Materials
for students for practical classes in pathological anatomy
at the Department of Pathological Anatomy

II year Faculty of Dentistry

Topic 3 (part 1): "Disorders of blood circulation: congestion, bleeding, hemorrhage".

1. The purpose of the lesson. To study the causes, mechanisms of development, types, clinical and morphological manifestations, values and consequences of arterial and venous plethora, ischemia, bleeding and hemorrhage.

2. Requirements for the level of the student for mastering the discipline - pathological anatomy. The student should know:

1. Determination of arterial and venous plethora.

2. Etiology, classification and morphological manifestations of arterial and venous plethora.

3. Complications and outcomes of arterial and venous plethora.

4. Definition, etiology, pathogenesis, classification, morphological manifestations, complications and outcomes of bleeding.

5. Definition, types, morphological manifestations, complications and outcomes of hemorrhages.

Theoretical aspects.

Classification of Disorders of blood circulation (Hemodynamic disorders):

1) Disorders of of blood filling (arterial and venous hyperemia; ischemia);

2) Disorders of the permeability of the vascular wall (bleeding (hemorrhage); plasmorrhage);

3) Disorders in blood flow (i.e. rheological properties) and blood conditions (stasis, sludge phenomenon, thrombosis and embolism).

Shock is a special place among circulatory disorders.

Arterial hyperemia and venous congestion

Arterial plethora (hyperemia) is an increase in the volume of circulating blood in the arterial system of an organ.

Depending on the causes and mechanisms of development, it can be physiological, corresponding to organ hyperfunction, for example, increased glandular secretion, 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 allergies, 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 enhancement of endocrine functions, arterial hyperemia causes an increased influx of hormones. In medical practice, locally acting active substances are widely used that enhance arterial hyperemia (heating pads, mustard plasters, banks, etc.).

At the same time, pathological arterial hyperemia due to excessive dilation 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 the microvasculature) are sharply dilated and full-blooded.

Venous congestion (congestion) can be acute or chronic. In acute venous congestion, 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 v. femoralis or facial inflammation v. apgilagis. However, much more often venous stasis is of a systemic nature and occurs with heart failure, diseases of the lungs and liver.

Chronic venous congestion 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 overflowing veins (diapedesis), their destruction (extravascular hemolysis) and capture by macrophages. In the latter, an iron-containing brown pigment is formed - hemosiderin, which is revealed with the help of the Perls reaction (the basis is the reaction of the formation of Prussian blue in the presence of iron). The second process that occurs simultaneously is the activation of fibrillogenesis and the emergence of newly formed connective tissue, leading to the compaction (induration) of the organ - sclerosis. The third process is characterized by the development of a canillar-arenchumatous 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) of 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 the 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). At the same time, the liver, kidneys, spleen are enlarged and dense. The edges of the liver become rounded, and the surface of the cut is variegated: there is an alternation of areas of yellow and red ("nutmeg"). 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 sinusoidal cells - lipocytes (Ito cells) and fibroblasts of the adventitia of the central and collecting veins. In the end, congestive fibrosis (sclerosis) of the liver develops, also called nutmeg, 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 stasis in the pulmonary circulation is the previous stagnation 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 congestion in the portal vein system (portal hypertension) is accompanied by the development of ascites, an increase in the spleen mass (up to 500 g or more), varicose expansion of extrahepatic port-caval anastomoses. In the 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, damage to the liver by schistosomes (schistosomiasis), compression of the portal vein mouth by a tumor.

Bleeding

Bleeding, hemorrhage - the release of blood from the blood vessels or the cavity of the heart, 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, surgery) 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, pedao - jumping), ie, the passage of erythrocytes in the places of contact of endotheliocytes.

Important factors, especially in the development of diapedetic bleeding, are hereditary blood diseases, 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, there is a pronounced hemorrhagic syndrome, which is a consequence of consumption coagulopathy, since in DIC syndrome, thrombin synthesis is inhibited, platelet aggregation, fibrin polymerization, proteolysis of factors V and VIII.

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 consequence 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 - 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, and the density of the surrounding tissue. A hematoma in the retroperitoneal tissue can contain up to 2 liters of blood, in the brain tissue - much less. On the periphery of the hematoma, where blood coagulation occurs, an organization of convolution occurs - 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 is called hemothorax (chest cavity), hemopericardium (pericardial cavity), hemoperitoneum (abdominal cavity), hemarthrosis (joint cavity).

Hemorrhagic impregnation - 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 point hemorrhages - petechiae (from Italian retechia - speck) and larger - ecchymosis (from Greek chytos - juice). Pronounced small punctate hemorrhages are called pur-pura (rrriga cuti).

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 lasts a long time or occurs periodically (hemorrhagic diathesis).

3.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, ie, a decrease in the volume of circulating fluid and anemia. A decrease in venous blood return leads to a drop in shock blood return and a subsequent decrease in blood pressure, as a result of which tissue perfusion with blood decreases and shock develops.

3. 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 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 connective tissue in its place are possible.

7. Accession of infection and suppuration of hemorrhage.

Shock

Shock is a generalized acute hemodynamic disorder that occurs after a super-strong effect on the body and leads to a sharply increasing drop in blood supply to tissues.

There are 5 main types of shock: hypovolemic, cardiogenic, septic, neurogenic, anaphylactic.

Hypovolemic shock occurs when the volume of circulating blood decreases. One of the most common causes is acute massive blood loss. In addition, with extensive burns, when more than 20% of the skin surface is affected, plasma sweating from the damaged microvasculature is observed, and hypovolemic shock may develop. The same mechanism for the development of hypovolemic shock was observed in allergic skin lesions, severe repeated vomiting and profuse diarrhea.

Cardiogenic shock develops in response to an acute decrease in the volume of cardiac output during myocardial infarction, massive non-coronary necrosis in the myocardium against the background of severe pain.

Septic shock most often appears when gram-negative microbes that release endotoxin are spread in the body, often occurs in people with immunodeficiency (primary and secondary). An important pathogenetic role is played by the effect of endotoxins and massive damage to the endothelium, followed by activation of the internal blood coagulation system (Hageman factor XII), complement, tumor necrosis factor (TNF), endorphins, interleukins and other cytokines included in the group of endogenous mediators, which participate in the pathogenesis of shock.

Neurogenic shock occurs when poisoning with hypnotics, drugs, ganglion blockers.

Anaphylactic shock develops under the influence of allergens against the background of sensitization. IgG and IgE antibodies are important in pathogenesis, followed by the involvement of mast cells, basophils in the process and the release of mediators, histamine, anaphylatoxin (C3 and Cs components of complement), kinins, prostaglandins.

The main morphological changes in shock are associated with circulatory disorders.

1. Disorders of coagulation - coagulopathy. During the pathological examination, liquid blood is determined in the large vessels and cavities of the heart. The process is based on an extreme increase in the function of the blood anticoagulant system and coagulation of consumption (the use of all coagulation factors).

2. Development of DIC syndrome.

3. Sequestration of blood in the microvasculature. At the same time, there is an unevenness of the blood filling of the organs. The cavities of the heart contain almost no blood ("empty" heart), and large vessels and parenchymal organs are filled with liquid blood.

4. Bypass blood flow - the inclusion of bypass routes in the kidneys, liver, lungs. In the kidneys, it is especially pronounced in the form of anemization of the cortical and hyperemia of the medulla, especially in the area of the pyramids.

Circulatory disorders lead to pronounced circulatory toxic tissue damage. In the vessels of organs (especially in the kidneys, adrenal glands, pituitary gland, pancreas), stasis, sludge phenomenon, blood clots are detected. In addition, there is an increase in capillary permeability, against the background of coagulopathy, hemorrhagic syndrome develops. There are petechial and large focal hemorrhages in the organs.

Lungs - bronchiolospasm, atelectasis (collapse of lung tissue), loss of fibrin in the lumen of the alveoli and the formation of hyaline membranes, serous-hemorrhagic edema.

Kidney - tubular epithelium necrosis.

Liver - dystrophy with the formation of Kraevsky cells (the rapid disappearance of the glycogen from hepatocytes gives the cytoplasm a foamy appearance) with their subsequent necrosis.

Adrenal glands - loss of lipids by cells of the cortex, hemorrhages.

Gastrointestinal tract - hemorrhages, necrosis, leading to the formation of acute erosions and ulcers.

Brain - Edema and tissue swelling.

Myocardium - non-coronary necrosis.

In the late period of shock, the inhibition of the phagocytic activity of leukocytes, sharp shifts in the number and population composition of lymphocytes and the development of secondary immunosuppression are noted.

3. Lesson plan

Macropreparations

1. **Cyanotic induration of the kidneys** - pay attention to the size, consistency and color of the organs.

2. **Cyanotic induration of the spleen** - pay attention to the size, consistency and color of organs.

3. **Nutmeg liver** - pay attention to the size, texture, color of the liver in the cut.

4. **Brown induration of the lungs** - pay attention to the size, texture and color of the lung tissue.

5. **Cerebral hemorrhage** - 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.

Micropreparations

1. **Chronic venous congestion 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 and epidermis.

2. **Chronic venous congestions of the liver** (nut meg liver) (staining with hematoxylin and eosin, erythrosin) - 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.

3. **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.

4. **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.

Electronograms

1. **Nut meg liver fibrosis** - pay attention to the localization of collagen fibers, the state of fibroblasts and stellate reticuloendotheliocytes (Kupfer cells).

Situation cases

Situation case 1

Patient L., 44 years old, from childhood suffered from rheumatism with mitral valve lesions. She was admitted to the cardiology department with signs of decompensation of mitral stenosis: acrocyanosis, shortness of breath, orthopnea, edema, abdominal enlargement due to the accumulation of edematous fluid. When coughing, sputum with a brown tint was released. Death came from chronic cardiovascular failure.

Questions to the situation case 1

1. What circulatory disorder was found on autopsy?

2. Characterize the macroscopic changes in the lungs. Explain

morphogenesis of lung changes.

3. Why did the patient's sputum have a rusty tint?

4. Name the changes in serous cavities characteristic of chronic cardiovascular insufficiency. Explain the morphogenesis of these changes.

5. Characterize the macroscopic changes in the liver in chronic cardiovascular failure. Explain the morphogenesis of liver changes.

6. Explain the occurrence of shortness of breath in the patient in the terminal period.

Situation case 2

Patient T., 23 years old, was admitted in serious condition to the neurological department. Despite intensive therapy and an attempt at neurosurgical treatment, death occurred. An autopsy revealed an extensive accumulation of blood in the subcortical nuclei of the right hemisphere of the brain.

Questions to the situation case 2

1. Name the type of circulatory disorders in the brain.

2. What is the mechanism of blood exit from the bloodstream in this observation?

3. Give the macro- and microscopic characteristics of the morphological changes in the brain found at autopsy.

4. What changes would the lesion focus undergo in case of a favorable outcome?

5. Name other mechanisms of blood exit from the bloodstream.

6. Give morphological characteristics to other forms of blood accumulation in tissue.

Situation case 3

Patient I., 50 years old, suffering from atherosclerosis of the left renal artery, developed dizziness, weakness in the left extremities. The patient died in an ambulance. An autopsy shows an accumulation of blood in the subcortical nuclei of the right hemisphere of the brain.

Questions for situational task number 3

1. What are the macroscopic features of the left kidney?

2. Name the reason and describe the energy metabolism of the cells of the left kidney.

3. Name the mechanisms of ischemia.

4. What is the mechanism of blood exit from the bloodstream in this observation?

5. Name the type of circulatory disorders in the brain.

Literature to the Topic 3

Basic literature:

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

Additional literature:

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