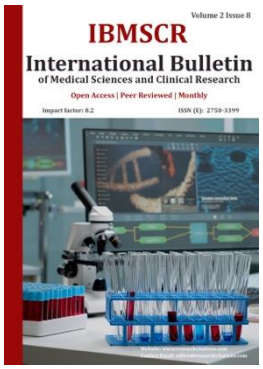

PATHOGENETIC MECHANISMS OF MICROCIRCULATION DISORDERS

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https://doi.org/10.5281/zenodo.10469496

Abstract: In the pathogenesis of many diseases requiring intensive treatment, hypoxia, ischemia and acidosis occur due to deterioration of blood rheological properties, microcirculatory disorders and capillary trophic insufficiency. Disturbances in the hemorheological system constitute a common mechanism in the pathogenesis of critical conditions. Therefore, improvement of blood rheological properties, i.e. optimization, is an important direction of intensive therapy

Key words: hemorheology, pathogenesis, erythrocyte aggregation, microcirculation, microthrombosis

In the pathogenesis of most diseases requiring intensive treatment, hypoxia, ischemia and acidosis occur due to deterioration of blood rheological properties, microcirculatory disorders and capillary trophic deficiency. Deterioration of blood rheological properties: increase in blood viscosity, increase in erythrocyte aggregation, change in erythrocyte deformation properties, increase in hematocrit index, blood separation due to conglomerates formed in the blood, increase in erythrocyte sedimentation rate due to changes in the ratio of plasma proteins albumin\globulins, albumin\fibrinogen, blood flow characterized by microthrombosis caused by slowing down and activation of coagulation factors, microcirculation disorders in the form of prestasis, stasis, and capillary trophic insufficiency.

Since the erythrocyte surface area has 40% excess surface area compared to the volume of 140 μm2, the membrane can change its shape without stretching, that is, it is deformed. The viscosity of the erythrocyte cytoplasm is 6.4 mPa− , and the time to return from deformation to its original shape is 0.12 seconds. An increase in blood viscosity creates additional resistance to blood flow, and therefore an additional afterload on the heart, resulting in increased microcirculatory disorders and tissue hypoxia. In a hemodynamic crisis, the viscosity of the blood increases due to the slowing of the blood flow, and a diseased population occurs. Stoppage of blood flow leads to stasis and blood shunting in the microcirculatory vein. Hemorheological stroke was also observed among 49-50-year-old patients. In metabolic syndrome, erythrocyte deformation characteristics change due to changes in surface tension and charge. As a result, irreversible aggregation of erythrocytes occurs, as a result, the function of oxygen delivery and the deterioration of blood rheology are observed.

Disturbances in the hemorheological system constitute a common mechanism in the pathogenesis of critical conditions. Therefore, improvement of blood rheological properties, i.e. optimization, is an important direction of intensive therapy.

Decreased blood viscosity accelerates blood flow, increases oxygen delivery to tissues, and improves heart function. With the help of rheologically active substances, the development of thrombosis, ischemia and infectious complications of the main diseases are prevented.

Hemorheology is the study of the physicochemical properties of blood flow. Blood fluidity is related to its viscosity. From a biomechanical point of view, macro and microrheological properties of blood differ.

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When comparing the results of hemorheological changes in a group of meteosensitive patients, changes were found in all microrheological indicators of erythrocytes (erythrocyte deformation, aggregation, geometry, membrane tension), and the fact that hematocrit is 2-3 times higher than the norm is due to erythrocyte deformability (50.1±2.1%; $p<0.01$), changes in the geometry of erythrocytes (decrease in the number of discocytes to 82.1±1.5 due to an increase in the amount of echinocytes; $p<0.05$); spontaneous aggregation rate of erythrocytes increased by 30-40% ($p<0.05$) and hematocrit indicators increased by 30-35% ($p<0.01$) from normal.

The viscosity of a normal liquid is constant (8 s Poise). The viscosity of blood varies depending on the conditions of blood flow (from 3 to 30 s Poise). The property of "internal" resistance to blood moving forces is called viscosity, and the unit of measurement is poise. Adhesion is based on inertial and drag forces. When the hematocrit index is equal to 0 (zero), blood viscosity approaches plasma viscosity. To measure and mathematically interpret blood viscosity, we need the concepts of motion resistance and motion velocity. The first concept is that motion resistance is equal to the ratio of friction between layers in laminar flow to its surface-F\S which is expressed in dynes\cm2 or pascals. The second indicator is the speed of movement, which is the gradient of the velocity of layers in laminar flow, delta V\L, which is measured in s-1. According to Newton's equation, the resistance to motion is directly proportional to the speed of motion. This means that the greater the speed difference between the layers, the higher their friction force, and on the contrary, the equalization of the movement speed between the liquid layers reduces the mechanical resistance along the line of the water separator. In this case, the viscosity is as a proportionality factor. Normal or Newtonian fluids (such as water) have a constant viscosity in any desired motion (slow, fast). For these fluids, there is a linear relationship between drag and velocity. Unlike normal fluids, blood viscosity is related to changes in blood flow velocity. For example, blood viscosity in the aorta and main arteries approaches 4-5 relative units.

In the venous part of the microcirculation, despite the low movement resistance, the level of viscosity increases 6-8 times compared to the level of blood viscosity in the artery. (up to 30-40 relative units). At extremely slow blood flow rates, which are physiologically incompatible, blood viscosity can increase up to 1000 times.

Thus, the relationship between movement resistance and movement speed for blood is not linear, but exponential in nature. Therefore, the rheology of blood is not Newtonian, or in other words, blood does not belong to Newtonian fluids. The reason for this is related to the rough dispersion of blood.

From a physicochemical point of view, blood is suspended in a liquid medium (water) with solid insoluble (form elements and high molecular substances) phases. Disperse phase

particles are sized to resist Brownian motion. A common feature for such systems is its inequality.

The components of the dispersed phase always tend to leave the dispersed medium and settle in the form of cell aggregates. The main and rheologically significant aggregates formed by blood cells are mainly erythrocyte aggregates. Erythrocyte aggregates have a typical "column of coins" shape.

Factors that ensure blood viscosity include: aggregation and deformation of erythrocytes; an increase in the hematocrit indicator is accompanied by an increase in blood viscosity; fibrinogen concentration, increasing the amount of soluble fibrin monomer complexes and fibrin/fibrinogen degradation products increases blood viscosity; a decrease in the ratio of albumin/fibrinogen and albumin/globulin is accompanied by an increase in blood viscosity; an increase in the amount of circulating immune complexes causes an increase in blood viscosity;

Increased blood flow reduces the size of the aggregates. Aggregation of erythrocytes necessarily requires fibrinogen or other high molecular protein or polysaccharide. They form a bridge between erythrocytes. In "coin columns", erythrocytes are parallel to each other at the same distance. For example: in the case of fibrinogen bridges, the distance between them is 25 nm. The electrostatic repulsive force caused by the same charge on the erythrocyte membrane does not allow this distance to approach. Bridges created by the fibrinogen molecule do not allow the distance to increase. Aggregation of erythrocytes is a reversible process, and erythrocytes in the aggregate separate when a certain movement speed occurs in the blood stream and can deform, that is, they can change their shape due to their plasticity.

In obvious disorders, microcirculation disorders occur due to pathological aggregation of sludge-erythrocytes. The hydrodynamic resistance of the formed erythrocyte aggregates increases. Aggregation of erythrocytes mainly depends on the following factors: ionic composition of the environment: when the total osmotic pressure in the plasma increases, erythrocytes do not swell and lose the property of aggregation; surface active substances that change the charge on the surface of the membrane, and their effects can be different; concentration of fibrinogen and immunoglobulins; contact with a foreign surface is usually accompanied by disruption of normal erythrocyte aggregation.

Erythrocyte deformation depends on the following main factors: plasma osmotic pressure; intracellular calcium magnesium ratio and ATP concentration; strength and duration of external effects (mechanical and chemical) that disrupt the lipid composition or spectrin structure of the erythrocyte membrane ; state of the erythrocyte cytoskeleton, which includes spectrin; intracellular adhesion in erythrocyte.

In short, the violation of the rheological properties of blood is directly related to the changes in the aggregation and deformation properties of erythrocytes. As a result of microcirculation and capillary trophic disorders, in many cases, it creates conditions for the formation of blood clots in patients. The main link of the vicious circle in the pathogenetic mechanism of microcirculatory disorders, which leads to the severity of hemorheological disorders, impaired tissue oxygenation, and disruption of the supply of nutrients and drugs, is the increase in blood viscosity and sludging, i.e., the use of drugs that improve blood rheology in the correction of hemorheological disorders can prevent many irreversible complications. helps to get.

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