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Chapter 1

Introductory Chapter: Limits of Aortic Aneurysm

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1. Introduction

The aorta, with its four-dimensional structure, is a complex organ that shows anatomical, functional, embryological, etiological, structural, pathological, dilatational, and spatial variations with limits. Geometrical inequality of the aorta in three-dimensional plane complicates measurement, diagnosis, and therapeutic interventions (open surgery, endovascular, or hybrid procedures) of pathologic segments. Fourth dimensional plane is dependent on cardiac cycles, which change functionally the form and configuration of each segment of the aorta. The requirements of this functional variation of the aorta are maintained by major components: extracellular matrix, elastin, and collagen (predominantly types I and III) [1]. During cardiac systole, the proximal aorta compensates pressure and volume loads (capacitance) and canalizes them distally during diastole through recoil (elastance). The proximal aorta is enrichment of elastic fibers and a relative paucity of collagen (elastin/collagen ratio is 2:1 in thoracal aorta), when compared with more distal aortic segments (collagen/elastin ratio is 2:1 in abdominal aorta). Upper parts of the aorta have more elastic structure to adapt this higher dynamic variation, and elastic fibers show dynamic lengthening and contraction in response to pressure and volume loads, as well as in main branches. This behavior in healthy proximal aorta absorbs and transfers kinetic energy resulted by the left ventricle to transport into the aorta-ejected blood volume toward distally, so that this energy does not cause any pathologic change (dilatation, dissection, outward compression) at the aortic wall. Collagens provide tensile strength and stiffness to the tissue to resist rupture and efficiently propagate pressure waves, especially in distal aorta and in large arteries. This resistance in healthy mid- and distal aorta adsorbs and weakens the kinetic energy carried by blood flow, so that this energy does not result mechanical complications (rupture, bleeding, inward occlusion) at the aortic wall.

The aorta shows different behavior above and below the diaphragm due to different embryologic development of vascular smooth muscular layer. Different segments of the aorta are comprised of cells originating from the neural crest, mesenchyme, and splanchnic mesoderm with a clear difference depending upon the segment. During embryologic development, the thoracic aorta above the ligamentum arteriosum (proximal thoracic aorta) consists by cells from the neural crest commonly with a constant ratio of aortic diameter to medial thickness, whereas the thoracoabdominal aorta below the ligamentum (distal thoracic and abdominal aorta) originates by cells from the mesoderm and the thickness of each unit is expanded during maturation. The media composed of concentric bands of elastin, collagen, and vascular smooth muscle cells provides viscoelasticity; on the other hand, that is the location of degradative remodeling responsible for aneurysm formation. The proximal aorta formed by neural crest is vulnerable against genetic malformation and degradation, which conduct primarily luminal enlargement of the aorta. Proximal aortic dilatation characterizes usually with true aneurysm formation including all aortic wall layers without rupture and clots. The distal aorta formed by mesenchyme is inclined to atherosclerotic changes, which induce first luminal narrowing of the aorta. Distal aortic dilatation characterizes often with pseudoaneurysm formation devoid of all aortic wall layers with rupture and clots.

The etiology of aneurysmal development is often nonatherosclerotic (genetic) in the thoracic aorta, but atherosclerotic in the abdominal aorta [2, 3]. Nonatherosclerotic aneurysm includes inflammation and degeneration. Especially, different aortitis (Takayasu, giant cell arteritis, isolated thoracic arteritis, Behçet disease, syphilis, rheumatic aortitis, etc.) causes granulomatous inflammation with coagulative necrosis and elasticophagia, intimal proliferation, and obliteration without fibrinoid necrosis of the vasa vasorum, and aneurysmal formation if fibrosis is delayed [4]. Genetic disorders cause weakness of the tissues due to loss of structural integrity via specific gene mutations (Marfan syndrome, Loeys-Dietz syndrome, etc.) resulting in cystic medial necrosis, elastic fiber fragmentation, contractile dysfunction, and collagen deposition [5]. Atherosclerotic aneurysm includes atheroma, calcifications, penetrating ulcers, and subadventitial pseudoaneurysms. When atheromatous lesion extends into the media with loss of elastic fibers, the aortic wall is thinned and results in aneurysmal formation. Dissection of blood between intima and media at the ulcer area causes associated psuedoaneurysmal dilatation. Inflammatory aneurysm is characterized by lipids or products of lipid oxidation in the aortic adventitia, extensive adventitial thickening, medial and adventitial fibrosis via lymphoplasmacytic infiltrate, and adhesion to the surrounding retroperitoneal structures.

Aortic aneurysm is a well-known pathology, which can be limited in a segment or spread whole the thoracoabdominal aorta with or without its major branches. Excessive enlargement causes compression symptoms and signs, but its fatal progress is dependent on mechanical complications such as rupture, dissection, fistulization, occlusion, etc. Because aortic aneurysm often has a silent clinical nature until fatal rupture (silent killer), no patient can have time to arrive to hospital when aneurysm is acutely ruptured; however, chronic healing of mechanical complications of aortic aneurysm can give a chance to be diagnosed and treated surgically or endovascularly. The prevalence of aortic aneurysm (>5 cm) is 0.16–0.34% in the general population and the annual incidence of first detected aortic aneurysm (>5 cm) is 5–10 patients/100,000 people (0.005–0.01%), which increases with time and age. At this decade, aortic aneurysm has

increased up to 19th leading cause of death (0.64% of the first 20 reasons) in all age groups, but it has reached up to 15th leading pathology of mortality (0.69% of the first 20 reasons) in individuals over 65 years [6]. On the other hand, the true ratio of aortic aneurysm resulting in sudden death must be higher than expected or recorded due to false classification as an acute coronary syndrome (estimated between 2 and 7.5%). That is the fact that aortic aneurysm has an increasing appearance rate in the last decades due to *de novo* cases and/or improvement of imaging modalities. Preventive medicine has a critical role for early diagnosis and reducing of the prevalence in general population. Preventive screening of population with positive family history, profusely predictors, and advanced age provides early diagnosis in asymptomatic patients with an unpredictable, but acceptable devastation of health budgets; whereas periodically screening of diagnosed patients is the gold standard to follow-up patients and to decide initiative time. In spite of excessive improvements in medical technology, health-care policy, interventional therapy techniques, and medical follow-up provide successfully results after aortic aneurysm repair. Sudden death is still the main risk and complication of aortic aneurysms. Avoidance from atherosclerotic risk factors, continuance of physical activity, and maintenance of healthy lifestyle are key points of preventive medical care.

There are two major spatial aneurysm types (**Table 1**). Thoracic aortic aneurysm (TAA) develops in all age groups and is not associated with cardiovascular risk factors, but it can occur as an isolated pathology or it can be associated with different pathologies (**Table 2**) [7]. Most of them are presented syndromic (part of a systemic connective tissue disorder) or nonsyndromic (part of a sporadic structural disorder). The aneurysmal dilatation of the proximal thoracic aorta can be often dependent on a genetic connective tissue disorder with mostly autosomal dominant inheritance or other types, which acts as multisystemic syndrome. The structural malformation of the aortic valve is a common situation, especially bicuspid aortic valve that increases the annular growing speed at least 50% than the normal tricuspid aortic valve. Intracranial aneurysm is the most important concomitant arterial dilatation, which may result in permanent or

1. Thoracic aortic aneurysm
- **a.** Sinus of Valsalva aneurysm
- **b.** Aortic root aneurysm
- **c.** Ascending aorta aneurysm
- **d.** Aortic arch aneurysm
- **e.** Descending aorta aneurysm
- **f.** Combined

2. Abdominal aortic aneurysm
- **a.** Suprarenal
- **b.** Infrarenal
- **c.** Combined
- **d.** Integrated (with iliac arteries)

3. Thoracoabdominal aortic aneurysm
- **a.** Type I (from LSA to CA)
- **b.** Type II (from LSA to IB)
- **c.** Type III (from sixth intercostal space to IB)
- **d.** Type IV (from subdiaphragmatic segment to IB)
- **e.** Type V (from sixth intercostal space to RA)

CA, celiac axis; IB, iliac bifurcation; LSA, left subclavian artery; RA, renal artery.

Table 1. Spatial variations of aortic aneurysms.

1. Intracranial aneurysm (up to 9%)
2. Multisegment involvement of the thoracoabdominal aorta (up to 20–25%)
3. Aortic arch anomalies (up to 20%)
  a. Bovine aortic arch (double carotid branching from the brachiocephalic trunk)
  b. Isolated left vertebral artery
  c. Aberrant right subclavian artery
4. Bicuspid aorta (up to 20–80%)
5. Familial history (up to 20%)
6. Several genetic transition signs (thumb-palm sign)
7. Temporal arteritis
8. Simple renal cyst, polycystic kidney
9. Inguinal hernias

Table 2. Associated pathologies with thoracic aortic aneurysms.

temporary neurologic events, and adversely affect the natural prognosis or surgical outcomes of TAA. Multilevel involvement of the whole aorta is not uncommon and combined aneurysmal enlargement of two different aortic segments worsens fatal risks or surgery. Aortic arch anomalies do not affect the natural course, but they can complicate the pathologic course after complications or surgical repair of TAA. Abdominal aortic aneurysm (AAA) is usually isolated, but it can be associated with TAA in 10–15% of cases. The etiology is different and multifactorial, which cannot affect thoracal aortic segments (**Table 3**) [8]. Smoking and familial history are the main predictors, but also several genetic and inflammatory factors can cause this pathology [9]. Other vascular aneurysms, especially involvement of iliac arteries, are not seldom due to spreading directly or separately [10]. Increased cardiovascular risk factors enforce combined surgical intervention for coronary artery disease [11]. The basic presentation of AAAs is a pseudoaneurysm developed after rupture or perforation of aneurysm sac, which wraps the pathologic segment and supports with attached surrounding tissues against fatal hemorrhage.

Aortic aneurysms grow very slowly during life and overall growth rate is 0.12 cm/year in adults, where it is slower at the ascending aorta (0.1 cm/year) than those at the descending aorta (0.3 cm/year) [12]. As a large elastic artery, definition of aneurysmatic development of the aorta is an enlargement greater than 1.5 times the expected threshold diameter, which are 4 cm for the suprarenal aorta and 3 cm for the infrarenal aorta. The dilatation reaches a hinge diameter in the whole aorta at which the rate of mechanical complications (rupture, dissection) increases exponentially. This borderline diameter is 6 cm for the ascending aorta and 7 cm for the descending aorta. These limits can be used in asymptomatic patients as cut-

1. Smoking
2. Advance age
3. Male
4. Family history
5. Atherosclerosis
6. Hypertension
7. Hypercholesterolemia
8. Other vascular aneurysm

Table 3. Risk factors for abdominal aortic aneurysms.

off value for surgery, but we take down both limits below 6 cm because of 95% of asymptomatic patients have death as a first symptom and we accept the upper limit 5.5 cm in patients without and 5 cm with a risk factor such as Marfan syndrome, bicuspid aortic valve, and family history of sudden death. On the other hand, any symptom caused by aneurysm dilatation (continue retrosternal pain for ascending or interscapular back pain for descending aneurysms) is the cut-off value for surgical treatment regardless of size.

Surgical repair of aortic aneurysms is the definitive treatment procedure with an acceptable hospital mortality and morbidity rates, complication-free long-term survival, and lesser requirement of any reintervention [13–17]. In spite of surgical treatment of aortic aneurysms based on open approaches, endovascular repair has gained widespread acceptance due to percutaneous applicability and is considered the first treatment option in the last years [18]. Open

1. Mortality
2. Stroke
3. Spinal cord injury
4. Renal insufficiency
5. Pulmonary insufficiency

Table 4. Major complications of surgical repair of aortic aneurysms.

conventional surgery remains the standard treatment, but technical complexity increases with more extensive dissection, higher clamp site, prolonged visceral ischemia, and more extensive reconstruction [19]. Major complications are the main drawbacks and hesitations of surgical repair (**Table 4**). To improve intraoperative and early postoperative outcomes, endovascular approaches for aortic aneurysms have evolved and revealed that total endovascular repair with or without hybrid surgical procedures allows reducing operative mortality and morbidity as well as simplifying the intervention and avoiding from complicated steps such as aortic cross-clamping, thoracotomy, single-lung ventilation, and prolonged ischemic time. On the other hand, the presence of several risk factors continues to limit the application of endovascular approaches (**Table 5**). Low- and high-risk patients are not true candidates for hybrid procedure due to high rates of mortality and morbidities and procedural complica-

1. Large aortic seals
2. Short neck (<2 cm)
3. Severe calcified aorta
4. Previously thoracic and/or abdominal surgery
5. Involvement of the visceral arteries
6. Limited life expectancy
7. High-risk patients

- **a.** Unstable angina, malign arrhythmia, recurrent congestive heart failure
- **b.** Left ventricular dysfunction (LVEF < 25%)
- **c.** Vital capacity < 1.8 L
- **d.** Resting pO_2 < 60 mmHg and pCO_2 > 50 mmHg
- **e.** Serum creatinine > 2.5 mg/dL

LVEF, left ventricular ejection fraction; pO_2, oxygen partial pressure; pCO_2, partial pressure of carbon dioxide.

Table 5. Factors complicating endovascular interventions.

tions, but this approach is useful in patients who require creation of definitive landing zone [20]. Another frequent choice of endovascular repair is ruptured AAAs due to easier and more noninvasive nature of the procedure, whereas open surgical treatment has similar or better outcomes [21].

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