ISSN-0972-1622
Vol. 25 (1-2) : 44-47

Case Report

Multiple Right Ventricular free wall aneurysms in chronic constrictive pericarditis : A case report

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Summary

Right ventricular aneurysms are rarely seen in clinical practice and their association with constrictive pericarditis is even rarer. We present a case of constrictive pericarditis (CCP) resulting in multifocal outpouching of the right ventricular free wall, resembling right ventricular free wall aneurysms.

A 16-year-old boy with 1 year history of progressive right heart failure and congestive hear failure was diagonosed with CCP. RV angio revealed multiple RV outpouchings mimicking aneurysms. The patient underwent pericardectomy and is doing fine.

Conclusion : Diffuse CCP with a focal areas of sparing overlying the free wall of the right ventricle can result in free wall aneurysm-like outpouching of structurally and functionally normal ventricular wall. (Indian J Cardiol 2022;25 (1-2):44-47)

Key words: Constrictive Pericarditis , Aneurysm, Right heart failure

Introduction

Chronic Constrictive pericarditis (CCP) is caused by scarring and loss of elasticity of the pericardium, resulting in external impedance to cardiac filling. Previous cardiac surgery, thoracic irradiation and viral or idiopathic pericarditis are among the most common aetiologies[1]. Aneurysms of the right ventricle are extremely rare in CCP and only a few cases have so far been described in the literature[2,3].

Case report

A 16 year old boy with 1 year history of progressive right heart failure and passive liver congestion was referred for evaluation of possible CCP. There was no history of palpitations or syncope. Family history was negative for any sudden cardiac death or arrhythmogenic right ventricular dysplasia. His past medical history was significant as he was operated at the age of 2 years for empyema thoracis and pyopericardium. His blood pressure was 120/70 mmHg with a pulse of 80 beats per minute. His physical examination was notable for marked jugular venous distension to the level of the angle of the jaw, abdominal fullness with moderate ascites and minimal peripheral oedema. Electrocardiogram showed normal sinus rhythm, and no epsilon waves or ventricular ectopics. Chest x-ray PA and lateral view revealed pericardial calcification over free RV wall (Figure 1a,b). Right heart catheterization revealed a normal cardiac output, equalisation of end diastolic pressures in both ventricles and Square root sign. RA tracing showed prominent "y" descent, without any respiratory variation. Pulmonary artery systolic pressure was 36 mmHg. RV angio revealed multiple aneurysmal dilatations of RV free wall((Figure 2A,B).

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Indian J Cardiol 2022
Vol. 25 (1-2) : 44-47

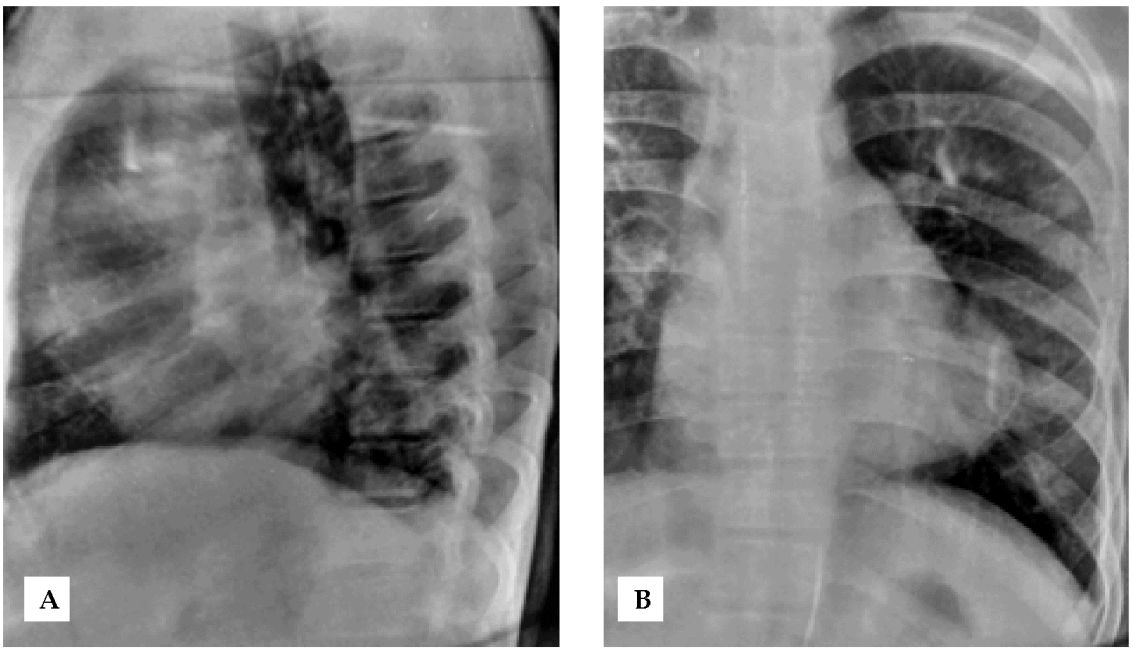

Fig. 1 A,B : X-RAY chest lateral and PA view depicting pericardial calcification(white bold arrows).

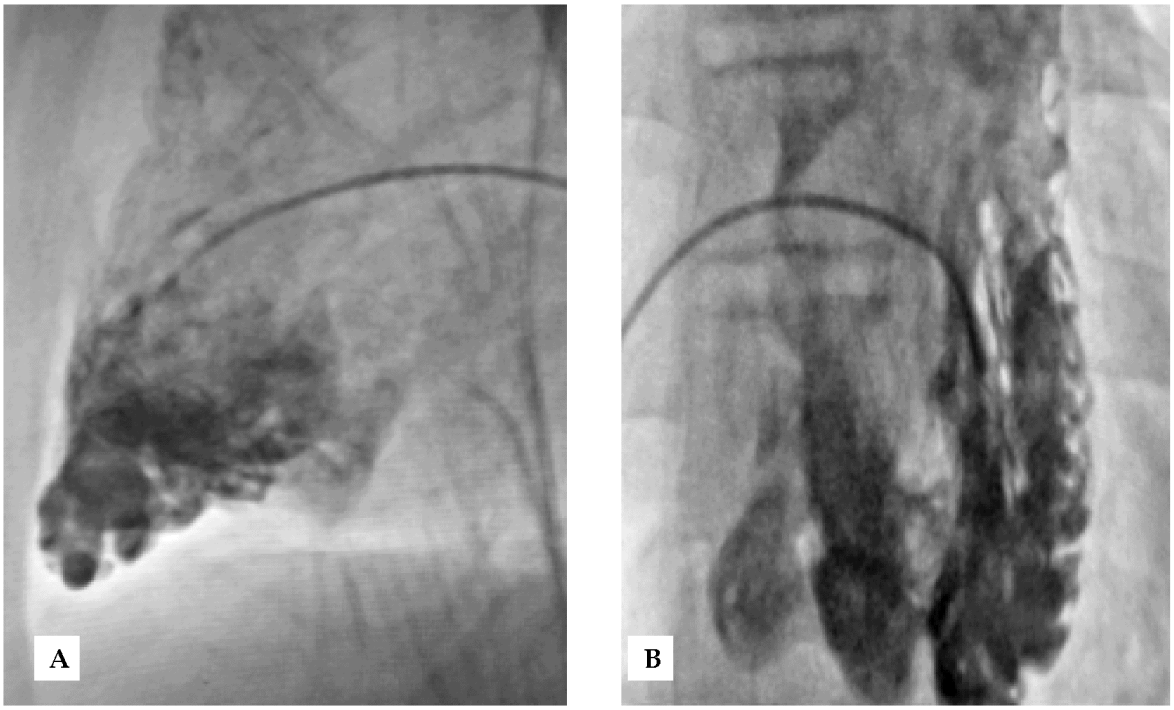

Fig. 2 A,B : RV angio(LAO cranial and RAO caudal view) demonstrating multiple aneurysm like outpouches.

The patient was referred for cardiac MRI. MRI was performed using a 1.5 T whole-body MR unit with a standardized cardiac MRI protocol. Double IR T2 weighted images showed diffuse pericardial thickening, maximum thickness of 8.0 mm. The pericardium anterior to right ventricular free wall outpouchings was measured as normal. In addition inferior vena cava and hepatic veins were grossly dilated and there was biatrial enlargement. Dyskinetic systolic motion of the right ventricle causing aneurysm-like outpouchings during systole in the right ventricle free wall, which was covered by normal pericardium (Figure 3A,B). Paradoxical septal motion was also noted, compatible with CCP. No thrombus within the right ventricle or aneurysms was observed. Left ventricular function, wall thickness and enhancement were normal.

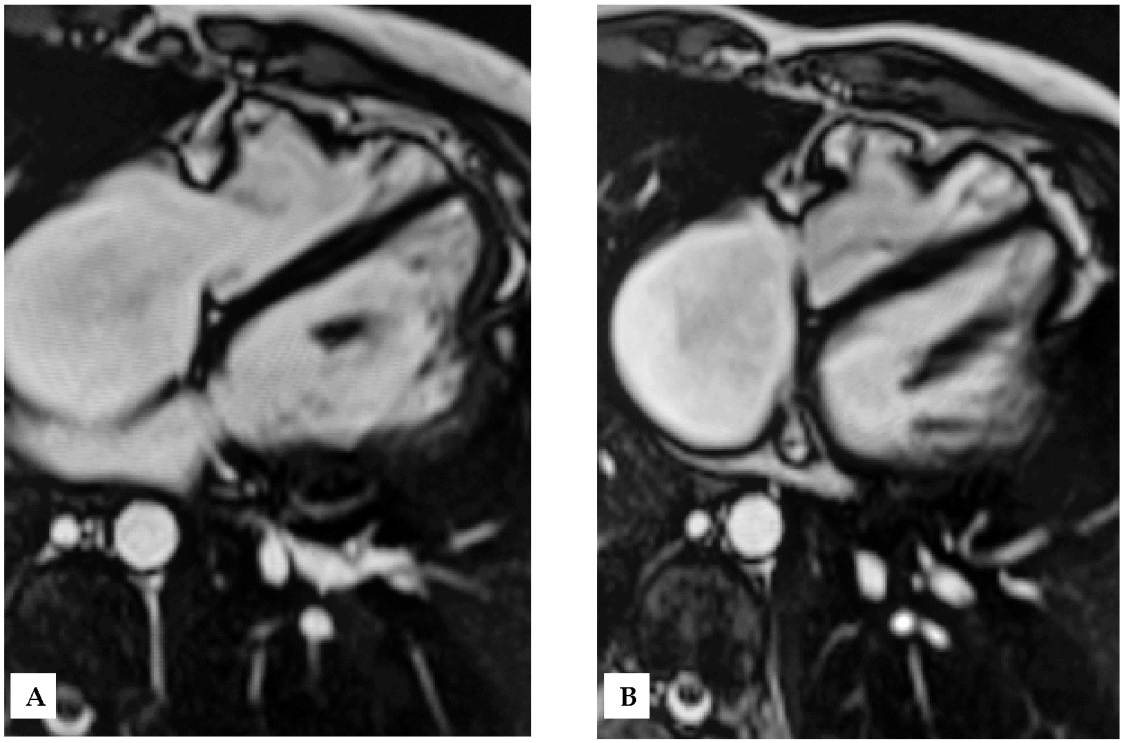

Fig. 3 A,B : Cardiac MRI showing thickened pericardium(solid arrow) and RV free wall aneurysms(stars) . Note can be made of relatively normal pericardium covering the outpouches(solid plus)

In order to evaluate pericardial calcification, noncontrast chest CT was performed using a 64 channel multidetector CT scanner. Non-contrast CT study showed similar findings to the MRI, including a thickened pericardium, measuring up to 6.8 mm. Dilated hepatic veins and inferior vena cava again demonstrated changes consistent with a right ventricular overload. Additionally, a moderate amount of abdominal ascites and passive hepatic congestion were seen. It was unclear why the right ventricle free wall had aneurysms. The patient was operated, pericardectomy was done and is doing fine on followup.

Discussion

The pericardium is a thin, avascular sac, composed of two layers: an inner serous membrane and an outer fibrous collagenous layer[4]. CT and MRI provide excellent visualisation of the pericardium. The thickness of the normal pericardium, measured on CT or MRI, is less than 2 mm[5]. A pericardial thickness of 4 mm or more indicates abnormal thickening and, when it is accompanied by clinical findings of heart failure, is highly suggestive of CP. MRI has a reported accuracy of 93% for differentiation between CP and restrictive cardiomyopathy on the basis of depiction of thickened pericardium (>4 mm)[5]. CT has a high

sensitivity in depicting pericardial calcification, which is also associated with CP. However, neither pericardial thickening nor calcification is diagnostic of CP unless the patient also has symptoms of physiological constriction. Clinical signs and symptoms of right heart failure, enhanced interventricular dependence, respiratory variation in the ventricular pressures and ventricular discordance are the pathophysiological hallmarks of CP. Arrhythmogenic right ventricular dysplasia, although rare, is the most common aetiology associated with focal right ventricular aneurysms[6]. Other reported, but less frequent, causes of isolated right ventricular aneurysm include acute myocardial infarction, acute myocarditis and iatrogenic injury and trauma[4]. Inoue et al[7] described MRI findings of a right ventricular aneurysm secondary to acute myocarditis. They noted diffuse transmural contrast enhancement throughout the right and left ventricle surrounding the aneurysm, suggestive of a transmural myocardial inflammation. They concluded that an aneurysm of the right ventricle can form after severe myocardial damage due to acute myocarditis[7]. Focal or localised CP is also a known entity. Hasuda et al[8] reported the first case of post-surgical focal CP confined to the left ventricle. After mitral valve replacement, the patient presented with heart failure. Cardiac catheterisation showed impaired diastolic filling but lacked the characteristic ventricular interdependence. MR tagging cine revealed pericardial adhesion, limited to the left ventricle, which was confirmed during pericardiectomy[8].

Our case is best considered as diffuse CP with multiple regions of sparing. The part of the right ventricle free wall that was covered with normal pericardium was not restrained and the adjacent thickened pericardium was stiff enough that this normal region protruded in between the edges of thickened pericardium, resembling an aneurysm.

Conclusion

Diffuse CP with a focal areas of sparing overlying the free wall of the right ventricle can result in a free wall aneurysm-like outpouching appearance of what is structurally and functionally normal wall.

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