CASE REPORT
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A GIANT ASCENDING AORTIC ANEURYSM PENDING RUPTURE

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ABSTRACT

Aneurysm of the aorta refers to abnormal dilatation of a portion of the aortic wall related to its weakening from any cause. It is a serious cardiovascular problem that can present as an acute life-threatening emergency due to dissection and rupture of the aortic wall. Though most common in the abdominal aorta, any portion of the vessel can be affected, with clinical features relevant to the area and extent of involvement. The risk of rupture increases with aneurysmal dilatations exceeding 5 cm, whereas giant aneurysms measure above 13 cm, and are rare. Reportedly, only 10-25% of patients survive rupture because of increased pre- and post-operative mortality.

A 40 years young hypertensive male presented to Rehman Medical Institute, Peshawar, Khyber Pakhtunkhwa, in December 2016, with symptomatic giant aortic aneurysm involving root and ascending aorta with aortic regurgitation. He was complaining of chest pain and dyspnea that was gradually increasing over time. Due to risk of rupture the patient was prepared for surgery and Bentall's procedure of aortic root replacement with coronary arteries button re-implantation was performed with successful postoperative uneventful course.

Keywords: Aortic Aneurysm; Aortic Rupture; Aneurysm, Ruptured; Vascular Disease.

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INTRODUCTION

The ascending aorta is a part of the aorta that starts from the aortic annulus and finishes at the take-off innominate artery. Microscopically, it can be seen as a three-layered structure that is composed of an inner smooth intimal layer, a medial layer and an outer layer of adventitia. Enlargement of the aorta is called an aneurysm. Aneurysms of the ascending thoracic aorta most often result from cystic medial degeneration (CMD); additionally, atherosclerotic vascular disease can lead to wall weakness causing subsequent dilation. CMD leads to weakening of the aortic wall which results in aortic dilatation and aneurysm formation; histologically, it appears as smooth muscle cell dropout and elastic fiber degeneration. CMD occurs normally to some extent with aging, but the process is accelerated by hypertension. Aortic aneurysms can happen without overt connective-tissue disorders. Moreover, it is now recognized that although cases of thoracic aortic aneurysms in the absence of overt connective-tissue disorders may be sporadic, they are often familial and are now referred to as the Familial Thoracic Aortic Aneurysm (FTAA) syndrome.[1,2]

It is now well established that the risk of dissection or rupture increases with aortic diameter. The aneurysmal thoracic aorta grows at an average rate of 0.10 cm per year (0.07 for the ascending aorta and 0.19 for the descending). By the time the thoracic aorta has reached a size of more than 6 cm, 31% of patients will have suffered rupture or dissection. The annual risk of rupture, dissection or death for patients with thoracic aortic sizes greater than 4 cm, 5 cm, or 6 cm, is 5.3%, 6.5%, and 14.1%, respectively.[3]

Aneurysm diameter is the main indication for elective surgical intervention, as it correlates strongly with the risk of the ascending aortic

aneurysm dissecting or rupturing.[4,5] Diameter of a normal aorta is influenced by age and body mass index.[6] Indications for replacement of ascending aorta are influenced by etiology, diameter and rate of growth of the aneurysm.[7,8]

CASE PRESENTATION

A 40 years old male presented to Rehman Medical Institute (RMI), Peshawar, Khyber Pakhtunkhwa, on December 02, 2016, with increasing chest pain and dyspnea. He was a known hypertensive patient. There was no family history of ischemic heart disease, diabetes mellitus and connective tissue disorders.

On clinical examination, pulse rate was 80 beats per minute and blood pressure was 150/80mm Hg bilaterally equal. Peripheral pulses were felt equal bilaterally. On auscultation, there was an early diastolic murmur at the base of the heart consistent with Aortic Regurgitation (AR). There were no signs of Marfan's syndrome or other connective tissue disorders.

A 12-lead surface electrocardiogram (ECG) showed left ventricular hypertrophy (LVH). On chest X–Ray, wide mediastinum and calcifications of ascending aorta were observed. Transthoracic echocardiography (TTE) revealed proximal aortic aneurysm with severe AR. CT aortography was performed for full evaluation of the aorta which showed about 8 cm of aneurysm involving root and ascending aorta touching the sternum with severe atherosclerosis and calcification (Figures 1,2).

The aortic arch diameter was in normal range. Based on aortic AHA guidelines, it was decided to do aortic replacement surgery for the patient.

The patient was prepared for Bentall-type aortic surgery. A median sternotomy was performed and right femoral artery exposed. The patient was heparinized and cardiopulmonary bypass was initiated by right femoral artery cannulation and right atrial venous cannulation. Resection and replacement of the aortic root, aortic valve and ascending aorta were done (Figure 3).

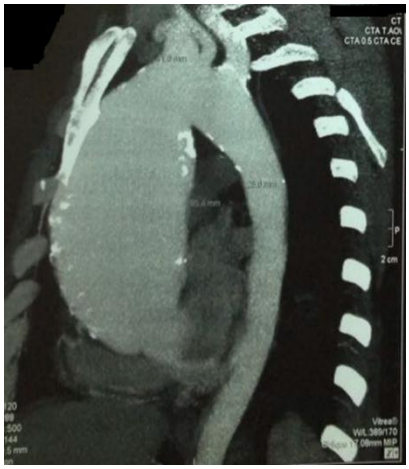

Figure 1: Large Aortic aneurysm touching the sternum with calcification of the wall

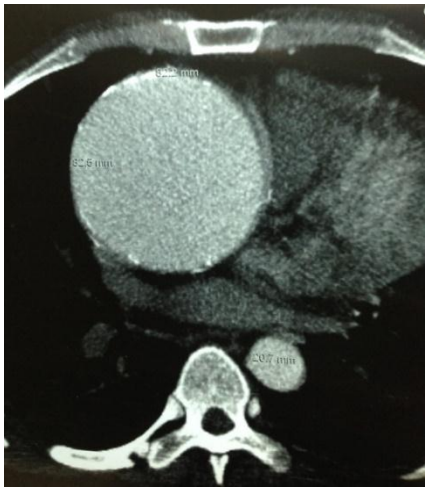

Figure 2: 8 cm ascending and aortic root aneurysm

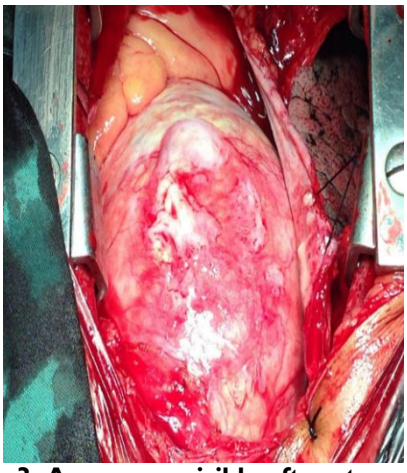

Figure 3: Aneurysm visible after sternotomy

Coronary arteries were attached to the aortic graft with the coronary button technique (Figure 4).

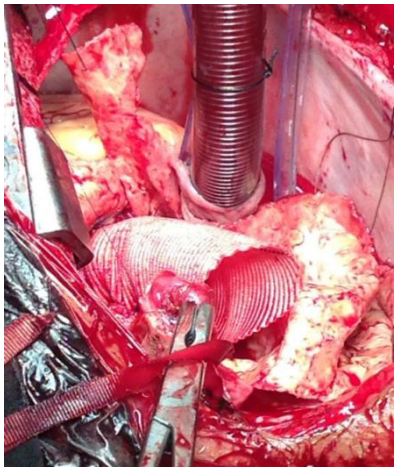

Figure 4: Aorta replaced with prosthetic graft.

Patient had an uneventful postoperative course and was discharged home.

DISCUSSION

Aortic aneurysm can affect any part of the aorta. Presence of dilation involving all three layers (intima, media and adventitia) of a segment of blood vessel, causing at least 50% increase in diameter compared to normal diameter defines a true aortic aneurysm. The natural history of aortic aneurysm is continuous growth leading to either rupture or aortic dissection. Both these can be a cause of sudden death. Aneurysm of the ascending aorta can cause aortic valve regurgitation in addition to the above-mentioned complications. Hence its operation is more complex and requires dealing with the coronary artery and the aortic valve itself. In some patients the aortic valve itself is normal but the regurgitation is because of the dilation of the sino-tubular junction. In these cases the native aortic valve can be spared. With recent advances in surgical care it can be cured by resection of the diseased aorta and replacing it with a prosthetic valve. According to the Griepp series the midterm survival of discharged patients was 93% at 1 year and 83% at 5 years. The type of the procedure is decided based on dividing the aorta into different components and then finalizing the reconstruction plan. First it is established which of the following parts are diseased (aortic valve, coronary arteries, aortic root, ascending aorta and arch of aorta). After that they all can be replaced with the help of cardiopulmonary bypass and hypothermia. Usually moderate hypothermia is enough but in cases where aortic arch is also involved then deep hypothermic arrest may also be utilized. Surgical repair is the only option available once large aneurysm are formed.

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