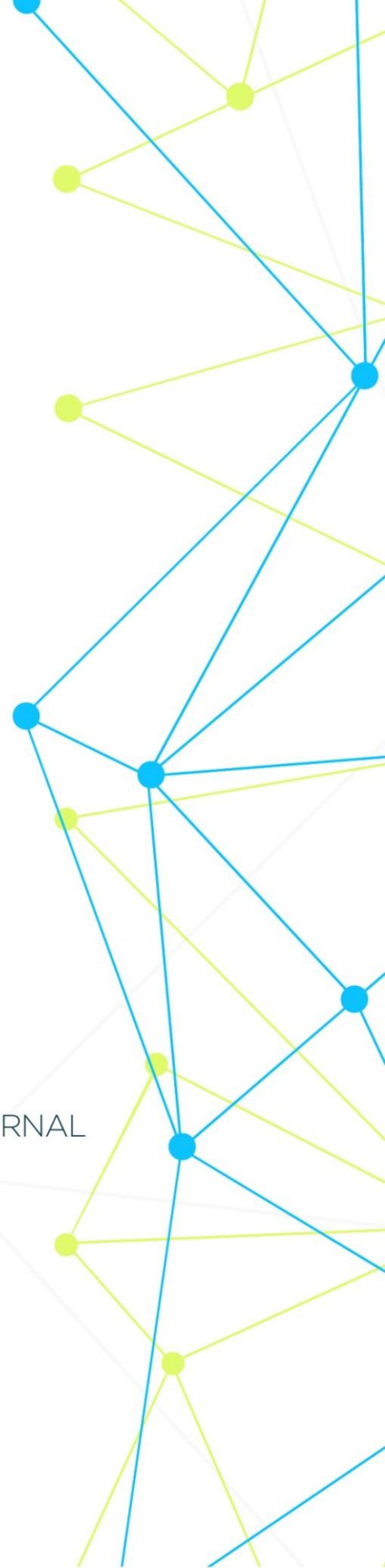




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STUDY OF RISK FACTORS FOR THE DEVELOPMENT OF THE SUSPENDENT PATHOLOGY (OBSERVATION)

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Abstract: Most of the known risk factors for the development of vascular pathology are realized through changes in the properties of the vascular wall. At the same time, large conducting arteries, and primarily the aorta, are subject to pathological influences to a greater extent than peripheral ones. Elastic properties of arteries, that is, pliability, extensibility and stiffness determine the structure of their walls. Experimental studies have shown that an increase in arterial stiffness is associated with structural changes [1,3,8,10]. A significant role in the formation of violations of the mechanical properties of the vessel wall is played by functional factors, such as an increase in shear stress on the endothelium, a temporary increase in the concentration of circulating vasoactive hormones, inflammatory mediators, exudative stress products, and others. Of particular importance among these factors is the functional activity of the vascular endothelium and smooth muscle cells. In response to various external stimuli, dysfunction of the autonomic nervous system can lead to paradoxical vasomotor reactions [2,4,5,9,11].

Introduction

In Uzbekistan, sufficient attention is paid to the surgical aspects of stenosing aortic lesions and branches departing from it in nonspecific aorto-arteritis, neurological aspects of NAA as well as pharmacogenetic aspects of antihypertensive drugs in the treatment of arterial hypertension [6,7,12,19,21].

Currently, there has been a trend of conservative treatment of some degenerative aortic diseases. At the same time, there is no morphological base for the administration of suitable treatment⁶. Since there is a pronounced fibrous-cellular complex in the middle aortic shell, which represents a single morphofunctional system that provides mechanical strength of the vascular wall, it is the structural characteristics of the middle aortic shell that are the key to understanding the processes of compensation and decompensation in the formation of aneurysms and dissection of the aortic wall [13,16,18].

The study of morphological changes in the aortic wall in various pathological conditions can reveal the main factors leading to pathological changes, as well as structures that support the maintenance of the biomechanical properties of the vascular wall [14,15,17].

Main Body

In the middle of the XVI century, the famous French physician Jean-Francois Fernel, who studied the structure of the aortic wall, in 1542 divided aneurysms into internal (thoracic and abdominal aorta and main branches) and external (peripheral vessels). His contemporary Antoine Saporta clarified the clinical course of aortic rupture and suggested distinguishing tumors and aneurysms by the presence of pulsation. In 1557, Andrei Vesalius diagnosed a saccoid aneurysm of the thoracic aorta [20,22,23].

For three centuries, various theories of the appearance of an aortic aneurysm have been put forward, some of which were subsequently confirmed.

Another direction was the theory of degenerative diseases leading to diffuse fusiform or cylindrical expansion of the aorta. So, the above-mentioned J. Fernel argued that with a true aneurysm, there is a change in all layers, and not in isolation of any layer.

J. Lancisi also suggested trauma and congenital pathology as an etiological factor. Already in the XIX century, Antonio Scarpa suggested that atherosclerotic changes may be the cause of aortic dilation.

The first descriptions of aortic dissection are of the greatest interest. In 1628, Daniel Sennertus described the dissection of the aorta with adventitia edema, but conclusions about the pathogenesis were made in favor of the formation of any aneurysms.

In 1760, Frank Nicholls revealed the presence of fenestration of the inner layer of the aortic wall, blood between the layers of the aortic wall and concomitant hemopericardium during the autopsy of the suddenly deceased King of Great Britain George II. Also, Giovanni Battista Morgagni was one of the first to describe the pathology in 1761.

And 60 years later, a detailed description of the process appeared, in 1819 Rene Laennec applied the term "delaminating aneurysm", determined the possibility of dilatation of the aorta in chronic dissection. However, the most correct term "dissection" of the aorta was used 17 years earlier by Jean Pierre Manuard. The concept proposed by Manuard was undeservedly forgotten, due to its lesser fame in comparison with Laennec, who was popular because of the invention of the stethoscope.

In addition, in 1904, Scarpa claimed that the dissection is associated with ulceration or tearing of the aortic wall and subsequent separation of the wall layers by blood. A. Barnes in 1809 and J. Hodgson in 1815 described isolated cases of aortic dissection with a hemopericardium, similar to the case with King George II.

The aorta is an example of elastic type arteries, the aortic wall consists of three layers: internal or intima (tunica intima), middle or media (tunica media) and external or adventitia (tunica adventitia). Intima is a basement membrane covered with endotheliocytes. At the border of the separation of intima and media is a fenestrated elastic membrane. Further, the fenestrated elastic membranes and smooth muscle cells are distributed concentrically, there is also an expanded network of connections between the membranes, including micro fibrils (elastic fibers), thus the middle layer is composed – the media, which occupies more than 60% of the thickness of the aortic wall. Outside, the middle layer is enveloped by the plexuses of its own blood and lymphatic vessels of the aorta, connective tissue. Thus, the main shock of the pulse wave takes over and distributes the media. Due to elastic fibers, the aortic wall has the ability to stretch and absorb the pulse wave. Also, collagen fibers are located in the media, providing a framework, which protects against overgrowth of the wall.

The main etiological factors of aneurysms and dissections of the ascending aorta include arterial hypertension, non-syndromic and syndromic (Marfan, Ehlers-Danlo, Loyes-Dietz, Shereshevsky-Turner syndromes) connective tissue dysplasia, Gzel-Erdheim syndrome, atherosclerotic and degenerative processes, infectious lesions (syphilitic, mycotic), autoimmune diseases (giant cell aorto-arteritis), nonspecific aorto-arteritis. In addition, cases of the appearance of this pathology in members of the same family are not uncommon. A connection between aneurysmal dilatation of the ascending aorta and congenital bicuspid aortic valve (DAC) was also revealed. At the moment, infectious lesions can be a fairly rare cause of the formation of aneurysms and dissections of the ascending aorta. In the presence of a penetrating atherosclerotic ulcer, the risks of dissection and/or rupture of the aorta also significantly increase.

The main triggers and factors of excessive load on the aortic wall include high blood pressure, pheochromocytoma, the use of stimulating narcotic drugs (cocaine), injuries (including auto-injuries and falls from a height), heavy lifting and excessive straining, aortic coarctation, pregnancy, iatrogenic dissection during X-ray endovascular or open cardiac surgery.

The main pangenetic feature of aortic dissection is the rupture of the intima, followed by the formation and spread of a subintimal hematoma. A hematoma spreading along the layers of the aortic wall usually occupies from 1/2 to 3/4 of its diameter, in some cases the dissection spreads circularly along the entire circumference of the aorta. As a result, a "false" lumen, or a two-light aorta, is formed, which significantly increases the risks of malperfusion of internal organs due to impaired blood flow through the main arteries extending from the aorta. When the dissection spreads to the ascending aorta, tamponade of the pericardial cavity may develop.

In general, aortic dissection occurs with a frequency of 0.2 to 25 per 100,000 populations per year. In the Russian Federation, this figure is 3-25 per 100,000 populations per year, In the USA, the incidence of aortic dissection is 1 case per 10,000 hospitalized patients (but it is worth remembering that a significant part of patients dies at the pre-hospital stage). Aortic dissection is detected in 1 case per 400 autopsies, in 1 out of 100 sudden deaths and in 3-4% of all sudden deaths from cardiovascular diseases. According to the literature, it is shown that the probability of acute dissection, rupture of the ascending aorta and death correlates with the diameter of the aorta. The size of 60 mm is critical, and after reaching such sizes, the risk of a complicated course of pathology increases significantly (dissection or rupture of the aorta, sudden death). In this group of patients, the annual risk of rupture, aortic dissection and sudden death is 3.6%, 3.7% and 10.8%, respectively, and the total risk of such outcomes is 14.1%. If we correlate the diameter of the ascending aorta with the body surface area, then the risk of adverse events associated with the aorta is minimal in patients with an aortic diameter index of less than 2.75 cm/m² (4% per year), moderate (8%) – with an aortic diameter index of 2.75 – 4.25 cm/m², and high (about 20% per year) with an index of more than 4.25 cm/m².

In the Republic of Uzbekistan, the first publications on aorto-arteritis appeared in the mid-60s. Of great importance in the study of this pathology was the establishment of close scientific ties between scientists of Uzbekistan and Russia. A number of studies have been carried out on thoracic aortitis and abdominal aorta.

According to the data of the International Registry of Acute Aortic Dissections (IRAD) from 1996 to 2005, in 59% of cases the average diameter of the aorta in patients was less than 55 mm, and in 40% – less than 50 mm; the maximum diameter of the aorta averaged 53 mm. It can be concluded that the diameter of the aorta itself is not decisive, and there are other factors that cause complications in the aneurysm of the ascending aorta.

The first lifetime diagnosis of aortic dissection in the middle of the nineteenth century was reported by K. Svein and P. Latham. However, real progress in clinical recognition emerged only with the advent of more effective radiographic methods, which provided clinical and pathological correlation before autopsy. By the time of clinical manifestations in 2/3 of patients, the ascending aorta is involved in the process, and in 1/3 – only the distal one. In men, stratification occurs 2-4 times more often and in most cases at the age of 40-60 years.

Aortic dissection is the most significant and frequent catastrophe among acute aortic diseases. In the absence of qualified assistance from specialists, early mortality in case of dissection is 1% per hour during the first two days, 75-85% – within two weeks, and 90-95% – during the first year. But it is important to know that the survival rate of patients can now be significantly increased thanks to timely diagnosis and early treatment of this formidable condition. Early clinical recognition and various diagnostic techniques for aortic imaging are an integral part of the management of patients with aortic dissection.

The most critical issue of diagnosis is in case of emergency admission of the patient and instability of hemodynamics. In this situation, standard algorithms for routine diagnostics can cost the patient his life. Therefore, the question of the reasonable sufficiency of the volume of research and preparation of the patient before surgery is acute. In order to get the patient to the operating table as quickly as possible and start the operation. At the same time, before the operation begins, the surgical team should have the necessary amount of information to anticipate the maximum range of potential risks and complications. There are currently no generally accepted algorithms of actions, there are only recommendations regarding individual research methods. Undoubtedly, the issue of developing an algorithm for the necessary and sufficient examination and preoperative preparation for various variants of the proximal aortic dissection is relevant.

For a number of centuries, the anatomy of the aortic valve has been studied. So Galen in 130 AD was the first to describe the function of AC and the aortic root, and Leonardo da Vinci in 1513 and Antonio Maria Valsalva in 1740 studied the mechanics of AC and hydrodynamics in the aortic root zone. Henle first used the term "artery root" instead of "aortic ring". But the greatest interest in this issue arose in the second half of the XX century, and was, at first, only descriptive. Since 1966, starting with the works of Jacob Zimmerman studies began to be morphofunctional in

nature, the lack of a direct assessment of the biomechanics of AK became a limitation. Studies of functional anatomy have made it possible to determine the morphofunctional boundaries of the aortic root and advance in the study of its function. Since then, the aortic root has been considered as an inseparable anatomical and functional complex consisting of supporting structures for fixing the aortic valves and being a bridge between the left ventricle and the aorta.

According to modern concepts, the structure of the AC should be considered together with the aortic root (valve-aortic complex). At the same time, the ascending aorta is functionally and anatomically a single formation consisting of two divisions: the aortic root and the aorta proper. The valvular-aortic complex is a bulbous structure, which normally includes strong fibrous elements of the aortic root (fibrous ring of the base of the valves; commissural rods; arched ring or sinotubular (distal) articulation), three semilunar flaps, corresponding to them three Valsalva sinuses, three interstitial Henle triangles between them, as well as ventriculoaortic (proximal) junction. The average diameter of the aortic mouth in adults is approximately 2.3 ± 0.3 cm, and the area of the aortic opening is 4.6 ± 1.1 cm². The aortic root is located almost in the anatomical center of the heart, its structures are wedged between the two atria and the trunk of the pulmonary artery. Unlike the right chambers of the heart, where the tricuspid valve is located at some distance from the pulmonal valve.

In the left chambers, the aortic and mitral valves are closely adjacent to each other. In this case, the anterior flap of the mitral valve continuously continues into the non-coronary sinus AK, the so-called mitral-aortic contact. Therefore, speaking of topographic-anatomical relationships, it should be mentioned that the heart has its own fibrous skeleton. Its formation involves three valve apparatuses with their fibrous rings (aortic, mitral and tricuspid), as well as the fibrous part of the interventricular septum of the LVP). However, the basis is the aorta-ventricular membrane, which by means of a close interweaving of connective tissue structures (including the right and left fibrous triangles, as well as the intervalvular triangle) unites all elements of the fibrous skeleton of the heart. The right fibrous triangle is functionally the center of the connective tissue formations of the heart, and in surgical terms corresponds to the projection of the passage of the Gis beam. Thus, the fibrous framework provides fixation of many layers of the myocardium, as well as cardiac septa, is a rigid support for the valve apparatus, and also separates the atrial cavities from the ventricles.

Ventriculoaortic junction (valve base ring, in foreign literature – "aortic ring") is a rounded anatomical connection between the LV outlet and the aorta, which is a fibrous and muscular structure. Formed on average by 45-47% from the myocardium of the arterial cone of the LV.

The sinotubular junction (arched ring, or arched ridge) is represented by three arched arches connecting the tops of three commissures, wave-shaped, located between the aortic root and the ascending aorta proper.

The semilunar valves of the AK are the locking elements of the valve that prevent the movement of blood in the opposite direction, their proximal edge departs from the semilunar part of the fibrous ring, which is a dense collagen structure. They

are located in the distal part of the LV exit tract and are separated from the mitral valve by right and left fibrous triangles with a sub aortic curtain (between the valve triangle). The name of the flaps is determined by the departure from the aorta of the main coronary arteries: right coronary (anterior), left coronary (posterior and left) and non-coronary (posterior and right) flaps. AK flaps consist of a body (the main loaded part), a surface of co-adaptation (closure) and a base. Each leaf has several folding surfaces that face the aorta, and a smooth surface facing the ventricle. The thickening of the triangular shape in the central part of the cooptation zone is called the Arantzi nodule.

The flaps divide the aortic root into sub- and supravalyvular components. The subvalvular elements are similar in their morphological structure to the myocardium of the left ventricle, and the supravalyvular elements are similar to the aorta. It is worth noting that the similarity is transitional, i.e. only dominant morphological structures are histologically marked, but in each of these areas there are opposite structures.

The line of attachment of the flaps does not follow a geometric ring, but has a crown-shaped shape. Semilunar flaps consist of connective tissue in the form of fibers and scattered cells lined on both sides with an endocardium forming a kind of duplication. The stroma of the valves does not contain blood vessels and belongs to tissues with a very low level of metabolism. The free edge of each leaf is slightly elongated and has a fibrous arantium nodule in the center. The rest of the half moon, especially near the U-shaped line of its fixation with the aorta, turns out to be thinned and almost transparent. The severity of each leaflet is very variable, but the perimeter of their free edge, taken together, significantly exceeds the circumference of the aorta at the same level.

Valvular half-moons form three commissures, in the area of which the aortic wall is thickened and compacted. Commissura is a line connecting adjacent flaps with its peripheral proximal edges on the inner surface of the distal segment of the aortic root and adjacent to the sinotubular junction with its distal end. Commissural rods (columns) are places of fixation of commissures on the inner surface of the aortic root. The commissural rods are a distal extension of the three segments of the fibrous ring. The sinuses of the Valsalva are expanded areas of the aortic root, limited by the valves and the sinotubular junction. At the base level, they are separated by Henle triangles. Their main role is the redistribution of blood tension during diastole and the establishment of an equilibrium position of the valves in the systole.

The sinuses of the Valsalva serve as a transitional link between the left ventricle and the aorta, as a result of which both beginnings are carried in their histological structure. The LV muscle elements create only a support for the base of the aortic root, mainly at the level of the right coronary sinus.

The sinuses of Valsalva consist only of intima and media in the proximal department, adventitia is added at the level of the sinotubular junction. The inner layer of the sinuses is covered, like the vessels, with endothelium. The middle layer has circular connective tissue structures (collagen, elastin), as well as smooth muscle cells. Adventitia consists mainly of type I collagen. In the overall structure, it is worth noting that the media of the Valsalva sinuses is thickened due to an increase in the

number of collagen fibers. At the same time, in the direction from the ventricle to the aorta, there is an increase in the number of elastic fibers and a decrease in collagen fibers. Such a change leads to an increase in functionality, since collagen is "responsible" for the strength and rigidity of the structure, and elastin is for the ability to stretch and "extinguish" excessive aortic tension during systole.

There are three functionally identical sinuses, while from an anatomical point of view there are their differences. Coronary arteries depart from two of them, so they are called the left (left - posterior) and right coronary sinuses, while the third is commonly called non-coronary (right - posterior).

The interstitial triangles of Henle are fibrous or fibrous-muscular components of the aortic root located proximal to the commissures between adjacent segments of the fibrous ring and the corresponding flaps. Anatomically, the interstitial triangles are part of the aorta, but functionally they provide exit routes from the LV and are affected by the hemodynamics of the ventricle, not the aorta. Henle triangles play an important role in the biomechanical function of the valve, allowing the Valsalva sinuses to function relatively independently, unite them and maintain a uniform geometry of the aortic root.

During the examination of the patient, it is important to know the condition of the aortic wall, the ratios and sizes of the structures of the root and ascending part, the arch and descending part of the aorta from the point of view of choosing a treatment strategy and determining the scope of the operation, if an active surgical tactic is chosen. As aortic pathology develops, regardless of the severity, the aortic valve may be involved in the process of aortic lesion with the formation of aortic regurgitation of varying severity. The causes of aortic regurgitation can be: A - dilation of the aortic root and the fibrous ring of the aortic valve (FC AK), B - rupture of the FC AK or valve flaps; C - displacement of one of the flaps below the valve closure line; loss of the area of fixation of the flaps; D - mechanical effect of detached intima on the closure of the aortic valve.

Morphological changes in the aortic wall outside the dissection zone concern all structural components of the vascular wall - elastic and collagen fibers, MMC and interstitial substance. However, the nature of these structural changes in RAA is heterogeneous, which allowed us to distinguish 3 variants. The first option is a predominant lesion of the elastic frame of the media with signs of hypoelastosis and a total violation of the structure of the EM with the disappearance of the fibrillar component and the absence of contact with collagen fibrils. This group mainly includes cases with differentiated and undifferentiated connective tissue dysplasia. The second option is severe focal necrotic changes and the formation of medianecrosis sites in the presence of severe damage to the adventitia vessels, including fibrinoid necrosis of their walls. This group mainly includes patients aged 45-55 with severe hypertension and, as a rule, without permanent treatment. However, unlike patients with hypertension without RAA, this group revealed insufficient collagen formation in the media and adventitia of the aorta. The third option is nonspecific diffuse changes in all structures of the aortic wall. It usually occurs in patients aged 60 years and older and is often combined with atherosclerosis. At the

same time, the stratification channel does not affect the media in the area of atherosclerotic plaques. In all observations, including the group with genetically determined hereditary diseases, hypertension seems to play a decisive role in the formation of RAA. It is possible that the process of stratification is associated with it in the presence of various variants of pre-existing injuries in the aortic wall, accompanied by a violation of both the contractility of the EM and the strength of the wall due to the inferiority of collagen formation. From these positions, the discrepancy between the frequency of AH and the rarity of the bundle becomes obvious.

Ascending aortic aneurysms are an absolute indication for surgical treatment. At the same time, several approaches are distinguished: wrapping of the ascending aorta and prosthetics of the ascending aorta, a more radical approach is also proposed - prosthetics of the ascending aorta with a plastic arch of the "semi-arc" type, which allows resecting a larger volume of the pathologically altered aortic wall. To date, there are no uniform recommendations on the level of formation of distal anastomosis during aortic prosthetics in patients with ascending aneurysm.

The pathogenesis of an aortic aneurysm is based on a change in its wall. Histological examination of intraoperative material is one of the most objective research methods that allow to assess in detail the morphological state of the aortic wall. At the same time, understanding the morphological status of the aortic wall at different sites can help in choosing an adequate level of resection of the ascending aortic aneurysm. In the available literature, we have not found any works devoted to morphological studies of the aortic wall, which would allow us to fundamentally substantiate the advantage of the procedure over traditional prosthetics. The identification of the morphological structure of the aortic wall at different levels will allow us to justify the necessary level of distal anastomosis in patients with ascending aortic aneurysms, which was the purpose of our work. In patients with ascending aortic aneurysm, the aortic wall at the level of the arch is morphologically more preserved compared to the distal zone of the ascending aorta. Prosthetics of the ascending aorta with arch plasty may be the preferred surgical option in these patients.

Reperfusion syndrome is an unavoidable phenomenon when restoring blood flow after prolonged ischemia. The article is devoted to the study of the severity of this condition. The aim is to compare the depth of morphological changes in the arterial endothelium against the background of ischemic and reperfusion lesions in the experiment. Materials and methods. The study was performed on 90 laboratory animals of the Wiztar line. Models of ischemia and reperfusion were created by squeezing the abdominal aorta (group 1) followed by conditioning (group 2). The animals were removed from the experiment and the vascular wall was taken on the 1st, 3rd, 5th, 7th day. The study of the preparations was performed on a Libra 120 transmission electron microscope with automatic image scanning. Results. Comparison of path morphological data obtained during the study of the aorta and iliac arteries of animals of two groups ("ischemia" and "reperfusion") shows that the cascade of path morphological changes includes several main stages. Transient ischemia leads to damage (alteration) of the main components of the vascular wall.

Endotheliocytes under the influence of this factor react in a non-specific way, changing their synthetic activity, which is manifested by a set of morphological features that capture the nucleus, karyolemma, cytoplasm and plasma lemma. In some cells, the changes become irreversible, accompanied by rupture of mitochondrial membranes, general-purpose organelles, plasma lemmas. Such endotheliocytes die and desquamate. Due to the insignificant duration of ischemia, these changes are not significantly pronounced. Sub endothelial structures undergo edema, which is natural due to the violation of the barrier function of the endothelium and a slightly pronounced inflammatory component (in response to the death of some endotheliocytes and vascular wall stroma cells). When studying the ultrastructure of the vascular wall in the ischemia-reperfusion group, adaptive and pathological changes in endothelial cells were detected. Data were obtained indicating a significant violation of microhemodynamics in tissues during reperfusion. Conclusion. There were no significant structural and ultrastructural differences in the pattern of damage and reactive changes in the "ischemia" and "reperfusion" groups. In this regard, for a subtler differentiation of the differences in pathomorphogenesis in these two conditions, it is advisable to apply higher-resolution research methods.

The work of Shilova M.A. presents the results of a study of 83 cases of sudden death of young people with external and internal signs of connective tissue dysplasia with aortic pathology (DST). During sectional and pathomorphological examination, it was found that the main pathology that is the cause of death was vascular pathology caused by cardiovascular syndrome in connective tissue dysplasia. Pathological changes in the vascular wall were detected in the aorta, the vessels of the brain and heart. Morphologically, the pathology of the aorta was represented by aneurysms of various localization with a rupture of the wall and the development of hemorrhagic shock. It has been established that the main pathogenetic mechanism of aneurysm formation in DST is a violation of the structure of the vascular wall caused by a congenital defect of the vascular muscle layer and damage to the internal elastic membrane. In conditions of physical exertion, sports, psychoemotional stresses, in violation of the usual rhythm of life, these factors lead to the risk of sudden death with rupture of blood vessels in areas with the least resistance.

Conclusion

Thus, knowledge of the mechanism of formation of aortic regurgitation, the structure and topographic anatomy of the aortic root plays a key role in choosing a method of treatment of aortic and aortic valve pathology. Ruptures and dissections of the ascending aorta remain an urgent problem of modern cardiology and cardiac surgery. The search for new methods of diagnosis and treatment is the main task, which will contribute to a better understanding of the mechanisms of ruptures and delamination and will allow for the prevention of these conditions.

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