

Review Article

Aneurysms: A Comprehensive Review

H.S Natraj Setty*, Raghavendra Murthy P, Rahul Patil, Shivanand S Patil, J.R Vijay Kumar, and C.N Manjunath

Sri Jayadeva Institute of Cardiovascular Sciences and Research, India

***Corresponding author**

HS Natraj Setty, Assistant Professor of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore, Karnataka, India, Tel: 9845612322; Fax: 080-22977261; Email: drnatrajsetty75@gmail.com

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OPEN ACCESS**Abstract**

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in basic to clinical and surgical approach, treatment and drug therapy are required. Health and Medicine emphasizes holistic approaches to understanding and preventing disease. A wide range of health care and surgical interventions is explored. Aneurysms are common manifestations in elderly patients especially with high blood pressure and arteriosclerosis. Rupture of an aortic aneurysm is a catastrophic event associated with a very high mortality. The etiological factors of aneurysms are not fully understood although some modifiable risk factors have been identified. Various arteries can be affected by aneurysms including peripheral arteries. Not all aneurysms need treatment but some can be monitored while risk factors are managed. Many aneurysms are asymptomatic, but ruptured aneurysms do produce life threatening bleeding, dissection that needs emergency hospital care. To review current literature of aneurysmal disease surveillance guidelines and management options.

Keywords

- Aneurysms
- High blood pressure
- Arteriosclerosis

INTRODUCTION

Aneurysmal degeneration can occur anywhere in the human aorta. By definition, an aneurysm is a localized or diffuse dilation of an artery with a diameter at least 50% greater than the normal size of the artery. Most aortic aneurysms (AAs) occur in the abdominal aorta; These are termed abdominal aortic aneurysms (AAAs) [1]. Although most AAAs are asymptomatic at the time of diagnosis. Aneurysmal degeneration that occurs in the thoracic aorta is termed a thoracic aortic aneurysm (TAA). Aneurysms that coexist in both segments of the aorta (thoracic and abdominal) are termed thoracoabdominal aneurysms (TAAAs) [2]. Aortic aneurysms are a complex multifactorial disease with genetic and environmental risk factors. Genetic factors have been shown to play a role in the etiology of TAA and AAA even when they are not associated with Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome, or other rare aortic syndromes. Aneurysm is the most common condition of the thoracic aorta that requires surgical treatment. This category includes congenital or developmental, degenerative, chronic traumatic, inflammatory, infectious, mechanical, and anastomotic aneurysms. Advancements in open surgical technique and the advent of endovascular treatment have supported a significant improvement in outcomes and survival for many with descending thoracic and thoracoabdominal aortic aneurysms. Since the inception of successful thoracic and thoracoabdominal aortic reconstruction by Etheridge and DeBakey in 1955 and 1956 [3]. Thoracoabdominal aortic aneurysms may be classified according to the anatomic extent of disease as proposed by the Crawford

classification system: type (or extent) I (24%) involving the proximal descending thoracic to proximal abdominal aorta, type II (26%) involving the proximal descending thoracic to infrarenal abdominal aorta, type III (26%) involving the distal descending thoracic and abdominal aorta, and type IV (24%) involving the abdominal aorta and including visceral vessel segments [4]. Principles of endovascular aortic treatment : The principles around endovascular treatment of the aorta involve the insertion of a stent graft across the diseased aorta, bridging two normal segments, the so-called proximal and distal landing zones. The stent graft is a metal meshwork tube covered by a material which relines the aorta. This facilitates the passage of blood through a conduit away from the diseased segment, thus reducing wall pressure, shear stress and subsequent disease progression.

EPIDEMOLOGY

Findings from autopsy series vary widely; the prevalence of aortic aneurysms probably exceeds 34% in individuals older than 65 years. Death from aneurysmal rupture is one of the 15 leading causes of death in most series. The estimated incidence of thoracic aortic aneurysms is 6 cases per 100,000 person years. In addition, the overall prevalence of AAs has increased significantly in the past 30 years. This is partly due to an increase in diagnosis based on the widespread use of imaging techniques. However, the prevalence of fatal and nonfatal rupture has also increased, suggesting a true increase in prevalence. Population based studies suggest an incidence of acute aortic dissection of 3.5 per 100,000 persons; An incidence of thoracic aortic rupture of 3.5 per 100,000 persons; And an incidence of abdominal aortic

ruptures of 9 per 100,000 persons. An aging population probably plays a significant role [5,6].

ETIOLOGY

Aneurysmal degeneration occurs more commonly in the aging population. Aging results in changes in collagen and elastin, which lead to weakening of the aortic wall and aneurysmal dilation. Pathologic sequelae of the aging aorta include elastic fiber fragmentation and cystic medial necrosis. Arteriosclerotic (degenerative) disease is the most common cause of thoracic aneurysms. False aneurysms are more common in the descending aorta and arise from the extravasation of blood into a tenuous pocket contained by the aortic adventitia. Authorities strongly agree that genetics play a role in the formation of aortic aneurysms. This appears especially true in first degree relatives of female patients with aortic aneurysms. Thus, inherited disorders of connective tissue appear to contribute to the formation of aortic aneurysms. Marfan syndrome is a potentially lethal connective tissue disease characterized by skeletal, heart valve, and ocular abnormalities. Patients with Marfan syndrome may develop annuloaortic ectasia of the sinuses of Valsalva, commonly associated with aortic valvular insufficiency and aneurysmal dilation of the ascending aorta. Type IV Ehlers Danlos syndrome results in a deficiency in the production of type III collagen, and individuals with this disease may develop aneurysms in any portion of the aorta. Imbalances in the synthesis and degradation of structural proteins of the aorta have also been discovered, which may be inherited or spontaneous mutations. Atherosclerosis may play a role. Whether atherosclerosis contributes to the formation of an aneurysm or whether they occur concomitantly is not established. Other causes of aortic aneurysms are infection (i.e., bacterial [mycotic or syphilitic]), arteritis (i.e., giant cell, Takayasu, Kawasaki, Behçet), and trauma. Aortitis caused by syphilis may cause destruction of the aortic media followed by aneurysmal dilation. Traumatic dissection is a result of shearing from deceleration injury due to high speed motor vehicle accidents (MVA) or a fall from heights. The true etiology of aortic aneurysms is probably multifactorial, and the condition occurs in individuals with multiple risk factors. Risk factors include smoking, chronic obstructive pulmonary disease (COPD), hypertension, atherosclerosis, male gender, older age, high body mass index (BMI), bicuspid or unicuspid aortic valves, genetic disorders, and family history [2].

INDICATIONS FOR SURGERY

Because of the risk of rupture, all patients with thoracic or thoracoabdominal aortic aneurysms are considered for surgical repair, taking into careful account the patient's overall health [7,8]. Asymptomatic patients with an aneurysm diameter that is twice the size of a normal, contiguous segment of aorta or greater than 6 cm should be evaluated for surgery. Patients with symptomatic aneurysms should undergo urgent surgical repair, even if smaller in size. Patients with evidence of progressive aneurysm enlargement should be offered an operation unless they have a prohibitively high operative risk or some other major medical problem limiting their life expectancy. Those with smaller asymptomatic thoracic or thoracoabdominal aortic aneurysms are followed with serial CT or MRI scans. Currently, surveillance-

imaging studies are performed at 6-month intervals, unless the aneurysm is approaching 6 cm in size, at which point these studies are done more frequently prior to surgical intervention [9,10]. Traditional open surgery using an inlay graft has been the 'gold standard' treatment for AAAs for over 50 years. Although the technical success of this operation has been fully optimised, it has been associated with mortality rates of up to 10% [11]. Typically, open repair is offered to young patients, patients who are anatomically unsuitable for EVAR, patients who are unable to comply with the lifelong postoperative surveillance necessary following EVAR. EVAR involves accessing the aneurysm from a peripheral vessel (usually the femoral artery), and placing an endograft inside the aneurysm to exclude it from the circulation. The principal advantage of EVAR centres on its significantly lower perioperative mortality and morbidity, [12] Thoracic endovascular repair (TEVAR) represents a valid treatment choice for thoracic disease, particularly those involving the descending aorta.

Advances in endovascular technology have addressed the challenges of the thoracic aorta by designing custom-made TEVARs that extend the length of the proximal landing zone, while maintaining flow to the great arch vessels aortic branches. These techniques include scalloped TEVAR (S-TEVAR), fenestrated TEVAR (F-TEVAR) and branched TEVAR (B-TEVAR). S-TEVARs are custom-made stent grafts with a scallop designed to accommodate the origin of a supra-aortic vessel which would otherwise be covered. F-TEVARs work on the same principle as F-EVARs and are stents grafts that are designed with customised carefully planned holes within the fabric, that allow perfusion of the great arch vessels. These are then catheterised and secured with stents to prevent movement of the fenestrations. B-TEVARs are an extension to the concept of F-TEVARs, but unlike F-TEVARs they are custom-built with stent graft branches that can be used to facilitate cannulation of the great vessels. S-TEVARs are becoming accepted for the treatment of thoracic aortic aneurysms with a short proximal landing zone [13] with encouraging short and mid-term results. F-TEVARs and B-TEVARs also look encouraging with short and mid-term reports of high technical success rate and lower morbidity and mortality, especially when compared to redo open aortic surgery. Long-term outcome data are required to establish wider use of modified thoracic endografts. Other surgical and hybrid endovascular methods have been described for treating challenges aortas, such as endovascular sealing, chimney and periscope techniques [14] (Figure 1-4).

TREATMENT

Treatment of AAAs, TAAAs, and TAAs involves surgical repair in good risk patients with aneurysms that have reached a size sufficient to warrant repair. Surgical repair may involve endovascular stent grafting (in suitable candidates) or traditional open surgical repair [15]. Most of these initial successful repairs involved the use of preserved aortic allografts, thus triggering the establishment of numerous aortic allograft banks. Simultaneously, Gross et al successfully used allografts to treat complex thoracic aortic coarctations, including those with aneurysmal involvement [16]. In 1951, Lam and Aram reported the resection of a descending thoracic aneurysm with allograft replacement [17]. Ascending aortic replacement required the development



Figure 1 CT aortogram showing marked dilation involving sino tubular junction, ascending aorta.



Figure 2a Aortogram showing Thoroco abdominal stenosis.

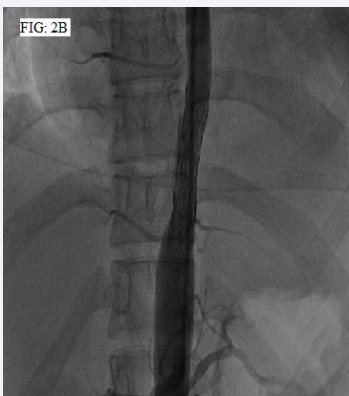


Figure 2b Aortogram showing post PTA with stent.

of cardiopulmonary bypass and was first performed in 1956 by Cooley and De Bakey [18]. although the use of aortic allografts as aortic replacement was widely accepted in the early 1950s, the search for synthetic substitutes was well underway. Dacron was introduced by De-Bakey. By 1955, Deterling and Bhonslay believed that Dacron was the best material for aortic substitution [19]. Numerous types of intricately woven hemostatic grafts have

since been developed and are now used much more extensively than their allograft counterparts. Such Dacron grafts are used to replace ascending, arch, thoracic, and thoracoabdominal aortic segments. Subsequently, combined operations that replaced the ascending aneurysm in conjunction with replacement of the aortic valve and re-implantation of the coronary arteries were performed by Bentall and De Bono in 1968, using a mechanical valve with a Dacron conduit [20]. Ross, in 1962, and Barratt Boyes, in 1964, successfully implanted the aortic homograft in



Figure 3a Aortogram showing descending thoracic aortic aneurysm.



Figure 3b Aortogram showing post Thoracic Endovascular Aortic Repair (TEVAR).

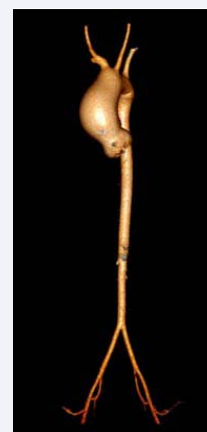


Figure 4a CT Aortogram showing aortic root and proximal ascending aortic aneurysm.



Figure 4b CT Aortogram showing infrarenal abdominal aortic aneurysm.



Figure 4c CT Aortogram showing isolated aortic root aneurysm.

the ortho topic position [21]. In 1985, Severs reported the use of stentless porcine aortic roots [22]. More recently, less invasive therapy for descending thoracic aortic aneurysm have been developed. Dake et al., reported the first endovascular thoracic aortic repair in 1994. In March 2005, the US Food and Drug Administration (FDA) approved the first thoracic aortic stent graft, the GORE TAG graft [23]. Since 2005, 2 other devices have gained FDA approval: the Talent Thoracic endograft and the Cook TX2 endograft.

CONCLUSION

Aortic aneurysms are an important cardiovascular disease, particularly in the aging population. They are a complex disease with both genetic and environmental factors contributing to the disease process, which involves formation, growth, and rupture. Aneurysms are often silent without symptoms until rupture occurs, but they can be detected effectively via imaging techniques. First degree relatives of aneurysm patients have an increased risk of the disease, and it is therefore important to offer appropriate advice to these individuals and counsel them to seek screening options. Current surgical treatments give excellent results; there is a need to develop nonsurgical approaches to manage small aneurysms.

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