

Unusual Case of Giant Ascending Aortic Aneurysm Evolution of an Acute Aortic Dissection Type A Unoperated: A Case Report

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Abstract

Survival after an acute aortic dissection type A unoperated is exceptional. We present the case of a 74-years-old male patient with multiple cardiovascular risk factors, presented mainly with severe dyspnoea of increasing intensity. Echocardiography and computer tomography angiography showed a large ascending aortic aneurysm (with a diameter of 10.8 cm) without aortic valve insufficiency, which started at 7 cm above the sinotubular junction, with a parietal thrombus occupying more than 30% of the circumference and its distal end at 5 cm after the aortic arch. This aneurysm was due to the evolution of an acute aortic dissection type A that was diagnosed five years prior to the current admission, which was not operated because the patient refused the surgical procedure at that time. Also, severe mediastinum displacement to the right with compression effect on the lung was revealed on the computer tomography. There are just a few cases in the literature diagnosed after acute aortic dissection type A unoperated who survived so long. In this context, he underwent surgical ascending aortic replacement with a prosthetic graft. The patient's postoperative evolution was uneventful.

Keywords: Aortic aneurysm; Ascending thoracic aortic aneurysm; Aneurysm rupture

Introduction

The evolution of acute type A aortic dissection to lethal complications is well-known. Exceptional cases which for various reasons are not operated and survive are rare. Evolution to false lumen thrombosis and transformation of dissection into AAA is a rare but favourable development. AAA has an incidence of 6 cases per year per 100,000 inhabitants [1] half of them, untreated evolving to dissection, rupture and death in 48 hours, and those undergoing emergency surgery have 15% to 26% mortality [2,3]. The main predictors of rupture are the aneurysm size >6.5 cm and its expansion rate >5.5 mm per year [4,5]. The AAA grows at an average rate of 0.10 cm per year [6]. Elective surgery for ascending aneurysm lowers mortality to only 2.5% [6]. The most common cause of ascending aortic aneurysms is the same as for acute type A aortic dissection, cystic medial degeneration [1]. Risk factors include atherosclerosis, connective tissue disorders (Marfan's syndrome, Ehlers-Danlos syndrome), vasculitis (giant cell arteritis, Takayasu's arteritis), infections (syphilis, tuberculosis, human immunodeficiency virus), cystic medial necrosis, and post-surgical. Some patients with AAA remain asymptomatic for a long period of time, more frequently symptoms in evolution are related to other pathology that accompanies the AAA like aortic insufficiency, heart failure, local compression of adjacent structures or even systemic embolization [7]. Some patients can present dull pain or tenderness in the chest, shortness of breath, pain in the back, jaw, or neck, cough or hoarseness. In other patients, the onset of symptoms corresponds to dissection or rupture, usually a sudden, sharp pain in the chest that extends to the back. Compression of surrounding structures has been reported to lead to complications such as lung collapse [8], superior vena cava (SVC) syndrome, and airway compression [9].

Case Presentation

A 74-years-old male patient with longstanding systemic hypertension. This patient had relevant cardiovascular risk factors (obesity, smoker, hyperlipidemia). 5-years prior to this admission he was diagnosed with acute aortic dissection type A. At that time transthoracic echocardiography (TTE) showed an intimal flap in the ascending aorta and the diameter of ascending aorta was 58 mm. At that time the patient

was advised for surgical management, but he and his relatives did not give consent for surgery. In evolution the patient presented dyspnea on exertion (NYHA class II-III), palpitation and moderate intensity chest pain. In the past six months he started complaining of intense chest discomfort and pain, shortness of breath, and current activities were difficult to do because of severe fatigue. At admission, the patient was hemodynamically stable with signs of hypoxia, anxiety, confusion, and restlessness. Oxygen saturation levels were lower than 88%. His pulse rate was 95 beats per minute and the respiratory rate was 25 breaths per minute. His blood pressure (BP) was 132/86 mmHg. The first imaging exam performed was a chest X-ray, postero-anterior view which showed widening of the mediastinum (enlarged cardiac silhouette and aortic notch). The trachea was deviated to the right side and a collapsed right lower lobe was noticed. In the case presented, AAA increased to partially obstructing the right main bronchus and producing atelectasis in the right lung. The TTE showed an aortic tricuspid valve with a mild aortic valve insufficiency, a vena contracta of 2 mm, and a large ascending aortic aneurysm, with the following 2D diameters: aortic ring=25.6 mm, Valsalva sinuses=39 mm, ascending aorta=80 mm, aortic arch=35 mm, descending aorta=30 mm, ascending aortic hematoma=55mm. The left ventricle was enlarged, with ejection fraction (calculated by Simpson's method) of 60%. Subsequently, the patient had a contrast-enhanced multi-slice computed tomography (CTA) of the chest for detailed evaluation and this also showed giant ascending aortic aneurysm (10.8 cm) partial thrombosed, compressing the right lung (Figure 1).

We proposed surgical intervention to the patient and this time he accepted. The patient underwent a median sternotomy, the aneurysm

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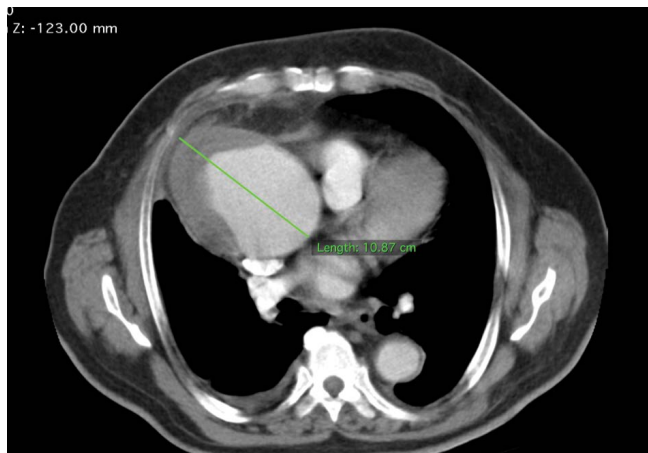


Figure 1: CTA of the chest showed giant ascending aortic aneurysm (10.8 cm) partial thrombosed, compressing the right lung.

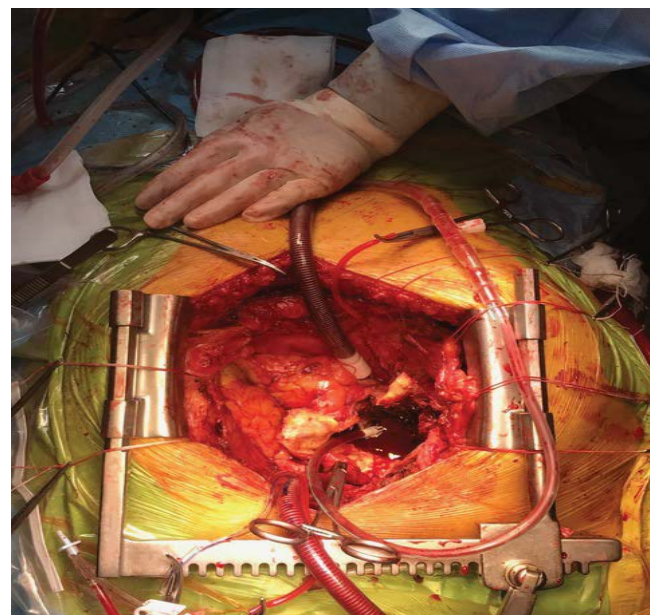


Figure 3: Intraoperative image of the opened aortic aneurysm.

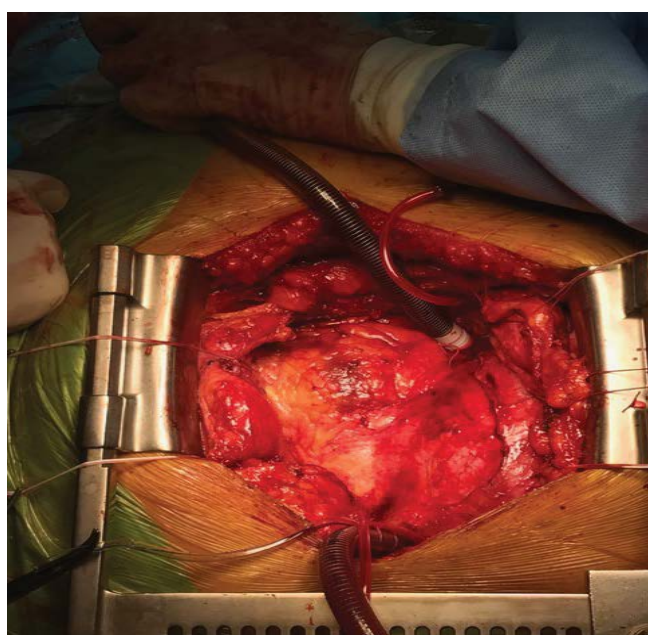


Figure 2: Intraoperative image of the aneurysm complete covering the right atrium.

being so large that it completely covered the right atrium and did not allow venous cannulation. Total cardiopulmonary bypass (CPB) was established by right femoral artery and left femoral vein cannulation. CPB was started and moderate cooling phase initiated. We continued with complete dissection of the aneurysm from the surrounding tissues and we dissected the distal portion of right atrium at the atriocaval junction and a second venous cannula with the tip facing towards the middle of the right atrium was inserted (Figure 2).

The heart was vented through the right superior pulmonary vein. The ascending aorta was cross-clamped distal in a normal sized portion when the heart fibrillated. The proximal ascending aorta was opened and we drained about 500 ml of clots (Figures 3 and 4).

Myocardial protection was insured by antegrade manner using cold



Figure 4: Evacuated aortic hematoma (approximately 500 ml).

blood cardioplegic solution. We performed complete surgical resection of the aneurysm, with implantation of a Dacron aortic graft Interguard no.32, with good surgical final result. The patient had favorable evolution and was discharged at 9 days postoperative (Figure 5).

Discussion

It is uncommon for a patient diagnosed with acute aortic dissection type A to refuse the treatment. In the literature, there are few informations regarding this type of patients. Moreover, it is known

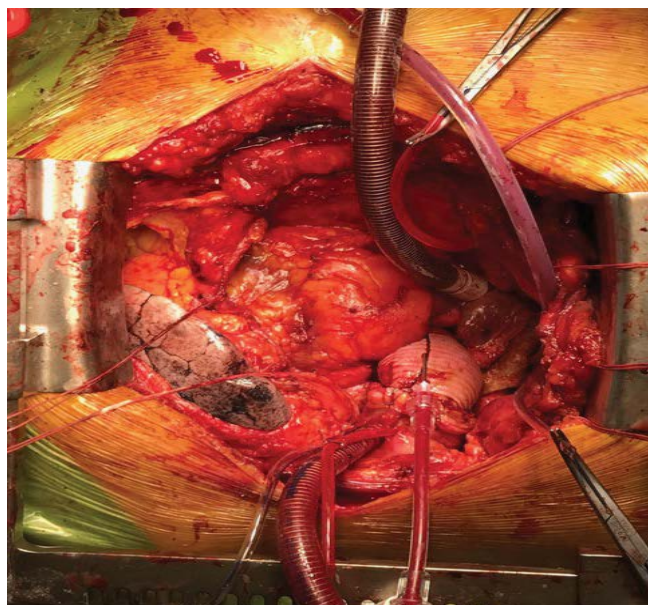


Figure 5: Intraoperative image of the Dacron prosthesis no. 32 used to replace the aortic aneurysm.

that in its natural evolution, without treatment, acute type A aortic dissection is reported to have a mortality rate to almost 80% by the end of the second week [10]. We admitted the patient 5-years after the original diagnosis of acute aortic dissection accusing mainly chest pain and worsening respiratory symptoms. 6 months previously to this admission the patient underwent a CT examination and in comparison, with the one performed in our clinic, the aortic aneurysm did not increase in dimensions, which confirms slow grow rate estimation given by literature [6]. Also, the false lumen was thrombosed, and the aortic hematoma remained at the same size. The giant proportion of the ascendent aortic aneurysm determined compression over superior vena cava without any clinical signs of superior vena cava syndrome, the deviation of trachea to right and compression on right inferior pulmonary lobe with atelectasis. The difficulty in performing every day activities and severe dyspnoea convinced the patient to accept the surgery. Open surgical repair remains the standard approach for treating giant aortic aneurysm and results from elective operation are more than satisfying [6]. To minimize embolic risks a series of measures should be taken. Stroke risks in surgical elective repair is estimated to about 8% [6]. Gentle aortic manipulation and total dissection of the aneurysm under extracorporeal circulation, complete resection of ascending aortic dilatation, and carbon dioxide flooding of the operative field can minimize the embolic risk. To address the partial obstructed right main bronchus, endobronchial stents may be used to relieve the bronchial

obstruction [8,11]. In our case because the pulmonary obstruction was extrinsically and incomplete the total surgical resection of the aneurysm and ventilation of the patient with high positive pressure in intensive care unit lead to complete expansion of the lungs.

Conclusion

Survival after a diagnose of acute aortic type A dissection without surgical treatment is extremely rare. If death does not occur through rupture of the free wall and tamponade, the subsequent dilatation of the aortic wall can reach impressive dimensions which leads to the compression of the mediastinal vital elements. In the presented case survival was possible due to the evolution towards not only dilatation of the ascending aorta, but also to thrombosis of the false lumen, thus conferring the aortic wall an additional resistance which allowed the ascending aorta to reach giant dimensions without rupture and severe respiratory failure completing the clinical symptomatology. Complete surgical resection of the aneurysm, with implantation of a Dacron aortic graft is the only surgical solution in such situations.

References

1. Bonow RO, Mann DL, Zipes DP, Libby P (2012) Braunwald's heart disease: A textbook of cardiovascular medicine. 9th ed. Philadelphia: Elsevier Saunders p: 7.
2. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, et al. (2000) The International Registry of Acute Aortic Dissection (IRAD): New insights into an old disease. *JAMA* 283: 897-903.
3. Ehrlich MP, Ergin MA, McCullough JN, Lansman SL, Galla JD, et al. (2000) Results of immediate surgical treatment of all acute type A dissections. *Circulation* 102 : 1-248.
4. Oladokun D, Patterson BO, Sobocinski J, Karthikesalingam A, Loftus I, et al. (2016) Systematic review of the growth rates and influencing factors in thoracic aortic aneurysms. *Eur J Vasc Endovasc Surg* 51: 674-681.
5. Yiu RS, Cheng SW (2016) Natural history and risk factors for rupture of thoracic aortic arch aneurysms. *J Vasc Surg* 63: 1189-1194.
6. John A (2002) Elefteriades, natural history of thoracic aortic aneurysms: Indications for surgery, and surgical versus nonsurgical risks. *Ann Thorac Surg* 74: 1877-1880.
7. Goldstein LJ, Davies RR, Rizzo JA, Davila JJ, Cooperberg MR, et al. (2001) Elefteriades, MD stroke in surgery of the thoracic aorta: Incidence, impact, etiology, and prevention. *J Thorac Cardiovasc Surg* 122: 935-945.
8. Yap KH, Sulaiman S (2009) Pulmonary atelectasis from compression of the left main bronchus by an aortic aneurysm. *Singapore Med J* 50: 247.
9. Duke RA, Barrett MR, Payne SD, Salazar JE, Winer-Muram HT, et al. (1987) Tonkin compression of left main bronchus and left pulmonary artery by thoracic aortic aneurysm. *AJR* 149: 261-263.
10. Coady MA, Rizzo JA, Goldstein LJ, Elefteriades JA (1999) Natural history, pathogenesis, and etiology of thoracic aortic aneurysms and dissections. *Cardiol Clin* 17: 615-635.
11. Heringlake M, Schumacher J, Sedemund-Adib B (2002) Bronchial stenting and high-frequency percussive ventilation treatment of the descending aortic aneurysm-induced atelectasis of the left lung. *Anesth Analg* 95: 1189-1191.