

## Chapter 25

# Thoracic Aortic Disease

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### ABSTRACT

*Thoracic aortic disease most commonly presents in the form of aneurysmal dilation or dissection of the ascending or descending thoracic aorta, most commonly secondary to degenerative disease and hypertension. Several genetic connective tissue disorders are commonly associated as well. Treatment is focused on blood pressure control and surgical repair. Surgical repair of thoracic aortic disease presents unique risks, including neurologic injury involving the brain or spinal cord, and several adjuncts are available to mitigate against this risk. This chapter discusses commonly associated risk factors, preoperative testing, surgical repair, and postoperative management of thoracic aortic disease along with strategies for minimizing neurologic injury.*

### INTRODUCTION

With current cardiopulmonary bypass techniques, surgery for thoracic aortic disease has become much more common, with low morbidity and mortality rates. Ascending aortic aneurysm repair can be performed with ~2% operative mortality rate (Cohn et. al. 1996). Increasingly complex procedures on the aortic root, including replacement with a valved conduit with coronary reimplantation and valve sparing root replacement are performed with similar outcomes. Aortic arch aneurysms requiring a period of circulatory arrest for repair pose additional neurologic risks, and several adjuncts to standard cardiopulmonary bypass techniques including deep hypothermia, antegrade, and retrograde cerebral perfusion, have been utilized to reduce stroke risks. Similarly, descending thoracic aortic aneurysm repair is associated with increased paraplegia risk due to spinal cord ischemia and again a variety of techniques to reduce this risk

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are similarly available. The decision to undertake repair requires an assessment of the risks of surgery versus those of rupture or dissection if observation is chosen. As operative risks decrease, this balance is shifted in favor of earlier operation.

## **BACKGROUND**

Aortic aneurysms are most commonly degenerative in nature, secondary to cystic medial necrosis in which there is a loss of elastic tissue in the aortic wall. Vascular myocytes in the media undergo necrosis, and are replaced by cystic accumulations. Risk factors are similar to those of atherosclerosis, including smoking, hypertension, hypercholesterolemia, and diabetes. Connective tissue diseases such as Marfan, Loewy-Dietz, and Ehlers-Danlos syndromes, bicuspid aortic valve disease, and certain infections also predispose to aneurysm formation.

Marfan syndrome is an autosomal dominant disease caused by mutation of the gene encoding fibrillin-1, a glycoprotein component of the extracellular matrix. Clinical features most commonly include aortic root dilation along with dilation and dissection of the remaining aorta, mitral valve prolapse, ocular and orthopedic manifestations, and spontaneous pneumothorax. These patients are prone to more rapid growth, rupture, and dissection of their aneurysms. A positive family history and pregnancy may increase this risk, and beta blocker therapy significantly reduces it. Most patients eventually die from complications of aortic aneurysm disease (Pomianowski & Elefteriades 2013).

Loeys-Dietz syndrome is an autosomal disease caused by mutation of the gene encoding the transforming growth factor beta receptor, and may present in a fashion similar to Marfan syndrome, although with heightened severity. Patients commonly demonstrate the triad of hypertelorism, bifid uvula, and widespread arterial aneurysm formation. Aneurysm formation may occur at an earlier age than in Marfan syndrome (Pomianowski & Elefteriades 2013).

Ehlers-Danlos syndrome is an autosomal dominant disease caused by mutation of the gene encoding type III collagen. Clinical manifestations include skin hyperelasticity and fragility leading to easy bleeding, joint hypermobility, aneurysms of medium and large sized vessels prone to rupture, and visceral perforation. Again, most patients eventually die of from arterial aneurysm rupture (Pomianowski & Elefteriades 2013).

Bicuspid aortic valve is the most common congenital cardiac malformation, occurring in 1-2% of the population. This entity appears to have a familial association, although no inheritable mutation has been identified. The bicuspid aortic valve may function normally, but is prone to stenosis or regurgitation. In addition, these patients have a poorly understood propensity to ascending and aortic root aneurysm and dissection formation (Verma & Siu 2014).

Mycotic aneurysms, most commonly from syphilis infection, is now uncommon with current antimicrobial therapy. The ascending aorta is most commonly involved, with destruction of the media and saccular aneurysm formation (Lopes, Almeida, Dias, Pinho, & Maciel 2008). Inflammatory aortitis is most commonly secondary to giant cell or temporal arteritis. Granulomatous inflammation of the media involving medium and large sized arteries can lead to aneurysm formation. Takayasu's arteritis most commonly leads to stenosis of the aortic arch and its branches, but may lead to aneurysm formation. Several other autoimmune inflammatory conditions may also lead to aortic aneurysm formation (Pagni, Ganzel, Williams, Slater, & Trivedi 2012).

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