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THE IMPORTANCE OF ENDOTHELIAL DYSFUNCTION IN THE DEVELOPMENT OF DISORDERS OF THE HEMOSTASIS SYSTEM IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Abstract

The literature review presents current data on the physiology and pathology of the endothelium. It has been noted that endothelial dysfunction is the main pathogenetic mechanism in the development of many diseases, and a more in-depth study of the morphofunctional structure of the vascular endothelium in various pathologies can contribute to early diagnosis and prevention.

Keywords: endothelium, endothelial dysfunction, endothelial factors, method.

INTRODUCTION

Endothelium is a single-layer layer of flat cells of mesenchymal origin, lining the inner surface of blood and lymphatic vessels and cardiac cavities. Its cells are connected by powerful tight junctions, the formation of which is induced by contact with astrocytes. The endothelium is the inner lining of blood vessels that separates the blood flow from the deeper layers of the vascular wall. This is a continuous monolayer of endothelial cells that form tissue, the mass of which in humans is 1.5 - 2.0 kg. The endothelium continuously produces a huge amount of important biologically active substances, thus being a giant paracrine organ distributed over the entire area of the human body.

MATERIALS AND METHODS

There are several phenotypes of endothelial cell structure: fenestrated, sinusoidal and continuous. The fenestrated type is observed in the vessels of the kidneys, intestines and endocrine glands; sinusoidal - in the vessels of the liver, spleen and bone marrow; continuous type – in the vessels of the brain [1,2].

In recent years, the understanding of endothelial function has expanded significantly. The participation of the endothelium in the regulation of vascular tone, vascular permeability, leukocyte adhesion, neoangiogenesis and other processes is

widely studied [3]. Impaired functional activity or endothelial dysfunction (ED) is considered an important pathogenetic link in diseases such as hypertension, atherosclerosis, diabetes mellitus and many others.

RESULTS AND DISCUSSION

The participation of the endothelium in the regulation of vascular tone is associated with the production of various vasoconstrictors and vasodilators [4]. Vasoactive substances that affect the endothelium are also produced by platelets, leukocytes, mast cells or are activated in the blood plasma. In addition, the endothelium is affected by certain hormones and neuropeptides. Some substances are synthesized in the endothelium itself and act either systemically or autocrine and paracrine. The effect of all these substances on endothelial cells is associated with the presence of specific receptors on the latter, stimulation of which causes the formation of secondary mediators, vasoconstrictors or vasodilators, which directly affect vascular smooth muscle cells. The formation of vasoactive substances in the endothelium is regulated primarily by two main mechanisms: the action of biologically active substances and shear stress.

Shear stress is the second factor that affects the formation of vasoactive substances in the endothelium, as well as thrombogenic and atrombogenic factors, and adhesive molecules. A significant change in shear stress occurs predominantly in arterial vessels when the blood flow velocity changes, and its increase leads to increased formation of all major vasodilators in the endothelium. Thus, vascular tone in each part of the vascular bed depends on the combination of vasoactive substances formed in the endothelium and circulating in the blood, the location of the vessels and the type of vessels.

Tissue and urokinase plasminogen activators and their inhibitors are formed in the endothelium. Tissue plasminogen activator is secreted constantly, but its release from endothelial cells can sharply increase in certain situations (physical exercise, catecholaminemia). The plasminogen inhibitor is also constantly produced and secreted by endothelial cells, and is found in the cell in large excess relative to the activator (in inflammation, coronary heart disease, idiopathic deep vein thrombosis and many other pathological processes, their content in the blood is increased).

The interaction of leukocytes with the endothelium occurs through special adhesive molecules, which are presented both on endotheliocytes and on leukocytes. The main regulator of the process of leukocyte adhesion is the endothelium itself. Under

normal conditions, a marginal pool of leukocytes is formed on the endothelium in the venous vessels of the gastrointestinal tract and lungs.

Leukocyte adhesion occurs in two stages: rolling and tight adhesion. The process of leukocyte adhesion ends with their migration outside the vessels, which is also ensured by adhesion molecules. The expression of adhesion molecules can be suppressed by a number of factors that are formed in the endothelium itself. Increased endothelial adhesiveness is of great importance in the pathogenesis of endothelial dysfunction during inflammation, atherosclerosis, septic shock and other pathological processes. The criterion for activating the adhesive properties of the endothelium is the soluble forms of adhesion molecules, the level of which can be measured in the blood.

The mechanisms of endothelial dysfunction are involved not only in the pathogenesis of many diseases, but also in the formation of hemostasis disorders, the immunoinflammatory process in the vascular wall, etc. Three main stimuli can be identified that cause "hormonal" reaction of the endothelial cell:

1) change in blood flow speed (increase in shear stress);
2) platelet mediators (serotonin, ADP, thrombin);
3) circulating and/or "intrawall" neurohormones (catecholamines, vasopressin, acetylcholine, endothelin, bradykinin, histamine, etc.) [2].

CONCLUSION

Thus, endothelial dysfunction is the main pathogenetic mechanism in the development of many diseases, and a more in-depth study of the morphofunctional structure of the vascular endothelium in various pathologies can contribute to early diagnosis and prevention.

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