A case report of successful endovascular repair of a giant 15 cm diameter asymptomatic thoracic aortic aneurysm

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ABSTRACT

INTRODUCTION: Giant thoracic aortic aneurysms (TAA) are extremely uncommon, and there are only a few cases reported in the literature. Most patients presented with symptoms before the size of the aneurysm reached a magnitude >10 cm, and most of the reported cases were treated with open repair. PRESENTATION OF CASE: Here we report a 15 cm asymptomatic thoracic aortic aneurysm of a 72-year-old male patient, treated successfully with thoracic endovascular aortic repair (TEVAR). The patient was discharged asymptomatic on postoperative day 2. DISCUSSION: Only 20 case reports of giant TAA were found in the literature, and this is the biggest TAA reported treated with TEVAR. This procedure is a promising treatment as morbidity and mortality is lower when compared with open aortic repair (OAR).

CONCLUSION: Even though there is limited documented experience, use of TEVAR seems a safe and promising option in the treatment of giant thoracic aneurysms as presented in this case.

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1. Introduction

Thoracic aortic aneurysm (TAA) is defined as a loss of parallelism of the aortic walls, resulting in saccular, fusiform, or diffuse dilation, 1.5 times greater than the superjacent aorta [1]. Giant TAA is considered when widening exceeds 10 cm in diameter [2]. Thoracic aneurysms affect 10 of every 100,000 elderly adults, and are less common than their abdominal counterparts [3,4].

We present a case of a 72-year-old male presenting a 15 cm asymptomatic TAA, successfully treated by TEVAR. A review of the literature is also presented.

The work has been reported in line with the SCARE criteria [5].

2. Presentation of case

A 72-year-old male with no past history of smoking, and with medical history of hypertension, dyslipidemia and benign prostate hyperplasia treated with angiotensin-converting-enzyme inhibitor, atorvastatin, tamsulosin, and finasteride respectively, presented with a giant descending thoracic aortic aneurysm. Diagnosis was incidentally found after a CT-scan was performed for polycystic kidney disease. Tomography revealed an unruptured aortic aneurysm, affecting the distal part of the aortic arch and the descending aorta, with a maximum diameter of 15.6 cm (Fig. 1A, B). The patient didn’t have any mass effect manifestation such as dyspnea, cough, chest pain, or any other symptom. At that time, the patient underwent a laparoscopic left nephrectomy, which went unremarkable, at a small town 1000 miles away from our teaching hospital.

The patient arrived to our hospital two months after the kidney surgery. He had a complete cardiac assessment with no abnormalities in cardiac function, and serum creatinine of 0.9 mg/dl. No contraindications for surgery were presented, and TEVAR was performed. An open approach to the right common femoral artery was done under general anesthesia. A Medtronic-Valiant 34-30-200 thoracic endograft (Santa Rosa, CA Medtronic) was placed distally to the left subclavian artery and a Medtronic-Valiant 30-30-200 thoracic endograft (Santa Rosa, CA Medtronic) was positioned proximally to the origin of the celiac trunk, with 5 cm overlapping. No blood pressure reduction during deployment was needed, nor CSF drainage. An angiogram showed complete exclusion of the aneurysm without endoleaks (Fig. 2).

Patient recovered satisfactorily and was discharged on postoperative day two. No complications were seen in the CT angiography at four-month follow-up (Fig. 3). At 24-month follow up the patient is doing well without complications.

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3. Discussion

Most TAAs occur in the ascending aorta followed by the descending thoracic aorta, and the aortic arch. The average age at the time of diagnosis is around the 60–70 years range [4,6,7]. Women with degenerative TAA have greater aneurysm growth rates than men, independently of body size or other clinical variables [7].

Pathophysiology of TAA formation involves the process of cystic medial necrosis, where focal degeneration of the elastic and muscle tissue within the tunica media of the aortic wall occurs. The aortic wall subsequently weakens and dilates as a result of the high pressure of intraluminal blood flow [6]. This definition differentiates an aneurysm from a false aneurysm, with the latter being a perivascular pulsatile hematoma secondary to a vessel injury often seen after endovascular procedures. A third type of aneurysm is the mycotic counterpart, which is defined by the presence of two or more of the following features: sepsis, positive blood culture, positive culture from the aneurysmal wall, or a characteristic radiological appearance [8]. Another type of aneurysm is the one following an acute event of aortic dissection.

In this case, the patient was diagnosed with hypertension 30 years before intervention, which constitutes the main risk factor predisposing for TAAs, aortic dissection and rupture, accounting for 50–60% of deaths. Patients with aneurysms greater than 10 cm have a 5-year survival of 15% [2,4,7]. In retrospect, our patient’s aneurysm should have been repaired first before performing the nephrectomy due to the high risk of rupture that it presented.

Signs and symptoms that might present with a TAA include a diastolic murmur or, less often, patients may present with congestive heart failure. On the other hand, giant TAAs may suffer a local mass effect, such as compression of the trachea or mainstem bronchus, provoking fatigue, nausea, cough, dyspnea, wheezing, chest pain, or recurrent pneumonitis [1,6,9]. Additionally, typical symptoms of aortic rupture include the abrupt onset of severe pain in the chest, neck, back, and/or abdomen [6]. Aortoesophageal fistulae (AEFs), which have also been described as complications in giant TAA, are classified as being either primary or secondary. The first are the results of intrinsic disease, such as TAA, esophageal cancer, mediastinal tubercular infections, or incurred injury from foreign body ingestion, trauma or caustic erosions as from lye consumption, whereas the latter are communications between the esophagus and the repaired aorta. Chiari’s triad for this fatal disor-

Fig. 1. a) Coronal MPR CT image obtained with intravenous contrast in arterial phase shows a sacular aneurism of the descending aorta, with the presence of an intramural thrombus (arrows). There is no extravasation of the contrast media from the true lumen (arrowhead). b) Axial MPR image shows calcification of the aortic wall (arrowhead). The true lumen (arrowhead) measures 7.4 cm; the aneurysm measured in the perpendicular plane has a diameter of 15.6 cm.

Fig. 2. TEVAR (A) Invasive angiography depicts the thoracic aneurism before repair. There is no extravasation of the contrast. (B) Invasive angiography posterior the placement of two endoprosthesis. There are no endoleaks.

Fig. 3. CT angiogram at a four-month follow up. The endoprosthesis is well positioned with no endoleaks.
| Age (yrs), Sex | Reference        | Size (transverse diameter), Location | Presentation                          | Comorbidities and risk factors | Type of repair                     | Outcome                        |
|---------------|------------------|--------------------------------------|---------------------------------------|--------------------------------|-----------------------------------|----------------------------------|
| 75, F         | Refaat et al.    | 10cm, ascending                      | Dyspnea and impaired consciousness    | None                           | Non-surgical                      | Recovers short-term. unknown     |
| 63, M         | Wang et al.      | 10.3 cm aortic arch                  | Cough, hoarseness and dyspnea for more than 1 week | None                           | Non-surgical                      | Dies same day                    |
| 61, M         | Enríquez-Puga et al. | 11.3 cm, ascending (root) | Dyspnea and chest pain asymptomatic | aortic prosthesis, aortic valve prosthesis | Open repair. Bentall procedure, Open repair. Hemashiel woven graft 34 mm | Discharged POD 8                 |
| 28, M         | Gónçalves et al. | 16cm, ascending                      | Dyspnea                               | Hypertensive                    | Non-surgical                      | Discharged POD 10                |
| 70, M         | Philippakis      | 10cm                                 | back pain                             | Renal failure and cardiac insufficiency smoker, hypertensive, CKD, COPD | Open repair. Bentall procedure | Discharged POD 3                 |
| 76, F         | Lamrani et al.   | 11cm, decending                      | Dyspnea, cardiac insufficiency        | None                           | Open repair. Bentall procedure | Discharged POD 7                 |
| 75, M         | Garrido et al.   | 15cm, arch                           | Cardio-vocal syndrome: dysphonia, dysphagia, dyspnea, chest pain | None                           | Open repair. Bentall procedure | Dies 12 days after evaluation    |
| 77, F         | Jmaa-Hela et al. | 13.97 cm, ascending (root)          | Dyspnea                               | Hypertension, aortic valve calcification, Marfan syndrome | Open repair. Bentall procedure | Discharged POD 7                 |
| 78, F         | Adebekanne et al.| 10.2 cm, ascending                   | Dyspnea                               | Syphilis.                       | Open repair. Bentall procedure | Died in OR                       |
| 33, M         | Shah et al.      | 13cm, ascending                      | Asymptomatic                          | None                           | Open repair. Bentall procedure | Died 12 days after evaluation    |
| 76, M         | Tomey et al.     | 11.5 cm, ascending                   | Dyspnea, presyncope; aortitis and atherosclerosis | None                           | Open repair. Bentall procedure | Died 12 days after evaluation    |
| 76, M         | Rajab et al.     | 11.4 cm ascending                    | Dyspnea and leg swelling, lumbalgia, nausea, fatigue | None                           | Open repair. Bentall procedure | Died in OR                       |
| 39, M         | Topcuoglu et al. | 15cm, descending                     | Dyspnea                               | Hypertension, aortic coarctation | Open repair. dacron graft 16 mm | Died in OR                       |
| 85, F         | Kaptanakis et al. | 14.8 cm, descending               | dyspnea, dysphagia,                   | None                           | Non-surgical                      | Died in OR                       |
| 88, F         | Okura et al.     | 10.5 cm, ascending                   | Asymptomatic                          | None                           | Non-surgical                      | Died in OR                       |
| 66, M         | Fatimah et al.   | 11cm, ascending                      | Dyspnea                               | aortic valve regurgitation, mitral valve regurgitant NYHA III, permanent AF, CKD, DMZ, AAA repair 10 years earlier | Open repair. Bentall procedure | Died 12 days after evaluation    |
| 64, M         | Pietrzyk et al.  | 10.5 cm, ascending and aortic arch. | Dyspnea                               | None                           | Open repair. bentall procedure | Died 12 days after evaluation    |
| 72, M         | Moutakalllah et al. | 11cm, ascending                    | Dyspnea, orthopnea, SVCS              | Heart failure                   | Open repair. bentall procedure | Died 12 days after evaluation    |
| 82, F         | Cerea et al.     | 11cm, ascending and aortic arch.    | Acute chest pain                      | Hypertension, Diabetes          | Open repair. bentall procedure | Died 12 days after evaluation    |
| Unknown       | Sansone et al.   | 13cm, ascending                      | Asymptomatic                          | Past history of aortic valve replacement | Open repair. bentall procedure | Died 12 days after evaluation    |

SVCS: Superior Vena Cava Syndrome. POD: Post Operative Day. AF: atrial fibrillation. AAA: Aortic Abdominal Aneurysm. CKD: Chronic Kidney Disease. DM2: Diabetes Mellitus Type II.
The patient was diagnosed during evaluation for polycystic kidney disease. It is known that most patients with TAA are symptomatic and diagnosis is made incidentally during imaging studies for additional reasons [1,2,6,9]. Contrast enhanced CT scan and MR angiography are the preferred imaging methods to assess aneurysms, being both the gold standard for diagnosis [1,6].

Patients with aneurysms smaller than 6 cm are generally not candidates for surgery, unless they have symptoms or they present comorbidities, therefore they may be treated medically. Elective surgery may be carried out at a size of 5.5 cm for ascending and 6.5 cm for descending aortic aneurysms, repair is also suggested for patients with documented aneurysm growth of >1 cm per year. Propranolol has shown significantly slower rate of aortic dilatation, fewer aortic events, and lower mortality than treatment with non-β-blocker therapy [6,9].

TEVAR has been successfully performed under either general anesthesia (GA) and regional anesthesia (RA). The advantage of RA is that it allows the patient to remain awake, avoid tracheal intubation, and provide postoperative pain relief. Factors favoring GA include an endovascular repair with planned fenestrated or branched endografts, expecting a long technique duration; a need for debranching procedures or for aortic/iliac artery access and planned hemodynamic manipulations to create a mobile field during stent placement [12].

The most feared nonfatal complication in TAA’s repair is postoperative paraplegia secondary to interruption of the blood supply to the spinal cord [6]. The incidence of spinal cord ischemia (SCI) after TEVAR is generally less when compared to open aneurysm repair (OAR) but still occurs with a reported incidence of 0–13%; this is because blood flow to the spinal cord via distal aortic branches is not compromised during TEVAR because there is no aortic cross-clamping [13].

Cerebrospinal fluid drainage (CFD) has proven that its useful in preventing SCI [14]. For many years, we performed this preoperative measure in cases with long endovascular coverage, until systematic reviews failed to show that CFD prevents SCI in TEVAR [15]. Furthermore, many complications related to CFD have been reported [16,17]. Now, our strategy is more selective, and we only perform preoperative CFD in patients with high risk of developing SCI [18].

Only 20 case reports of giant TAA were found in the literature and detailed in Table 1. The average age was of 67.5 years (28–88). The mean diameter of aneurysm was 12 cm (10–16 cm). Sixteen (80%) patients reported symptoms. Thirteen (65%) of the patients were treated with open approach, and six (30%) patients were not operated on and only one (5%) of the patients was successfully treated with TEVAR. This case to our knowledge is the largest thoracic aneurysm treated with TEVAR.

4. Conclusion

TEVAR is an emergent therapy to successfully treat TAA. Morbidity and mortality has been reportedly decreasing by this surgical approach. Even though there is limited documented experience, use of TEVAR seems a safe and promising option in the treatment of giant thoracic aneurysms as presented in this case. There is still a lot of work to do regarding minimally invasive vascular procedures, since this may play an important role on the future of vascular surgery.
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