UNIVERSAL JOURNAL OF MEDICAL AND NATURAL SCIENCES
ISSN 2992-8826
https://sr-journals.org/index.php/UJMANS

MORPHOLOGICAL FEATURES OF AN ASCENDING AORTIC WALL ANEURYSM (A CASE FROM CLINICAL PRACTICE)

[1] Choriev Beruniy Akbarovich, [2] Babajanova Visola Marifjon qizi,
[3]Maxmudova Umida Rustam qizi
[1] Tashkent State Medical University, Assistant, Department of Pathological Anatomy, PhD
[2] Tashkent State Medical University, Master's student, Department of Pathological Anatomy
[3] Tashkent State Medical University, Master's student, Department of Pathological Anatomy

Abstract. This study is devoted to the investigation of the macro- and microscopic morphological features of a clinical case associated with an aortic aneurysm. The tissue of the aortic wall obtained during autopsy was taken as the object of the study. Histological analysis was performed on the basis of specimens stained with hematoxylin-eosin and by the Van Gieson method. Microscopic examination revealed a decrease in smooth muscle cells in the medial layer of the aortic wall, necrobiotic changes, as well as fragmentation of elastic fibers. Van Gieson staining demonstrated diffuse destruction of collagen fibers and the predominance of a fibrotic process. The weak expression of inflammatory infiltration indicates the degenerative nature of the pathological process. The obtained results confirm that medial necrosis and a decrease in the mechanical strength of the wall play an important role in the pathogenesis of aortic aneurysm. Morphological changes were evaluated in correlation with clinical data.

Keywords: Aortic aneurysm, ascending aorta, pathomorphology, medial necrosis, fibrosis, degradation of elastic fibers, Van Gieson stain, hematoxylin-eosin, military.

Аннотация. Ушбу тадқиқот аорта аневризмаси билан боғлиқ клиник ҳолатнинг макро-ва микроскопик морфологик хусусиятларини ўрганишга бағишланган. Тадқиқот объекти сифатида аутопсия вақтида олинган аорта девори тўқимаси олинди. Гистологик таҳлил гематоксилин-эозин ва Ван Гизон усулида бўялган препаратлар асосида амалга оширилди. Микроскопик текширувда аорта деворининг медиа қаватида силлиқ мушак ҳужайраларининг камайиши, некробиотик ўзгаришлар ҳамда эластик толаларнинг парчаланиши аниқланди. Ван Гизон бўёғида коллаген толаларининг диффуз парчаланиши ва фиброз жараён устунлиги намоён бўлди. Яллиғланиш инфильтрациясининг суст ифодаланиши патологик жараённинг дегенератив хусусиятга эга эканини кўрсатади. Олинган натижалар аорта аневризмаси патогенезида медиа некроз ва деворнинг механик мустаҳкамлиги пасайиши муҳим аҳамиятга эга эканини тасдиқлайди. Морфологик ўзгаришлар клиник маълумотлар билан уйғун ҳолда баҳоланди.

Калит сўзлар: Аорта аневризмаси, кўтарилувчи аорта, патоморфология, медианекроз, фиброз, эластик толалар деградацияси, Ван Гизон бўёғи, гематоксилин-эозин, ҳарбий.

Аннотация. Данное исследование посвящено изучению макро- и микроскопических морфологических особенностей клинического случая, связанного с аневризмой аорты. В качестве объекта исследования была взята ткань стенки аорты, полученная во время аутопсии. Гистологический анализ проводился на основе препаратов, окрашенных гематоксилин-эозином и по методу Ван Гизона. При микроскопическом исследовании в медиальном слое стенки аорты выявлены уменьшение количества гладкомышечных клеток, некробиотические изменения, а также фрагментация эластических волокон. При окраске по Ван Гизону обнаружены диффузное разрушение коллагеновых волокон и преобладание фиброзного процесса. Слабая выраженность воспалительной инфильтрации указывает на

дегенеративный характер патологического процесса. Полученные результаты подтверждают, что в патогенезе аневризмы аорты важное значение имеют медианекроз и снижение механической прочности стенки. Морфологические изменения оценивались в сопоставлении с клиническими данными.

Ключевые слова: Аневризма аорты, восходящая аорта, патоморфология, медианекроз, фиброз, деградация эластических волокон, окраска по Ван Гизону, гематоксилин-эозин, военный.

Introduction. The aneurysm of the ascending aorta is considered one of the most dangerous pathologies of the cardiovascular system. The main risk of this disease depends on the damage of the aortic wall - a condition that is related with a very high risk of death. Therefore, timely diagnosis and treatment of ascending aortic aneurysm are of great importance [2, 7].

Typically, thoracic aortic aneurysms emerge in elderly patients as a result of atherosclerosis and degenerative changes. However, analyses from the past decade show that increasing aortic aneurysms are also found out among young individuals (20-35 years) and even among professional athletes [1, 3, 9]. In such young patients, congenital connective tissue disorders that weaken the aortic wall are often present. In particular, hereditary conditions such as Marfan syndrome, Loeys-Dietz syndrome, congenital aortic valve abnormalities, and Ehlers-Danlos syndrome may lead to pathological dilation of the aortic wall and aneurysm formation in young athletes. In addition, cases have been reported in which excessive physical exertion and long-term use of anabolic steroid-type doping substances caused damage even to a previously healthy aortic wall in young athletes, resulting in early aneurysmal changes. Evidence suggests that individuals engaged for many years in heavy athletic sports may demonstrate an increase in aortic dimensions beyond normal reference values [4, 7]. Specifically, a study conducted in 2020 showed that 21% of master-level athletes exhibited clinically significant dilation of the ascending aorta (≥40 mm), a figure noted to be substantially higher than the expected population-based prevalence. These findings underscore the need to reassess the impact of physical activity on the aortic wall and emphasize the importance of preventive monitoring even in young athletes [5].

Clinical and morphological studies confirm that pathological alterations in the structure of the vascular wall play a decisive role in the pathogenesis of aortic aneurysms. In particular, a morphological condition known as cystic medial degeneration occupies a central place in ascending aortic aneurysms. This condition is characterized by fragmentation of elastic fibers, loss of smooth muscle cells, and collection of pathological glycosaminoglycan substances within the medial layer of the aorta. As a result of medial degeneration, the mechanical strength of the aortic wall decreases; the vessel wall weakened and, over time, its susceptibility to aneurysmal dilation progressively increases. In congenital pathological conditions observed in young individuals, such as Marfan syndrome, these degenerative changes are more pronounced: the structural organization of the medial layer of the aortic wall is completely disrupted, and marked cystic degeneration is observed [3, 11]. If an aortic aneurysm develops in elderly patients on the background of atherosclerosis, changes such as atheromatous plaques and calcium deposition predominate in the vessel wall, while destruction of the medial layer is relatively less pronounced. Thus, in Marfan syndrome-associated aneurysms, computed tomography of the aorta commonly reveals a steady, smooth dilation of the vessel, which histologically corresponds to diffuse cystic medial degeneration. On the other hand, atherosclerotic aneurysms are characterized by vessel walls rich in calcifications, and histological examination reveals extensive calcium deposits and fibrous tissue within the wall. Therefore, alongside the clinical manifestations of aortic aneurysms, their morphological characteristics also differ significantly depending on the underlying etiological factors [11, 13].

Over the past years, scientific study on aortic aneurysms has primarily focused on the hereditary and molecular foundations of the disease, as well as on methods for early diagnosis. In particular, both the 2022 guidelines of the European Society of Cardiology (ESC) and the recommendations of the American Heart Association/American College of Cardiology (AHA/ACC)

emphasize the implementation of specific screening programs and genetic testing for the timely identification of hereditary syndromes such as Marfan and Loeys-Dietz syndromes [11, 15]. It is also highlighted that first-degree relatives of patients with aortic aneurysms should undergo periodic echocardiographic examinations or genetic analyses, as the possibility of aortic dilation is known to be significantly higher in hereditary situations. In addition, researchers have conducted comparative morphological studies of aortic aneurysms of various etiologies. Some previously published works have mainly focused on isolated descriptions of individual syndromes and nosological entities, while comparative clinical and pathoanatomical analyses of aortic diseases with diverse etiologies have been addressed partially. However, morphological examination of the aneurysmal wall enhances the ability to identify the true cause and mechanisms of disease development, thereby facilitating the selection of the most appropriate treatment and monitoring strategy for each patient. From this perspective, an in-depth investigation of the clinical and pathological features of ascending aortic aneurysms occurring in young athletes and military personnel represents an issue of substantial relevance for both theoretical understanding and clinical practice [15, 16].

Typically, the main factors contributing to the development of aortic fusiform aneurysms are long-standing, uncontrolled arterial hypertension, which leads to degeneration of the aortic wall, as well as atherosclerosis, which plays an important role. Consequently, this disease is predominantly observed in middle-aged and elderly individuals. It occurs more frequently in men than in women, with a ratio of approximately 3:1. In young patients, aortic fusiform aneurysms are often associated with congenital cardiovascular abnormalities, such as aortic coarctation, aortic stenosis, bicuspid aortic valve, and other aortic valve defects, as well as connective tissue disorders. In this population, the condition may also be related to hereditary medial dysplasia, infectious causes (syphilis, bacterial thrombo-aortitis, athero-arteritis, bacterial-embolic processes), and non-infectious aortitis [5, 9, 13]. Non-infectious aortitis can develop in the context of spondyloarthropathies (associated with HLA-B27), psoriatic arthritis, Crohn's disease, indeterminate ulcerative colitis, and rheumatoid arthritis. Etiological factors for stenosing forms of aortic inflammation include systemic vasculitides, particularly nonspecific aorto-arteritis and giant cell arteritis. In many cases, aortic fusiform aneurysms progress insidiously and are clinically manifested only after vascular wall disruption [4, 14].

Objective of the Study. The aim of this study is to investigate the morphological changes in the ascending aortic wall and to determine their characteristics, based on the lifetime medical records, as well as histological and autopsy materials, of 23-year-old military personnel.

Research Methodology. The study was conducted at the Forensic Medical Examination Center of the Armed Forces. The patient was a 23-year-old male who had been in military service since 2024. Previous examinations revealed 120 ml of serous fluid on the right and 100 ml on the left side of the thoracic cavity. Upon inspection of the pericardium, a clotted blood mass weighing 410g was found in the pericardial cavity. Measurements of the ascending aorta revealed a perforation measuring 1.2 × 0.9 cm. The edges of the perforation were irregularly torn. In this region, the aortic wall was sac-like dilated, with dimensions of 5.5 × 6.5 cm; the wall was thin, the intima detached, and a thrombus was detected below the intima. The thrombus was thick and firmly connected between the intimal and adventitial layers. The ruptured portion of the aneurysm involved all layers of the vessel wall. Hemorrhages were observed in the surrounding tissues. During the examination, all internal organs of the deceased, as well as the area of the aortic aneurysm, were subjected to forensic histological analysis. Tissue samples from the internal organs were fixed in 10% formalin, embedded in paraffin blocks, and sectioned onto microscope slides. The sections were then stained with hematoxylin and eosin. *Medical History:* No prior serious chronic diseases were recorded. Family history was unremarkable for cardiovascular diseases. The patient led a healthy lifestyle and had not used prohibited substances (doping) in sports. Occasionally, mild chest pain was experienced during heavy physical exertion, but the patient did not consider it necessary to seek medical evaluation.

UNIVERSAL JOURNAL OF MEDICAL AND NATURAL SCIENCES
ISSN 2992-8826
https://sr-journals.org/index.php/UJMANS

1. Medical Records. The patient's medical records were reviewed. According to the medical card provided by the outpatient clinic where the patient lived, he had developed normally since birth, without any congenital defects. All scheduled vaccinations had been administered. No congenital or acquired pathologies were identified. The military conscription medical card indicated that the patient had undergone examinations by all narrow specialists and was classified as "Healthy." According to the medical certificate from the Angren City Medical Association, the following diagnosis was recorded: hypertonic type of vegetative-vascular dystonia. Clinical charts and notes from the patient's medical history were also examined. Instrumental examinations performed during the patient's lifetime-including X-rays, electrocardiography, blood tests, and others-along with physician observations, were included in the analysis.

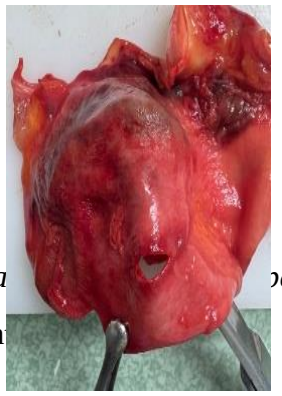
2. Morphological Examination Data. *Autopsy Materials:* Following a forensic autopsy, tissue samples were collected from the area of the aneurysm in the ascending aorta. The dilated part of the aortic wall and adjacent tissues were preserved for morphological analysis. *Macroscopic characteristics of the Aortic Aneurysm:* The intimal surface of the vessel wall was irregular; the medial layer was thin, and the adventitia was attenuated. A perforation was observed in the central area of the aneurysm.



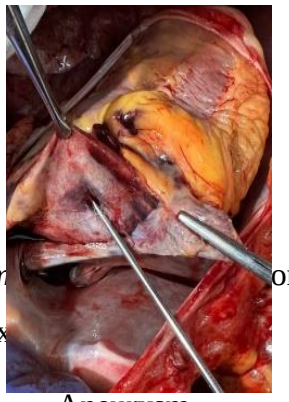
l Sta *ecim* ortic

In Ex F

w



Aneurysm



Aneurysm



10% formalin solution. The tissue was then embedded in paraffin blocks, and sections with a thickness of 4-5 μm were prepared. Histological sections were added with the standard hematoxylin-eosin method. Additionally, to allow a more detailed analysis of specific tissue elements, supplementary special staining techniques were applied. In particular, the Van Gieson method was used to stain collagen fibers.

3. Histological Specimens. Permanent microscopic slides (histological blocks) were prepared from the aortic tissue samples under laboratory conditions. These slides were subsequently used for microscopic analysis. The prepared histological specimens were examined using a light microscope. During microscopy, various magnifications (ranging from ×4 to ×400) were done to evaluate microscopic differencs in the aortic wall, including fragmentation of elastic lamellae, loosening of collagen tissue, inflammatory infiltration, and other alterations. The observed tissue changes were documented through microphotography.

UNIVERSAL JOURNAL OF MEDICAL AND NATURAL SCIENCES
ISSN 2992-8826
https://sr-journals.org/index.php/UJMANS

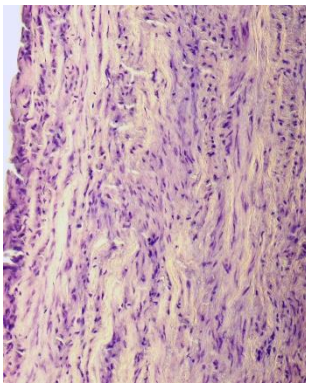
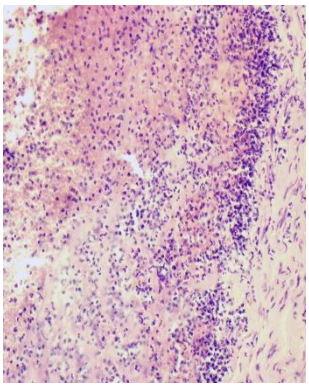
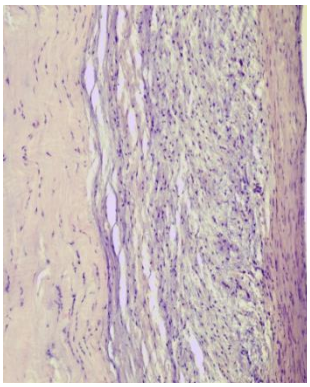
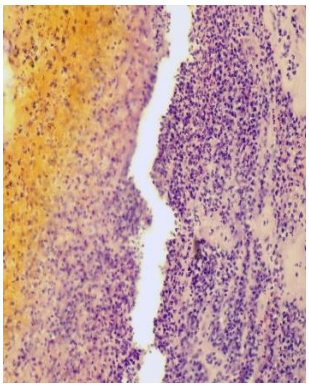





Figure 1 **Figure 2** **Figure 3** **Figure 4**

Microscopic analysis revealed pronounced pathomorphological changes in all layers of the aortic wall (intima, media, and adventitia). In the intima, fibrosis, endothelial disruption, and thrombotic elements were observed. In the media, destruction of elastic fibers and medial necrosis were prominent, representing the primary alterations. The structural integrity of the medial layer was markedly compromised. Specifically, fragmentation, disintegration, and disorganization of elastic lamellae were observed (1), along with a reduction in smooth muscle cells (medial necrosis) (2). In certain regions, loss of cell nuclei, homogenized eosinophilic masses, and necrotic foci were detected (3). In the adventitia, an increase in fibrous tissue accompanied by a mild inflammatory reaction, characterized by lymphocytic and plasmacytic infiltration, was noted (4). In several specimens, signs of chronic fibrosis were evident, indicating decreased mechanical strength of the aortic wall. The observed morphological changes are characteristic of aneurysmal dilation and wall weakening developing on the background of medial necrosis, with the inflammatory component being secondary in nature (2-4).

The morphological changes observed in the aortic wall associated with the aneurysm were grouped and classified. The alterations were divided into degenerative changes-characterized by loosening of the aortic wall structure and fragmentation of elastic fibers-and inflammatory changes. This classification allowed for a systematic comparison of the findings and facilitated the formulation of relevant conclusions.

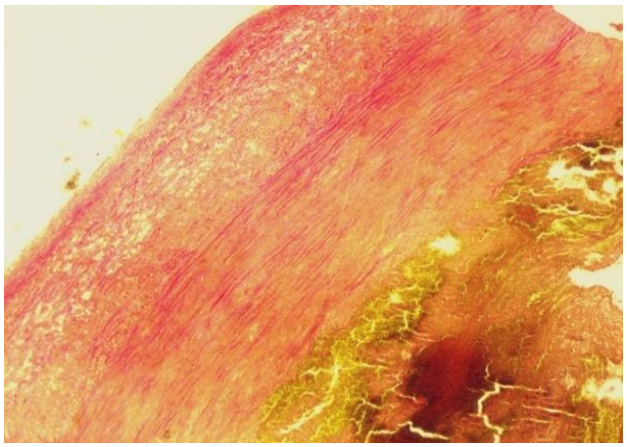


Figure 5. Although the layered structure of the aortic wall is preserved in the specimen, the medial layer shows marked disorganization. *Van Gieson Staining:* Collagen fibers appear bright red, while muscle and elastic elements are seen in yellowish-reddish shades. In the medial layer, there is a diffuse increase in collagen fibers, which compress the average elastic and muscular elements, indicating fibrotic transformation. In some regions, the boundaries between the wall layers are not clear. These changes provide a morphological basis for the mechanical weakening of the aneurysmal aortic wall.

UNIVERSAL JOURNAL OF MEDICAL AND NATURAL SCIENCES
ISSN 2992-8826
https://sr-journals.org/index.php/UJMANS

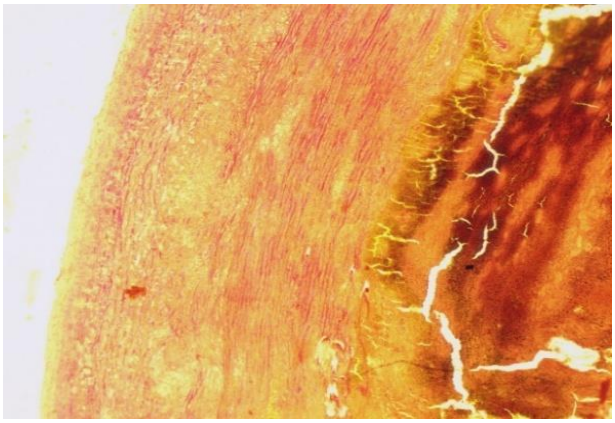


Figure 6. In the medial layer, fragmentation and rupture of elastic fibers are observed, replaced by thickened, irregularly arranged collagen fibers, along with a reduction or loss of smooth muscle cells.

Between the collagen fibers, there are acellular, homogeneous areas, characteristic of medial necrosis. Van Gieson staining clearly shows that the fibrotic process developed after the medial necrosis.

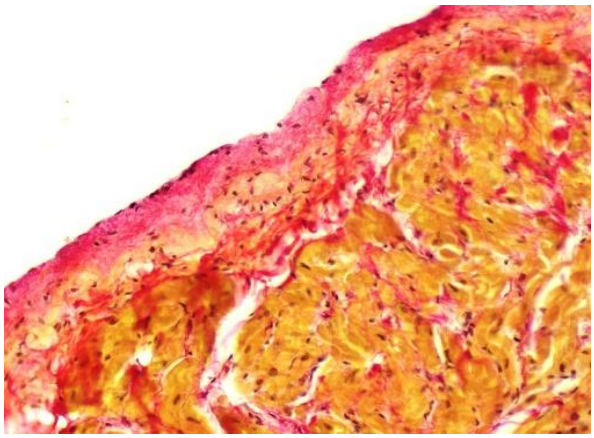


Figure 7. Collagen fibers are densely packed, forming large bundles. Only a few fibroblast nuclei and inflammatory cells are observed.

This indicates that there is no active inflammatory process, confirming that the changes in the aortic wall are of a degenerative-dystrophic nature. Thus, the aneurysm developed on the basis of primary medial necrosis, rather than as a result of aortitis.

Analysis of Hematoxylin-Eosin and Van Gieson Staining Results

In this study, morphological changes in the aortic wall were analyzed using two histological staining methods: hematoxylin-eosin (H&E) and Van Gieson, allowing for a comprehensive assessment of both structural and functional aspects of the pathology. H&E staining revealed a thinning of smooth muscle cells in the medial layer, loss of cell nuclei, eosinophilic homogeneous masses, and necrobiotic changes. There was mild lymphocytic infiltration in the intima and adventitia. These findings indicate a predominance of degenerative-dystrophic processes in the aortic wall. Van Gieson staining further highlighted the structural consequences of the changes identified with H&E. To be specific, fragmentation and destruction of elastic fibers in the medial layer, accompanied by a diffuse increase in collagen fibers, were clearly observed, confirming a marked reduction in the mechanical strength of the vessel wall. Van Gieson staining demonstrated that fibrotic processes predominated over inflammation. Thus, H&E staining revealed the cellular-degenerative stage of the pathological process, while Van Gieson staining clearly demonstrated its tissue-structural remodeling.

The patient's age of 23 and active engagement in sports are clinically significant. The hereditary or primary degenerative pathology of the aortic wall caused the development of an aneurysm in a young patient, although typically aortic aneurysms occur in older individuals.

Microscopic examination revealed medial necrosis, destruction of elastic fibers, and fibrosis-changes that are clinically relevant in the context of constant high hemodynamic stress on the aortic wall during physical activity. Areas of the wall weakened by high arterial pressure and mechanical load are predisposed to aneurysmal dilation.

Additionally, the mild expression of inflammatory infiltration corresponds with clinical data showing no evidence of infectious or autoimmune disease. This indicates that the observed changes in the aortic wall are not related to aortitis but rather developed on the basis of primary medial necrosis.

Analysis and results. Microscopic analysis revealed that the main changes were located in the medial layer of the aorta, manifested by fragmentation of elastic fibers, loss of smooth muscle cells, and necrotic foci. It was found out that fibrosis and mild inflammatory infiltration in the intima and adventitia.

The examination demonstrated features of aortic changes without atherosclerotic damage, consistent with a non-inflammatory process. These changes resulted in dilation and thinning of the aortic wall, as well as rupture of blood into the pericardial cavity, causing the development of cardiac tamponade. Based on perimeter measurements, the diameter of the ascending aorta in the dilated area reached 6.5 cm, allowing the aneurysm to be classified as a saccular aneurysm.

Comparison: The histological findings were analyzed in comparison with normal (healthy) aortic structure and similar cases reported in the scientific literature. Comparative analysis highlighted both the distinctive features and similarities of the changes observed in this patient.

The identified morphological alterations reduce the mechanical strength of the aortic wall, creating conditions conducive to aneurysmal dilation. The mild expression of the inflammatory response indicates that aortitis is secondary, and that medial necrosis serves as the primary pathogenetic basis for the development of the disease.

Conclusion. The primary pathogenetic factor in aortic aneurysm identified medial necrosis is caused by structural disorganization and mechanical weakening of the vessel wall. The microscopic changes increase the risk of clinical complications and are of critical importance for timely diagnosis.

References

1. Balraj Singh et al. Aortic Dissection in a Healthy Male Athlete: A Unique Case with Comprehensive Literature Review. Hindawi Publishing Corporation Case Reports in Cardiology Volume 2016, Article ID 6460386, 5 pages.
2. BA Choriev, MT Valieva, DR Sobirova. Forensic histological assessment of fat embolism in combined injury death cases. Galaxy International Interdisciplinary Research Journal. - 2024. - Vol. 11. - P. 577-81.
3. Beruniy A. Choriyev, Xasan Z. Tursunov. Morphological features of pleomorphic tumors of the salivary glands. american journal of applied medical science. - 2025. Issue-1. - P. 53-64.
4. Clouse WD, Hallett JW Jr, Schaff HV, Spittell PC, Rowland CM, Ilstrup DM, Melton LJ 3rd. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clin Proc. 2004;79(2): 176-80. doi: http://dx.doi.org/10.4065/79.2.176.
5. Erbel R., Aboyans V., Boileau C. et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J 2014; 35(41): 2873-2926.
6. Suziku Toru, Hirohisa Katoh, Ryozo Nagai (1999). Biochemical diagnosis of aortic dissection: from bench to bedside // Japanese Heart Journal. - 2013. - №40(5). - P. 527-534.
7. Tchartchan ER, Abugov SA, Polyakov RS, Zaklyazminskaya EV, Rumyantseva VA, Khovrin VV, Skvortsov AA, Kabanova MN, Khatchatryan ZR, Oparina NV. Shereshevsky-Turner syndrome in

aortic surgery: an example of hybrid aneurism treatment and dissection of thoracoabdominal aorta. Russian Journal of Cardiology. 2015;(5): 111-7. Russian. doi: 10.15829/1560-4071-2015-5-111-117.
8. John Papagiannis. Sudden death due to aortic pathology. Sudden Cardiac Arrest in the Young: A Contemporary Perspective. - Vol. 27. - 2017.
9. KZ Tursunov, BA Choriyev - Morphological Features of Middle Neck Cysts. Journal of education and scientific. - 2024. - Vol. 3. - P. 51-56.
10. Timothy W. Churchill, MD. Endurance Exercise Among Older Masters-Level Athletes. JAMA Cardiology. - Vol. 5. - №. 5.
11. T. Paul Tran, Ali Khoynezhad. Current management of type B aortic dissection // Vasc. Health Risk Manag. - 2009. - Vol. 5. - P. 53-63.
12. Von Kodolitsch Y, Nienaber C, Dieckmann C, et al. (2004). Chest radiography for the diagnosis of acute aortic syndrome // Am J Med. - 116(2). - P. 73-77. Graham S. A., Williams A. Marfan's syndrome and the heart // Archives of Disease in Childhood. - 2007. - Vol. 92(4). - P. 351-356.
13. Weigang E., Nienaber C. A., Rehders T. C. et al. Management of Patients With Aortic Dissection // Dtsch. Arztebl. Int. - 2008. - 105. - P. 639-645.
14. Кошелева Н.А., Горохова Е.А., Резяпкина М.С. Особенности течения расслаивающей аневризмы аорты. Архив внутренней медицины. 2016; 3 (29): 68-70.
15. Кузнечевский Ф.В., Осипов А.Х., Евсиков Е.М. и др. Распространенность и природа аневризм и расслоений аорты по данным анализа последовательных патологоанатомических вскрытий в течение десяти лет в ГКБ № 15 им. О.М. Филатова. Российский кардиологический журнал. 2004; 6 (50): 5-13.
16. Струков А.И., Серов В.В. Патологическая анатомия. 5-е изд., стер. -М.: Литтерра, 2010; 880 с.