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HYPEREMIA: PATHOPHYSIOLOGICAL MECHANISMS, HEMODYNAMIC CHANGES AND CLINICAL SIGNIFICANCE

Bahodirova Robiyakhon Lutfullo qizi
Emu University 2nd Year Dentistry 205 Group A

Istamova Sitorabonu Anvar qizi
Scientific Supervisor

Abstract
Hyperemia is a hemodynamic condition characterized by an increase in blood volume in tissues and organs, which develops under the influence of physiological or pathological factors. The article comprehensively discusses the etiology, pathogenesis, microcirculatory changes and clinical significance of hyperemia.

Keywords: Hyperemia, arterial hyperemia, venous hyperemia, microcirculation, stagnation, hypoxia.

Introduction

The circulatory system supplies all tissues of the body with oxygen, glucose, amino acids and other metabolites, and removes carbon dioxide and metabolic waste. And hemodynamic disorders cause a disruption of cell metabolism.

Hyperemia is one of the quantitative changes in blood circulation, which is characterized by an increase in the volume of blood in the tissue. From a pathophysiological point of view, hyperemia is divided into two main types:

1. **Arterial (faol) giperemiya**
2. **Venoz (passive) giperemiya**

Whereas arterial hyperemia manifests as a more adaptive reaction, venous hyperemia is usually associated with a pathological process.

Materials and methods

This article was prepared on the basis of a theoretical and analytical method. Modern textbooks and scientific sources on pathophysiology, internal medicine, histology and hemodynamics were analyzed. Clinical examples were illuminated based on data on heart failure, inflammation, and thrombosis.

The concept of hyperemia

The circulatory system supplies all tissues and organs of the body with oxygen, glucose, amino acids and other essential metabolic substances, while removing carbon dioxide and metabolic products. The metabolic processes that take place in tissues are directly related to the level of their blood supply, that is, perfusion, and a violation of the hemodynamic balance causes a disruption of cell metabolism.

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Hyperemia is one of the quantitative changes in the circulatory system, characterized by an increase in the volume of blood in a specific anatomical area. Pathophysiologically, it is formed by two main mechanisms: either by increased arterial blood flow or as a result of increased difficulty in venous blood flow

Arterial giperemiya

Hemodynamically, the volume of blood flow depends on the pressure difference and vascular resistance, and according to Poasley's law, the flow is proportional to the fourth level of vascular radius. Even a small expansion of the arterioles leads to a significant increase in perfusion. In arterial hyperemia, blood flow increases precisely due to a decrease in vascular resistance, and in venous hyperemia, blood accumulates in the capillaries as a result of blocking blood flow, and pressure rises. Arterial (active) hyperemia is associated with vasodilation of the arterioles. Vasodilation occurs through neurogenous, metabolic and endothelial mechanisms. A decrease in the tone of the sympathetic nervous system, or increased parasympathetic exposure, leads to relaxation of the vascular smooth muscles. And the metabolic mechanisms are related to an increase in CO_2, hydrogen ions, lactate, adenosine and potassium ions. For example, during physical work, ATP breakdown in muscle increases, and adenosine breakdown causes the arterioles to dilate. Nitric oxide (NO) and prostatycline, which are produced by endothelial cells, also promote vasodilation. NO guanylate activates cyclase, thereby increasing the amount of cGMP in smooth muscle cells and induces muscle relaxation.

Arterial hyperemia is often a physiological adaptation reaction. For example, redness of the skin in a hot environment or blood filling of muscles during exercise. Also, arterial hyperemia plays an important role in the inflammatory process. In acute inflammation, histamine, bradykinin, prostaglandins and cytokines (TNF-α, IL-1) are released. These mediators increase vascular permeability and lead to dilation of the arterioles. Clinically, this process causes symptoms of redness (rubor), heat (calor), swelling (tumor), and pain (dolor). In arterial hyperemia, the tissue turns bright red because the content of oxyhemoglobin in it is high, blood flow is accelerated, and oxygen delivery increases. [1]

Venoz giperemiya

Venous (passive) hyperemia, on the other hand, is a pathological process that develops as a result of dysphoria of venous blood. Its main causes include heart failure, venous thrombosis, impaired blood flow as a result of embolism or external compression. For example, in right ventricular insufficiency, venous dampening occurs in the circle of large blood circulation, as a result of which swelling occurs in the lower extremities. In left ventricular insufficiency, increased venous pressure in the lungs develops, and alveolar edema develops.

In venous hyperemia, blood flow slows down, hydrostatic pressure in the capillaries increases, and fluid passes into the interstitial tissue. This leads to the formation of a tumor. The slow flow of blood increases hypoxia because the amount of deoxyhemoglobin increases and the tissue turns blue (cyanosis). Prolonged hypoxia causes a decrease in ATP at the cellular level, disruption of the Na^+/K^+ pump, accumulation of sodium and water intracellularly. Anaerobic glycolysis is strengthened, lactate accumulates and metabolic acidosis develops.

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Chronic venous hyperemia leads to atrophy of parenchyma and an increase in connective tissue. For example, in chronic heart failure, blood accumulation occurs in the liver around the central veins, and peripheral zones remain relatively preserved. As a result, the appearance of liver on the surface of the section is reminiscent of nutmeg, a condition that is called "muscat liver". And in the lungs, prolonged dimming is characterized by the accumulation of hemosiderin and the appearance of "heart cells". So,
Causes: heart failure, thrombosis, embolism, vascular constriction.
Symptoms: cyanosis, cold, pain, swelling.

Microcirculatory changes

At the level of the microcirculatory system, hyperemia is characterized by an increase in capillary pressure, increased filtration and increased lymph flow. In venous hyperemia, stasis may develop, in which erythrocytes accumulate in capillaries and undergo aggregation. This increases the risk of thrombosis. Endothelial dysfunction is also an important factor, since the imbalance of vasoregulatory substances produced by the endothelium leads to pathological changes in vascular tone.

Clinical Significance

Hyperemia is an important diagnostic sign in clinical practice. Arterial hyperemia is observed in the foci of inflammation, allergic reactions and during functional overload. Venous hyperemia, on the other hand, manifests as a major pathogenetic factor in heart failure, thrombosis, and other hemodynamic disorders. Clinically, arterial hyperemia is manifested by hot and red skin, while venous hyperemia is manifested by cold and bluish skin.

Differential diagnosis

It is important to distinguish between arterial and venous hyperemia from a clinical point of view:

	Characters	Arterial	Venosis
1	Tint	Bright Red	Ko'kim
2	Temperature	Warm	It's cold
3	Blood Flow	Fast	Slow
4	Oxygen	A lot	Cam
5	Aftermath	Adaptation	Hypoxia

Conclusion

Thus, hyperemia is an important form of hemodynamic disorders, with the arterial type having a more physiological and adaptive character, while the venous type can cause pathological complications. In arterial hyperemia, increased vasodilation and perfusion are observed, in venous hyperemia, dimming, hypoxia, and metabolic disorders develop. An in-depth understanding of pathogenesis plays an important role in early diagnosis and the selection of the right treatment strategy in clinical practice.

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