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Constrictive pericarditis following Dressler's syndrome induced by percutaneous coronary arteriography

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ABSTRACT

Constrictive pericarditis (CP) is a chronic inflammatory process characterized by chronic scarring, fibrosis and calcification of the pericardium associated with diastolic dysfunction, eventually leading to low cardiac output and heart failure. Idiopathic pericarditis remains the predominant cause of CP in the western world, followed by surgery and radiation therapy. Tuberculous pericarditis is still the leading cause of CP in developing countries and immunocompromised patients. Dressler's syndrome is an autoimmune pericarditis which has been rarely reported following myocardial infarction and cardiotomy cases and lately after percutaneous coronary intervention (PCI). However, CP following PCI is exceedingly rare. Herein, we report a case of CP following an uncomplicated coronary angioplasty and stenting.

Keywords: constrictive pericarditis, Dressler's syndrome, percutaneous coronary intervention, pericardiectomy.

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INTRODUCTION

Constrictive pericarditis (CP) is a chronic inflammatory process characterized by chronic scarring, fibrosis and calcification of the pericardium associated with diastolic dysfunction, eventually leading to low cardiac output and heart failure. Idiopathic pericarditis remains the predominant cause of CP in the western world, followed by surgery and radiation therapy. Tuberculous pericarditis is still the leading cause of CP in developing countries and immunocompromised patients. Other causes include; uremia, systemic lupus erythematosis, tumors, incomplete drainage of pulrulent pericarditis, fungal and parasitic infections and post-viral and cardiac injury [1]. The latter has been referred to as Dressler's syndrome (DS). It includes two distinct entities, the post-myocardial infarction syndrome which occurs after myocardial infarction, and the post-cardiotomy syndrome which occurs after cardiac surgery or trauma [2]. Few cases of DS have been described after percutaneous coronary intervention (PCI) yet CP is exceedingly rare. Herein, we report a case of CP following an uncomplicated coronary angioplasty and stenting

THE CASE

A 71 year-old man had angina on mild effort. Routine tests at that time showed normal peripheral leucocytic and platelets counts, hemoglobin, renal and liver profile as well as urine routine and microscopy. ECG showed T-wave inversion in all precordial and inferior leads with mild rise in serum troponin. Coronary arteriography was done followed by angioplasty and stinting of 4 vessels at that time. One month later, he developed progressive shortness of breath with massive lower limbs oedema. He was afebrile with blood pressure at 90/40 mm Hg. He did not have lymphadenopathy yet had evident jugular venous distension. Systemic examination did not show abnormality. Laboratory investigations were normal. Chest x-ray showed right-sided pleural effusion with enlarged heart. Echocardiogram showed massive pericardial effusion with thickened pericardium. Tape of the pleural effusion showed high protein content with 200 white cell/HPF, of which 75% were lymphocytes. Pericardial fluid culture was sterile and Genexpert test for mycobacterium tuberculosis was negative. He was given Colchicine for his pericarditis and Lasix was started on Lasix 80mg/day for his massive lower limbs oedema.

Corticosteroids were not given by the cardiologists for fear of worsening his cardiac remodeling. With time, his shortness of breath, lower limbs oedema and hypotension did not improve. Moreover, his serum urea and creatinine had increased to 20 mmol/L and 213 umol/L, respectively. On his initial evaluation by the renal unit, his chest x-ray showed right sided pleural effusion with enlarged heart. ECG was normal except for low voltage. Abdominal and pelvic ultrasound was normal except for ascitis. Serum complements (C3 & C4), IgA level and protein electrophoresis were normal. ANA, anti-ds DNA, ANCA, anti-GBM-antibodies, RA, hepatitis B surface antigen and anti-HCV antibodies were negative. Transthoracic echo-cardiogram showed thickened pericardium (13 mm) while normal is < 3 mm (fig.1). Moreover, it showed significant respiratory transmitral Doppler variation with 63% decrease in mitral inflow during inspiration (fig.2) and an increased tissue Doppler imaging (TDI) E septal velocity which was more than TID E lateral wall velocity (fig.3). The latter features were suggestive of significant constrictive pericarditis. He was treated with Prednisone 60 mg daily. Four weeks later, his condition did not improve; hence he was subjected to pericardiectomy. Unfortunately, he died during surgery.

DISCUSSION

DS is an autoimmune pericarditis induced by reaction to myocardial neo-antigen from an injured heart or pericardium. It was first characterized by William Dressler at Maimonides Medical Center in 1956 [3]. DS consists of a persistent low-grade fever, retrosternal chest pain and pericardial rub with or without effusion. The symptoms tend to occur 2–3 weeks after myocardial infarction, but can also be delayed for a few months. It tends to subside in a few days, and very rarely leads to pericardial tamponade [4]. DS was, historically, a phenomenon complicating about 3.5% of myocardial infarctions [5]. However, in the era of primary PCI, it is very uncommon [2]. Bonello et al, reported a case of CP diagnosed six months after PCI [6]. However, their patient's PCI was complicated by a type 3 coronary rupture associated with well-tolerated hemopericardium. The coronary rupture was successfully sealed with a polytetrafluoroethylene-covered stent [6]. Review of the medical history, showed Only 1 case report similar to ours. The article described a patient who had developed CP 3 months following an uncomplicated PCI [7]. The authors attributed such phenomenon to micro-perforation with small, unrecognized blood leakage, which may have included contrast material, into the pericardial sac. Contrary to our patient, theirs did not have delayed development of a pericardial effusion and early tamponade. The latter confirms the localized autoimmune process. Our patient had progressive DS following PCI yet corticosteroids were used at a later stage of his disease. Optimizing, at the stage of pericardial effusion and subacute CP, with corticosteroids may have decreased the need for pericardiectomy which remains a high-risk surgery even in the best centers [8]. In-conclusion; PCI can induce DS and even fatal CP.

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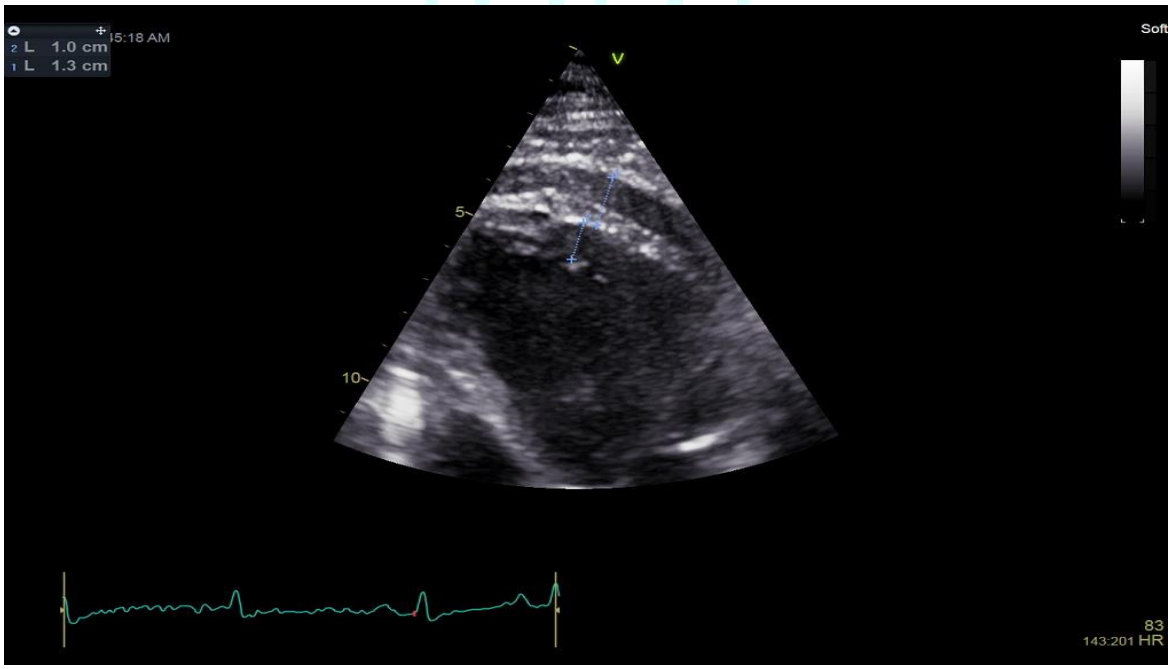

Figure 1: Transthoracic echocardiogram showing the thickened pericardium (13 mm) while normal is < 3 mm.

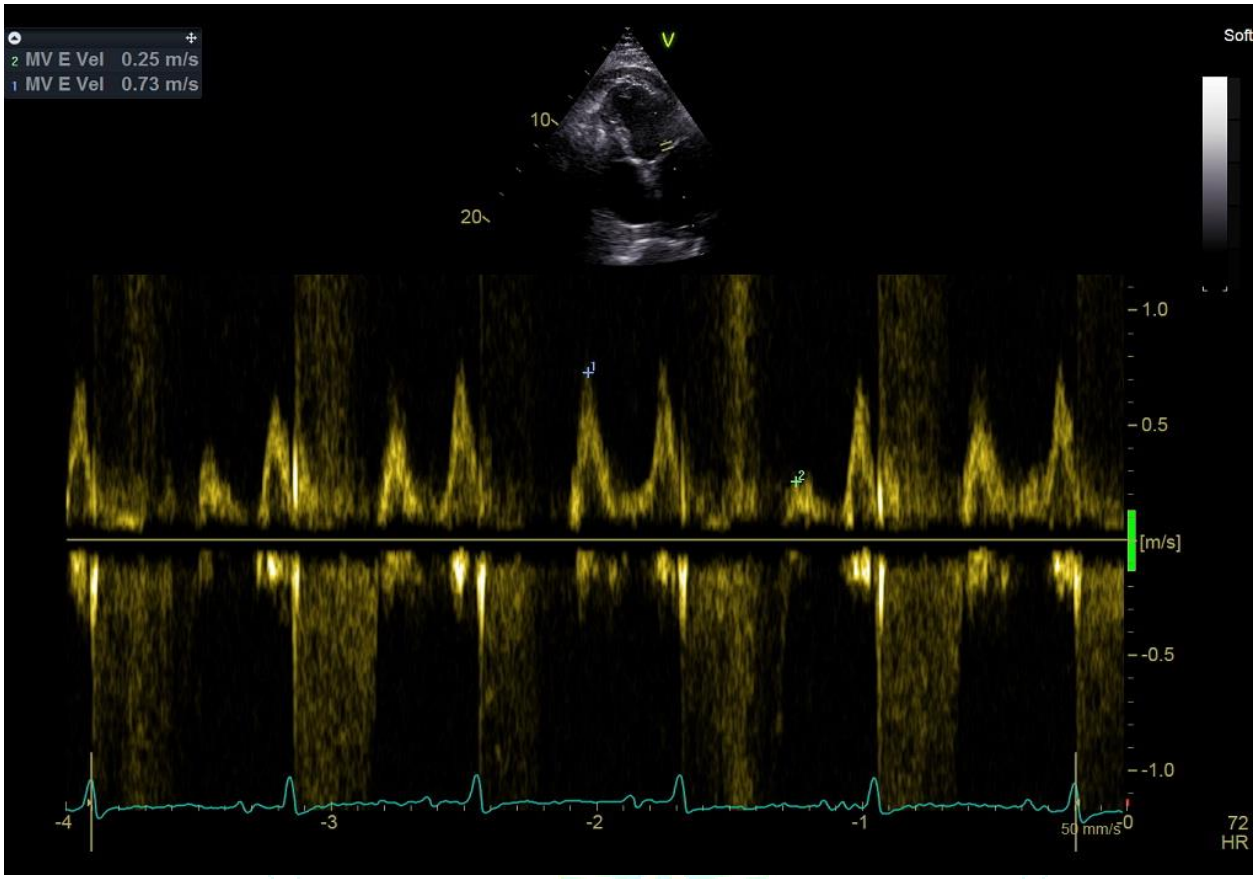

Figure 2: Transthoracic echocardiogram showing the significant respiratory transmitral Doppler variation (63% decrease in mitral inflow during inspiration).

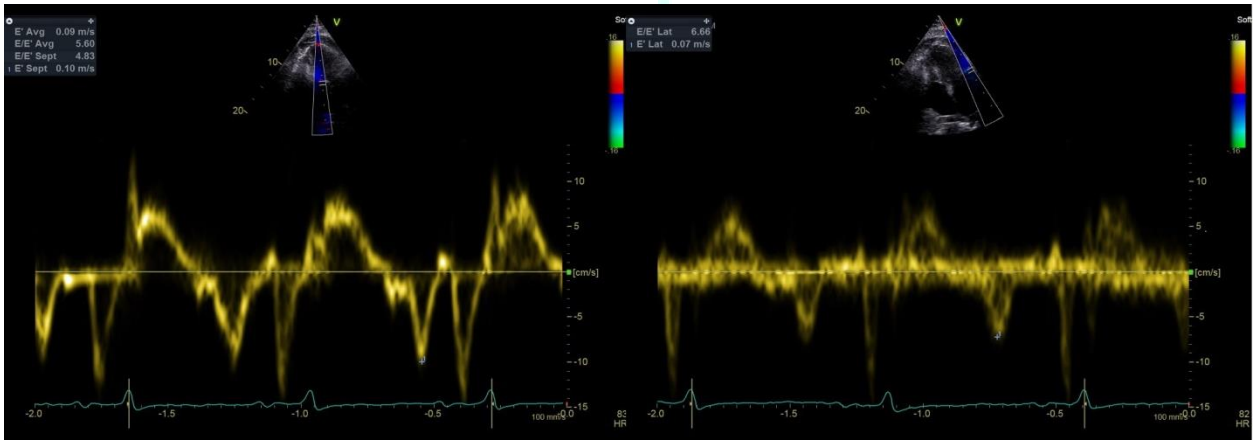

Figure 3: Transthoracic echocardiogram showing the increased tissue doppler imaging (TDI) E septal velocity more than TID E lateral wall velocity.