Endovascular Treatment of Atherosclerotic and Other Thoracic Aortic Aneurysms

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ABSTRACT

The incidence of thoracic aortic aneurysms (TAAs) is increasing with the present rate of occurrence at 10.9 cases per 100,000 people per year. The estimated 5-year risk of rupture of a TAA with a diameter between 4 and 5.9 cm is 16%, but it rises to 31% for aneurysms ≥ 6 cm. Despite increasing awareness of the importance of early diagnosis and treatment options, there are no clear guidelines available at the time of writing. Nor is there any clear evidence for specific pharmacological treatment able to resolve or delay the disease progression. Endovascular treatment (EVT), proposed as an alternative to surgery, has been considered a therapeutic innovation, especially because it is minimally invasive, which allows treatment even in high surgical risk patients. Vascular imaging is crucial for patient selection, endoprosthesis choice, and planning of the treatment because not all aneurysms are suitable. Early and midterm results are encouraging, but long-term results are necessary to definitively assess reliability of stent-graft materials and improvement in patient survival. In the choice between surgical or endovascular repair of TAAs, many factors must be considered, including the clinical situation, comorbidities, anatomy, choice of equipment, and last, but not less important, experience of the clinical team.

KEYWORDS: Thoracic aortic aneurysm, endovascular treatment and follow-up

Objectives: Upon completion of this article, the reader should understand the indications for inserting endovascular stent grafts in the thoracic aorta as a treatment option for atherosclerotic and other thoracic aortic aneurysms.

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In the past decade, the incidence of thoracic aortic aneurysm (TAAs) was estimated to be 3.5 cases per 100,000 people per year. A population-based study of thoracic and thoracoabdominal aortic aneurysm in 1998 reported an incidence of 10.9 cases per 100,000 people per year, revealing an increased rate of occurrence of the disorder.¹ Although this may be a function of more sophisticated and available imaging techniques, the phenomenon is not completely explained: The

complex multifactorial mechanism that leads to alteration of the tunica media and aneurysm formation is still under investigation. Most aneurysms are atherosclerotic, usually long and fusiform. Saccular false aneurysms may also develop in patients with atherosclerosis as a result of a penetrating ulcer. The natural history is quite diverse, reflecting a broad spectrum of underlying causes. Although many studies have identified risk factors related to the formation and

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progression of aortic aneurysms, none has fully explained the etiology of the disorder.

Atherosclerosis could be considered a concomitant process and not a direct cause of aneurysm formation and growth. Recently, the role of inflammation has also been investigated in promoting aneurysm growth and rupture. Aortic medial degeneration has been demonstrated in most aneurysms, regardless of their cause and location. The structural integrity of the adventitia is also lost as an aneurysm forms and expands.

In the ascending aorta, gradual degenerative changes of the media can be related to congenital disorders of the extracellular matrix associated with an alteration of the elastic fibers, such as Marfan's syndrome; similarly, families with a genetic predisposition to the development of thoracic and abdominal aneurysms, without evidence of collagen-vascular disease, have been documented.

Inflammatory diseases of the aorta may also result in thoracic aneurysms. Aortitis of nonspecific or unknown etiology (Takayasu's aortitis, Behçet's syndrome, giant cell aortitis, Kawasaki disease, ankylosing spondylitis), and specific aortitis, in which the aortitis is the consequence of an inflammatory disease of known origin (e.g., syphilitic aortitis), are common in Asian countries and rare in white populations. This disparity suggests an element of genetic transmission. Histologically, marked irregular mural thickening of the aortic wall and a fibrous lesions are the result of the inflammatory process of the media. These alterations can lead to stenotic lesions (Takayasu's disease), aneurysms of the aorta and its major branches, or aortic insufficiency as a consequence of dilation of the aortic root.

The overall survival of patients with TAA has improved significantly in the past few years. The estimated 5-year risk of rupture of a TAA with a diameter between 4 and 5.9 cm is 16%, but it rises to 31% for aneurysms ≥ 6 cm. Despite increasing awareness of the important role of early diagnosis in treatment options, no clear guidelines about therapeutic strategies exist. For example, there are no clear guidelines for pharmacological treatment to resolve or delay disease progression.

TREATMENT OPTIONS FOR THORACIC AORTIC ANEURYSMS

Thoracic aortic diseases historically represent a challenge for surgeons due to procedural complexity and clinical problems related to surgical access and anesthetic techniques. Since the first experiences of Swan in 1950² and DeBakey and Cooley in 1953,³ continuous improvements in thoracic aortic surgery have been supported by diagnostic and etiopathogenetic knowledge, advances of visceral and cerebral perfusion systems, and improvement of surgical materials and technique. Because atherosclerosis is the major leading cause, affected people

are mostly elderly, often with comorbidities such as ischemic heart disease, hypertension, chronic obstructive pulmonary disease, or diabetes, which negatively influence surgical risk and patient outcome.

Surgical access, consisting in thoracotomy with associated phrenolaparotomy, is particularly invasive and carries substantial risks of serious cardiovascular and respiratory dysfunctions. Hemorrhage, coagulation disorders, medullary ischemia, cerebrovascular accident, and renal failure often complicate surgery and postoperative outcome and therefore patient prognosis. Mortality and morbidity range from 5 to 15% in elective cases and up to 50% in emergency situations.^{4,5}

The first thoracic endovascular stent-graft insertion was described in 1986 by Volodos et al,⁶ but the history of thoracic EVT really began in 1992 when the Stanford group published a clinical trial of 103 patients (1992 to 1997) with TAA caused by different pathologies such as atherosclerotic aneurysms, aortic dissection, penetrating ulcers, and post-traumatic lesions;⁷ using an experimental homemade endovascular stent graft, 30-day mortality was 9% and 1-year survival was 81%.

In 1996, the first commercial TAA endoprosthesis became available and focused the interests of radiologists, cardiologists, and vascular-cardiac surgeons with a fusion of different experiences and clinical cultures. In the thoracic aorta, EVT is now accepted for TAAs and other disorders such as penetrating atherosclerotic ulcers, traumatic partial thickness tears, pseudoaneurysms, intramural hematomas, and aortic type B dissections.⁸⁻¹¹ Furthermore, the use of EVT is increasing in emergency situations (acute aortic syndromes) because of the high surgical mortality and ineffectiveness of medical therapy.¹²⁻¹⁶

CONTRAINDICATIONS AND INDICATIONS FOR TREATMENT

The anatomy of the thoracic aorta is the limiting factor as to whether endovascular treatment is possible: Vascular imaging is crucial for patient selection, endoprosthesis choice, and planning.

Anatomy

To assess EVT feasibility, a complete visualization of the entire aorta, from supra-aortic arteries to femoral arteries, is mandatory to evaluate lesion morphology, the state of the aortic wall, and anatomy of the vascular access site. To obtain a good seal with the aortic wall, oversizing of the prosthesis diameter from 10 to 30% is required. An accurate measurement of aortic diameters is mandatory in EVT planning. The diameter of stent graft delivery systems ranges from 22 to 27F (7 to 9 mm) and the femoroiliac access must be dimensionally adequate,

Figure 1 Multidetector computed tomography volume-rendered (A) and curved multiplanar reconstruction (B) images showing a severely calcified (large arrow), tortuous (arrows), and thin (arrowhead) femoroiliac axis. These features suggest that percutaneous insertion of the delivery catheter will be difficult or impossible and are a predictor of serious vascular damage. A surgically constructed iliac or aortic conduit may be necessary.

especially in the presence of atherosclerosis, calcifications, and tortuosity (Fig. 1).

Aortic wall assessment at the level of landing zone is also very important. An atheromatous, thrombotic, or heavily calcified wall could cause a failure to seal or late slippage (Fig. 2). It is generally accepted that the length of the proximal and distal neck (a portion of normal aortic wall between the origin of the left subclavian artery/celiac trunk and the proximal/distal extremities of the aneurysm) should be at least 1 to 1.5 cm. If the aortic neck is shorter than 1 cm, a graft may not exclude

an aneurysm from the blood flow and lead to a high risk of device migration in the long term.

When the TAA is at the origin of the left subclavian artery, the proximal neck can be extended by creating an extra anatomical bypass of the supra-aortic vessels. Left subclavian artery bypass (from ipsilateral carotid artery) or transposition can be performed from a supraclavicular surgical approach; this technique is minimally invasive and can also prevent potential complications like cerebellar infarction (vertebral artery occlusion) and subclavian-vertebral steal syndrome.¹⁷

Figure 2 Multidetector computed tomography volume-rendered (A) and multiplanar reconstruction (B) images showing a large thoracic aortic aneurysm (large arrows) with a short and atherosclerotic proximal neck (arrows), which is unlikely to allow a proximal seal without covering the left subclavian artery. This is acceptable as long as there is a good right vertebral artery. Only 10% of patients develop symptoms relating to left arm ischemia, and this group will require a carotid subclavian bypass operation.

Furthermore, reperfusion of aneurysm sacs from the subclavian artery (type II endoleak) can be avoided. Extra-anatomic bypass of the right carotid or innominate artery requires a sternotomy and aortic clamp. Because the mortality ranges from 6.8 to 15%,¹⁸⁻²¹ this should be reserved for patients with a very high risk for traditional arch surgery.

IMAGING EVALUATION

Several excellent imaging techniques are currently available for pretreatment evaluation, during surgery, and postprocedural follow-up. Computed tomography (CT) is generally accepted as the first-choice modality in the evaluation of acute and chronic aortic disease.^{22,23} Its advantage is its ability to explore the entire thoracic-abdominal aorta with the inclusion of femoral access in a few seconds and with only one contrast media injection (70 to 80 mL). With multidetector CT (MDCT), spatial resolution is submillimeter, and the aorta can be visualized from any angle and anatomical plane (Fig. 3). Software reconstructions such as maximum intensity projection and volume rendering are useful for displaying complex 3D anatomy, including the aortic arch and supraortic vessels.

Magnetic resonance (MR) is also useful in aortic disease evaluation, even if calcifications are not visualized. The possibility of functional analysis and the

anatomical detail provided by MR angiography are particularly useful in conditions such as post-traumatic lesions or congenital aortic disorders.

Intra-arterial digital subtraction angiography is infrequently necessary in the preliminary evaluation but is occasionally useful for a precise assessment of morphology of the aortic arch or femoral arteries. Its role is still essential during the actual stent graft insertion for fluoroscopic guidance and road mapping.

Transesophageal echocardiography (TEE) is very useful during endovascular procedure.²⁴⁻²⁸ With this technique it is possible to visualize the correct position of guide, catheters, and stent graft system. Furthermore TEE provides a real-time evaluation of the aortic wall at the drop and landing zones. After stent graft delivery, TEE with color Doppler is able to identify residual blood flow inside the aneurysm sac (endoleak) or initial thrombosis (echocontrast effect) as an indicator of the efficacy of the procedure.²⁸ When balloon inflation for stent apposition is necessary, TEE can establish the correct grade of balloon distension and the contact with the aortic wall, avoiding the risk of damage by overinflation. TEE, therefore, can be useful in all phases of thoracic endovascular procedures. It also allows a reduction in contrast load and radiation exposure.

Figure 3 Multidetector computed tomography volume-rendered (A) and multiplanar reconstruction (B) images showing dilation of the descending thoracic aorta (arrows). Proximal and distal necks, as well as the femoroiliac access site, are free from thrombosis and/or calcifications.

POSTOPERATIVE FOLLOW-UP

CT is the imaging modality of choice also in the post-procedural follow-up (Fig. 4). When contraindications are present (e.g., renal failure, allergy to iodine, pregnancy), MR is generally employed although not with ferromagnetic devices. In young patients, MRI may be also preferable to avoid excessive radiation exposure. Regular imaging follow-up is recommended during the first year (1, 3, 6, and 12 months) after the procedure.^{8,29}

The most important morphological parameters that should be evaluated are the presence or absence of blood flow inside the aneurysm sac (endoleak), aortic dimension, morphology of prosthesis (position and structural integrity), diameter and morphology of proximal and distal neck, and aortic wall abnormalities. The presence of blood flow inside the aneurysm sac (endoleak) is an indicator of procedural failure and has an important clinical impact in thoracic aortic disease, with a potential risk of aortic rupture.^{8,13,29} Endoleaks in general may have slow flow, which could be missed on arterial phase CT. Therefore, biphasic CT or MR acquisition, with an early (30 to 40 seconds) and delayed (60 to 80 seconds) scan,³⁰ are recommended.

Five types of endoleak have been identified.^{31–36} Endoleak is defined as type I when blood flow originates from proximal or distal extremities of the graft, with reperfusion of the aneurysm sac; type II endoleak is caused by a retrograde blood flow arising from a tributary artery of the excluded portion (left subclavian artery, intercostal arteries). Type III endoleak originates from a prosthetic material defect (holes, laceration) or from segment disconnection; type IV endoleak is caused by stent macroporosity and generally resolve spontaneously, and type V (endotension) derives from a pressure rise inside the excluded sac without evidence of blood flow. Endoleaks may occur early or late during follow-up: The former appear often at the end of the endovascular procedure, the latter during follow-up examinations.

Type I and type III endoleaks that cannot be repaired endovascularly by graft extension need immediate surgery (conversion) because they usually lead to aneurysm sac expansion and rupture. Treatment of type II endoleaks (Fig. 5) is not urgent because most seal spontaneously.²⁹

Migration of the stent graft is rarely reported in thoracic series. Migration of a thoracic stent graft may cause aortic branch occlusion such as celiac trunk and other visceral vessels with severe ischemic consequences. Therefore CT images of the stent graft position with respect to left subclavian or celiac artery should be compared in each CT follow-up examination.

RESULTS

Technical and clinical results of TAA endovascular stent grafting are encouraging for both acute and chronic

aortic diseases. The Stanford Clinical Trial, the first performed with thoracic stent graft,^{7,37,38} with a large population (103 patients) and 10 years of follow-up, showed a perioperative mortality rate of 9% and a survival rate of 82%, 49%, and 27%, respectively, after 1, 5, and 8 years. However, most of the deaths (57/65; 87.7%) were due to nonaortic causes such as cancer, stroke, acute myocardial infarction, or heart failure. Independent risk factors for death were older age, previous stroke, and an American Society of Anesthesiologists (ASA) III grading. Freedom from aortic rupture was 99%, 94%, and 91% at 1, 5, and 8 years, and freedom from aortic reintervention was respectively 84%, 80%, and 78%. The philosophical question has been raised whether it is right to provide such an expensive treatment that does not improve quality of life because many patients are asymptomatic when they have such a dismal prognosis. This is not an easy question to answer, but for many patients living with the “time bomb” of a TAA is unacceptable. As long as patients are fully informed about their prognosis and the risks they undertake by having or not having a repair, then most physicians are comfortable that a positive approach is ethically justified.

Results of TAAs for second-generation stent grafts arise from multicenter international registries such as EUROSTAR, GORE-TAG, and TTR^{33,39,40} In the EUROSTAR (European Collaborators on Stent Graft Techniques for Thoracic Aortic Aneurysm and Dissection Repair)³⁹ registry, 249 of 443 patients had a degenerative thoracic aneurysm. Primary technical success was obtained in 87%; device-related complications occurred intraoperatively in 16% of patients and arterial injuries in 2.4% of patients. Neurological events consisted of paraplegia/paraparesis (4.0%) or stroke (2.8%). Postoperative systemic problems included cardiac, pulmonary, and renal function impairment and occurred in ~25% of patients. Perioperative mortality rates ranged from 5.3% in elective procedures and from 28% in emergency endovascular repair. Late survival after 1-year follow-up was 80% with 2.1% of aneurysm-related deaths.

In the GORE-TAG registry,⁴⁰ 139 of 142 patients (98%) had successful implantation of endovascular device with a perioperative mortality rate of 1.5%. Major complications included stroke (4%), paraplegia-paraparesis (3%), and vascular trauma (14%). Device-related complications such as migration, components disconnection or wire fractures were reported in 10 to 14% of cases during the 2 years of the follow-up period. Overall 2-year survival rate was 75% with an aneurysm-related mortality of 2%.

The TTR (Talent Thoracic Retrospective Registry)²⁹ registry collected data from 457 patients (344 elective and 113 emergency): 137 of them had a degenerative aortic aneurysm. Perioperative overall mortality rate was 5% with an in-hospital complication rate of

Figure 4 Multidetector computed tomography volume-rendered (A,B), axial (C,D), and multiplanar reconstruction (E,F) images of a large thoracic aortic aneurysm before (A,C,E) and after (B,D,F) endovascular treatment. The aneurysm sac is completely excluded from blood flow by the endograft (star).

Figure 5 Multidetector computed tomography volume-rendered (A) and multiplanar reconstruction (B) images showing a type II endoleak (arrow) arising from a tributary intercostal artery (arrowhead). This type of endoleak generally resolves spontaneously.

3.7% for stroke, 1.7% for paraplegia-paraparesis, and 3.3% for vascular injuries. Device-related complication has been reported in 2.4% of cases. Survival rates at 1, 3, and 5 years were, respectively, 91%, 85%, and 77.5%, with an aneurysm-related mortality of 2.6%.

Only a few single-center series are focused on thoracic atherosclerotic aneurysms.^{41–44} In general, the technical success rate ranges from 91% to 100% and 1-year survival rate from 91 to 97.5%. Similar favorable results were reported by other authors, even if single-center series mostly report data from different aortic disease such as dissection and aneurysm or penetrating atherosclerotic ulcer.^{45,46} The most frequently reported complications in EVT were stroke and paraplegia, ranging, respectively, from 2.8 to 3.7% and from 1.7 to 14%.^{29,39,40}

In our personal experience, founded on 43 patients affected by TAA, 41 elective and 2 emergency, technical success rate was 95% with no in-hospital mortality. Major complications consisted of paraparesis in 2 patients and paraplegia in 1 patient. Stent-related mortality (aortic rupture and aortoesophageal fistula) occurred in 2 patients during follow-up. Aortoesophageal fistula is a recognized complication due to the close contact of the large aneurysm sac with the esophagus: It is thought that peri sac inflammatory reaction to the stent graft as a foreign body leads to adventitial erosion and fistula formation.

There is still insufficient data in the literature about the efficacy of EVT in thoracic inflammatory aneurysm (only few case reports), but data on EVT in abdominal inflammatory aneurysms suggest that EVT is feasible, excludes the aneurysm effectively, and reduces periaortic fibrosis and renal impairment in most patients

with very low periprocedural and midterm mortality and an acceptable reintervention rate.^{47–49} Even if results of different published experiences are encouraging, there is still no good scientific evidence about impact on long-term survival of EVT in chronic aortic diseases.

CONCLUSION

In the choice between surgical or endovascular repair of atherosclerotic TAAs, many factors must be considered, including clinical conditions, comorbidities, anatomical situation, efficacy of materials, and last, but not less important, experience of working groups.

Early and midterm results are encouraging. But long-term results are necessary to assess definitively the reliability of stent-graft materials and improvement in patient survival.

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