Ascending Aortic Aneurysm

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Introduction

Definition

True aortic aneurysm is commonly defined as a localized, permanent aortic dilation diameter of 50% or greater than normal, and is contained by all the layers of the normal aortic wall [1]. False aortic aneurysm is a focal dilation that consists of adventitia, some or all of the media, as well as compressed periaortic tissue, and is most frequently seen in traumatic aortic injury.

Historical Note

Denton Cooley and Michael De Bakey reported the first reported modern surgical repair of an aortic aneurysm in 1952, which described the lateral resection of a descending aortic saccular aneurysm without cardiopulmonary bypass [2]. Four years later in 1956, Cooley and De Bakey performed a replacement of the ascending aorta with a homograft with cardiopulmonary bypass [3]. Polyester conduits were introduced by De Bakey and quickly became the material of choice for artificial conduits. In 1964, Myron Wheat Jr. and colleagues resected the ascending aorta and aortic root while leaving aortic tissue around the coronary ostia, followed by the insertion of a mechanical valve tailored to accommodate the in situ coronary arteries [4]. The first composite aortic root repair with an aortic graft with attached valve was described in a patient with Marfan syndrome by Hugh Bentall and Antony De Bono in 1968 [5]. Further technical advances were developed by Christian Cabrol and colleagues, as well as Nicholas Kouchoukas and Robert Karp who described the modern technique that comprises individual coronary button reimplantation with end-to-end anastomosis [6, 7]. Valve-sparing aortic root replacement techniques, including a remodeling technique described by Sir Magdi Yacoub and a reimplantation technique developed by Tirone David, are currently performed in specialized centers.

Surgical Anatomy

The aortic root is located between the left ventricle and ascending aorta, and is an extension of the left ventricular outflow tract containing the aortic valve. The aortic root contains four distinct anatomic components: the aortic annulus and subcommissural triangles, the aortic valve cusps, sinuses of Valsalva, and the sinotubular junction (Fig. 11.1). The aortic annulus is a combination of fibrous and muscular tissue that attaches the aortic valve to the left ventricle, with approximately 55% of the circumference comprising of fibrous attachments to the anterior leaflet of the mitral valve and membranous septum, and the remaining 45% containing muscular attachment directly to the myocardium [8]. The aortic valve normally has three semilunar-shaped cusps, containing a base and a free margin, that attach to the aortic annulus forming three commissures. The area between the aortic cusps and the superior commissure are known as the subcommissural triangles.

The superior aspects of the commissures interrelate with the sinotubular junction, which is a ridge that marks the

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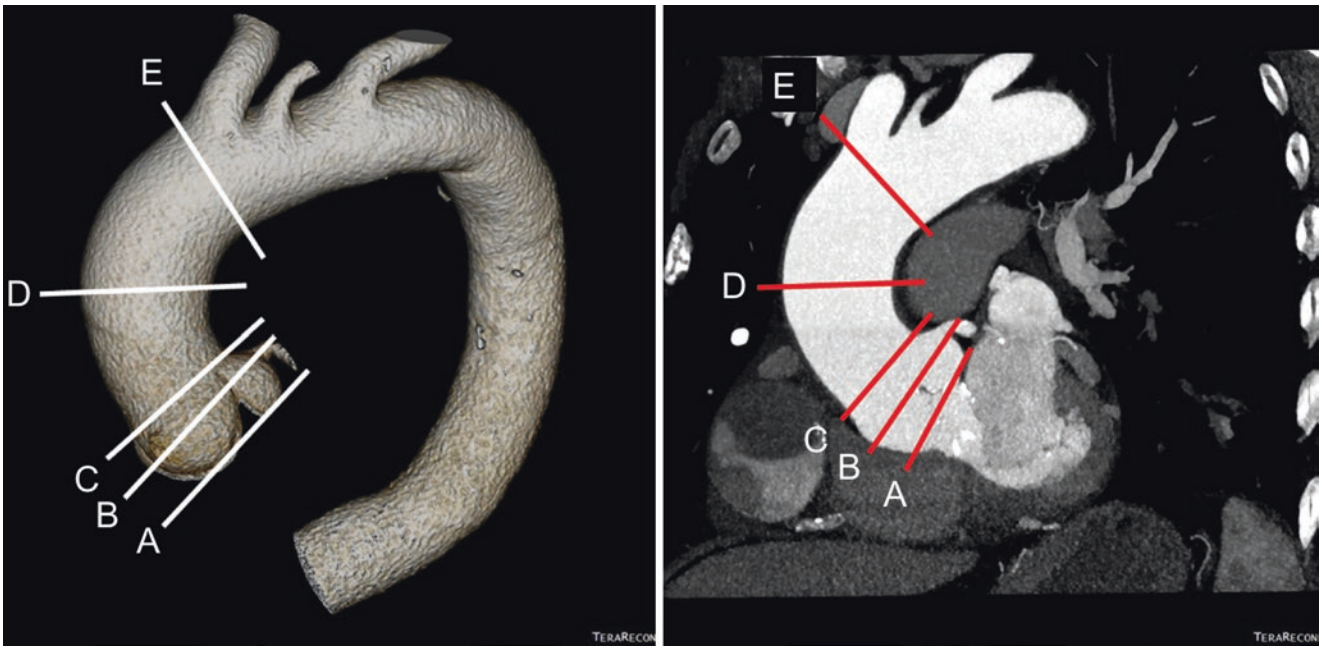

Fig. 11.1 Anatomy of the aortic root and ascending aorta (mean dimension (cm), male and female). A: aortic annulus (2.6 ± 0.3 and 2.3 ± 0.2), B: sinuses of Valsalva (3.4 ± 0.3 and 3.0 ± 0.3), C: sinotubular junction (2.9 ± 0.3 and 2.6 ± 0.3), A–C: aortic root, D: mid ascending aorta (3.0 ± 0.4 and 2.7 ± 0.4), E: aortic arch [12]

origin of the ascending aorta [9]. Slight dilation of the sinotubular junction relative to the aortic annulus frequently results in aortic insufficiency as the aortic valve cusps no longer coapt centrally. The three sinuses of Valsalva, sometimes referred to as the aortic sinuses, are located between the aortic annulus and the sinotubular junction and are associated with corresponding aortic valve cusps—the left cusp and sinus containing the ostium of the left main coronary artery, the right cusp and sinus containing the ostium of the right coronary artery, and the noncoronary cusp and sinus. The subcommissural triangles bordering the noncoronary associate with the anterior leaflet of the mitral valve, and the subcommissural triangle between the right and noncoronary sinuses mark the conduction system within the membranous septum. The relative dimensions of the three cusps are variable; however, the right and noncoronary cusps are typically larger than the left cusp [10, 11]. Larger cusps have a proportionately large annulus, sinus, and sinotubular junction. Men on average have slightly larger aortic root dimensions than women.

A study of two-dimensional (2D) echocardiographic aortic root dimensions found the mean aortic root dimensions (cm) in males and females to be 2.6 ± 0.3 and 2.3 ± 0.2 at the annulus, 3.4 ± 0.3 and 3.0 ± 0.3 at the sinuses of Valsalva, 2.9 ± 0.3 and 2.6 ± 0.3 at the sinotubular junction, and 3.0 ± 0.4 and 2.7 ± 0.4 at the proximal ascending aorta, respectively [12]. The thoracic aorta normally decreases in diameter from the aortic root distally to the diaphragmatic descending thoracic aorta. Measurement of the ascending aorta in males using computed tomography was utilized to report a mean diameter of the root, ascending, mid-descending, and diaphragmatic aorta to be 3.63, 2.85, 2.39, and 2.43 cm, respectively [1].

Epidemiology

The prevalence of ascending aortic aneurysms has been historically difficult to ascertain due to large amount of cases that go undiagnosed, as well as an underreporting in mortality statistics. Literature investigating the epidemiology of ascending aortic aneurysms is scarce. Historically, ascending aortic aneurysms were more prevalent than thoracoabdominal aortic aneurysms in the first half of the twentieth century due to increased prevalence of syphilis; however, thoracoabdominal aortic aneurysms are now more common after the advent of antibiotics [7]. A study examining thoracic aortic disease from 1987 to 2002 in Sweden, including ruptured and nonruptured thoracic aneurysm as well as acute and chronic aortic dissection, found the prevalence of aortic disease to be 16.3 per 100,000 for men and 9.1 per 100,000 for women in 2002 [13]. The incidence of aortic dissection was estimated to be six per hundred thousand persons per year in the Oxford Vascular study [14]. A trend in increased prevalence of aortic disease may be partially attributable to improved imaging techniques and screening.

Etiology and Pathophysiology

Pathology

The wall of the ascending aorta is comprised of three layers: the intima, media, and adventitia. The intima is a fragile layer of endothelial cells on a thin basal membrane. The media is a thick layer that contains an extracellular matrix containing elastin sheets, collagen, and smooth muscle. The vasa vasorum, which is a network of small blood vessels that supplies nutrients to the media, is located in the thin fibrous adventitia. The aorta contains a high degree of elastic tissue, which allows for the aorta to expand and contract, minimizing the physiologic stress of pressure shifts during the cardiac cycle. The ascending aorta has approximately twice the elastin content as the abdominal aorta and is more susceptible to age-related degeneration [15]. There are many known causes of aortic aneurysm formation, which are commonly separated into groups including age-related degeneration, congenital syndromes such as bicuspid aorta disease, Marfan syndrome, Ehlers–Danlos syndrome and Loeys–Dietz Syndrome, and infectious and noninfectious inflammatory syndromes (Fig. 11.2).

Degenerative

Degeneration of the elastic media is the most common biologic cause of aneurysm formation, and is caused by fragmentation of the extracellular matrix of the media by matrix metalloproteinases and cathepsin groups [16, 17]. As elastic layers fragment, smooth muscle cells become dysfunctional and are eventually replaced with cystic appearing mucoid material, known as cystic medial degeneration [18]. The Young–Laplace equation explains the relationship between aortic diameter and wall stress and comparable blood pressures (wall tension = pressure × radius) and helps to explain the progressively increasing rate of aortic dilation in aneu-

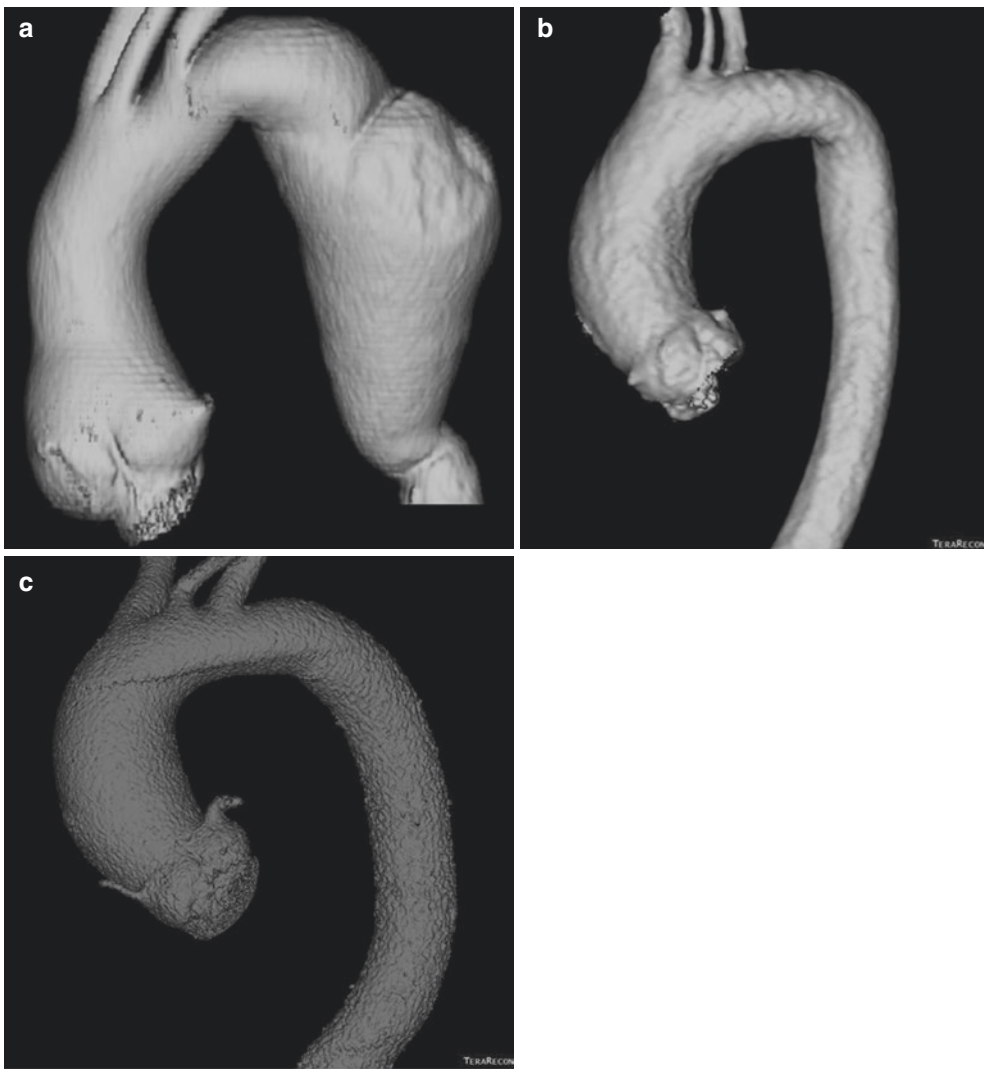

Fig. 11.2 Ascending aortic aneurysm caused by congenital syndromes. (**a**): Marfan syndrome. (**b**): Loeys–Dietz syndrome. (**c**): Bicuspid aortic valve

rysm progression. Ascending aortic aneurysms are often asymmetrical with anterior and rightward protuberance because the inner curvature of aorta is adhered to the pulmonary artery, which provides additional structural support. Dilation of the sinotubular junction and aortic annulus disturbs the coaptation of the aortic leaflets, which can progress to aortic insufficiency. Risk factors for degenerative aneurysms include smoking, hypertension, and hyperlipidemia.

Congenital

Marfan Syndrome

Marfan syndrome is a rare, autosomal-dominant multisystem disorder with high penetrance. The Ghent criteria were developed in 1996, and later revised in 2009, which comprise major and minor manifestations to aid in the diagnosis of Marfan syndrome [19]. The two most common cardiovascular features of Marfan syndrome include dilation of the ascending aorta and mitral valve prolapse, in addition to aortic arch and descending aortic aneurysms, aortic valve degeneration, and cardiomyopathy. Other manifestations include pectus excavatum, glaucoma and cataracts, hepatic and renal cysts, obstructive sleep apnea, myopathy, degenerative arthritis, osteoporosis, dural ectasia, and truncal obesity [20]. The prevalence of Marfan syndrome is about 1 per 3000–5000 individuals, without geographic or ethnic predilection. Nearly 25% of cases are sporadic due to de novo mutations [21]. Historically, Marfan syndrome was thought to be caused by mutations in the gene FNM1 coding the glycoprotein fibrillin-1 [22], resulting in elastin derangement, degeneration, and aortic aneurysm formation [23, 24]. More recently, the discovery of abnormal levels of activation of transforming growth factor β (TGF-β), which causes increased stimulation of inflammation, fibrosis, and metalloproteinases, are responsible for the clinical features of Marfan syndrome in combination with structural microfibril matrix and cell–matrix interaction abnormalities [21, 25, 26].

Aortic root dilation is often progressive and requires close surveillance. It is recommended that patients with a clinical diagnosis of Marfan syndrome and normal aortic diameter undergo annual screening transthoracic echocardiography (Fig. 11.3) with additional CT angiography or MRI every 5 years [27]. Patients with rapid progression or diameters close to surgical thresholds may require more frequent imaging. Both the absolute size of the aorta in the greatest diameter and the rate of expansion are evaluated. A recent study of the risk of an event (death/dissection) in a cohort of patients with Marfan syndrome reported risks of 0.09%/year, 0.30%, 1.33%, and 8.14% for aortic root diameters of <4.0 cm, 4.5–4.9 cm, 5.0–5.4 cm, and >5.5 cm, respectively [28]. Due to the increasing risk of dissection in larger aortic root aneurysms, it is widely accepted that prophylactic surgery should be performed when aortic diameter is greater

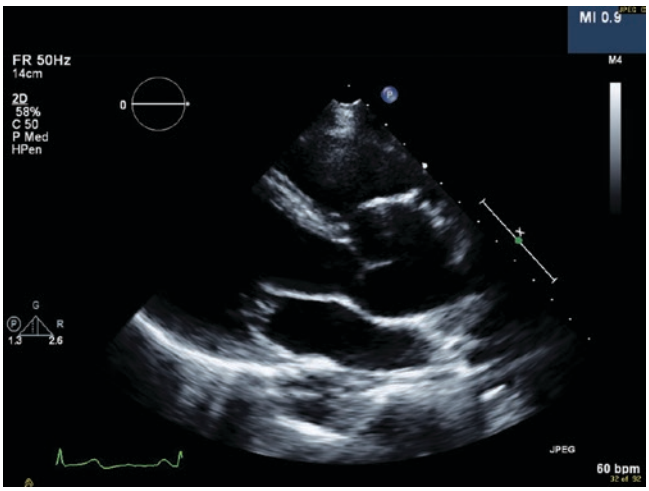

Fig. 11.3 Transthoracic echocardiogram of aortic root aneurysms in patient with Marfan syndrome

than 5.0 cm. Surgery should be considered for >4.5 cm in the presence of risk factors including family history of dissection, severe aortic regurgitation, and diameters >4.0 cm in those who desire to become pregnant [29, 30]. The average rate of aortic root dilation in patients with Marfan syndrome is variable, but is usually less than 1–2 mm per year [31]. Guidelines recommend more frequent surveillance and consideration for prophylactic aortic root replacement when dilation growth is greater than 2–5 mm/year [19, 29]. Without surgery, life expectancy in patients with Marfan syndrome is approximately 35–40 years of age, with leading causes of death mostly cardiovascular, including aortic dissection, rupture, and aortic insufficiency [32].

Medical treatment to prevent or delay the development of aortic dissection in patients with Marfan syndrome includes β-adrenergic receptor blockade, angiotensin II blockade, and historically calcium-channel blockers. The initiation of β-blocker is recommended for all Marfan patients regardless of aortic diameter if clinically appropriate [33]. Angiotensin II-receptor blockers, in addition to their well-known antihypertensive properties, act as a TGF-β antagonist and have been shown to significantly slow the rate of progressive aortic-root dilation in Marfan syndrome [34]. Calcium-channel blockers have been used in those who have had unacceptable side effects or limited response to β-blockers; however, a recently published study found that Marfan mice treated with calcium-channel blockers showed accelerated aneurysm expansion, rupture, and premature lethality [35]. In addition, retrospective analysis of a registry of humans with Marfan syndrome revealed preliminary evidence that aortic dissection and aortic surgical repair were more common in patients who had received a calcium-channel blocker than those who had been treated with other antihypertensive medication [35]. A multicenter randomized control trial compared losartan treatment in both operated and unoperated

adults with Marfan syndrome and reported a significantly decreased aortic root dilation rate in patients receiving additional losartan treatment compared to those with no additional treatment in combination with previously prescribed beta or calcium-channel blockers [36]. Lifestyle modification counseling should include not engaging in contact sports or anaerobic exercise that pronouncedly increases systemic blood pressure due to the risk of acute aortic dissection and sudden death.

Ehlers–Danlos Syndrome

Ehlers–Danlos syndrome (EDS) comprises a group of heterogeneous connective tissue disorders caused by defects in the synthesis of type III collagen. Symptoms classically include hyperelasticity and fragility of skin, joint hypermobility, obstetrical complications, platelet dysfunction, gastrointestinal rupture, and cardiovascular complications. The prevalence of EDS is estimated to be from 1 in every 10,000 to 1 in every 20,000 births [37]. The most common forms of EDS include *classical* (EDS types I, II), *hypermobile* (EDS type III), and *vascular* (EDS type IV) [38]. Vascular EDS type IV is a rare autosomal-dominant inherited connected disuse disorder resulting from mutation of the COL3A1 gene encoding type III collagen.

Cardiovascular complications of EDS type IV include dilation and/or rupture of the aortic sinus and rupture of the aorta, mitral valve prolapse, rupture of medium-sized arteries including the carotid artery, and varicose veins (Fig. 11.4).

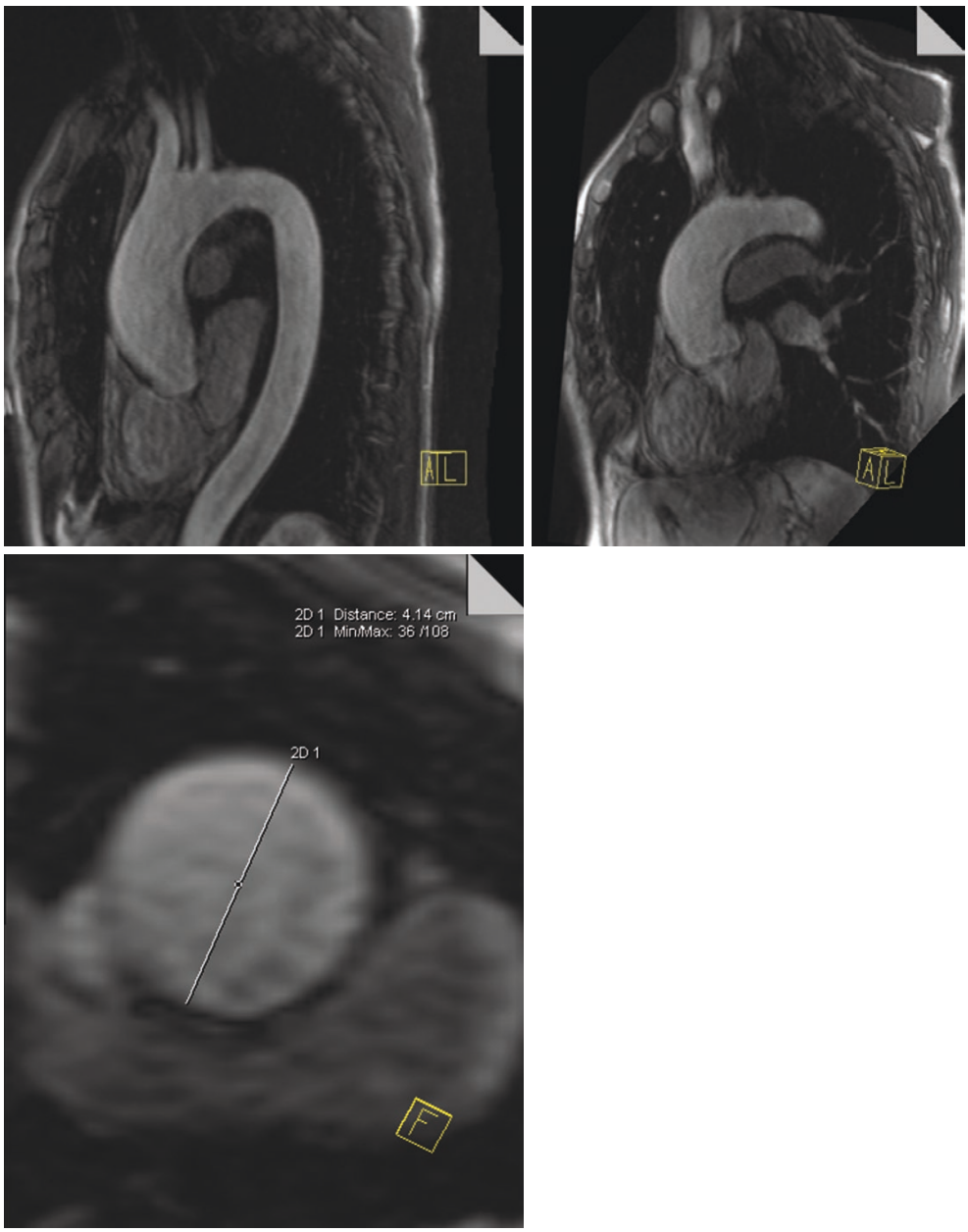

Fig. 11.4 MRI of Loeys–Dietz syndrome and ascending aortic aneurysm

Complications are rare during infancy, but occur in 25% of affected persons before the age of 20 years and 80% before the age of 40 years [39]. The presence of aortic root dilation was 28% in a study of 71 patients with EDS of any type [38]. Spontaneous rupture without dissection of the thoracic and abdominal aorta, as well as medium-sized arteries including carotid and vertebral, as well as branches of the abdominal aorta are the leading causes of death in patients with EDS type IV. Due to the risk of sudden death, close surveillance of aortic dilation is required, and prophylactic surgical repair of rapidly enlarging aneurysm may be indicated. Special precautions include delicate and atraumatic handling of tissues, use of prosthetic material, reinforced pledgeted anastomosis, and close postoperative monitoring with noninvasive imaging [40].

Loeys–Dietz Syndrome

Loeys–Dietz syndrome (LDS) is an autosomal-dominant disorder first reported to result from mutations in either transforming growth factor receptor type I or type II (TGFBR1 or TGFBR2) genes [41]. Subsequently, four different mutations have been identified and correspond to LDS type 1 through type 4, all of which alter TGF-β signaling [42]. This results in increased medial collagen and diffuse elastic fiber fragmentation and extracellular matrix deposition. Characteristics include developmental abnormalities, cleft palate, bifid uvula, scoliosis, pectus deformities, congenital heart defects, persistent patent ductus arteriosus, and atrial septal defects, in addition to aortic root aneurysm and dissection [41]. The progression of aneurysm enlargement is more rapid than other congenital syndromes and often requires prophylactic aortic root repair at smaller diameters and younger ages. Therefore, at least yearly echocardiography is recommended, and more frequently depending on the severity of the aortic disease. Medical management includes strict blood pressure control with antihypertensive medications including β-blockers, angiotensin receptor blockers, and angiotensin-converting enzyme inhibitors. Exercise restrictions should be recommended, including avoidance of contact or competitive athletics and isometric exercises.

MacCarrick et al. recommend surgical thresholds in adults with LDS type 1, 2, and 3, to be an aortic root dimension >4.0 cm or rapidly expanding (>0.5 cm per year) or an ascending aorta/aortic arch diameter of >4.0 cm, with a low threshold for surgical intervention with growth. It is also recommended to delay surgery in children until aortic annulus is 2.0–2.2 cm to accommodate adult-sized grafting [42]. Postoperative echocardiography is recommended at 3–6 month intervals for the first year, then every 6 months to 1 year thereafter.

Bicuspid Aortic Valve Disease

Bicuspid aortic valve is a common congenital heart anomaly that occurs in approximately 1–2% of the population [43]. It more commonly affects males and is associated with Turner syndrome. Bicuspid aortic valve disease is prevalent in patients with first-degree relatives of patients with bicuspid aortic valve disease [44]. There are many suspected mechanical properties of bicuspid valves that increase the propensity of ascending aortic dilation, including significantly increased wall stress, larger dimensions of the aortic annulus and ascending aorta, independent of aneurysmal disease formation. The aortic wall in bicuspid aortic valve disease has been shown to contain increased levels of matrix metallic proteinases, elastic fragmentation, matrix disruption, and deficiency of fibrillin-1 [22, 45, 46]. Patients with bicuspid valves have a higher rate of aortic root dilation, often including the aortic arch [47] (Fig. 11.5).

Infectious

Infections may occasionally cause damage the aortic wall, which can occasionally lead to the formation of mycotic ascending aortic aneurysms. They are often associated with left-sided endocarditis, therefore sharing some of the most common organisms, such as *Staphylococcus aureus, Staphylococcus epidermidis, Salmonella,* and *Streptococcus pneumoniae* [48–50]. Syphilis was a common cause of ascending aortic aneurysm prior to the use of antibiotics. Untreated syphilis can result in the degeneration of the medial elastic fibers of the aorta, which is not reversible with antibiotic treatment. Mycotic ascending aortic aneurysms may also form in atherosclerotic aortic disease with the seeding of intraluminal clot with bacteria [49]. Computed tomography (CT) with contrast enhancement is the gold standard for diagnosis, with findings of periaortic density and adjacent gas collection signs impeding rupture [50]. Transesophageal echocardiography is often the initial imaging technique due to its role in the evaluation of infective endocarditis. Magnetic resonance imaging (MRI) with gadolinium contrast may also be beneficial. Prompt initiation of antibiotic therapy and complete surgical excision of the infected aorta is widely recommended; however, few controlled studies have been performed to guide the management of infective aortitis.

Noninfectious Inflammatory Syndromes

The most common causes of noninfectious aortitis include large-vessel vasculitides giant cell arteritis (GCA) and Takayasu arteritis, but other rheumatologic disorders such as system lupus erythematosus, rheumatoid arthritis, Wegener granulomatosis, and polyarthritis nodosum are known causes [51]. Antigen-driven cell-mediated autoimmune infiltration of the aortic media, adventitia, and vasa vasorum are present in GCA and Takayasu arteritis, which causes scarring and progresses to the destruction of the elastic lamina. Immunosuppressive therapy is the primary treatment of large-vessel vasculitis, and glucocorticoid therapy should be initiated at the time of diagnosis. Patients should be monitored closely for the common complications of

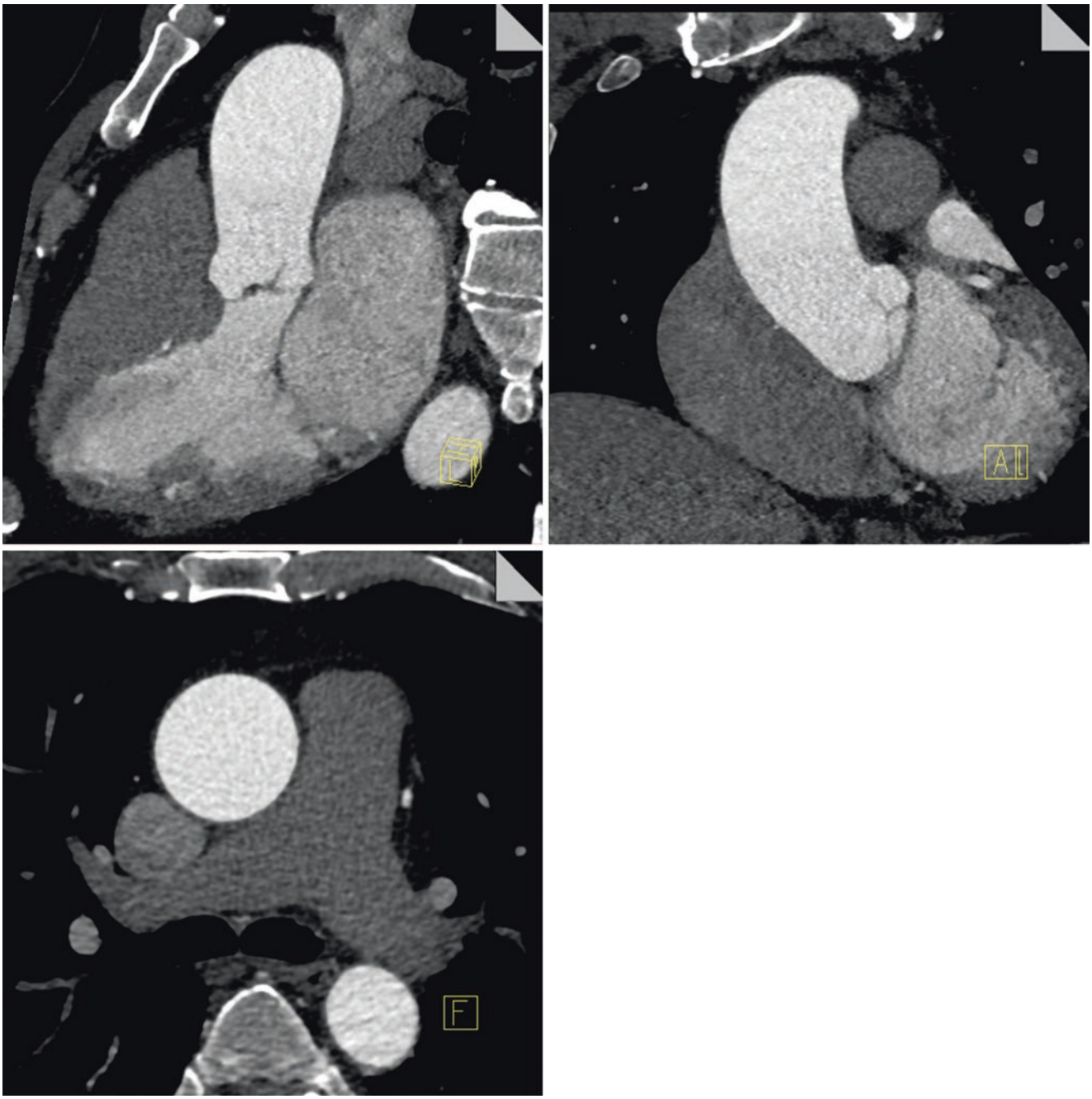

Fig. 11.5 Bicuspid aortic valve and ascending aortic aneurysm

long-term glucocorticoid therapy. Surveillance of aortic aneurysms is necessary and open aortic aneurysm repair remains the standard of treatment for enlarging aneurysms.

Clinical Presentation

Ascending aortic aneurysms are commonly asymptomatic, and are frequently discovered incidentally on radiographic studies. However, as aneurysms progress in size or involve the aortic arch they more frequently become symptomatic, with chest pain, back pain, dyspnea, dysphagia, and transient neurologic deficit. Hoarseness due to stretching of the recurrent laryngeal nerve may occur. Symptoms of heart failure may develop as dilation of the aortic root or ascending aorta disrupts coaptation of the aortic valve leaflets causing aortic regurgitation, evident by a widened pulse pressure and diastolic murmur. Acute dissection or rupture of the ascending aorta is a surgical emergency and often manifests as sudden severe anterior chest pain. Myocardial infarction, stroke, and

cardiac tamponade may result, as well as dissection down throughout the thoracoabdominal aorta causing ischemia of the kidneys or abdominal viscera. Chest X-ray is frequently obtained as initial imaging and may contain a widened mediastinum. Diagnosis is commonly obtained with chest computed tomography.

Imaging Modalities

Imaging of the aorta has evolved dramatically in the past 50 years. The advancement of echocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) have allowed for many options for clinical assessment. However, each imaging modality has evolved largely independent of the others, leading to lack of standardization across the different modalities. This requires that evaluation between modalities and amongst serial studies be done cognizant of the potential limitations of each testing strategy. The best approach is to compare images directly, remeasuring if necessary to ensure consistency in approach and timing of measurement. Developing a good working relationship with advanced imaging colleagues, both Cardiology and Radiology, will ensure that clinical decisions are being made based on disease progression and not variability in measurement.

Transthoracic echocardiography is a common initial imaging strategy at most institutions due to the ease of acquisition, safety, tolerability, the ability to concurrently evaluate cardiac structure and function, and identify concurrent pathology. Compared to other aortic segments, transthoracic echocardiographic imaging of the ascending aorta is both accurate and usually reproducible, and correlates well with both transesophageal echocardiography and CT aortic diameter measurements. In addition, the negative predictive value for aortic root dilation of an optimized, diagnostic-quality transthoracic echocardiogram is high, suggesting no additional testing needed for normal aortic roots. In addition, both transthoracic echocardiography and transesophageal echocardiography have outcome data, which has driven the current guideline recommendations regarding its use. Extensive data in normal populations has allowed for gender-specific cutoffs to be developed, which are indexed to body surface area. This allows for more precise interpretation of aortic measurements tailored to individual patients. Furthermore, echocardiography allows for reliable assessment of aortic physiology as well as anatomy, specifically around distensibility and regional stiffness. This allows for assessment of dysfunction despite normal anatomy. Despite imaging-based guideline documents, there are common sources of variability that are important to identify.

Firstly, current guidelines recommend all measurements be in end-diastole. This is largely due to the relative stability of aortic hemodynamics as well as easy identification using the QRS complex, both leading to improved reproducibility. However, aortic dimensions are often the largest in end-systole, and CT and MR do not have similar guidelines as to the timing of measurement (many are nongated studies, removing this as an issue altogether). This can make comparisons of reports difficult, especially if discrepancies are found.

Secondly, current guidelines recommend measuring "leading edge to leading edge." Leading edge measurements were largely related to previous technology that made clear identification of the inner wall of the vessel difficult. However, with improved technology, harmonic imaging, and use of contrast agents, this has become less of an issue. While true inner diameter may be a more accurate representation of the size of the vessel, outcome data is largely linked with the older "leading edge to leading edge" methods. As such, guideline documents have not adjusted to reflect a change in practice. This "leading edge to leading edge" method overestimates the diameter by an average of 2 mm when compared to inner diameter measurements.

Thirdly, 2D echocardiography is angle-dependent. Depending on the orientation of the ultrasound (US) probe to the vessel, the measured diameter may underestimate or overestimate the true diameter if looked at in cross section.

Transthoracic Echocardiography

Transthoracic echocardiographic (TTE) imaging of the ascending aorta is largely done via the parasternal long-axis and suprasternal views. The parasternal long-axis view allows for the left ventricular outflow tract, aortic valve, and aortic root and a portion of the ascending aorta to be viewed in continuity. Generally, optimization of the imaging plane is achieved when both the mitral valve and aortic valve excursion are seen at the highest possible intercostal level. However, for imaging of the aorta itself efforts should be taken to maximize both aortic root and aortic dimensions given the above concerns. In this view, the aortic valve leaflets of trileaflet valve are generally seen coapting in the center of the left ventricular outflow tract, and the long axis of the aorta is seen.

Allowing for easy measurement can introduce a source of variability between different modalities. In most patients, this represents the distance between the right coronary sinus to the non-coronary sinus. Sinus-to-sinus measurements, as opposed to sinus-to-commissure measurements, tend to overestimate the aortic root size by a mean of 2 mm. Measuring in this fashion may still be helpful clinically, because it may identify sinus of Valsalva aneurysms. In addition, asymmetric enlargement of the sinuses will not necessarily be appreciated from this two-dimensional view. Three-dimensional echocardiography can help address this

issue, and some have advocated the use of an average measurement of the three individual sinus-to-sinus measurements in three-dimensional modalities. This source of variability is not inconsequential, and has been the impetus for CT-guided imaging of the aortic annulus and aortic root in most structural heart programs for transcatheter aortic valve replacements (TAVR).

Frequently, the distal ascending aorta is not completely appreciated from the parasternal long-axis view due to acoustic impedance from the left lung, and the suprasternal view is needed to identify the distal ascending aorta and arch. This view is not always obtained due to patient comfort and anatomy, but provides visualization of the distal ascending aorta and aortic arch in the vast majority of patients (>90%). Right parasternal views are needed, especially in patients with significant dilatation. Finally, apical and subcostal views can be used in selected patients, but the complete ascending aorta is not usually completely visualized in these views.

Transesophageal Echocardiography

Transesophageal echocardiography (TEE) allows for increased spatial resolution due to higher frequency probes and the proximity of the esophagus to both the ascending and descending aorta. Furthermore, multiplane TEE probes allow for both long-axis and short-axis visualization of the aorta. TEE has been studied largely in its role in acute aortic syndromes, but can also be used in the assessment of ascending aortic aneurysms. The same issues in reproducibility occur with TEE as they do with TTE, although diagnostic image quality is generally more reliable. A specific issue with the TEE is visualization of the distal ascending aorta, arch, and great vessels due to acoustic impedance of the ultrasound image from the trachea. This theoretical "blind spot" can largely be overcome via imaging from different angles and depths to avoid air-filled structures.

Computerized Tomography

Computed tomography (both ECG gated and non-ECG gated) have rapidly become the gold standard for imaging of the entire aorta. The increased spatial resolution and rapid and reliable imaging in varied clinical scenarios have allowed for routine use of CT in this setting. The major limitation of CT-based imaging approach is the need for ionizing radiation (especially in younger patients or for serial imaging). Contrast agents are less of a concern now, especially given that iso-osmolar agents and intravenous injection allow for adequate mixing prior to the renal artery, minimizing the risk of nephrotoxicity.

Specific issues regarding imaging the aortic root and the ascending aorta largely surround artifact created by normal cardiac motion, often mimicking an aortic root dissection. This has necessitated the need for ECG gating to allow for imaging at a time when cardiac motion is minimal. In most patients with regular heart rhythm, this is achieved in end-diastole, but is adjustable, and can lead to variability in measurements.

Furthermore, the method of measurement has not been standardized. Ideally, all measurements will be made after centerline reconstructions to allow for a true cross section to be visualized. Measurements made from axial slices alone can often overestimate true dimensions. In addition, there is variability in how measurements are made. Inner diameter to inner diameter measurements likely reflect the true lumen and are most accurate. However, this can be challenging, especially in the setting of intramural hematomas, previous interventions (endovascular or open), or aortic wall thickening. Previous guidelines had suggested outer dimension to outer dimension measurements to allow for reproducibility with contrast and noncontrasted studies. However, this can significantly overestimate the true diameter. Measuring axial slices with an outer diameter to outer diameter approach will lead to the largest dimensions.

Magnetic Resonance Imaging

Magnetic resonance angiography (MRA) has been frequently been used in imaging the thoracic aorta, especially for serial assessments or a more comprehensive assessment in younger patients. The spatial resolution is less than either CT or echocardiography, especially TEE, but allows for reconstructions, as well as the lack of ionizing radiation has made it an attractive option for many patients. In addition, time-of-flight imaging allows for an MRA to be done without the need for gadolinium-based contrast agents. Patient comfort and time can limit those that tolerate MRA as an imaging modality, but evolution of this technology has limited this issue.

Unfortunately, the same issues with measurement in CT apply to MRA as well. Cardiac motion can lead to lack of spatial resolution and increased artifact, especially at the aortic root. This can be largely avoided with ECG gating and appropriate timing of acquisition. Centerline reconstruction and inner diameter to inner diameter will likely give the smallest diameter, while axial slices and outer diameter measurement will tend to overestimate the dimension.

Selecting the Appropriate Imaging Modality

Selecting the optimal imaging modality for the ascending aorta is dependent on many factors: age of patient, body habitus, need for serial testing, concurrent disease, expected

location of disease, patient compliance, and renal function. In general, an initial assessment with transthoracic echocardiography is most appropriate, especially if the disease is expected to be limited to the aortic root or proximal ascending aorta, concurrent valvular disease is suspected, or the patient has concurrent cardiovascular disease that needs to be assessed. If the transthoracic echocardiogram is normal and the ascending aorta is well visualized, additional testing should be reserved for specific clinical questions. For aortic root or ascending aortic dilation or for nondiagnostic images, assessment with CT or MRI is reasonable. The decision of which to pursue should largely be dependent on patient-specific considerations of age, need for serial testing, and patient comfort. In patients at risk for syndromic aortas, initial testing with CT or MRI is reasonable depending on patient-specific factors.

Surgical Indications

Prompt surgical evaluation and intervention is indicated in patients with symptoms suggestive of aneurysm expansion independently of size, except in cases of limited life expectancy or quality of life from comorbid conditions [30]. Indications for asymptomatic aortic aneurysm resection and replacement have developed over time. Elefteriades and colleagues at the Yale Center for Thoracic Aortic Disease have reported a threshold for dramatically increasing risk of dissection with progressive aneurysmal dilation at a size of 6 cm in the ascending aorta, and 7 cm in the descending aorta [52–54]. However, patient size is an important factor in evaluating aortic aneurysms, especially when considering operative guidelines that would apply to both small and large patients. A calculation was developed by Elefteriades et al. based on a retrospective review of patients who developed aortic dissection or rupture that incorporated body surface area and aortic aneurysm diameter [55]. Body surface area (BSA) in m^2 is calculated by the Dubois and Dubois formula:

$$\mathrm{BSA} = 0.20247\left(wgt^{0.425} \times \left(\frac{hgt}{100} \right)^{0.725} \right)$$

The aortic size index (ASI) is described as the aortic diameter (cm) divided by body surface area (m^2). Patients with ASI less than 2.75 cm/m^2 are classified as low risk (approximately 4% per year), 2.75–4.24 cm/m^2 are considered medium risk (approximately 8% per year), and greater than 4.25 cm/m^2 are high risk (approximately 20% per year) [55].

For asymptomatic adults with degenerative thoracic aneurysms, it is generally accepted that resection of the ascending aortic aneurysm is indicated at 5.5 cm or growth rate of more than 0.5 cm/year, aortic arch aneurysms should be resected at 5.5 cm, and descending aortic aneurysms may be monitored until a diameter of 6.0 cm prior to open repair [30, 56]. Lower thresholds should be considered in patients of small stature or in the cases of aortic regurgitation, and pregnancy. Surgical thresholds for repair of ascending aortic aneurysms are smaller in genetic syndromes (Table 11.1) [30, 56], such as Marfan's syndrome at 5.0 cm (4.0 cm if contemplating pregnancy) or family history of aortic dissection at smaller diameter, LDS at between 4.2 and 4.6 cm depending on imaging modality, bicuspid aortic valve at less than 5.0–5.5 cm, and EDS less than 5.0 cm but limited data is available to establish a threshold [30, 56]. Arguments have been presented that early treatment of thoracic aortic aneurysms may be beneficial in appropriate patients due to the difficulty in predicting aortic dissection, high rates of early and late mortality, emotional burden to patients, and relative safety of elective aortic surgery in the present era [57].

Table 11.1 Indications for surgical repair on asymptomatic ascending aortic aneurysms

Underlying etiology of aneurysm	Diameter (cm)
Degenerative thoracic aneurysm	5.5
If also indication for aortic valve surgery	4.5
Marfan	4.5–5.0
If desiring pregnancy	4.0
Ehlers–Danlos	4.2–4.4
Loeys–Dietz	5.0
Turner syndrome	5.0
Bicuspid valve	5.0–5.5
Mycotic aneurysm	5.5

Data from [30, 56]

Operative Technique

Ascending aortic aneurysm is a surgical disease and operative repair is essential to prevent dissection and rupture. Indications for repair are based on size, symptoms, and rate of growth. Multiple preoperative procedures are commonly performed by anesthesiologists. Venous access is obtained with a large bore central catheter, typically via the internal jugular vein with pulmonary artery pressure monitoring, as well as several large peripheral catheters. An arterial catheter is placed for hemodynamic monitoring, and both right and left radial artery access is commonly preferred in patients with aneurysms involving the aortic arch. If hypothermic circulatory arrest is planned, bilateral electroencephalographic monitoring is also employed. Transesophageal echocardiography is performed intraoperatively to evaluate cardiac function, size of aorta, and valvular function.

All patients are placed on cardiopulmonary bypass prior to repair of the aneurysm. Many repairs require concomitant aortic valve replacement, either as a separate prosthesis or using a composite graft. Choice of operative technique

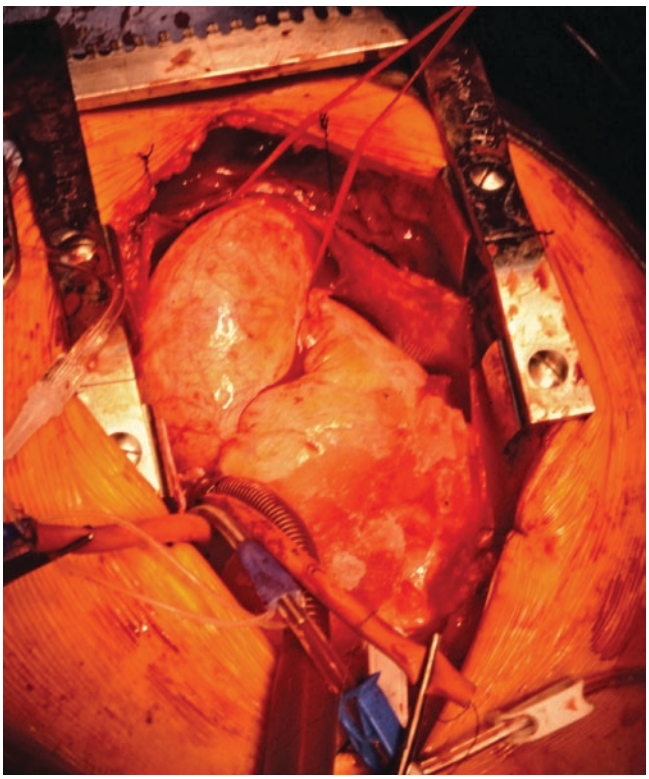

Fig. 11.6 Ascending aortic aneurysm repair

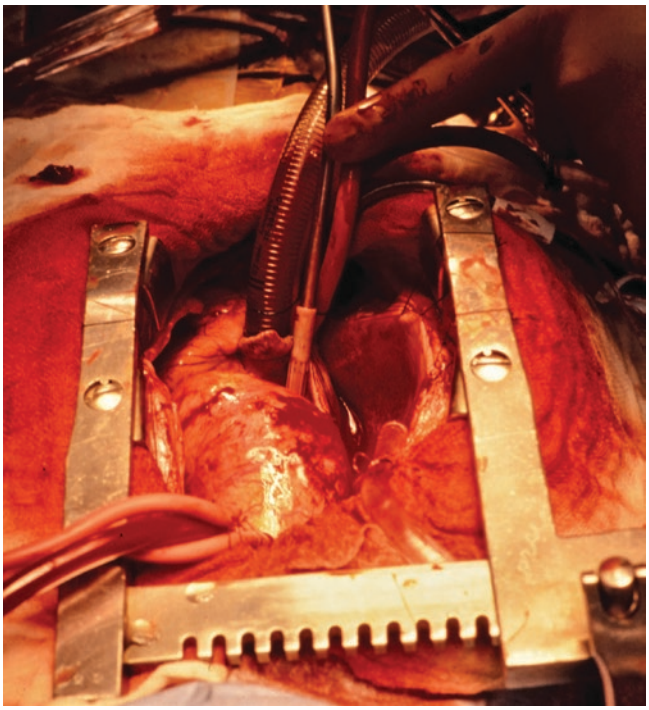

Fig. 11.7 Ascending aortic aneurysm

depends on the etiology and extent of aortic disease, as well as patient anatomy and preoperative condition (Figs. 11.6 and 11.7).

A. Isolated ascending aortic aneurysm is frequently excised and replaced with a Dacron tube graft. This procedure carries a low risk with good long-term results. However, the aortic valve is often involved and requires repair or replacement.
B. Aortic valve replacement and ascending aortic replacement with a tube graft without requiring coronary reconstruction may be performed in ascending aortic aneurysms not involving the root with concomitant aortic valve pathology, such as bicuspid aortic valve.
C. Aortic root aneurysms may require composite root replacement, which involves excision of the ascending aorta, coronary artery reimplantation, and placement of a composite valve, either mechanical (Bentall procedure) or implanted bioprosthetic. This technique is often necessary when the aneurysm involves the sinotubular junction resulting in aortic insufficiency.
D. If the aortic valve is incompetent but the cusps are normal, valve competence can be reestablished by repair of the aortic root. Valve-sparing aortic valve repairs such as the David and Yacoub procedures can be performed, which require advanced technical skill and experience and are predominantly performed at large aortic centers.

Outcomes

Early hospital mortality after elective repair of chronic ascending aortic aneurysm ranges to a high degree and is reported to be as high as 9% and recent surgical advancements have reduced operative mortality to below 3% [58–61]. Causes of early mortality and morbidity include postoperative hemorrhage, stroke, respiratory failure, myocardial dysfunction, and renal dysfunction and failure. Reoperation for bleeding ranges widely between 3% and 12%, depending on type of repair [58, 59, 62]. Preoperative risk factors for stroke include prior stroke, severe carotid artery occlusive disease, age greater than 65 years, and peripheral vascular disease [63]. Cardiopulmonary bypass is associated with acute respiratory distress syndrome and the incidence is reported to be between 0.4% and 1.7% [64, 65]. Aortic disease involving the arch carries a higher mortality [66]. Ascending aortic surgery due to diffuse atherosclerosis, which is commonly seen in cases of ascending aortic aneurysm and frequently requires concomitant coronary artery bypass grafting has higher early mortality and morbidity [67, 68]. Late mortality is variable and is dependent on patient characteristics and surgical technique. Survival rates vary from 80% to 95% at 1 year, 70% to 90% at 5 years, 60% to 75% at 10 years, and 60% at 14 years [69–71]. McCarthy et al. published a study of patients at the University of Pennsylvania who underwent elective aortic root replacement for aortic insufficiency with aneurysm and found that 95% were free of reoperation at 10 years [72]. Late mortality

in patients undergoing valve-sparing aortic root replacement at high volume centers is comparable to repair with prosthetic valves [73, 74].

Conclusion

Ascending aortic aneurysms are not uncommon and are potentially asymptomatic, and deadly.

Healthcare providers need to be aware of the patient profiles and their clinical presentations. An understanding of the strengths and limitations of the diagnostic tools available at their institutions is warranted. Recognizing the indications for surgical intervention is necessary to offer the patient the best chance for improved clinical outcomes. Timely diagnosis and delivery of appropriate medical therapy and surgical procedures is essential not only to improve survival, but reduce comorbidities and improve quality of life. However, despite a much greater understanding of the underlying etiologies, diagnostic options, and surgical techniques for ascending aortic aneurysms over the past 50 years, there remains much to learn regarding an individual patient's risk. Therefore, all prophylactic decisions for surgery should be made together with the healthcare provider and the patient, taking into consideration individual clinical and nonclinical factors.

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