

**MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

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**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Emergency conditions in clinic of Internal Medicine
<i>Content module</i>	Emergency conditions in clinic of Internal Medicine
<i>Study subject</i>	Curation of the patient with acute coronary syndrome
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

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I. The urgency of the problem

Acute coronary syndrome (GCS) — one of the most dangerous manifestations of coronary heart disease, mortality from which is one of the first places in Ukraine and around the world. That is why, the study of the main etiological factors, pathogenetic links the formation of disease, modern approaches to treatment and rehabilitation is given special attention.

II. Learning Objectives.

- ☐ To analyze the prevalence of SCS.
- ☐ Categorize GCS and analyze the typical clinical picture.
- ☐ To familiarize students with the survey methods that are used for the diagnosis and differential diagnosis of GCS, the indications for their use of the technique, the diagnostic value of each of them.
- ☐ To teach students to interpret the results of their own studies.
- ☐ To combine individual scheme of diagnostic search, identify and provide the necessary volume and consistency of patient examination methods of GCS.
- ☐ conduct differential diagnostics and substantiate the clinical diagnosis.
- ☐ To know the stages of an emergency when the SCS and the possible complications, principles of treatment, rehabilitation and prevention.

III. Content of the topic.

By GCS include unstable angina (UA), myocardial infarction (MI), non-Q wave and Q-wave and acute occlusion of coronary arteries after invasive interventions (transluminal balloon coronary angioplasty, rotational atherectomy).

Unstable angina - is the transition of severe myocardial ischemia, unstable stroke, which in its clinical manifestations and prognostic value is intermediate between stable angina, myocardial infarction and acute myocardial infarction. If MI and sudden cardiac death (SCD) is preceded by the National Assembly in 80% of patients.

The etiology and pathogenesis of unstable angina.

NA etiological factor is the destabilization of atherosclerotic plaque with the following thrombosis or microembolisms.

Reasons NA and MI without ST segment lift:

1. thrombosis or thromboembolism, which develops as a result of strain or erosion of atherosclerotic plaque:

- Oklyuziruyuschy blood clot in the vessels of the collateral;
- Subtotal thrombotic occlusion of the plaque, which originated before;

- The distal microvascular thrombosis caused by a blood clot margin associated with plaque.
2. thromboembolism due to reasons different from plaque rupture.
 3. Dynamic obstruction (coronary spasm or vasoconstriction) epicardial and / or capillary vessels.
 4. Progressive mechanical obstruction of the coronary blood flow.
 5. Inflammation of the coronary artery.
 6. Stratification of coronary artery.
 7. Secondary NA.

Development of pain in the National Assembly comes as a result of ischemia in the blood supply to the affected area venochnogo vessel, which leads to the emission of biologically active substances and the stimulation of pain receptors.

There are 3 HC development mechanism:

1. Rupture of an atherosclerotic plaque. The growth of atherosclerotic plaque leads to progressive narrowing of the lumen of the coronary vessel with a suitable blood circulation disorders. Then there is the anguish of its tire with the formation of a blood clot or plaque microemboli content.
2. Thrombosis predetermined increase in activity of the blood coagulation system at the systemic and local levels. Platelet formed first (white) and then erythrocyte, fibrin (red) thrombus.
3. vasoconstriction is platelet-dependent and endothelium-dependent.

The classification of unstable angina.

Distinguish primary (for CHD) and secondary (off-coronary causes, which lead to myocardial ischemia and increase its oxygen demand, such as rheumatic fever, diffuse connective tissue disease, anemia, infections, hyperthyroidism, and so on.) Of the National Assembly.

By the NA include:

- For the first time emerged angina.
- progressive angina.
- Early postinfarction angina and angina after coronary artery bypass grafting.

The recommendations for the diagnosis and treatment of the ACC / AHA NA (2007) proposed to distinguish these clinical variants:

1. tranquility Angina (the attack lasted more than 20 minutes.)
2. For the first time emerged angina (at least III functional class (FC))

I. The main clinical symptoms of various forms of the National Assembly:

- For the first time emerged angina lasts up to 28 days from the beginning of development and accompanied by compressing and burning pain behind the breastbone, which is associated with physical and psycho-emotional load of referred in the left arm and shoulder blade lasting 1-20 minutes. and stopped nitroglycerin in 1 -10 minutes.

- Progressive angina is accompanied by an increase in the frequency and duration of angina attack, which occurs when less physical or psycho-emotional load, which is always docked nitroglycerin (patients can take 30-50 tablets per day).

- The early post-infarction angina - is anginal attacks in the tranquility of a small or exertion in patients with and which arise after 3-28 days from the beginning of its development and significantly worsen the prognosis of the disease.

- Angina which originated in the state of rest.

II. ECG criteria during the attack NA

ST segment observed abnormal offset horizontally above or below. isoline with possible sploschenieem, two-phase or negative prong T.

III. Biological markers in blood

Troponin T and And isoenzyme CK-MB, AST, ALT, aldolase are at the upper limit of normal or increased by no more than 50%.

The most promising are the newly developed test systems to determine kardiospetseficheskih isoforms of troponin T and I. Each of these markers has a high specificity for a myocardial infarction and may be detected in the blood at 3 hours after the onset of the pain syndrome. As a result of their high sensitivity and specificity of the use of troponin in the diagnosis of acute myocardial infarction is increasing rapidly.

According to the European Society of Cardiology, the American College of Cardiology and the American Heart Association (2007), the diagnosis of acute coronary syndrome without ST elevation is set in the following cases:

- if the patient long-term (> 20 min) anginal pain at rest;
- in the event for the first time severe angina (at least class III according to the classification of the Canadian Association of cardiovascular disease);
- when the newly emerging destabilize previously stable angina, and increasing at least until III FC (progressive angina);
- angina, occurred after MI.

Treatment of unstable angina

NA treatment is carried out in the intensive care unit. The main drugs are anticoagulants, antiplatelet agents, β -blockers, statins and nitrates.

Unfractionated heparin - is the most common antitrombolitichesky drug for the treatment of patients with unstable angina. It should be used in the first 20 min after hospitalization. The mechanism of action of unfractionated heparin is its binding to antithrombin III inhibition of thrombin and Xa activity and blood clotting factor in a ratio of 1: 1. During intravenous effect occurs immediately. Typically, the first dose of heparin is 70 - 80 U / kg or 000 IU 5000-10. it

is administered by bolus, and then pass to infusional at 15 U / (kg·d) or an average of 1,000 units / h under the control of the activated partial thromboplastin time (APTT). Control of the APTT is performed every 6 h infusion to establish a therapeutic level, that is 1.5 - 2.5 times higher than normal (but not indicators baseline!) In the following two definitions. After that APTT is determined every 24 hours. Continuous intravenous infusion of heparin lasts 1-3 days in the first three days of treatment is necessary to control the ratio of hemoglobin to hematocrit (II / III) and platelet count once per day. APTT should be determined in all cases where the clinical condition of the patient changes significantly (recurrent ischemia, hemorrhage, arterial hypotension), or change the dose of heparin. Treatment of intravenous unfractionated heparin should be started on the first day and continue to 48 - 72 hours. Appointment of unfractionated heparin is particularly important in patients receiving ticlopidine or clopidogrel (via delayed the start of their antiplatelet effects). Subcutaneous (periomphalic) heparin at a dose of 12 500 IU every 12 hours. Prior to this, the drug is administered by intravenous bolus at a dose of 5000 IU. Monitoring the effectiveness of the prescribed dose unfractionated heparin during percutaneous administration is also carried out in terms of APTT, like the clock intravenous administration.

Concomitant use of unfractionated heparin with aspirin, ticlopidine or clopidogrel reduces the risk of acute myocardial infarction or sudden cardiac death. However, an alternative subcutaneous unfractionated heparin (12 500 IU 2 times a day) makes it impossible to predict and maintain the desired level of anticoagulation. On the other hand, proved the possibility of reactivating the process of thrombus formation in the event of an abrupt discontinuation of unfractionated heparin due to increased formation of thrombin (withdrawal or rebound). In this regard, it recommended gradual withdrawal (transition from intravenous (if performed) on the subcutaneous route of administration and the gradual reduction in dose).

Over the past 10 years, entered the clinical practice safer antithrombin agents - low molecular weight heparins: enoxaparin calcium (Clexane / Lovenox), nadroparin calcium (fraxiparine) delteparin calcium (Fragmin), reviparin (klevargin) sandoparshi (mono emboleks) tinzaparin (lozhiparin / innogep) ardeparin (normoflo).

Because in the process of thrombus formation in the arteries leading role belongs to platelets, it is this component of hemostasis new antiplatelet agents influence.

Aspirin. The basis of aspirin mechanism of action is the ability to irreversibly inhibit COX-1, which is contained in platelets and promotes the conversion of arachidonic acid to endoperoxide prostaglandins and hence for thromboxane A₂ in the vessel wall - the formation of prostacyclin I₂ (thromboxane A₂ is a potent vasoconstrictor and activator adhesion and aggregation platelets and prostacyclin I₂ - a vasodilator and antiplatelet agents).

Aspirin prescribed with caution if there is a risk of gastrointestinal bleeding, an exacerbation of peptic ulcer disease, gastralgia, hemorrhagic vasculitis, hematopoiesis disorders, allergic reactions. In cases of aspirin intolerance or contraindications to it ticlopidine sulfate used (tiklid). It inhibits platelet aggregation induced by ADP, collagen, thrombin, serotonin, epinephrine. Unlike aspirin, ticlopidine does not affect the metabolism of arachidonic acid and cyclic AMP. However, ticlopidine causes and adverse effects - diarrhea, gastric dyspepsia, neutropenia, hepatosplenomegaly syndrome, vasculitis, that, in fact, are relative contraindications for too long application of the drug (even with monitoring the number of leukocytes in the blood). Clopidogrel (Plavix) is a potent selective blocker of platelet aggregation induced by ADP. The antithrombotic effect of clopidogrel is irreversible binding to platelet ADP receptors membrane, resulting in platelet aggregation is inhibited stimulated by ADP. Clopidogrel at a dose of 75 - 150 mg per day inhibits platelet aggregation within 2

hours after oral administration, and permanent inhibition of aggregation of 40 - 60% recorded after 3 - 7 days of treatment, and maintained for a long time treatment. Side effects are negligible and rarely require discontinuation of the drug. The most common side effects are diarrhea, skin rashes, hemostatic disorders.

Blockers (antagonists) receptor glycoprotein II / IIIa - a fundamentally new class of antitrombotic drugs. It is proved that the receptor glycoprotein (GP) are a family of the platelet integrins (adhesion proteins) located on the platelet membrane. Two subunit thereof - GP and GP IIb IIIa - form stoichiometrically Ca^{2+} -dependent complex in 1: 1 ratio, which is found on the platelet surface, even inactive. As a result of the stimulation of biologically active substances (blood clots, collagen) there is a significant conformation of the receptor, thereby increasing the affinity of the structure of the GP IIb / IIIa to fibrinogen and communicate with him. It should be noted that due to the fibrinogen molecule structurally joined to both receptor GP IIb / IIIa platelet two adjacent causing their coalescence (aggregation). Von Willebrand Factor (adhesive protein of blood plasma and extracellular spaces) can also be connected to the GP receptor IIb / IIIa (the more typical is its interaction with the receptor GP IIb). The first drug from a group blockers (antagonists) receptor GP II / IIIa was abciximab (Reo Pro). This monoclonal antibody to the receptor GP II / IIIa. It is administered intravenously in a bolus dose of 0.25 mg / kg and for 18 - 24 h infusion at a dose of 10 mg / kg to percutaneous transluminal coronary angioplasty. The structure of the other GP II / IIIa receptor antagonists based on the modification of the CSR chain amino acid sequence (the tripeptide arginine - glycine aspartic acid). So there were drugs in which CSR arginine chain is replaced by lysine (a sequence called CSR) - integrilin, tirofiban and lamifiban. Combination lamifiban - aspirin proved to be quite effective. This significantly reduces the frequency of the transition in unstable angina myocardial infarction, reduced mortality and the need to use invasive methods. Inhibitors GP II / IIIa administered in addition to aspirin and heparin in patients with preserved signs of myocardial ischemia, high-risk groups and patients with planned percutaneous transluminal coronary angioplasty.

The role of β -blockers in the treatment of patients with unstable angina is not a subject of discussion. The possibility of vasospastic angina in response to the use of β -blockers is unlikely, because the vast majority of patients with stable angina not vasospastic etiologies. β -blockers are most effective in patients with tachycardia and high blood pressure.

NA patients with pain syndrome prescribe **nitroglycerin** - 5 mg every 5 minutes. If the use of pills nitroglycerin pain does not subside, to be administered nitrates intravenously clock: 1% solution of nitroglycerin 6 mL (60 mg) to 250 ml of isotonic sodium chloride solution, the dosage - from 2 to 17 drops in 1 minute (every 10 min to 1 drop more frequencies reaching 17 drops per 1 minute infusion continue round the clock) or 0.1% solution perlinganita 100 ml (100 mg) in 400 ml of isotonic sodium chloride solution; Izoketa 0.1% solution - 100 ml (100 mg) in 400 ml of isotonic sodium chloride solution administered the same procedure as nitroglycerin.

Preparations of nitroglycerin depot (nitrogl-forte, forte Susak, nitrogranulong, sustanit, nitret) take 20 - 60 mg per day, mononitrates (isosorbide-5-mononitrate - I5-5-M) - 60 mg per day - 80, dinitrate (isosorbide dinitrate - Iza -) at 80-120 mg per day dinitrate are pharmacologically inactive compounds only after biotransformation in the liver, they become active mononitrates Molsidomine (korvaton, Sydnopharm, korvasol) take 8 - 24 mg per day.

During the introduction of Nitrate headache restaurants as an unwanted effect, but at the same time it is a sufficient criterion for vasodilation, so the nitrate dose should be increased until moderate headache.

Calcium antagonists at the National Assembly is not applicable. In accordance with the recommendations of evidence-based medicine dihydropyridines (nifedipine and its analogues) are able to increase the development of MI, SCD and cardiac death (in some studies only). Moreover, diltiazem and verapamil should not be used in patients with pulmonary edema and severe left ventricular dysfunction.

Thrombolytics in UA are not assigned.

Standards of treatment NSA

Step 1. Relief of pain:

- nitroglycerin sublingual dose of 0.5-1 mg better nitroglycerin / in for 1-2 days;
- talamonala: 2.1 ml of 0.005% solution of fentanyl 1.2 ml of 0.25% solution in droperidol / or in V / m;
- promedol, morphine, tramadol, seduksen in optimal doses.

Step 2. Prevention of thrombosis with anticoagulants or antiplatelet agents:

- not fractionated heparin bolus dose of 60-70 u / kg (maximum 5000 U / hr. / In infusion followed by 12-15 units. / Kg (maximum of 1 250 units / hr.) Under control of the activated partial thromboplastin time (APTT), which should be increased by 1.5-2 times;
- dalteparin - 120 U / kg every 12 hours (maximum 1000 units twice a day.).
- enoxaparin - 1 mg / kg n / k every 12 hours and the first dose can be administered in / bolus;
- first ASA 325 mg / day, then - 100 mg / day;
- clopidogrel 75 mg / day (loading dose - 300-600 mg), followed by maintenance - 75-100 mg / day;
- abciximab / v bolus dose of 0.25 mg / kg, followed by infusion at 0.125 mg / kg / min (maximum 10 mg / min) for 12-24 hours (12 hours after the percutaneous coronary intervention) patients who are scheduled myocardial revascularization; medication is particularly indicated when the threat of complications and a high content of troponin T and I.

Step 3. Warnings pain attacks using antianginal drugs:

- mononitrates: isosorbide mononitrate by 40-80 mg / day;
- dinitrate: isosorbide dinitrate, nitrosorbid, izoket, sustak, kardiket in optimal doses;
- β -blockers metoprolol succinate of 100-200 mg / day; nadolol - 40-180 mg / day; carvedilol - 25-100 mg / day; nebivolol - 5.10 mg / day (Prinzmetal angina, these drugs are not shown).

Step 4. The correction of lipid metabolism using antisklerotichnih means for a long time:

- lovastatin 20-40 mg / day or
- simvastatin 20-40 mg / day or
- Pravastatin 10-40 mg / day or

- Atorvastatin 10-20 mg / day.

Step 5. myocardial revascularization (the need for urgent revascularization is determined by the results of the risk assessment for the GRACE scale) (via transluminal balloon angioplasty, coronary artery stenting and coronary artery bypass grafting).

Acute myocardial infarction

Myocardial infarction (MI) - is necrosis of the heart muscle due to coronary circulation acute disorders.

According to the data of ECG diagnosed macrofocal (transmural) and melkoochagovyj (subendocardial, intramural) MI. When transmural infarction necrosis develops the bulk of the ischemic myocardium. Meanwhile, when most of the small-focal myocardial infarction ischemic cardiomyocytes remains viable. In fact, the pathological changes is the discrepancy between the histological and ECG - data. In this connection, in recent years the use of such terms as "MI with tooth Q (macrofocal) and" IM without tooth Q (melkoochagovyj).

If the new myocardial necrosis develops within the first 28 days of the onset of acute myocardial infarction, it is called recurrent MI. After 28 days of onset of MI diagnosis was replaced with "myocardial infarction" reinfarction deem that develops later than 28 days after suffering a first myocardial infarction.

In 95% of cases the cause is coronary infarction, which causes acute focal ischemia and necrosis of cardiomyocytes. Aggravating factors are giperkateholamiya, blood hypercoagulation, hyperlipidemia, retrograde thrombosis, physical and psycho-emotional overload.

In 80-85% of cases it is the classic version of MI (status anginosus). The disease starts with an acute attack of angina in the chest lasting more than a minute LP feeling approaching death. When collecting history, more than half Patients indicate that during the last 1-3 weeks before the onset of myocardial infarction have been prolonged angina (unstable), which were not removed by nitrates. Most long-anginal pain occurs at rest in the first half of the day, in waves, lasting up to several hours or even days. The pain may spread to the left side of the chest, the left arm, jaw, epigastrium. Patients feel a contraction or a burning sensation in the chest. The young and middle-aged persons are pronounced vegetative disorders: sweating, tachycardia, arrhythmia, nausea, vomiting, hypertension or hypotension. Consciousness is usually preserved, although in some cases there are anxiety, dizziness, or excitement.

Myocardial infarction accompanied by neutrophilic leukocytosis up to $10,0-12,0 \times 10^9 / L$ with eozinopenieyu and lymphopenia Increasing the number of white blood cells occurs within 2 hours after the onset of anginal attack with a maximum at 2-4-th day. Intensity leukocytosis depends on the initial number of leukocytes, extensive necrosis, fever In most cases, the ESR increases with 2-4-day and lasts for 2-4 weeks. Simultaneously increased level of C-protein and fibrinogen to blood hypercoagulability. The revealed changes are of limited diagnostic value.

By rezorbtivno necrotic syndrome in myocardial infarction, in addition to changes in the blood, referred hyperfermentemia, which is quite often pathognomonic sign of illness infarction myocardial necrosis enzymes enter the blood and increases their activity.

Enough is sensitive myocardial enzyme creatine phosphokinase (CPK), its activity increases within 2-3 hours after the onset of pain, reaches a maximum at 24-36 hours and returns to the original level at 3-4 days when there is no recurrence. High levels of CK indicate the extensiveness of necrosis. More sensitive and specific marker of myocardial infarction is the growth fraction (CK-MB). Maximum activity of the isoenzyme CK-MB is for 12-24 hours, so it is necessary to conduct a study in dynamics. After successful reperfusion (thrombolysis, angioplasty) has been rapid growth in the activity of CK-MB. Within 24-36 hours after the onset of anginal attack to carry out research on the activity of CK-MB is inappropriate. Meanwhile isoenzyme activity can be high during cardioversion, myocarditis, massive trauma or muscular dystrophy, rhabdomyolysis, and the like.

blood concentration of lactate dehydrogenase (LDH) begins to grow after the 8th hour after myocardial infarction, with a maximum of 3-5 days and returning to normal values in the 8-12th day. The false increase in the activity of the enzyme occurs in liver disease, leukemia, lung and kidney infarction, myocarditis. That's why more valuable diagnostic test is to determine the activity of the isoenzyme LDG1 contained mainly in the heart muscle. Value $LDG1 / LDG2 > 1.0$ is a sign of acute myocardial infarction. Isoenzyme activity LDG1 increases by 12-24 hours, and remains high for 2-3 days with a gradual return to normal values.

Increased activity of the third enzyme aspartate aminotransferase (ACT) is even less specific diagnostic value. enzyme levels in the blood increases after 6-10 h with a peak at 18-36 hours and return to normal on day 4-5. In addition to acute myocardial infarction, an increase of activity ACT is celebrated in myocarditis, aggressive hepatitis, pericarditis, pulmonary infarction, after defibrillation, and paroxysms of tachyarrhythmias.

Consequently, the increase in LDH activity and ACT provides significantly longer in MI compared with CK. Meanwhile, the high concentration of CPK in the blood is much more reliable and specific diagnostic test.

Studies show that certain diagnostic importance is the definition of myoglobin in the blood during acute myocardial infarction. Its concentration is determined by ELISA or radioimmunoassay. The concentration of myoglobin in the blood rises in 1-2 hours after the onset of ischemic pain with the achievement of maximum for 10-12 hours. Its level may exceed 8-12 times normal levels return to baseline levels within 48-72 hours. Repeated increase in the concentration of myoglobin suggests recurrent MI.

The most affordable and the most reliable method of diagnosis is the ECG diagnosis. Reliable diagnostic criteria for acute myocardial infarction is the appearance of pathological Q waves with progressive changes in the ST segment and T wave. In 80-85% of cases of early diagnostic test macrofocal infarction (MI with tooth Q) is ST-segment elevation on the contour line (curve Purdy) in one or more leads a decline from the R-wave appearance of pathological tooth Q. Still later comes the decline in ST segment with the formation negative ("coronary") T wave anterior MI when these changes are in I and II, V2-V5 leads, MI and at the back - in the II and III, AVF leads. The shortness of diagnostic use cases precordial mapping (ECG in 35 leads). The above ECG changes persist for several months or even years.

When MI without tooth Q (microfocal MI) typical ECG changes does not happen. In most cases, the QRS complex configuration is not changed, except for the possible reduction of R wave height observed ST segment depression with T-wave inversion.

Identified on the ECG changes with suspected myocardial infarction require careful study. In acute myocardial infarction ST segment increased by more than 2 mm above the contour line.

with a change in form. At the same time, these changes happen at Prinzmetal angina, ventricular aneurysm, pericarditis, early repolarization syndrome. Reciprocal changes are also typical for MI. By reducing the ST comes inversion of the T wave, which is becoming a deep and balanced. At the same time we must remember that changes in the T wave happen at very different pathology (hypokalemia, acute infection, uremia, myocarditis, endocrine disorders, and so on. Etc.) and even in young healthy individuals.

According to the dynamic changes in the ECG distinguish the following stages of acute myocardial infarction:

I. Stage damage (acute) - from the beginning of the ST segment elevation to the formation of pathological tooth Q.

II. The acute stage - the presence of tooth Q, ST elevation and merge it with the tooth T.

Subacute Stage

III - the presence of tooth Q, ST return to the contour line, a negative, "coronary" T.

IV Scar stage - the presence of Q wave with positive tooth T

According to ECG signs of acute myocardial infarction with Q wave the following basic localization

- 1) Front MI - availability of Q or QS in V, -V ^,
- 2) Lower (posterolateral diaphragmatic) - the presence of Q or QS in II, III and AVF leads,
- 3) Side - the presence of Q or QS in i, AVL, V ^ -V ^ leads.
- 4) The back (posterior-basal dorsal) - Reciprocal ECG changes in V ^ -V ^ leads.

Differential diagnosis of MI.

The diagnosis of myocardial infarction is difficult in atypical cases of the disease. Without the pain of MI is diagnosed on the basis of ECG examination with a detailed survey of the presence of discomfort in the recent past. Status gastralgicus occurs in 2% of patients with posterolateral diaphragmatic MI. In these patients, there is a sharp pain in the upper abdomen, nausea, vomiting, diarrhea, fever. To exclude appendicitis, perforated ulcer, cholecystitis, intestinal obstruction is necessary to provide dynamic observation with repeated ECG studies. Importance has collected detailed medical history, presence hyperenzymemia, ischemic ECG changes. Where necessary, conduct an ultrasound examination of the abdomen organs gastroduodenoscopy. At the same time, the combination of the two diseases. Thus, acute pancreatitis can cause exacerbation of ischemic heart disease until the development of myocardial infarction.

In acute dissecting aneurysm of the thoracic aorta intense chest pain occurs suddenly, often against a background of hypertension. Pain spreading to the neck, back, both arms and, unlike myocardial infarction with time shifts in the lumbar region of the spine and pelvis. As a result of internal bleeding diagnosed with anemia. Some patients develop cardiogenic shock. No ECG changes characteristic of myocardial infarction. Differential diagnostic significance radiology, X-ray and echocardiography. If the patient does not die suddenly, then 1-2 weeks developed abdominal organ ischemia with heart failure. Difficulties arise in the differential diagnosis of myocardial infarction with pulmonary embolism, which are the cause of

thrombophlebitis, phlebothrombosis, atrial fibrillation, IE, chronic non-specific lung disease. Most thromboembolism occurs in the postoperative period, especially after abdominal, obstetric and urological surgery, childbirth. For it is characteristic more acute onset, chest pain combined with shortness of breath, cyanosis, sinus tachycardia. When massive thromboembolism is a shock. Hemoptysis., Pockets of moist rales, pleural friction assist in the diagnosis. Later appear fever, leukocytosis, increased ESR. In the acute weakness of the right ventricle to the 2-3 th day there is the risk of right ventricular failure. The ECG - blockade of right bundle branch block, P-pulmonale in the II and III leads to the negative T in V1-V3, pathological QIII, but there are no pathological tooth QII.

Unlike infarction in acute pericarditis, pericardial friction occurs in the first hours, enhanced by pressing a stethoscope on the chest. Pain occurs simultaneously with fever, leukocytosis, increased erythrocyte sedimentation rate, without the typical irradiation. ST segment over the isoline in all standard leads, no pathological Q waves with constant R.

When left-sided spontaneous pneumothorax pain is sharper with the progressive increase in shortness of breath. On the affected side of the percussion - tympanic sound, breathing weakened or not to hear. No ECG changes characteristic of myocardial infarction. The activity of cardiac enzymes and myoglobin levels within normal limits.

The clinical picture of acute pancreatitis like. Acute unbearable pain in the upper abdomen extends to the left side of the chest, left arm, interscapular region. ECG changes characteristic of melkoochagovogo MI - negative T, depression ST, arrhythmia. Diagnostic value has pain that is acute pancreatitis more constant, not wavy, as in myocardial infarction. In hemorrhagic pancreatitis develops shock with a sharp drop in blood pressure. Assist in the diagnosis of determining the activity of diastase, repeated ECG monitoring of patients.

Complications of myocardial infarction.

The severity and frequency of complications of myocardial infarction associated with the degree of violation of the coronary blood flow and myocardial contractility. Short-term circulatory disorders lead to the spread of necrosis subendocardial zone to epikardialnoy. If the duration of the blood flow stops more than 3-6 hours, then comes the death of 60-80% of ischemic myocardium. At ECHO it looks like the area of akinesia or hypokinesia. It promotes the spread of necrosis hypotension and loss of several coronary atherosclerosis.

In acute myocardial ischemia of the affected land ceases to fall, there comes a passive extension of cardiomyocytes with their bulging during systole. The elongation and tensile ischemic segments and necrotic myocardial infarction in the early period was called the "expansion of the myocardium, which is the basis for the emergence of LV dilatation. When LV dilatation and reduced cardiac output index, ejection fraction with the increase in LV end-diastolic pressure, which leads to left ventricular systolic dysfunction and congestive heart failure.

In addition to left ventricular systolic dysfunction, the cause of heart failure when it can be diastolic dysfunction, the mechanism of which is myocardial relaxation disorder with high rigidity. This leads to a stagnation in the left atrium and pulmonary veins, leading to stagnation in the pulmonary circulation with ejection fraction within normal limits.

Reduced left ventricular pump function leading to acute left ventricular failure, in which the organs and tissues do not receive the necessary blood supply. Early clinical signs of acute left ventricular failure are sinus tachycardia, shortness of breath, cough, crackles in the lungs.

Such severe manifestations of heart failure as cardiac asthma and pulmonary edema are more often in the second half of the first day of MI against the backdrop of myocardial infarction, heart failure, Cardiosclerosis stenosis in arterial hypertension and protodiastolic gallop rhythm. When cardiac asthma patient complains of dyspnea, cough, and sometimes wet, orthopnea. Cardiac auscultation deaf, listens protodiastolic gallop. In the lungs breathing hard, both dry and moist rales. If asthma goes into cardiac pulmonary edema, it appears klekochuschee breath with the release of pink frothy sputum. The skin is covered with cold sweat, there is peripheral cyanosis, tachycardia. Throughout lung crackles are heard krupnopuzyrchatye When radiography of the chest diagnose bilateral pulmonary edema, Kerley lines.

The most formidable and fatal complication of myocardial infarction is cardiogenic shock, mortality in which more than 80-90%. It develops necrosis more than 35-40% of left ventricular muscle mass. Cardiogenic shock occurs most often during the first days as a result of left ventricular systolic dysfunction due extensive ischemia or necrosis. Quite rarely the cause is ventricular septal rupture or mitral insufficiency. Later, the development of cardiogenic shock due to the expansion of areas of necrosis and progression of weakness of the left ventricle. If it is the case of tissue hypoperfusion with renal insufficiency, arterial hypoxemia and tissues acidosis.

External myocardial rupture is the second fatal complication of acute myocardial infarction, it is the cause of death in 10% of all cases. Rupture occurs at the boundary of the slit necrosis on 1-9 th day of illness. It promotes the appearance of complications of advanced age, hypertension, female. Sometimes the rupture is preceded by pain in grudi.rt.st. with pulmonary hypertension and peripheral vascular resistance high.

Dressler syndrome (Dressler's syndrome) diagnosed in 3% of patients at 2-6 weeks by him in his main role belongs to the pathogenesis of autoimmune reactions with the body autoserotherapy autoantigens necrotic cardiomyocytes. Dressler's syndrome is manifested by fever, pericarditis, pleurisy and pnevmoshtom. For it is peculiar to relapsing course. In some cases, there is a hemorrhagic vasculitis, joint disease and kidney failure. By atypical variants include "front chest wall", "brush syndrome ", "shoulder syndrome syndrome ".In the period of acute fever, leukocytosis, eosinophilia, increased ESR.

The early complications include myocardial infarction early postinfarction angina that occurs at rest for 3 weeks from the onset of acute myocardial infarction. At the heart of it lies the pathogenesis of restenosis, infarct-related vessel with an increase in parietal thrombosis. Most often it is diagnosed in patients with small-focal myocardial infarction. In the early post-infarction angina at high risk of reinfarction and PKC. Among the adverse prognostic factors in this are the expansion of the necrotic zone, the occurrence of ventricular arrhythmias, hemodynamic deterioration.

Earlier thromboembolic complications in acute myocardial infarction was diagnosed in 10% of cases, when used in the early days of heparin, antiplatelet agents, they are 3% of all cases. The probability of occurrence of thromboembolism during the first 10 days high at extensive anterior infarction with dyskinesia apex of the heart and the formation of aneurysms. Contribute to the emergence of complications of congestive heart failure, mural thrombosis.

IV. Means of self-control:

Tests:

1. Patient K., 65 years old, sought medical assistance complaining of squeezing chest pain that lasts more than an hour, radiating to the left arm, sweating, general weakness, taking nitroglycerin, but the pain has not diminished, the ECG ST-segment elevation, T wave is not differentiated in more than two leads, a diagnosis takes place:

- A. Sinus tachycardia
- B.. The attack of angina pectoris
- C. Myocardial infarction
- D. Atrial Fibrillation
- E. AV block

2. Patient N., 72, was admitted to the intensive care unit with a diagnosis of acute myocardial infarction, thrombolytic therapy is possible with:

- A. The presence of concomitant aortic dissecting aneurysm;
- B. a stroke in the past 4 weeks;
- C. Massive surgical operations for the past 3 weeks;
- D. gastrointestinal bleeding within the last 3 weeks;
- E. The systolic blood pressure below 100 mm Hg

3. Patient S. 54 years delivered an emergency basis in the BRIT with intense burning pain behind the breastbone. The ECG-arc-shaped lifting ST V2-V5 10 mm, tooth QS in V2-V5. What is the diagnosis of the patient?

- A. The acute stage of Q-positive front widespread myocardial infarction;
- B. Acute Q-stage front common adverse myocardial infarction;
- C. The acute stage of Q-positive transmural anterior myocardial infarction common;
- D. The acute stage of Q - positive, rear transmural myocardial infarction;

4. A patient .. 60 years, was admitted to the ICU with complaints about the unbearable pain in the heart, sweating, general weakness, which lasts for 3 hours. ECG ST elevation in III and aVF, S1 S2 leads, pathological tooth Q. What is the diagnosis of the patient?

- A. Q - positive basal transmural posterolateral myocardial infarction;
- B. Q-positive transmural anterior myocardial infarction common;
- C. Q-negative common front myocardial infarction;
- D. Q - positive, rear transmural myocardial infarction;

5. The long-term secondary prevention of myocardial infarction include:

- A. aspirin, beta-blockers, nitrates
- B. aspirin, beta-blockers, statins, IAPF
- C. aspirin, dihydropyridine calcium antagonists, statins
- D. aspirin, vitamins E and A, nitrates

Recommended literature:

A. Main:

1. "Harrison's principles of internal medicine", Editors: Anthony S. Fauci, Dennis L. Kasper, Stephen L Hauser, Dan L. Longo, Joseph Loscalzo, McGraw-Hill Education / Medical; 19 edition (April 8, 2015), 1-2 volumes, 3000 p.
2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. Davidson's Principles and Practice of Medicine: With STUDENT CONSULT Online Access, 21e (Principles & Practice of Medicine (Davidson's)) by Nicki R. Colledge BSc FRCP(Ed), Brian R. Walker BSc MD FRCP(Ed) and Stuart H. Ralston MB ChB MD FRCP FMedSci FRSE (Paperback - Mar 11, 2010)Kumar and Clark's Clinical Medicine, 7e (Kumar, Kumar and Clark's Clinical Medicine) by Parveen J. Kumar (Paperback - Jul 2, 2009)
4. 1000 Questions and Answers from Kumar & Clark's Clinical Medicine, 2e [Paperback] Parveen Kumar CBE BSc MD FRCP FRCP(Edin) (Editor), Michael L Clark MD FRCP (Editor)
5. Differential Diagnosis in Internal Medicine: From Symptom to Diagnosis by Walter Siegenthaler (Mar 21, 2007)
6. Symptom to Diagnosis: An Evidence Based Guide, Second Edition (LANGE Clinical Medicine) by Scott D. C. Stern (Sep 16, 2009)
7. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
8. Harrison's Gastroenterology and Hepatology by Dan Longo and Anthony Fauci (May 3, 2010)
9. Mayo Clinic Gastroenterology and Hepatology Board Review (Mayo Clinic Scientific Press) by Stephen Hauser (Jun 23, 2011)
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. – 2012. p.
12. Sonographer's Handbook of Diagnostic Ultrasound by Jason R. Young M.D. (Feb 23, 2011)

Additional literature:

1.Kovalyova O.M., Asheulova T.V. Propedeutics to internal medicine. Part 1, Diagnostics. Vinnytsya, Nova Knyha, 2006, 424 p

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