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Giant Ascending Aortic Aneurysm: A Case Report and Review of the Literature

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Abstract **Case Report**

Giant aortic aneurysm is defined as aneurysm in the aorta greater than 10 cm in diameter. It is a rare finding since most patients will present with complications of dissection or rupture before the size of aneurysm reaches that magnitude. Etiological factors include atherosclerosis, Marfan's syndrome, giant cell arteritis, tuberculosis, syphilis, HIV-associated vasculitis, hereditary hemorrhagic telangiectasia, and medial agenesis. Once diagnosed, prompt surgical intervention is the treatment of choice. Although asymptomatic unruptured giant aortic aneurysm has been reported in the literature, there has not been any case of asymptomatic giant dissecting aortic aneurysm reported in the literature thus far. We present a case of a 62 year old man with a 14 cm of diameter aneurysm of the ascending aorta and aortic arch without impact on the aortic root.

Keywords: Aortic; Aneurysm; Vascular surgery.

INTRODUCTION

Giant ascending aortic aneurysm, defined as an aneurysm more than 10cm in diameter [1]. This entity is still very rare with an atypical clinical presentation, and few cases have been reported in the literature. A giant aneurysm of the ascending aorta is an urgent indication for surgery. Because this vessel is already fragile, it receives the systolic volume in all its ejection force without any damping, hence a high risk of rupture. Surgical management of these aneurysms is challenging with high perioperative mortality [2].

We present the case of a 62 year old man with a 14 cm of diameter aneurysm of the ascending aorta and aortic arch without impact on the aortic root.

CASE REPORTS

A 62-year-old male admitted to the emergency for dyspnea and chest pain persistent by six months. In his medical history the only cardiovascular risk factors were smoking, there was no significant family history. No notions of syphilis or HIV, no Marfan's disease or aortic bicuspid, no notion of thoracic trauma, no known arteritis. On physical examination, her room air saturation was 96%, her heart rate was 78 beats per minute, and her blood pressure was 110/80 mm Hg.

The cardiac, vascular and respiratory examination was normal. The electrocardiogram noted a regular and sinus rhythm with heart rate at 75 cpm.

Chest X ray revealed a mediastinal widening. Thoracic CT scan showed a huge thrombosed ascending aortic aneurysm measuring 140mm × 68mm × 130mm pushing back the right lung with chronic arterial dissection (Fig 1).

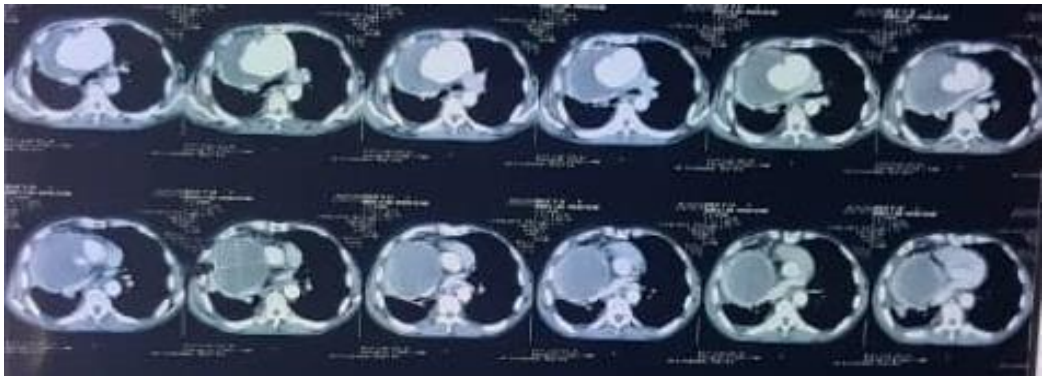

Fig 1: Thoracic CT scan: Giant aneurysm of the ascending aorta

Transthoracic echocardiography (TTE) revealed an the heart chambers that vary in size and conserved features, an ejection fraction of the left ventricle in 65%, aortic insufficiency and tricuspid regurgitation minimal, no pulmonary hypertension , initial aorta not dilated, the Valsalva sinus at 28 mm, the sino-tubular junction at 31 mm and the tubular portion at 31 mm.

Coro-scanner was not performed for technical reasons. Echodoppler of the arteries of the supra-aortic trunks does not find aneurysm or stenosis. Abdominal aortic ultrasound does not show any detectable vascular abnormalities. Echodoppler of the arteries of the lower limbs is without abnormality. The serologies, syphilitic, HIV 1 and 2 and viral hepatitis B and C are negative.

Because of the huge diameter of the aneurysm, we decided to perform emergent surgical repair. Femoral arteriovenous cannulation was established to decompress the aneurysm before sternotomy and systemic cooling was started. Then, a median sternotomy was performed and the pericardium was opened, cannulation of the trunk brachiocephalic (TABC) providing cerebral perfusion, starting CEC moderate hypothermia at 28°C. On exploration, the trunk of the pulmonary artery is compressed, the heart is pushed back downwards by a giant aneurysm of the ascending aorta and the right hemi-arch (Fig 2). The aortic valve, Valsalva sinus, and sino-tubular junction were normal. Clamping of the descending aorta at its origin, anterograde cardioplegia via the coronary ostia. The procedure consisted of flattening the aneurysm and interposing a right dacron prosthesis between the ascending aorta and the sino-tubular junction. Distal anastomosis between the prosthesis, the ascending aorta and the hemi-arch upstream of the TABC. Proximal anastomosis between the prosthesis and the sino-tubular junction (Fig 3). CEC time at 155 min and ischemia time 64 min with 10 min of circulatory arrest.

The patient left the block under stable hemodynamic conditions under low inotropic support dose which was stopped four hours after the block.

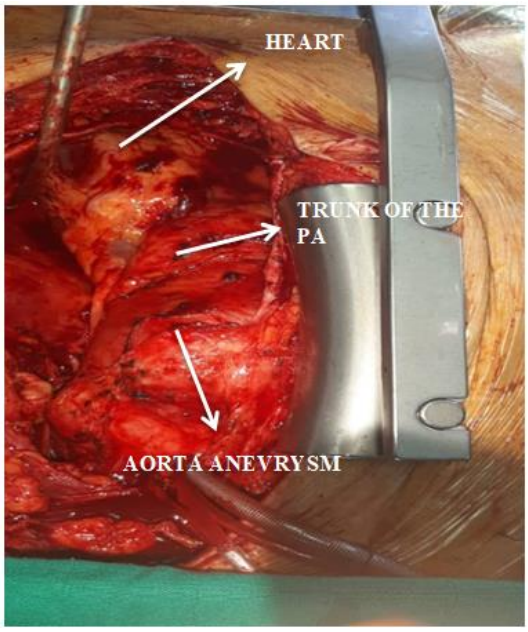

Fig 2: Giant aneurysm of the ascending aorta

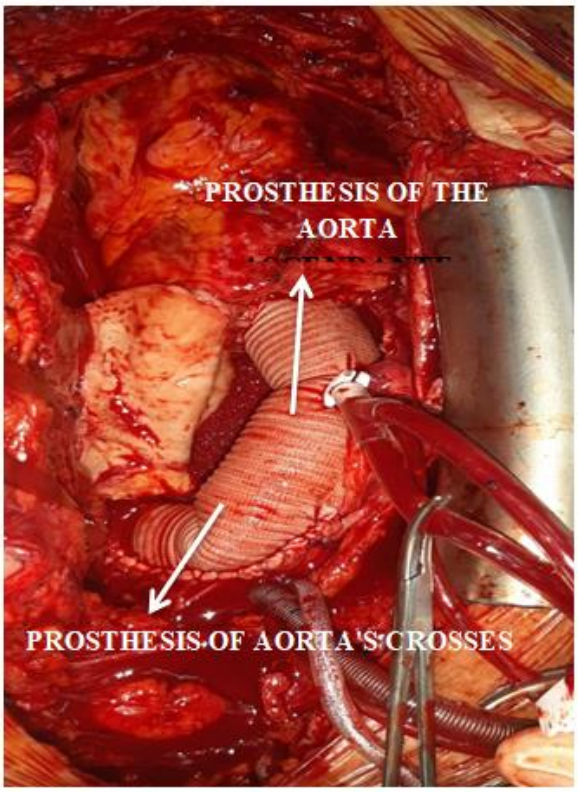

Fig 3: Prosthesis of the ascending aorta and the aortic arch in Dacron

The patient was extubated five hours after leaving the operating room. He presented with a cough with hemoptoid expectoration. A chest x-ray showed moderate pleural effusion on the left with no indication of drainage and low abundance on the right. A thoracic CT scan was performed, revealed an aortic prosthesis in place, a liquefied periprosthetic hemo-mediastinum without sign of active extravasation, measuring 140 mm × 95 mm extended over 168 mm and a bilateral pleural effusion (Fig 5). TTE showed a normal left ventricular function., minimal IAo, no pericardial effusion. Histological study revealed a strongly reworked arterial wall, with a partially preserved endothelial coating, with hyperplasia of the connective tissue of the intima and fibrosis and edema of the muscle layer, suggesting an atheromatous cause (Fig 4). The cough and sputum resolved and the patient was discharged after an 8 day hospital stay.

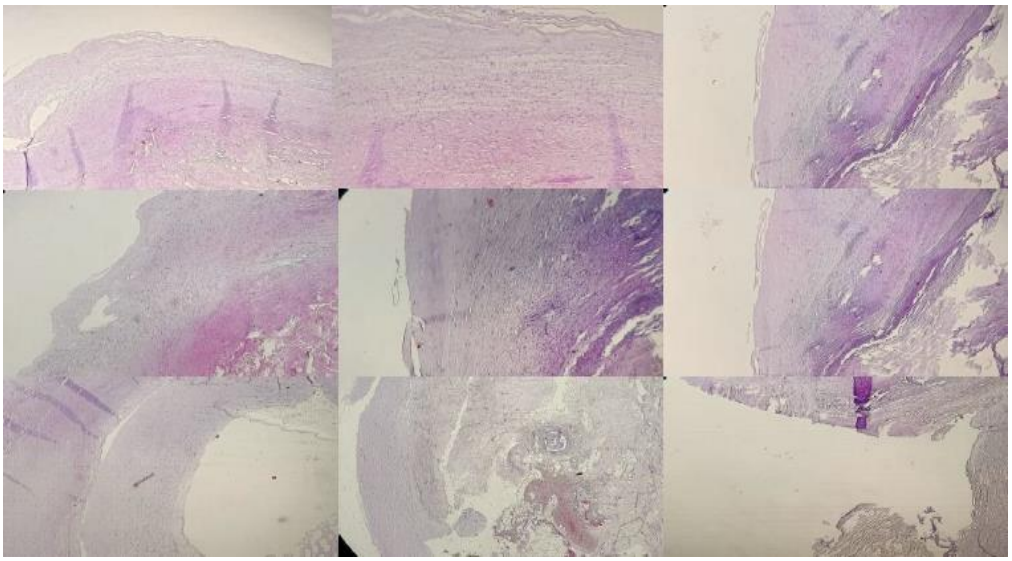

Fig 4: Histology: Strong reworking of the arterial wall. Partially preserved endothelial coating. Hyperplasia of the connective tissue of the intima and fibrosis and oedema of the muscle layer

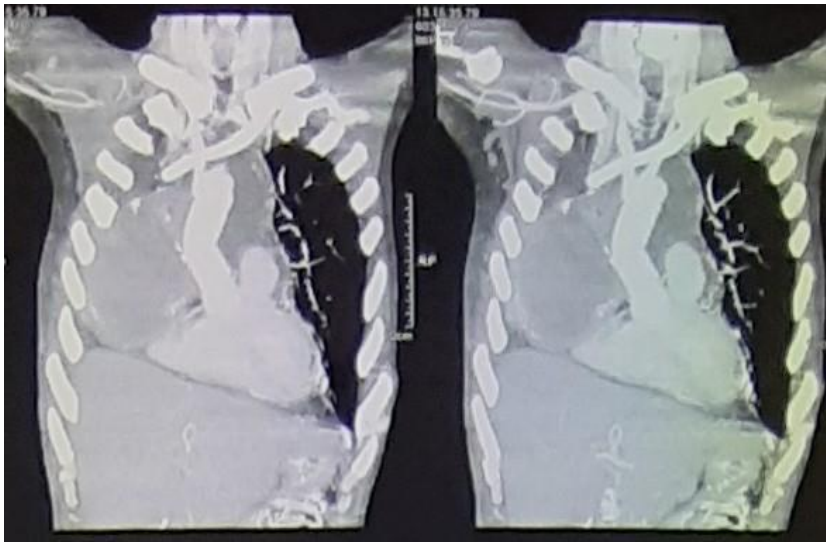

Fig 5: Hematoma in a residual cavity of the aneurysm of the ascending aorta and the arch

DISCUSSION

Giant aneurysm of the ascending aorta, defined by an aneurysm diameter greater than 10 cm, is rare. With an atypical clinical presentation. In our case, the patient was admitted to the emergency for dyspnea and chest pain persistent by six months Vuckovic [3] reported cases of giant aneurysms that were completely asymptomatic and presented with cyanosis. Moutakiallah *et al*., [2, 4] reported a case of an aneurysm of the ascending aorta measuring 11 cm of diameter revealed by a superior vena cava syndrome. The risk of rupture or dissection was found to be 31% when the diameter of the ascending aorta reaches 6 cm [5]. So, surgical repair is necessary, and it is a technical challenge. The most common etiological reason of AAAs in older patients is arteriosclerotic degeneration, Marfan syndrome or bicuspid aortic valve that may be associated with aortic pathology are the most common reasons in younger patients. Other etiological reasons of AAAs are trauma, aortic pseudoaneurysms, aortic dissection and several forms of vasculitic diseases such as Takayasu arteritis and giant cell arteritis. CT scan and echocardiography are the most common investigations to confirm the diagnosis of the aneurysm and precise its dimensions.

The treatment of these aneurysms is a technical challenge and carries a high morbidity and mortality. It is difficult to lay down principles for treating them as they are rare and need to be treated on an individual basis. There are nevertheless issues as in any aneurysm surgery such as approach to the aneurysm, establishment of cardiopulmonary bypass, cerebral protection, and control of bleeding and postoperative care.

Belov *et al*., [5] propose femoro-femoral circulation and cooling of patients and circulatory arrest as an approach to these patients. In our series, surgery to the ascending aorta was associated with surgery to the arch. Arch surgery is burdened with a rate of neurological sequelae of 3 to 5% [5]; mortality is 6-15% in the event of stroke [6, 7]. We operated on the aortic arch in hypothermia at 28 ° using a selective cerebral perfusion coupled with a perfusion of the viscera by the femoral route as recommended by Preventza *et al*., [5]. We made a circulatory arrest at 28° C for a safe duration of 10 min.

The mortality rate after surgery of these aneurysms remains high, and complications could be life-threatening especially neurological deficit and multi-organ failure (7). Our patient was extubated five hours after leaving the operating room, and inotropic support was stopped four hours after the block.

CONCLUSION

Giant aneurysm of the ascending aorta and lacrosse without impact on the aortic root is very rare. His surgery is very problematic because such an aneurysm has a very high risk of rupture and death in the operative field. It also requires circulatory arrest with a risk of cerebral ischemia or stroke increasing mortality by 9-15%. So the only proven means of reducing neurological sequelae during circulatory arrest are three in number: Limit the duration of the arrest; maintain minimal cerebral blood flow and Cool the brain.

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