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COMPREHENSIVE REVIEW OF AORTIC ARCH ANEURYSM MANAGEMENT: FROM PATHOPHYSIOLOGY TO ADVANCED SURGICAL TECHNIQUES

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ABSTRACT

Introduction and purpose: Aortic arch aneurysm is a rare life threatening condition; with in-hospital mortality of 50% following their rupture it is imperative that patients are informed by their medical providers of the risk factors, and available treatments. The purpose of this literature review is to provide an overview of possible surgical interventions, pathophysiology, and presentation of aortic arch aneurysms.
Description of the state of knowledge: With prevalence at just 0.16 - 0.34% aneurysms of the thoracic aorta are less common than those located within the abdominal aorta. Of those located in the thorax only 10% locate within the aortic arch. Genetic disorders such as Loeys-Deitz syndrome, Marfan syndrome, and bicuspid aortic valve, as well as non-genetic factors such as hypertension, inflammatory cell infiltration of the aortic wall, and SMC phenotype switching can all be linked with aortic arch aneurysm onset and progression. The clinical presentation of this aortic pathology is often scarce, however progressive enlargement of the aneurysmal sack may in time cause symptoms stemming from compression of neighboring structures. Surgical techniques used in aortic arch aneurysm management range from very invasive procedures requiring cardio-pulmonary bypass and deep hypothermia, to minimally invasive, fully endovascular approaches, with hybrid procedures situated between them.
Conclusions: Ongoing advancements in imaging techniques and relentless innovation in the field of vascular surgery continue to bring new, less invasive aortic arch aneurysm treatment options. As the surgical repertoire expands, more patients - previously deemed not suitable for intervention - are able to take advantage of the life saving aortic arch repair.

KEYWORDS

Aneurysm, Aortic Arch, Aortic Rupture, Vascular Surgical Procedures, Endovascular Aneurysm Repair

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1. Introduction and purpose

Only one it ten aneurysms of the thoracic aorta affect primarily the aortic arch [1]. Their rarity however, should not diminish our vigilance in looking out for patients affected by this condition. More than 80% of ruptured aortic arch aneurysms treated conservatively lead to in-hospital patient death, while up to 71% of individuals treated with current vascular surgical techniques survive, which puts the overall mortality of ruptured arch aneurysms at around 50% [2]. As such, it is imperative for medical providers to be aware of this aortic pathology, and know that there are treatment options, that sufficiently early employed, may greatly increase the odds of survival. The constant innovation in the field of vascular surgery has greatly broadened the availability of minimally invasive procedures, providing those who would be disqualified from any intervention just two decades ago an option for life-saving treatment [3]. The purpose of this paper is to review the pathophysiology behind the formation of aortic arch aneurysms, provide its clinical presentations, showcase imaging modalities used in aortic arch aneurysm diagnosis and perioperative planning, as well as outline surgical therapeutic procedures employed in its treatment.

2. Description of the state of knowledge

2.1. Anatomical considerations

The main artery within the human body is the Aorta, which can be divided into two major sections - the thoracic aorta originates at the aortic valve and then arches downward towards the diaphragm, and the abdominal aorta which continues past the diaphragm supplying organs of the abdominal cavity. For the purposes of this paper we will focus solely on further topological classification of the thoracic aorta (TA).

TA can be divided in two ways - anatomically into three sections listed in the antegrade blood flow direction. The ascending thoracic aorta, the aortic arch, and the descending thoracic aorta; each with distinctive branches (although they may be individually variable) [4,5]. And according to the reporting standards for thoracic endovascular aortic repairinto so called Ishimaru zones encompassing sections from/to:

- zone 0: sinotubular junction/distal attachment of the brachiocephalic trunk
- zone 1: distal attachment of the brachiocephalic trunk/distal attachment of left carotid artery
- zone 2: distal attachment of the left carotid artery/distal attachment of the left subclavian artery
- zone 3: 2 cm distally from the distal attachment of the left subclavian artery
- zone 4: end of zone 3/mid point of the descending thoracic aorta
- zone 5: mid point of the descending aorta/proximal attachment of the celiac trunk

Understanding of the Ishimaru zones will prove useful while describing endovascular repair approaches in further reading [6].

The physiological diameter of the aortic arch varies between individuals, with age and sex being the primary determinants influencing its dimensions. Mean aortic arch diameter within general population is 2.77 ± 0.37cm for proximal and 2.61 ± 0.41cm for distal arch; and for population >30 and <70 years of age, the diameter grows by around 0.4 mm/year. According to EACTS/STS guidelines, an aortic aneurysm is defined as 1.5x increase in its normal vessel diameter for descending and abdominal aortic aneurysms and >45 mm for the aortic root and ascending aorta [7-9].

2.2. Epidemiology

Population-based studies put prevalence of aneurysmal abdominal aortic disease affecting elderly males at 4 to 7%, while aneurysms of the thoracic aorta are less common at just 0.16 to 0.34%. Within those located in the TA only one in ten patients was observed to have an aortic arch aneurysm [1,10].

2.3. Pathophysiology

The aortic wall consists of three distinct layers - intima, media, and adventitia. Progressive weakening of those layers is prerequisite for onset and progression of the aortic aneurysmal disease. Factors underlying the degenerative process of aneurysm formation are multiple and encompass genetic variance, comorbidities, inflammation and neovascularization [1,11].

2.3.1. Genetics

There are at least 29 identified genes predisposing to thoracic aortic aneurysms (TAA). Depending on genes affected we can distinguish two subsets of familial TAAs. They can be either syndromic or non-syndromic (although there can be overlaps of the affected genes in both groups) [12]. One of the largest groups of syndromic TAAs are those connected to Loeys-Deitz syndrome in which different TGFβ/SMAD pathway proteins are affected (specific genes involved determine the LDS type). Another genetic disorder pointing towards the importance of TGF βpathway dysregulation in aneurysm formation is Marfan Syndrome in which mutated fibrillin-1 proteins disrupt sequestration of TGFβ, causing its signal to be stronger [13]. Patients with collagen mutations consistent with Ehlers-Danlos syndrome, as well as mutations of other proteins, present in connective tissues disorders such as Cutis Laxia and Meester-Loeys syndrome are also at a higher risk of developing TAA at younger age than general population. Mutations of genes (FOXE3, LOX, MAT2A, MFAP5, MYH11, MYLK, NOTCH1, PRKG1) leading to TAA formation, that fall under non-syndromic label, affect different cell types found in the arterial wall, but are not associated with a defined genetic condition [12]. Individuals suffering from Autosomal Dominant Polycystic Kidney Disease, as well to a lesser extent Turner Syndrome, and Neurofibromatosis type 1 should also be considered more prone to developing TAA than general population [14]B .icuspid aortic valve has been shown to predispose to ascending aorta dilation and abnormalities within elastic fibers of the arterial wall [15]. Although no singular gene responsible for BAV has been identified, there is evidence suggesting polygenic heritability of the malformation [16,17].

2.3.2. Comorbidities and risk factors

Although AAA and TAA are often considered just different locations of the same disease, risk factors linked to each condition differ. Hypertension has stronger association with TAA, whereas hypercholesterolemia and smoking are more related to AAA. This suggests higher relevance of physical factors, such as sheer stress in degeneration of thoracic aortic wall. Male sex, increasing age, and body surface area are of similar significance in aneurysm pathogenesis in both locations. Additionally, history of cardiovascular events, aortic dissection, aortic ulcers, and other vascular pathologies increase the likelihood of aneurysm formation in the future [1,3,18].

2.3.3. Inflammation and phenotype switching

Tissue samples of human aortic aneurysms provide substantial evidence of inflammatory cell infiltration. Aortic wall of patients suffering from TAA had a higher count of T-lymphocytes, macrophages, and NK cells as a percentage of of individual cells in overall tissue composition, conversely, the control group showed higher non-immune to immune cells ratio. Additionally, non-immune cells fount in the aneurysmal tissue had altered gene expression, most likely associated with their adaptation to stress. Smooth muscle cells (SMC) present within degenerated aortic wall had switched to proliferative phenotype, produced more ECM and showed a decrease in contractile protein production (α-smooth muscle actin, SM-specific myosin heavy chain, smooth muscle 22α, and SM-calponin). In conjunction with mechanical stress, higher local activity of proteolytic enzymes, higher ROS concentrations caused by inflammatory response, and cell apoptosis, the lowered mechanical resistance of this SMCs phenotype exacerbates aortic wall degradation [11,19].

2.3.4. Thrombus mediated neovascularization

Atherosclerosis, arterial tortuosity, and degradation of the aortic wall structure induced by the aforementioned mechanisms induce turbulent flow within the vessel, and in turn promote coagulation and intramural thrombus formation. Such thrombi act as a barrier in oxygen and nutrient exchange between intravascular fluid and aortic wall. Consequently this stimulates proliferation of vasa vasorum via VEGF. Additionally to promotion of new capillary formation, the cytokine causes an increase in MMP-2 gene expression and inflammatory cell migration into the area, which in turn aggravates connective tissue degradation and further weakens the aortic wall, causing aneurysm progression [11].

2.4. Clinical presentation

Most aortic arch aneurysms are asymptomatic and are discovered incidentally during imaging performed for unrelated reasons. The presenting symptom at the time of hospital admission, therefore, is often related to an acute, catastrophic event, most commonly the rupture or dissection of the aneurysmal segment. When this happens, the presenting clinical symptom can be the sudden onset of severe, tearing pain - most often in the chest, back, or neck - followed rapidly by signs of hypovolemic shock due to massive intrathoracic hemorrhage.

While the clinical symptoms of aortic aneurysm are often absent or minimal prior to rupture, the confined anatomical space of the mediastinum imposes physical constraints such that, with aneurysmal enlargement, patients may develop a constellation of symptoms brought about by the mechanical displacement or compression of nearby thoracic structures. Aortic arch aneurysms may cause hoarseness due to left recurrent laryngeal nerve involvement, dysphagia due to compression of the esophagus, dyspnea or stridor due to narrowing of the airway, and venous congestion of the upper body due to superior vena cava obstruction. Patients may also complain of poorly localized, dull chest discomfort most likely caused by compressive forces acting on the thoracic wall. In addition to mass effect, the clinical presentation can include distal thromboembolic events or symptoms of aortic valve insufficiency in the event the aneurysm extends proximally and distorts the aortic root [9,20].

2.5. Imaging

The gold standard for imaging TAA, preoperative planning, and the assessment of acute aortic conditions is computed tomography angiography (CTA), ideally performed with three-dimensional reformatting to enable comprehensive anatomical evaluation. A complete CTA protocol should encompass the circle of Willis, supra-aortic branches, and extend down to the femoral heads. In addition to standard arterial phase imaging, ECG-gated acquisition or dual-source CT may be employed to capture dynamic changes in the aortic wall throughout the cardiac cycle. For accurate measurement of aortic dimensions, multi-planar reconstruction (MPR) is essential, as it provides high-resolution cross-sectional views aligned with the true axis of the vessel [3,21].

Magnetic resonance imaging (MRI) offers a more comprehensive assessment of thoracic aortic function, and nature of atherosclerotic plaques compared to other modalities; however, its relatively long acquisition time restricts its utility in acute clinical scenarios. Magnetic resonance angiography (MRA) has become the de facto standard for both preoperative and postoperative evaluation, with contrast-enhanced (CE) MRI providing the most reliable imaging due to minimal flow-related artifacts. Additionally, MRI affords superior visualization of circle of Willis patency, delivering critical information regarding the risk of cerebral malperfusion – a key consideration in planning surgical or endovascular interventions involving the aortic arch [3,22].

2.6. Available surgical techniques

The first successful reconstruction of the aortic arch was performed by DeBakey and associates in 1957, a landmark event in the evolution of cardiovascular surgery. This feat established the possibility of surgical treatment for complex aortic pathology and set the stage for future developments in the field. During the next several decades, ongoing improvements in perioperative care, imaging techniques, and surgical infrastructure progressively expanded the potential for the treatment of aortic arch disease. The contemporary surgical treatment of aortic arch aneurysms has become a multidisciplinary undertaking with a diverse array of operative options based on patient-specific anatomic and clinical profiles. Consequently, the therapeutic armamentarium has increased substantially, including a range of approaches that can be chosen on the basis of risk profile, comorbidities, and disease extent [3,23].

2.6.1. Traditional open approach

Traditional open aortic arch replacement is undertaken via median sternotomy, with the patient on cardiopulmonary bypass (CPB), accompanied by systemic cooling to deep or moderate hypothermia. Cerebral protection in patients with fully patent circle of Willis is provided by antegrade selective cerebral perfusion through cannulation of the brachiocephalic trunk or right axillary artery, and in those whose anatomy does not permit effective unilateral brain perfusion, supplementary cannulation of left common carotid artery can be used. After circulatory arrest is established, the aortic arch is transected, and the diseased aorta segment is resected. Reconstruction is begun by anastomosis of a vascular prosthesis to the distal aorta, typically just beyond the origin of the left subclavian artery. The supra-aortic branches are then reimplanted into the graft either en bloc as a single island patch incorporating the brachiocephalic trunk, left common carotid, and left

subclavian arteries, or separately utilizing separate branched grafts. Proximal aortic reconstruction is then carried out by anastomosing to the ascending aorta or aortic root. The operation is completed by gradual rewarming and weaning from CPB, with attention to hemostasis and neurological stability prior to chest closure [3,24-27].

While traditional open aortic arch replacement has been accomplished successfully for many decades, a large number of patients who have aortic arch pathology also have concomitant disease of the descending thoracic aorta. In addition, the extreme invasiveness of this procedure, coupled with the elderly age and frailty of the majority of patients, makes a substantial number of patients unsuitable for open surgery. As such, modern practice has moved toward less invasive approaches, and elephant trunk, hybrid and totally endovascular methods are progressively supplanting the conventional open repair [3].

2.6.2. Elephant trunk

The elephant trunk technique (ET) varies from the traditional aortic arch replacement mainly in the nature and design of the graft implanted. In this method, a longer prosthetic graft is used, with its distal portion intentionally left unanchored and invaginated into the descending thoracic aorta - ideally extending as far distally as zone 4.

This can be accomplished either with a standard graft sewn circumferentially to the aortic wall proximal to its free-floating segment or with a specially designed graft equipped with a collar to facilitate secure positioning. This free-floating graft portion serves as a pre-positioned landing zone, simplifying future interventions. It creates an ideal proximal graft-to-graft anastomosis site for planned second-stage open thoracic aortic replacement or thoracic endovascular aortic repair (TEVAR), making it especially useful in patients with widespread multisegmental aortic pathology in need of staged repair [3,28-30].

2.6.3. Frozen elephant trunk

The next significant evolution in the surgical management of aortic arch disease after the advent of the conventional ET technique was the advent of the frozen elephant trunk (FET) procedure. The FET technique is a hybrid strategy involving open replacement of the aortic arch and endovascular treatment of the descending thoracic aorta as a single-stage procedure. In contrast to the ET technique, where a free-floating Dacron graft segment is left invaginated into the descending aorta as a future anastomotic site, the FET method involves the use of a composite prosthesis with a stented distal segment integrated with a proximal conventional vascular graft. This provides the advantage of excluding pathology in both the arch and proximal descending aorta immediately, enabling more definitive and durable repair in those with extensive or multisegmental disease. The operation is carried out through median sternotomy under CPB with moderate or deep hypothermia to enable a period of circulatory arrest for replacement of the arch. Selective antegrade cerebral perfusion is utilized routinely to maintain neurological function, most commonly accomplished by direct cannulation of the right axillary artery or innominate artery, and, if required, the left common carotid artery. Distal aortic perfusion can also be added during the procedure to augment organ protection. Following resection of the aortic arch and meticulous identification of suitable landing zones, the delivery system of the stent-graft portion of the hybrid prosthesis is introduced into the descending thoracic aorta via the open aortic arch. In most instances, a guide wire introduced via femoral artery is utilized to facilitate positioning and deployment. Fluoroscopic imaging may be utilized to ensure precise placement.

After proper alignment of the stent-graft, it is deployed in the descending aorta, where it expands to fit the vessel wall. The proximal aspect of the stented segment is then attached to the native aortic tissue with reinforced surgical suturing, a measure that eliminates the risk of proximal type I endoleak - a complication seen in purely endovascular repairs. The fabric part of the graft, which is left in the mediastinum, is then anastomosed to the ascending aorta, completing the aortic reconstruction. Depending on the surgeon's preference and anatomy, the supra-aortic vessels are reimplanted into individual branches of the graft or as an island. The FET technique not only facilitates future reinterventions by creating a stable landing zone for TEVAR but also encourages aortic remodeling, of particular value in chronic dissection or multifocal aneurysmal disease. Consequently, it has become the operation of choice in many centers for patients who need extensive arch and descending aortic repair [3,31-33].

2.6.4. Hybrid repair

Widespread adoption and refinement of total endovascular abdominal aortic repair, especially for infrarenal abdominal aortic aneurysms, have driven the development of endovascular approaches to treating more proximal aortic pathologies, including those located within the aortic arch. Nevertheless, the anatomical arrangement and inherent biomechanical forces of the ascending aorta and aortic arch present formidable obstacles to the simple application of these modalities. The ascending aorta and arch are uniquely subjected to

dynamic physiologic stress, with prominent pulsatility, cyclic bending, and high shear forces imposed by proximity to the left ventricle and the high-velocity outflow of systolic blood ejection. These forces increase the mechanical strain on endovascular devices, heightening the risk of stent-graft migration or collapse. Additionally, the complex three-dimensional curvature and branching anatomy of the aortic arch make device navigation, positioning, and fixation within the aortic arch far more complicated. Achieving a durable proximal and distal seal is particularly challenging within the arch because of the acute angulation and short segmental lengths between critical branch vessels. These limitations greatly heighten the risk of type I endoleaks and incomplete aneurysm exclusion if not fastidiously addressed. To avoid these hazards, current-generation thoracic endografts necessitate strict adherence to manufacturer-recommended landing zone lengths - often exceeding 20 mm both proximally and distally - to achieve secure fixation and avoid complications. Yet, such anatomical criteria are seldom fulfilled in the native arch without sacrificing the patency of one or more supra-aortic branch vessels. To transcend these limitations, hybrid strategies using surgical supra-aortic debranching have emerged as crucial adjuncts to endovascular arch repair. Debranching operations allow transposition and bypass of the brachiocephalic trunk, left common carotid artery, and left subclavian artery, thus increasing the length of available landing zones within 0, 1, and 2 zones of the aorta, while maintaining cerebral and upper extremity perfusion. This strategy effectively reconfigures the arch anatomy to make proper endograft deployment more feasible and decreases perioperative risk connected to more invasive approaches in high-risk surgical candidates [3,34,35].

2.6.5. Fully endovascular approach

In the endovascular treatment of aortic arch aneurysms, two primary stent-graft design strategies have emerged: fenestrated and branched endografts. These approaches aim to achieve both effective aneurysm exclusion and preservation of perfusion to critical supra-aortic branch vessels while overcoming the unique anatomical and hemodynamic challenges posed by the aortic arch. Fenestrated stent-grafts are designed with one or more precisely located openings (fenestrations) in the graft fabric, which correspond to the origins of supra-aortic vessels. These fenestrations allow for either direct alignment with the branch ostia or for bridging stents to be inserted from the fenestration into the target vessel to secure patency and prevent migration. Proper alignment during deployment is critical, and therefore fenestrated devices often require extensive preoperative imaging, advanced planning software, and sometimes custom manufacturing tailored to individual patient anatomy. The use of fenestrated grafts is typically limited to patients with relatively stable anatomy and favorable aortic arch geometry. Branched stent-grafts, on the other hand, feature integrated side branches or portals within the main aortic endograft body. These are specifically designed to accommodate additional bridging stent-grafts that are deployed retrograde through surgical or percutaneous access of the supra-aortic arteries. The branched design allows for more flexible revascularization strategies and is particularly advantageous in complex anatomies or when more than one supra-aortic vessel requires preservation. Deployment of branched systems is technically demanding and may require adjunctive debranching procedures or extracorporeal circulation in select cases. Additionally, in certain clinical scenarios where the proximal landing zone of the stent-graft lies in close proximity to a supra-aortic branch, and complete branch coverage is not necessary, a scalloped endograft design may be employed. A scallop refers to a semicircular or U-shaped indentation at the proximal edge of the stent-graft fabric, which is intentionally left uncovered to preserve blood flow through an adjacent supra-aortic vessel. Unlike fenestrations, scallops do not allow for bridging stents and are thus only appropriate when anatomical alignment can reliably maintain flow without additional support. Scalloped grafts offer a less complex alternative to fenestrated or branched configurations, particularly in cases with limited landing zone length or minimal vessel overlap.Collectively, these evolving stent-graft technologies have significantly expanded the applicability of fully endovascular approaches in aortic arch pathology. However, careful patient selection, meticulous preoperative planning, and operator experience remain critical determinants of procedural success and long-term outcomes [3,36-38].

2.6.6. Parallel grafts

Stent-graft deployment within the aortic arch poses considerable technical challenges, and despite careful planning, unintentional coverage of supra-aortic branch ostia can occur. One of the solutions to this situation is that of parallel grafting. Parallel grafts can be divided into two species - periscopes and chimneys. A chimney is a bare or covered stent deployed in between the stent-graft and the aortic wall so that its opening allows for antegrade blood flow through the chimney into the covered aortic branch. Conversely a periscope allows for retrograde blood flow from the area of the distal landing zone of the stent-graft “upwards ”towards the covered branch. A related method known as the “sandwich ”technique, involves positioning the inflow of the parallel graft between two overlapping stent-grafts, effectively “sandwiching ”the branch conduit. While

these techniques can restore flow, they come with important drawbacks. Deployment of such parallel grafts weakens the stent-graft's apposition to the aortic wall and makes type I endoleaks more likely. Additionally parallel grafting inadvertently leads to creation of gutters - small triangular gaps appearing on the tri-border of the aorta, stent-graft, and the parallel graft which additionally worsens the likelihood of leakage. The current body of evidence on the use of parallel grafting techniques in the aortic arch is limited, and clinical outcomes remain uncertain. Therefore, these approaches should only be used as the last resort [3,39].

2.6.7. In situ fenestrations

In situ fenestration is an emerging technique under investigation in the field of aortic arch pathology repair, particularly in cases where the available proximal landing zone for stent-graft deployment is inadequate. This method involves the creation of a fenestration in situ - that is, after deployment of the aortic stent-graft - by perforating the graft fabric retrogradely through the ostium of a covered supra-aortic vessel. Fenestration can be achieved using mechanical instruments or laser-based devices, allowing re-establishment of flow to the occluded branch. At present, this technique remains largely limited to bailout scenarios .While promising, broader clinical adoption of in situ fenestration requires further refinement of technique and accumulation of long-term outcome data [3].

3. Conclusions

Aortic arch aneurysms, though relatively rare, carry a high risk of mortality. Pathophysiology underlying the condition is multifaceted, disease onset and progression mostly silent, and acute rupture deadly. Prompt diagnosis and appropriate intervention are critical to improving outcomes .Advances in imaging modalities have enabled more precise preoperative planning, while the evolution of surgical techniques - from conventional open repair to hybrid and fully endovascular approaches - expanded treatment possibilities for a broader patient population. Despite these innovations, the management of aortic arch aneurysms remains technically challenging and requires a tailored approach based on anatomical considerations and patient comorbidities. Continued research and refinement of endovascular strategies may further reduce perioperative risk and extend life-saving therapies to those previously deemed inoperable.

All authors have read and approved the manuscript. The authors declare no conflict of interest.

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