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Aortic aneurysm – current state of knowledge analysis

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ABSTRACT

Introduction:

Thoracoabdominal aortic aneurysms (TAAAs) are rare vascular pathologies involving the aorta from the descending thoracic to the abdominal segments. Due to their asymptomatic progression and high risk of rupture, they present significant diagnostic and therapeutic challenges. Although endovascular repair has become increasingly common, open surgical repair remains essential for complex or extensive aneurysms.

Aim of the study:

This study aims to summarize current knowledge on the etiology, classification, diagnosis, and classical surgical treatment of TAAAs.

Materials and methods:

A literature review was conducted focusing on diagnostic modalities, surgical techniques, and clinical outcomes. Special emphasis was placed on Crawford's classification and protective measures during open repair.

Results:

TAAAs represent ~3% of all aortic aneurysms. Risk of rupture rises significantly with diameter, exceeding 14% annually for aneurysms >6 cm. CT angiography remains the diagnostic gold standard. Point-of-care ultrasound and MRI are useful adjuncts. Open surgery, although technically demanding, remains essential for extensive disease. Perioperative complications include spinal cord injury and renal failure. Techniques such as cerebrospinal fluid drainage and left heart bypass improve safety and outcomes.

Conclusion:

Open repair of TAAAs remains a key therapeutic approach. Early detection, optimal imaging, and tailored intraoperative strategies are essential to reduce complications and improve prognosis.

Keywords:

Thoracoabdominal aortic aneurysm; open surgery; aneurysm rupture; perioperative management.

Introduction:

Thoracoabdominal aortic aneurysms (TAAAs) constitute a rare but highly complex vascular pathology involving the aortic segment that spans both the thoracic and abdominal regions. Although representing only about 3% of all aortic aneurysms, their silent progression and high rupture risk make them a significant clinical challenge. In untreated cases, mortality can reach up to 87%, while even with surgical intervention, perioperative mortality remains substantial.

Historically, treatment of TAAAs has advanced from traditional open repair to a hybrid of endovascular and open surgical techniques, guided by enhanced imaging modalities and risk stratification systems. Among these, the Crawford classification remains central for evaluating aneurysm extent and surgical planning.

The predominant cause of TAAAs is degenerative atherosclerosis, which leads to the fragmentation of elastin fibers and loss of smooth muscle cells in the tunica media. Other etiologies include chronic dissection, connective

tissue disorders such as Marfan and Loeys-Dietz syndromes, and infectious (mycotic) aneurysms. This diverse pathogenesis underscores the need for individualized diagnostic and therapeutic strategies.

Epidemiological data indicate that aneurysm size and expansion rate are critical determinants of rupture risk. Aneurysms exceeding 6 cm in diameter have a significantly increased annual rupture rate. Risk factors such as hypertension, smoking, and genetic predisposition further contribute to disease progression.

Diagnostic evaluation relies heavily on imaging, with contrast-enhanced computed tomography (CT) serving as the gold standard. Magnetic resonance imaging (MRI), echocardiographic techniques, and contrast-enhanced ultrasound (CEUS) also play important roles in assessing aneurysm morphology, complications, and treatment planning.

While endovascular repair is increasingly used in anatomically favorable cases, open surgical treatment remains the primary option for extensive aneurysms. Despite being technically demanding and associated with high perioperative risk, surgical outcomes can be optimized through techniques such as left heart bypass, cerebrospinal fluid drainage, and intraoperative monitoring.

This review synthesizes current knowledge on the etiology, classification, diagnosis, and classical management of TAAAs, aiming to support evidence-based decision-making in the care of patients with this challenging vascular disease.

1. Historical outline

Since ancient times, the aorta has been the subject of study by physicians. The term "aorta" itself is derived from the Ancient Greek verb suspend/lift, and the noun "belt," as it was believed that the aorta was the "belt" from which the heart hung. The first specific reference to the aorta is found in Homer's Iliad (700-705 BCE), followed by the work of Hippocrates of Kos, who used the term aorta to describe the large vessels of the heart - both veins and arteries. It was only Aristotele who used this term to refer to a single vessel – which corresponds to the modern aorta.

The first mention of aortic aneurysms as a "pulsating swelling in the dorsal vertebral region" was described by Vesalius in 1557. In 1572, French surgeon Ambroise Paré described a thoracic aortic aneurysm and linked its presence to the presence of syphilis in a patient[1]. Another important figure of the 17th century who leaned towards the topic of aortic aneurysms is Valsalva, who, based on observations, suggested a therapy consisting of rest and starvation. He based this treatment on the assumption that hypotension would reduce the impulse of blood and thus eliminate one of the causes of stretching and rupture of the artery wall. Morgagni - a disciple of Valsalva initiated the idea of the existence of pathological changes in the organ as the cause of the disease. It is impossible not to mention Morgagni's work, published, in 1761, in which he included more than 700 clinical case descriptions of thoracic aortic aneurysms and thoracoabdominal aortic aneurysms[2].

A better understanding of the pathophysiology of aneurysms has encouraged surgeons to deepen their knowledge of the factors that predispose to their occurrence, as well as to seek surgical methods of treating them. Antonio Scarpa (1747-1832), an anatomist from Pavia who lived in Italy in the 19th century, considered aortic aneurysms to be an atherosclerosis-related degeneration of the vessel wall. He considered syphilis to be one of the risk factors

for aneurysms. Scarpa also identified the causes that cause aneurysm rupture: a vigorous blow inflicted on the patient, an accelerated heartbeat, progressive slow degeneration or ulceration of the artery's inner membrane[3].
Two French surgical pioneers: Alexis Carrel (1873-1944) and RenéLeriche (1879-1955), due to the lack of highly effective treatment of aneurysms, attempted to wrap the aneurysm with cellophane to prevent further growth [4].
In the 1950s, French surgeon Charles Dubost (1914-1991) operated on an ascending aortic aneurysm by resecting it and suturing the edges of the aortic wall. In 1951, Dubost became the first surgeon to resect an abdominal aortic aneurysm and replace it with a homograft with a 50-year-old patient. He obtained a 15-centimeter homograft from a 20-year-old woman who had died three weeks earlier. This was the first successful operation to resect an aortic aneurysm and implant a homograft, making it the basis for modern aortic reconstructive surgery [5]. In the following years, a synthetic artery substitute, Vinyon-N, was developed, followed by Orlon, Nylon, Teflon and Dacron. In 1953, Charles DeBakey performed the first thoracic aortic aneurysm surgery using a vascular prosthesis made of Dacron to replace the homograft[6]. In 1966, New Orleans native Oscar Creech (1916-1967) used and described a method that is still used today. It involves replacing the dilated segment of the aorta with a vascular prosthesis and then covering it with an aneurysm sac[7].
Despite advances in surgical technique, the introduction of extracorporeal circulation and better postoperative care, the surgical treatment of aortic aneurysms has still not achieved satisfactory results, so the search for a less invasive method of treatment has continued. The first reports on a minimally invasive method of treating aortic aneurysms were published in 1991, describing a procedure performed in Kharkiv in 1987 by Ukrainian surgeon Nicholas Volodos. At the time, he treated a thoracic aortic aneurysm using a stentgraft of his own design[8]. Around the same time, reports emerged of an abdominal aortic aneurysm repair performed in 1990 in Buenos Aires performed Juan Parodi. He inserted a vascular prosthesis into the lumen of the abdominal aorta through an access from the femoral artery and fixed it in the aorta with stents. Parodi and his team pioneered the treatment of aortic aneurysms with stentgrafts in the early 1990s [9].

2. Definition, causes of formation, Crawford classification.

A thoracoabdominal aortic aneurysm is characterized by an enlargement of the aortic lumen exceeding 50% of the normal diameter at the level of the diaphragmatic aortic hiatus. The correct aortic diameter depends on a number of factors such as age, gender, height and weight, and mean blood pressure. [10]

Causes of formation:

The classification of thoracoabdominal aortic aneurysms can be based on their underlying cause. The primary etiological factor is the atherosclerotic process, which gradually leads to **degeneration of the tunica media**, involving both the loss of smooth muscle cells and the fragmentation of elastin fibers. [11]

In contrast, the second most common cause is **aortic dissection**. A classic aortic dissection develops as a result of a tear in the intima, which initiates a progressive cleavage plane within the tunica media. The torn and weakened aortic wall predisposes to the formation and rupture of aneurysms.

The extent of the dissection is classified based on which segments of the aorta are involved in the pathological process. Commonly used classification schemes are the DeBakey classification and the Stanford classification scheme. [10]

Genetic conditions may predispose to the formation of thoracoabdominal aortic aneurysms, especially connective tissue disorders, including:

- **Marfan syndrome** - Mutation of the fibrillin-1 gene results in dysregulation of transforming growth factor beta (TGF-β) signaling, which contributes to the gradual degradation of the extracellular matrix within the aortic wall.
- **Loeys-Dietz syndrome** - determined by mutations in genes encoding TGF-beta receptors.

Fungal aneurysms (also termed mycotic aneurysms), on the other hand, are conditioned by infection of the aortic wall and often occur in the region of the visceral branches, assuming a saccular shape. In contrast, among bacterial pathogens, the following are distinguished: Staphylococcus aureus, Staphylococcus epidermidis, Salmonella and Streptococcus.

Crawford Classification:

There is a classification according to Crawford, which serves several important functions. This approach permits precise patient risk stratification, supports the selection of a treatment modality tailored to the expected extent of aortic replacement, and ensures standardized reporting of postoperative results This classification distinguishes:

Type I - involves extensive disease of the aorta, requiring replacement from the origin of the left subclavian artery to the origin of the renal arteries.

Type II - includes replacement of most or the entire descending thoracic aorta, as well as the suprarenal and infrarenal segments of the abdominal aorta. These repairs typically begin distal to the origin of the left subclavian artery and extend to the bifurcation into the common iliac arteries.

Type III - is characterized by dilation of the distal half of the descending thoracic aorta (below the level of the sixth rib), extending through the abdominal aorta down to the bifurcation into the common iliac arteries.

Type IV - the aneurysm is limited to the aorta below the diaphragm. Repairs in this category begin at the diaphragmatic hiatus and frequently involve the entire abdominal aorta. [10]

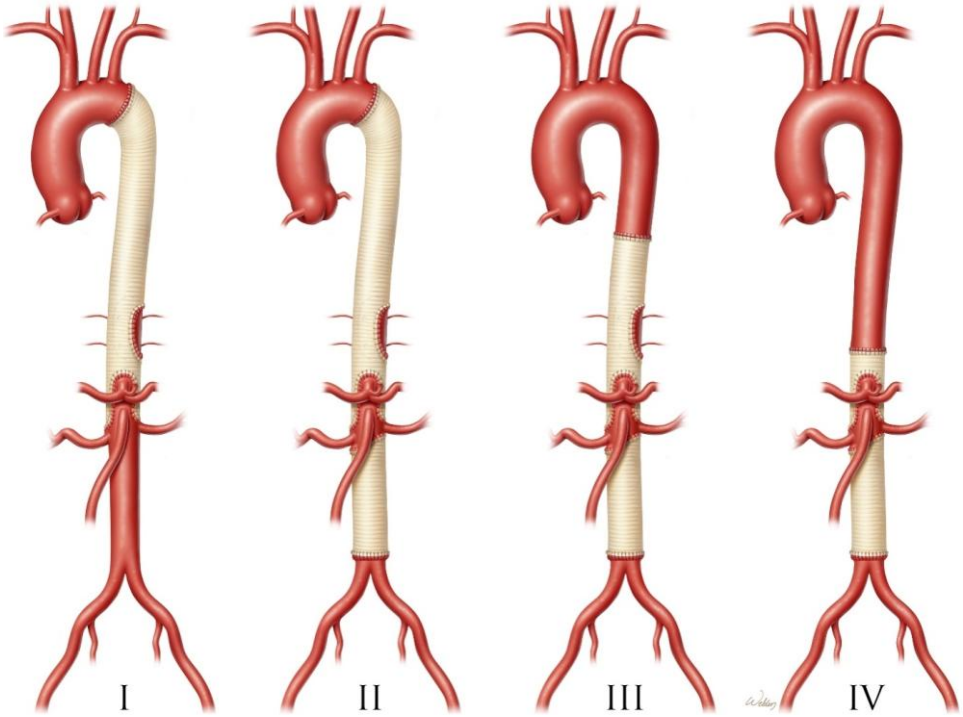

Fig. 1. Crawford classification scheme for thoracoabdominal aortic aneurysms. Reproduction from [12] under a Creative Commons Attribution 4.0 International License.

In addition, Crawford's classification was expanded to include a fifth type proposed by Safi and Associates.
Type V - It includes repairs that begin in the lower segment of the descending thoracic aorta (again, below the level of the sixth rib) and terminate proximal to the origin of the renal arteries.

3. Epidemiology

Thoracoabdominal aortic aneurysms are relatively rare and account for ~3% of all aortic aneurysms and ~6% of abdominal aortic aneurysms. For many years they may not give any symptoms, but when they rupture they pose a serious risk to the patient's life. Mortality in those who do not undergo surgery ranges from 20% to as high as 87% [14-16], meanwhile, in those who do, perioperative mortality ranges from 9% to 21.7% (sequentially for elective and emergency procedures) [17]. Data from Germany show that the morbidity is 5.9/100 000/year [13].

The aortic arch is derived from the embryonic pharyngeal arches formed by neural crest cells, whereas the descending thoracic and abdominal aorta develop from dorsal mesenchymal cells originating in the endocardial cushion. The aforementioned dissimilarities may account for the differences in the development of aneurysms. For example, the presence of atherosclerosis is positively correlated with the occurrence of aneurysms of the descending and abdominal aorta, and negatively correlated with aneurysms of the ascending aorta. The best indicator in assessing the risk of aneurysm rupture is the diameter of the vessel. Aneurysms of the thoracoabdominal aorta have a larger diameter than aneurysms of the ascending aorta (5.9cm and 4.8cm)[18]. Aneurysms with a diameter of 6 cm are associated with a fivefold higher risk of rupture, with an estimated annual rupture rate of 14.1% [17,18]. Complication risk rises progressively with increasing aneurysm diameter, reaching

over 40% when the diameter exceeds 7 cm [17]. Aneurysms of the descending aorta tend to grow faster, compared to aneurysms of the ascending aorta, and this is 0.19cm/year and 0.07cm/year, respectively [18]. One of the complications of an aneurysm can be dissection, which most often occurs at an average size of about 5cm, with a mortality rate estimated at 9%.

Due to the relatively small number of patients described in the literature, risk factors for thoracoabdominal aortic aneurysm development are poorly defined. So far, genetic factors, COPD, hypertension and smoking have been classified[10].

4. TAPB diagnostic methods

During the diagnosis of an aneurysm, we focus on patient-reported symptoms, family history and cardiovascular risk factors.

Starting with the symptoms, there may be pain or throbbing in the chest. Pain localized to the anterior chest is more commonly associated with Stanford type A dissections, while pain in the back and abdominal regions is more indicative of Stanford type B. Dissections involving the descending aorta may additionally present with interscapular pain. Less typical symptoms such as cough, shortness of breath or hoarseness (with aneurysmal laryngeal nerve palsy) may also occur, however, clinicians should also be aware of the possibility of clinically silent aortic disease.

Rupture of an aortic aneurysm presents with acute, intense pain and marked hypotension progressing to shock and, frequently, death. Atypical symptoms such as hemoptysis may occur if the aneurysm penetrates the trachea or lung parenchyma, while hematemesis may be observed in cases of fistulization into the esophagus [20]

Imaging methods are of utmost importance in the diagnosis of aneurysms. Methods such as transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE), computed tomography, magnetic resonance imaging and conventional angiography are used.

In the initial diagnosis, ultrasound plays a major role due to its availability, ease, speed of use, low price and non-invasiveness. With the help of transthoracic and more accurate transesophageal ultrasound in the case of a dissecting aneurysm, we can detect the true and false lumen. The undoubted advantages of ultrasound are especially appreciated in emergency medicine. Point-of-care ultrasound (POCUS) protocols—such as RUSH (Rapid Ultrasound for Shock and Hypotension), UHP (Undifferentiated Hypotension Protocol), and RCT—incorporate the assessment of the aorta to detect potential aneurysmal dilation. They allow assessment of aneurysm enlargement with a sensitivity and specificity of 98% with an accuracy of +-0.2 cm. It also allows assessment of the anatomical relationships of the aneurysm in relation to the renal and visceral vessels. In addition, echo-doppler allows assessment of blood flow through the aneurysm. However, the disadvantages of this diagnostic method are a high dependence on the experience of the examiner and variable examination conditions caused, for example,

by the presence of a large amount of air in the intestinal loops. It is a useful method for the prospective evaluation of aneurysms because, unlike CT, it does not expose the patient to ionizing radiation.

Contrast-enhanced ultrasound is becoming an increasingly widely used tool for detecting peri-implant leakage and is being used in patients after endovascular treatment. It is slowly becoming an alternative to CT scanning due to the fact that it is faster, cheaper and does not expose the patient to ionizing radiation. However, it still cannot be used in place of CT, especially when a leak or the presence of aneurysmal lesions is detected.

CT still remains the gold standard because of its high accuracy, good availability, relatively low price and reproducibility of results. Aortic CT angiography has effectively supplanted traditional catheter-based angiography as the primary diagnostic modality. It is now the reference method for monitoring a patient with a dilated vessel wall and after treatment of aortic aneurysms. Computed tomography enables detailed evaluation of aneurysm dimensions, active bleeding, presence of a false lumen, mural thrombus formation, ulcerative changes, and vascular wall calcifications. MIP and MPR reconstructions help visualize the morphology of the aneurysm and its relationship to other structures. The use of reconstructions helps in surgical planning and postoperative evaluation. It serves as an excellent imaging method for the detection and assessment of periprosthetic endoleaks. It allows its detection and appropriate classification. The sensitivity of leak detection is 92% which is more than traditional angiography but still less than MR. On the other hand, unlike ultrasound, it is a more objective method, and its diagnostic value does not depend on the experience of the examiner.

MRI, unlike CT, does not involve patient exposure to ionizing radiation and iodine contrast agents, which is particularly important in children and pregnant women. Magnetic resonance imaging provides high-resolution assessment of the aneurysm in relation to adjacent structures, enables detection of inflammatory edema, and allows for determination of the age of the inflammatory process. The use of special sequences in MR imaging allows imaging of intravascular blood flow. Magnetic resonance angiography serves as a reliable non-invasive alternative to traditional catheter-based angiography in diagnostic evaluation. The downsides of this method include its time-consuming nature, less availability than ultrasound or CT, and its relatively high price. It is not suitable for use in emergencies, but with the development of technology, images are obtained more and more quickly and therefore its importance in this matter is gradually increasing. Due to its reliance on strong magnetic fields, this imaging technique is generally not suitable for patients with implanted cardiac devices such as pacemakers or implantable cardioverter-defibrillators (ICDs).

With the advancement of modern imaging techniques, the role of aortography has diminished owing to its invasive nature and its limited sensitivity for detecting subtle aneurysms. Nevertheless, it remains a valuable tool in cases of ambiguous imaging results or when used directly before initiating therapeutic procedures.

In choosing an imaging method, we should consider its diagnostic value, potential exposure to contrast and ionizing radiation, availability of the method, and, of course, potential cost, so we try to skillfully choose the type of examination to make an accurate diagnosis. [10]

In the differential diagnosis, it is important to exclude emergency conditions such as acute coronary syndrome, pericarditis or pulmonary embolism. Pain characteristics may aid in distinguishing acute aortic syndromes from acute coronary syndromes, as the former typically present with continuous pain independent of exertion. Electrocardiography (ECG) is an essential component of initial evaluation. Although D-dimer levels may be helpful in the pre-imaging assessment of suspected aortic dissection, they should not be relied upon as the sole basis for exclusion.[21]

5. Treatment methods - classical treatment

The problem of treating abdominal and thoracic aortic aneurysms should be considered in a multifaceted manner, from prevention, to pharmacotherapy, to complex surgical treatment, which is now giving way to endovascular techniques, although there are also situations in which classical treatment is preferred:

- **Emergency indications:** ruptured aneurysms (perioperative mortality is 40-70% [20])
- **Urgent indications:** symptomatic aneurysms (indication for hospitalization and performance of full diagnostics, which if it confirms or does not exclude rupture is an indication for immediate surgery)
- **Elective indications:** asymptomatic aneurysms of the ascending aorta and aortic arch >55 mm in diameter, >60 mm aneurysms of the descending aorta (here endovascular treatment is preferred if possible). Smaller values include patients with Marfan syndrome or bicuspid aortic valve and risk factors. Asymptomatic abdominal aortic aneurysms >55 mm in diameter and patients with rapid aneurysm enlargement (greater than 10 mm/year [20]) confirmed by imaging [30].

For every patient, a case-by-case evaluation is required to determine whether the risk associated with intervention exceeds the potential risk of aneurysmal rupture.

Contraindications to surgery:

1. Age > 85 years
2. Canadian Cardiovascular Society (CCS) Class III or IV
3. Left ventricular ejection fraction (LVEF) <30%
4. Exacerbation of heart failure within the past 30 days
5. Recent myocardial infarction
6. Complex ventricular arrhythmias
7. Large left ventricular aneurysm
8. Severe valvular heart disease
9. Chronic heart failure or ischemic heart disease following coronary artery bypass grafting (CABG)
10. Extensive atherosclerotic lesions in the coronary arteries
11. Serum creatinine ≥ 3 mg/dL
12. Advanced pulmonary disease
13. Biopsy-confirmed liver cirrhosis with ascites
14. Retroperitoneal fibrosis
15. Inoperable malignant tumor
16. Severe systemic disease
17. Critical general condition [20] [29].

Conventional surgical treatment of thoracoabdominal aortic aneurysms is associated with considerable technical complexity and a high risk of serious complications, including distal ischemia, spinal cord injury resulting in paraplegia, and acute kidney injury or renal failure. To reduce the risk and severity of perioperative complications, several protective measures are utilized, such as systemic and local hypothermia, controlled heparinization,

reattachment of segmental arteries, staged aortic cross-clamping, cerebrospinal fluid drainage, left heart bypass to maintain distal aortic perfusion, selective visceral perfusion, and intraoperative neuromonitoring of spinal cord function. These techniques are selected depending on the anatomy and risk factors present in a particular patient. In Crawford's range I, II, III surgeries, aortic fragment replacement involves the abdominal as well as thoracic segments.

The procedure begins with induction of general anesthesia, after which a cerebrospinal fluid drainage catheter is inserted to facilitate spinal cord protection. The patient's position is important, controlled by an inflatable bag (the right side of the body facing down and the left side positioned so that the shoulders are at a 60° angle and the hips are 30° above the level of the operating table). The incision is most commonly made through the sixth intercostal space, extending posteriorly between the scapula and the spinal processes, and terminating to the left of the umbilicus. A single lung ventilation is initiated. The aortic segment is exposed transperitoneally moving the colon, spleen and kidney while protecting the phrenic nerve. After exposing the aorta, its clamp sites are prepared. The vagus nerve and recurrent laryngeal nerve are identified and protected, and then bypassing is performed (between the left common carotid artery and the subclavian artery), which prevents cardiac ischemia, using an aortic clamp. Intravenous heparin (1 mg/kg) is administered. Cannulation of the left atrium is performed via the inferior pulmonary vein, while the return cannula is positioned in the distal segment of the descending thoracic aorta or alternatively in the left femoral artery. A flow of 500 ml/min through the LHB (left heart bypass) is initiated. Proximal aortic clamping is performed across the distal arch, between the origin of the left common carotid and left subclavian arteries, with distal clamping applied to the descending thoracic aorta at the T4–T7 level. The proximal aorta is incised using electrocautery and debrided of intraluminal thrombus. The upper intercostal arteries are secured to minimize the risk of spinal cord ischemia. A gelatin-impregnated dacron graft is usually used to replace the aortic fragment. The aorta is transected 2-3 cm distal from the proximal clamp to make the anastomosis with a continuous polypropylene suture and to prevent damage to the esophagus. Removal of the artery follows and the proximal clamp is placed on the aortic implant. After cessation of flow through the left heart bypass (LHB), the distal aortic clamp is removed, and any remaining thrombotic material is evacuated. If the outlet of the superior mesenteric artery is visible then flow through the LHB is restarted at 200 ml/min to maintain perfusion. If the renal artery outlet is also visible then cold crystalloid 200-400 ml boluses are similarly administered every 15-20 minutes. When replacing the vessel from proximal to distal, the aortic clamp is moved downward whenever possible to restore blood flow. The aortic graft is vented, the clamps are removed, and protamine sulfate is administered to reverse the effects of heparin. Renal perfusion is confirmed by intravenous administration of indigocarmine dye, if blue dye appears in the urine it means satisfactory renal perfusion. The surgical field is doused with warm physiological saline to reverse hypothermia. Blood flow in the lower extremities is also assessed. The aneurysm wall is wrapped around the graft, the diaphragm is sutured with continuous polypropylene suture, and drains are placed before closure. The thoracic opening is closed with polyester sutures and stainless steel wires.

Postoperative management consists of: administration of anesthesia, monitoring of patient parameters, optimization of oxygen therapy, control of mean blood pressure in the range of 80-90 mmHg, as too high can damage the suture lines causing bleeding, and in turn too low can cause paraplegia and renal failure. After awakening from anesthesia, leg motility, and neurological status are assessed. Cerebrospinal fluid pressure is

maintained at 10-12 mmHg. If postoperative complications occur, they are treated as soon as possible at the onset of the first symptoms. [10]

An important intraoperative issue is organ protection to prevent their ischemia. This is especially true for the spinal cord and abdominal organs [24].

The elements that allow for the preservation of the aforementioned protection are:

- No more than 60 min aortic clamping
- Preventing blood pressure drops during surgery
- Continuous measurement of PMR pressure (possible drainage), and cooling of the PMR
- Use of opioid antagonists
- Sewing in as many intercostal artery departures as possible

It is also believed that renal ischemia should not exceed 25 min [25] [32].

Complications of the Classical Method are divided into three main types:

- **Intraoperative**: hemorrhages caused by damage to the veins: inferior vena cava, iliac vein, renal vein can subsequently cause temporary or permanent failure of the organs supplied, anesthetic complications such as acidosis, hypovolemia and myocardial ischemia.
- **Early:**(up to 30 days after surgery): hemorrhages at vascular anastomosis sites, thrombosis of the implanted graft, peripheral arterial embolism, infections, respiratory failure, circulatory failure, acute kidney failure, gastrointestinal bleeding, neurological injury, disseminated intrasvascular coagulation (DIC)
- **Late**: obstruction of the vascular prosthesis usually caused by thrombosis due to technical error or progression of atherosclerosis, occurrence of aneurysms at anastomotic sites, fistulas between the aorta and inferior vena cava or duodenal lumen [23] [26] [27].

The key factors affecting the number and severity of complications are the size and location of the aneurysm. A large number of complications are caused by aneurysms extending above the renal arteries in such cases renal failure is very common due to their temporary ischemia caused by the operation [31].

Results:

Clinical and diagnostic observations confirm the complexity and heterogeneity of thoracoabdominal aortic aneurysms (TAAAs). Analysis of epidemiological data indicates that TAAAs account for approximately 3% of all aortic aneurysms, with an estimated incidence of 5.9 per 100,000 population annually. Despite frequent asymptomatic presentation, the risk of rupture increases sharply with aneurysm diameter—reaching 14.1% annually for aneurysms exceeding 6 cm and over 40% beyond 7 cm. The highest growth rate is observed in descending aortic aneurysms, averaging 0.19 cm per year.

Imaging played a pivotal role in diagnosis and treatment planning. Computed tomography angiography (CTA) proved to be the most reliable modality, enabling precise assessment of aneurysm extent, morphology, and relation to visceral branches. Ultrasound-based methods, especially point-of-care ultrasound (POCUS), demonstrated high

sensitivity (98%) in emergency settings. Magnetic resonance imaging offered a non-ionizing alternative for high-resolution evaluation, particularly in complex or contraindicated cases.

In the surgical cohort, classical open repair was associated with considerable perioperative risk, particularly in Crawford type II and III aneurysms. Postoperative complications included acute kidney injury, spinal cord ischemia, and respiratory failure, with perioperative mortality ranging from 9% in elective procedures to over 21% in emergency cases. Protective measures such as cerebrospinal fluid drainage, visceral perfusion, and left heart bypass contributed to improved outcomes by reducing ischemic injury.

The findings highlight the importance of accurate preoperative imaging, individualized risk assessment, and multidisciplinary perioperative management in optimizing patient outcomes in TAAA treatment.

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Formal Analysis: [WK][BJ][JD]
Investigation: [WK][BJ][JD]
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Software: [WK][BJ][JD]
Supervision: [WK][BJ][JD]
Validation: [WK][BJ][JD]
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