

VOLGOGRAD STATE
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**Blood circulation disorders. Venous congestion. Arterial hyperemia. Bleedings.
Hemorrhages.**

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1. Purpose: to study various types of circulatory disorders by macroscopic and microscopic pictures, as well as using electron microscopy:

arterial and venous congestion (congestion), bleeding, hemorrhage.

2. Requirements for the level of the student for mastering the discipline - pathological anatomy.

The student should know:

1. The concept and types of arterial plethora (hyperemia), pathogenesis, morphogenesis.

2. The concept and types of venous plethora (stagnation), pathogenesis, morphogenesis.

H. Concept, classification, causes of bleeding, degree of blood loss. Outcomes of bleeding.

Arterial and venous congestion

Arterial plethora (hyperemia) - an increase in the volume of circulating blood in the arterial system of the organ. Depending on the causes and mechanisms of development, it can be physiological, corresponding to organ hyperfunction, for example, increased secretion of glands, etc., and pathological: inflammatory, postischemic (after local ischemia), decompression (after a sharp change in barometric pressure). With arterial hyperemia, the supply of oxygen to tissues increases.

In case of inflammation and allergy, arterial hyperemia promotes increased entry of leukocytes into the tissue, provides a high level of delivery of antibodies and other effectors of the immune system. It is essential for the removal of decay products and stimulation of regenerative processes. With the strengthening of endocrine functions, arterial hyperemia causes an increased influx of hormones. In medical practice, locally active substances are widely used, which enhance arterial hyperemia (heating pads, mustard plasters, banks, etc.)

However, pathological arterial hyperemia due to excessive expansion of blood vessels and increased pressure can lead to rupture of the vessel and hemorrhage. It can cause the spread of infection, damage to cell membranes, and excessive release of hormones.

Macroscopically hyperemic tissues have a red color, a higher temperature. Microscopically, the vessels (mainly of the microvasculature) are sharply dilated and full-blooded.

Venous congestion (congestion) can be acute or chronic. In acute venous stasis, local tissue edema is observed in accordance with the vein involved in the pathological process, for example, edema of the lower limb with inflammation of the walls in v. femoralis or face with inflammation in v. angularis, however much more often venous stasis is of a systemic nature and occurs with heart failure, diseases of the lungs and liver.

Chronic venous plethora is characterized by chronic hypoxia of an organ, tissue, as a result of which three stereotypical processes arise in various organs. The first is characterized by an increase in vascular permeability, displacement of erythrocytes from crowded veins (diapedesis), their destruction (extravascular hemolysis) and capture by macrophages. In the latter, an iron-containing brown pigment, hemosiderin, is formed, which is detected using the Perls reaction (based on the reaction of the formation of Prussian blue in the presence of iron). The second process, occurring simultaneously, is the activation of fibrillogenesis and the appearance of a newly formed connective tissue, leading to the compaction (induration) of the organ. sclerosis. The third process is characterized by the development of a capillary-parenchymal block caused by thickening of the basement membranes of the endothelium and epithelium due to the activation of fibroblasts, smooth muscle cells and lipocytes.

The morphological picture of acute venous stasis in the pulmonary circulation is characterized by an increase in vascular permeability and the development of pulmonary edema (cardiac asthma), and in chronic venous plethora by the appearance of brown induration of the lungs. The lungs become dense due to the growth in the interalveolar septa and around the vessels of the connective tissue, acquire a brown color due to the accumulation of hemosiderin.

The reasons leading to venous stasis in the pulmonary circulation are as follows:

- failure (decreased contractility) of the left ventricle of the heart;
- defect of the mitral and aortic valves with a predominance of narrowing (stenosis) at the atrioventricular opening or deformation of the leaflets (insufficiency) of the valve;
- compression of the pulmonary veins by a mediastinal tumor.

With venous congestion in the systemic circulation, due to increasing hypoxia, plasmorrhage increases, edema of the subcutaneous fatty tissue (anasarca) occurs, as well as accumulation of fluid in the serous

cavities: in the pleural cavity (hydrothorax), in the cavity of the heart bag (hydropericardium), in the abdominal cavity (ascites). In this case, the liver, kidneys, spleen are enlarged and dense. The edges of the liver become rounded, and the incision surface becomes variegated: there is an alternation of areas of yellow with red ("nutmeg liver"). Yellow foci are areas of fatty degeneration of hepatocytes against the background of a sharp plethora of the centers of the hepatic lobules (red foci).

The progression of venous stasis in the liver leads to the death of hepatocytes and the proliferation of connective tissue in the area of hemorrhage. The mechanism of sclerosis is associated with the proliferation of lipocyte sinusoid cells (Ito cells) and adventitia fibroblasts of the central and collecting veins. In the end, congestive fibrosis (sclerosis) of the liver develops, also called muscat, or cardiac, cirrhosis.

In the spleen and kidneys with chronic venous stasis, cyanotic induration develops. The organs are slightly increased in size, dense, bluish-claret color.

The most common cause of venous congestion in the systemic circulation is the previous congestion in the pulmonary circulation. In addition, diffuse sclerotic changes in the lungs that occur in some forms of tuberculosis and defects of the right heart may be of importance.

Venous plethora in the portal vein system (portal hypertension) is accompanied by the development of ascites, an increase in spleen weight (up to 500 g or more), varicose expansion of extrahepatic portocaval anastomoses. In an enlarged spleen, sharply dilated sinusoids, periarterial hemorrhages are microscopically found, followed by the development of fibrous nodules in their place (Gandhi-Gamna nodules). The causes of portal hypertension can be a diffuse sclerotic process (cirrhosis of the liver), Budd-Chiari syndrome with blockage or compression of the hepatic veins, liver damage by schistosomes (schistosomiasis), tumor compression of the portal vein mouth.

Bleeding

Bleeding, hemorrhage is the release of blood from the blood vessels or the heart cavity, in which blood can accumulate in the tissues (hemorrhage).

The reasons for bleeding are as follows.

1. Rupture of the vessel wall during injury (trauma, surgical operations) or with the development of pathological processes in it (atherosclerosis with the formation of aneurysms, hypertension with fibrinoid necrosis of the arteriole wall).
2. Corrosion of the vessel wall during exacerbation of peptic ulcer disease, tissue necrosis in tuberculosis, inflammation (especially purulent), tumor growth and its decay.
3. Increase in the permeability of the vessel wall, accompanied by diapedesis of erythrocytes (dia-through, redao - jumping), ie the passage of erythrocytes in the places of contact of endothelial cells.

Important factors, especially in the development of diapedetic bleeding, are hereditary blood disorders such as hemophilia or hemorrhagic diathesis, when spontaneous bleeding occurs in response to minor damage. This condition may be due to quantitative or qualitative changes in platelets, insufficiency of one or more coagulation factors, pathological fragility or increased permeability of the vascular wall.

In addition, with DIC syndrome, a pronounced hemorrhagic syndrome is observed, which is a consequence of coagulopathy of consumption, since in DIC syndrome, thrombin synthesis is inhibited, platelet aggregation, fibrin polymerization, proteolysis of factors Y and YIII.

The principles underlying the classification of bleeding are as follows.

1. Depending on the place where the blood is poured, external bleeding is distinguished, the result of which is blood loss and the development of anemia or hypovolemic shock, and internal, ending in hemorrhage.

There are the following types of hemorrhages.

Hematoma is an accumulation of coagulated blood in tissues with a violation of their integrity and the formation of a cavity. The sizes of the hematoma are different, they depend on the caliber of the damaged vessel, the level of blood pressure, density

surrounding tissue. The hematoma in the retroperitoneal tissue can contain up to 2 liters of blood, in the brain tissue it is much less. Along the periphery of the hematoma, where blood coagulation occurs, there is an organization of the convolution of an enclosed or cystic hematoma. A hematoma of the brain can break through into its ventricles or into the subarachnoid space. Hemorrhage in various cavities and the accumulation of blood in them are called hemothorax (chest cavity), hemopericardium (pericardial cavity), hemoperitoneum (abdominal cavity), hemarthrosis (joint cavity).

Hemorrhagic impregnation of hemorrhage while maintaining the integrity of tissue elements. It can be in the form of an accumulation of erythrocytes around the vessel, the so-called punctate hemorrhages of petechiae (from the Italian pechelia speck) and larger ecchymosis (from the Greek sigutos juice). Severe small punctate hemorrhages are called purpura (purpura cutis, segebry).

2. Distinguish between arterial, venous and capillary (parenchymal) bleeding. Arterial bleeding is most often acute, sometimes instantaneous, if the aorta, carotid artery ruptures. Bleeding can be chronic, then it continues for a long time or occurs periodically (hemorrhagic diathesis).

Taking into account the degree of blood loss, small (up to 10% 7-10 ml / kg), moderate (up to 25% - 15-20 ml / kg), massive (30-45% 20-30 ml / kg) and fatal (50 - 60% 40-60 ml / kg) blood loss.

The outcomes of bleeding are different.

1. The outflowing blood causes tissue compression, disrupts the delivery of substrates, which leads to cell death and dysfunction of the organ. For example, compression of the heart and large vessels during rupture of the heart and bleeding into the pericardial cavity (hemopericardium, pericardial tamponade) leads to cardiac arrest. Fresh hemorrhage consists of normal blood. The old hemorrhage is hemolyzed blood, partially absorbed.

2. Loss of blood leads to hypovolemia, i.e. a decrease in the volume of circulating fluid and anemia. A decrease in venous return of blood causes a drop in shock return of blood and a subsequent decrease in blood pressure, as a result of which the perfusion of tissues with blood decreases and shock develops.

H. A decrease in the number of erythrocytes and their dilution due to the compensatory release of fluid from the tissues into the vessels causes anemia and subsequent hemic hypoxia, leading to alteration and dysfunction of organs, primarily the brain, kidneys, liver and myocardium.

4. Cessation of bleeding as a result of increased blood clotting and thrombus formation at the site of vessel damage and normalization of the circulating fluid volume.

5. Resorption of blood with the formation of a cyst containing a brown liquid

- "rusty" cyst. The brown color is due to the accumulation of hemosiderin contained in the cytoplasm of macrophages in the cyst wall or outside the cells.

6. The organization of a hematoma and the formation of a connective fabrics.

7. Accession of infection and suppuration of hemorrhage.

3. Lesson plan.

1. **Macropreparation** To study chronic venous plethora of the kidney and spleen by the macroscopic picture. Describe the macro-preparations 'Cyanotic induration of the kidneys' and "Cyanotic induration of the spleen". Pay attention to the size, consistency and color of the organs.

2. **Micropreparation** To study chronic venous plethora of the skin on a microscopic picture. Describe the micropreparation "Chronic venous plethora of the skin" (staining with hematoxylin and eosin). Pay attention to the condition of the veins, capillaries and lymphatic vessels, as well as the connective tissue of the dermis, epidermis.

3. **Macropreparation** To study chronic venous plethora of the liver according to the macroscopic picture. Describe the macropreparation "Nutmeg liver". Pay attention to the size, consistency, color of the liver in the section.

4. **Micropreparation** To study chronic venous plethora of the liver by a microscopic picture. Describe the micropreparation "Chronic venous plethora of the liver (nutmeg liver)" (staining with hematoxylin and eosin, erythrosine). Pay attention to the blood filling of the central vein and sinusoids of the centers of the lobules, the state of hepatocytes in these parts of the lobules, as well as the blood filling of the sinusoids and the state of hepatocytes of the peripheral parts of the lobules.

5. **Electronogram** To study the mechanism of sclerosis in the development of muscat liver fibrosis using electron microscopy. Describe the electronogram 'Muscat liver fibrosis'. Pay attention to the localization of collagen fibers, the state of fibroblasts and stellate reticuloendotheliocytes (Kupffer cells).

6. **Macropreparation** To study chronic venous congestion of the lungs on the basis of the macroscopic picture. Describe the macropreparation "Brown induration of the lungs". Pay attention to the size, consistency and color of the lung tissue.

- 7. Micropreparation** To study chronic venous plethora of the lungs on a microscopic picture. Describe the micropreparation “Brown induration of the lungs” (staining with hematoxylin and eosin, Perls reaction). Pay attention to the localization of sideroblasts and siderophages, the color of the pigment in the Perls reaction, the thickness of the interalveolar septa.
- 8. Macropreparation** To study cerebral hemorrhage according to the macroscopic picture. Describe the cerebral hemorrhage macro-preparation. Pay attention to the location and form of hemorrhage, the color of coagulated blood, the state of the brain tissue in the area of hemorrhage.
- 9. Micropreparation** To study the hemorrhage in the brain according to the microscopic picture. Describe the micropreparation ‘Hemorrhage in the brain’ (staining with hematoxylin and eosin). Pay attention to the localization of erythrocytes in the brain tissue, note the state of erythrocytes and brain tissue in and around the hemorrhage zone, the state of the arteriole walls.
- 10. Macropreparation** To study anemia of the kidney on a macroscopic picture. Describe the macro-preparation “Kidney ischemia”. Pay attention to the size, consistency and color of the organ.

4. QUESTIONS

Select all correct answers

1. A 23-year-old woman had a mitral valve defect with a predominance of stenosis, which led to the development of venous stasis in the pulmonary and systemic circulation. Systemic manifestations of chronic venous stasis:
 - a) brown induration of the lung,
 - b) nutmeg liver,
 - c) anasarka,
 - d) fibrosing alveolitis,
 - e) chronic hepatitis.
2. Chronic venous congestion of the lungs is characterized by:
 - a) inflammation,
 - b) diffuse pneumosclerosis,
 - c) perivascular sclerosis,
 - d) diffuse hemosiderosis,
 - e) blood clots in the microvasculature.
- H. Portal hypertension is characterized by:
 - a) hypertrophy of the right ventricle of the heart,
 - b) splenomegaly,
 - c) ascites,
 - d) varicose expansion of the superior vena cava,
 - e) varicose expansion of extrahepatic portocaval anastomoses.
4. The variegated appearance of the liver in chronic venous stasis is caused by:
 - a) expansion and plethora of sinusoids,
 - b) proliferation of connective tissue,
 - c) fatty degeneration of hepatocytes,
 - d) regeneration of hepatocytes,
 - e) balloon dystrophy of hepatocytes.
5. The death of a 43-year-old man, suffering from chronic hepatitis B with an outcome in cirrhosis, occurred from bleeding from the veins of the esophagus. Causes of ruptured veins of the esophagus:
 - a) varicose veins of the round ligament of the liver,
 - b) varicose veins of the esophagus and cardia of the stomach,
 - c) pulmonary hypertension,
 - d) venous congestion in the portal vein system.
- b. Portal hypertension leads to:
 - a) the development of ascites,
 - b) cirrhosis of the liver,
 - c) kidney enlargement,
 - d) enlargement of the spleen,
 - e) intravital blood coagulation in the cavities of the heart.
7. Complications of massive blood loss:
 - a) hypovolemic shock,
 - b) a decrease in the amount of iron,
 - c) lowering blood pressure,
 - d) DIC syndrome,
 - e) increasing tissue hypoxia.
8. Complications of pilots during high-speed starts:
 - a) fat embolism,
 - b) air embolism,
 - c) gas embolism,
 - d) lung infarction,
 - e) obstruction of blood vessels by air bubbles.
9. Unfavorable outcomes of hematoma:
 - a) anemia,
 - b) hypovolemic shock,
 - c) organization,
 - d) suppuration,
 - e) petrification.
10. Choose the correct statements:
 - a) venous congestion - increased blood supply to an organ or tissue due to a decrease in blood outflow,
 - b) venous congestion - increased blood filling of an organ or tissue due to increased blood flow,
 - c) bleeding the release of blood from a blood vessel or heart cavity into tissue or into the environment,

d) hematoma - an accumulation of coagulated blood in tissues with a violation of their integrity,

e) petechiae are planar hemorrhages in the subcutaneous tissue.

Choose one correct answer

and. a 63-year-old woman has ascites on the background of venous congestion in the portal vein system; when fluid was evacuated from the abdominal cavity, collapse developed. The reason for the drop in blood pressure:

- a) turning off the main artery,
- b) fast evacuation of ascitic fluid,
- c) hypovolemic shock,
- d) a large loss of proteins and ions,
- e) varicose expansion of portocaval anastomoses.

12. A 22-year-old man with rheumatic mitral valve disease had heart disease cells in his sputum. It:

- a) plasma cells filled with immunoglobulins,
- b) macrophages filled with hemosiderin,
- c) macrophages filled with lipofuscin,

d) multinucleated giant cells,

e) leukocytes filled with pigment.

13. A 70-year-old man suffers from hypertension, 10 years ago suffered a cerebral hemorrhage. The brain determines:

- a) blood in the ventricles of the brain,
- b) hemosiderosis of the subcortical nuclei,
- c) a focus of gray softening,
- d) a cyst with rusty walls,
- e) blood clots in the brain tissue.

14. Precise hemorrhages are:

- a) purple,
- b) ecchymosis,
- c) petechiae,
- d) hematomas.

15. Purpura is:

- a) small punctate hemorrhages,
- b) large multiple hemorrhages.

16. Hemorrhagic diathesis is:

- a) progressive activation of coagulation,
- b) spontaneous bleeding in response to minor damage,
- c) multiple punctate hemorrhages,
- d) common petechiae and ecchymosis.

5. List of recommended literature:

Basic literature:

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

Additional literature:

1. "Pathology. Quick Review and MCQs" Harsh Mohan, 2004.
2. "Textbook of Pathology" Harsh Mohan, 2002.
3. "General and Systemic Pathology" Joseph Hunter, 2002.
4. "General and Systematic Pathology" Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2th).
5. "Histology for Pathologist" Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2th).
6. "Histopathology. A Color Atlas and Textbook" Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
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).
9. "Pathology Illustrated" Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).
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).

11. "Wheater's Basic Histopathology. A Color Atlas and Text" Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
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.
16. "Short lectures on pathology" Zagoroulko A., 2002
17. "Robbins pathologic basis of diseases" Cotran R., Kumar V., Collins T.
18. "General pathology" Dr. Fatma Hafez, 1979.
19. "Anderson's Pathology" Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

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