Federal State Budgetary Educational Institution of Higher Education "North Ossetian State Medical Academy" of the Ministry of Health of the Russian Federation

Department of Internal Diseases No. 4

Methodological guidelines for conducting practical classes with students of the 6th year of the Faculty of Medicine on the topic: "Acute coronary syndrome"

Vladikavkaz 2021

Guidelines for conducting practical classes

with students of the 6th year of the Faculty of Medicine on the topic:

"Acute coronary syndrome"

The purpose of the lesson: Be able to build a diagnostic search program for the detection of acute coronary syndrome, choose rational methods of treatment of acute coronary syndrome both at the stage of emergency medical care and in a specialized hospital.

Motivation for the relevance of the topic: In recent years, emergency cardiology has been changing especially dynamically. Currently, acute coronary syndrome is the central topic of emergency cardiology. ACS is a fairly frequent clinical condition that every doctor has to deal with to one degree or another.

The widespread introduction of endovascular methods of treatment opens up new opportunities in the treatment of acute coronary syndrome, which requires students to have in-depth modern knowledge, as well as real interdisciplinary interaction during the provision of emergency care to patients

The student should know: etiopathogenesis of acute coronary syndrome. diagnosis and differential diagnosis of acute coronary syndrome in an emergency room, principles of treatment of acute coronary syndrome in an emergency room; must be able to: choose rational methods of treatment of acute coronary syndrome; must possess the skills of: first aid in acute coronary syndrome.

Experts of the All-Russian Scientific Society of Cardiology in 2001 adopted the following definition of ACS and unstable angina: "ACS is a term denoting any group of clinical signs of symptoms that allow one to suspect myocardial infarction, unstable angina.

ACS includes the following concepts:

1 Acute myocardial infarction.

2 Myocardial infarction with ST segment elevation on ECG (STEMI).

3 Myocardial infarction without ST segment elevation on ECG (STEMI).

4 Myocardial infarction, diagnosed by changes in enzymes, by

other biomarkers, by late ECG signs.

5 Unstable angina.

The term ACS is not a diagnosis and can be used only in the first hours and days of the disease, when diagnostic information is still insufficient to make a final judgment about the presence or absence of foci of necrosis in the myocardium. With an increased level of biomarkers of myocardial necrosis (mainly we are talking about highly sensitive troponin – Tn), a diagnosis of MI is established, in the absence of necrosis markers – unstable angina

The nature and urgency of the intervention to restore coronary perfusion is determined by the position of the ST segment relative to the isoelectric line on the ECG. When the ST segment is shifted (lifted) upwards

- ACS with the ST segment rising on the ECG (STEMI) – the restoration of coronary blood flow should be carried out immediately. The method of choice for restoring coronary blood flow is coronary angioplasty; if it is impossible to carry it out in the appropriate time, TLT is effective and indicated.

With ACS without ST segment elevation on an ECG (OXBPST) TLT is ineffective, and the timing of coronary angioplasty or coronary artery bypass grafting (CABG) depends on the degree of risk (prognosis) of the disease.

Etiology: With a wide variety of potential etiopathogenetic factors, the cause of ACS is mainly atherosclerosis of the coronary artery at the stage of destabilization of the atherosclerotic plaque and the formation of an intravascular thrombus above it. ACS develops as a result of ulceration or rupture of an "unstable" atherosclerotic plaque and subsequent thrombosis in the area of intimal damage. A detailed study of the mechanisms of thrombogenesis showed that the thrombotic process begins with the adhesion of platelets to the sites of damaged atherosclerotic plaque. At the same time, young plaques with a thin surface shell are the most vulnerable. Plaque rupture is detected in 75% of cases of acute coronary thrombosis.

Diagnosis of ACS is carried out on the basis of clinical picture data, ECG indicators recorded during development clinical picture, and the results of the study of cardiospecific enzymes.

Classifications of ACS and acute MI

At the stage of preliminary diagnosis:

1 ACS with ST segment elevation – IM with ST segment elevation (this group also includes acute blockade of LDL).

2 ACS without lifting the ST segment.

Clinical diagnosis (including final) after confirmation/exclusion

1 IM with the rise of the ST segment (this group also includes the acute blockade of the CNPG).

2 IM without lifting the ST segment.

3 Unstable angina.

Classification of MI based on subsequent ECG changes (optional):

1 MI with the formation of pathological Q teeth.

2 IM without the formation of pathological Q teeth .

Classification of MI based on the depth of the lesion of the muscle layer (is a priority for pathoanatomic / forensic diagnosis):1 Subendocardial IM. 2 Transmural IM.

Classification of MI based on the localization of the focus of necrosis.

After 28 or more days after the onset of the symptoms of STEMI, the diagnosis is not applied to them. In this case, it is customary to indicate the transferred wound IM, designating it as post-infarction cardiosclerosis.

Classification of MI based on the presence of MI in the anamnesis:1The

recurrent MI -MI, developed after 28 days and later (after the preceding MI).

2 Relapse of MI - MI that developed within 28 days after the previous MI.

Clinic. STEMI is diagnosed in patients with an anginal attack with pressing, squeezing pains behind the sternum (a feeling of discomfort) lasting more than 20 minutes, not passing after taking nitroglycerin. Most often, these pains radiate to the left arm, left shoulder, lower jaw on the left, or other sensations (discomfort) in the chest cell and persistent (persisting for at least 20 minutes) ST segment elevation or "new" (first-time or presumably first-time) blockade of the left leg of the Gis bundle (LNPG) on the ECG. As a rule, if the disease begins as STEMI, signs of myocardial necrosis appear later – an increase in the levels of biomarkers of ECG changes, including the formation of Q teeth.

ECG for ACS with ST segment elevation

1.If the rise of the ST segment is caused by occlusive thrombosis of the CA, then in the absence of restoration of blood flow, a pathological Q wave is formed and a characteristic dynamics is observed in the form of an approximation of the ST segment to the isoline, which occurs gradually over several days.

2 At the same time, the formation of a negative T-wave is observed and then its deepening

3. If, as a result of reperfusion therapy, the blood flow in the SC is restored, the dynamics on the part of the ST segment is fast - by 50% within 60-90 minutes, which is the criterion for the success of reperfusion.

4. If the ST segment elevation persists for a long time, it is necessary to suspect the development of an aneurysm.

ECGpri ACS without ST segment elevation

1.A new horizontal or oblique depression of the ST segment exceeding 0.5 mm in leads V2 and V3 and 1 mm in the remaining leads.

2.A new inversion of the tooth T>1 mm in two adjacent leads with a predominant tooth R or R/S>1

3.The coronary T is usually symmetrical.

The appearance of signs of necrosis means that the patient has developed a myocardial infarction. The term "myocardial infarction" reflects the death (necrosis) of heart muscle cells (cardiomyocytes) as a result of ischemia. In accordance with international conciliation documents, myocardial infarction is diagnosed if there is a clinical picture of ACS and markers of necrosis. MV KFK,troponin I,Troponin T.

Classification of IM types:

Type 1 MI, developed as a result of rupture or erosion of atherosclerotic A B c.

Type 2 MI developed as a result of ischemia caused by causes unrelated to thrombotic complications of coronary atherosclerosis.

Type 3 MI type 3 corresponds to cases of symptoms indicating myocardial ischemia, accompanied by ECG changes or ventricular fibrillation. The diagnosis is confirmed based on the detection of acute MI at autopsy.

Type 4a. MI associated with complications that occurred during the PCI procedure and in the next 48 hours after it.

Type 4b. MI associated with coronary stent thrombosis, documented during KG or autopsy.

Type 4c. MI associated with restenosis after PCI is established if pronounced restenosis is detected in the artery corresponding to the MI zone, when there are no signs of thrombosis and other lesions of the infarct-related artery.

Type 5 MI associated with coronary bypass surgery.

COMPLICATIONS OF MI

■ Disturbances of heart rhythm and conduction.

■ Acute heart failure.

■ Cardiogenic shock.

■ Mechanical complications: ruptures (interventricular septum, of the free wall of the left ventricle), detachment of the chords of the mitral valve,

detachment or dysfunction of the papillary muscles).

■ Pericarditis (epistenocarditis and Dressler syndrome).

Prolonged or recurrent pain attack, postinfarction angina

Treatment

Reperfusion treatment of the disease.

Reperfusion therapy to reduce the risk of death is recommended for all patients with STEMI and duration of symptoms < 12 hours.

Currently, reperfusion treatment involves the use of two strategies: primary PCI and a pharmacoinvasive approach, including the sequential use of TLT and PCI. The choice between these two strategies is determined primarily by the availability of units capable of organizing timely PCI.

Thrombolytic therapy in the absence of contraindications is recommended to reduce the risk of death in patients with STeMI with symptoms lasting < 12 hours, in whom primary PCI is not expected to be performed within 120 minutes after diagnosis.

Absolute contraindications to TLT: hemorrhagic stroke or blood circulation of unknown etiology;- ischemic stroke in the previous 6 months;- damage or neoplasms of the central nervous system.

Streptokinase - fibrin is a non-specific drug, administered in a vein at a dose of 1500,000 IU for 30-60 minutes in a small amount of 0.9% sodium chloride solution.

Unlike streptokinase, fibrin-specific drugs have a high tropicity to thrombus fibrin, which increases the frequency of restoration of coronary blood flow when using them up to 70%. These drugs are not antigens, and this allows them to be re-administered at any time, as soon as it becomes necessary.

A recombinant tissue plasminogen activator (alteplase) is injected into a vein (the drug is previously dissolved in 100-200 ml of distilled water or 0.9% sodium chloride solution) according to the "bolus + infusion" scheme.

Relative contraindications to TLT:

- transient ischemic attack in the previous 6 months;

- taking oral anticoagulants;

- pregnancy and the 1st week after delivery;

- refractory hypertension (SAD > 180 mmHg and/or DAD > 110 mmHg);

- severe liver disease;

- infectious endocarditis;

- traumatic or prolonged cardiopulmonary resuscitation;

- exacerbation of peptic ulcer disease.

Anticoagulants

With conservative treatment tactics, the drug of choice is fondaparinux (2.5 mg per day n / a), as a drug with an optimal safety profile. With invasive tactics, the drug of choice is enoxaparin, which is injected into the vein bolus at a dose of 30 mg or unfractionated heparin in the form of a bolus at the rate of 60-70 IU / kg (maximum 4000 IU).

Disaggregants

Patients with ACS, starting from the moment of first contact with medical personnel, should receive double disaggregant (antiplatelet) therapy in the form of a combination of acetylsalicylic acid (ASA) + adenosine diphosphate — P2U12 receptor blockers (clopidogrel or ticagrelor). A loading dose of ASA is prescribed in a simple form (without a protective shell) at a dose of 250-500 mg. In the case of planned thrombolytic therapy, clopidogrel should be prescribed at a loading dose of 300 mg, followed by 75 mg per day. In persons over 75 years of age, the loading dose is not used.With the planned primary percutaneous intervention , it is preferable to prescribe ticagrelor at a loading dose of 180 mg . If it is impossible to prescribe ticagrelor, the loading dose of clopidogrel should be 600 mg, then 150 mg per day for 7 days.

Subsequently, acetylsalicylic acid is prescribed for an indefinitely long period in a maintenance dose of 75-100 mg (it can be in the form of intestinal soluble).

Maintenance doses of inhibitors (clopidogrel 75 mg per day, ticagrelor 90 mg 2 times a day) are prescribed at 12 months after an acute event.

Pain relief

The most important therapeutic measure is adequate anesthesia. Anesthesia can begin with the introduction of nitroglycerin under the tongue (tablets, sprays). If 2-3 doses are ineffective (the effect should be achieved within a few minutes), narcotic analgesics are prescribed. The drug of choice is morphine, which has a pronounced vasodilating effect, thereby reducing pre- and post-loading on the heart. Morphine

helps to reduce fear, arousal, reduces sympathetic activity, increases the tone of the vagus nerve, reduces the work of breathing, causes the expansion of peripheral arteries and veins (the latter is especially important for pulmonary edema). It is also possible to use fentanyl, which has a more pronounced analgesic, but more short-term effect.Administered intravenously by 0.05-0.1 mg (1-2 ml of 0.005% solution) diluted in 10 ml of 0.9% sodium chloride solution. To enhance and prolong the effect, as well as for the purpose of sedation, the administration of fentanyl is combined with intravenous administration of a neuroleptic (neuroleptanalgesia) - droperidol (1 ml of 0.25% solution - 2.5 mg). With the introduction of narcotic analgesics, complications may occur in the form of respiratory depression (stopped by the introduction of naloxone and ventilation of the lungs with an Ambu bag), vomiting (stopped by the introduction of metroclopramide), bradycardia (stopped by the introduction of atropine

Nitrates

Sublingually, nitrates are used in the acute period to relieve pain (especially during the first medical contact).Intravenous administration of nitrates in patients in the acute period is indicated for recurrent anginal pain, pulmonary edema (acute left ventricular failure - Killip III class), arterial hypertension. In all other cases, intravenous administration of nitrates is impractical.

Beta-blockers reduce the risk of myocardial rupture and ventricular fibrillation. Beta-blockers in an effective dose should be administered orally as early as possible, taking into account the manifestations of left ventricular failure and bronchospasm. It is important to titrate doses of beta-blockers until the target heart rate values are reached - 50-60 beats per minute.

ACE inhibitors are prescribed in the early stages of myocardial infarction to reduce the degree of myocardial remodeling and prevent the development of chronic heart failure. Especially in patients with a low ejection fraction, in the presence of hypertension, diabetes mellitus.

Statins. Early administration of statins in maximum therapeutic doses (40 mg of rosuvastatin or 80 mg of atorvastatin) has been proven to improve the prognosis in patients with MI. In the future, it is required to titrate the dose according to the level of low-density lipoproteins (the target level is less than 1.8 mmol / l).

Rehabilitation of patients who have undergone ACS

Cardiorehabilitation involves three main stages:

* Inpatient stage, which can begin in the conditions of the hospital and continue in the ward of the cardiology department of the hospital or vascular center.

* An early inpatient rehabilitation stage, conducted in an inpatient cardiorehabilitation department of cardiological or multidisciplinary hospitals, or a rehabilitation center.

* Outpatient rehabilitation stage. At this stage, the patient is defined as a subject with post-infarction cardiosclerosis who needs to perform a complex of rehabilitation measures and prolonged secondary prevention. In the first months after discharge from the hospital, these activities are carried out under medical supervision, and then – under self-control at home

Test tasks on the topic:

"Acute coronary syndrome"

1. Unstable angina pectoris includes:

A. First-time angina pectoris

B. Progressive angina pectoris

B. Variant angina

pectoris G. All of the above

2. Angina pectoris is considered to have arisen for the first time, which lasts:

A. One month

B. Three months

B. Four months

G. Six months

3. The most important additional method of diagnosis of myocardial infarction is

A. chest radiography

B. electrophysiological examination of the heart

B. echocardiography

G. myocardial scintigraphy

4. The gradation of risk in patients with acute myocardial infarction with ST segment elevation is determined using

the A. QRISK 2 scale;

B. HEART;

B. ATP III;

G. GRACE.

5. The concept of acute coronary syndrome without ST segment elevation includes:

A. postinfarction angina;

B. postinfarction cardiosclerosis;

B. progressive angina pectoris;

G. Stable angina pectoris;

D. Angina pectoris after aorto-coronary

6. How many days before the planned surgical intervention is recommended to stop taking tikagrelor?

A. for 3 days;

B. for 5 days;

B. for 7 days;

G. per day.

7. To detect unstable plaques

, A. intravascular optical coherence tomography is used;

B. intravascular ultrasound examination;

B. Contrast computed tomography;

G. Myocardial scintigraphy.

8. Risk factors for acute coronary syndrome without ST segment elevation are:

A. hyperthyroidism;

B. dyslipidemia;

B. Infections;

G. disorders of carbohydrate metabolism;

D. Chronic kidney disease.

9. The main causes of the development of acute coronary syndrome

A. microvascular dysfunction;

B. coronary artery thrombosis;

B. spasm of the coronary artery affected by atherosclerosis;

G. Metabolic disorders.

10. Which of the biochemical markers of myocardial necrosis has the greatest sensitivity (100%)?

A. myoglobin;

B. troponin T or I;

B. CFK general;

G. KFK MV.

11. Preferred combinations for double disaggregant therapy

A. acetylsalicylic acid + ticagrelor;

B. acetylsalicylic acid + prasugrel;

B. acetylsalicylic acid + clopidogrel;

G. acetylsalicylic acid + warfarin.

12. An indication for emergency coronary artery bypass grafting in a patient with acute myocardial infarction is

A. successfully performed endovascular treatment of an infarct-related artery, but with persistent hemodynamically insignificant stenoses of other coronary arteries

B. the presence of cardiogenic shock if the anatomy of the coronary arteries does not allow endovascular treatment

B. stenosis

13. What should be done first of all if an anginal attack has developed?

A. to perform anesthesia, sedation therapy

B. to start oxygen

therapy B. to give antiplatelet agents

D. give anticoagulants

14. The equivalent of pain with angina pectoris (subject to its short duration) may be:

A. Heartburn

B. Numbness of the 4th-5th fingers of the left hand

B. Short-term feeling of weakness and sweating

G. All of the above

15. Complications of myocardial infarction include:

A. Cardiogenic shock

B. Cardiac asthma

B. Rhythm disturbances

G. All of the above

16. Unfractionated heparin in myocardial infarction with ST segment elevation is used

A. only after percutaneous coronary intervention with stenting;

B. during percutaneous coronary intervention;

B. with thrombolytic therapy;

D. only at the prehospital stage;

d. for the prevention and treatment of arterial or venous thrombosis.

17. According to the classification of unstable angina, class II severity (according to Braunwald) includes

A. first-time angina pectoris; angina pectoris lasting less than 2 months, except for angina pectoris at rest during the last 2 months;

B. angina pectoris at rest, acute; one or more episodes of angina pectoris at rest in the last 48h;

B. angina pectoris at rest, subacute; one or more episodes of angina pectoris at rest for a month, except for angina pectoris at rest for the last 48h;

D. severe angina pectoris three or more times a day, or definitely more frequent and provoked by less exertion.

19. What age is estimated by the minimum score on the GRACE scale?

A) 60-69;

B) ≤30;

C) ≥90.

20. Specific signs for aortic wall dissection:

A. asymmetry of blood pressure on the extremities;

B. Chest pain;

B. pain along the spine;

G. Headache;

18. According to the classification of unstable angina pectoris, class III severity (according to Braunwald) refers

to A. first-time angina pectoris of tension, except for angina pectoris at rest during the last 2 months;

B. angina pectoris at rest, acute; one or more episodes of angina pectoris at rest in the last 48h;

B. angina pectoris at rest, subacute; one or more episodes of angina pectoris at rest for a month, except angina pectoris

D. shortness of breath.

Response standards

1-G

2-A

3-In

4-G

5-AVD

6-A

7-ABC

8-BGD

9-BV

10-B

eleventh

12-B

13-A

14-G

15-G

16-BVD

17-In

18-B

19-B

20-AV

. Task 1 Patient A., 48 years old, works as a shop supervisor at the enterprise. He turned to the emergency department of the hospital on duty with complaints of intense burning pains behind the sternum, radiating into the left half of the neck, the left shoulder. The pain lasts about 2 hours, accompanied by a feeling of fear of death. The patient took 4 tablets of nitroglycerin without effect.

From the anamnesis of the disease. Connects the occurrence of a pain attack with a stressful situation at work. From the anamnesis of life: has a long experience as a smoker, smokes up to 20 cigarettes a day. The patient's older brother suffered a myocardial infarction at the age of 52.

On examination: the skin is pale, the patient is over-nourished, BMI is 37 kg/m2. There are no peripheral edema. There is vesicular breathing in the lungs, wheezing is not heard. BH - 20 in 1 min. Pulse 104 beats per minute. Visually, the area of the heart is not changed. The boundaries of the heart are not shifted. The heart tones are deaf, the rhythm is correct, the heart rate is 104 in 1 min, noises are heard during auscultation. Blood pressure =105/69 mm Hg. The abdomen is soft, painles liver at the edge of the costal arch.

General blood test: hemoglobin 134 g/l, leukocytes 11,3x109 / l, neutrophils -7%, neutrophils – 64%, e – 1%, l/f – 25%, m – 3%, ESR – 10 mm/h. Biochemical blood test: creatinine – 52 mmol/l, urea -5.4 mmol/l, total protein - 63 g/l, total cholesterol – 9.2 mmol/l, HDL cholesterol – 0.7 mmol/l, LDL cholesterol – 5.6 mmol/l; troponin test positive; myoglobin – 620 mcg/l (at a rate of up to 69 mcg/l); fibrinogen – 4,600 mg /l; PTI – 105%.

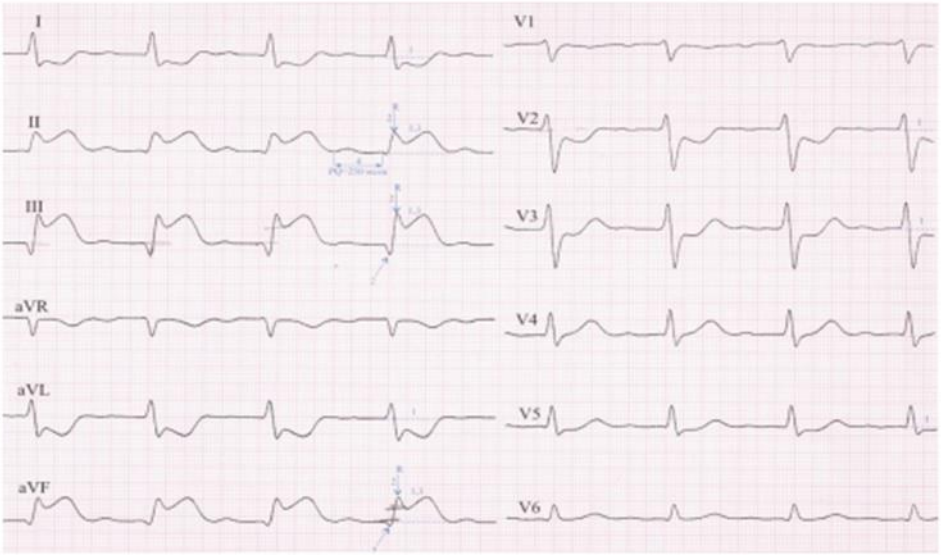
1. Formulate a preliminary diagnosis.

2. What are the signs of this disease found on the ECG?

3. What atypical clinical variants of the onset of this disease do you know?

4. What is the basis of the pathogenesis of this disease?s, hepatic

ЭКГ.



Task 2. A 52-year-old patient was taken to the emergency room by ambulance with complaints of sharp pressing pains behind the sternum, radiating to both arms and the left shoulder blade, lasting more than two hours, the pain is not stopped by taking nitroglycerin and injectable analgesics, accompanied by sharp weakness, fear of death, pronounced sweating.

It was found out from the anamnesis that the patient had been bothered for about 10 years by paroxysmal headaches, tinnitus, dizziness, "flashing of flies" in front of his eyes, short-term stabbing pains in the heart area, without irradiation. Sometimes there was an increase in blood pressure to 200/120 mm Hg, constant blood pressure - 160/100 mm Hg.

When examined, the general condition is severe. The skin is pale, acrocyanosis. Breathing is frequent (26 per minute). Percussion over all fields of the chest is a pulmonary sound, during auscultation - vesicular breathing, single wet wheezes in the lower parts of the lungs. The pulse is the same on both hands, 120 beats / min, rhythmic, weak filling and tension. AD - 90/70 mm Hg art . P Percutorially, the left border of relative cardiac dullness

is shifted to the left. Auscultatively, the heart tones are deaf. The accent of the second tone above the aorta. The abdomen is soft, painless, the liver is not enlarged. There is no swelling.

1 Is your preliminary diagnosis?

2 Assign the necessary informative research methods.

3 Your treatment tactics.