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HYPERTENSION AND ITS PATHOGENESIS

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Abstract: This article analyzes the anatomical and morphological changes of the heart that occur in the context of hypertension. Prolonged elevation of arterial blood pressure leads to structural remodeling of the heart, particularly left ventricular hypertrophy and myocardial thickening. Structural alterations also develop in the atrial and ventricular cavities, affecting the functional activity of the heart. The article compares normal cardiac anatomy with pathological changes and explains their pathogenetic mechanisms. The findings highlight the importance of anatomical knowledge in the early detection of hypertension and prevention of its complications.

Keywords: hypertension, heart ventricles, heart, pathological anatomy, morphological changes, myocardium, atria.

WHAT IS HYPERTENSION?

Hypertension is a chronic disease characterized by a persistent increase in blood pressure within the arteries (above 140/90 mmHg). It represents a dysfunction of the cardiovascular system and often progresses without noticeable symptoms, gradually causing serious damage to the heart, brain, and kidneys.

The causes include acute or chronic emotional stress, hereditary and occupational factors, unhealthy dietary habits, and other contributing conditions.

According to its clinical course, hypertension is classified into two main forms:

1. A mild, slowly progressing form that lasts for a long period.
2. A severe, rapidly progressing form that may quickly lead to cerebral damage, renal failure, and sudden visual impairment.

According to the classification adopted by the World Health Organization in 1978, hypertension is divided into three stages:

Stage I:

Blood pressure exceeds 140/90 mmHg (for example, 150–180/90–105 mmHg) without signs of damage to the central nervous system, cardiovascular system, or kidneys. This stage is often temporary, and blood pressure may normalize after rest, stress relief, or antihypertensive therapy.

Stage II:

Systolic pressure ranges between 160–179 mmHg and diastolic pressure between 100–109 mmHg or higher. At this stage, pathological changes are primarily characterized by left ventricular hypertrophy. Retinal arterial narrowing (diffuse or focal), microalbuminuria (presence of protein in urine), and a moderate increase in blood plasma creatinine levels (1.2–2.0 mg/dL) may also be observed. Ultrasound or angiographic examination may reveal atherosclerotic plaques in the aorta and peripheral arteries. Clinical symptoms include frequent headaches, dizziness (hypertensive crises), and chest discomfort.

Stage III:

Blood pressure levels are persistently high (systolic 180–209 mmHg or higher; diastolic 110–119 mmHg or higher). The clinical presentation becomes more severe and diverse. Complications may include angina pectoris, myocardial infarction, hemorrhagic stroke, hypertensive encephalopathy, renal failure (nephroangiosclerosis), and retinal hemorrhage. During severe complications, blood pressure may temporarily decrease or even normalize.

PATHOGENESIS OF THE DISEASE

Blood pressure is the force exerted by circulating blood on the walls of blood vessels and depends on three main hemodynamic factors: cardiac output, total circulating blood volume, and total peripheral vascular resistance. Changes in one or more of these parameters may lead to elevated arterial pressure.

Dysfunction of the central and autonomic nervous systems plays a significant role in the development of hypertension. Under emotional stress, the sympathetic nervous system becomes activated, increasing the release of adrenaline and noradrenaline. This leads to an increased heart rate and vasoconstriction, ultimately raising blood pressure.

Activation of the renin–angiotensin–aldosterone system (RAAS) is another crucial mechanism. Reduced renal perfusion stimulates renin secretion, leading to the formation of angiotensin II. Angiotensin II is a potent vasoconstrictor that increases peripheral resistance and stimulates aldosterone secretion. Aldosterone promotes sodium and water retention, increasing blood volume and arterial pressure.

Endothelial dysfunction also contributes to hypertension. Under normal conditions, the endothelium produces vasodilators such as nitric oxide. A reduction in nitric oxide production impairs vascular relaxation, resulting in persistent vasoconstriction and increased peripheral resistance.

Disruption of water–salt balance is another important factor. Excessive salt intake and impaired kidney function lead to sodium and water retention. Increased blood volume raises cardiac workload and further elevates arterial pressure.

Long-standing hypertension causes structural changes in the vascular walls: thickening, reduced elasticity, and narrowing of the lumen. As a result, the vessels lose their ability to dilate properly, and blood pressure remains chronically elevated.

Thus, in the pathogenesis of hypertension, increased cardiac activity, elevated vascular tone, water–salt imbalance, and neurohumoral activation interact to form a pathological "vicious cycle," contributing to the chronic progression of the disease.

CONCLUSION

The main pathogenetic factors in hypertension include increased cardiac output, elevated vascular tone, and disturbances in water–salt metabolism. Activation of the central and autonomic nervous systems, as well as the renin–angiotensin–aldosterone system, leads to vasoconstriction and increased blood volume. These mechanisms raise total peripheral resistance and cause persistent elevation of arterial pressure. Chronic hypertension results in structural vascular remodeling and damage to vital organs such as the heart, brain, kidneys, and eyes. Therefore, early diagnosis and timely treatment are essential to prevent severe complications.

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