboratory assessment of thyroid function is carried out once every 3-6 months, then every 6-12 months.

• Pregnant women with HD should use the lowest doses of thyreostatics, ensuring that thyroid hormone levels are slightly above the reference range, with suppressed TSH. Thyroid function during pregnancy should be assessed monthly and the thyreostatic dose adjusted as needed.

Treatment effectiveness indicators:

• reduction or elimination of symptoms of thyrotoxicosis, allowing the patient to be transferred to outpatient treatment;
• reduction in the size of the goiter;
• reduction of the dose of thyreostatics necessary to maintain euthyroidism;
• disappearance or decrease in the content of antibodies to TSH receptors.

INDICATIONS FOR HOSPITALIZATION:

Indications for planned hospitalization:

• newly diagnosed thyrotoxicosis;
• decompensation of thyrotoxicosis;
• conducting radioiodine therapy;
• surgical treatment (thyroidectomy).

Indications for emergency hospitalization:

• thyrotoxic crisis.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:

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I. Theme № 12: Organization of medical and preventive care at the PHC level.

II. Learning goals: Organization of medical and preventive care for newborns, young children.

III. Lecture thesis:

Organization of pediatric care at the outpatient-polyclinic level

Healthcare organizations providing outpatient care for children carry out their activities in accordance with the Regulations on the activities of health care organizations providing outpatient care, approved by order of the Acting Minister of Health of the Republic of Kazakhstan dated January 5, 2011 No. 7 (registered in the Register of State Registration of Regulatory and Legal Acts under No. 6774). Monitoring of the child's development from the day of discharge from the obstetric care organization or the maternity ward of a multidisciplinary hospital is carried out in accordance with Form No. 112, approved by Order No. 907.

At the outpatient level, in accordance with the Rules for the provision of primary health care and the Rules for attaching citizens to primary health care organizations, approved by order of the Minister of

Health and Social Development of the Republic of Kazakhstan dated April 28, 2015 No. 281 (registered in the Register of State Registration of Regulatory and legal acts under No. 11268) is carried out:

1) advisory, diagnostic, medical assistance, medical examination and medical rehabilitation for children;

2) patronage and active visits to pregnant women, newborns and young children in accordance with the universally progressive model of the patronage service;

3) planning, organizing and conducting vaccination in accordance with the terms of preventive vaccinations approved by the Government of the Republic of Kazakhstan dated December 30, 2009 No. 2295 "On approval of the list of diseases against which preventive vaccinations are carried out, the Rules for their conduct and population groups subject to routine vaccinations" ;

4) referral of children for consultations to specialized specialists if indicated;

5) identification of acute and chronic diseases, timely implementation of emergency and planned treatment measures;

6) referral of children to a round-the-clock hospital, day hospital and organization of a hospital at home if indicated;

7) dynamic observation of children with chronic diseases who are registered with dispensaries, treatment and health improvement;

8) rehabilitation treatment and medical rehabilitation;

9) conducting screening examinations in accordance with the order of the Minister of Health of the Republic of Kazakhstan dated September 9, 2010 No. 704 "On approval of the Rules for organizing screening" (registered in the Register of State Registration of Normative Legal Acts under No. 6490) to identify congenital pathology and disorders of psychophysical development, visual and auditory functions in young children;

10) organization of children's health improvement before their admission to preschool or school institutions;

11) information work with parents and family members or with their legal representatives on the issues of rational nutrition, prevention of childhood diseases and the formation of a healthy lifestyle.

The provision of emergency medical care to children in a medical organization (or a medical center) is carried out in accordance with the clinical protocols for diagnosis and treatment approved by the Joint Commission for the Quality of Medical Services of the Ministry of Health of the Republic of Kazakhstan. For the provision of emergency medical care to children in a medical organization (or a medical center), the availability of medicines and medical devices is ensured in accordance with clinical protocols

diagnostics and treatment approved by the Joint Commission for the Quality of Medical Services of the Ministry of Health of the Republic of Kazakhstan.

Medicines and medical products for the provision of emergency medical care at the outpatient level are in special plastic boxes - "suitcases" made of processed material, which are easily carried and placed in a convenient and accessible place.

The volume of emergency medical care provided to the patient is recorded in the prescription and observation sheet indicating the heart rate, blood pressure, pulse, body temperature, name and dose of the drug, methods and time of administration.

The provision of medical care to students and pupils of educational organizations is carried out in accordance with the Rules for the provision of medical care to students and pupils of educational organizations, approved by order of the Minister of Health of the Republic of Kazakhstan dated April 7, 2017 No. 141 (registered in the Register of State Registration of Normative Legal Acts under No. 15131). When chronic diseases are detected, dispensary registration is carried out according to the indications with a record of the examination in the medical card of an outpatient in the form No. 025 / y, approved by Order No. 907, drawing up a management plan, drawing up a control card for dispensary observation in form No. 30 / y, approved by Order No. 907.

Anti-epidemic and preventive measures are being taken at the site to prevent the spread of infections among children.

The registration of preventive vaccinations is carried out by the corresponding entries in the registration forms, which are stored at health facilities at the place of vaccination, at educational facilities,

preschool education and training: a register of preventive vaccinations in form No. 064 / y, history of the child's development in form No. 112 / y , a card of preventive vaccinations according to the form No. 063 / y, a medical card of the child according to the form No. 026 / y, approved by Order No. 907.

A pediatrician or GP carries out the execution of medical documentation for children for sanatorium treatment.

Registration and issuance of sheets (certificates) on temporary disability to parents (guardians) for caring for a sick child according to indications, a certificate of temporary disability to release a child from attending preschool and school institutions for a period of illness is carried out in accordance with the Rules for the examination of temporary disability, issuance of a sheet and certificates on temporary disability, approved by order of the Minister of Health and Social Development of the Republic of Kazakhstan dated March 31, 2015 No. 183 (registered in the Register of State Registration of Normative Legal Acts No. 10964).

The issue of exempting children studying in educational institutions from transfer and final exams for diseases is decided by a medical advisory commission.

When a child is found to have persistent disorders of the body's functions with the results of an examination in the medical record of an outpatient at the VKK, the issue of referral to a medical and social examination is considered, in accordance with the Rules for conducting a medical and social examination, approved by order of the Minister of Health andsocial development of the Republic of Kazakhstan dated January 30, 2015 No. 44 (registered in the Register of State Registration of Normative Legal Acts No. 10589).

When disability is established, the development and implementation of individual rehabilitation programs for children with disabilities is carried out, including with the involvement of social services.

A pediatrician, a GP and a paramedical worker maintains accounting and reporting documentation with the provision of reports on the main medical and statistical indicators of morbidity, disability and mortality in children of the served area to the head of the unit (head of the department).

Provision of preventive care for children at the level of the Ministry of Health of the Primary Health Care is carried out by organizing:

1) the work of the child development room (hereinafter referred to as the CRC) in all medical organizations providing primary health care to the child population, the CRC is equipped in accordance with Appendix 1 to this Standard;

2) neonatal screening, audiological screening of newborns and young children, screening of psychophysical development of young children, ophthalmological screening of premature newborns for the purpose of early diagnosis of congenital and hereditary diseases in children, reducing childhood morbidity and disability;

3) patronage supervision at home of pregnant women, newborns and young children.

The doctor or paramedical worker of the CRC conducts activities for:

advising on the issues of caring for young children and the formation of parenting skills for mothers and their families, explaining the importance of games, reading, communication for the development of the child;

informing a pregnant or lactating mother about proper nutrition, family planning, pregnancy, breastfeeding support;

teaching the parent (s) the skills of timely introduction of complementary foods and their practical preparation, taking into account sanitary safety standards and energy requirements;

explaining to the parent (s) measures to create a safe environment for children, preventing injury, poisoning and accidents;

monitoring the child's psychomotor and speech development and counseling parents on identified problems;

counseling on the issues of caring for sick children at home and for children with developmental disabilities;

prevention of domestic violence and child abuse.

Preventive admission (observation) of children in medical care includes a comprehensive assessment and monitoring of the child's development: physical, sexual development, assessment of hearing, vision, fine

and gross motor skills, expressive and receptive speech, emotions, the ability to self-regulate and establish relationships, play and mutual participation.
When detecting chronic diseases, hearing and vision impairments, anomalies in the development of the sense organs, as well as stenosis of the larynx and trachea, the local doctor directs the child to specialized specialists to clarify the diagnosis and prescribe treatment.
When detecting children with a risk of retardation in psychophysical development, with a decrease in hearing and vision, neurological symptoms, a medical worker directs them to psychological, medical and pedagogical consultation.

Patients with delayed neuropsychic development due to somatic pathology, syncope, sluggish child syndrome, febrileconvulsions after exclusion of the pathology of the nervous system by a doctor specializing in "Neurology (children's)" are observed by pediatricians.
In the absence of a doctor specializing in Neurology (Pediatric), the issues of organizing and conducting additional examination or hospitalization in the specialized department of the hospital for the provision of inpatient specialized medical care are decided by the district pediatrician or GP.
KDP for children is provided by specialized specialists in a consultative and diagnostic center or polyclinic (department) in the direction of a PHC doctor or other specialist, except in cases of emergency and urgent medical care.
The organization of home visits for pregnant women, newborns and young children is carried out on the basis of a universally progressive model recommended by the United Nations Children's Fund (UNICEF) in order to identify and reduce medical or social risks that threaten the life, health, development of the child, as well as reduce the number compulsory visits to families that do not have risks. With the universally progressive model of patronage, along with mandatory scheduled visits (universal approach), additional active visits according to an individual plan (progressive approach) are introduced for pregnant women, newborns and children in need of special support due to the presence of medical or social risks to life. health or development of the child.

Universal (compulsory) patronage supervision is provided to all pregnant women and children under 5 years of age and consists of 2 antenatal visits to a pregnant woman (up to 12 weeks and 32 weeks of pregnancy) and 9 visits to children according to the General Scheme of observation of pregnant women, newborns and children up to 5 years as a doctor / feldsher and paramedical worker at home and at the reception at the Ministry of Health at the PHC level in accordance with Appendix 2 to this Standard.
The progressive approach provides for patronage supervision of pregnant women and children who have identified risks of a medical or social nature that pose a threat to their life, health, development and safety according to the Scheme of a universal progressive model of patronage of pregnant women and children under 5 years of age (home visits by average healthcare professional) in accordance with Appendix 3 to this Standard.
When a moderate risk is identified (including problems with breastfeeding, complementary feeding, difficulties with hygiene skills, with playing, communicating, and others), the paramedical worker carries out work to eliminate it independently or in conjunction with the local doctor. In case of high risk (abuse, violence, neglect, child disability, etc.), when the family needs social support, information is transferred to a social worker, psychologist or representatives of other sectors if necessary (education, social protection, internal affairs, akimats, non-governmental organizations and others).
When visiting newborns and young children at home, a paramedic or nurse takes with them a child's tonometer, measuring tape, thermometer.
When visiting a pregnant woman, a paramedical worker:

1) asks the pregnant woman for complaints, measures blood pressure, examines for edema and signs of anemia;

2) assesses mood (presence of depression), safety of the home environment and living conditions, hygiene of the premises and personal hygiene, risk factors for pregnancy;

3) informs about the physical and mental changes associated with pregnancy; the harmful effects of stress during pregnancy; about the harmful effects of smoking and the use of alcohol and drugs;

4) gives recommendations on balanced nutrition, weight control, physical activity, oral hygiene, personal hygiene;

5) teaches the family about the alarming signs of pregnancy, when it is necessary to immediately consult a doctor and plan antenatal care with a therapist and gynecologist;

6) advises and teaches preparation for childbirth, how to prepare a room, place, care items and clothing for a newborn, basic newborn care, the importance of exclusive breastfeeding and breastfeeding techniques.

46. When visiting a newborn baby, a paramedical worker:

1) assesses the signs of a disease or local bacterial infection in a newborn and, if any, informs the doctor immediately;

2) assesses the mood of the mother (parent or other legal representative) in order to identify depression, the safety of the home environment and the needs of the newborn;

3) asks about the state of health of the parturient woman (complaints, condition of the mammary glands, physical activity, nutrition, sleep, contraception);

4) informs, consults and teaches the mother (parent or other legal representative) the basic care of the newborn: breastfeeding, temperature regime, developmental care and attitude towards the child, the participation of both parents (if any) in raising the child, hygiene and washing issues hands, safety when bathing, safety in sleep, prevention of sudden death syndrome, hygienic care of the umbilical cord and skin;

5) teaches the family the danger signs of diseases in which it is necessary to immediately contact a medical organization: feeding problems, decreased activity of the newborn, rapid breathing of more than 60 per minute, difficulty breathing, fever or lower temperature, convulsions, chills and others;

6) promotes timely vaccination;

7) conducts an initial assessment of social risks that threaten the life, health, safety and development of the child and, if risks are identified, informs the social worker of the Ministry of Health providing outpatient care;

8) identifies newborns in need of additional assistance and plans individual visits for them (children with low birth weight, sick or born to HIV-infected mothers).

When visiting young children, the paramedical worker:

1) assesses the general signs of danger, the main symptoms of diseases (cough, diarrhea, fever, and others), checks for anemia or low weight; assesses the mood of the mother (parent or other legal representative) in order to identify depression; the needs of the child depending on the age; home safety in terms of injuries and accidents; signs of neglect, abuse and violence against the child;

2) monitors physical, motor, psychosocial development;

3) asks about the state of health of the parturient woman (complaints, the condition of the mammary glands, physical activity, nutrition, sleep, contraception), the parturient woman on issues of exclusive breastfeeding up to 6 months, the introduction of complementary foods at 6 months;

4) train the parent (s) or legal representative to assist in the development of the child through play, communication, reading; hygiene issues, joint participation in the upbringing of a child of both parents (if any), dangerous signs of diseases in which you must immediately seek medical help;

5) informs the parent (s) or legal representative about the signs of childhood illnesses (cough, diarrhea, fever, etc.) and gives recommendations if they occur;

6) promotes vaccination;

7) assesses social risks that threaten the life, health, safety and development of the child and, if risks are identified, inform the social worker of the polyclinic.

If a moderate risk is identified, a paramedical worker, together with a social worker, a psychologist and with the participation of a pregnant woman or the child's parent (s) or legal representative, draws up an individual plan of patronage follow-up measures within the framework of a progressive approach in order to reduce or eliminate risks to life, health, development and child safety includes an assessment of the child's needs, analysis of the child's situation in the family, informing the head nurse (s) / brother, the district doctor, the head of the department and the social worker.

IV. Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 13: Rickets.
II. Learning goals: Diagnosis and treatment of Rickets on ambulatory level.
III. Lecture thesis:
Rickets is a group of diseases of the child's body associated with an insufficient intake of vitamin D or a violation of its metabolic processes, leading to a violation of many types of metabolism, primarily calcium-phosphorus metabolism, which causes damage to many organs and systems, but mainly, bone skeleton (Maidannik V.G., 2014).
Vitamin D-deficient rickets is a disease of an intensively growing child's body caused by dysregulation of calcium-phosphorus metabolism and bone mineralization as a result of vitamin D deficiency.
Clinical classification

Severity	**Course of the disease**	**Period**
I light II moderate III severe	Acute Subacute Recurrent	Initial manifestations Culmination Reconvalescence Residual changes

Secondary rickets often occurs:
• with malabsorption syndromes;
• in chronic diseases of the kidneys or biliary tract;
• with metabolic diseases (tyrosinemia, cystinuria, etc.);
• caused by prolonged use of anticonvulsants (phenobarbital), diuretics, glucocorticoids, as well as parenteral nutrition.

Vitamin D-dependent rickets:
• Type I - a genetic defect in the synthesis of 1,25-dihydroxyvitamin D - 1,25 (OH) 2D in the kidneys.
• Type II - genetic resistance of target organ receptors to 1,25 (OH) 2D.

Vitamin D-resistant rickets:
• Phosphate-diabetes;
• de Toni-Debré-Fanconi syndrome (de Toni-Debré-Fanconi);
• Hypophosphatasia;
• Renal tubular acidosis.
Diagnostic criteria

Complaints: anxiety, fearfulness, irritability, hyperesthesia, loss of appetite, sleep disturbance, flinching when falling asleep, loud sound, flash of light; increased sweating, the child rubs his head on the pillow, baldness of the back of the head; deformation of the bones of the head, chest, spine, limbs; lag in physical development, delayed teething

Anamnesis: the disease is detected from 3-4 months of age, although the first symptoms may appear in 1 - 1.5 months. Identification of risk factors

Clinical criteria:

• Symptoms of osteomalacia (softening, calcium depletion of the bone -
prevail in the acute course of rickets) - compliance of the bones of the skull, the edges of the fontanelle, craniotabes, brachycephaly, deformation of the bones of the skull, limbs, collarbones, flat pelvis, erosion and dental caries.

• Symptoms of osteoid hyperplasia (prevailing in the subacute course of rickets) - frontal and parietal tubercles, rib supracondylar thickening of the legs, on the forearms, "pearl threads" on the fingers.

• Symptoms of bone hypoplasia - growth retardation with a characteristic "short-legged", late eruption of deciduous and permanent teeth, late fontanelle closure.

• Symptoms of muscular hypotension - curvature of the spine with lumbar kyphosis, scoliosis, deformity of the chest with a deployed lower aperture, lethargy and flabbiness of muscles, looseness of joints, "frog" abdomen.

• Delay of static and locomotor functions.

Clinical picture

The**initial period** of rickets corresponds to minor disturbances in the general state: nervous excitability, anxiety, flinching at a sharp sound, flash of light, disturbances in the rhythm of sleep, superficial "disturbed" sleep. Sweating (sticky sweat with a sour smell), itching of the skin, the child rubs the back of the head against the pillow, which leads to baldness of the back of the head. On the part of the skeletal system: compliance of the edges of the large fontanelle.

With moderate severity of the disease, there are moderately pronounced changes in the skeletal system and internal organs: distinct deformities of the skull, chest, slight enlargement of the liver and spleen, moderate anemia;

• on the part of the central nervous system - the development of static and locomotor functions is delayed, later begins to raise his head, sit, stand up and walk, passive movements usually cause a negative reaction in them, behavior, sleep, appetite are disturbed;

• on the part of the muscular system - pronounced muscular hypotonia and weakness of the ligamentous apparatus: in the supine position, they easily pull the leg to the head, suck the first toe on the foot, put the foot on the shoulder; large, flattened "frog" abdomen, flabbiness of the muscles of the abdominal wall; divergence of the rectus abdominis muscles; predisposition to pneumonia, dystrophic changes in the heart are revealed;

• hypochromic anemia, mainly iron deficiency, but may be due to a deficiency of amino acids, copper and zinc;

• from the side of the bone tissue: symptoms of osteoid hyperplasia: frontal and parietal tubercles, costal "rosary", supracondylar thickening of the legs, "bracelets" on the wrists, "string of pearls" on the fingers;

• symptoms of bone hypoplasia: growth retardation due to lagging growth of tubular bones in length, late eruption of deciduous and permanent teeth, late closure of fontanelles, flat pelvis;

• bone deformities: deformities of the chest with an expanded lower aperture, "Harrison's groove", keeled ("chicken" breast) or funnel-shaped ("shoemaker's chest") deformity.

In severe rickets - damage to several parts of the skeletal system, severe damage to internal organs and the nervous system, lag in physical and mental development, deformation of the lower extremities - O-shaped (genuvarum), X-shaped (genuvalgum) and others (coxavara, genurecurvatum) ... Curvature of the spine in the form of scoliosis, lordosis, lumbar kyphosis.

The initial period most clearly begins to manifest itself at the age of 3-4 months, however, the first symptoms may appear earlier - at 1-1.5 months, but they are not specific and can often pass by the attention of parents. In the first place are neurological and autonomic changes. The child shows anxiety,

moodiness, sleep is disturbed - children fall asleep poorly and often wake up, fearfulness, irritability appear, children often startle from a loud sound or bright light. Appetite is noticeably reduced - the child is reluctant and takes a breast for a short time, a sluggish sucker - sometimes there are constipation. In addition, such vegetative manifestations as sweating, especially during sleep, and an increase in vascular excitability of the skin, which manifests itself in the form of an increase in the intensity and duration of red dermographism, draw attention to themselves. The scalp sweats most intensively, causing severe itching in the child, with which the child constantly rubs against the pillow, this leads to a baldness of the occiput specific to rickets. Also noteworthy is the characteristic sharp and sour smell of sweat. There may be some decrease in the muscle tone of the child. Bone changes are not typical for the initial period of rickets, but sometimes some suppleness of the edges of the large fontanelle can be found. The initial period of illness usually lasts 2 to 4 weeks.

During the **culminationperiod**, changes in the skeletal system progress: osteomalacia of the chest, lower extremities, excessive osteogenesis (rickety "rosary", "bracelets", frontal and parietal cranial tubercles). The child may lag behind in physical and mental development.

In severe rickets, the functional state of the liver, gastrointestinal tract, protein, lipid metabolism are disturbed, there is a deficiency of vitamins B1, B6, B5, A, E, C, copper, zinc, magnesium.

Most children with rickets of 1 and 2 degrees have symptoms of hypochromic anemia.

During the period of convalescence (recovery), the clinical and laboratory symptoms of rickets gradually disappear. The skeletal deformities arising from rickets remain in adulthood: impaired posture, changes in the chest, bones of the lower extremities. Postponed rickets can contribute to tooth damage (the development of multiple caries), the development of myopia, flat feet, narrowing, deformation of the pelvic bones.

• **Acute course** - rapid development of all symptoms, vivid neurological and autonomic disorders, significant hypophosphatemia, high alkaline phosphatase level, predominance of osteomalacia processes.

• **Subacute course** - characterized by moderate or subtle neurological and autonomic disorders, the prevalence of osteoid hyperplasia.

• **Recurrent course** - typical changes in periods of exacerbation and subsiding of the process with persisting residual effects. X-ray of growth zones reveals several bands of calcification in the metaphyses.

Laboratory research methods: there is no strict correlation between clinical signs of rickets and laboratory changes, so only 50% of children with hypovitaminosis D have a complete correspondence of biochemical and radiological data.

• The concentration of phosphorus in the blood serum can be reduced to 0.65-0.8 mmol /L (the norm in children under 1 year old is 1.5-1.8 mmol /L).

• Calcium concentration - up to 2.0-2.2 mmol / l (at a rate of 2.2-2.7 mmol /L).

• Concentration of ionized calcium less than 1.0 mmol /L.

• The ratio between the levels of calcium and phosphorus in the blood serum is normally equal to 2: 1, during the height of rickets it rises to 3: 1-4: 1. In the initial period of rickets and during the height of osteomalacia, the amount of calcium may be normal.

• Increase in the activity of alkaline phosphatase in the blood serum by 1.5-2 times (the absolute indicators of the norm depend on the method of its determination).

• A decrease in 25-OH-D in the blood (normally 20 ng / ml), a decrease to 10 ng /ml indicates a vitamin D deficiency, and a content below 5 ng / ml indicates avitaminosis (II B).

• Increased excretion of amino acids (aminoaciduria - above 10 mg / kg per day) is an early sign of rickets.

• Hyperphosphaturia and increased clearance of urine phosphates (normally 0.1-0.25 ml /sec, with rickets up to 0.5-1.0 ml /sec).

• Increase in serum osteocalcin content up to 90-170 ng /ml.

Instrumental research methods:

• **X-ray of bones.** On the roentgenogram of bones in places of the most intensive growth, especially enchondral ossification, osteoporosis is revealed and intensified; the calcification zone becomes not convex, but more horizontal, gradually smoothes out, becomes uneven, fringed; the gap between the pineal gland and the diaphysis increases due to the expanding metaphysis; the pineal gland becomes

saucer-shaped. Ossification points of small bones are detected in a timely manner, but on the roentgenogram they are less clearly detected. Subperiosteal fractures of the green twig type are often detected. In severe rickets, Loser zones are observed - transversely located transparent zones (stripes) several millimeters wide. In the initial period - minor osteoporosis.

• **Densitometry** (informative test without radiation exposure, according to indications, to determine the severity of osteoporosis).

Indications for consultation of specialists:

• consultations of specialists (according to indications) - concomitant somatic diseases and / or pathological conditions.

Indications for planned hospitalization:

• Rickets of the III degree of severity during the peak period.

• Rickets of II degree of severity during the peak period, not amenable to conventional complex therapy.

• Severe rickets deformities that appear in a child at the end of the first and at the beginning of the second year of life in order to differentiate with rickets-like diseases. The severity of the child's condition, polyuria, polydipsia, pronounced signs of damage to the vegetative part of the central nervous system, severe muscle hypotonia in a child in the first months of life with symptoms of rickets also for differentiating diagnosis with rickets-like diseases.

Indications for emergency hospitalization:

• Hypervitaminosis D, developed in the specific treatment of rickets of any degree.

• Spasmophilia.

Treatment goals:

• elimination of violations of the calcium-phosphorus metabolism;

• elimination and minimization of musculoskeletal disorders; • stabilization of the patient's condition.

Treatment tactics:

Treatment of rickets is usually outpatient treatment, severe hospitalization.

Non-drug treatment:

• diet therapy;

• massage, baths, stay in the fresh air;

The therapeutic effect for rickets is achieved only when vitamin-D therapy is combined with the rationalization of feeding the child, a balanced intake of calcium and phosphorus salts, normalization of sleep and wakefulness, prolonged stay in the fresh air, and other recreational activities

In the organization of nutrition in the treatment of rickets, a balanced diet is recommended in terms of the content of proteins, fats and carbohydrates, the predominance of foods rich in vitamins and minerals, in particular, calcium, phosphorus, magnesium, etc. vegetable and fruit juices, mashed potatoes. Hard-boiled yolk, rich in fat-soluble vitamins and B vitamins, as well as phosphorus, calcium, microelements should be added to vegetable puree.

In calcium for a healthy child the first 6-12 months is 500-600 mg per day.

For rickets during the period of reparation and with pronounced residual phenomena, salt, coniferous, salt-coniferous baths, sand, sea and sun baths are useful. Salt baths are indicated for pasty children, conifers - for children with reduced nutrition. General massage and gymnastics are required

Prescribing massage prior to prescribing drug therapy can have a negative effect.

Drug treatment:

For treatment, vitamin D preparations are used in the form of an aqueous and oil solution. Prescribe an aqueous solution of vitamin D3 (500 IU in one drop) or

oil solutions of vitamin D3 (in one drop of 500 IU) and Ergocalciferol (in one drop of 625, 1250 IU) at a dose of 2000-5000 IU in courses of 30-45 days.

After achieving the clinical effect (manifested by the normalization of muscle tone, the disappearance of craniotabes and autonomic disorders, the absence of progression of bone deformities, the onset of teething) and the normalization of laboratory parameters (alkaline phosphatase, calcium, phosphorus in the blood), they switch to a prophylactic dose of vitamin D (400-500 IU)), which the child receives daily during the first 2 years of life and in winter in the 3rd year.

For children from social risk groups, from the category of frequently and long-term illnesses, it is possible to carry out an anti-relapse course of specific treatment for rickets 3 months after the end of the main course in similar doses for 3-4 weeks.
At the time of fever in acute diseases in patients with rickets (ARVI, pneumonia, intestinal infection, otitis media and others), vitamin D intake should be stopped for 2-3 days. After the temperature has returned to normal, continue treatment.
Contraindications to the appointment of a prophylactic dose of vitamin D:
• idiopathic calciuria (Williams-Burnet disease);
• hypophosphatasia;
• organic lesion of the central nervous system with symptoms of microcephaly and craniostenosis.
Children with a small fontanelle have only relative contraindications to the appointment of vitamin D. Specific prophylaxis of rickets in them is carried out, starting from 3-4 months under the control of the size of the big town and the circumference of the head.
Further management (after hospitalization):
children who have had rickets of I degree are observed by a general practitioner (family doctor) or pediatrician for up to 2 years, and those who have had rickets of P-IP degree - for 3 years. Inspections are carried out once every 3 months.
According to indications, a general practitioner (family doctor) or pediatrician may prescribe a biochemical blood test (determination of total and ionized calcium, phosphorus and alkaline phosphatase content), bone densitometry or radiography, consult a child with an orthopedist, surgeon. Specific prophylaxis is carried out during the 2nd year of life in the autumn-winter-spring periods, and at the 3rd year of life only in winter.

Rickets is not a contraindication for prophylactic vaccinations.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 14: Immunoprophylaxis in children.
II. Learning goals: Organization of work on immunization in the Republic of Kazakhstan. National vaccination calendar.
III. Lecture thesis:
Immunoprophylaxis in Kazakhstan, as in all countries, occupies a special place. With the aim of the epidemic well-being of children and the general population, as well as to prevent infection, the **Vaccination Schedule** was developed and approved.
Immunity is a biological process that is formed at the cellular level after the introduction of a vaccine into the body. As practice shows, in people who have been infected and become ill, despite the presence of vaccinations, the disease is mild, and complications and deaths are rare.
Categories of vaccinations
All vaccinations are divided into 2 categories: **mandatory** and **voluntary**.

Mandatory preventive vaccinations within the guaranteed volume of medical care, in turn, consist of those vaccinations that are carried out at the expense of the republican budget and vaccinations at the expense of the local budget.

At the expense of the republican budget, children are given the entire a list of vaccines specified in the Vaccination Calendar of the Republic of Kazakhstan. But from the local budget, "Viral hepatitis" A "at 2 years old and influenza vaccination for children registered with dispensaries were added to the category of mandatory preventive vaccinations. (Decree of the Government of the Republic of Kazakhstan dated September 24, 2020 No. 612" On approval of the list diseases against which mandatory preventive vaccinations are carried out within the guaranteed volume of medical care ... ".

The amendments and additions made to the Code of the Republic of Kazakhstan "About people's health and the health care system" in 2020 came into force. Basic moments:

• The Code provides for the possibility of refusal of vaccinations by legal representatives of the child.

• The Code limits the admission of children who have not received routine vaccinations to preschool organizations.

"Admission to preschool organizations of children who have not received routine preventive vaccinations is carried out only when the threshold level of collective immunity in preschool education is reached."

"Collective immunity is an indirect protection of unvaccinated persons by reaching a threshold level of coverage with routine vaccinations of the population against vaccine-preventable infections."

This means that a threshold minimum percentage (number) of children who have vaccinations has been approved for preschool organizations. This is an indicator of collective immunity. If this indicator is not reached, the kindergarten is obliged to refuse to accept the unvaccinated child. For violation of the norm, consequences are provided. If the indicator is reached, then the preschool organization can accept unvaccinated children. Kindergartens need to monitor the herd immunity score, especially when dropping and enrolling children.

Routine vaccines of the Republic of Kazakhstan

According to the Decree of the Government of the Republic of Kazakhstan No. 2295 of 12/30/2009 and with amendments and additions of 02/12/2013 - planned preventive vaccinations for children are carried out in accordance with the National Vaccination Calendar of the Republic of Kazakhstan and in accordance with the approved sanitary and epidemiological requirements for conducting preventive vaccinations for the population - Order of the Minister Healthcare of the Republic of Kazakhstan No. 361 dated June 13, 2018.

National immunization calendar for children

- still in the maternity hospital, at **1 - 4 days of life** - are vaccinated against:
tuberculosis
B hepatitis.

- At **2 months**, babies receive a comprehensive vaccination against:
hepatitis,
poliomyelitis,
pertussis,
diphtheria,
tetanus,
hemophilus influenza type b,
pneumococcal infection

At **3 months** - repeated vaccination against:
pertussis,
diphtheria
tetanus,
poliomyelitis,
hemophilus influenza b.

At **4 months -** stage 3 vaccination against:
hepatitis,
poliomyelitis,
hemophilus influenza type b
pertussis,
diphtheria
tetanus,
and second vaccination against**pneumococcal infection**
At the age of **12-15 months** – against:
measles,
rubella
mumps,
repeat the vaccination against **poliomyelitis** and **pneumococcal infection.**

at 1.5 years - revaccination of:
pertussis,
diphtheria
tetanus,
poliomyelitis
hemophilic infection type b

7.At **2 years old -** vaccination against **viral hepatitis "A"**

8.at **2.5 years -** revaccination against **hepatitis "A"**

at school age:
- at the age of 6 years - revaccination against:
tuberculosis,
measles,
rubella
mumps
pertussis,
diphtheria
tetanus;

- 16 years old - revaccination(which in the future will need to be repeated every 10 years) against**:**
1) tetanus
2) diphtheria.

Both local and foreign vaccines are used for vaccination. The only condition is the availability of registration and certificate of the National Center for Expertise of Medicines. It is recommended to be vaccinated only in medical institutions, since all vaccines require special storage conditions.

Despite the fact that the Calendar of Vaccinations for Children in Kazakhstan provides for certain terms for carrying out certain vaccinations, the final decision on the time of vaccination is made by the local pediatrician. This is due to the peculiarities of the child's immune system: when the vaccine is administered, the body must be ready to develop the necessary antibodies in order to easily tolerate vaccination.
Otherwise (if the vaccination was made ahead of time), the child's body, due to the lack of formation of immunity, will not be able to properly respond to the vaccine.
Procrastination - puts the child at risk of catching the disease.

If the child missed the vaccination period, he will have to "catch up" with his peers according to the general rules. For example, those who do not have the hepatitis B vaccine are given the following procedure: the first procedure, the second in a month, and the third after 5 months.

How to prepare for vaccination:
There are no special requirements. Vaccinations usually do not require prior preparation.

How to get a vaccination permit:
A prerequisite for admission is that the child must be healthy.
Before the first vaccinationagainst **pertussis, diphtheria** and**tetanus,** it is necessary to consult a pediatric neuropathologist and obtain permission from a neuropathologist for vaccination, and also be sure to pass general urine and blood analyzes.
Before being vaccinated, the child must be examined by the attending pediatrician. Important are not only general clinical indicators (temperature, absence of complaints), but also the opinion of the mother - if the baby slept poorly, was capricious, there are changes in behavior, appetite, then it is better to postpone the procedure for now.
An additional safety net will be a general blood analyzes (special attention should be paid to ESR and platelets).

Who is prohibited from vaccinations?
Contraindications:

There are **temporary** (relative) and **permanent** (absolute) contraindications for vaccination.

Permanent contraindications:

vaccination against**pertussis, diphtheria, tetanus** - a history of convulsions, severe allergic diseases, complications to other vaccines, neurological pathology, detection of neoplasms in the body;
vaccination against**tuberculosis**- the presence of HIV infection in children, malignant blood diseases, primary immunodeficiency, neoplasms, as well as a strong reaction or complications with the introduction of other vaccines;
vaccination against**measles, mumps and rubella** - malignant tumor, blood diseases, neoplasms, allergy to aminoglycosides and quail eggs, strong reactions to other vaccinations, primary and severe immunodeficiency;
vaccination against**Poliomyelitis** - if children have HIV infection, primary immunodeficiency, complications associated with neurology, blood diseases and neoplasms in the body.

Temporary contraindications are exacerbation of acute chronic diseases, allergies, recent acute colds or intestinal infections. From the moment of recovery to vaccination, at least 14 days must pass. If agreed with the doctor, this period can be extended to 6 weeks or reduced to 1 week. Mild cough and runny nose are not considered contraindications.
Temporary contraindications for vaccination also include a deterioration in the child's well-being on the day of vaccination or during the previous few days. It can be high fever, SARS, runny nose. If a child has diabetes, then vaccination is carried out only after the conclusion of an endocrinologist.
Contraindications can only be during an exacerbation. During the onset of remission, need to be vaccinated immediately.
Even with mental illness, Down syndrome and cerebral palsy, during the period of remission or improvement in the condition, it is necessary to vaccinate. These children have very low immunity, they need to be strengthened.
In any case, the decision to withdraw from vaccinations: permanent or temporary - must be made by the doctor.
Refusal from routine vaccinations.

Are they admitted to school or kindergarten according to the law of the Republic of Kazakhstan?
According to the law of the Republic of Kazakhstan, parents under 18 years of age are responsible for the health of the child and have the right to independently decide whether to make routine preventive vaccinations for their child or refuse them or some.
Refusal of vaccination, a child who does not have a specific vaccination or vaccinations - has the right to attend school, kindergarten.

However, on July 17, 2020, amendments and additions made to the Code of the Republic of Kazakhstan "On people's health and the health care system" came into force.
• The Code provides for the possibility of refusal of vaccinations by legal representatives of the child.
• The Code limits the admission of children who have not received routine vaccinations to preschool organizations (EC). The term "collective immunity" is introduced.

Why? it is an increased responsibility for such a child. When an outbreak of an infectious disease begins in a children's institution, unvaccinated children are suspended from visiting until the disease stops. A child who has not received preventive vaccinations is not protected from infection, can quickly become infected, get sick and become a source of infection himself.
In addition, if an unvaccinated child gets sick, then more often the disease passes in a more severe form, with severe complications. Since the child has no immunity against a formidable infection.
There is a high risk of tetanus and rabies, when injured in violation of the skin and mucous membranes, and animal bites. Any child is not guaranteed against injury or an animal bite, which has a high risk of contracting these infections. Tetanus can only be protected from tetanus vaccination and timely emergency administration of sera.
Add to this the fact that in some countries entry is prohibited unless there are specific preventive vaccinations, according to international health regulations or international treaties.

If the parents still **refuse vaccinations**, then the refusal must be properly formalized so that in the future there are no problems with clinics, kindergartens and schools. At the clinic at the place of attachment of the child, you will need to fill out a form, where it will be written that the doctor acquainted you with the consequences of your decision, that you, having received full information, refuse to carry out preventive vaccination (name of the drug).

IV.Visual material: are in the appendix

V. Bibliography: at the end

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Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 15: Chronic eating disorders in children.
II. Learning goals: Diagnosis and treatment of chronic eating disorders in children on ambulatory level.
III. Lecture thesis:
Chronic nutritional disorders (dystrophies) have a significant proportion in the pathology of an early age and pose a serious threat to the health and life of the child due to a decrease in immunological

reactivity and the body's resistance to infection. Fasting, which took place at an early age in experimental animals, leads to a decrease in the mass of the brain substance, depletion of its cellular elements, and a decrease in nucleic acids and myelin. Therefore, severe malnutrition in early childhood can have long-term consequences, leading to a lag in the child's neuropsychic and intellectual development.
Chronic nutritional disorders - dystrophies (*distrophia; dis – disorders; trohpe - nutrition*) - is characterized by impaired absorption of nutrients by the tissues of the body. At the same time, eating disorders cannot be considered only as a result of improper feeding.

Classification

According to the classification of G.N. Speransky there are 4 types of dystrophies:

1. hypotrophy, proceeding with a decrease in body weight in relation to its length (or protein-energy deficiency);
2. parotrophy occurring with overweight;
3. hypostatura, proceeding with a proportional decrease in body weight and length;
4. polyhypovitaminosis.

HYPOTROPHY

Hypotrophy (**protein-energy malnutrition (PEM**)) is a chronic nutritional disorder accompanied by impaired metabolic and trophic functions of the body and characterized by a deficiency in body weight at close to average growth rates, decreased food tolerance and immunobiological reactivity. This condition is mainly observed in young children. age due to high growth rates and the activity of metabolic processes, requiring a sufficient intake of nutrients.
The term PEM was proposed in 1961 by the Joint FAO / WHO Expert Committee on Nutrition to denote such severe nutritional deficiency diseases as kwashiorkor or marasmus, as well as transitional states to them. PEM develops as a result of prolonged protein (kwashiorkor) and / or protein-energy (marasmus) starvation and is manifested by a deficiency in body weight and / or growth, complex disruption of homeostasis in the form of changes in basic metabolic processes, water-electrolyte balance, impaired nervous regulation, endocrine imbalance , suppression of the immune system, dysfunctions of the gastrointestinal tract and other organs and systems.

Etiology

There are 3 main groups of reasons leading to the development of malnutrition in children:
1.insufficient intake of nutrients (nutritional deficiency or difficulty eating);
2. violation of digestion and assimilation of food (malabsorption syndrome);
3. inadequate provision of increased requirements for nutrients (premature babies, congenital heart defects, chronic lung pathology, severe infections accompanied by catabolic stress, etc.).

By the time of occurrence, 2 groups of factors are distinguished:

Prenatal	**Postnatal**
a) gene and chromosomal mutations; b) malnutrition of the mother during pregnancy, causing starvation of the maternal organism; c) pathology of pregnancy (toxicosis of the first half of pregnancy, preeclampsia, placental abruption, diseases of a pregnant woman, etc.), leading to placental insufficiency, chronic fetal hypoxia, delayed development of some functional systems and the formation of metabolic processes.	**1. Exogenous factors:** **- alimentary:** quantitative and qualitative underfeeding (hypogalactia, poverty of mother's milk in constituent ingredients in case of improper nutrition of a nursing mother, insufficient volume of formula during artificial feeding, mainly one-sided feeding, etc.), improper introduction of complementary foods, as well as unbalanced and poorly organized nutrition; **-toxic:** hypervitaminosis D and A, massive and long-term drug therapy, unfavorable environmental conditions;

	-defects in care and organization of the environment: insufficient attention to the child and a negative emotional impact on him, non-observance of the daily regimen, neglect of the simplest hygiene procedures (timely change of diapers, daily bathing), as well as physical inactivity. **2.Endogenous factors:** **-** congenital and acquired lesions of the central nervous system; - malformations of the digestive system (pyloric stenosis, atresia of the biliary tract, etc.), heart, lungs, diaphragmatic hernia. - primary immunodeficiencies (hypogammaglobulinemia, deficiency of individual components of the complement system, hereditary pathology of phagocytosis); - hereditary metabolic anomalies (galactosemia, fructosemia, Niemann-Pick, Tay-Sachs, etc.); - malabsorption syndrome (lactase deficiency, celiac disease, cystic fibrosis, exudative enteropathy); - endocrine pathology (adrenogenital syndrome, diabetes mellitus, thyroid dysfunction, etc.); - severe acute infectious diseases, accompanied by intoxication, vomiting, frequent stools; - prolonged intoxication in severe chronic infectious diseases (tuberculosis, brucellosis, etc.) and purulent processes (abscesses, suppurating bronchiectasis, osteomyelitis); - severe non-infectious diseases (severe respiratory, heart failure, malignant tumor); - severe thermal and concomitant injury.

Classification of hypotrophy (according to E.V. Neudakhin, 2001)

Form	**Degree of hypotrophy depending on body weight deficit (%)**
Prenatal (intrauterine) **Potsnatal** (acquired)	I–10-20% II–20-30% III–30%и>

Prenatal hypotrophy

Intrauterine hypotrophy is diagnosed in newborns with insufficient birth weight in relation to their gestational age, that is, when the body weight is below 10% of the centile.

At the heart of prenatal malnutrition are violations of intrauterine development of the fetus due to insufficient placental circulation, exposure to infectious factors, hereditary and constitutional characteristics of the mother, as well as unfavorable socio-economic, production and environmental factors.

Clinically distinguish between mild, moderate and severe intrauterine malnutrition.
With a **mild degree**, the deficit in body weight in relation to body length is below 10% of the centile. There is a decrease in skin elasticity, thinning of the subcutaneous fat layer, tissue turgor is not changed or slightly reduced, muscle mass is preserved. The early neonatal period proceeds without complications, or with symptoms of excessive birth stress, mild metabolic disorders, signs of energy deficiency. Children tend to lose more of their original body weight and regain it slowly.
With a **moderate degree**, the deficit in body weight in relation to body length is below 5% of the centile. There is a significant decrease in elasticity, the skin is dry, peeling, and there may be cracks. The subcutaneous fat layer is absent on the abdomen, is thinned everywhere, tissue turgor is flabby, there are transverse folds on the limbs, muscle mass is reduced. The course of the early neonatal period is usually complicated: asphyxia or symptoms of chronic intrauterine hypoxia, sometimes convulsions, respiratory disorders, edema, hemorrhagic syndrome, muscle hypotension, hyporeflexia.
In severe cases, the deficit in body weight in relation to body length is below 1% centile. The skin is wrinkled, dry with lamellar peeling, often cracks, the subcutaneous fat layer is absent everywhere, the muscle mass is reduced. Turgor of tissues is flabby, there are folds on the face, buttocks, trunk, thighs. The course of the early neonatal period, as a rule, is complicated with the dominance of signs of damage to the brain, cardiovascular system, infection, often metabolic disorders, hemorrhagic syndrome.
In modern conditions, antenatal diagnostics of intrauterine hypotrophy is possible, which will make it possible to carry out therapeutic measures even before the birth of a child. Such diagnostics is carried out taking into account the history, features of the antenatal period, assessment of the weight gain of the pregnant woman, ultrasound examination, which will allow early diagnosis of the initial signs of slow fetal growth for correction.
Postnatal hypotrophy
There are three degrees of malnutrition.
The evaluation criteria are:
- underweight;
- the degree of depletion of the subcutaneous fat layer (determined by the thickness of the subcutaneous fat fold on the abdomen, thighs, face);
- the state of skin elasticity, tissue turgor, muscle mass;
- psychomotor development;
- immunological reactivity;
- change on the part of internal organs; - the nature of the chair.

Hypotrophy I degree
It is characterized by a 10-20% body weight deficit, a flattening of the weight curve, a thinning of the subcutaneous fat layer on the abdomen, a decrease in skin elasticity and soft tissue turgor. Psychomotor development is age appropriate, anxiety and frequent negative emotions are noted. Internal organs without pathology, stool is not changed. Children are prone to frequent illnesses.
Hypotrophy II degree
It is characterized by a body weight deficit of 20-30%, the weight curve is moderately flat, the subcutaneous fat layer is thinned on the abdomen and limbs. Skin elasticity is sharply reduced, tissue turgor is significantly reduced, muscle mass is reduced, there are signs of polyhypovitaminosis. There is a lag in psychomotor development, alternation of bouts of anxiety and depression, sleep disturbance, loss of appetite, growth retardation by 3-4 cm. Internal organs without pronounced deviations. The chair is unstable. Immunological protection is reduced, protracted infectious and inflammatory diseases, often with an atypical course, are characteristic.
Hypotrophy III degree
It is characterized by an extreme degree of exhaustion, the appearance of the child resembles a skeleton covered with skin. The body weight deficit is more than 30%, the weight curve is flat. The subcutaneous fat layer is depleted everywhere, including on the face, the skin hangs in folds. A sharp decrease in the turgor of soft tissues and muscle mass is noted, signs of polyhypovitaminosis (seizures and cracks in the corners of the mouth) are clearly expressed. Characterized not only by a lag in psychomotor development, but also by the disappearance of previously acquired skills. The child is depressed, indifferent to others,

paradoxical reactions are noted, lags behind in growth by 5-6 cm. There are changes in the internal organs: superficial arrhythmic breathing, muffling of heart sounds, tachycardia. The stool is hungry, scanty, lumpy with a putrid and fetid odor. The immunological reactivity of the body is sharply reduced, severe toxic-septic conditions are characteristic, often with a fatal outcome.

Diagnostic criteria for hypotrophy

1. Anamnestic.

Allow to identify etiological and predisposing factors of the disease: congenital malformations, enzymopathies, neuroendocrine disorders, alimentary (quantitative deficit or qualitative imbalance of the daily diet, violation in the organization of the feeding regimen), infectious (acute and chronic diseases, including intestinal infections), improper care for the child (poor emotional impact, insufficient stay in the fresh air, lack of bathing, massage and gymnastics, etc.), toxic (hypervitaminosis D, massive and prolonged drug therapy, unfavorable environmental conditions).

In the diagnosis of prenatal hypotrophy, an important place is occupied by the study of the course of the antenatal period, hereditary burden.

2. Anthropometric methods with the calculation of body mass index (the ratio of body weight in kg to the square of height in m^2) and Z-score - the deviation of the values of an individual indicator (body weight, height) from the average value for a given population, divided by the standard average value.

In the standard population, the mean Z-score is zero with a standard deviation of 1.0. Positive Z-score values indicate an increase in the anthropometric indicator compared to the standard, and negative values indicate a decrease in parameters compared to the standard value.

3. Clinical.

A) Syndrome of trophic disorders: body weight deficit, to a lesser extent - body length, trophic changes in the skin (violation of elasticity, signs of polyhypovitaminosis), sequential depletion of the subcutaneous fat layer (first on the abdomen, then on the limbs and face), decreased turgor of soft tissues , amyotrophy.

B) Symptoms of changes in the functional state of the central nervous system: disturbance of emotional tone and neuro-reflex excitability, muscle hypotension, retardation of psychomotor development, sleep disturbance, thermoregulation.

C) Syndrome of decreased food tolerance: decreased appetite up to anorexia, flattening and the wrong type of weight curve, the development of dyspeptic disorders (regurgitation, unstable stool).

D) Syndrome of decreased immunobiological reactivity: a tendency to frequent infectious and inflammatory diseases, their atypical course, the development of toxic-septic conditions, dysbiocenoses and secondary immunodeficiencies.

4. Paraclinical:

a) blood analysis - anemia, with severe malnutrition, ESR is slowed down;

b) biochemical blood analysis - hypo- and dysproteinemia, dyslipidemia;

c) coprogram - signs of insufficiency of gastric, small and large intestinal digestion and absorption;

d) analysis of feces for intestinal biocenosis - signs of intestinal dysbiosis.

e) comprehensive immunological examination - partial immunodeficiency, dysimmunoglobulinemia, a decrease in the indices of nonspecific resistance - lysozyme, bactericidal activity of the skin and mucous membranes, phagocytic activity of neutrophils.

Diseases with hypotrophy syndrome

• Syndrome of malabsorption (celiac disease, lactase deficiency, cystic fibrosis, exudative enteropathy).
• Hereditary metabolic defects (galactosemia, fructosemia, glycolipidosis).
• Hypotrophy with malformations (pyloric stenosis, biliary atresia).
• Hypotrophy against the background of endocrine diseases (adrenogenital syndrome).
• Hypotrophy against the background of primary immunodeficiencies.

Treatment of hypotrophy

Disease therapy should be comprehensive and include:

1) identification of the causes of malnutrition, their correction and elimination;

2) organization of a rational daily routine and child care;

3) diet therapy, taking into account the etiology of the disease;

4) drug treatment (enzyme therapy, vitamin therapy, stimulating therapy, etc.);
5) identification and rehabilitation of foci of infection, correction of anemia and other concomitant diseases.

Organization of the regime

The child should be in a bright, spacious, regularly ventilated room, preferably in an insulated box with a germicidal lamp irradiation. The air temperature in the ward should not be lower than 24 and not higher than 26 ° C. If there are no contraindications to walking, you should walk several times a day at a temperature not lower than -5 ° C. It is obligatory to carry out massage, gymnastics, exercise therapy.

Optimal diet therapy

It is necessary to strive to meet the age-related needs of the child in basic nutrients, energy, macro- and micronutrients by gradually increasing the nutritional load, taking into account the child's tolerance to food.

In building a diet for hypotrophy of II-III degrees, the principle of multiphase nutrition is applied:
- adaptation - the period of clarification of food tolerance;
- reparation - including a transitional period;
- a period of increased nutrition.

During the period of clarification of food tolerance, they resort to diet rejuvenation - they use breast milk or adapted milk formulas, increasing the frequency of feeding up to 7-10 times a day. In the most severe cases, a period of continuous tube enteral nutrition is used in combination with partial parenteral nutrition. Further, complementary foods are sequentially introduced, gradually increasing their volume. It is recommended to use commercially produced complementary foods.

When building a diet, you must follow a number of principles:
- use only easily digestible food at the initial stages of treatment (breast milk, and in its absence, adapted formulas);
- more frequent feedings from 7 to 10 times a day;
- adequate systematic control of nutrition with the maintenance of a food diary, where the volume of food eaten at each feeding, the nature of the stool, diuresis, the amount of liquid drunk and parenterally administered is noted;
- Regular calculation of the food load for the main ingredients - once every 7 days.

With **hypotrophy of the I degree**, it is necessary to eliminate feeding defects. Preference should be given to breast milk, and with mixed and artificial feeding, adapted milk formulas enriched with probiotics and prebiotics, which favorably affect the digestion processes and normalize the intestinal microflora, as well as nucleotides that improve the absorption of nutrients and stimulate the child's immune system. Unadapted fermented milk products (kefir, etc.) should not be prescribed to children earlier than 8 months. To increase the energy value of the diet and correct protein, foods and complementary foods (cereals, cottage cheese, meat puree) are prescribed 2 weeks earlier than healthy children. Calculation and correction of nutrition is carried out for the required weight.

In case of **hypotrophy of the II degree** during the adaptation period, which lasts 3-5 days, the calculation of nutrition is carried out on the actual body weight (Table 4). The amount of food is ½ of the norm. The number of feedings increases by 1-2 per day with a decrease in the volume of each feed, additional liquid is introduced (5% glucose solution, saline solutions for oral rehydration). During this period, it is preferable to use breast milk, and in case of its lack or absence, adapted mixtures enriched with probiotics, oligosaccharides and nucleotides. It is recommended to use a mixture with a higher protein content.

With normal tolerance of the prescribed nutrition, gradually over the course of a week, the amount of feeding is increased to the physiological norm. In the reparative period, with a sufficient rate of weight gain, the absence of dispatching phenomena, the calculation of nutrition is carried out for the required weight, first of the protein, carbohydrate component and, last of all, fat.

During the period of increased nutrition, complementary foods are introduced, starting with cereals of industrial production, followed by the introduction of cottage cheese, meat, yolk.

With **hypotrophy of III degree**, all types of metabolism are sharply disturbed, the condition of the child is usually very serious, therefore, such children need to use parenteral and enteral nutrition, which

requires inpatient treatment. Degree III hypotrophy caused by alimentary factors is extremely rare and develops in severe somatic pathology. Therefore, the diagnosis and treatment of the underlying disease leading to the development of malnutrition is a fundamental factor in the treatment of this pathology. Parenteral nutrition of the initial period should be carried out gradually using exclusively amino acid preparations and glucose solutions. Fat emulsions are added only after 5-7 days from the start of therapy due to their insufficient absorption and a high risk of side effects. Parenteral nutrition should be minimal and balanced due to the development of serious metabolic disorders. In parallel, correction of dehydration, acidosis and electrolyte disturbances is carried out.

Prophylaxis

Antenatal prophylaxis.

Includes maternal health care. Preventive measures are aimed at the timely detection and treatment of a woman's diseases, at the prevention and treatment of pregnancy toxicosis, as well as the organization of the correct diet for a pregnant woman. Preventive measures should be carried out by antenatal clinics in conjunction with children's clinics.

Postnatal prophylaxis.

Postnatal prophylaxis should be aimed at organizing optimal conditions of the external environment, nutrition and creating a physiological and sanitary-hygienic environment and ensuring the daily regimen of the mother and child.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:

Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 16: Neonatal jaundice.

II. Learning goals: Diagnosis and treatment of neonatal jaundice on ambulatory level.

III. Lecture thesis:

Neonatal Jaundice, also known as **icterus,** is the appearance of a visible icteric coloration of the skin and mucous membranes of the newborn due to an increase in the level of total bilirubin in the blood serum.

Jaundice becomes visible when the serum total bilirubin (SBB) level reaches 80 μmol / L.

Clinical classification

Physiological jaundice of newborns:

• Usually appears after 36 hours of a child's life.
• Peak serum total bilirubin levels usually occur on days 3–4 in a term baby and on days 5–7 in a premature baby.

• The peak serum total bilirubin level is ± 205 mkmol / L (12 mg / dL).

• The level of total serum bilirubin decreases in a full-term baby by the 14th day of life, and in a premature baby by the 21st day of life.

• The child's clinical condition is satisfactory: he is active, has clearly distinguishable periods of sleep and wakefulness, a good sucking reflex; the child has a stable temperature; the liver and spleen are of normal size, the urine is light, the stool is normally colored.
• Jaundice associated with breastfeeding may be accompanied by two peaks in the rise in bilirubin, the first at 4-5 days and the second at 14-15 days, decreases slowly and can be detected at 12 weeks of age.

Pathological jaundice of newborns:

Jaundice onset within the first 24 hours of life or on the feet and palms at any time.

• Jaundice that appears after the 7th day of life.
• The rate of increase in total serum bilirubin> 85 μmol / L / day or 5 mg / dL / day.

• Direct bilirubin level> 34 μmol / L (2 mg / dL), or ³ 20% of the total serum bilirubin level.

• The child's condition is unsatisfactory, regardless of the level of bilirubin, the presence of pathological clinical symptoms.

• Hepatomegaly and / or splenomegaly.
• Discolored stools or dark colored urine.
• Jaundice without a tendency to decrease after 14 days of life in a full-term newborn or after 21 days of life in a premature newborn is called “lingering jaundice”.

Diagnostic measures

The main diagnostic measures:
A. In the antenatal period, determine the blood group and Rh factor in all pregnant women, as well as the presence of a burdened transfusion and vaccination history.

B. At birth:

• In a child from a mother with Rh-negative affiliation in the umbilical cord blood, determine the group and Rh-affiliation, Coombs' test;

• In a child born to a mother with a blood group O (1) Rh-positive, determine the blood group and Coombs' test in the umbilical cord blood.

• The level of total bilirubin in the umbilical cord blood and its fraction (if necessary) in dynamics.

Additional diagnostic measures:
A. If you suspect the presence of neonatal jaundice caused by excessive hemolysis (R 58-58.9) - determine the level of hemoglobin, hematocrit, the number of erythrocytes, reticulocytes, microcytes.

B. If you suspect the presence of jaundice in hypothyroidism, examine the level of thyroid hormones.
C. If you suspect the presence of hereditary pathological jaundice:

• with genetic testing;
• hereditary hemolytic anemias (membranopathies, hemoglobinopathies, enzymatic deficiency anemias, etc.) - examine a smear of peripheral blood with a Price-Jones curve, calculate the thickness, sphericity index, average volume of erythrocytes and average concentration of hemoglobin in erythrocytes, osmotic resistance.

Diagnostic criteria:

Complaints and anamnesis

Medical history.

• Rh-affiliation and blood group of the mother.

• Infections during pregnancy and childbirth.

• Hereditary diseases (G6PDG deficiency, hypothyroidism, other rare diseases).
• Parents have jaundice.

• Jaundice in a previous child.

• Weight and gestational age of the baby at birth

• Feeding the baby (underfeeding and / or vomiting).

• Whether the child received resuscitation at birth.

Risk factors:

• Acute hemolysis.

• Premature birth.

• Bruising and cephalohematomas.
• Decrease in body weight (> 10%.)

• Neonatal asphyxia, acidosis.

• Hypoglycemia.

• Neonatal infection.

• Hypoalbuminemia (<30 g / l).

Physical examination

• Assess the color of the skin.

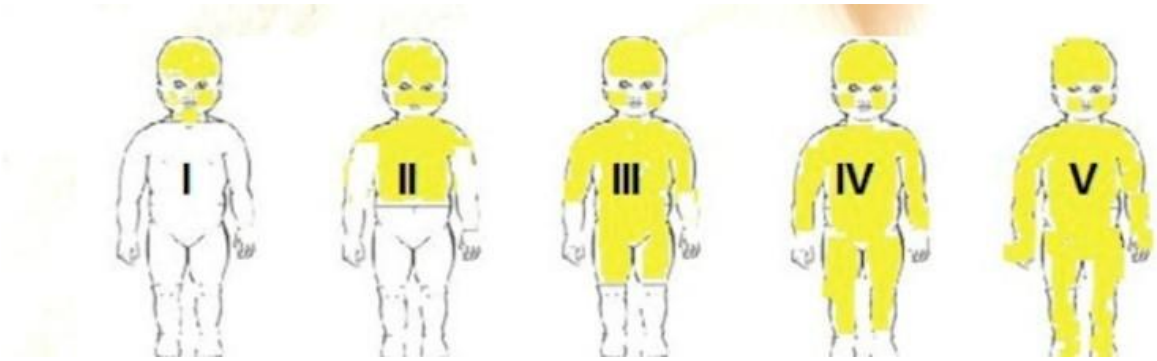
• Examine a fully undressed child in good lighting (preferably daylight).
• Provide the child with thermal protection during the examination.

• Assess the color of the skin after light finger pressure to the level of subcutaneous fat.

• Determine the localization of jaundice according to the modified Kramer scale.

	Zone	**Jaundice**	**Indirect plasma**

			bilirubin, on average
	1	**limited to the head and neck**	**100**
	2	**including the upper torso**	**150**
	3	**including the lower torso**	**200**
	4	**including arms, legs below the knees**	**250**
	5	**hands, feet**	**>250**



Laboratory researchmethods:
There are various causes of pathological jaundice, therefore, it is necessary to determine, in addition to total bilirubin in the blood, the fraction of bilirubin (direct /conjugated bilirubin and indirect / non-conjugated bilirubin).

For newborns with jaundice and persistent hyperbilirubinemia that lasts more than 3 weeks, a galactosemia test is recommended.

If there is a family history of jaundice associated with G6PDH deficiency, the level of this enzyme should be determined.

Newborns with high levels of direct bilirubin should be screened for sepsis (laboratory and bacteriological blood tests).

Newborns with elevated levels of direct bilirubin and hepatomegaly need to measure the level of transaminases ALT and AST to exclude hepatitis.

Differential diagnosis

In the overwhelming majority of cases, neonatal jaundice is physiological, but it is very important to control each case for the timely detection and appropriate treatment of "dangerous" jaundice in order to prevent complications:
- bilirubin encephalopathy: acute lesion of the central nervous system.
- kernicterus: irreversible chronic damage to the central nervous system.

• For early detection of signs of dangerous jaundice, it is important to record the time of onset of any jaundice and its regular clinical assessment (with an interval of 8-12 hours).

• Jaundice after day 7 may be a sign of cholestasis, glucose-6-phosphate dehydrogenase (G6PDH) deficiency, Crigler-Najjar disease, or late neonatal sepsis.

• Dangerous jaundice appears within the first 24 hours of life or is defined on the feet and palms at any time.

Treatment:

Non-drug treatment:

A. Physiological jaundice

Children with "**physiological jaundice**" do not need special treatment, but they do need exclusive breastfeeding. It is necessary to explain to the mother of the child that exclusive breastfeeding (as often and for as long as the child wants, day and night, but at least 8-12 times a day) helps the child to overcome jaundice faster.

Pathological jaundice

• **Phototherapy** is the most effective method for reducing
bilirubin levels in case of neonatal jaundice.
Timely and correct phototherapy reduces the need for replacement blood transfusions by up to 4% and reduces the risk of complications of neonatal jaundice.

Indications for phototherapy and replacement blood transfusion in a full-term newborn:

- If jaundice appears in the first 24 hours after birth, or it is "dangerous" ─ phototherapy should be started immediately and the indication for replacement blood transfusion should be considered.

- In other cases, decide whether to start phototherapy.
- If risk factors are present, the decision to initiate phototherapy should be made on the basis of a lower total serum bilirubin level.

- If the child's general condition worsens, phototherapy should be started with lower total serum bilirubin levels.

The indications for starting phototherapy in a premature baby are the same as for newborns with hemolytic disease.

- Phototherapy is performed using an incubator or in a warm bed.

- The child's eyes must be protected by an opaque bandage.

- Monitor the baby's body temperature and the air temperature under the lamp every 3 hours. Maintain body temperature at 36.5–37.5 ° C.

- Turn the baby over every 3 hours.

- Weigh your child at least once a day.

- During phototherapy, your child's stool may be liquid and yellow. This is normal and does not require treatment.

- Continue breastfeeding on demand, without night breaks, at least 8 times a day.

- If the baby receives expressed breast milk or intravenous fluids during phototherapy, it is recommended to increase the volume of milk / fluids to 10% of the total daily requirement.

- Determine the level of total serum bilirubin every 12 hours.

- Phototherapy in a newborn is discontinued in the event of a steady decrease in the concentration of serum bilirubin within 24-36 hours below the values that became the basis for starting phototherapy and the presence or absence of risk factors.

Indications for replacement blood transfusion:

• If phototherapy fails, replacement blood transfusion is recommended.
• In the event of clinical symptoms of acute bilirubin encephalopathy (muscle hypertonicity, opisthotonus, fever, "brain" cry), replacement blood transfusion is performed regardless of the bilirubin level.

• Replacement blood transfusion in case of hemolytic disease of newborns caused by isolated Rh-conflict, Rh-negative erythrocyte mass and plasma, one-group with the child's blood, and plasma are used, but, if possible, AB (IV) blood groups in a ratio of 2: 1.

• Replacement blood transfusion in case of incompatibility between the mother's blood and the child's blood due to rare factors, it is necessary to use blood from individually selected donors.

It must be remembered that for children with hemolytic disease of newborns, erythromass is used with a shelf life of no more than 72 hours.

Replacement blood transfusion, which is an unsafe procedure, should only be performed by trained personnel on strict indications following phototherapy.

Indications for hospitalization:
• hospitalization of a pregnant woman in an obstetric organization of any level in connection with the onset of labor;

• planned hospitalization of a pregnant woman with Rh-negative blood, with blood group O (1) Rh-positive, with a burdened transfusion and vaccination history in the obstetric organization of the 3-4th levels;

• emergency hospitalization of a newborn with pathological jaundice.

Discharge criteria:

• Discharge of a newborn with jaundice from the hospital should be carried out individually, taking into account the severity of jaundice and the results of treatment.

• Each mother / family needs to be trained on how to assess jaundice and how to recognize “danger signs” in a baby, and how to exclusively breastfeed.

Preventive measures:
to prevent Rh sensitization in subsequent pregnancies, women with Rh-negative blood factor who have given birth to a Rh-positive baby should be administered anti-Rh immunoglobulin in the first 72 hours after childbirth.

Treatment effectiveness indicators:

• Normalization of the level of bilirubin in the blood and prevention of bilirubin brain damage in the child.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
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Prevention

I. Theme № 17: Allergies in children.
II. Learning goals: Diagnosis and treatment of allergies in children on ambulatory level.
III. Lecture thesis:
Allergy is a state of increased sensitivity of the body in relation to a certain substance (allergens), which develops with repeated exposure to these substances, that is, a pathologically increased and perverse reaction of the body to certain substances of an antigenic nature, leading to its self-harm.

Classification
There is no single classification of allergens, but, as a rule, allergens are classified according to their origin and pathways of entry into the human body:
1. Household allergens - house and library dust, house dust mites, warehouse mites.
2. Pollen allergens - pollen of trees (birch, oak, etc.), meadow grasses (meadow fescue, hedgehog, bluegrass, etc.), weeds.
3. Animal allergens - allergens of dandruff, secretions (urine, saliva, secretions of sebaceous and sweat glands) and animal hair, more often cats, dogs, guinea pigs, horses, hamsters.
4. Fungal allergens - Alternaria, Cladospoium, Pleurotus, Penicillium, Aspergillus, etc. entering the human body both by inhalation and enteral.
5. Insect allergens - the poison of wasps, bumblebees, bees, hornets, horseflies, gadflies, mosquitoes.
6. Food allergens - more often glycoproteins, less often polypeptides and haptens. The most allergenic properties are citrus fruits, honey, nuts, seafood, tomatoes, chicken eggs, cow's milk, etc.
7. Medicinal allergens are divided into full-fledged allergens (insulin, tetanus toxoid) and haptens, which include most drugs or products of their metabolism (antibiotics of the penicillin group, sulfonamides).
8. Industrial allergens - turpentine, oils, nickel, chromium, arsenic, tar, resins, tannins, dyes, tannin, pyrogallol, varnishes, insectofungicides, phenoplasts, aminoplasts, formalin, urea, epoxy resins and other substances.

Classification of allergic reactions
Allergic reactions are immediate, delayed and mixed. In the pathogenesis of immediate-type allergic reactions, A.D. Ado distinguishes three stages: immunological, pathochemical and pathophysiological. In this case, allergic reactions by the mechanism of development are divided into several types.

Types of allergic reactions.
1. Allergic reaction of type I (reaction of immediate type, reaginic, anaphylactic, atopic type). It develops with the formation of antibodies (AB) - reagins, belonging to the class IgE and lgG4. They are fixed on mast cells and basophilic leukocytes. When reagins are combined with an allergen, mediators are released from these cells: histamine, heparin, serotonin, platelet-activating factor, prostaglandins, leukotrienes, etc., which determine the clinical picture of an immediate allergic reaction. After contact with a specific allergen, clinical manifestations of the reaction occur in 15-20 minutes.
2.Allergic reaction of type II (cytotoxic type). The type is characterized by the fact that AB are formed to tissue cells and are represented by IgG and IgM. This type of response is only triggered by ABs capable of activating complement. AB connects with the modified cells of the body, which leads to the reaction of complement activation, which also causes damage and destruction of cells, followed by phagocytosis and their removal. It is by the cytotoxic type that drug allergy develops.
3.Allergic reaction of type III (tissue damage by immune complexes, immunocomplex type). It arises as a result of the formation of circulating immune complexes, which include IgG and IgM. ABs of this class are called precipitating, since they form a precipitate attached with AG. This type of reaction is leading in the development of serum sickness, allergic alveolitis, drug and food allergies, in a number of autoallergic diseases.

R**isk factors for allergic diseases.**
With all the diversity of views on the causes of the development of allergic diseases among the population in practice, from the point of view of evidence-based medicine, their list is rather limited and needs further study.
1**. Genetic predisposition.** The presence of an atopic disease in the family is considered a genetic factor of predisposition to the development of atopic diseases. 2. Active and passive smoking, especially during pregnancy (A), prolonged active contact with various external and internal air pollutants (B).
3. Infringement of the feeding of the child (especially in the risk group AD: use of cow's milk in early childhood or standard milk formula in the AD risk group (A), lack of breastfeeding (A)), unbalanced nutrition.
4. Inadequate drug therapy.
5.Cesarean section subsequently affects the increased risk of developing AD in children (B).
6. Unfavorable environmental situation and occupational hazards at work (D, C).
7.Frequent infectious diseases and chronic inflammatory diseases (C), etc.

Diagnostics of allergic diseases.
Examination of a patient suffering from allergic diseases includes.
I. By questioning collecting an allergic history.
II. Physical examination.
III. Laboratory and instrumental examination:
1. General clinical methods.
2.Specific allergological examination.

Allergic history
The main purpose of collecting an allergic history is to establish the allergic nature of the disease and the presumptive nosological form. When collecting it, you need to find out the following:
1. To establish a hereditary predisposition to AD;
2. To identify the relationship between environmental factors and the development of the disease;

3. Determine allergens that could cause the onset of this disease and identify diagnostically significant;
4. What diseases were noted in the patient's family;
5. How the patient reacts to the intake of food and drugs;
6. Whether the seasonality of the disease, its connection with the flowering of plants, moving from place to place, with infectious diseases have been noted;
7. Identification of other allergic diseases in the patient;
8. Where and when there are exacerbations of an allergic disease and how they are stopped;
9. Housing and working conditions;
10. Food regimen;
11.Analysis of the course of pregnancy (gestosis, smoking, etc.);
12.Previously conducted laboratory examinations;
13. To evaluate the clinical effect of antiallergic agents and / or elimination of an allergen, etc.

Physical examination.
Physical examination is carried out according to the standard examination scheme for a therapeutic patient:
inspection,
palpation,
percussion,
auscultation.
This allows you to assess the general condition of the patient, to identify violations of the patient's organs and systems. At the same time, special emphasis is placed on the examination of the skin, respiratory organs, mucous membranes, and lymphoid organs. The rest of the examination is identical to that of a therapeutic patient.

Laboratory and instrumental examination.
General clinical examination
Patients suffering from AD should be carefully examined in order to conduct differential diagnostics, assess the severity and select the volume of treatment and prophylactic measures.
For this, the following methods are usually used:
1 General blood analysis;
2. General urine analysis;
3. Biochemical blood analysis;
4.Cytological and bacteriological examination of secretions of the nose, pharynx, etc ;
5. General analysis of sputum;
6. Virological examination;
7. Parasitological examination;
8. Coprogram;
9.Ultrasound of the abdominal cavity;
10.Hormonal examination;
11.Rheumatological tests;
12. If necessary, assessment of the function of the respiratory system;
13. Consultations of related specialists.

Specific allergy examination
1. Skin testing.
2. Prick and Scarification Testing.
3. Provocative tests
4. Elimination test

Principles of the treatment of allergic diseases.
There are several basic principles of AD treatment:

1. Elimination measures.
2. Pharmacotherapy.
3. Allergen-specific immunotherapy.
4. Education of the patient.

Elimination activities
Elimination measures in allergists play an almost central role. Completely excluding the allergen from contact with the patient is the first thing to start with treatment measures (A). Below are the data on the reasonable features of limiting contact with allergens of various groups.

Household sensitization
In this case, it is necessary to limit contact with household dust, library dust and especially house dust mites (A).

Food sensitization
In case of food allergy, it is necessary, first of all, to exclude from the contact a food product for which there is a sensitization, as well as products that have cross-reactions with it with proven clinical significance.

Patient education.
As part of patient education, it is necessary to ensure the participation of patients or patients at risk of developing AD in special educational programs. This will allow listeners to get acquainted with the causes, development mechanisms, principles of diagnosis, treatment and prevention of AD, as well as improve mutual understanding in the doctor-patient system. Forms of education can be full-time (allergy schools, patient associations) or correspondence (brochures, information sheets, etc.).

Pharmacotherapy of allergic diseases.
Antihistamines
Antileukotriene drugs
Glucocorticosteroid drugs
Immunosuppressive therapy

Prevention of allergic diseases.
Primary prevention.
1. Breastfeeding at least up to 4-6 months of age, and if it is impossible to carry out it, in children with a high risk of AD, use hydrolysis mixtures (A) in the diet, and not soy mixtures due to the risk of developing allergic reactions to them (B).
2. During pregnancy and lactation, a balanced diet containing all the necessary nutrients is recommended (A).
3. Exclusion of active and passive smoking (A), as well as limiting contact with other external and internal air pollutants, such as exhaust gases (B).
4. It is not recommended to keep animals, especially cats, in an apartment where a child with a high risk of developing AD (B) lives.
5. Carrying out organizational and methodological work among the population, especially pregnant women, people with a high risk of AD, as well as health workers (A).
6. It is recommended to reduce body weight for overweight children to prevent the development of allergies (A).
7. Elimination of contact with professional sensitizers and irritants from the first month of pregnancy (A).
8. Maintaining a low humidity in the room (B) and reducing air pollution in the room (C).

The measures of secondary and tertiary prevention of AD include the timely elimination of identified allergens from contact, observation by an allergist-immunologist, regular examination of the patient, and treatment of the patient taking into account the recommended standards for each nosological form.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 18: Pneumonia in children.
II. Learning goals: Diagnosis and treatment of pneumonia in children on ambulatory level.
III. Lecture thesis:
Definition:Pneumonia in children is an acute infectious and inflammatory disease of the lung parenchyma, clinically manifested by respiratory disorders of varying severity, fever, intoxication, characteristic physical changes and radiological evidence of "fresh" focal-infiltrative changes in the lungs.

Classification:
1. by occurrence:
- community-acquired;
- nosocomial;
- intrauterine, in children with immunodeficiency;
- aspiration in children with encephalopathies;

2.forms:
- focal,
- segmental;
- croupous;
- interstitial;

3.course:
- acute;
- protracted;

4. by severity:
- mild;
- severe;
- very severe;

5.complications:
- respiratory failure I-III;
- pulmonary (pleurisy, abscess, bullae, pneumothorax, pyopneumothorax);
- extrapulmonary (toxicosis, neurotoxicosis ...).

Risk factors:
the most common causative agent of Streptococcus pneumoniae;
prematurity;
severe malnutrition;

immunodeficiency states;
congenital developmental anomalies;
recent upper respiratory tract infection;
foreign body;
low socio-economic status;
tobacco smoke;
pre-pubertal age.

Diagnostic criteria:

Complaints: cough

Physical examination
1.Fortened or labored breathing:
up to 2 months> 60 per minute;
from 2 months-12 months> 50 per minute;
12 months to 5 years> 40 per minute;
over 5 years, more than 28 per minute;
2. retraction of the lower chest;
3. fever;
4. grunting breathing (in infants);
5. auscultatory signs (weakened or bronchial breathing, fine bubbling rales, pleural friction);
6. symptoms of intoxication;

Laboratory research
- Complete blood count (leukocytosis with neutrophilic shift to the left, leukopenia, acceleration of ESR)

Instrumental research
X-ray data ("fresh" focal-infiltrative changes in the lungs).

Indications for specialist consultation
- a pulmonologist with a protracted course or the presence of asthmoid breathing

Differential diagnosis

DIAGNOSIS or cause of the disease	In favor of the diagnosis
Pneumonia	-Cough and rapid breathing: age <2 months> 60 / min age 2 - 11 months> 50 / min age 1 - 5 years> 40 / min - Retraction of the lower chest - Fever - Auscultatory signs - weakened breathing, wet wheezing - Inflating the wings of the nose - Grunting breathing (in young infants)
Bronchiolitis	- Astmoid breathing during the period of seasonal increase the incidence of bronchiolitis - Expansion of the chest - Long expiration - Auscultatory - weakened breathing

	- Weak or no reaction to bronchodilators
Tuberculosis	- Chronic cough (> 30 days); - Poor development / underweight or weight loss; - Positive Mantoux reaction; - Contact with a patient with tuberculosis in history - Radiological signs: primary complex or miliary tuberculosis - Detection of mycobacterium tuberculosis in the study sputum in older children
Pertussis	- Paroxysmal cough accompanied by characteristic convulsive wheezing inhalation, vomiting, cyanosis or apnea; - Well-being between coughing fits; - Lack of fever; - Lack of DPT vaccination in history.
Foreign object	- Sudden development of mechanical obstruction of the airways paths (the child "choked") or stridor - Sometimes asthmoid breathing or abnormal expansion of the chest on one side; - Air retention in the airway with amplification percussion sound and mediastinal displacement - Signs of lung collapse: impaired breathing and dullness of percussion sound - Lack of reaction to bronchodilators
Pleural effusion / empyema	- "Stone" dullness of percussion sound; - Lack of breathing sounds
Pneumothorax	- Sudden onset; - Tympanic sound with percussion on one side chest; - Displacement of the mediastinum

List of main diagnostic measures:
1. General blood analysis (6 parameters);
2. General urine analysis

List of additional diagnostic measures:
X-ray examination of the chest.

Treatment tactics
Goals of treatment
1. relief of the inflammatory process in the lungs;
2. elimination of Respiratory Insufficient symptoms, general intoxication;
3. restoration of lung excursion;
4. the disappearance of cough, rapid breathing, pneumonia auscultatory data;
5. improvement of health and appetite.

Non - drug treatment
1. For the period of temperature rise - bed rest.

2. Adequate hydration (plenty of warm drinks)
3. Promotion of breastfeeding and adequate nutrition according to age

Drug treatment
Adequate hydration (plenty of warm drinks) and nutrition,
Relief of fever (> 38.5) - paracetamol 10-15mg / kg up to 4 times a day,

It is not recommended to use antitussive drugs,

In the presence of asthmoid breathing, bronchodilator therapy (for example, salbutamol in age dosage 3-4 times a day for 5 days). Preference is given to inhalation forms.

Antibacterial therapy:
Antibiotics are prescribed empirically with preferential use oral forms. Selection of antibacterial agents according to the sensitivity of the flora in vitro is carried out only if empirical tactics are ineffective.

Drugs of choice are semi-synthetic penicillins, macrolides,
Alternative - cephalosporins II-III generation.
Amoxicillin 15 mg / kg x 3 times a day for 5 days, or protected penicillins (amoxicillin + clavulanic acid 20-40 mg / kg 3 a day)
Azithromycin 10mg / kg 1 day, 5mg / kg daily for the next 4 days orally
or clarithromycin - 15 mg / kg orally for 10-14 days in divided doses
or erythromycin - 40 mg per kg orally for 10-14 days in divided doses. Ambroxol syrup 1mg / 5 ml; 30mg / 5ml; 7.5mg / ml solution.
Cefuroxime 40 mg / kg / day, divided into 2 divided doses, 10-14 days orally, in cefuroxime maximum dose in children 1.5 g
Ceftazidime 1-6 g / day-10 days

For the treatment and prevention of mycosis with prolonged massive antibiotic therapy **itraconazole** oral solution at the rate of 5 mg / kg / day, children over 5 years old.

Indications for hospitalization
1. an increase in respiratory failure with retraction of the lower part of the chest and increased breathing;
2. central cyanosis;
3. convulsions;
4. severe toxicosis (refusal to eat and drink, breastfeeding, impaired consciousness);
5. ineffectiveness of outpatient treatment.

Preventive actions
1.limitation of contacts with patients and virus carriers, especially during the seasons increasing respiratory morbidity.
2. wearing masks and washing hands of family members with ARVI disease.
3.Maintaining optimal air conditions in the room
4.carrying out hardening activities

Further management
1. Re-examination by the local doctor after 2 days or earlier if the child has become worse or unable to drink or suckle, has a fever, increased or shortness of breath (to teach the mother in what situation it is urgently necessary again consult a doctor).

2. Children who have had pneumonia are under dispensary observation for 1 years (examinations are carried out after 1, 3, 6 and 12 months).

Treatment effectiveness indicators:
1. Normalization of respiratory rate
2. Disappearance of fever
3. Positive percussion and ascultative dynamics
4. The disappearance of intoxication

List of essential medicines:
1. Paracetamol 200 mg, 500 mg table; 2.4% syrup in a bottle; 80 mg suppositories;
2. Amoxicillin 500 mg, 1000 mg table; 250 mg, 500 mg capsule; 250 mg / 5 ml
Oral suspension;
3. Amoxicillin + clavulanic acid, coated tablets 500 mg / 125 mg, 875 mg / 125 mg, powder for solution for intravenous administration in vials 500mg / 100mg, 1000mg / 200mg;
4. Cefuroxime 250 mg, 500 mg tablet; 750 mg in vial, powder for cooking injection solution;
5. Ceftazidime - powder for solution for injection in a bottle of 500 mg, 1g, 2g
6. Azithromycin 125 mg, 500 mg tablets; 250 mg capsule
7. Ambroxol syrup 1 mg / 5 ml; 30mg / 5ml; 7.5mg / ml solution.
8. Itraconazole oral solution 150 ml - 10 mg / ml.

List of additional medicines:
1. Salbutamol 100 mcg / dose, aerosol; 2 mg, 4 mg tablet; 20 ml solution for nebulizer;
2. Clarithromycin 250 mg, 500 mg tal.;
3. Erythromycin-250 mg, 500 mg table; 250 mg / 5 ml oral suspension.

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

I. Theme № 19: Features of management of children with obstructive syndrome.
II. Learning goals: Diagnosis and treatment of of children with obstructive syndrome on ambulatory level.
III. Lecture thesis:
Obstructive bronchitis is an acute diffuse inflammation of the bronchial mucosa of various etiologies, occurring with reversible airway obstruction.

Classification:
1. obstructive bronchitis;
2. recurrent bronchitis;
3. acute bronchitis;

4. chronic bronchitis.

Risk factors:
1. air pollution;
2. low socio-economic status;
3. hereditary predisposition;
4. allergic mood;
5. adenovirus infection.

Complaints and anamnesis
1. noisy breathing, expiratory dyspnea;
2. obsessive, constantly progressive cough, more often occurring at night, associated with a cold;
3. increase in body temperature;

Physical examination
• rapid or difficult breathing (children under 2 months RR ≥60 per minute; from 2 months to 1 year ≥50 per minute; 1-5 years ≥40 per minute; older than 5 years >28 per minute);
• retraction of the lower part of the chest;
• auscultatory signs (bronchial (hard) breathing, the presence of difficult expiration, against this background - whistling dry rales, (localization and nature of rales change after coughing. Good response to bronchodilators).
• percussion box sound.

Laboratory research:
• complete blood count (leukocytosis with neutrophilic shift to the left, leukopenia, accelerated ESR).

Instrumental research:
• spirometry of changes in indicators of the function of external respiration (in older children).

Indications for expert advice:
• consultation of an otorhinolaryngologist - according to indications;
• consultation of a neurologist - according to indications;
• consultation with a cardiologist - according to indications;
• consultation of a phthisiatrician - according to indications.

List of main diagnostic measures:
1. X-ray of the chest organs;
2. Complete blood count;
3. General analysis of urine;
4. Researches a calla on I/worm.

List of additional diagnostic measures:
1. Peak flowmetry;
2. Study of the functions of external respiration;
3. Allergist's consultation;
4. Analysis and culture of sputum.

TACTICS OF TREATMENT AT OUTPATIENT LEVEL:
Treatment goals:
1. Relief of respiratory failure and asthma attacks, restoration of respiratory functions.
2. Elimination of symptoms of intoxication, improvement of well-being, normalization of appetite and body temperature.

Non-drug treatment
1. Adequate hydration (plentiful warm drink)
2. Encouragement of breastfeeding and adequate nutrition according to age
3. Bed rest for a period of fever and respiratory failure.

Drug treatment
Ambroxol tablets 30.0 mg 3 times a day for 7-10 days, children under 1 year old -
syrup 15 mg/5 ml 3 times a day.
Antibiotic therapy is prescribed only in case of prolonged fever and the presence of other signs of pneumonia: amoxicillin 15 mg / kg x 3 times a day for 5 days or protected penicillins (amoxicillin + clavulanic acid 20-40 mg / kg 3 times a day).
Relief of fever - above 38.5o (paracetamol - 10-15 mg / kg X 4 times a day). Treatment with salbutamol is used only for children from 1 year old in doses: inhalations 0.2-0.3 mg / kg / day or 1-2 doses 2-4 times a day, as well as in capsules 2.0 mg, 1 caps. 3r per day.

Treatment effectiveness indicators:
1. Relief of obstructive syndrome
2. Normalization of body temperature
3. Disappearance of cough

Indications for hospitalization
1. asthmatic status;
2. failure of bronchodilators within 3 days;
3. signs of severe respiratory failure;
4. stridor at rest.

Preventive actions
- limiting contact with patients and virus carriers
- wearing masks and washing hands of family members with SARS.
- maintaining optimal air conditions in the room
- carrying out hardening events

Further management, principles of clinical examination
1. Re-examination by the local doctor after 2 days or earlier, if the child becomes worse or cannot drink or breastfeed, fever, rapid or difficult breathing appears (teach the mother in which situation it is urgent to return to the doctor).
2. If the obstructive syndrome recurs, the child needs consultation and further treatment by a pulmonologist and an allergist.
IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.

Prevention

I. Theme № 20: Functional disorders of the gastrointestinal tract in young children.
II. Learning goals: Diagnosis and treatment of functional disorders of the gastrointestinal tract in children on ambulatory level.
III. Lecture thesis:
Functional dyspepsia is a symptom complex in children over one year old, including pain, discomfort or a feeling of fullness in the epigastric region, whether or not associated with eating or exercise, early satiety, bloating, nausea, regurgitation, intolerance to fatty foods, etc. , lasting at least 3 months over the last 6 months, in which the examination process fails to reveal any organic disease.

Classification of functional dyspepsia:
• **postprandial distress syndrome** (pain or discomfort that occurs after eating the usual amount of food, often combined with fast satiety);
• **syndrome of epigastric pain** (recurrent pain in the epigastric region, not associated with eating, defecation and in the absence of signs of disorders of the biliary tract).

Diagnostic criteria:

Complaints:

Postprandial Distress Syndrome:
• disturbing feeling of fullness after eating, which occurs after taking the usual amount of food;
• fast saturation (satiety);
• bloating;
• nausea after eating;
• belching.

Symptoms of epigastric pain syndrome:
• pain or heartburn, localized in the epigastrium, without a retrosternal component;
• periodic pain, burning, not associated with food intake;
• lack of relief from bowel movements or passing gas.

Postprandial distress syndrome and epigastric pain syndrome can be combined.

Anamnesis:
• genetic predisposition;
• psychosocial factors (stress, physical activity).

Physical examination:
• overlapping of the tongue;
• pain on palpation in the epigastrium, in the navel and pyloroduodenal zone, right hypochondrium, left hypochondrium;
• dyspeptic manifestations;
• absence of symptoms of "anxiety" (unexplained weight loss, repeated vomiting, progressive dysphagia, bleeding from the gastrointestinal tract);
• syndrome of vegetative dystonia (hyperhidrosis of the palm and feet, persistent red dermographism, arterial hypotension).

Basic diagnostic examinations:
• General blood analysis;
• Biochemical blood analysis (ALT, AST, thymol test, bilirubin);

• General urine analysis;
• Non-invasive diagnosis of H. pylori;
• Analysis of feces for protozoa and helminths;
• Analysis of feces (coprogram);
• ultrasound of the abdominal organs.

Additional diagnostic examinations:

• Determination of H. pylori;
• FEGDS;
• ECG;
• Bacteriological examination of feces for dysbiosis;
• Study of the perianal scraping.

Indications for specialist consultation
• consultation with an otorhinolaryngologist - in order to identify chronic foci of infection and their sanitation;
• consultation with a dentist - in order to identify chronic foci of infection and their sanitation;
• consultation of a neurologist - in order to exclude neurological diseases;
• consultation of an endocrinologist - in order to exclude endocrinological diseases;
• consultation of a psychologist - in order to exclude psychosomatic symptoms.

Differential diagnosis:

Criteria for the differential diagnosis of functional dyspepsia

Disease	**Clinical criteria**	**Laboratory and instrumental indicators**
Catarrhal esophagitis	Pain behind the sternum or high in the epigastrium at the xiphoid process of the sternum of a compressive character or burning while eating or after eating, with fast walking, running, deep breathing. Heartburn, worse when bending the trunk, lying down, when lifting severity	During endoscopy - mucosal hyperemia lining of the esophagus, thickening of the folds
Chronic gastroduodenitis	Localization of pain in the epigastrium; pain in the navel and pyloroduodenal zone; severe dyspeptic manifestations (nausea, belching, heartburn, less often vomiting); combination early and late pain;	Endoscopic changes in the gastric mucosa and DC (edema, hyperemia, hemorrhage, erosion, atrophy, hypertrophy folds, etc.)
Chronic cholecystitis	Pain in the right hypochondrium, tenderness on palpation in the projection of the gallbladder, subfebrile condition or periodic	In the blood - leukocytosis, neutrophilia, accelerated ESR. With ultrasound - thickening of the wall of the gallbladder, flakes

	rises in temperature to febrile digits, intoxication	of mucus in it, stagnation of bile, perivascular reaction.
Chronic pancreatitis	Localization of pain on the left above the navel with irradiation to the left, may be girdle pain, intoxication	Increased amylase in urine and blood, activity of trypsin in feces, steatorrhea, creatorrhea. With ultrasound - an increase in the size of the gland and a change in its echo density
Chronic enterocolitis	Localization of pain around the navel or throughout the abdomen, reducing them after bowel movements, bloating, poor tolerance to milk and dairy products, fruit vegetables, unstable stools discharge of gases	In the coprogram - amilorrhea, steatorrhea, creatorrhea, mucus, leukocytes, erythrocytes are possible, signs of disturbed intestinal microbiocenosis
Peptic ulcer	Pain on an empty stomach, "Mostly" late, 2-3 hours after eating. They appear acutely, suddenly, tenderness on palpation is pronounced, the tension of the abdominal muscles, zones of cutaneous hyperesthesia, a positive symptom is determined Mendel.	With endoscopy - a deep defect of the mucous membrane surrounded by a hyperemic shaft, there may be multiple ulcers.

Treatment goals:
• Relief of pain syndrome;
• Normalization of motor disorders.
The choice of therapeutic measures depends on the clinical variant of functional dyspepsia.

Treatment tactics
Drug-free treatment

Diet number 1 (during an exacerbation);
Diet No. 5 (in remission), depending on the individual tolerance of the food.

It is necessary to exclude:
• animal fats, smoked foods,
• strong meat, fish and mushroom broths, cabbage soup, borscht,
• rye bread, fresh baked goods, pancakes,
• carbonated drinks, coffee,
• radish, hot spices.

Drug treatment
• Antisecretory therapy;
• Correction of motor disorders;

• Eradication therapy in the presence of H.p.
• Normalization of the activity of the central nervous system.

Drug treatment for functional dyspepsia

Therapeutic measures:	**Groups of drugs**	**Name of the drug, dosage, duration**
Antisecretory group	Proton pump inhibitors	**Omeprazole**, 0.5-1 mg / kg / day, 10 days **Rabeprazole** 0.5-1 mg / kg / day, 10 days
	Antacids	**Algeldrat + magnesium hydroxide**, 1-3 sachets, 3-5 days
Correction of motor-evacuation disorders	Prokinetics	**Domperidone** - at a dose of 0.5-1 mg / kg body weight / day 7-10 days **Metoclopramide** at a dose of 0.5-1 mg / kg body weight / day 1-3 of the day **Trimebutin** for children from 12 years old, 50 mg 3 times a day 7-10 days
	Antispasmodic drugs	**Mebeverin** from 6 years old at a dose of 2.5 mg / kg in 2 divided doses 20 minutes before meals for 5-7 days **Papaverine** is prescribed for children over 10 years old at 0.005-0.06 d 2 times a day for 3-5 days **Drotaverin** is prescribed children under 6 years of age by mouth 0.01-0.02 g 1-2 times a day, children 6-12 years old, 0.02 1-2 times a day for 3-5 days.
Eradication therapy	Antibiotic therapy	**Clarithromycin** 7.5 mg / kg / day, 10 days **Metronidazole** 20-40 mg / kg / day, 10 days **Amoxicillin** 25 mg / kg / day, 10 days
Normalization of central nervous system activity	Sedation therapy	**Valerian** extract, 100-200 mg / day, 10-12 days

Improvement of digestion	Enzyme therapy	**Pancreatin** at 500-700 U of lipase per kg of body weight per day, 7-10 days

List of essential medicines (100% likely to be used)
• Omeprazole, capsules, tablets 20 mg;
• Rabeprazole capsules, tablets 20 mg;
• Domperidone tablets 10 mg;

List of additional medicines (less than 100% likely to be used).
• Clarithromycin tablets 250 mg, 500 mg;
• Metronidazole 250 mg;
• Amoxicillin 250 mg, 500 mg, 1000 mg;
• Algeldrat + magnesium hydroxide suspension, sachet;
• Valerian extract tablets 0.02.

Indications for planned hospitalization:
• duration of the disease (more than 3 months);
• ineffectiveness of outpatient treatment;
• severe pain syndrome (with the exclusion of acute surgical pathology);
• severe dyspeptic syndrome.

Preventive measures

Primary prevention:
• Proper nutrition in accordance with the age of the child (regime, rhythm, balance);
• rejection of bad habits;
• examination for helminthiasis;
• sanitation of the oral cavity;
• minimizing the impact of hazards;
• compliance with hygiene rules.
Secondary prevention:
• dietary food in accordance with the characteristics of the functional state of the stomach

IV.Visual material: are in the appendix

V. Bibliography: at the end

VI. Post-lecture feedback:
Epidemiology.
Classification.
Diagnostics.
Outpatient management tactics.
Indications for hospitalization.
Dynamic observation.
Issues of medical and labor expertise.
Prevention

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VI. Control questions (feedback):

1. Epidemiology of the disease.
2. Classification of the disease.
3. Diagnosis of the disease.
4. Management tactics on an outpatient basis.
5. Indications for hospitalization.
6. Dynamic observation.
7. Questions of medical and labor expertise.
8. Prevention of the disease.