Secondary Pulmonary Arterial Hypertension Caused By Organized Hematoma Compressing The Pulmonary Trunk

S Pedamallu, S Habib

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Abstract

INTRODUCTION

Secondary pulmonary arterial hypertension is a devastating long-term complication following aortic root replacement surgery. Extrinsic compression of pulmonary vasculature as a cause of pulmonary arterial hypertension is well established 1,2,3,4,5. Organised haematoma due to leakage of aortic root graft compressing the pulmonary trunk has not been previously reported. A high index of suspicion is needed to make the diagnosis.

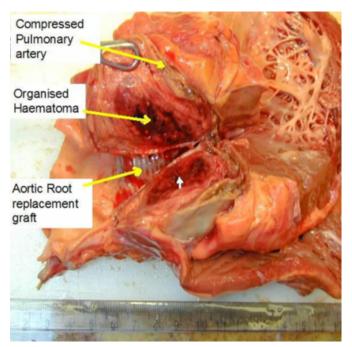
CASE REPORT

45-year-old lady known to have congenital heart disease underwent aortic root grafting and metallic valve replacement 10 years back. She subsequently developed complete heart block and required an implanted permanent pacemaker. She was on long term anticoagulation. She had laparoscopic cholecystectomy for chronic cholecystitis during the current admission. Anticoagulation was discontinued a week before the surgery. Preoperative INR was 1.5. In the immediate postoperative period, she was hypothermic with low blood pressure, very low urine output and a central venous pressure of 25mm. Bedside Echocardiogram revealed hypertrophied left ventricle, right ventricle is dilated with impaired systolic contraction and pulmonary arterial pressure of 60 mm Hg suggesting moderate to severe pulmonary arterial hypertension. She died in the immediate postoperative period with severe pulmonary hypertension.

The postmortem gross pathology heart specimen revealed moderately dilated heart with distorted appearance at the aortic root area. There was a large organized hematoma surrounding the aortic root and compressing the pulmonary trunk as the cause of pulmonary arterial hypertension (Figure). The left atrium was compressed and distorted but the left ventricle was found to be normal. The remainder of the aorta was normal.

Figure 1

Figure 1: Post-mortem specimen of the heart showing large organized hematoma surrounding the aortic root and compressing the pulmonary trunk.



DISCUSSION

Anatomically, the ascending aorta and pulmonary trunk share a common adventitia. Therefore, extravasation of blood from the ascending aorta extends into the adventitial space around the pulmonary trunk, and finally around the left and right pulmonary arteries. Due to low pressure in the pulmonary trunk and the arteries, they are easily compressible.

Non-dissecting chronic thoracic aortic aneurysm is one of

the common causes of pulmonary artery compression ₁. In particular, chronic inflammation secondary to syphilis damages the aortic wall and forms a huge aneurysm that compresses or penetrates the pulmonary artery or other adjacent structures.

On the other hand, dissecting thoracic aortic aneurysm usually ruptures into the pericardial space, pleural cavity or mediastinum because the outer wall of the false channel is much thinner. There are a few published case reports confirming pulmonary artery obstruction as a result of hematoma secondary to acute dissection of the thoracic aorta 2, 3, 4. There are no published case reports of secondary pulmonary arterial hypertension presenting as a long-term complication of aortic root replacement surgery

There is a published case report regarding successful bilateral percutaneous stenting of the pulmonary arteries in a patient with non small cell lung cancer and severe right ventricular hypertension due to mediastinal lymphadenopathy compressing both pulmonary arteries. Although the natural course of the disease was not altered, there was significant symptomatic relief without adverse effects 5.

A large organized haematoma compressing the pulmonary trunk, with an evidence of leakage of the aortic root replacement graft is a unique complication distinct from the above mentioned causes.

CONCLUSION

This case report will provide insight into long-term problems of anticoagulation after aortic valve and root replacement surgery. It also emphasizes the importance of suspecting graft leakage and possibility of haematoma causing compression of pulmonary vasculature, in all cases of new onset pulmonary hypertension.

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CORRESPONDENCE TO

Dr. Srinivas B Pedamallu 8, Stewart Street, Nuneaton CV11 5SA United Kingdom Email: pbs6vas@yahoo.com Mobile: 00447886141004

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Author Information

Srinivas B. Pedamallu, M.B.B.S, M.S

Department of General Surgery, George Eliot Hospital

Sherif Habib, FRCS

Department of General Surgery, George Eliot Hospital