VOLGOGRAD STATE MEDICAL UNIVERSITY Department of Pathological Anatomy

Blood circulation disorders. Stasis. Thrombosis. Embolism. Shock. Ischemia	
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Purpose of the lesson: Studying the causes, pathogenesis, and significance for the body of the main types of circulatory disorders (thrombosis, embolism, disseminated intravascular coagulation syndrome, ischemia, heart attack). Mastering the methods of morphological diagnostics of these pathological processes.

The student should know:

- 1. Terminology used in the section on circulatory disorders.
- 2. Etiology, pathogenesis, morphogenesis, main types of circulatory disorders (thrombosis, embolism, disseminated intravascular coagulation syndrome, ischemia, heart attack).
- H. Principles of classification of these general pathological processes.
- 4. Typical morphological changes and diagnostic methods for thrombosis, embolism, disseminated intravascular coagulation syndrome, ischemia.
- 5. Basics of clinical and anatomical analysis and principles of constructing a diagnosis in identifying signs of circulatory disorders.

The student must be able to

- 1. Describe morphological changes in the studied macro and micropreparations, electron diffraction patterns.
- 2. Based on the description, give a conclusion about the nature of the circulatory disorder and its clinical manifestations.

Block of information on the topic:

Thrombosis

Normal blood circulation and the rheological properties of blood are maintained by a regulatory system that ensures a fluid state of the blood, affects the permeability of the vascular wall and prevents the release of plasma elements into the interstitium.

Impaired regulation of hemostasis, i.e., the balanced interaction of its 4 systems of coagulation, fibrinolysis, endothelial structure and platelets leads to intravital pathological coagulation of blood in the lumen of the bloodstream, thrombosis.

Reasons leading to development

- 1. Violation of the integrity of the vascular wall endothelium.
- 2. Violation of blood flow
- H. Changes in blood composition leading to hypercoagulation.

In structure and color, blood clots can be white, red, hyaline and mixed. White blood clots are more common in arteries, blood clots are dense, fragile, grayish-white masses. If they are parietal, they have a rough corrugated surface, which reflects the rhythmic prolapse and adhesion of platelets and fibrin and blood flow conditions. Red blood clots. In addition to platelets and fibrin, they are represented by a large number of erythrocytes, such blood clots are soft, gelatinous, dark red in color, their surface is uneven, dull. More often, red blood clots are found in the veins in conditions of slowing blood flow. Mixed blood clots often form in the veins, in the cavity of the aortic and heart aneurysms. Hyaline blood clots form in small vessels. They are mainly composed of destroyed erythrocytes, platelets, and precipitating plasma proteins.

In relation to the lumen of the vessel or the cavities of the heart, thrombi are parietal, obstructing and spherical. Blood clots can form in arteries, veins, cardiac cavities, aneurysms of the heart and blood vessels.

Outcomes of blood clots include:

- an increase in the size of a thrombus by layering thrombotic masses on the primary thrombus
- contraction (compression) of the thrombus tissue by reducing the mass of fibrin in it,
- lysis of a thrombus with the participation of plasmin and proteolytic enzymes of neutrophilic leukocytes;
- organization and sewerage of a thrombus, ie its replacement by connective tissue and the development of a network of capillaries;
- the formation of fibrous tissue at the site of a thrombus and its calcification (petrification);
- detachment of a thrombus and the development of thromboembolism;
- purulent septic fusion thromboembolism

Embolism

An embolism is the transfer of material or particles by the blood stream that are absent in normal blood.

An embolus is a particle that moves about in our blood vessels, either in the veins or arteries. Most emboli are composed of clotted blood cells. A blood clot is called a thrombus and a moving blood clot is called a thromboembolus.

Types of embolism

There are several types of embolism:

pulmonary embolism: An embolus, usually formed in the leg (sometimes known as a deep vein thrombosis or DVT), lodges in one of the arteries of the lungs. Many emboli are broken down by the body and go away by themselves; however, serious pulmonary embolism may cause death.

brain embolism: If a blood clot travels to the brain, this causes an ischemic stroke or TIA (transient ischemic attack).

retinal embolism: Small clots that wouldn't block a major artery can block the smaller blood vessels feeding the retina at the back of the eye. The result is usually sudden blindness in one eye.

septic embolism: This occurs when particles created by infection in the body reach the bloodstream and block blood vessels.

amniotic embolism: Not all emboli are made of clotted blood. In pregnancy, the womb is filled with amniotic fluid, which protects the fetus. Amniotic fluid can embolize and reach the mother's lungs, causing pulmonary amniotic embolism.

air embolism: Scuba divers who rise to the surface too rapidly can generate air embolism, bubbles in the blood that can block arterial blood flow.

fat embolism: If fat or bone marrow particles are introduced into the blood circulation, they may block blood vessels the way a blood clot or air bubble can.

Causes

Most embolisms happen to people who have risk factors for blood clot formation, such as smoking and heart disease. Other risk factors for other types of emboli include high blood pressure, atherosclerosis (buildup of fatty plaque in the blood vessels), high cholesterol, and obesity.

The primary cause of most pulmonary embolisms is deep vein thrombosis (DVT). This is a condition in which the veins of the legs develop clots. Natural agents in the blood often dissolve small clots without causing any effects of blockage. Some clots are too big to dissolve and are big enough to block major blood vessels in the lungs or in the brain.

Factors that slow blood flow in the legs may promote clotting. People can develop a DVT or pulmonary emboli after sitting still on long flights or after immobilization of the leg in a cast, or after prolonged bed rest without moving the legs. Other factors associated with DVT or pulmonary embolism include cancer, previous surgery, a broken leg or hip, and genetic conditions affecting the blood cells that increase the chance of blood clot formation.

As an embolus moves through the body's blood vessels, it's likely to come to a passage it can't fit through. It lodges there, backing up blood behind it. The cells that normally get their blood supply via this passage are starved of oxygen (ischemia) and die. This condition is called an embolism.

Thromboembolism is one of the most common adverse outcomes of thrombosis, when a thrombus or parts of it break off and begin to circulate through the bloodstream (orthograde embolism). Much less often, thromboembolism, as a result of its severity, moves against the bloodstream (retrograde embolism). Prognostically, the most unfavorable is thromboembolism of the pulmonary artery and its branches, which can be the cause of a sudden cessation of the blood supply to the lungs or, which is more common, a sudden cardiac arrest due to the pulmonary coronary reflex. In this case, spasm of bronchioles, branches of the pulmonary artery and

coronary arteries occurs. Blockage of the branches of the pulmonary artery by parts of the embolus leads to the development of hemorrhagic pulmonary infarction, especially against the background of venous stasis. Also, quite often there is a separation of blood clots in the presence of parietal blood clots at the site of ulcerated atherosclerotic plaques. The source of thromboembolism can be blood clots in the heart cavities or thrombotic overlays on the valve cusps -

In addition to detached blood clots, tumor cells can enter the blood when a malignant tumor of the vessels grows. During their engraftment (implantation), daughter nodes develop

metastases. Microbial embolism is also possible (microbes circulating in the blood obturate the smallest vessels), fat embolism (fat particles enter the vessels, for example, with a fracture of long tubular bones). Most often, fat particles enter the capillaries of the lungs, leading to acute pulmonary failure and cardiac arrest. During thoracic operations, venous or arterial catheterization, during decompression, air embolism is possible, when air bubbles (if their volume in the blood is more than 100 ml) cause blockage of the capillaries of the pulmonary circulation and lead to sudden cardiac death.

DIC syndrome

Disseminated intravascular coagulation DIC syndrome is characterized by widespread coagulation of blood in the vessels of the microvasculature, due to the progressive activation of coagulation. This condition is characterized by a combination of hypercoagulation in small vessels with the simultaneous development of hemorrhagic diathesis and, as a consequence, the occurrence of acute, often fatal bleeding. Hemorrhagic phenomena are due to pronounced consumption of blood clotting factors and an excessive increase in fibrinolysis, which occurs in response to an increase in blood clotting, DIC syndrome develops in severe conditions of the body:

- embolism by amniotic fluid;
- placental abruption;
- hypoxia of newborns;
- burns;
- acute pancreatitis;
- infectious and septic conditions:
- poisoning with hemocoagulating snake venom;
- malignant tumors of the lung, pancreas and prostate, colon, stomach;
- transfusion of incompatible blood;
- with almost all types of shock (very rarely with cardiogenic shock).

Ischemia

Ischemia (local anemia) tissue condition with a decrease or insufficient blood supply. The causes of ischemia are occlusion, obstruction, or obstruction (i.e., blockage) of the arteries by a thrombus, embolus, and prolonged vasospasm. Ischemia can occur when an artery is compressed by a tumor, ligature, or as a result of blood redistribution.

Heart attack. This is tissue necrosis due to the cessation and significant decrease in arterial blood flow. Macroscopically, heart attacks are divided into white (anemic) and red (hemorrhagic).

The shape of the heart attack depends on the angioarchitectonics of the organ, the severity of collateral blood supply and can be wedge-shaped (triangular) and irregular. If the heart attack reaches the capsule or the serous membrane of the organ, then fibrin often appears on them. Final diagnosis and determination of the time of development of a heart attack are possible with microscopic examination. On the periphery of the focus of necrosis, edema, hyperemia develop; leukocytes, macrophages and mast cells accumulate, a zone of demarcation (i.e., restrictive) inflammation is formed. The accumulation of leukocytes containing proteolytic enzymes and partial resorption promote softening, dissolution and can lead to rupture of necrotic tissue. Along

with infiltration, the number of newly formed vessels increases and granulation tissue is formed, the maturation of which leads to the formation of a scar at the site of the infarction. This process is called organization. In the kidneys in the area of the scar, retractions are formed, and the surface of the kidney becomes uneven, coarse. In the brain, with a small size of the necrosis zone, a small glial scar can form, with a large cavity filled with fluid (cyst). Hemorrhagic heart attacks of the lung with the addition of an infection and the development of purulent inflammation can melt. Hemorrhagic bowel infarctions are usually complicated by bowel gangrene, wall perforation, and fecal peritonitis.

Lesson plan.

Explore and describe:

1. Macropreparation "Mixed obstructing thrombus in the aorta."

Examine the mixed thrombus in the aorta on a macroscopic picture. Describe the macro-preparation "Mixed obstructing thrombus in the aorta". Mark the color, characterize the surface of the thrombus. Determine its relation to the lumen of the vessel and intima.

2. Macropreparation "Metastasis of melanoma in the liver".

Examine tissue embolism in the liver by a macroscopic picture. To describe the macropreparation "Metastasis of melanoma in the liver". Reveal the presence and number of metastases. Mark the color, shape, size of the nodes. Describe the cut surface.

3. Additionally. Macropreparation "Myocardial infarction with rupture".

To study myocardial infarction on a macroscopic picture. Describe the macropreparation "Ruptured myocardial infarction." Determine the zone of infarction by the characteristic macroscopic features. Identify signs of myocardial rupture and cardiac tamponade.

4. Macropreparation "Pulmonary embolism".

To study pulmonary thromboembolism according to the macroscopic picture. Describe the macropreparation "Pulmonary embolism". Mark the color, size, characterize the surface of the thromboembolus. Determine its relation to the lumen of the vessel and intima.

5. Macropreparation "Hemorrhagic pulmonary infarction".

To study hemorrhagic pulmonary infarction according to the macroscopic picture. To describe the macro-preparation "Hemorrhagic pulmonary infarction". Determine the shape, color, relation to the pleura of the infarction zone.

6. Additionally: Macropreparation "Ischemic spleen infarction".

To study ischemic spleen infarction according to the macroscopic picture. To describe the macropreparation "Ischemic spleen infarction". Determine the shape, color, relation to the capsule of the infarction zone.

Micropreparations:

Explore and draw:

1. Micropreparation"Mixed thrombus in a vessel."

Examine a mixed blood clot in a vessel using a microscopic picture. Describe the micropreparation "Mixed thrombus in a vessel" (staining with hematoxylin and eosin). Pay attention to the state of the vessel lumen, the structure of the thrombus, the presence of fibrin, platelets, erythrocytes, leukocytes, the site of endothelial damage.

2. Micropreparation "Organizing blood clot".

To study the organization of a blood clot in a vessel using a microscopic picture. Describe the micropreparation "Organizing thrombus" (staining with hematoxylin and eosin). Pay attention to the state of the vessel lumen, the structure of the thrombus, the presence of fibrin, platelets, erythrocytes, leukocytes, the area of endothelial damage, as well as the growth of connective tissue in the thrombus (the presence of fibroblasts and collagen fibers), the formation of new vessels - vascularization.

3. Micropreparation "Fatty embolism of the lung."

To study fatty pulmonary embolism in a microscopic picture. Describe the micropreparation "Fatty embolism of the lung" (coloring Sudan III). Pay attention to the state of the vessel lumen, the color of the emboli in an orange-yellow color.

4. Additionally: Micropreparation "Myocardial infarction".

To study myocardial infarction on a microscopic picture. Describe the micropreparation "Myocardial infarction" (staining with hematoxylin and eosin). Pay attention to the state of cardiomyocytes, karyolysis, the presence of a zone of demarcation inflammation with neutrophilic infiltration.

5. Micropreparation «Hemorrhagic pulmonary infarction».

To study hemorrhagic pulmonary infarction on a microscopic picture. Describe the micropreparation "Hemorrhagic pulmonary infarction" (staining with hematoxylin and eosin). Pay attention to the state of the interalveolar septa, karyolysis, the presence of a large number of erythrocytes in the lumen of the alveoli.

6. Micropreparation"Stasis in the capillaries of the brain."

Examine blood stasis using a microscopic picture. Describe the micropreparation "Stasis in the capillaries of the brain" (staining with hematoxylin and eosin). Pay attention to the state of the lumen of the capillaries, sludge of erythrocytes, edema of the brain tissue.

7. Micropreparation "Tumor emboli in a vessel".

Examine tumor emboli in a vessel using a microscopic picture. Describe the micropreparation "Tumor emboli in a vessel" (staining with hematoxylin and eosin). Pay attention to the state of the vessel lumen, to the presence of tumor cells in the lumen, to the infiltration of the vessel wall by tumor cells.

Test QUESTIONS

Select all correct answers

- 1. The formation of blood clots in small vessels during stasis causes:
- a) deformation of the walls of blood vessels,
- b) violations of the axial layer of blood flow,
- c) accumulation of activated coagulation factors,
- d) changes in blood composition,
- e) blood hypercoagulability

Choose one correct answer

2.A young man who died suddenly, an autopsy revealed a rupture of the wall of the middle cerebral artery in the area of the

aneurysm (protrusion) and a rounded lesion containing blood in the frontal lobe of the brain. This type of hemorrhage is called.

- a) bruising,
- 6) hematoma,
- c) petechiae,
- d) purple.

Select all correct answers

- 3, Imbalance between stimulants and inhibitors of platelet aggregation can lead to:
- a) hyperemia,
- b) bleeding,
- c) stasis,

d) thrombosis.

Choose one correct answer

- 4. White blood clots are described as.
- a) dense grayish-red masses,
- b) elastic grayish-white masses,
- c) elastic grayish-red masses,
- d) dense grayish-white masses.

Select all correct answers

- 5. The most common causes of fatty embolism:
- a) excessive accumulation of neutral fat,
- b) fractures of tubular bones,
- c) fractures of cancellous bones,
- d) injury with crushing of fatty tissue.

Select all correct answers

- b. Pulmonary embolism can result in.
- a) sudden death,
- b) fatty degeneration of the myocardium,
- c) rupture of the vessel wall,
- d) lung infarction.

Select all correct answers

- 7. The death of a 23-year-old woman occurred during childbirth from amniotic fluid embolism. Histological examination revealed:
- a) pulmonary vasospasm,
- b) DIC syndrome,
- c) hemosiderosis of the lungs,
- d) lung infarctions,
- e) vein thrombosis of the pelvic tissue.

Select all correct answers

- 8. Participate in coagulation and fibrinolysis.
- a) macrophages,
- b) endothelium,
- c) platelets,
- d) erythrocytes,
- e) mesothelium.

Establish compliance

- 9. Type of blood clot: Localization:
- 1) white, a) capillaries,
- 2) red, b) arteries,
- H) hyaline, c) aneurysm cavity,
- 4) mixed, d) lymphatic vessels,
- e) veins.

Select all correct answers

- 10. A 27-year-old man with polycythemia developed small-vessel thrombosis in the brain, leading to cerebral heart attacks and death. The main causes of thrombus formation in this case.
- a) increased blood viscosity and stasis,
- b) decreased adhesion and aggregation of platelets
- c) accumulation of activated coagulation factors,

Choose one correct answer

- 11. "Thrombus of stagnant blood flow":
- a) white,
- b) red,
- c) mixed,
- d) hyaline.

Establish compliance

- 12. Education 1-2: Sign a-e:
- 1) a blood clot,
- 2) postmortem blood clotting
- a) dull surface,
- b) smooth surface, c) attached to the vessel wall, d) lies in the vessel lumen, e) dense, fragile.

Select all correct answers

- 13. The formation of a red heart attack corresponds to:
- a) occlusion of the venous bed,
- b) arterial thrombosis
- , c) double type of organ blood supply,
- d) venous stasis,
- e) arterial hyperemia

Select all correct answers

- 14. When the lumen of the renal artery narrows by an atherosclerotic plaque in the kidney tissue:
- a) ischemic infarction,
- b) cyst,
- c) atrophy,
- d) sclerosis,
- e) hemorrhagic infarction.

Choose one correct answer

- 15. Outcome of spleen infarction:
- a) organization,
- b) sewerage,
- c) vascularization,
- d) cyst formation,

e) formation of a glial scar.

Select all correct answers

- 16. A 54-year-old man on the 3rd day after the onset of myocardial infarction died of heart rupture in the affected area. The reason for the rupture of the muscle wall:
- a) autolysis,
- b) proteolysis,
- c) organization,
- d) hyperemia, edema,
- e) secondary colliquation.

Select all correct answers

- 17. Outcomes of a heart attack:
- a) scar,
- b) cyst,
- c) resorption,
- d) softening,
- e) tutelage.

Select all correct answers

- 18. Fatal complication of myocardial infarction:
- a) organization,
- b) acute aneurysm,
- c) heart rupture,
- d) chronic aneurysm,
- e) cardiogenic shock.

Select all correct answers

- 19. To identify changes in tissues caused by acute ischemia, use:
- a) the Perls reaction,
- b) picrofuchsin,
- c) tetrazolium salts,
- d) SHGK (RA) -reaction.

Select all correct answers

- 20. Low resistance to ischemia of the brain and heart is due.
- a) features of the structure of blood vessels,
- b) high metabolic rate,
- c) limited reserves of blood supply,
- d) the aerobic nature of metabolism,
- e) the anaerobic nature of metabolism.

Choose one correct answer

21. The reason for the development of a lung infarction is:

- a) pulmonary vasospasm,
- b) arterial hypertension,
- c) chronic venous plethora,
- d) pulmonary hypertension,
- f) thrombosis of small branches of the pulmonary artery.

Select all correct answers

- 22. The main mechanisms of hemorrhagic complications in disseminated intravascular coagulation:
- a) alteration of the organ parenchyma,
- b) massive flow of procoagulants into the bloodstream,
- c) activation of platelets,
- d) increased fibrinolysis,
- e) consumption coagulopathy.

Select all correct answers

- 23. The development of DIC syndrome is determined by:
- a) increased production of thromboplastins,
- b) soluble tissue factors in the bloodstream,
- c) insufficiency of coagulation factors,
- d) damage to the endothelium,
- e) hemorrhagic diathesis.

Select all correct answers

- 24. DIC syndrome most often occurs when:
- a) embolism by amniotic fluid,
- b) placental abruption,
- c) sepsis,
- d) shock,
- e) venous congestion of internal organs.

Select all correct answers

- 25. A 54-year-old man with disintegrating pancreatic cancer developed a massive cerebral hemorrhage, which was the cause of death. The autopsy revealed signs of disseminated intravascular coagulation and hemorrhagic diathesis. The mechanism of development of disseminated intravascular coagulation and bleeding.
- a) the presence of vitamin K antagonists,
- b) thromboplastin production,
- c) production of tissue factors,
- d) decreased platelet adhesion,
- e) consumption coagulopathy.

List of recommended literature:

Basic literature:

1. "Basic pathology" Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

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- 7. "Muir's Textbook of Pathology" Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14th).
 - 8. "Pathology" Eds. Rubin, J.L. Farber Philadelphia: Lippincott Raven Publ, 1998 (3th).
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- 10. "Robbins Pathologic Basic of Disease" Eds. R.S.Cotran, V.Kumar, T.Collins Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
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- 12. "Color Atlas of Anatomical Pathology" Cooke R.A., Steward B. Edinburgh: Churchill Livingstone, 1995 (10th).
- 13. "General Pathology" Walter J.B., Talbot I.C. Edinburgh: Churchill Livingstone, 1996 (7th).
 - 14. "Concise Pathology" Parakrama Chandrasoma, Glive R. Taylor.
- 15. "Pathology" Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
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 - 17. "Robbins pathologic basis of diseases" Cotran R., Kumar V., Collins T.
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 - 19. "Anderson's Pathology" Damjanov I., Linder J. St. Louis: Mosby Inc., 1995 (10th).

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