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Case Report

A rare presentation of intrapericardial hematoma 20 months post aortic valve replacement

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ABSTRACT

We report the successful treatment of a rare case of chronic intrapericardial hematoma which presented with congestive cardiac failure 20 months after aortic valve replacement surgery for severe calcific aortic stenosis. Chest roentgenograph demonstrated paracardiac mass over lower left ventricle border, left pleural effusion and pulmonary venous hypertension. An echocardiographic study demonstrated intrapericardial mass posterolateral to left ventricle compressing left ventricular cavity both during systole and diastole causing septum to bulge into right ventricle. Computed tomography showed a loculated pericardial mass in left heart margin, left pleural effusion, bilateral axillary and mediastinal lymphadenopathy. Surgical resection was planned to relieve the symptoms and to confirm the diagnosis of the mass. The mass was completely resected through left anterolateral thoracotomy and histopathology findings confirmed the diagnosis of hematoma with cystic degeneration. Postoperative course was uneventful, and his symptoms improved markedly.

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1. Introduction

Chronic expanding hematomas a rare benign complication of chest surgery, chest trauma, or tuberculosis. Chronic expanding hematoma can occur at any location in the body, and the symptoms are related to the anatomical location. We describe a case of chronic expanding intrapericardial hematoma 20 months after aortic valve replacement surgery presenting as congestive heart failure.

2. Case report

A 61 year-old gentleman with history of aortic valve replacement 20 months back, was admitted to our hospital with recurrent episodes of syncope, dyspnea on exertion, decreased urine output and bilateral pedal edema of 6 months duration. A chest roentgenography showed paracardiac mass over left ventricle border, left pleural effusion and evidence of pulmonary venous hypertension. Echocardiography demonstrated intrapericardial mass [Fig. 1a] measuring 6.54 cm × 3.12 cm posterolateral to left ventricle compressing left ventricular cavity both during systole and diastole causing septum to bulge into right ventricle. Computed tomography showed loculated pericardial mass [Fig. 2a–c] in left heart margin, left pleural effusion, and bilateral axillary and mediastinal lymphadenopathy. Patient was stabilized with antifailure medications and planned for surgical removal of the mass. Under general anesthesia, through left anterolateral thoracotomy, the pericardium was opened. Partially organized hematoma with focal areas of necrosis was seen located posterior to the left ventricle, severely adhered to the myocardium which was carefully dissected out. Partial pericardiectomy and complete removal of the hematoma was successfully performed. Thorough saline wash given and source of bleeding could not be found. Intraoperative echocardiography study showed significant expansion of left ventricle after complete evacuation of hematoma. Post procedure, central venous pressure became normal. Histopathological examination [Fig. 3a–b] of the mass showed predominantly hematoma and fibrinous material with entrapped RBCs and WBCs. Necrotic areas showed the presence of inflammatory cells consisting of polymorphs, lymphocytes and reactive mesothelial cells. Areas of cystic degeneration with thick fibrocollagenous wall showing dilated congested vessels, hemorrhage and inflammatory cells. Histopathological examination of mediastinal lymph node showed reactive lymphadenitis. Microbiological examination of specimen revealed no infective organism. Postoperative course was uneventful and his symptoms improved markedly. Postoperative echocardiography [Fig. 1b] demonstrated significant expansion of left ventricle with minimal residual pericardial collection.

3. Discussion

Chronic expanding hematoma was first described by Reid et al[1] According to Reid a hematoma that persists and increases in size more than one month after the incident haemorrhagic event is a chronic expanding hematoma. This disease can occur at any location of the body. Only few cases have been reported after open heart surgery, chest trauma or epicardial injury.[2–5] In a literature review of Brown & Ivey, the time from injury to presentation was a wide range from 3 to 20 years.[6] This long latent period highlights the persistent insidious nature of the disease. This chronic expanding intrapericardial hematoma can result in diastolic dysfunction, heart failure, constrictive pericarditis or restrictive physiology. In this case, the patient underwent aortic valve replacement 20 months back. Symptoms were related to the anatomical location of the hematoma. Chronic expanding hematomas occur in many locations, often simulating neoplasms. All have the same structure with a central mass of blood, a wall of granulation tissue and dense fibrous tissue at the periphery.

Various theories have been put forth to explain the cause of sudden expansion. One such theory is that a high osmotic pressure gradient is produced by the breakdown products of the hematoma, resulting in a localized inflammatory reaction.[7–9] Factors in the coagulation cascade along with release of vasoactive substances are believed to be associated with an inflammatory reaction which may cause additional bleeding

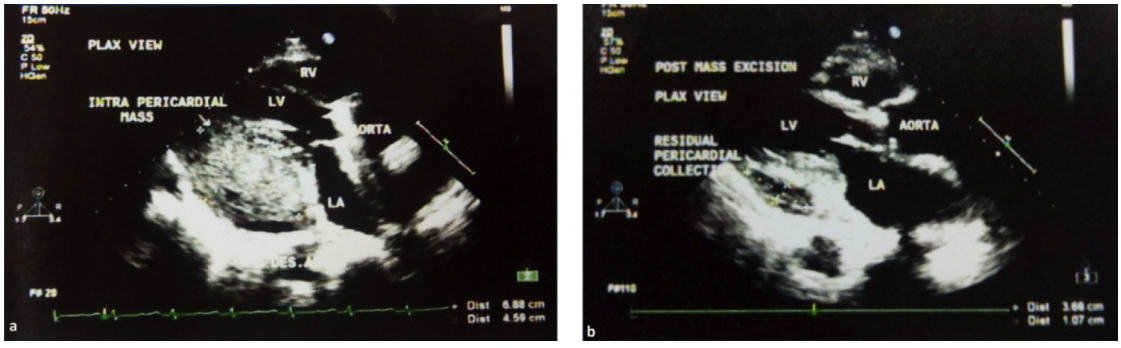

Fig. 1 – a: 2-dimensional echocardiography-parasternal long axis view showing intrapericardial mass posterior to left ventricle compressing left ventricular cavity causing septum to bulge into right ventricle. b: 2-dimensional echocardiography – parasternal long axis view after resection of the intrapericardial mass showing normal left ventricle and right ventricle chambers with minimal pericardial collection.

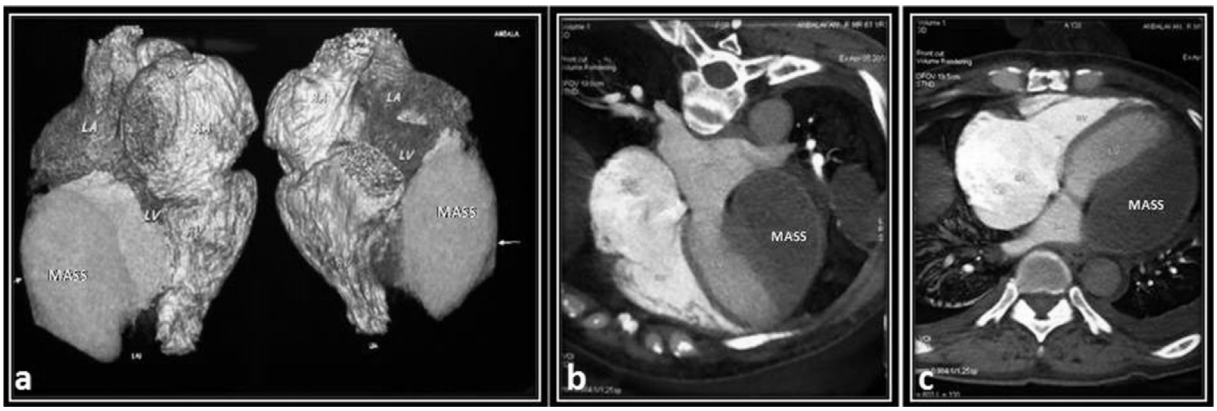

Fig. 2 – a–c: Computed tomography showing loculated pericardial mass in left cardiac border compressing left ventricle in multiple views.

from fragile capillaries, and bleeding leads to further inflammation in a cycle that allows the development of a chronic expanding hematoma in virtually any anatomical location.[8–10]

In a patient 20 months post aortic valve replacement presenting with signs and symptoms of heart failure and X-ray chest showing a paracardiac mass over left ventricular border, the differential diagnosis that one can think of are loculated pericardial effusion, left ventricular aneurysm, left ventricular clot, benign and malignant tumors of the heart. Echocardiography would further narrow down by telling us whether the mass arises within the heart, in the pericardium or extra cardiac. The investigations that would help to differentiate intrapericardial hematoma from other differential diagnosis are tumor markers, echocardiography, computed tomography, Magnetic Resonance Imaging (MRI), FDG PET Scan (Fluoro De-oxy Glucose Positron Emission Tomography).

Tumor markers like CarcinoEmbryonic Antigen (CEA), Carbohydrate Antigen 19-9 (CA19-9), Neuron Specific Enolase (NSE), Squamous Cell Carcinoma related antigen (SCC) and interleukin 2-receptor would help to identify the corresponding tumors.

Echocardiography can differentiate intracardiac hematoma from all other conditions except intracardiac thrombus. Echocardiography features that suggest intrapericardial hematomas are superolateral location, smooth rounded margins that project into the adjacent chamber and an echo lucent center surrounded by a highly refractile margin that compresses the edge of the hematoma and the contiguous pericardium or LV wall. Echocardiographically a hematoma may appear anywhere from sonolucent to highly refractile mass.

Computed tomography scan can detect, localize and characterize hematoma. Such characterization is best accomplished if both precontrast and post contrast images are obtained. The extension of the extrinsic hematoma beyond the boundaries of the involved chamber differentiates an intracardiac thrombus from an intrapericardial hematoma. Several Computed tomography characteristics are useful in differentiating intrapericardial hematoma from other soft tissue masses namely, the hematoma remain more dense than soft tissues, the pericardium is visualized along the lateral border of the mass and vascular extra cardiac masses such as tumors usually show an increase in radiographic density after the administration of contrast medium when compared to avascular masses such as hematomas.

On MRI a chronic expanding hematoma with peripheral capsule and central contents will have signal intensities ranging from high to low also called mosaic pattern. The

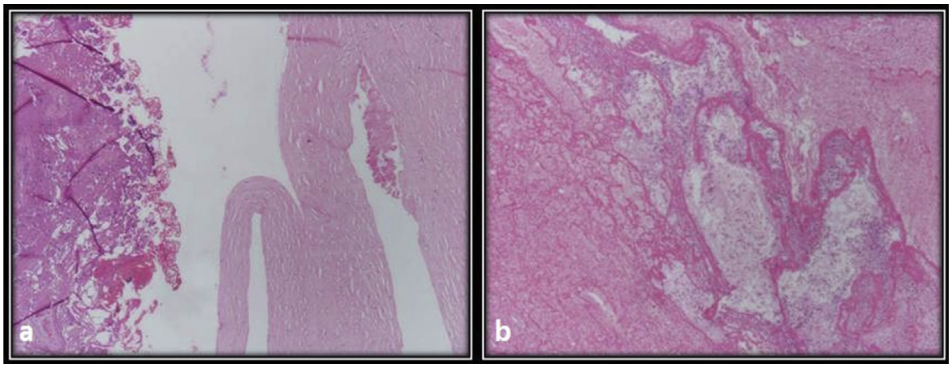

Fig. 3 – a–b: Histopathology slide showing predominantly hematoma and fibrinous material with entrapped RBC and WBC. Areas of cystic degeneration with thick fibrocollagenous wall showing dilated congested vessels, hemorrhage and inflammatory cells.

mixture of high and low intensity areas on T2 weight images correspond with the fresh and old haemorrhages caused by repeated bleeding overtime.

There is not much experience or information about FDG PET imaging for intrapericardial hematoma. FDG PET is an evolving diagnostic modality for tumor detection, staging and therapeutic monitoring of various malignant tumors. A chronic intrapericardial hematoma is not supposed to take up FDG like tumors, but the hematoma sometimes take up FDG at the periphery because of inflammatory cell uptake of FDG. This should not be mistaken for malignancy.

In the present case, the mechanism was supposed to be associated with inflammation, which is confirmed by histopathological findings. The management of such hematomas should be complete surgical resection at an early stage before compression of mediastinal structures. Since the patient described here presented with congestive heart failure resulting from compressive effect of the mass, surgery is warranted despite his advanced age and the risk of re-do surgery. In conclusion, chronic expanding hematoma remains a rare disease, but should be considered when an expanding mass is found in the chest after cardiac surgery. An earlier surgical resection may be recommended to make a definitive diagnosis and relieve the symptoms associated with compression by the mass.

Conflicts of interest

All authors have none to declare.

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