PATHOLOGICAL PHYSIOLOGY OF POSTHEMORRHAGIC ANEMIA

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Annotation. Blood loss is a complex of pathological disorders and compensatory reactions directed against a decrease in the volume of circulating blood and hypoxia caused by a decrease in the respiratory function of the blood. Posthemorrhagic shock is a shock that occurs as a result of abundant acute blood loss. A decrease in the volume of circulating blood, a drop in blood pressure, a violation of microcirculation, disorders of the blood supply to vital organs (brain, heart, kidneys), the development of hypoxia, acidosis and intoxication, "vicious circles" in the pathogenesis leads to death. Post-hemorrhagic anemia-develops as a result of blood loss.

Keywords: blood vessels, posthemorrhagic anemia, hemoglobin, hematocrit, blood pressure.

Introduction: Acute post-hemorrhagic anemia occurs after rapid massive blood loss when blood vessels are injured or damaged by a pathological process.

Chronic post-hemorrhagic anemia develops as a result of repeated blood loss caused by damage to blood vessels in a number of diseases (dysmenorrhea, gastric ulcer, hemorrhoids, etc.) and a violation of vascular-platelet and coagulation hemostasis (hemorrhagic diathesis). The loss of iron with frequent bleeding gives this anemia an iron deficiency character.

Etiology:

1) violation of the integrity of blood vessels when injured or affected by a pathological process (atherosclerosis, tumor, tuberculosis);

2) increased permeability of the vascular wall (acute radiation sickness);

3) decrease in blood clotting (hemorrhagic diathesis).

A serious danger to human life is the loss of 50% of the volume of circulating blood, fatal is the loss of blood over 60%.

Pathogenesis:

I. Initial stage. A decrease in the volume of circulating blood, a decrease in blood pressure, hypoxia.

II. Compensatory stage. Activation of protective and compensatory reactions.

III. Terminal stage. The increase of pathological changes in the body up to a fatal outcome.

Protective and compensatory reactions in case of blood loss:

I. Centralization of blood circulation:

1) spasm of arterioles of the skin, muscles, organs of the digestive system;

2) opening of arteriovenous anastomoses of these organs and tissues as a result of spasm of precapillary sphincters;

3) venoconstriction (contraction of the smooth muscles of the veins), an increase in blood flow to the heart. These changes are based on the following mechanisms:

a) lowering of blood pressure - + excitation of baroreceptors - + activation of the sympathoadrenal system -+ action of catecholamines on a-adrenoreceptors of smooth muscles of arteries, arterioles, precapillary sphincters and veins;

b) a decrease in the volume of circulating blood and blood pressure - + excitation of volumo-and baroreceptors - + activation of neurosecretory cells of the hypothalamus that produce vasopressin -+ action on V1-receptors of vascular smooth muscles with subsequent vasoconstriction;

c) decrease in the volume of circulating blood and activation of the sympathoadrenal system - + release of renin by the cells of the juxtaglomerular apparatus of the kidneys -+ activation of the renin-angiotensin system with the formation of angiotensin II -+ spasm of the smooth muscles of blood vessels.

II. Increase in the volume of circulating blood:

1. The transfer of tissue fluid into the blood vessels. The Starling mechanism is a decrease in the volume of circulating blood, a decrease in hydrostatic pressure in the capillaries, a decrease in water filtration in the arterial part of the capillaries and an increase in fluid reabsorption in the venous.

2. Increased reabsorption of water and sodium ions in the kidneys. Prevents the loss of fluid in the urine:

a) the effect of vasopressin (antidiuretic hormone) on the V2-receptors of the epithelium of the distal convoluted tubules and collecting tubes of the kidneys;

b) activation of the renin-angiotensin system, the release of aldosterone, which increases the reabsorption of sodium in the distal convoluted tubules of nephrons;

c) activation of the sympathoadrenal system, redistribution of blood flow in the nephron.

3. The output of blood from the depot into the bloodstream. Activation of the sympathoadrenal system and the effect of catecholamines on the vessels of the liver, spleen, subcutaneous fat.

III. Restoration of blood composition-from several days to 1-2 weeks after bleeding. Circulatory (decrease in the volume of circulating blood) and hemic (anemic) hypoxia of the kidneys, an increase in the number of renal erythropoietins, an effect on erythropoietin-sensitive cells of class III of the red bone marrow, an increase in young regenerative forms of red blood cells in peripheral blood.

Pathological changes in blood loss:

1. A decrease in the volume of circulating blood, a drop in blood pressure, shock.
2. Acute posthemorrhagic anemia.
3. Hypoxia. Initially, circulatory, and then hemic (anemic).
4. Non-gas acidosis. It is caused by hypoxia and the intake of lactic acid into the blood.
5. Violation of the excretory function of the kidneys, acute renal failure, oligo - and anuria, intoxication (azotemia), death.

Acute post-hemorrhagic anemia - there are three periods, each of which is characterized by a certain picture of peripheral blood:

I. The first few hours after acute blood loss. During this period of time, the total volume of blood decreases, as well as the total number of red blood cells in the body.

II. A period of time from several hours to several days after acute blood loss. As a result of the transfer of fluid from the interstitial space to the blood vessels, blood dilution (hemodilution) occurs. The number of red blood cells and hemoglobin decreases, the hematocrit decreases. The color indicator remains unchanged (normochromic anemia). Qualitative changes in red blood cells in the blood smear have not yet been detected.

III. The time period is from several days to 1-2 weeks after acute blood loss. The appearance of a large number of regenerative forms of red blood cells, which is associated with increased erythropoiesis in the red bone marrow. Since young immature red blood cells contain less hemoglobin compared to mature cells, the color index decreases and anemia becomes hypochromic.

Chronic posthemorrhagic anemia - due to loss of iron with frequent bleeding, signs of iron deficiency anemia develop: the concentration of hemoglobin and color index decreases, degenerative forms of red blood cells appear in the blood smear (micro-and poikilocytosis, hypochromia). The number of red blood cells and hematocrit may remain unchanged.

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