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Encased Heart – A Very Rare Post Myocardial Infarction Complication Sequela

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Abstract
Post cardiac injury syndrome complicating acute myocardial infarction is well known but its progression to constrictive pericarditis is extremely rare. We report a case of 60 year old male who underwent a primary percutneous intervention for acute myocardial infarction. Subsequently he developed chronic constrictive pericarditis which improved dramatically after pericardiectomy.
Keywords: *Chronic constrictive pericarditis, Myocardial rupture, pericardiectomy.*

Introduction

Post cardiac injury syndrome (PCIS) is a condition characterized by injury to the pericardium leading to its inflammation along with accumulation of fluid in the pericardial space.[1] It includes post myocardial infarction related pericarditis (Dressler syndrome), post traumatic pericarditis [post percutaneous cardiac intervention (PCI), post cardiac implantable electric device placement] and post pericardiotomy syndrome.[2] In the pre reperfusion era, the incidence of PCIS after an acute myocardial infarction was found to be 3%.[3] The incidence of PCIS after a PCI procedure is 0.5%.[1] Data on progression of acute pericarditis to chronic constrictive pericarditis (CCP) is scant. Imazio et al found such progression in 1.8% of overall population of acute pericarditis while progression to CCP was seen in 2.8% of acute pericarditis due to pericardial injury syndrome.[4] This case report describes a 60 year old male who started developing breathlessness within 6 months after recovering from PCIS.

Case

History of Presentation: A 60-year-old diabetic, non-hypertensive male, presented to our hospital with acute inferior wall myocardial infarction and underwent primary percutaneous intervention with stenting to right coronary artery (RCA) with a drug eluting stent (Promus Elite, Boston Scientific, USA). 2 months later, he developed stent thrombosis which was managed percutaneously with re-stenting to RCA with switch over to newer antiplatelets (Ticagrelor). During this admission, he was detected to have moderate pericardial effusion without tamponade which was managed conservatively. It resolved

completely within two weeks. On follow up at 6 months, he started complaining of insidious onset breathlessness on climbing 2 flights of stairs. There was no chest pain, orthopnoea or history of allergy. Blood N terminal pro bnp was normal. Repeat echocardiogram revealed ejection fraction of 50% with mild hypokinesia in inferior wall and complete resolution of pericardial effusion. This breathlessness was attributed to ticagrelor which was changed back to clopidogrel. At 1 year follow up, breathlessness was gradually increasing and he started developing abdominal distention. He was readmitted for a detailed evaluation. On examination, jugular venous pressure was raised 3cm above the clavicle on sitting position with prominent X and Y descent. Heart sounds were normal and chest was clear. He had bilateral pitting pedal edema extending up to mid shin. There was gross ascites which was out of proportion to the pedal edema. There were no new changes in the electrocardiogram (ECG) and cardiac biomarkers were normal. Transthoracic echocardiography revealed thickening of in inferior wall extending to apex and anterior wall. Inferior vena cava was dilated, interventricular septal bounce present, annulus paradoxus, respiratory variation of mitral and tricuspid inflow velocity of 40% and 50% respectively and hepatic vein expiratory diastolic reversal ratio >0.8 suggestive of constrictive pericarditis. Patient was subjected to Computed tomography (Figure-1) which revealed maximum pericardial thickness of 4.9mm along the right ventricle, 3.7mm along the left ventricle and 2.1 mm at apex. After a futile trial of high dose aspirin (1.8gm/day) followed by colchine for 3 months, patient was advised pericardiectomy. Due to the Covid-19 pandemic, he could not undergo surgery. There was further worsening of breathlessness from NYHA II to NYHA III/IV over the next 2 years. Ascites became so tense that he started to develop umbilical hernia. With the recession of the pandemic, he underwent pericardiectomy.

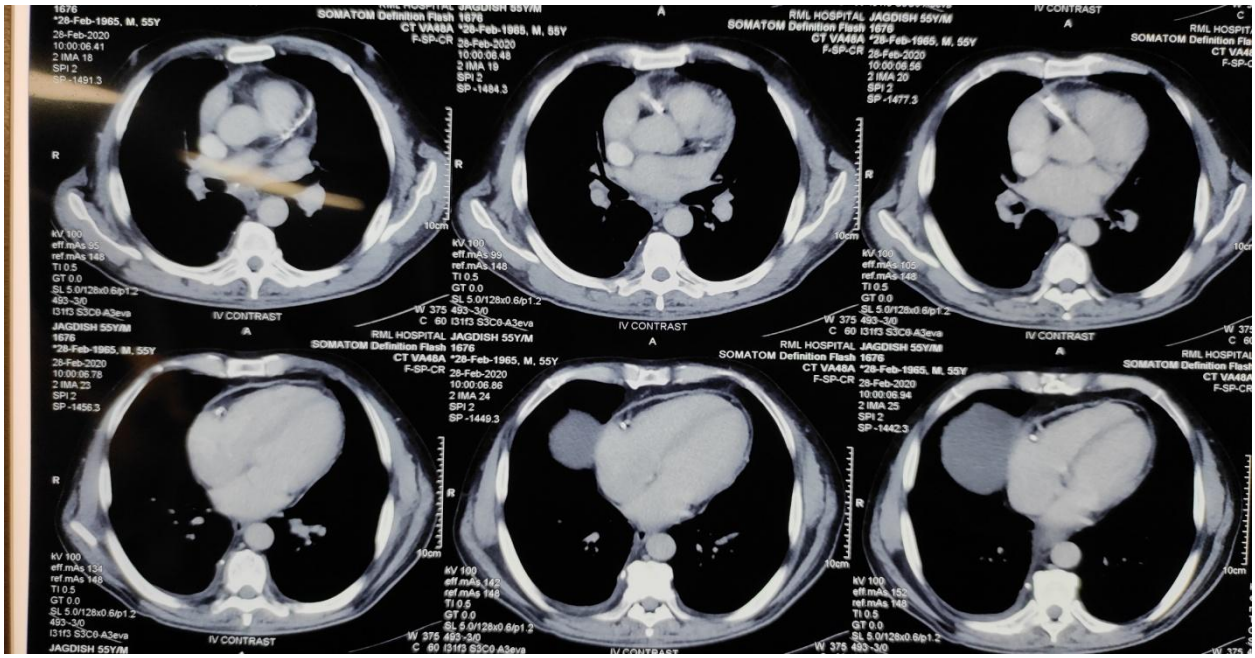

Figure 1: Cardiac CT showing pericardial calcification

Investigations

Echocardiography findings were similar and consistent with the diagnosis of CCP. A cardiac MRI revealed septal bounce and thickened pericardium with features of old inferior wall myocardial infarction. Pericardial thickening was more prominent in the inferior wall and apex with extension into the anterior wall. Preoperative, coronary angiogram revealed proximal RCA (stented segment)-100% stenotic (retrogradely

filling through left coronary system) and left anterior descending (LAD) artery having 50% stenosis. RCA was totally fixed and there was no movement at all, it seemed that RCA was totally encased by the thickened pericardium.

Management

Patient was planned for pericardiectomy along with grafts to distal RCA and LAD. During surgery, pericardium was found to be thickened and adherent to the underlying myocardium which was stripped off. Occluded RCA stent was seen in atrio-ventricular groove densely adherent to overlying thickened pericardium (Figure-2). Due to difficulty in mobilizing the arteries in view of dense adhesion, only pericardiectomy had been performed. Pre operative central venous pressure was 30 millimeters of mercury with came down to 16 millimeters immediately post operatively.

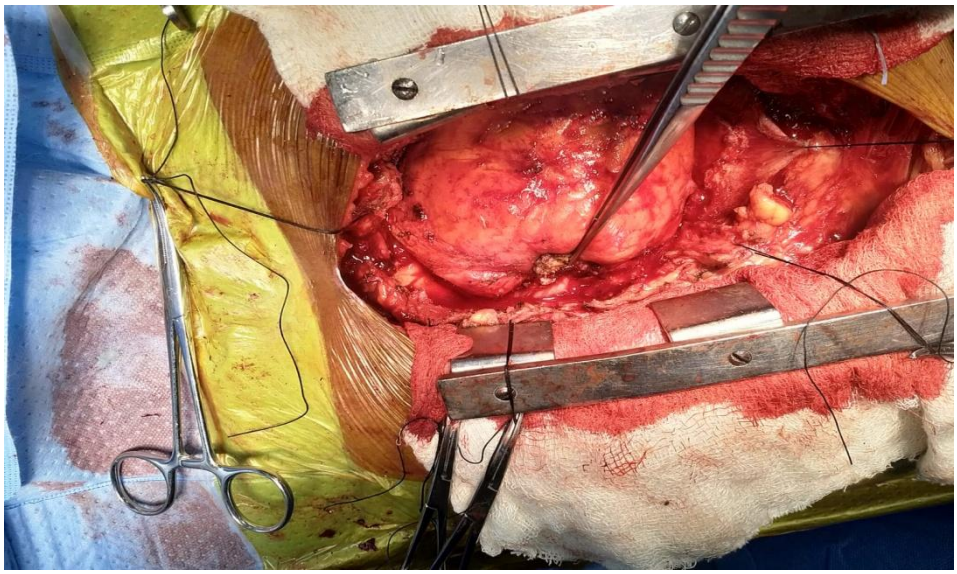

Figure 2: Thickened pericardium with stent seen coming out of RCA adherent to the pericardium

Discussion

Constrictive pericarditis is a condition in which granulation tissue formation in the pericardium results in loss of pericardial elasticity leading to restriction in the ventricular filling.[5]The risk of progression to constriction is related to the etiology: low (1%) in viral and idiopathic pericarditis, intermediate (2–5%) in immune-mediated pericarditis, neoplastic pericardial diseases and high (20–30%) in bacterial pericarditis, especially purulent pericarditis.[2]The delay between the initial pericardial inflammation and the onset of constriction is variable and is possibly a direct evolution from subacute or chronic pericarditis to constrictive pericarditis.[2] Despite advances in diagnostic modalities, the mean time between symptom onset and diagnosis of pericardial constriction is 24 months.[6]The most common cause of CCP in the developing world is tuberculosis while it is post cardiac surgery in the developed world[7]

Our patient had stent thrombosis at 2 months after a primary PCI, detected to have moderate pericardial effusion at that time which resolved spontaneously within 14 days but he started developing symptoms after 6 months which progressed to full blown clinical features consistent with CCP at 1 year. Early pericarditis following an acute myocardial infarction is a common entity within the first 4 days. Primary PCI has shown to reduce this early pericarditis if presentation is early (within 3 hours of symptom onset), however late pericarditis after a primary PCI is a rare entity reported to be around 0.5%.[8] Mohamed et al reported a case of CCP 4 months after an uncomplicated PCI, attributing it to micro perforation with a small unrecognized blood leakage into the pericardial sac.[9] Our case looks

like a case of PCIS leading to moderate pericardial effusion which finally led to CCP.

The current hypothesis of PCIS stands with an autoimmune pathogenesis with the first event being damage to the pericardial or pleural mesothelial cells by either necrosis of myocardium, surgical trauma, blunt thoracic trauma, or an iatrogenic injury to the pericardium.[1] Iatrogenic trauma caused during PCI is minimal albeit can elicit a hyperinflammatory state culminating to PCIS in susceptible individuals.

Follow-up: At 2 weeks post pericardiectomy, there was drastic reduction of patient's breathlessness and abdominal distension. At 4 weeks patient was totally asymptomatic(Figure 3) with absence of echocardiographic features of CCP. (Figure 4)

Figure 3: A- Before pericardiectomy ascites. B – 4 weeks post pericardiectomy resolution of ascites

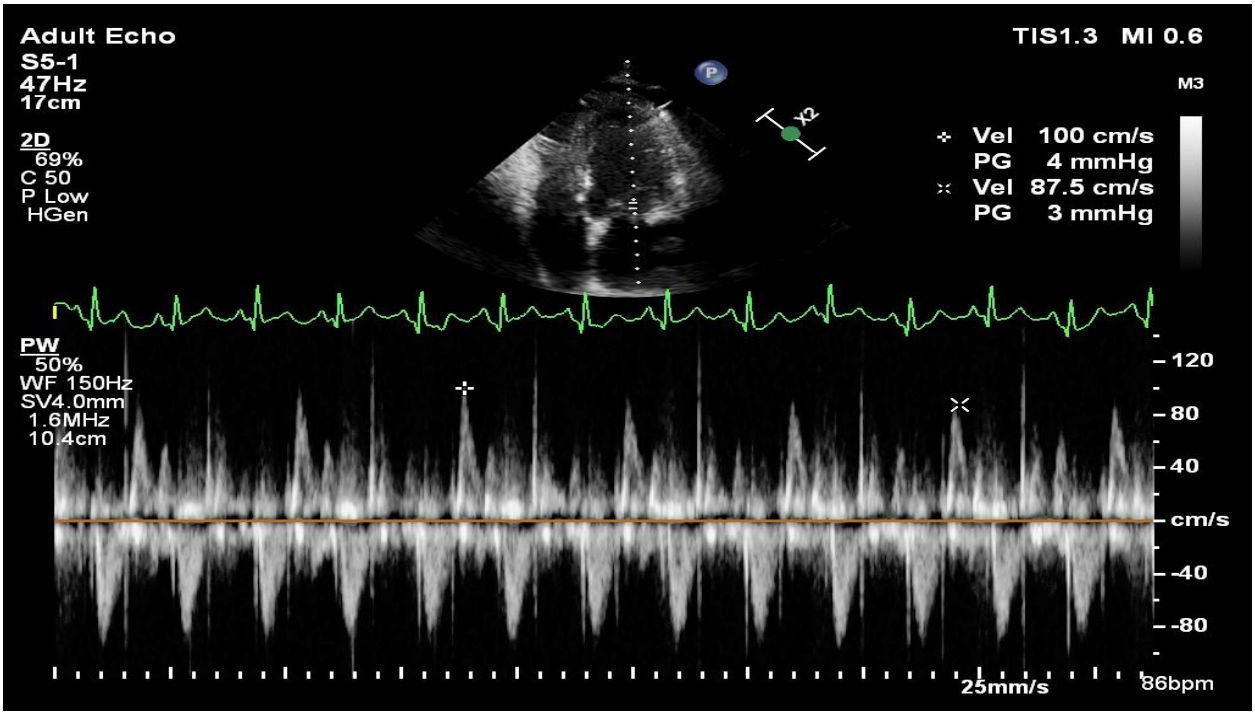

Figure 4: Echocardiogram post pericardiectomy showing no significant respiratory variation in mitral inflow velocity

Conclusion

Constrictive pericarditis should be suspected in individuals developing ascites disproportion to the pedal edema and raised jugular venous pressure with prominent X/Y descent after acute MI. Possible mechanisms are micro haemorrhage into

pericardium, sealed perforation or development of PCIS complicating into constriction. Pericardiectomy in these patients have a good result and should be done promptly.

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Abbreviations

PCIS (Post Cardiac Injury Syndrome)
PCI (Percutneous Cardiac Intervention)
CCP (Chronic Constrictive Pericarditis)
RCA (Right Coronary Artery)
ECG (Electrocardiogram)