

Module 1. The principles of diagnosis, treatment, and prophylaxis of the major diseases of circulatory system

Topic 1. Arterial Hypertension

Duration – 4 hours.

Actuality

Hypertension is sustained elevation of resting systolic blood pressure (≥ 130 mm Hg), diastolic blood pressure (≥ 80 mm Hg), or both.

In the US, about 65 million people have hypertension. Only about 70% of these people are aware that they have hypertension, only 59% are being treated, and only 34% have adequately controlled BP. In adults, hypertension occurs more often in blacks (32%) than in whites (23%) or Mexican Americans (23%); morbidity and mortality are greater in blacks.

BP increases with age. About $\frac{2}{3}$ of people > 65 have hypertension, and people with a normal BP at age 55 have a 90% lifetime risk of developing hypertension. Because hypertension becomes so common with age, the age-related increase in BP may seem innocuous, but higher BP increases morbidity and mortality risk. Hypertension may develop during pregnancy. Hypertension may be primary (85 to 95% of cases) or secondary. Untreated hypertension is notorious for increasing the risk of mortality and is often described as a silent killer. Mild-to-moderate hypertension, if left untreated, is associated with a risk of atherosclerotic disease in 30% of people and organ damage in 50% of people after only 8–10 years of onset.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with AH;
- make out a plan of laboratory and instrumental examination, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (modification of diet in renal disease (MDRD) formula), haemoglobin and haematocrit, C-reactive protein, urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Carotid ultrasound. Roentgenological signs of LV hypertrophy. Ophthalmoscopy;
- carry out differential diagnosis of AH with secondary hypertension;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment depending on the stage, grade, and cardiovascular risk of the patient.

Test questions for testing in practical classes:

1. What links in the pathogenesis do not matter in the development of hypertension:

- A. Disruption of transport Na
- B. Endothelial dysfunction.
- C. Decreased RAAS activity.
- D. Increased activity of the sympathetic nervous system.
- E. Decreased depressive function of the kidneys.
- F. Insulin resistance

2. What stage of hypertension corresponds to the presence of the patient during the examination of the following indicators: Left ventricular mass index over 125 g / m², hypertrophic narrowing of the retinal arteries, microalbuminuria?

- A. Stage I
- B. Stage III
- C. Stage II

3. The factors influencing the prognosis in patients with hypertension do not include:

- A. Diseases of the pancreas.
- B. Heart disease.
- C. Dyslipidemia (total cholesterol over 6.5 mmol / l, HDL cholesterol less than 1.0 mmol / l).
- D. Left ventricular hypertrophy.
- E. Abdominal obesity.

4. ECG criteria for left ventricular hypertrophy with its systolic overload are (3 correct answers):

- A. High R in the left chest leads.
- B. High R in the right chest leads.
- C. Deep S in the right chest leads.
- D. ST depression, T inversion in the left chest leads.
- E. ST depression, T inversion in the right chest leads.

5. The ability to prevent stroke in patients with hypertension have (3 correct answers):

- A. Alpha-blockers.
- B. Aspirin.
- C. Thiazide diuretics.
- D. Statins.
- E. Indirect anticoagulants.

6. What stage of hypertension corresponds to the presence of the patient during the examination, in addition to high blood pressure, signs of posterior diaphragmatic myocardial infarction on the ECG?

- A. Stage I
- B. Stage II
- C. Stage III

7. First-line drugs for the treatment of hypertension include all, except:

- A. Calcium antagonists.
- B. Alpha-blockers.
- C. Diuretics.
- D. ACE inhibitors.
- E. Beta-blockers.

8. Which drugs are contraindicated during pregnancy:

- A. Beta-blockers.
- B. Calcium antagonists.
- C. ACE inhibitors.
- D. Dopegit.

9. What concomitant clinical situation is not attributed to a complicated hypertensive crisis?

- A. Unstable angina.
- B. Transient ischemic attack.
- C. Acute hypertensive encephalopathy.
- D. Bleeding.
- E. Increasing the CAT to 240 mm Hg. Art., or DBP up to 140 mm Hg

10. What are the drugs of choice In case of hypertensive crisis on the background of acute heart failure and myocardial infarction:

- A. Nitroglycerin.
- B. Nicardipine hydrochloride.
- C. Furosemide.
- D. Clonidine.

E. Verapamil.

Answers to control questions:

1. – C 2. – C 3. – A 4. – A, C, D 5. – B, D, E 6. – C 7. – B 8. – C 9. – E 10. – A.

To know:

The main theoretical issues of the topic:

1. Definition of arterial hypertension.
2. The pathogenesis of hypertension.
3. Classification of hypertension by severity and stage of the disease.
4. Risk factors for hypertension.
5. The program of examination of the patient with hypertension.
6. Methods of measuring blood pressure.
7. Risk stratification to assess the prognosis in hypertension.
8. Principles of treatment of hypertension. Lifestyle modification.
9. Drugs for the treatment of hypertension: their pharmacological characteristics, doses and regime of administration, contraindications.
10. Definition of hypertensive crisis, classification of hypertensive crises.
11. Treatment of complicated and uncomplicated crises.
12. Primary and secondary prevention of AH.
13. Prognosis and performance in AH.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Arterial Hypertention (AH).	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwld. – 10-th edition. Copyright 2016.
2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
3. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016.
4. Internal medicine: cardiology / V.F. Orlovskyy, L.N. Prystupa, N.M. Kyrychenko, O.S.

- Pogorelova, Yu.O. Ataman. – Sumy: Sumy State University, 2013 – 243 p.
5. <https://www.merckmanuals.com/professional/cardiovascular-disorders/hypertension/overview-of-hypertension>
 6. 2018 ESC/ESH Guidelines for the management of arterial hypertension // *European Heart Journal*, Volume 39, Issue 33, 01 September 2018, Pages 3021–3104, <https://doi.org/10.1093/eurheartj/ehy339>

Topic 2. Secondary Hypertention

Duration – 4 hours.

Actuality

Hypertension with an identified cause (secondary hypertension) is usually due to sleep apnea, chronic kidney disease, primary aldosteronism, diabetes, or obesity. Renovascular disease is one of the most common causes of curable hypertension but accounts for < 2% of all cases of hypertension. Overall, about 80% of cases are caused by atherosclerosis and 20% by fibromuscular dysplasia. Atherosclerosis is more common among men > 50 and affects mainly the proximal one third of the renal artery. Fibromuscular dysplasia is more common among younger patients (usually women) and usually affects the distal two thirds of the main renal artery and the branches of the renal arteries. Rarer causes include emboli, trauma, inadvertent ligation during surgery, and extrinsic compression of the renal pedicle by tumors.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with AH;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein, urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Carotid ultrasound;
- carry out a differential diagnosis of AH with secondary hypertension;
- make diagnosis formulation and individual plan of treatment of the supervised patient.

Test questions for testing in practical classes:

1. Hypokalemia, hypernatremia, hyporeninemia, hyperkaliuria, hypoisostenuria were revealed in the laboratory analysis of a patient. The level of which substances must be determined for diagnosis:

- A. Aldosterone;
- B. Vanyl almond acid;
- C. Catecholamines;
- D. 17-ACS;

2. Renovascular hypertension is characterized by everything except:

- A. High systolic-diastolic hypertension, refractory to treatment;
- B. Positive test with α -blockers;
- C. High plasma renin content;
- D. Auscultation of systolic murmur over the abdominal artery;

3. Pheochromocytoma is characterized by everything except:

- A. Positive test with α -blockers;

- B. No effect of β -blocker therapy;
- C. Transient arterial hypertension;
- D. Decreased plasma renin;
- E. Hyperglycemia;

4. Primary aldosteronism is characterized by everything except:

- A. High plasma renin content;
- B. Neuromuscular disorders;
- C. Hypokalemia;
- D. Decreased glucose tolerance;
- E. Tumor of the adrenal glands;

5. Renal-parenchymal arterial hypertension is characterized by everything except:

- A. History of glomerulonephritis or pyelonephritis;
- B. In the analysis of urine leukocytes, erythrocytes, cylinders;
- C. Positive hypotensive effect of specific therapy of renal disease;
- D. Lack of positive antihypertensive effect from specific therapy of renal disease;

6. For hypertension in Cushing syndrome is characterized by everything except:

- A. hypertension in overweight people;
- B. Decreased glucose tolerance;
- C. Increased levels of cortisol in the blood;
- D. Decreased cortisol levels in the blood;

7. Which symptoms doesn't help in suspicion of Cushing's syndrome (disease) in a patient with hypertension:

- A. Polycythemia;
- B. Centripetal obesity;
- C. Muscle weakness;
- D. Hyperkalemia;
- E. Stria gravidarum;

8. Hypertension in coarctation of the aorta is characterized by all except one:

- A. Increasing blood pressure in the arms and lowering it in the legs;
- B. Rough systolic murmur in the interscapular region;
- C. Accent II tone and systolic murmur in the aorta;
- D. Rough systolic murmur over the thoracic aorta, irradiating the vessels (carotid, subclavian);

9. The drug of which group is used to relieve hypertensive crisis in pheochromocytoma?

- A. β -blockers;
- B. α -blockers;
- C. ACE inhibitors;
- D. Diuretics;

10. For the treatment of hypertension in case of hyperthyroidism drugs of choice are:

- A. Beta-blockers + antihypertensive central action;
- B. Beta-blockers + calcium antagonists;
- B. Diuretics + beta-blockers;
- D. ACE inhibitors + antihypertensive central action.

Answers to control questions:

1. – A 2. – B 3. – D 4. – A 5. – D 6. – D 7. – E 8. – C 9. – B 10. – A

To know:

The main theoretical issues of the topic

1. Definition. Main causes.

2. Clinical features, diagnostic of renal (renovascular, renoparenchymal), endocrinal (Cushing's syndrome, pheochromocytoma, Conn' syndrome, hyperthyroidism) and hemodynamics arterial hypertensions.
3. Arterial hypertensions during pregnancy, metabolic disorders (metabolic syndrome).
4. The role of laboratory and instrumental methods for differential diagnosis and diagnosis verification.
5. Therapeutic and surgical treatment.
6. Primary and secondary prevention.
7. Prognosis and employability.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Secondary AH.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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5. 2018 ESC/ESH Guidelines for the management of arterial hypertension // *European Heart Journal*, Volume 39, Issue 33, 01 September 2018, Pages 3021–3104, <https://doi.org/10.1093/eurheartj/ehy339>
6. 2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults.
7. <https://www.merckmanuals.com/professional/genitourinary-disorders/renovascular-disorders/>

Topic 3. Atherosclerosis. Chronic coronary syndrome

Duration – 4 hours.

Actuality

Atherosclerosis (dyslipidemia) is an elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high density lipoprotein level that contributes to the development of

atherosclerosis. Causes may be primary (genetic) or secondary. Diagnosis is by measuring plasma levels of total cholesterol, TGs, and individual lipoproteins. Treatment is dietary changes, exercises, and lipid-lowering drugs.

Coronary artery disease (CAD) affects approximately 12.9 million Americans and is the primary cause of death in both men and women. In 2000, 681,100 people (more than one in five) died in the result of CAD. Dyslipidemia is one of the most important modifiable risk factors for CAD. Many patients with CHD or those who are at risk for CHD have more than one lipid abnormality, each of which increases cardiovascular risk.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with atherosclerosis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein, urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Carotid ultrasound. Roentgenological signs of LV hypertrophy. Ophthalmoscopy;
- carry out differential diagnosis of atherosclerosis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment depending on dyslipidemia's type.

To know:

The main theoretical questions

1. Definition of Atherosclerosis;
2. Pathophysiology of Atherosclerosis;
3. Risk factors for Atherosclerosis;
4. Features of clinical symptoms depend on localization (aorta, coronary, mesenteric, and renal arteries, arteries of low extremities);
5. Role of laboratory and instrumental methods for differential diagnosis;
6. Differential diagnosis;
7. Complications of Atherosclerosis;
8. General principles of treatment. Treatment tactics for different variants of atherosclerosis;
9. Primary and secondary prevention of Atherosclerosis;
10. Prognosis and employability.

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwld. – 10-th edition. Copyright 2016.
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5. <https://www.merckmanuals.com/professional/cardiovascular-disorders/arteriosclerosis/>

3. Chronic Coronary syndrome

Cardiovascular diseases are presently the leading causes of death in industrialized countries and expected to become so in emerging countries by 2020. Among them, coronary artery disease (CAD) is the most prevalent manifestation and is associated with high mortality and morbidity. The clinical presentations of ischaemic heart disease (IHD) include silent ischaemia, stable angina pectoris, unstable angina, myocardial infarction (MI), heart failure, and sudden death. Patients with chest pain represent a very large proportion of all acute medical hospitalizations in Europe.

The diagnosis of chronic stable angina pectoris includes predictable and reproducible left anterior chest discomfort after physical activity, emotional stress, or both; symptoms are typically worse in cold weather or after meals and are relieved by rest or sublingual nitroglycerin. The presence of one or more obstructions in major coronary arteries is likely; the severity of stenosis is usually greater than 70%.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with angina;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: cardiac markers: cardiac markers are cardiac enzymes (CPK-MB) and cell contents (troponin I, troponin T, myoglobin) (cTnT or cTnI), creatine kinase-MB level (CK-MB), myoglobin. Markers of neurohumoral activation: B-type natriuretic peptide (BNP) or its N-terminal prohormone fragment (NT-proBNP); potassium and magnesium levels, creatinine, C-reactive protein (CRP), serum lactate dehydrogenase (LDH). Fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, urine clinical analysis (proteins, erythrocytes, leukocytes). Electrocardiogram. Echocardiogram. Technetium-99m sestamibi scan. Coronarography;
- carry out differential diagnosis of angina with chest pain: ischaemic cardiac pain and non-ischaemic chest pain;
- make diagnosis formulation and individual plan of treatment of supervised patient;
- prescribe differential treatment.

Test questions for testing in practical classes:

1. Limited ability to self-care within the apartment is typical for patients with stress angina:

- A. I FC;
- B. II FC;
- C. III FC;
- D. IV FC;

2. The drug of choice in patients with spontaneous angina is:

- A. Verapamil;

- B. Dihydropyridine calcium antagonists;
 - C. Diltiazem;
 - D. All answers are correct;
 - D. There is no correct answer;
- 3. For urgent elimination of myocardial ischemia prescribe everything except:**
- A. Nitroglycerin under the tongue, spray and intravenously;
 - B. Morphine sulfate or hydrochloride intravenously;
 - C. Beta-blockers in the absence of contraindications;
 - D. Short-acting dihydropyridine calcium antagonists as monotherapy;
 - E. Diltiazem and verapamil in the absence of left ventricular dysfunction;
- 4. The antianginal effect of trimetazidine (product) is related to:**
- A. With a decrease in the preload on the left ventricle;
 - B. With a decrease in postload on the left ventricle;
 - C. With a decrease in myocardial contractility;
 - D. With cytoprotective action;
- 5. For stable angina is not typical:**
- A. Increased blood cholesterol;
 - B. The appearance of the Q wave;
 - C. Transient depression of the ST segment;
 - D. Positive test with dipyridamole;
- 6. Coronary syndrome X is not characterized by:**
- A. stable tension angina clinically;
 - B. Ischemic changes on the ECG during the stress test;
 - C. The presence of typical atherosclerotic changes in the coronary arteries during coronary angiography;
 - D. No tendency to coronary vasospasm;
- 7. In angina pectoris, the minimum effective single dose of amlodipine is:**
- A. 2.5 mg;
 - B. 10 mg;
 - C. 5 mg;
 - D. 20 mg;
- 8. Trimetazidine is recommended to prescribe:**
- A. 35 mg per day;
 - B. 35 mg 3 times a day;
 - C. 35 mg 2 times a day;
 - D. 35 mg 4 times a day;
- 9. Coronary angiography is recommended for:**
- A. Confirmation of the presence of atherosclerotic lesions in coronary arteries;
 - B. Mandatory for patients with stable angina I-II FC.;
 - C. Patients older than 65 years;
 - D. Asymptomatic patients with a burdensome family history of coronary heart disease;
- 10. In patients with CAD have a favorable effect on the prognosis:**
- A. Short-acting nitroglycerin;
 - B. Calcium antagonists;
 - C. Isosorbide dinitrate;
 - D. Hypolipidemic drugs;

Answers to control questions:

- 1. - D 2. - B 3. - D 4. - D 5. - B 6. - C 7. - A 8. - C 9. - A 10. - D

To know:

1. Definition of coronary artery disease (CAD). Definition of Chronic coronary syndrome.
2. Classification of CAD.
3. Etiology and pathogenesis of coronary artery disease. Mechanisms of coronary insufficiency.
4. Give details of the pain syndrome with stable angina.
5. Classification of stable angina pectoris of the Canadian Society of Cardiology. Clinical picture.
6. Features of the clinical picture of vasospastic angina, microvascular angina (coronary syndrome X).
7. Diagnosis of angina (objective methods of examination, ECG diagnosis in during anginal attack and at rest). Daily ECG monitoring.
8. Functional stress tests: tests with dosed physical activity: methods, interpretation of results; pharmacological tests.
9. Echocardiography. Coronary angiography: indications, contraindications, methods, interpretation of results. CT angiography. Echocardiography.
10. Differential diagnosis of coronary artery disease.
11. Stable angina treatment program. The main antianginal drugs, their pharmacological characteristics.
12. Features of treatment of vasospastic angina, microvascular angina.
13. Role, types, indications for percutaneous coronary interventions in patients with chronic coronary artery disease (Chronic coronary syndrome).
14. The essence of primary and secondary prevention of coronary heart disease. The program of treatment of angina. Main antianginal drugs, their pharmacological properties.
15. Surgical treatments for CAD
16. Primary and secondary prevention of CAD.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Chronic coronary syndrome.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
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Topic 4. Acute Coronary syndrome. Myocardial infarction

Duration – 6 hours.

Actuality

Cardiovascular diseases are presently the leading causes of death in industrialized countries and expected to become so in emerging countries by 2020. Among them, coronary artery disease (CAD) is the most prevalent manifestation and is associated with high mortality and morbidity.

Acute coronary syndrome (ACS) – is the group of clinical signs and symptoms of CAD, which give the basis to suspect developing of acute MI or unstable angina, in the basis of which lays uniform pathophysiology process – thrombosis of a various degree, formed above area of rupture of atherosclerotic patch or damage (erosion) of endothelin.

ACS results from acute obstruction of the coronary artery. Consequences depend on degree and location of obstruction and range from unstable angina to ST-segment elevation MI (STEMI) or new or for the first time arisen of left bundle-branch block (LBBB); non-ST-segment elevation MI (NSTEMI), and sudden cardiac death.

Myocardial infarction (MI) is the myocardial necrosis resulting from abrupt reduction in coronary blood flow to the part of the myocardium. Infarcted tissue is permanently dysfunctional; however, there is a zone of potentially reversible ischaemia adjacent to infarcted tissue.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with ACS and MI;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: cardiac markers: cardiac markers are cardiac enzymes (CPK-MB) and cell contents (troponin I, troponin T, myoglobin) (cTnT or cTnI), creatine kinase-MB level (CK-MB), myoglobin. Markers of neurohumoral activation: B-type natriuretic peptide (BNP) or its N-terminal prohormone fragment (NT-proBNP); potassium and magnesium levels, creatinine, C-reactive protein (CRP), serum lactate dehydrogenase (LDH). Fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, urine clinical analysis (proteins, erythrocytes, leukocytes). Electrocardiogram. Echocardiogram. Technetium-99m sestamibi scan. Coronarography;
- carry out differential diagnosis of ACS and MI;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Test questions for testing in practical classes:

1. Acute coronary syndrome includes:

- A. Ischemic cardiomyopathy;
- B. Hypertensive cardiomyopathy;
- C. Non-Q-myocardial infarction;
- D. Coronary syndrome X;
- E. Stable tension angina, FC II;

2. The leading mechanism of development of acute coronary syndrome is:

- A. Interstitial myocardial fibrosis;
- B. Diffuse cardiosclerosis;
- C. Thrombosis;
- D. Stable atherosclerotic plaque;

3. Acute coronary syndrome do not include:

- A. Progressive angina;
- B. He-Q myocardial infarction;
- C. Q-myocardial infarction;
- D. Acute angina pectoris;
- E. Stable angina pectoris, FC III;

4. Morphological variants of atherosclerotic plaque are as follows, except:

- A. Lipid-rich, eccentric;
- B. Lipid-rich, concentric;
- C. Rich in glycoprotein GP IIb / Sha;
- D. Mostly fibrous, concentric;
- E. Mostly fibrous, eccentric;

5. Atherosclerotic plaque should be considered unstable:

- A. "Young", rich in lipids;
- B. Fibrous, eccentric;
- C. Fibrous, concentric;
- D. With a tight "tire";
- E. With calcinates;

6. Rupture of atherosclerotic plaque contributes to:

- A. Hyperadrenergic;
- B. Bradycardia;
- C. Raynaud's syndrome;
- D. Coronary syndrome X;
- E. Neurocirculatory dystonia;

7. The most powerful vasoconstrictor is:

- A. Endothelium relaxing factor;
- B. Prostacyclin I₂ .;
- C. Endothelium I;
- D. Angiotensin I;
- E. Angiotensin II;

8. The mechanism of action of aspirin is:

- A. Binding of plasmin to a plasminogen activator inhibitor - IAP-1;
- B. Irreversible inhibition of cyclooxygenase-1;
- C. Inhibition of cyclooxygenase-2;
- D. Stimulation of NO synthetase;
- E. Binding to GP IIb / IPa receptors

9. Inhibits ADP-induced platelet aggregation:

- A. Aspirin;
- B. Absiximab;
- C. Clopidogrel;
- D. Dipyridamole;

E. Heparin;

10. **Heparin therapy should be carried out under the control of:**

A. Blood clotting time;

B. Partially activated thromboplastin time;

C. Prothrombin index;

D. Blood pressure;

E. Electrocardiograms.

Answers to control questions:

1. - C 2. - C 3. - E 4. - C 5. - A 6. - A 7. - C 8. - B 9. - C 10. - B

To know:

The main theoretical issues of the topic

1. Definition of the terms "acute coronary syndrome", "unstable angina and myocardial infarction".

2. The role of atherosclerosis, destabilization of atherosclerotic plaque, thrombosis and functional factors in the pathogenesis of various forms of coronary artery disease.

3. Classification of Unstable angina.

4. Classification of MI.

5. Features of the clinical course and diagnosis of acute myocardial infarction (to characterize the clinical variants of myocardial infarction).

6. Diagnostic methods used for patients with ACS (laboratory, instrumental). Values of ECG, coronary angiography, echocardiography, myocardial scintigraphy, MRI.

7. Differential diagnosis of different forms of ACS.

8. ECG changes in MI depends on stages.

9. Sequence of measures in ACS, risk assessment of cardiovascular death. Indications for percutaneous coronary interventions, concomitant therapy.

10. Drug treatment of MI: groups of drugs, mechanisms of action. Thrombolytic therapy, indications, drugs, contraindications to its implementation.

11. Complications of MI and their treatment (acute left ventricular failure, cardiac arrhythmia and conduction, myocardial rupture, acute cardiac aneurysm, post-infarction Dressler's syndrome, etc.).

12. Rehabilitation of postinfarction patients.

13. Primary and secondary prevention of MI.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients ACS: MI and Unstable angina.	280	Case history		Ward, Class room
3	Check survey. Summing up	65	Control questions		Class room

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1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwald. – 10-th edition. Copyright 2016.
2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
3. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016.
4. <https://www.merckmanuals.com/professional/cardiovascular-disorders/coronary-artery-disease/>
5. Guidelines on the management of stable angina pectoris. Available on-line from European Heart Journal: <http://www.escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/guidelines-angina-FT.pdf>.
6. 2018 ESC/EACTS Guidelines on myocardial revascularization // *European Heart Journal*, Volume 40, Issue 2, 07 January 2019, Pages 87–165, <https://doi.org/10.1093/eurheartj/ehy394>
7. Fourth universal definition of myocardial infarction (2018) // *European Heart Journal*, Volume 40, Issue 3, 14 January 2019, Pages 237–269, <https://doi.org/10.1093/eurheartj/ehy462>
8. Acute coronary syndrome: Study guide / O.S. Pogorelova. – Sumy: Sumy State University, 2021 – 73 p.

Topic 5. Chronic Heart Failure

Duration – 4 hours.

Actuality

Heart failure (HF) is a syndrome of ventricular dysfunction.

Heart failure affects about 6.5 million people in the US; > 960,000 new cases occur each year. About 26 million people are affected worldwide.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with HF;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: cardiac markers: cardiac markers are cardiac enzymes (CPK-MB) and cell contents (troponin I, troponin T, myoglobin) (cTnT or cTnI), creatine kinase-MB level (CK-MB), myoglobin. Markers of neurohumoral activation: B-type natriuretic peptide (BNP) or its N-terminal prohormone fragment (NT-proBNP); potassium and magnesium levels, creatinine, C-reactive protein (CRP), serum lactate dehydrogenase (LDH). Fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, urine clinical analysis (proteins, erythrocytes, leukocytes). Electrocardiogram. Echocardiogram. Technetium-99m sestamibi scan. Coronarography;
- carry out differential diagnosis of HF;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Test questions for testing in practical classes:

1. **The main cause of CHF at present is:**
 - A. Arterial hypertension;
 - B. Valvular disease;
 - C. Cardiomyopathy;
 - D. coronary heart disease;
2. **Which drug should be prescribed for patients with CHF with preserved EF:**
 - A. Prazosin;
 - B. Beta-blockers;
 - C. Minoxidil;
 - D. Cardiac glycosides;
3. **The most effective in heart failure diuretic thiazide group is:**
 - A. Hypothiazide;
 - B. Aquaphor;
 - C. Brinaldix;
 - D. Hygroton;
4. **Which cardiac arrhythmias is most typical for cardiac glycoside intoxication?**
 - A. Atrial fibrillation;
 - B. Atrial tachycardia with blockade;
 - C. Reciprocal AV-nodal tachycardia;
 - D. Sinus tachycardia;
5. **Which cardiac arrhythmias is most typical for cardiac glycoside intoxication:**
 - A. Atrial fibrillation;
 - B. Ventricular tachycardia type "pirouette";
 - C. Polymorphic atrial tachycardia;
 - D. "Sinusoidal" ventricular tachycardia;
 - E. "Nonparoxysmal" tachycardia from AV junction with AV dissociation.
6. **The drug of choice in the treatment of arrhythmias in patients with glycosidic intoxication are:**
 - A. Novocainamide;
 - B. Propafenone;
 - C. Lidocaine;
 - D. Amiodarone;
7. **The effectiveness of diuretics in patients with chronic heart failure may decrease with therapy:**
 - A. Indomethacin;
 - B. Ibuprofen;
 - C. prednisolone;
 - D. All answers are correct;
 - E. There is no correct answer.
8. **Which of the following drugs improves the survival of patients with chronic heart failure according to evidence-based medicine:**
 - A. Diuretics;
 - B. ACE inhibitors;
 - C. β -blockers;
 - D. Slow calcium channel blockers;
 - E. Dihydropyridines;
 - F. Sympathomimetics;
9. **The drug of choice in patients with chronic heart failure with severe mitral regurgitation is:**
 - A. Digoxin;
 - B. Isosorbide dinitrate;
 - C. Furosemide;

D. Hydralazine.

10. **The initial dose of captopril in CHF is:**

A. 2.5 mg 3 times a day;

B. 6.25 mg 3 times a day;

B. 12.5 mg 3 times a day;

G. 25 mg 3 times a day.

Answers to control questions:

1. - A 2. - B 3. - A 4. - C 5. - D 6. - C 7. - D 8. - B 9. - B 10. - B

To know:

The main theoretical issues of the topic:

1. Chronic heart failure (CHF) - definition, etiology.

2. Pathogenesis of CHF: the mechanism of systolic and diastolic dysfunctions. Extracardiac compensation mechanisms.

3. What risk factors contribute to the development and progression of heart failure.

4. Clinical manifestations of CHF.

5. Classification of CHF: stages and functional class (New York classification, NYHA).

5. Diagnostic criteria for CHF

6. Examination program for patients with CHF.

7. Laboratory diagnosis of CHF.

8. Instrumental diagnosis of CHF.

9. Non-drug treatment of CHF.

10. Drug treatment of CHF.

11. Standards of treatment of patients with different clinical variants of HF.

12. Complications of CHF.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients HF.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwald. – 10-th edition. Copyright 2016.
2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
3. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition.

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4. Internal medicine: cardiology / V.F. Orlovskyy, L.N. Prystupa, N.M. Kyrychenko, O.S. Pogorelova, Yu.O. Ataman. – Sumy: Sumy State University, 2013 – 243 p.
5. <https://www.merckmanuals.com/professional/cardiovascular-disorders/heart-failure/>
6. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure // European Heart Journal (2016) 37, 2129–2200.

Topic 6. CONGENITAL HEART DISEASE

Duration – 4 hours.

Actuality

Congenital heart disease is the most common congenital anomaly, occurring in almost 1% of live births (1). Among birth defects, congenital heart disease is the leading cause of infant mortality.

The most common congenital heart diseases diagnosed in infancy are muscular and perimembranous ventricular septal defects (VSD) followed by secundum atrial septal defects (ASD), with a total prevalence of 48.4 in 10,000 live births. The most common cyanotic congenital heart disease is tetralogy of Fallot, which is twice as prevalent as transposition of the great arteries (4.7 vs. 2.3/10 000 births). Overall, bicuspid aortic valves are the most common congenital defects with a prevalence reported to be as high as 0.5% to 2.0%. Non cyanotic defects include also patent ductus arteriosus, coarctation of the aorta.

Study Objective

To know:

The main theoretical question for topic:

1. Etiology and pathogenesis, hemodynamic disorders in congenital heart disease.
2. Classification of congenital heart defects.
3. Congenital heart defects with primary cyanosis (tetrad and triad of Fallot), clinic, diagnosis.
4. Congenital heart defects of the pale type with secondary cyanosis (Lutembasche syndrome, Eisenmenger complex), clinic, diagnosis.
5. Congenital heart defects of pale type without cyanosis (open ductus arteriosus, atrial and interventricular septal defect), clinic, diagnosis.
6. Aortic coarctation: pathogenesis, clinical manifestations, diagnosis, treatment.
7. Differential diagnosis of congenital heart defects and acquired heart defects.
8. Laboratory and instrumental diagnosis of congenital heart disease.
9. Complications of congenital heart disease and their treatment.

To be able to:

- collect medical history;
- carry out clinical examination of the patients with ASD, VSD, PDA, coarctation of the aorta;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein; blood coagulation, prothrombin. Urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Roentgenological chest with contrast of esophagus;
- carry out differential diagnosis of ASD with functional systolic murmur, stenosis of the pulmonary artery, mitral stenosis and regurgitation, chronic core pulmonale, VSD. VSD with

mitral regurgitation, obstructive cardiomyopathy, stenosis of the pulmonary artery, Fallot's tetrad, ASD and with phase Eisenmenger's syndrome with idiopathic lung hypertension, thromboembolism, systemic vasculitis, and pulmonary artery changes. PDA with functional systolic murmur, ASD, VSD, aortic valve diseases, stenosis of pulmonary artery. Coarctation of the aorta with arterial hypertension;

- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Congenital heart disease.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwald. – 10-th edition. Copyright 2016.
2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
3. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016.
4. Internal medicine: cardiology / V.F. Orlovskyy, L.N. Prystupa, N.M. Kyrychenko, O.S. Pogorelova, Yu.O. Ataman. – Sumy: Sumy State University, 2013 – 243 p.

Topic 7. VALVULAR HEART DISEASE

Duration – 4 hours.

Actuality

Aortic regurgitation is incompetency of the aortic valve causing flow from the aorta into the left ventricle during diastole. Causes include idiopathic valvular degeneration, rheumatic fever, endocarditis, myxomatous degeneration, congenital bicuspid aortic valve, aortic root dilatation or dissection, and connective tissue or rheumatologic disorders. Aortic regurgitation (AR) may be acute or chronic.

Aortic stenosis (AS) is narrowing of the aortic valve obstructing blood flow from the left ventricle to the ascending aorta during systole. Causes include a congenital bicuspid valve, idiopathic degenerative sclerosis with calcification, and rheumatic fever. The most common cause of AS in patients < 70 yr is a congenital bicuspid aortic valve. Congenital AS affects more males.

Mitral regurgitation (MR) is incompetency of the mitral valve causing flow from the left ventricle (LV) into the left atrium during systole. MR may be acute or chronic. Causes of acute MR include: ischaemic papillary muscle dysfunction or rupture; infective endocarditis; acute rheumatic fever; spontaneous, traumatic, or ischaemic tears or rupture of the mitral valve leaflets or subvalvular apparatus; acute dilation of the LV due to myocarditis or ischaemia; mechanical failure of a prosthetic mitral valve. Common causes of chronic MR include: those of acute MR; myxomatous degeneration of the mitral leaflets or chordae tendineae; mitral valve prolapse (MVP); mitral annular enlargement; nonischaemic papillary muscle dysfunction; calcification of the mitral annulus (mainly in elderly women).

Mitral stenosis (MS) is narrowing of the mitral orifice impeding blood flow from the left atrium to the left ventricle. Aetiology and pathophysiology: rheumatic fever, less common causes include bacterial endocarditis, SLE, atrial myxoma, RA malignant carcinoid syndrome with an atrial right-to-left shunt. Occasionally, MS is congenital.

Study Objective

To know:

The main theoretical question topics:

1. Definition of valvular heart disease.
2. The main mechanisms of hemodynamic disorders in heart disease. What is resistance and volume overload?
3. Classification of acquired heart defects.
4. Etiology, features of hemodynamics, diagnostic criteria of mitral valve stenosis (data of objective examination, instrumental methods). Tactics of management of a patient with mitral stenosis, indications for surgical treatment.
5. Etiology, features of hemodynamics, diagnostic criteria of mitral valve regurgitation (data of objective examination, instrumental methods). Tactics of management of the patient with mitral insufficiency, indications for surgical treatment.
6. Etiology, hemodynamics in aortic valve stenosis. Clinical manifestations, classification of aortic stenosis. Instrumental diagnostics. Management of a patient with aortic stenosis, indications for surgical treatment.
7. Etiology, hemodynamic features, diagnostic criteria of aortic valve insufficiency (data of objective examination, instrumental methods). Tactics of management of the patient with aortic insufficiency, indications for surgical treatment.
8. Criteria for the diagnosis of pulmonary artery valve defects.
9. Criteria for diagnosing defects of the tricuspid valve.
10. Complications in valvular heart disease.
11. Types of surgical treatment of patients with acquired heart disease.
12. Prevention of thrombotic complications in patients who have undergone surgical correction of heart defects.
13. Rehabilitation of patients in the postoperative period.

To be able to:

- collect medical history;
- carry out clinical examination of the patients with valve diseases;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein; blood coagulation, prothrombin. Urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram.

Echocardiogram. Roentgenological chest with contrast of esophagus;

- carry out differential diagnosis of MS with chronic core pulmonale, thyrotoxicosis, mixom of LA. Mitral regurgitation with VSD, aortic stenosis, relative mitral regurgitation at the dilatation of LV. AS with relative stenosis of aorta at its dilatation, murmur at the stenosis of carotic and subclavion arteries, stenosis of lung artery, VSD, mitral regurgitation, obstructive cardiomyopathy. AR with Stil's murmur at pulmonale regurgitation, with Flint's murmur at MS;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Valvular disease.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwld. – 10-th edition. Copyright 2016.
2. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
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5. <https://www.merckmanuals.com/professional/cardiovascular-disorders/valvular-disorders/>
6. 2017 ESC/EACTS Guidelines for the management of valvular heart disease // European Heart Journal (2017) 00, 1–53.

Topic 8. INFECTIVE ENDOCARDITIS

Duration – 4 hours.

Actuality

Infective endocarditis (IE). If it is not treated, there is a fatal disease. The achievement of recent years in diagnosis and treatment have improved that the prognosis of the patients with IE is poor. The reason of high death from IE is later establishment of the diagnosis or later treatment. Therefore it is very important to: 1) consider the opportunity of IE occurrence at early stages of examination of each patient having a fever or septicemia and cardio murmur; 2) carry out urgent cardiography of each patient with suspicion on IE; 3) ensure cooperation of cardiologists, microbiologists, and cardio surgery at suspicion on IE or at establishment of this diagnosis.

Density of IE in structure of the acquired heart defects in the operated patients have been

increased 8 times for recent ten years. The reason of such increase is the growth of narcomania. In those who inject drugs intravenously, IE arises 30 times more often, than among people in general and 4 times is more often, than in the patients with rheumatic defects of heart. IE arises in 1–4% of the patients with prosthetic heart valve in the first year after the operation. In 10% IE arises after the realization of diagnostic and medical manipulations.

IE affects the men 2–3 times more often, than the women. There is a tendency for IE “aging” in the industrially advanced countries for the recent 30 years. The average age of such patients is 50 years and 25% of the patients is older than 60 years.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with IE;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein; blood coagulation, prothrombin, increase of alanine aminotransferase, lactate dehydrogenase, creatine phosphokinase, α 2- and γ -globulin, cialic acid, fibrinogen, seromucoid, haptoglobin, C-reactive protein, rheumatoid factor. Immunologic examination: titre of circulating immunity complex, C3 and C4 complements, blood culture. Urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Roentgenological chest;

- carry out differential diagnosis of IE in the patient with fever and nonspecific murmur in heart without signs of the valves diseases (at sepsis, tuberculosis, osteomyelitis, focal purulent infection in abdomen), acute rheumatic fever, autoimmunity diseases (systemic lupus erythematosus, systemic vasculitis, dermatomyositis), tumor, and paraneoplastic syndrome (lymphomas);

- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Test questions for testing

1. It is advisable to carry out antibiotic prophylaxis in a patient with:

- A. Implanted pacemaker;
- B. History of rheumatic fever without defect;
- C. History of infectious endocarditis;
- D. Operated atrial septal defect;
- E. Operated ventricular septal defect;

2. It is advisable to carry out antibiotic prophylaxis in a patient with:

- A. "Innocent" heart murmur;
- B. Isolated secondary defect of the interventricular septum;
- C. Operated open ductus arteriosus;
- D. Implanted artificial valve;
- E. Mitral valve prolapse without regurgitation;

3. It is advisable to carry out antibiotic prophylaxis during manipulation:

- A. Intubation of the trachea;
- B. Esophageal echocardiography;
- C. Bronchoscopy with a flexible bronchoscope;
- D. Catheterization of the heart;
- E. Tooth extractions;

4. It is advisable to carry out antibiotic prophylaxis during manipulation:

- A. Childbirth;
- B. Stenting;
- C. Catheterization of the urethra in the absence of infection;
- D. Tympanostomy;
- E. Tonsillectomy;

5. At what manipulations antibiotic prophylaxis will be inexpedient?

- A. Interventions at the root of the tooth;
- B. Angioplasty;
- C. Dilatation of the esophageal structure;
- D. Cystoscopy;
- E. Bronchoscopy with a rigid bronchoscope;

Answers to control questions: 1. - C 2. - D 3. - E 4. - E 5. - B

To know:

The main theoretical issues of the topic:

1. Define the term "infectious endocarditis".
2. Classification of infectious endocarditis.
3. Etiology and pathogenesis of infectious endocarditis. Risk factors for the development of IE.
4. Clinical manifestations of infectious endocarditis.
5. The main syndromes of infectious endocarditis.
6. Laboratory and instrumental diagnosis of IE. Features of blood test in case of suspected IE.
7. Duke-criteria for the diagnosis of IE.
8. Principles of treatment of IE depending on the infectious agent.
9. Indications for surgical treatment of IE.
10. Treatment of IE of staphylococcal etiology.
11. Principles of treatment of IE of streptococcal etiology.
12. Indications for surgical treatment.
13. Prevention of infectious endocarditis.

Topic 8. PERICARDITIS

Actuality

Pericarditis (pericardial syndromes) is inflammation of the pericardium, often with fluid accumulation. Pericarditis may be caused by many disorders but is often idiopathic. In progressive of human immunodeficiency virus (HIV) infection the incidence of echocardiographically detected pericardial effusion is up to 40%. Purulent pericarditis in adults is rare, but always fatal if untreated. Mortality rate in treated patients is 40%, mostly due to cardiac tamponade, toxicity, and constriction. In the recent decade, TBC pericarditis in the developed countries has been primarily seen in immunocompromised patients (AIDS). The mortality rate in untreated acute effusive TBC pericarditis approaches 85%. Pericardial constriction occurs in 30–50%.

Pericardial constriction may happen in up to 20% of patients with radiation-induced pericarditis, requiring pericardiectomy. The operative mortality is high (21%) and the postoperative five years survival rate is very low (1%) mostly due to myocardial fibrosis.

Study Objective

To know:

The main theoretical question topics:

1. Define the term "pericarditis".
2. Classification of pericarditis.
3. Etiology and pathogenesis of pericarditis.

4. Clinical manifestations of dry pericarditis and data of objective examination of a patient with dry pericarditis.
5. Laboratory and instrumental diagnosis of dry pericarditis.
6. Treatment of dry pericarditis.
7. Clinic and data of objective examination of a patient with exudative pericarditis.
8. Instrumental and laboratory diagnosis of exudative pericarditis.
9. Treatment of exudative pericarditis. Methods and indications for pericardial puncture.
10. Clinical signs of cardiac tamponade.
11. Clinical manifestations, objective examination of a patient with constrictive pericarditis.
12. Laboratory and instrumental diagnosis of constrictive pericarditis.
13. Principles of treatment of a patient with constrictive pericarditis.
14. Cardiac tamponade: causes, clinical manifestations, treatment.

To be able to:

- collect medical history;
- carry out clinical examination of the patients with pericarditis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination. Chest X-ray (signs are atelectasis or infiltrate, pleural effusion, pleural-based opacity(infarction), elevated diaphragm, decreased pulmonaryvascularity, amputation of hilar artery. Blood gases (signs of hypoxaemia). ECG (signs of right ventricular overload). Signs in lung scintigraphy, pulmonary angiography and spiral computed tomography, echocardiography. Signs of ultrasonic investigation;

- carry out differential diagnosis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Infective endocarditis and Pericarditis.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwld. – 10-th edition. Copyright 2016.
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 5. <https://www.merckmanuals.com/professional/cardiovascular-disorders/myocarditis-and-pericarditis/>
 6. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases // European Heart Journal Advance Access published August 29, 2015.
 7. <https://www.merckmanuals.com/professional/cardiovascular-disorders/endocarditis/infective-endocarditis>.
 8. <https://www.merckmanuals.com/professional/cardiovascular-disorders/endocarditis/infective-endocarditis>.

Topic 9. Myocarditis and Cardiomyopathies

Duration – 4 hours.

Actuality

Myocarditis is inflammation of myocardium with necrosis of cardiac myocyte cells. Myocarditis may result from infectious or noninfectious causes. Infectious myocarditis is most often viral in the US and other developed nations. The most common viral causes in the US are parvovirus B19 and human herpes virus 6 . In developing nations, infectious myocarditis is most often associated with rheumatic carditis , Chagas disease , or AIDS . Direct myocardial injury due to SARS-CoV-2 infection , with symptoms ranging from mild chest discomfort to fulminant myocarditis, may occur in COVID-19 cases.

Prognosis at Dilated Cardiomyopathy generally is poor: up to 70% die at the age of < 5 yr; about 50% of deaths are sudden, due to a malignant arrhythmia or an embolic event. Most cases of Hypertrophic Cardiomyopathy are inherited. At least 50 different mutations that are inherited in an autosomally dominant pattern have been identified; spontaneous mutations are common. Perhaps 1 in 500 people is affected; phenotypic expression varies markedly. Typically, symptoms appear between age 20 and 40 and are exertional. Prognosis at Restrictive Cardiomyopathy is poor, similar to that with dilated congestive cardiomyopathy, because the diagnosis is often made at a late stage. No treatment is available for most patients; symptomatic, supportive care can be provided.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with myocarditis and DCM;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, C-reactive protein; blood coagulation, prothrombin, increase of alanine aminotransferase, lactate dehydrogenase, creatine phosphokinase, α 2- and γ -globulin, fibrinogen, seromuroid, haptoglobin, C-reactive protein, rheumatoid factor. Immunologic examination: titre of circulating immunity complex, C3 and C4 complements, blood culture. Urine clinical analysis (proteins, erythrocytes, leukocytes), Reberg test. Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination). Electrocardiogram. Echocardiogram. Roentgenology. MRI.
- carry out differential diagnosis of DCMP with coronary artery disease with HF, with

myocarditis, with mitral regurgitation of rheumatic and nonrheumatic aetiology, with aortic stenosis, with exudative pericarditis, specific cardiomyopathy at the amyloidosis, haemochromatosis and sarcoidosis. HCM with “sport’s heart”. RCM with DCM, HCM, disease with syndrome restrictive cardiomyopathy (constrictive pericarditis at tuberculosis, chest trauma, acute pericarditis);

- make diagnosis formulation and individual plan of treatment of the patient;
- prescribe differential treatment.

Test questions for testing in practical classes:

1. What are the indications for the appointment of nonsteroidal anti-inflammatory drugs in myocarditis?

- A. In the first two weeks of the acute phase of the disease;
- B. From the third week of the acute phase of the disease;
- C. At a chronic current without the expressed dilatation of cavities of heart and heart failure;
- D. Situations are specified in items B and C;
- E. In the first three months from the onset of the viral disease;

2. What can most provoke myocardial dysfunction in myocarditis?

- A. Low calorie diet
- B. Unlimited salt intake;
- C. Unlimited fluid intake;
- D. Ignoring medications;
- E. Physical activity in severe disease

3. Method (s) of verification of the diagnosis of myocarditis:

- A. Signs of inflammation on biopsy
- B. Increased levels of CF isoenzyme CPK in the blood;
- C. Depression of the ST segment and the negative T wave on the ECG;
- D. Acute occurrence of complete blockade of the left leg of the His bundle;
- E. Dilatation of the left ventricle and reduction of PV according to Echo-CG;

4. The major criteria for myocarditis do not include:

- A. Occurrence of congestive heart failure after infection;
- B. Occurrence of a positive blood culture after infection;
- C. Occurrence after a complete infection of complete blockade of the left leg of the His bundle;
- D. Increasing the activity of cardiospecific enzymes;

5. What tactics should be chosen in patients with asymptomatic hypertrophic cardiomyopathy?

- A. Drug treatment for prophylactic purposes;
- B. Dynamic observation;
- C. Non-drug treatment;
- D. Surgical treatment;

Answers to control questions:

1. - B 2. - E 3. - A 4. - B 5. - B

To know:

The main theoretical issues of the topic

1. Define the term "myocarditis".
2. Classification of myocarditis.
3. Etiology and pathogenesis of myocarditis.
4. Name and describe the clinical variants of myocarditis.
5. Laboratory and instrumental diagnosis of myocarditis, indications for endomyocardial biopsy.
6. Diagnostic criteria for myocarditis.

7. Know the elements of differential diagnosis of myocarditis.
8. To make the program of treatment of myocarditis. Indications and conditions for specific therapy.
9. Symptomatic therapy of myocarditis. Non-drug treatment.
10. What is the prevention of myocarditis?
11. Define the term "cardiomyopathy" (CM).
12. Classification of CM.
13. Etiology, pathogenesis and clinical manifestations of dilated cardiomyopathy.
14. Data of objective examination, instrumental methods in the diagnosis of dilated CM.
15. Treatment of dilated CM: groups of drugs, the mechanism of their action. Non-drug treatment (surgical methods).
16. Pathogenesis, clinical manifestations of hypertrophic CM.
17. Data of objective examination and instrumental methods in the diagnosis of hypertrophic CM.
18. Drug treatment of hypertrophic cardiomyopathy, surgical methods.
19. Clinic, diagnosis and treatment of restrictive CM.
20. Clinical manifestations, diagnosis and treatment of arrhythmogenic dysplasia of the right ventricle.
21. Metabolic CM, classification, clinical manifestations, diagnosis.
22. Treatment of metabolic cardiomyopathies.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients myocarditis and cardiomyopathies.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwald. – 10-th edition. Copyright 2016.
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Topic 10. Cor Pulmonale and Pulmonary Embolism

Duration – 4 hours.

Actuality

Cor pulmonale (CP) is right ventricular enlargement secondary to a lung disorder that produces pulmonary artery hypertension. Cor pulmonale results from a disorder of the lung or its vasculature or deformation of chest.

Cor pulmonale is estimated to account for 6–7% of all types of adult heart disease in the United States, with chronic obstructive pulmonary disease (COPD) due to chronic bronchitis or emphysema the causative factor in more than 50% of cases. At present, cor pulmonale accounts for 10–30% of decompensated heart failure related admissions in the United States. Acute massive pulmonary embolism is the most common cause of acute life-threatening cor pulmonale in adults.

Acute pulmonary embolism (PE) is a major cause of complications and death associated with surgery, injury, and medical illnesses, and it may occur after long-distance air travel. Venous thromboembolism is responsible for up to 15% of all in-hospital deaths, and it also accounts for 20 to 30% of deaths associated with pregnancy and delivery in the United States and Europe.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with angina;
- carry out laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: markers of myocardial injury, D-dimer level. Chest X-ray (signs are atelectasis or infiltrate, pleural effusion, pleural-based opacity (infarction), elevated diaphragm, decreased pulmonary vascularity, amputation of hilar artery. Blood gases (signs of hypoxaemia). ECG (signs right ventricular overload). PE signs in lung scintigraphy, pulmonary angiography and spiral computed tomography, echocardiography, and detection of deep vein thrombosis;
- carry out differential diagnosis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.
-

Test questions for testing in practical classes:

1. The most common groups among the main causes of chronic Cor pulmonale are:

- A. Diseases of the lung parenchyma;
- B. Diseases of the airways;
- C. Lesions of the pulmonary vascular bed;
- D. Thoracodiaphragmatic diseases;
- E. Dysfunction of the respiratory center;

2. Among the diseases of the lung parenchyma, the most common cause of chronic Cor pulmonale are:

- A. Pneumosclerosis;
- B. Pulmonary fibrosis due to tuberculosis;
- C. Pulmonary fibrosis due to pneumoconiosis;
- D. Fibrous alveoli;
- E. Chronic pneumonitis and pulmonary fibrosis in diffuse connective tissue diseases;

3. Among the lesions of the pulmonary vascular bed the most common cause of chronic Cor

pulmonale is:

- A. Primary pulmonary hypertension;
- B. Vasculitis in diffuse connective tissue diseases;
- C. Nodular peri arteritis and other generalized vasculitis;
- D. Recurrent thromboembolism and thrombosis of small branches of the pulmonary artery;
- E. The main reason is not named;

4. Among the disorders of the respiratory center, the most common cause of chronic Cor pulmonale are:

- A. Obesity (Pickwick syndrome);
- B. Idiopathic alveolar hypoventilation;
- C. Syndrome of nocturnal apnea of central origin;
- D. Prolonged stay in the highlands;
- E. The main reason is not named;

5. The main mechanism of pulmonary arterial hypertension in patients with chronic Cor pulmonale is:

- A. Hypoxic pulmonary vasoconstriction;
- B. Effects of hypercapnia and acidosis;
- C. Anatomical changes of the pulmonary vascular bed;
- D. Disorders of bronchial patency;
- E. All together;

6. The main changes in hemodynamics in chronic Cor pulmonale are:

- A. Hypertrophy of the right ventricle;
- B. Gradual decrease in systolic function of the right ventricle;
- C. The tendency to increase the volume of circulating blood, sodium and water retention in the body;
- D. In the later stages of the disease - a decrease in cardiac output and blood pressure;
- E. All together;

7. The main clinical objective manifestations of compensated chronic Cor pulmonale are all except:

- A. Central cyanosis;
- B. Dilation and increase in the number of vessels in the skin and conjunctiva;
- C. Right-sided hydrothorax;
- D. Tachypnea, dry and / or wet rales, crepitation;
- E. Signs of emphysema;

8. Decompensated chronic Cor pulmonale is characterized by all symptoms except one:

- A. Severe cyanosis of mixed nature;
- B. Swelling of the lower extremities;
- C. Edema of the jugular veins;
- D. Abdominal-jugular reflux (increased swelling of the jugular veins when pressing the hand on the anterior abdominal wall);
- E. Cold limbs;

9. For patients with chronic Cor pulmonale during the examination is characterized by all these symptoms, except:

- A. Systolic murmur over the apex of the heart
- B. Expansion of absolute dullness of the heart;
- C. Tachycardia;
- D. Accent II tone over the pulmonary artery;
- E. Common pulsation to the left of the sternum and in the epigastrium;

10. What method of treatment of patients with chronic Cor pulmonale confirmed the increase in duration and quality of life?

- A. Vasodilators;
- B. Anticoagulants;

- C. Diuretics;
- D. Cardiac glycosides;
- E. Oxygen therapy;

Answers to control questions:

1. - B 2. - A 3. - D 4. - B 5. - E 6. - E 7. - C 8. - E 9. - A; 10. - E

To know:

The main theoretical issues of the topic

1. Define the term "Cor pulmonale".
2. The main causes of drug development.
3. Pathogenesis of drugs. Mechanisms of pulmonary hypertension formation.
4. Clinical picture of chronic Cor pulmonale.
5. Clinic of decompensated Cor pulmonale
6. Define the term "Pulmonary embolism".
7. Etiology and pathogenesis of pulmonary embolism.
8. Risk factors for the development of pulmonary embolism.
9. Classification of pulmonary embolism according to the level of occlusion of the pulmonary arteries. Estimation of the probability of pulmonary embolism according to clinical data.
10. Clinical manifestations of pulmonary embolism and complications.
11. Laboratory and instrumental diagnostics.
12. Differential diagnosis of pulmonary embolism.
13. Treatment of patients with pulmonary embolism.
14. Prevention of pulmonary embolism.
15. Instrumental diagnosis of chronic Cor pulmonale (echocardiography, catheterization of the right heart, examination of the function of external respiration)).
18. ECG criteria chronic Cor pulmonale
19. Diagnostic criteria for acute and chronic Cor pulmonale.
20. Differential diagnosis of chronic Cor pulmonale.
21. Chronic thromboembolic pulmonary hypertension.
22. Treatment of chronic Cor pulmonale: correction of pulmonary arterial hypoxemia, pulmonary vascular resistance, hemorheological disorders.
23. Features of treatment of right ventricular heart failure.
24. Oxygen therapy in the treatment of chronic Cor pulmonale.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Cor pulmonale and PE.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L.

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 3. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016.
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Topic 11. Cardiac arrhythmias and Conduction Disorders

Duration – 6 hours.

Actuality

Cardiac arrhythmia is a term for any of a large and heterogeneous group of conditions in which there is abnormal electrical activity in the heart. The heart beat may be too fast or too slow, and may be regular or irregular.

Some arrhythmias are life-threatening medical emergencies that can result in cardiac arrest and sudden death. Others cause aggravating symptoms such as an abnormal awareness of heart beat (palpitations), and may be merely annoying. Others may not be associated with any symptoms at all, but predispose toward potentially life threatening stroke or embolism.

Atrial fibrillation (AF) is the most common arrhythmia. The frequency of its occurrence depends on age, it is more often noted at men. Among people under the age of 40, its frequency is less than 0.5%, over 70 years - 10%.

Study Objective

To be able to:

- collect medical history;
- carry out clinical examination of the patients with arrhythmias;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood examination: cardiac markers: cardiac markers are cardiac enzymes (CPK-MB) and cell contents (troponin I, troponin T, myoglobin) (cTnT or cTnI), creatine kinase-MB level (CK-MB), myoglobin. Markers of neurohumoral activation: B-type natriuretic peptide (BNP) or its N-terminal prohormone fragment (NT-proBNP); potassium and magnesium levels, creatinine, C-reactive protein (CRP), serum lactate dehydrogenase (LDH). Fasting plasma glucose, serum total cholesterol, serum LDL-cholesterol, serum HDL-cholesterol, fasting serum triglycerides, serum potassium, serum uric acid, serum creatinine, estimated creatinine clearance (Cockcroft-Gault formula) or glomerular filtration rate (MDRD formula), haemoglobin and haematocrit, urine clinical analysis (proteins, erythrocytes, leukocytes). Electrocardiogram. Echocardiogram. Technetium-99m sestamibi scan. Coronarography;
- carry out differential diagnosis of arrhythmias and conduction disorders;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment.

Test questions for testing in practical classes:

1. The drugs of first choice in paroxysmal supraventricular tachycardia are:

A. ATP;

- B. Novocainamide;
- C. Verapamil;
- D. Obzidan;

2. **The drug of first choice in paroxysmal ventricular tachycardia are:**

- A. Novocainamide;
- B. Lidocaine;
- C. Giluritmale;
- D. Amiodarone;

3. **Which drug is contraindicated for reliving a paroxysm of a ventricular tachycardia at patients with organic changes in the heart:**

- A. Novocainamide;
- B. Disopyramide;
- C. Verapamil;
- D. Amiodarone;

4. **In case of overdose of calcium antagonists the antidote is:**

- A. Unithiol;
- B. Calcium chloride;
- C. Obzidan;
- D. Sodium lactic acid;

5. **Lupus-like syndrome can develop during therapy:**

- A. Amiodarone;
- B. disopyramide;
- C. Quinidine;
- D. Novocainamide;

Answers to control questions:

1. - C 2. - B 3. - B 4. - B 5. – D

To know:

The main theoretical issues of the topic

1. Define the term "cardiac arrhythmia".
2. The main causes of arrhythmias.
3. Function of automaticity of heart, centers of automaticity Electrophysiological mechanisms of arrhythmias.
4. Conducting system of the heart. Violation of the impulse correspondence.
5. Classification of cardiac arrhythmias.
6. Antiarrhythmic drugs, classification, mechanisms of action, indications for their use.
7. Methods of diagnosis of arrhythmias: physical, examination, ECG, Holter monitoring, electrophysiological study, esophageal pacing.
8. Diagnosis and treatment of extrasystolic arrhythmia, paroxysmal tachycardia: ECG signs, clinic, treatment.
9. Atrial fibrillation, etiopathogenesis, classification, ECG signs.
10. Treatment of a patient with persistent atrial fibrillation.
11. Treatment of a patient with a permanent form of atrial fibrillation.
12. Atrial fibrillation, etiopathogenesis, classification, ECG signs, treatment.
13. Radiofrequency ablation, pacing, electrical defibrillation in the treatment of arrhythmias: methods, indications. Resynchronizing therapy.
14. Sinoatrial blockade: ECG diagnosis, clinical manifestations and treatment.
15. Intratrial block: ECG signs, clinical manifestations and treatment.
16. Atrioventricular block: classification, ECG signs, clinical manifestations.
17. Tactics of management of the patient with atrioventricular blockade.
18. Intraventricular blockade: ECG diagnosis, clinical manifestations, treatment.
19. ECG signs of blockade of the left and right leg of the His bundle.

20. Syndromes of premature ventricular excitation: ECG signs, clinical significance and treatment.
21. Combined arrhythmias: ECG signs, clinical manifestations, treatment:
 - a) AV-dissociation; b) Frederick's syndrome; c) parasystole.
22. The role of Electrical methods in the treatment of arrhythmias (indications, contraindications).
23. Morgan-Adams-Stokes syndrome: clinical manifestations, pathogenesis, emergency care. Indications for electrical stimulation, types of electrical stimulation.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	30	Tests		Class room
2	Thematic analysis of patients arrhythmias and conduction disorders.	250	Case history		Ward, Class room
3	Check survey. Summing up	80	Control questions		Class room

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1. Braunwald's Heart disease: a textbook of cardiovascular medicine / edited by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow, Eugene Braunwald. – 10-th edition. Copyright 2016.
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Module 2. The principles of diagnosis, treatment, and prophylaxis of the diseases of musculoskeletal system and connective tissue

Topic 12. The main symptoms of rheumatological pathology and methods of investigation in rheumatology. Rheumatic fever.

Duration – 4 hours.

Actuality

Rheumatic fever (RF) is common worldwide and responsible for many cases of damaged heart valves. In Western countries, it became fairly rare since the 1960s, probably due to widespread use of antibiotics to treat streptococcus infections. While it has been far less common in the United States since the beginning of the 20th century, there have been a few outbreaks since the 1980s. Although the disease seldom occurs, it is serious and has a mortality of 2–5%. Rheumatic fever primarily affects children between ages 5 and 17 years and occurs approximately 20 days after strep throat. In up to a third of cases, the underlying strep infection may not have caused any symptoms. The rate of development of rheumatic fever in individuals with untreated streptococcal infection is estimated to be 3%. The incidence of recurrence with a subsequent untreated infection is substantially greater (about 50%). The rate of development is far lower in individuals who have received antibiotic treatment. Persons who have suffered a case of rheumatic fever have a tendency to develop flare-ups with repeated strep infections. The recurrence of rheumatic fever is relatively common in the absence of maintenance of low dose antibiotics, especially during the first three to five years after the first episode. Heart complications may be long-term and severe, particularly if valves are involved. Survivors of rheumatic fever often have to take penicillin to prevent streptococcal infection which could possibly lead to another case of rheumatic fever that could prove fatal.

Study Objective

To know:

- 1) Definition of acute rheumatic fever and chronic rheumatic heart disease;
- 2) Classification of acute rheumatic fever;
- 3) Etiopathogenesis of acute rheumatic fever;
- 4) Clinical manifestations (carditis, polyarthritis, chorea, skin lesions);
- 5) Diagnostic criteria;
- 6) Laboratory and instrumental diagnosis of ARF;
- 7) Differential diagnosis, complications;
- 8) Treatment of ARF with regard to the level of activity;
- 9) Primary and secondary prevention

Test questions for testing in practical classes:

1. Rheumatic fever is caused by

- A. Staphylococcus
- B. beta-hemolytic streptococcus group C
- C. pneumococcus
- D. 4 beta-hemolytic streptococcus group A .
- E. 5 the causative agent is unknown

2. The most common signs of rheumatic fever are

- 1 small chorea
 - 2 "Flying" arthritis
 - 3 annular erythema
 - 4 erythema nodosum
 - 5 carditis
- A. true 1, 2
 - B. true 1, 2, 3, 5 .
 - C. true 2, 4, 5
 - D. all of the above is true
 - E. all of the above is not true

3. After a streptococcal infection, rheumatic fever occurs through

- A. 1-2 years
- B. 2-3 weeks .
- C. 4 days
- D. 5 months
- E. 6 weeks

4. Rheumatic polyarthritis is characterized by

- 1 Persistent deformity of the joints
- 2 Unstable joint deformity
- 3 Lesion of large and medium joints
- 4 Pain volatility
- 5 The disappearance of pain after taking NSAIDs

- A. true 1, 2
- B. true 1, 2, 3, 5
- C. true 3, 4, 5 .
- D. all of the above is true
- E. all of the above is not true

5. Laboratory diagnostics used for rheumatic fever allows

- 1 Clarify the nature of electrolyte disturbances
- 2 Diagnose rheumatic fevera
- 3 Determine the severity of the inflammatory process
- 4 Detect immunological disorders
- 5 Determine connective tissue disorder

- A. true 1, 2
- B. true 1, 2, 3, 5
- C. true 3, 4 .
- D. all of the above are true
- E. all of the above is not true

6. Mechanisms are involved in the pathogenesis of rheumatic fever

- 1 Sclerosis
- 2 Thrombus formation
- 3 Toxic-inflammatory
- 4 Immune
- 5 Allergic

- A. true 1, 2
- B. true 1, 2, 3, 5
- C. true 3, 4 .
- D. all of the above are true
- E. all of the above is not true

7. Typical signs of subcutaneous rheumatic nodules are

- 1 dense consistency
- 2 painlessness
- 3 localization in the area of the outer surface of the elbow joints, tendons of the hand, Achilles tendons, scalp, sacrum
- 4 reverse development within 2 weeks to 1 month

5 localization in the area of the ankles, Achilles tendons, spinous processes of the vertebrae, the occipital region of the tendon helmet (gallea aponeurotica), on the extensor surfaces of the joints

- A. true 1, 3, 5
- B. true 1, 2, 3, 4
- C. true 1, 2, 3, 4, 6
- D. true 1, 2, 4, 5 .
- E. 5 all of the above is true
- F. 6 all of the above is not true

8. In a patient with mitral stenosis, the electrocardiogram may show:

- 1 wide serrated P wave and deviation of the electrical axis of the heart to the right
- 2 wide serrated P wave and right bundle branch block
- 3 wide serrated P wave and displacement of the transition zone to the left chest leads
- 4 wide serrated P wave and signs of left ventricular hypertrophy

- A. answers 1, 2 and 3 are correct .
- B. answers 1 and 3 are correct
- C. answers 2 and 4 are correct
- D. the correct answer is 4
- E. answers 1, 2, 3 and 4 are correct

9. X-ray examination in patients with mitral stenosis is possible:

- 1.increase in the second arc of the left contour (pulmonary artery trunk);
- 2. an increase in the third arc of the left contour (left atrial appendage);
- 3. the presence of signs of active pulmonary hypertension;
- 4. an increase in the fourth arc along the left contour.

- A. answers 1, 2 and 3 are correct .
- B. answers 1 and 3 are correct
- C. answers 2 and 4 are correct
- D. the correct answer is 4
- E. answers 1, 2, 3 and 4 are correct

Correct answers:

1 D; 2 B; 3 B; 4 C; 5 C; 6 C; 7 D; 8 A; 9 A;

To be able to:

- collect medical history;
- carry out clinical examinations of the patients with acute rheumatic fever;
- make out a plan of instrumental, laboratory methods of examination and interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromuroid, creatinine, urea, aminotransferases, RF. Serological examinations of blood: antistreptolysin O titre, ASG, ASK, anti-DNase B antibody level, immunological blood test: B- and T-lymphocytes, subpopulation of T-lymphocytes, immunoglobulins. Urine clinical analysis (proteins, red blood cells, white blood cells). To describe the ECG, echocardiography data, X-ray examination of the heart;
- carry out differential diagnosis of ARF, rheumatoid arthritis, reactive arthritis, haemorrhagic vasculitis, nonrheumatic myocarditis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;

- prescribed differential treatment for patient with ARF and chronic rheumatic heart disease.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment Place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with Rheumatic Pathology, with Chronic Rheumatic heart disease	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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1. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
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3. <https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/joint-disorders/>
4. https://www.eular.org/recommendations_management.cfm#rec16
5. <http://www.rheumatology.org>

Topic 13. Rheumatoid arthritis.

Duration – 4 hours.

Actuality

Rheumatoid arthritis affects women three times more often than men, and it can first develop at any age. The risk of first disease developing (the disease incidence) appears to be greatest for women between 40 and 50 years of age, and for men somewhat later. RA is a chronic disease, and although rarely, a spontaneous remission may occur, the natural course is almost invariably one of persistent symptoms, waxing and waning in intensity, and a progressive deterioration of joint structures leading to deformations and disability.

Study Objective

To know:

1. Definition.
2. Etiopathogenesis of RA.
3. Joints and internal organs manifestations of RA.
4. Classification of RA.
4. Diagnostic criteria of RA.
5. Differential diagnosis of RA.
6. Disease modifying antirheumatic drugs (DMARD), characteristic of these drugs and symptomatic treatment.

7. Complications of RA and their therapy.
8. Surgical methods of correction and other therapies of RA.
9. "Pulse" therapy during RA, indications for it.
10. Biological agents for RA treatment.

Test questions for testing in practical classes:

- 1. The characteristics of the early stage of rheumatoid arthritis are:**
 - A. Duration of the disease up to 6 months.
 - B. Duration of the disease up to 3 months.
 - C. Duration of the disease from 6 months to 1 year .
 - D. The duration of the disease is more than 1 year
 - E. The duration of the disease is more than 2 years.
- 2. Systemic manifestations of rheumatoid arthritis include:**
 1. Rheumatoid nodules
 2. Necrotizing ulcerative vasculitis
 3. Neuropathy
 4. Dry syndrome
 5. Retinal vasculitis
 - A. Correct 1, 2 and 3
 - B. Correct 1, 3 and 4
 - C. Correct 2, 3 and 5
 - D. Correct 1, 4 and 5
 - E. All of the above is true.
- 3. Signs of stage III radiological manifestations in rheumatoid arthritis according to Steinbrocker are:**
 1. periarticular osteoporosis,
 2. constriction of the joint space
 3. single erosion
 4. multiple erosion
 5. Subluxation of the joints
 - A. Correct 1, 2 and 3
 - B. Correct 2, 4 and 5
 - C. Correct 1, 2, 4 and 5.
 - D. Correct 1, 2, 3 and 5
 - E. All of the above is true
- 4. The characteristic radiological signs of rheumatoid arthritis are:**
 1. Narrowing of the joint space
 2. Periarticular osteoporosis
 3. Subchondral osteosclerosis
 4. Bone erosion
 5. Cystoid enlightenment
 - A. Correct 1, 2 and 4
 - B. Correct 2, 4 and 5
 - C. Correct 2 and 4 .
 - D. Correct 1 and 2
 - E. All of the above is true
- 5. Prevalence of rheumatoid arthritis in the population**
 - A. 0.5-1.5%.
 - B. 0.2-0.5%
 - C. 0.01-0.1%
 - D. 2-5%

E. 0.01-0.02%

6. Complications of rheumatoid arthritis are

- A. osteonecrosis
- B. systemic osteoporosis
- C. secondary systemic amyloidosis
- D. secondary osteoarthritis
- E. all of the above is true.

7. Joints are most common affected in rheumatoid arthritis.

- A. Spine
- B. Knee
- C. Proximal interphalangeal .
- D. Sacroiliac joint
- E. Distal interphalangeal

8. What symptoms are important for early diagnosis of rheumatoid arthritis?

- 1. Morning stiffness
- 2. Tenderness to palpation of the Achilles tendon
- 3. Positive symptom of contraction
- 4. Lateral deviation of the joints of the hands
- 5. Subcutaneous nodules
- 6. Swelling of the proximal interphalangeal joints
 - A. true 1, 2, 3
 - B. true 1, 4, 5
 - C. true 1, 3, 6 .
 - D. true 2, 4, 6
 - E. all of the above is true

9. Among the criteria for the diagnosis of rheumatoid arthritis,

- 1 Morning stiffness
- 2 Arthritis of 3 or more articular areas
- 3 Arthritis of the joints of the hands
- 4 Symmetrical arthritis
- 5 Rheumatoid nodules
- 6 Rheumatoid factor
- 7 Radiological changes

- A. true 5, 6, 7
- B. true 1, 2, 3.
- C. true 3, 4, 5
- D. true 1, 3, 5
- E. true 2, 4, 6

Correct answers

1 C; 2 E; 3 C; 4 C; 5 A; 6 E; 7 C; 8 C; 9 B;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with rheumatoid arthritis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromuroid, creatinine, urea, rheumatoid factor, anti-CCP. Urine clinical analysis (proteins, red blood cells, white blood cells). To describe the ECG, echocardiography data, X-ray examination

of the joints;

- carry out differential diagnosis of rheumatism, psoriatic arthropathy, infectious arthropathies, gout, ankylosing spondylitis, osteoarthritis, Sjogren's syndrome;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment for patients with rheumatoid arthritis.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment Place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with RA.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
2. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016. – Vol. I.
3. <https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/joint-disorders/>
4. https://www.eular.org/recommendations_management.cfm#rec16
5. <http://www.rheumatology.org>

Topic 14. Connective tissue disease. Systemic lupus erythematosus.

Duration – 4 hours.

Actuality

Systemic lupus erythematosus is a chronic, multisystem, inflammatory disorder of autoimmune etiology, occurring predominantly in young women. Common manifestations may include arthralgias and arthritis; malar and other skin rashes; pleuritis or pericarditis; renal or CNS involvement; and haematologic cytopenias. Of all cases, 70 to 90% occur in women (usually of child-bearing age). SLE is more common among blacks and Asians than whites. It can affect patients of any age, including neonates. Increased awareness of mild forms has resulted in a worldwide rise in reported cases. In some countries, the prevalence of SLE rivals that of RA. SLE may be precipitated by currently unknown environmental triggers that cause autoimmune reactions in genetically predisposed people. Some drugs (e.g., hydralazine, procainamide, isoniazid) cause a reversible lupus-like syndrome.

Study Objective

To know:

- 1) Definition of systemic lupus erythematosus;

- 2) Etiological factors and pathogenesis of SLE;
- 3) Classification of SLE;
- 4) Clinical manifestations depending on the damage of organs and systems, disease activity;
- 5) Significance of laboratory and immunologic research methods;
- 6) Differential diagnosis of SLE;
- 7) Complications;
- 8) The main treatment principles for patient with SLE. Indications for prescription of cytostatic therapy. Pulse-therapy;
- 9) Prognosis and working capacity examination.

Test questions for testing in practical classes:

- 1. Systemic lupus erythematosus occurs mainly**
 - A. in men
 - B. in girls, young women .
 - C. in older women
 - D. the disease is not sex-related
- 2. In the pathogenesis of systemic lupus erythematosus,**
 - A. direct effects of infection on tissues
 - B. toxic effects of drugs
 - C. antibody mechanism
 - D. immunocomplex inflammation .
 - E. combination of mechanisms
- 3. The most common morphological type of kidney damage in systemic lupus erythematosus is**
 - A. diffuse lupus glomerulonephritis .
 - B. focal lupus glomerulonephritis
 - C. membranoproliferative glomerulonephritis
 - D. membranous glomerulonephritis
 - E. interstitial nephritis
- 4. For rapidly progressive lupus nephritis, the least common:**
 - A. proteinuria up to 1 g / l .
 - B. nephrotic syndrome
 - C. hypertension
 - D. rapid decline in kidney function
- 5. The most reliable laboratory sign of the lupus nature of nephritis is the detection of**
 - A. increase in ESR
 - B. anemias
 - C. antibodies to native DNA .
 - D. LE cells
 - E. leukopenia
- 6. Differential diagnosis of systemic lupus erythematosus with kidney damage must be carried out**
 - A. with acute glomerulonephritis
 - B. with chronic nephritis
 - C. with extracapillary nephritis
 - D. with bacterial endocarditis
 - E. with all the listed diseases .
- 7. For the treatment of active lupus nephritis, mainly used**
 - A. antibiotics
 - B. immunostimulants
 - C. steroids and cytostatics .
 - D. aminosinoline preparations

E. nonsteroidal anti-inflammatory drugs

8. Pulse therapy for systemic lupus erythematosus allows

A. get a quick effect

B. improve kidney function with activity

C. reduce maintenance dose of corticosteroids

D. reduce the number of complications of steroid therapy

E. all of the above.

Correct answers:

1 B; 2 D; 3 A; 4 A; 5 C; 6 E; 7C; 8 E;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with systemic lupus erythematosus;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea, rheumatoid factor. Antinuclear antibody, antibody to DNA. Urine clinical analysis (protein, red blood cells, white blood cells). To describe the ECG, echocardiography data, X-ray examination of the joints, heart, lungs;
- carry out differential diagnosis of rheumatoid arthritis, polyarthritis nodosa, dermatomyositis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment of SLE.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with SLE.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
2. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016. – Vol. I.
3. <https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/joint-disorders/>
4. https://www.eular.org/recommendations_management.cfm#rec16
5. <http://www.rheumatology.org>

Topic 15. Connective tissue disease. Scleroderma and Dermatomyositis.

Duration – 4 hours.

Actuality

Systemic sclerosis (SSc) is about 4 times more common among women than men. It is most common in the 3rd to 5th decades of life and is rare in children. SSc can develop as part of mixed connective tissue disease.

Immunologic mechanisms and heredity (certain HLA subtypes) play a role in aetiology. SSc-like syndromes can result from exposure to vinyl chloride, bleomycin, pentazocine, epoxy and aromatic hydrocarbons, contaminated rapeseed oil, or l tryptophan.

For polymyositis and dermatomyositis the female : male ratio is 2:1. These diseases may appear at any age but occur most commonly from age 40 to 60 or, in children, from age 5 to 15. The cause seems to be an autoimmune reaction to muscle tissue in genetically susceptible people. Familial clustering occurs, and HLA subtypes DR3, DR52, DR6 seem to be the genetic predisposition. Possible inciting events include viral myositis and underlying cancer. Picornavirus-like structures have been found in muscle cells, but their significance is not known, and viruses can trigger similar disorders in animals. The association of cancer with dermatomyositis (much less so with polymyositis) suggests that a tumor may incite myositis as the result of an autoimmune reaction against a common antigen in muscle and tumor.

Study Objective

To know:

The main theoretical question topics

1. Definition of systemic sclerosis and dermatomyositis;
2. Etiology, pathogenesis;
3. Classification of SS and dermatomyositis;
4. Clinical manifestations depending on the damage of organs and systems, disease activity;
5. Diagnostic criteria of SS and dermatomyositis;
6. Differential diagnosis;
7. Complications;
8. Treatment;
9. Prognosis and working capacity examination.

Test questions for testing in practical classes:

1. **For the clinical picture of systemic scleroderma, in addition to kidney damage, the most characteristic is:**
 - A. Raynaud's syndrome
 - B. heart damage
 - C. pulmonary fibrosis
 - D. damage to the nervous system
 - E. defeat of all specified organs .
2. **In the development of systemic scleroderma, the least importance is attached to:**
 - A. refrigerated
 - B. injuries
 - C. endocrine disorders
 - D. streptococcal infections .
 - E. chemical attack
3. **In the pathogenesis of systemic scleroderma, the most likely is**

- A. direct effects of infection
- B. antibody mechanism
- C. immunocomplex damage .
- D. chemical effects on the skin
- E. mechanical action on the skin

4. The most severe type of kidney injury in systemic scleroderma is

- A. chronic hypertensive glomerulonephritis
- B. chronic nephritis of the nephritic form
- C. true scleroderma kidneys .
- D. chronic nephritis of latent form

5. In true scleroderma kidney, the most rare symptom is:

- A. hematuria
- B. arterial hypertension
- C. retinopathy
- D. intact kidney function .
- E. encephalopathy

6. The prognosis for the life of patients with systemic scleroderma usually determines the lesion

- A. lungs
- B. hearts
- C. kidneys .
- D. serous membranes
- E. nervous system

Correct answers

1 E; 2 D; 3 C; 4 C; 5 D; 6 C;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with systemic scleroderma and dermatomyositis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea, rheumatoid factor. Aminotransferase, bilirubin, phosphatase, CPK, AST, LDH. Immunoglobulins, B- and T-lymphocytes and their populations, RF. Titles antinuclear antibodies (ANA), SCL-70 (topoisomerase I), and anticentromere antibodies, LE-cells. Urinalysis: proteinuria, microhematuria, cylindruria. Investigation of biopsies of skin-muscular graft. To describe the data of ECG, X-ray and ultrasound of internal organs (heart, lungs, bones, gastrointestinal tract, liver);
- carry out differential diagnosis of SLE, polyarthritis nodosa;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment for patients with systemic scleroderma and dermatomyositis.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with Systemic sclerosis, Dermatomyositis.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

REFERENCES

1. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
2. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016. – Vol. I.
3. <https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/autoimmune-rheumatic-disorders>
4. https://www.eular.org/recommendations_management.cfm#rec16
5. <http://www.rheumatology.org>

Topic 16. Systemic Vasculitis.

Duration – 4 hours.

Actuality

Polyarteritis nodosa is a vasculitis of medium and small-sized arteries, which become swollen and damaged by attack of rogue immune cells. Polyarteritis nodosa is also called Kussmaul disease or Kussmaul-Maier disease. The condition affects adults more frequently than children and males more frequently than females. Most cases occur between the ages of 30 and 49. It damages the tissues supplied by the affected arteries because they don't receive enough oxygen and nourishment without a proper blood supply. Polyarteritis nodosa is more common in people with hepatitis B infection.

Study Objective

To know:

The main theoretical question topics

- 1) definition of haemorrhagic vasculitis (Henoch-Schönlein purpura);
- 2) aetiology and pathogenesis;
- 3) clinical signs of haemorrhagic vasculitis;
- 4) classification and diagnostic criteria of hemorrhagic vasculitis;
- 5) the importance of laboratory methods for the diagnosis of hemorrhagic vasculitis.
- 6) the diagnosis structure of hemorrhagic vasculitis.
- 7) differential diagnosis of hemorrhagic vasculitis with other vasculitis;
- 8) varied schemes of pathogenetic and symptomatic treatment of hemorrhagic vasculitis;
- 9) treatment, prognosis and working capacity examination;
- 10) definition of polyarteritis nodosa;
- 11) aetiology and pathogenesis;
- 12) prognosis and working capacity examination;
- 13) definition of polyarteritis nodosa;
- 14) aetiology and pathogenesis;
- 15) clinical signs of polyarteritis nodosa;
- 16) diagnostic criteria;
- 17) differential diagnosis;
- 18) treatment;
- 19) prognosis and working capacity examination.

Test questions for testing in practical classes:

The most common etiological factor of periarteritis nodosa is

- A. infectious
- B. medicinal
- C. genetic
- D. HBs-antigenemia .
- E. chemical

2. The most common pathogenetic mechanism for the development of periarteritis nodosa is

- A. direct action of the microorganism on the vessel wall
- B. direct effect of chemical products on the vessel wall
- C. antibody mechanism of damage
- D. immunocomplex lesion .

3. The main clinical manifestations of periarteritis nodosa are

- A. fever and weight loss
- B. kidney damage
- C. arterial hypertension
- D. peripheral neuritis
- E. all of the above .

- 4. In addition to kidney damage by the type of glomerulitis, periarteritis nodosa may occur**
- A. renal infarction
 - B. ruptured kidneys
 - C. cortical necrosis with acute renal failure
 - D. all of the listed states .
 - E. true A and B
- 5. When morphological examination in the kidneys with periarteritis nodosa is found**
- A. membranous glomerulonephritis
 - B. proliferative glomerulonephritis
 - C. damage to the arteries with the formation of aneurysms and heart attacks in the kidneys
 - D. fibroplastic glomerulonephritis
 - E. mesangioproliferative glomerulonephritis
- 6. In the group of systemic vasculitis, the most common**
- A. periarteritis nodosa
 - B. Wegener's granulomatosis
 - C. hemorrhagic vasculitis .
 - D. nonspecific aortoarteritis (Takayasu disease)
- 7. In the pathogenesis of hemorrhagic vasculitis,**
- A. direct toxic effects
 - B. allergic inflammation
 - C. immunocomplex damage .
 - D. atherosclerotic lesion
- 8. In the pathogenesis of glomerulonephritis in hemorrhagic vasculitis, the greatest importance is**
- A. toxic effects
 - B. allergic effects
 - C. immunocomplex effect .
 - D. bacterial factor
- 9. Glomerulonephritis with hemorrhagic vasculitis most often has to be differentiated**
- A. from Berger Jade .
 - B. from acute glomerulonephritis
 - C. from chronic nephrotic glomerulonephritis
 - D. from chronic hypertensive glomerulonephritis
- 10. Drug therapy of active glomerulonephritis in hemorrhagic vasculitis consists in the predominant appointment**
- A. glucocorticoids in high doses
 - B. nonsteroidal anti-inflammatory drugs
 - C. cytostatics .
 - D. anticoagulants
 - E. symptomatic therapy
- 11. In the pathogenesis of Wegener's granulomatosis, the main role is played by**
- A. allergic inflammation
 - B. toxic damage .
 - C. immune disorders
 - D. circulating immune complexes
 - E. infection
- 12. The main clinical sign that makes it possible to isolate Wegener's granulomatosis in a separate nosological form is**
- A. kidney damage
 - B. lung damage

- C. damage to the upper respiratory tract .
- D. joint damage
- E. skin lesion

13. Morphological signs of nephropathy in Wegener's granulomatosis are

- A. necrotising vasculitis of small and medium arterioles
- B. fibrinoid necrosis
- C. polymorphic cell infiltration .
- D. giant cells
- E. all the above signs

14. The prognosis of patients with Wegener's granulomatosis and their survival depends on:

- A. from timely started treatment
- B. from the choice of drugs
- C. of the duration of the treatment
- D. from drug tolerance and complications
- E. for all named reasons .

Correct answers:

1 D; 2 D; 3 E; 4 D; 5 C; 6 C; 7 C; 8 D; 9 A; 10 C; 11 B; 12 C; 13 C; 14 E;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with systemic vasculitis;
- make out a plan of laboratory and instrumental examination methods, interpret their data

(to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea. Immunological examinations of blood: HBSAg or antibodies to it. Urinalysis: proteinuria, microhaematuria, cylindruria. Investigation of biopsies of skin-muscular graft. To describe the data of ECG, X-ray and ultrasound of internal organs (heart, lungs, bones, gastrointestinal tract, liver);

- carry out differential diagnosis of Wegener's granulomatosis, Churg-Strauss syndrome, Vinivarther-Burger disease, Wegener's granulomatosis, Takayasu's disease, Behchetdisease;

- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment for patients with systemic vasculitis.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients Systemic vasculitis.	125	Case history		Ward, Class room

3	Check survey. Summing up	60	Control questions		Class room
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REFERENCES

1. Davidson's Principles and Practice of Medicine. Edition 21-st The Editors: Nicki R. Colledge, Brian R. Walker, Stuart H. Ralston. – 2010.
2. Goldman-Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer. – 25-th edition. Copyright 2016. – Vol. I.
3. . <https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/vasculitis/>
4. https://www.eular.org/recommendations_management.cfm#rec16
5. <http://www.rheumatology.org>

Topic 22. Ankylosig Spondilitis. Reactive arthritis. Psoriatic arthritis

Duration – 4 hours.

Actuality

Ankylosing spondylitis is a chronic inflammatory disease of the axial skeleton with variable involvement of peripheral joints and nonarticular structures. AS is a form of spondyloarthritis. It is chronic, inflammatory arthritis and autoimmune disease. It mainly affects joints in the spine and sacroiliac joint in the pelvis, and can cause eventual fusion of the spine.

Ankylosing spondylitis (AS) is 3 times more frequent in men than in women and begins most often between ages 20 and 40. It is 10 to 20 times more common among 1st-degree relatives of AS patients than in the general population. The risk of AS in the 1st-degree relatives with the HLA-B27 allele is about 20%. Increased prevalence of HLA-B27 in whites or HLA-B7 in blacks supports a genetic predisposition. However, the concordance rate in identical twins is only about 50%, suggesting that environmental factors contribute.

Study Objective

To know:

The main theoretical question topics

- 1) Definition of ankylosing spondilitis;
- 2) Etiological factors and pathogenesis of AS;
- 3) Classification;
- 4) Clinical signs;
- 5) Significance of additional laboratory and instrumental methods;
- 6) Diagnostic criteria of AS;
- 7) Differential diagnosis of Ankylosing spondylitis and Osteoarthritis;
- 8) Different approaches to symptomatic and pathogenetic treatment of ankylosing spondylitis;
- 9) Treatment of AS;
- 10) Prevention, prognosis and working capacity examination.

Test questions for testing in practical classes:

1. The group of seronegative spondyloarthritis includes:

- 1 ankylosing spondylitis (Bechterew's disease)
- 2 psoriatic arthritis
- 3 pyrophosphate arthropathy
- 4 reactive arthritis
- 5 arthritis associated with bowel disease

- A. true 2, 3, 4, 5
- B. true 1, 2, 3, 4
- C. true 1, 2, 3, 5

- D. true 1, 2, 4, 5 .
- E. all of the above is true
- F. all of the above is not true

2. All seronegative spondyloarthritis have the following similar features

- 1 absence of RF
- 2 arthritis of peripheral joints (often asymmetric)
- 3 signs of sacroiliitis
- 4 association with HLA antigen B 27
- 5 detection of antinuclear antibodies
 - A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4 .
 - C. true 1, 2, 3, 5
 - D. true 1, 3, 4
 - E. all of the above is true
 - 6 all of the above is not true

3. Ankylosing spondylitis (Bechterew's disease) is characterized by:

- 1 night pain in the lumbar region
- 2 bilateral sacroiliitis
- 3 Heberden's nodules
- 4 enthesopathies
- 5 HLA antigen B 27
 - A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5
 - D. true 1, 2, 4, 5 .
 - E. all of the above is true
 - F. all of the above is not true

4. What radiological signs are characteristic of ankylosing spondylitis (Bechterew's disease)?

- 1 symptom of "squaring of the vertebrae"
- 2 presence of syndesmophytes
- 3 osteoporosis of the spine
- 4 unilateral sacroiliitis
- 5 ankylosis of the spine
 - A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5 .
 - D. true 1, 3, 4
 - E. all of the above is true
 - 6 all of the above is not true

5. Persistent unilateral sacroiliitis is characteristic of:

- 1 Reiter's syndrome
- 2 ankylosing spondylitis (Bechterew's disease)
- 3 gout
- 4 deforming osteoarthritis
- 5 systemic lupus erythematosus
 - A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5
 - D. true 1 .

- E. all of the above is true
- F. all of the above is not true

6. Bechterew's disease (ankylosing spondylitis) often affects:

- 1 children
 - 2 old men
 - 3 adolescents and young men (15-30 years old)
 - 4 women in menopause
 - 5 young girls
- A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5
 - D. true 3 .
 - E. all of the above is true
 - F. all of the above is not true

7. Bone cysts are more common for:

- 1 rheumatoid arthritis
 - 2 osteoarthritis
 - 3 Reiter's syndrome
 - 4 ankylosing spondylitis
 - 5 systemic lupus erythematosus
- A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5
 - D. 4 true 2 .
 - E. 5 all of the above is true

8. Ligament and tendon rupture is more common in:

- 1 rheumatoid arthritis
 - 2 osteoarthritis
 - 3 Reiter's syndrome
 - 4 ankylosing spondylitis
 - 5 systemic lupus erythematosus
- A. true 2, 3, 4, 5
 - B. true 1, 2, 3, 4
 - C. true 1, 2, 3, 5
 - D. 4 true 1 .
 - E. 5 all of the above is true
 - F. 6 all of the above is not true

9. Treatment of ankylosing spondylitis includes:

- 1 prescription of NSAIDs in various dosage forms
 - 2 sulfasalazine
 - 3 infliximab
 - 4 chondroprotectors
 - 5 glucocorticosteroids by mouth
- A. 1 is true 2, 3, 4, 5
 - B. 2 true 1, 2, 3 .
 - C. 3 is true 1, 2, 3, 5
 - D. 4 is true 1, 3, 4
 - E. 5 all of the above is true

F. 6 all of the above is not true

10. Specify the main clinical forms of psoriatic arthritis:

- 1 asymmetric oligoarthritis
- 2 arthritis of the distal interphalangeal joints
- 3 symmetrical rheumatoid arthritis
- 4 rhizomelic form
- 5 mutilating arthritis
- 6 psoriatic spondylitis

- A. true 2, 3, 4, 5, 6
- B. true 1, 2, 3, 4, 6
- C. true 1, 2, 3, 5, 6 .
- D. true 1, 3, 4, 5, 6
- E. 5 all of the above is true
- F. 6 all of the above is not true

11. Characteristic X-ray Signs of Psoriatic Arthritis

- 1 narrowing of joint spaces
- 2 edge erosion of articular surfaces
- 3 osteophytes
- 4 osteolysis
- 5 the symptom of the "punch"

- A. true 2, 3, 4, 5
- B. true 1, 2, 3, 4
- C. true 1, 2, 3, 5
- D. true 1, 2, 4 .
- E. 5 all of the above is true
- F. 6 all of the above is not true

12. The clinical signs of reactive arthritis are:

- 1 asymmetric arthritis of the lower extremities
- 2 eye damage
- 3 urethritis or diarrhea
- 4 symmetrical hand arthritis
- 5 keratoderma

- A. true 2, 3, 4, 5
- B. true 1, 2, 3, 4
- C. true 1, 2, 3, 5 .
- D. true 1, 3, 4, 5
- E. 5 all of the above is true
- F. 6 all of the above is not true

13. Typical radiological changes in reactive arthritis are:

- 1 loose heel spurs
- 2 unilateral sacroiliitis
- 3 ankylosis of the spine
- 4 erosive arthritis
- 5 subchondral sclerosis

- A. true 2, 3, 4, 5
- B. true 1, 2 .
- C. true 1, 2, 3, 5
- D. true 1, 3, 4

- E. 5 all of the above is true
- F. 6 all of the above is not true

Correct answers:

1 D; 2 B; 3 D; 4 C; 5 D; 6 D; 7 D; 8 D; 9 B; 10 C; 11 D; 12 C; 13 B;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with ankylosing spondylitis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea, rheumatoid factor. Determination of levels of antigen HLA B27. Urinalysis: proteinuria, microhematuria, cylindruria. To describe ECG, X-ray findings of lumbar, sacral and thoracic spine;
- carry out differential diagnosis of rheumatoid arthritis, psoriatic arthropathy, infectious arthropathies, gout, osteoarthritis, Sjogren's syndrome;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment of patients with AS.

REACTIVE ARTHRITIS

Two forms of reactive arthritis are common: sexually transmitted and dysenteric. The sexually transmitted form occurs primarily in men aged 20 to 40. Genital infections with *Chlamydia trachomatis* are most often implicated. Men or women can acquire the dysenteric form after enteric infections, primarily Shigella, Salmonella, Yersinia, or Campylobacter. Reactive arthritis probably results from joint infection or postinfectious inflammation. Although there is evidence of microbial antigens in the synovium, organisms cannot be cultured from joint fluid.

Epidemiology: the prevalence of the HLA-B27 allele in patients is 63 to 96% vs 6 to 15% in healthy white controls, thus supporting a genetic predisposition.

Psoriasis is a systemic chronic immune-mediated disease that mainly affects the skin and joints. According to the International Federation of Psoriasis Associations, its prevalence in the world ranges from 1.2 to 5% in general populations, averaging about 3%. In Western European countries, the incidence of psoriasis is more than 2%, in other European countries - does not exceed 6% (in Ireland - 5.5%, Spain - 3.7%, Sweden - 2.3%, USA and Canada, these figures 2, 2% and 4.7%, respectively). Simple (vulgar) or plaque psoriasis is the most common form of the disease, which is registered in 80-90% of patients.

In Ukraine, the statistics on the incidence of psoriasis differ significantly from the average in Europe and the world, as the prevalence of the disease has increased steadily in recent decades - from 114.8 per 100 thousand population in 1994 to 222.5 per 100 thousand in 2014 g., among whom were more than 6,000 children. This may be due to both the imperfection of medical and statistical systems, and the underdiagnosis of psoriasis due to the low level of patient treatment

Study Objective

To know:

The main theoretical question topics

- 1) Definition of reactive arthritis (ReA);
- 2) Classification of reactive arthritis;
- 3) Etiology and pathogenesis of reactive arthritis;

- 4) Clinical signs of reactive arthritis depend on different etiology;
- 5) Reiter's syndrome;
- 6) Significance of additional laboratory and instrumental methods;
- 7) Differential diagnosis;
- 8) Modern methods of treatment of reactive arthritis
- 9) Primary and secondary prevention;
- 10) Prognosis, working capacity.
- 11) Definition of psoriatic arthritis (PsA);
- 12) Classification of psoriatic arthritis;
- 13) Etiology and pathogenesis of psoriatic arthritis;
- 14) Additional laboratory and instrumental methods;
- 15) Differential diagnosis;
- 16) Modern methods of treatment of psoriatic arthritis.

To be able to:

- collect medical history;
- carry out clinical examination of the patients with reactive arthritis and psoriatic arthritis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea, rheumatoid factor. Blood test for HLA-antigen. Bacteriological and serological evidence of infection (Yersinia, Disenteria, Salmonella, Chlamidia, Gonorrhoea, etc.). Coprological and serological examination to detect the parasite. Investigation of synovial fluid, X-ray examination of the joints. Urine clinical analysis (proteins, red blood cells, white blood cells). To describe the ECG, echocardiography data;
- carry out differential diagnosis of rheumatism, psoriatic arthropathy, rheumatoid arthritis gout, ankylosing spondylitis, osteoarthritis, Sjogren's syndrome;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment for patients with reactive arthritis.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment Place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with AS, ReA, PsA.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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- disorders/joint-disorders/
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5. <http://www.rheumatology.org>

Topic 18. Gout. Osteoarthritis.

Duration – 4 hours.

Actuality

Gout is precipitation of monosodium urate crystals into tissue, usually in and around joints, most often causing recurrent acute or chronic arthritis. Gout is more common among men than women. Usually, gout develops during middle age in men and after menopause in women. Gout is rare in younger people but is often more severe in people who develop the disorder before age 30. Gout often runs in families. Gout has increased in frequency in recent decades affecting approximately one to two percent of the Western population at some point in their lives. The increase is believed to be due to increasing risk factors in the population, such as metabolic syndrome, longer life expectancy, and changes in diet. Gout was historically known as “the disease of kings” or “rich man's disease”.

Study Objective

To know:

- 1) Definition of gout;
- 2) Etiology and pathogenesis of gout;
- 3) Classification of gout;
- 4) Features of articular syndrome and damage of the internal organs;
- 5) Complications of gout. Principles of gout differential treatment.
- 6) Diagnosis of gout;
- 7) Radiological and laboratory diagnosis of gout.
- 8) Differential diagnosis of gout;
- 9) Principles of gout differential treatment;
- 10) Prevention, prognosis and working capacity

Test questions for testing in practical classes:

1. Select radiological signs characteristic of gouty arthritis:

- 1 round, well-contoured, epiphyseal defects
 - 2 subcortical cysts
 - 3 erosion of articular surfaces
 - 4 compaction of soft periarticular tissues
- A. true 2, 3, 4
 - B. true 1, 3, 4 .
 - C. true 1, 2, 3
 - D. true 1, 2, 4
 - E. 5 all of the above is true
 - F. 6 all of the above is not true

2. Specify changes characteristic of acute gouty arthritis synovial fluid:

- 1 good mucin clot formation
 - 2 low viscosity
 - 3 the presence of urate crystals
 - 4 cytos from 1000 to 5000 leukocytes / ml, neutrophils up to 10%
 - 5 cytos up to 25,000 leukocytes / ml, neutrophils up to 65%
 - 6 presence of rocytes
- A. true 2, 3, 4, 5, 6
 - B. true 1, 2, 3, 4, 6

- C. true 2, 3, 5 .
- D. true 1, 2, 4, 5, 6
- E. all of the above is true
- F. all of the above is not true

3. What diseases are often associated with gout:

- 1 ischemic heart disease
 - 2 stomach ulcer
 - 3 hyperlipidemia
 - 4 obesity
 - 5 arterial hypertension
 - 6 chronic pyelonephritis
- A. true 2, 3, 4, 5, 6
 - B. true 1, 2, 3, 4, 6
 - C. true 1, 2, 3, 5, 6
 - D. true 1, 3, 4, 5 .
 - E. all of the above is true
 - F. all of the above is not true

4. What are the features of gouty arthritis in women:

- 1 development of monoarthritis in onset
 - 2 development of oligo- or polyarthritis in the debut
 - 3 more frequent than in men, the defeat of the joints of the hands in the debut
 - 4 more frequent lesions of the joints of the feet in the debut than in men
- A. true 2, 3, 4
 - B. true 2, 3 .
 - C. true 1, 2, 3
 - D. true 1, 2, 4
 - E. all of the above is true
 - F. all of the above is not true

5. What changes in laboratory parameters are characteristic of an acute attack gout:

- 1 leukocytosis
 - 2 increased C-reactive protein
 - 3 leukopenia
 - 4 increased ESR
- A. true 2, 3, 4
 - B. true 1, 2, 3
 - C. true 1, 3, 4
 - D. true 1, 2, 4 .
 - E. all of the above is true
 - F. all of the above is not true

6. Specify the type of hyperuricemia, the concentration of uric acid in serum lower:

- A. 1 metabolic
- B. 2 renal .
- C. 3 mixed

7. What are the side effects of colchicine:

- 1 diarrhea
- 2 myelosuppression

3 impaired liver function

4 edema

5 depression

6 heartbeat

A. true 2, 3, 4, 5, 6

B. true 1, 2, 3, 4, 6

C. true 1, 2, 3, 5 .

D. true 1, 2, 4, 5, 6

E. all of the above is true

F. all of the above is not true

8. Please note the correct provisions regarding the tactics of prescribing allopurinol:

1 allopurinol is contraindicated in nephrolithiasis

2 allopurinol is contraindicated in an acute attack of gout

3 allopurinol is prescribed at a dose of 50-100 mg per day with titration until the level of uric acid in blood serum normalizes

4 allopurinol is contraindicated in hyperuricosuria

5 it is necessary to provide alkaline diuresis

6 the dose of allopurinol depends on the level of glomerular filtration

A. true 2, 3, 4, 5, 6

B. true 1, 2, 3, 4, 6

C. true 2, 3, 5, 6 .

D. true 1, 2, 4, 5, 6

E. all of the above is true

F. all of the above is not true

9. To stop an acute gout attack, use:

1 colchicine

2 corticosteroids intra-articular

3 GCS systemically

4 probenecid

5 NSAIDs

A. true 2, 3, 4, 5

B. true 1, 2, 3, 4

C. true 1, 2, 3, 5 .

D. true 1, 2, 4, 5

E. all of the above is true

F. all of the above is not true

10. Specify the joints most commonly affected by pyrophosphate arthropathy:

1 hand joints

2 joints of the spine

3 sacroileal joints

4 joints of the foot

5 knee joints

6 hip joints

A. true 2, 3, 4, 5

B. true 1, 2, 3, 4

C. true 1, 2, 3, 5

D. true 1, 2, 5, 6 .

E. all of the above is true

F. all of the above is not true

11. Indicate which joints are rarely affected by gouty arthritis:

- 1 joints of the spine
- 2 elbow joints
- 3 small hand joints
- 4 hip joints

- A. true 2, 3, 4
- B. true 1, 3, 4
- C. true 1, 2, 3
- D. true 1, 4 .
- E. all of the above is true
- F. all of the above is not true

12. Indicate which disease most often leads to the development of secondary gout:

- A. pernicious anemia
- B. chronic myeloid leukemia
- C. chronic renal failure .
- D. erythremia

13. The reasons for the delayed excretion of uric acid by the kidneys include:

- A. chronic renal failure
- B. taking diuretics
- C. dehydration
- D. all of the above .
- E. none of the above

14. Indicate the most frequent localizations of tofus:

- 1 heel tendon
- 2 auricles
- 3 elbow area
- 4 area of the fingers of the hands (around the joints)
- 5 eyelids

- A. true 2, 3, 4, 5
- B. true 2, 3, 4 .
- C. true 1, 2, 3, 5
- D. true 1, 2, 4, 5
- E. 5 all of the above is true
- F. 6 all of the above is not true

Correct answers:

1 B; 2 C; 3 D; 4 B; 5 D; 6 B; 7 C; 8 C; 9 C; 10 D; 11 D; 12 C; 13 D; 14 B;

To be able to:

- collect medical history;
- carry out clinical examination of the patients with gout; osteoarthritis
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, uric acid. To describe the X-ray findings of the joints. The results of synovial fluid. Zimnitsky's, Reberg test;
- carry out differential diagnosis of rheumatoid arthritis, psoriatic arthropathy, infectious arthropathies, ankylosing spondylitis, osteoarthritis;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment for patients with gout.

Osteoarthritis

Osteoarthritis (OA) also known as degenerative arthritis or degenerative joint disease, is a group of mechanical abnormalities involving degradation of joints, including articular cartilage and subchondral bone.

Osteoarthritis affects nearly 27 million people in the United States, accounting for 25% of visits to primary care physicians, and half of all NSAID prescriptions. It is estimated that 80% of the population have radiographic evidence of osteoarthritis by age 65, although only 60% of those will have symptoms. In the United States, hospitalizations for osteoarthritis increased from 322,000 in 1993 to 735,000 in 2006. Osteoarthritis is the most common form of arthritis, and the leading cause of chronic disability in the United States. It affects about 8 million people in the United Kingdom and nearly 27 million people in the United States.

Study Objective**To know:****The main theoretical question topics**

- 1) Definition of osteoarthritis;
- 2) Etiology and pathogenesis;
- 3) Classification of OA;
- 4) Clinical signs of OA depending on the localization;
- 5) Diagnosis of OA;
- 6) Differential diagnosis;
- 7) Treatment;
- 8) Prevention, prognosis and working capacity examination

To be able to:

- collect medical history;
- carry out clinical examination of the patients with osteoarthritis;
- make out a plan of laboratory and instrumental examination methods, to interpret their data (to describe and to estimate changes in clinical blood examinations: parameters of red blood, leukocytes, thrombocytes, erythrocyte sedimentation rate (ESR)). Biochemical blood tests: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, describe the X-ray findings of the joints. The results of synovial fluid;
- carry out differential diagnosis of rheumatic diseases, psoriatic arthropathy, infectious arthropathies, gout, ankylosing spondylitis, osteoarthritis, Sjogren's syndrome;
- make diagnosis formulation and individual plan of treatment of the supervised patient;
- prescribe differential treatment of patients with osteoarthritis.

Educational Objectives

To develop a sense of responsibility for the accuracy of professional activities that help to

establish the diagnosis with regard to all cause-effect relations that will improve treatment effectiveness and, consequently, duration and quality of patients' life.

1. Process map of classes

№	Stage	Minutes	Study Materials		Employment place
			learning equipment	equipment	
1	Determining the initial level by using the test control	15	Tests		Class room
2	Thematic analysis of patients with Gout, Osteoarthritis.	125	Case history		Ward, Class room
3	Check survey. Summing up	60	Control questions		Class room

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- <http://www.rheumatology.org>

Module 3. Basics of diagnosis, treatment and prevention of major diseases of the genitourinary system

Topic 19. Glomerulonephritis and nephrotic syndrome

Duration: 6 hours

Actuality. Glomerulonephritis (GN) is a heterogeneous group of diseases characterized by an inflammatory process that affects only or mainly the glomeruli. Changes in other structures of the kidneys (tubules, stroma and blood vessels) are secondary and are the result of disorders (of proteinuria mainly) caused by dysfunction of the glomeruli. The basis for the inflammatory process is a violation of immunological processes, and the causes and pathogenesis in many cases are unknown.

In Ukraine, the incidence of acute glomerulonephritis is 10.9 per 100 thousand population in urban and 13.8 in rural areas, chronic - 15.1 and 21.9, respectively. In recent years, there has been an increase in the number of patients with glomerulonephritis both worldwide and in Ukraine. Medico-social significance of glomerulonephritis is determined by the lesion of mostly young people, the severity of the course and, often, an unfavorable prognosis. Mortality from glomerulonephritis in different countries ranges from 3.1 to 5-6 people per 1 million population per year, with the maximum being at the age of 20-40 years.

Nephrotic syndrome is a clinical condition characterized by daily protein loss in the urine > 3.5 g / 1.73 m², as well as hypoalbuminemia, lipidation, hyperlipidemia and edema.

NS can accompany both primary and secondary kidney diseases. NS usually accompanies acute and chronic forms of GN and rapidly progressing GN, but its development can be observed in diabetic nephropathy, systemic connective tissue lesions and amyloidosis.

The prognosis is due to the peculiarities of the disease, on the background of which kidney damage develops, the duration and frequency of NS relapses, the addition of arterial hypertension and complications.

Thus, the problem of effective diagnosis and treatment of GN and NS is relevant and requires detailed study.

The general goal is to be able to make a preliminary diagnosis and determine the tactics of management of patients with glomerulonephritis and nephrotic syndrome.

Specific goals

Students should know: 1. Definition of the term "glomerulonephritis". Etiology, role of streptococcal infection and immunological disorders in the development of the disease. Pathogenesis of the main clinical syndromes. Classification. Clinical manifestations and diagnosis of individual forms. Differential diagnosis. Complications (eclampsia, acute renal and chronic renal failure, etc.). Treatment, considering the morphological variant and clinical course. Primary and secondary prevention. Forecast and efficiency.

2. Definition, etiology, pathogenesis of nephrotic syndrome. Clinical manifestations. Diagnostic criteria and differential diagnosis. Complication. Treatment. Primary and secondary prevention. Forecast and efficiency.

Students must be able to:

- collect the anamnesis, to carry out its analysis;
- conduct a clinical examination of patients with glomerulonephritis and nephrotic syndrome;
- make a plan of laboratory and instrumental methods of examination and interpret their data (describe and evaluate changes during a clinical blood test: blood counts; leukocytes; platelets; ESR indicators). Blood biochemical data: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromucoid, creatinine, urea, aminotransferases, cholesterol, β -lipoproteins, type of lipidemia, bilirubin. Immunological examination of blood: B- and T-lymphocytes, subpopulations of T-lymphocytes, immunoglobulins, RF, LE-cells. Determination of blood electrolytes (potassium, calcium, phosphorus, sodium, chlorine). Clinical examination of urine (protein, erythrocytes, leukocytes), Reberg tests, urine analysis according to Nechiporenko, Zymnitsky, determination of daily proteinuria, urinary cultures;
- evaluate radiographs of the kidneys and urinary tract (review and contrast), intravenous urography, analysis of ultrasound of the kidneys and urinary tract, radioisotope examination of the kidneys, kidney biopsy;
- make a differential diagnosis with pyelonephritis, amyloidosis, diffuse connective tissue diseases, renal tuberculosis, malignant hypertension;
- formulate the final clinical diagnosis and to make the individual plan of treatment of the supervised patient, being based on inspection of the patient;
- prescribe differentiated therapy to a patient with glomerulonephritis, nephrotic syndrome.

Examples of control questions for testing in practice

1. Patient M., 26 years old, complains of general weakness, itchy skin, headache, decreased visual acuity, constant nausea and vomiting. He has been ill for a year. Objective: the condition is severe, the skin is pale and dry. The boundaries of the heart are shifted to the left, the heartbeat is diffuse. Heart sounds are weakened, the "gallop rhythm" and systolic murmur. Rightside, in the subscapular area - the noise of friction of the pleura, hard breathing. In the blood test: hemoglobin 76 g / l, erythrocytes 2.8×10^{12} / l, leukocytes 11.4×10^9 / l. ESR 56 mm / year. In urine: relative density 1008, protein 1.65 g / l, erythrocytes - 15-20, leukocytes - 5-6 in the field of view, hyaline and granular cylinders. Creatinine 1.1 mmol / l, glomerular filtration 15 ml / min What is your diagnosis?

- A) Exacerbation of chronic pyelonephritis
- B) Acute glomerulonephritis

- C) Acute pyelonephritis
- D) Chronic glomerulonephritis
- E) Renal amyloidosis

2. A 45-year-old patient has been suffering from chronic glomerulonephritis for 8 years. Blood pressure - 180/120 mm Hg, serum creatinine - 970 $\mu\text{mol} / \text{l}$, blood urea - 28 mmol / l, glomerular filtration - 5 ml / min. What treatment tactics are indicated for this patient?

- A) Hemosorption
- B) Peritoneal dialysis
- C) Hemodialysis
- D) Plasmapheresis
- E) Hemofiltration

3. A patient with a long course of GN has facial edema, proteinuria (2.5 g / day), blood creatinine - 0.108 mmol / l, urea - 8.3 mmol / l, relative urine density - 1024, daily diuresis - 600 ml, night - 1200 ml during the year. Assess the functional state of the kidneys:

- A) Not violated
- B) Nocturia
- C) CRF stage I
- D) CRF stage II
- E) CRF stage III

4. The patient is 37 years old, has been suffering from chronic glomerulonephritis for the last 3 years. Swelling of the face, lower back, legs, shortness of breath at night, severe itching of the skin, loss of appetite appeared two weeks ago after a viral infection. Objective: dry skin, pericardial friction noise. BP - 200/120, HB-86 g / l; blood creatinine-1.03 mmol / l. Which of these drugs are contraindicated for this patient?

- A) Glucocorticosteroids
- B) Anabolic hormones
- C) Calcium gluconate
- D) Reosorbilact
- E) Furosemide

5. A 48-year-old woman complains of weakness, weight loss, loss of appetite, headache. In her youth she suffered from acute glomerulonephritis. From the age of 25 she suffers from arterial hypertension. She was not treated systematically, she rarely consulted a doctor. The study revealed signs of chronic renal failure stage I. What dietary recommendations are most justified for this patient?

- A) restriction of carbohydrates
- B) protein restriction
- C) fat restriction
- D) fluid restriction
- E) no restrictions are required

6. A 68-year-old patient has been suffering from chronic GN for 15 years. In the last 3 months he has been complaining of inspiratory dyspnea during exercise, palpitations, intermittent pain in the heart without irradiation, general weakness. Objectively: Acrocyanosis on the background of pale skin. Pulse 104 / min, rhythmic, intense. BP 190/110 mm. The left border of the heart is 2 cm outward from the left midclavicular line, above the apex of the first tone of the heart is weakened, systolic murmur, the accent of the second tone above the aorta. On the ECG there was a deviation of the electrical axis of the heart to the left, dysmetabolic changes. Blood: Hb - 66 g / l, creatinine 1.1 mmol / l. Relative density of urine 1003. The most probable cardiac diagnosis?

- A) Insufficiency of the aortic valve
- B) uremic myocardial dystrophy
- C) Subacute myocarditis

- D) Exudative pericarditis
 E) Hypertensive disease
- 7. A patient with chronic glomerulonephritis with arterial hypertension complained of headache, shortness of breath during exercise. On examination: blood pressure 170/110 mm Hg. Art., glomerular filtration 60 ml / min., blood creatinine 0.2 mmol / l, potassium 4.7 mmol / l, cholesterol 5.6 mmol / l. The appointment of which antihypertensive agent is most appropriate?**
- A) Enalapril
 B) Nifedipine
 C) Hypothiazide
 D) Atenolol
 E) Clonidine
- 8. What is the most characteristic of chronic glomerulonephritis on ultrasound?**
- A) Enlargement of the cortical layer
 B) The presence of urate crystals
 C) The presence of oxalate crystals
 D) Increased kidney size
 E) Thinning of the cortical layer
- 9. Patient P., 31 years old, complained of headache, nausea, and swelling of the legs. The hospital diagnosed chronic glomerulonephritis with chronic renal failure. Which of the following drugs has antiazotemic action?**
- A) Prednisolone
 B) Furosemide
 C) Riboxin
 D) Lespenephriil
 E) Spironolactone
- 10. A 42-year-old man suffers from chronic kidney disease with hypertension. In recent months, he has noted weakness, apathy, dry and itchy skin, nocturia. At examination - blood creatinine 920 μmol / l, glomerular filtration rate - 10 ml / min. Determine the functional state of the kidneys**
- A) Not violated
 B) Acute renal failure
 C) Terminal uremia
 D) Chronic renal failure stage I
 E) Chronic renal failure stage II

Correct answers

Question number	1	2	3	4	5	6	7	8	9	10
Correct answer	E	C	A	A	B	B	A	E	D	C

The main theoretical issues of the topic

1. Define the term "glomerulonephritis".
2. Classification of glomerulonephritis.
3. Etiology, role of streptococcal infection and immunological disorders in the development of the disease.
4. Clinical manifestations of glomerulonephritis.
5. The main syndromes of glomerulonephritis.
6. Differential diagnosis.
7. Complications (infectious complications, atherosclerosis, stroke, acute renal and chronic renal failure, etc.).

8. Treatment, considering the morphological variant and clinical course.
9. Primary and secondary prevention of glomerulonephritis.
10. Definition, etiology, pathogenesis of nephrotic syndrome.
11. Clinical manifestations.
12. Criteria for diagnosis of nephrotic syndrome and differential diagnosis
13. Complications (thrombosis, thromboembolism, cerebral edema, acute renal failure, nephrotic crisis).
14. Treatment of nephrotic syndrome.
15. Primary and secondary prevention

Technological map of the practice session

№	Stage	Minutes	Training manuals		Venue
			Teaching aids	Equipment	
1	Determination of the initial level using test control.	15	Tests		Classroom
2	Thematic analysis of patients with glomerulonephritis.	200	Medical histories		Ward, study room
3	Control survey. Summing up.	25	Control questions		Classroom

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Topic 20. Pyelonephritis, tubulointerstitial nephritis, renal amyloidosis

Duration: 4 hours

Actuality. Pyelonephritis is an infectious-inflammatory disease of the kidneys with a predominant lesion of the pelvi-calyceal system (PCS), tubulointerstitial tissue and often involving the glomerular apparatus.

Pyelonephritis is the most common disease of the urinary system in all age groups. 0.9-1.3 million new cases of acute pyelonephritis are registered annually among the population of Ukraine.

Chronic pyelonephritis is a delayed bacterial inflammation that periodically worsens and leads to irreversible changes in PCS with subsequent sclerosis of the parenchyma and kidney shrinkage. This disease is characterized by focal and polymorphism of the inflammatory process in the kidney.

Tubulointerstitial nephritis (TIN) is a heterogeneous group of nonspecific lesions of the tubules and interstitial tissue of the kidney with subsequent spread of the inflammatory process of infectious, allergic or toxic origin to all structures of renal tissue, characterized by acute or chronic course. According to clinical studies, the proportion of TIN ranges from 4 to 12%.

Amyloidosis - a group of diseases, the main feature of which is the deposition of fibrillar glycoprotein – amyloid in the tissues and organs.

The average incidence of amyloidosis in the population is 1 case per 50 thousand population. Amyloidosis usually develops in people older than 50-60 years.

All these factors require correct and timely diagnosis of pyelonephritis, tubulointerstitial nephritis and renal amyloidosis, which has not only clinical but also social significance.

The general goal is to be able to make a preliminary diagnosis and determine the tactics of management of patients with pyelonephritis, tubulointerstitial nephritis and renal amyloidosis..

Specific goals

Students should know: 1. Definition of pyelonephritis. The role of infection in inflammatory diseases of the kidneys and urinary tract. Primary and secondary pyelonephritis. Clinical manifestations. Instrumental and laboratory diagnostic methods. Differential diagnosis. Complication. Treatment. Primary and secondary prevention. Forecast and efficiency.

2. Definition, etiology, pathogenesis of tubulointerstitial nephritis. Clinical manifestations. Diagnostic criteria and differential diagnosis. Complication. Treatment. Emergency care for acute renal failure. Primary and secondary prevention. Forecast and efficiency.

3. Definition, etiology, pathogenesis of amyloidosis. Classification. Clinical manifestations of renal amyloidosis. Diagnostic criteria. Differential diagnosis. Complication. Treatment. Primary and secondary prevention. Prognosis and efficiency.

Students must be able to:

- collect the anamnesis, to carry out its analysis;
- conduct a clinical examination of patients with pyelonephritis and amyloidosis;
- make a plan of laboratory and instrumental methods of examination and interpret their data (describe and evaluate changes during a clinical blood test: red blood cells; leukocytes; platelets; ESR). Blood biochemical data: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromuroid, creatinine, urea, aminotransferases, cholesterol, β -lipoproteins, type of lipidemia, bi. Immunological examination of blood: B- and T-lymphocytes, subpopulations of T-lymphocytes, immunoglobulins, RF, LE-cells. Clinical examination of urine (protein, erythrocytes, leukocytes), Reberg test, analysis by Nechiporenko, Zymnitsky, determination of daily proteinuria, urine culture with determination of sensitivity to antibiotics, uroleukogram;
- evaluate radiographs of the kidneys and urinary tract (review and contrast), analyse the ultrasound of the kidneys and urinary tract, radioisotope examination of the kidneys, kidney biopsy;
- make a differential diagnosis of pyelonephritis with glomerulonephritis, renal tuberculosis, asymptomatic bacteriuria; differential diagnosis of amyloidosis;

- formulate the final clinical diagnosis and to make the individual plan of treatment of the supervised patient, being based on inspection of the patient;
- appoint differentiated therapy to the patient with pyelonephritis, TIN, amyloidosis.

Examples of control questions for testing in practice

1. The 36-years-old patient became acutely ill. Temperature is 38.50 C, chills, dull pain in the lumbar region, frequent painful urination. Objectively: tension of muscles of lumbar department, a positive symptom of Pasternatsky from both parties is noted. General blood test: leukocyte. $20.0 \times 10^9 / l$, neutrophilia. In the analysis of urine: protein 1.6 g / l, leukocytes - the whole field of view, bacteriuria 2.5×10^6 microbial bodies in 1 ml of urine. What is your previous diagnosis?

- A) Acute pyelonephritis
- B) Acute glomerulonephritis..
- C) Exacerbation of chronic pyelonephritis
- D) Acute cystitis
- E) Urolithiasis.

2. A 55-year-old man complains of general weakness, decreased urination, itchy skin. He has been suffering from chronic pyelonephritis for 15 years. Objectively: the skin is dry, with a yellowish tinge. PS -80 per minute, rhythmic, blood pressure -100/70 mm Hg At auscultation heart tones are deaf, the noise of friction of a pericardium is listened. Blood creatinine -1.1 mmol / l, glomerular filtration 5 ml / min. What treatment is indicated for the patient??

- A) Hemodialysis
- B) Plasmapheresis
- C) Neohemodesis
- D) Enterosorbent
- E) Diuretics

3. All of these organs are most often affected in senile amyloidosis, except:

- A) The brain
- B) Hearts
- C) Aorta
- D) Kidney
- E) Pancreas

4. What does prevent the preservation of the pathogen in the urinary tract and chronization of pyelonephritis?

- A) the presence of protoplasts and L-forms of bacteria;
- B) the phenomenon of bacterial adhesion;
- C) "physiological" obstruction of the urinary tract;
- D) the appointment of antibiotics;
- E) synthesis of urinary antibodies.

5. In the first trimester of pregnancy for the treatment of pyelonephritis can be prescribed:

- A) semi-synthetic penicillins;
- B) tetracycline;
- C) biseptol;
- D) furagin;
- E) fluoroquinolones.

6. A 27-year-old woman complains of general weakness, swelling of the eyelids in the morning. She has been ill for 3 years. She had a fever and a headache three days ago. Objectively: body temperature is 37.8 ° C, breathing rate is 18 per 1 min, pulse 86 per 1 min, blood pressure 150 \ 95 mm Hg. The skin is pale, the face is brittle, the feet are pasty. Moderate pain on palpation in the costal-vertebral angle on both sides. In the blood: Hb 115 g \ l, erythrocytes $3.3-10^{12} \ l$; $1.9,9 * 10^9 \ l$; n.9%; p.58%; lymph.31%; mon.2%; ESR 21 mm / year. Urine: cloudy, relative density 1015; glucose-negative; protein 0.132 g \ l.

Sediment microscopy: l. 55-60 in sight; er. 5-7 in sight. What antibacterial treatment should be prescribed:

- A) erythromycin;
- B) gentamicin;
- C) penicillin;
- D) tetracycline.

7. The 32-year-old patient consulted a physician about almost constant low-grade fever, dull pain in the left lumbar region, and increased diuresis. She notes nocturia, suffers from chronic adnexitis, has a child of 2 years. Blood pressure 160/110 mm Hg, diuresis 1900 ml. Blood test: Hb- 105 g \ l; er. 3.6 * 10¹² \ l; ESR 18 mm / year. Urine analysis: relative density 1010; protein 0.066 g \ l; l. 20-25 in sight; erythrocytes 1-2 in the field of view. The most likely diagnosis:

- A) acute glomerulonephritis;
- B) chronic glomerulonephritis;
- C) chronic pyelonephritis;
- D) renal amyloidosis;
- E) chronic cystitis.

8. A 50-year-old patient complains of pain in the left lumbar region, low-grade fever. The excretory program revealed an enlarged and deformed pelvi-calyceal apparatus of both kidneys, more on the left, a violation of their tone. Which disease is most likely:

- A) chronic pyelonephritis;
- B) chronic glomerulonephritis;
- C) amyloidosis of the kidneys;
- D) diabetic glomerulosclerosis;
- E) polycystic kidney disease.

9. The patient, 24 years old, was treated for hypostatic pneumonia after polytrauma. Massive doses of antibiotics, sulfonamides, NSAIDs, antihistamines, expectorants, cardiotonics were prescribed. On the 20th day, the acute renal failure developed. What did cause it:

- A) sulfonamides;
- B) cardiotonics;
- C) antibiotics;
- D) expectorants;
- E) NSAIDs.

10. The 42-year-old patient complained of aching low back pain, more on the right side, sometimes a rise in body temperature to subfebrile numbers, and a headache. Ten years ago, during pregnancy, the patient had an attack of low back pain on the right side, which was accompanied by a sharp rise in temperature. Five years ago, there was an increase in blood pressure to 200/110 mm Hg. Urine analysis: protein 0.99 g / l, l. 50-60 in sight, er. 2-4 in the field of view, hyaline cylinders 1-2 in the field of view. Creatinine 102 μmol / l. Diagnose:

- A) renal amyloidosis,
- B) chronic pyelonephritis,
- C) renal tuberculosis,
- D) chronic pyelonephritis,
- E) hypertensive nephropathy.

Correct answers:

Question number	1	2	3	4	5	6	7	8	9	10
Correct answer	A	A	E	E	A	C	C	A	A	D

The main theoretical issues of the topic

1. Determination of pyelonephritis.
2. The role of infection in inflammatory diseases of the kidneys and urinary tract.
3. Primary and secondary pyelonephritis.
4. Clinical manifestations of pyelonephritis.
5. Instrumental and laboratory methods for the diagnosis of pyelonephritis.
6. Differential diagnosis of pyelonephritis.
7. Complications of pyelonephritis.
8. Treatment of pyelonephritis.
9. Primary and secondary prevention. Prognosis and efficiency in pyelonephritis.
10. Definition, etiology, pathogenesis of tubulo-interstitial nephritis.
11. Clinical manifestations of tubulo-interstitial nephritis.
12. Diagnostic criteria and differential diagnosis of tubulo-interstitial nephritis.
13. Complications of tubulo-interstitial nephritis.
14. Treatment of tubulo-interstitial nephritis.
15. Emergency care for acute renal failure.
16. Primary and secondary prevention. Prognosis and efficiency of tubulo-interstitial nephritis.
17. Definition, etiology, pathogenesis of amyloidosis.
18. Classification of amyloidosis.
19. Clinical manifestations of renal amyloidosis.
20. Criteria for the diagnosis of amyloidosis.
21. Differential diagnosis of amyloidosis.
22. Complications of amyloidosis.
23. Treatment of amyloidosis.
24. Primary and secondary prevention. Prognosis and efficiency in amyloidosis.

Technological map of the practice session

№	Stage	Minutes	Training manuals		Venue
			Teaching aids	Equipment	
1	Determination of the initial level for the diagnosis of kidney disease	15	Tasks		Classroom
2	Thematic analysis of patients with pyelonephritis, tubulo-interstitial nephritis and amyloidosis	120	Medical histories		Ward, classroom
3	Summing up	25	Tasks		Classroom

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Topic 21. Acute and chronic renal failure

Duration: 4 hours

Actuality. Acute renal failure (ARF) is a syndrome that occurs due to an acute decrease in glomerular filtration and is manifested by acute disorders of kidney-regulated homeostasis parameters. ARF often develops in patients during a critical condition and, being in such cases an independent factor of risk for death, is associated with high mortality. The total incidence of renal failure is about 200 people per 1 million population per year, of which the most able-bodied age (18-50 years) is about 100 people, which determines the social significance of the disease.

Chronic renal failure (CRF) is a pathological symptom complex that develops in chronic kidney disease due to a gradual decrease in the number and function of nephrons, which leads to impaired excretory and incretory functions of the kidneys, homeostasis, disorders of all types of metabolism, acid-base balance, activity of all organs and systems

Thus, the problem of effective diagnosis and treatment of acute and chronic renal failure is relevant and requires detailed study.

The general goal is to be able to make a preliminary diagnosis and determine the management of patients with acute and chronic renal failure.

Specific goals

Students should know: 1. Definition of the term «acute renal failure». Etiology. Pathogenesis of the main clinical syndromes. Classification. Clinical manifestations and diagnosis. Differential diagnosis. Complication. Treatment. Primary and secondary prevention. Forecast and efficiency.

2. Definition, etiology, pathogenesis of chronic renal failure. Clinical manifestations. Diagnostic criteria and differential diagnosis. Complication. Treatment. Primary and secondary prevention. Forecast and efficiency.

- Students must be able to:

-collect the anamnesis, to carry out its analysis;

-conduct a clinical examination of patients with acute and chronic renal failure;

- make a plan of laboratory and instrumental methods of examination and interpret their data (describe and evaluate changes during a clinical blood test: red blood cells; leukocytes; platelets; ESR). Blood biochemical data: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromuroid, creatinine, urea, cholesterol, β -lipoproteins, type of lipidemia, bilirubin. Determination of blood electrolytes (potassium, calcium, phosphorus, sodium, chlorine). Clinical study of urine (protein, erythrocytes, leukocytes, glucose), Reberg tests (determination of GFR), analysis by Nechiporenko, Zymnysky, determination of daily protein excretion, urine culture with determination of sensitivity to antibiotics, uroleukogram;

- evaluate radiographs of the kidneys and urinary tract (review and contrast), analysis of ultrasound of the kidneys and urinary tract, radioisotope examination of the kidneys, kidney biopsy;

- make a differential diagnosis with kidney diseases that led to the development of CRF (glomerulonephritis, pyelonephritis, amyloidosis, diabetes, systemic connective tissue diseases, renal tuberculosis);
- formulate the final clinical diagnosis and to make the individual plan of treatment of the supervised patient, being based on inspection of the patient;
- prescribe differentiated therapy to a patient with CRF and ARF.

Examples of control questions for testing in practice

1. Patient V., 46 years old, complains of increased fatigue, periodic headache, general weakness. Objectively: pulse 88 beats / min., Blood pressure 140/80 mm Hg, swelling of the legs. In the blood test, creatinine 0.13 $\mu\text{mol} / \text{l}$, urea 9.0 mmol / l. glomerular filtration 80 ml / min What can you think about?

- A) CRF stage III.
- B) CHF stage II.
- C) CHF stage I.
- D) CRF stage II.
- E) CRF stage I.

2. A 42-year-old man suffers from chronic kidney disease with hypertension. In recent months, he has noted weakness, apathy, dry and itchy skin, nocturia. At examination - blood creatinine 520 $\mu\text{mol} / \text{l}$, glomerular filtration rate - 40 ml / min. Determine the functional state of the kidneys:

- A) Not violated
- B) Acute renal failure
- C) Terminal uremia
- D) Chronic renal failure stage I
- E) Chronic renal failure stage II

3. The clinical and laboratory signs of CRF include everything except:

- A) Hypercreatininemia
- B) Erythrocytosis
- C) Acceleration of ESR
- D) Insomnia
- E) Itchy skin

4. Facial edema, proteinuria (2.5 g / day), blood creatinine - 0.238 mmol / l, urea - 18.3 mmol / l are observed during the year in a patient with a long course of GN. Assess the functional state of the kidneys:

- A) CRF stage I
- B) CRF stage II
- C) Not violated
- D) CRF stage III
- E) Terminal uremia

5. The morphological substrate of chronic renal failure is:

- A) Nephrosclerosis.
- B) Proliferation of mesangial cells
- C) Destruction of small processes of podocytes
- D) Deposition of immune complexes in the glomerular basement membrane
- E) Obliteration of the external arterioles.

6. A headache, shortness of breath when walking, poor appetite, nausea, weakness appeared in a 40-year-old man 4 months ago. He has been suffering from chronic glomerulonephritis for 18 years. It was suspected that the course was complicated by chronic renal failure. Which laboratory indicator will be the most informative in this case?

- A) uric acid - 0,350 mmol / l
- B) blood potassium - 5.2 mmol / liter

- C) blood creatinine 0.36 mmol / liter
- D) blood urea - 9.2 mmol / liter
- E) blood sodium-135 mmol / liter

7. In a patient with chronic GN, facial edema, proteinuria (2.5 g / day), blood creatinine - 0.338 mmol / l, urea - 18.3 mmol / l are registered during the year. Assess the functional state of the kidneys:

- A) Not violated
- B) Renal failure
- C) Nephrotic syndrome
- D) Impaired concentration function of the kidneys
- E) Violation of homeostatic function of the kidneys

8. Hyperkalemia in acute renal failure can be eliminated by:

- A) Isotonic solution;
- B) Hypotonic solution;
- C) Intravenous introduction of glucose solution with insulin;
- D) The introduction of a solution of sodium bicarbonate;
- E) The introduction of a solution of calcium chloride.

9. What does the term "hypostenuria" mean?

- A) Decrease in relative density of urine
- B) Monotony of the relative density of urine;
- C) Decrease in minute diuresis;
- D) Increase in relative density of urine;
- E) Decrease in minute diuresis at the increased relative density of urine.

10. Prerenal factor of acute renal failure is:

- A) Acute pyelonephritis;
- B) A sharp decrease in the volume of circulating blood;
- C) Obstruction of the urinary tract by a stone;
- D) Thrombosis and embolism of the renal arteries.

The main theoretical issues of the topic

1. To define, name etiological factors of development of acute renal failure.
2. Clinic and changes in laboratory parameters depending on the stage
3. Treatment of ARF at different stages: diet, drug treatment, dialysis therapy
4. To define, name etiological factors of development of chronic renal failure.
5. Pathogenesis of lesions of organs and systems, their clinical manifestations.
6. Define the concept of "chronic kidney disease". Classification depending on GFR and urea, creatinine levels.
7. Clinic and changes in laboratory parameters depending on the stage.
8. Laboratory and instrumental diagnosis of chronic renal failure
9. Differential diagnosis (glomerulonephritis, pyelonephritis, amyloidosis, diabetes mellitus).
10. Treatment of CRF at different stages: diet, drug treatment, dialysis therapy.
11. Renal replacement therapy: hemodialysis, peritoneal dialysis, kidney transplantation.
12. Indications and contraindications to renal replacement therapy, complications.
13. Features of management of patients with end-stage renal disease.
14. Primary and secondary prevention of CRF and renal failure.
15. Forecast and efficiency.

Answers to control questions.

№	1	2	3	4	5	6	7	8	9	10
Correct answe	E	E	B	B	C	C	B	C	A	B

Technological map of the practice session

№	Stage	Minutes	Training manuals		Venue
			Teaching aids	Equipment	
1	Determination of the initial level using test control	15	Test		Classroom
2	Thematic analysis of patients with ARF and CRF	120	Medical histories		Ward, classroom
3	Control survey. Summing up	25	Control questions		Classroom

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6. Protocol of medical care for patients with chronic renal failure. Access mode: <http://medstandart.net/browse/1660>.

Module 4. General issues of internal medicine

Topic 22. Obesity and its consequences. Diagnostics and treatment of the elderly

Duration: 4 hours

Actuality. The World Health Organization views obesity as an epidemic that has affected millions of people. Obesity is a metabolic disorder and a serious social problem in economically developed countries. Currently, in most countries of Western Europe and the United States, 20-25% of the population suffer from obesity (BMI > 30). The social significance of the problem of obesity is determined by the threat of disability of young patients and the reduction of overall life expectancy due to the frequent development of severe comorbidities. These include type 2 diabetes, hypertension, dyslipidemia, atherosclerosis and related diseases, sleep apnea, hyperuricemia, gout, reproductive dysfunction, gallstones, osteoarthritis, varicose veins of the lower extremities. Weight loss helps reduce the risk of coronary heart disease and cerebral infarction.

Treatment of elderly patients is often quite a difficult task, as it is complicated by a large

number of comorbidities, the need to prescribe many drugs (polypragmatism), social factors (loneliness, helplessness, poverty), cognitive dysfunction, low ability to learn and lack of habituation to constant medication (low compliance). The course of most diseases in old age is characterized by rapid deterioration, frequent development of complications caused by both the disease and carried treatment.

Thus, given the increase in the number of obese patients in Ukraine, the problem of effective diagnosis and treatment remains relevant and requires detailed study.

The general goal is to be able to diagnose and determine the tactics of management of obese and elderly people.

General goals

Students should know: 1. Definition, etiology, pathogenesis, forms of obesity. Clinical manifestations. Diagnostic criteria and differential diagnosis. Complication. Treatment. Primary and secondary prevention. Forecast and efficiency. Features of metabolism in old age. Frequency of comorbid pathology in the elderly. Features of action of drugs on an organism of the elderly person. Features of diagnostics of diseases at advanced age. Features of treatment in old age.

Students must be able to:

- collect the anamnesis at the patient with obesity, to carry out its analysis;
- conduct a clinical examination of obese patients, identify the main syndromes and symptoms;
- make the plan of laboratory-instrumental methods of inspection and to interpret their data (to describe and estimate changes at clinical research of blood: indicators of red blood; leukocytes; thrombocytes; indicators of ESR). Blood biochemical data: total protein, protein fractions, fibrinogen, fibrin, C-reactive protein, haptoglobin, sialic acid, seromuroid, creatinine, urea, aminotransferases, cholesterol, β -lipoproteins, type of lipidemia, white. Clinical examination of urine (protein, erythrocytes, leukocytes, glucose). Describe ECG, echocardiography, ultrasound of the abdominal cavity, X-ray examination of joints.
- conduct a clinical examination of elderly patients;
- formulate the final clinical diagnosis and to make the individual plan of treatment of the supervised patient of advanced age, being based on inspection of the patient;
- prescribe differentiated therapy to an elderly patient with comorbid pathology.

Examples of control questions for testing in practice

1. Body mass index - an indicator that is calculated using the formula:

- | | |
|-------------------|------------|
| A. $W \times H/2$ | C. W/H^2 |
| B. $W \times H^2$ | D. $W/2H$ |

2. What groups of drugs should not be used in the treatment of patients with metabolic syndrome?

- | | |
|----------------------------------|------------------------------------|
| A. ACE inhibitors + diuretic; | B. Calcium antagonists + diuretic; |
| C. β -blockers + diuretic; | D. α -blockers + diuretic. |

3. To which β -blockers the preference is given in the treatment of hypertension in metabolic syndrome::

- | | |
|----------------|---------------|
| A. Anaprilin; | B. Atenolol; |
| C. Bisoprolol; | D. Nebivolol. |

4. Which antihypertensive drug is preferred in the treatment of metabolic syndrome with insulin resistance?

- | | |
|----------------|----------------|
| A. Losartan; | B. Carvedilol; |
| C. Furosemide; | D. Koraksan. |

5. What body mass index (kg / m²) corresponds to the second degree of obesity?

- | | |
|---------------|---------------|
| A. 2,0-29,9; | B. 18,5-24,9; |
| C. 30,0-34,9; | D. 35,0-39,9. |

6. The concentration of leptin in the blood of patients with insulin resistance is:

- A. Decreased
- B. Increased
- C. Doesn't change
- D. A and B are correct answers

7. Risk factors for cardiovascular diseases include:

- A. The fasting plasma glucose level is 5.6-6.9 mmol / l;
- B. Violated carbohydrate tolerance test;
- C. Consumption of alcoholic beverages;
- D. Lack of hormone replacement therapy in women.

8. The risk factors for cardiovascular disease are the following indicators of the lipid profile:

- A. Total cholesterol > 6.0 mmol / l;
- B. Total cholesterol > 6.2 mmol / l;
- C. Triglycerides > 1.7 mmol / l;
- D. Triglycerides > 1.2 mmol / l;

9. Age-related changes in the endocrine system are:

- A. Decreased plasma concentrations of renin, aldosterone
- B. Increased synthesis and elimination of thyroxine
- C. Decreased concentration of ADH
- D. Increased testosterone levels in the blood
- E. Increased absorption and synthesis of vitamin D.

10. Basic principles of prescribing drugs to elderly patients:

- A. Treatment begins with the maximum doses of drugs, reducing them later
- B. Treatment begins with small doses of drugs, slowly increasing them to the minimum effective
- C. Simultaneous administration of several drugs is recommended
- D. Insist on self-control while taking medication by the patient.

Answers to control questions.

№	1	2	3	4	5	6	7	8	9	10
Correct answer	C	C	D	A	D	B	A	C	A	B

The main theoretical issues of the topic

1. The urgency of the problem.
2. Methods of calculating overweight and determining obesity.
3. Classification of obesity.
4. The main medical consequences of obesity - metabolic syndrome, diabetes, cardiovascular and gastrointestinal diseases.
5. Modern approaches to dietary and medical treatment.
6. The role of bariatric surgery in the treatment of obesity.
7. Features of metabolism in old age.
8. Frequency of comorbid pathology in the elderly.
9. Features of the drug on the body of the elderly.
10. Features of diagnosis of diseases in old age.
11. Features of treatment in old age.

Technological map of the practice session

№	Stage	Minutes	Training manuals		Venue
			Teaching aids	Equipment	
1	Determination of the initial level in the diagnosis of obesity	15	Tasks		Classroom
2	Thematic analysis of patients with obesity	120	Medical histories		Ward, classroom

3	Summing up	25	Tasks		Classroom
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