

ENDOVASCULAR REPAIR OF POST-TRAUMATIC PSEUDO-ANEURYSM OF DESCENDING THORACIC AORTA

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ABSTRACT:

Traumatic aortic aneurysms are rare but significant lesions with catastrophic consequences. A case of chronic aortic aneurysm of descending aorta following a blunt chest trauma presenting with extrinsic compression of left pulmonary artery, a rare and unique complication has been reported. Here we have also attempted to emphasize on the importance of early diagnosis and treatment of post traumatic aortic aneurysm to avoid lifelong misery.

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INTRODUCTION:

Thoracic aortic aneurysms resulting from non-penetrating traumas are uncommon lesions causing an immediate morbidity and mortality of 80 to 90 %. Around 1-2% of the patients who survive become life time sufferers due to the development of chronic aortic aneurysms¹. Manifestations of these aneurysms by no means are uniform and may occur years later due to incidental finding, varied symptoms or late complications (rupture or dissection)². A case of a 14 years old boy who underwent thoracic endovascular repair for a false aneurysm of proximal descending aortic thoracic aneurysm one year following a significant chest trauma.

CASE PRESENTATION:

A 14-year old boy came to our attention in emergency department of Punjab Institute of Cardiology, Lahore, Pakistan in 2016, complaining of haemoptysis for last three months. He had three to four bouts of cough containing gross amount of fresh blood. There was no associated history of fever, cough, wheeze, dysphagia, dyspnoea or any weight loss. However, on further probing, he revealed to have a chest trauma almost one year back. He was cycling when he fell on the ground and had a serious side-impact collision, followed by chest pain that gradually resolved over few days. He was otherwise fit and well; therefore, denied any hospital admission since the time of his accident. Moreover, he is a non-smoker and has no risk factor for cardiovascular diseases, and has no

family history of congenital or cardiac issues.

On examination, he was an average built young boy of normal height and weight but markedly pale. His blood pressure was 110/70 (equal in both arm), heart rate 80 bpm, temperature 36.8C and Oxygen saturation was 92%. On chest examination, there were diminished breath sounds in infra-scapular and inter-scapular regions, without any tracheal deviation. Rest of the examination was unremarkable.

Routine blood tests and cardiac enzymes were normal, except low haemoglobin and MCV. Electrocardiogram (ECG) revealed normal sinus rhythm and no evidence of ischemia. Chest X-ray (CXR) displayed a smooth superior mediastinal widening extending into the left hemi-thorax. Another important finding was an elevated left hemi-diaphragm raised up to the level of mid thorax, with underlying gastric contents (figure 1). Echocardiography showed a huge pseudo-aneurysm arising from descending thoracic aorta, distal to the origin of subclavian artery. It was curling around anterior, lateral and medial aspects of proximal thoracic aorta compressing main and left pulmonary artery (PG at MPA=30mmHg). In addition to that, there was a large thrombus lining the inner wall of aneurysm with a central patent lumen, communicating with aortic lumen. Rest of the aorta was normal from its origin till bifurcation. There were structurally normal valves and good left ventricular systolic function with an ejection fraction of 60% (figure 2).



Fig 1: Chest X-ray PA view:
Superior mediastinal widening with elevated left hemi-diaphragm



Fig 2: Echocardiography



Fig 3: 3D image CT-angiogram(axial view):
A large Pseudo-aneurysm, arising from proximal descending Aorta.



Fig 4: CT-Angiogram:
Axial view, showing relation of Pseudo-aneurysm to its surrounding structures.

Patient was then referred for CT- angiogram which also exhibited a massive pseudo-aneurysm of proximal descending and distal aorta, measuring 7mm at the neck and 82x70mm for the aneurysmal dilatation (figure 3). There were clots and calcification in the wall of aneurysm, which were evident of its chronicity. It also revealed compression of main and left pulmonary artery; however, both lung fields were clear with normal Broncho-vascular markings. There were no features suggestive of rupture or any peri-aneurysmal fluid collection. Importantly, any aorto-esophageal or aorto-bronchial communication could not be identified (figure 4).

The differential diagnosis for the cause of an

aneurysm in a young boy definitely requires consideration. There was no documented history suggestive of chronic infectious state or atherosclerosis. Additionally, his examination was not favouring Marfan's syndrome. So the most likely aetiology would be chest trauma leading to pseudo-aneurysm of descending thoracic aorta compressing left pulmonary artery. Additionally patient also had unilateral left phrenic nerve injury (asymptomatic), most likely a chronic manifestation of trauma.

Patient was admitted for surgical intervention under the care of cardiology surgical department and was transfused with 6 units of whole blood to maintain haemoglobin levels. Four weeks

after his initial presentation, he underwent stent implantation with balloon expandable 8x34mm covered CP stent. Overall the procedure was partially successful as repeat check Aortogram showed a small leakage into the aneurysmal sac. CT-angio was repeated after 48 hours which further confirmed a tiny leak at the site. It was decided to leave it as such and review the patient after two weeks' time.

DISCUSSION:

Aortic injuries following blunt traumas can be easily missed transforming into infrequent but mortal chronic aneurysms of all the injuries resulting in chronic aortic aneurysm the most frequent ones result from high speed motor vehicle collisions and fall from height. The exact mechanism hasn't been determined yet but it would be safe to say that aortic injury is a complex interplay of different mechanisms like rapid deceleration, shearing forces, osseous pinch and hydrostatic forces³.

Thoracic aortic aneurysms are distinctive in aspect that they are relatively discrete and are typically localized, calcified and saccular shaped. The most vulnerable location for aortic injury is the aortic isthmus distal to left subclavian artery. It is a site where ligamentum arteriosum attaches and it also serves as a junction between the fixed aortic arch and the mobile descending aorta. It is subjected to maximum strain due to its inflexibility and any lethal trauma can easily lead to its partial or total disruption. Among all the patients presenting with post traumatic aortic aneurysms, 80% cases involve the aortic isthmus and 70% of them are saccular in shape.

Clinical manifestations are highly dependent on the nature of injury and its impact on local haemodynamic and intrinsic factors. Majority of the patient suffer instant morbidity and mortality, and those who survive, may silently develop false aortic aneurysm. Most of them remain asymptomatic until found incidentally on imaging studies; whereas, some may manifest symptoms years later. The most common symptom is chest pain either from compression or distention, or due to an impending rupture or dissection. Aneurysm may compress surrounding structures (trachea, bronchi or oesophagus) producing symptoms like haemoptysis, stridor, wheeze, cough or dysphagia. It may even erode into surrounding structures leading to bleed. Another rare presentation is thromboembolism involving distal viscera and lower extremities.

In this case, patient had three to four episodes

of haemoptysis which can be explained by left pulmonary artery compression. The underlying mechanism can be the compromise of pulmonary circulation due to extrinsic compression of pulmonary artery which leads to compensatory over flow in the bronchial arterial tree. This causes eventual hypertrophy and thinning of bronchial arteries which can easily rupture into alveoli and bronchi causing haemoptysis. Another possibility is the turbulent flow in compressed pulmonary artery territory that favours the thrombi formation that subsequently breakoff and coughed out manifesting as haemoptysis. Furthermore, chronic turbulent flow does favour inflammatory process that further promotes neo-vascularization. These new vessels are fragile and have the tendency to rupture into the airways⁴. This needs evaluation by imaging like bronchoscopy which couldn't be attempted due to the risk of aneurysmal rupture.

The most common complications of ATAI (acute traumatic aortic injury) that have to be taken care of are dissection and aneurysmal rupture². Aneurysmal injuries of descending thoracic aorta have greater risk of complications as compare to ascending aortic due to its increased growth rate (0.19cm/year vs 0.14cm/year respectively).

ATAI demands multimodality imaging and broad knowledge of both clinician and the radiologist. Multidetector CT has proved to be an excellent imaging technique if aortic aneurysm is suspected. Further transthoracic or transoesophageal echocardiography, angiography, aortography and MRA are equivocal investigations for assessment of surrounding structures especially in cases with unusual presentations⁵.

Few guidelines focus on the management of chronic thoracic aneurysms. However, medical management is only limited for the haemodynamic stability (heart rate and blood pressure maintenance) and the mainstay of treatment is surgical intervention. According to the guideline the first line option for a descending thoracic aneurysm exceeding 5.5cm in size or a false aneurysm would be endovascular repair using graft stents. Second line choice would be the conventional open surgical repair which involves resection of the culprit lesion and graft placement with or without cardiopulmonary bypass. Complications like operative mortality, paraplegia, and vascular lesions are definitively higher with conventional approach⁶. Whereas endovascular approach has demonstrated high technical success rate, greater feasibility and low side effect profile^{7,8}. However, there are certain challenges linked to

TEVAR like endoleak, stent fracture and stent graft endothelial migration, thrombosis and improper placement of stent, which have to be taken care of.

A limitation with present evidence is that all the present knowledge is based on retrospective cohort studies and it would not be possible to conduct randomized trial on such cases due to ethical consideration. Further prospective population based studies can guide on attaining the best evidence on this issue.

In this case, we decided to go for TEVAR(thoracic endovascular aortic repair) using covered CP stent (cheatham-platinum) intended for permanent implant, which is balloon expandable. It is made of platinum/iridium, arranged in a zig pattern, covered with an expandable sleeve of ePTEF

allowing a barrier around the stent⁹. Femoral percutaneous approach was used to pass the stent. There were no intraoperative complications. Check aortogram revealed a small leak into the sac. As we over-expanded the balloon to completely cover the aneurysmal neck which might lead to reduced stent length leading to this minor residual leak. Another possibility could be a small gap between the stent boundary and aortic wall.

In conclusion, this case illustrates significant but rare consequences of a blunt, non-penetrating thoracic trauma. High level of suspicion is mandatory to avoid missing the diagnosis and timely management is necessary before fatal sequela ensues. Further research is required to have specific upto date guidelines for the management of chronic aortic injuries.

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