CASE REPORT

Constrictive pericarditis presenting with an outpouching of the right ventricle free wall simulating an aneurysmal dilatation

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ABSTRACT. We present a case of constrictive pericarditis resulting in an outpouching of the right ventricular free wall, simulating a right ventricular free wall aneurysm. The present case is, to the best of our knowledge, the first reported right ventricular free wall aneurysm-like outpouching adjacent to surrounding regions of thickened pericardium in a patient with constrictive pericarditis.

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Constrictive pericarditis (CP) 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, and only a few cases have so far been described in the literature [2, 3].

Case report

A 48-year-old man with a 1 year history of progressive right heart failure and passive liver congestion was referred for evaluation of possible CP. His medical history was essentially unremarkable. There was no history of palpitations or syncope. Family history was negative for arrhythmogenic right ventricular dysplasia. His blood pressure was 128/84 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 significant ascites and minimal peripheral oedema. Electrocardiogram showed normal sinus rhythm, and no epsilon waves. Right heart catheterisation revealed a relatively normal cardiac output, but equalisation of pressures with the right atrial pressure, the pulmonary artery diastolic pressure and the wedge pressure were all approximately 20 mmHg. Pulmonary artery systolic pressure was 37 mmHg; therefore, the patient was referred for cardiac MRI.

MRI was performed using a 1.5 T whole-body MR unit (Sigma; GE Medical Systems, Milwaukee, WI) with a standardised cardiac MRI protocol. After triplanar localising images, left ventricular function was assessed utilising a steady-state free precession (SSFP) sequence in the two-, three- and four-chamber long-axis orientations and in the short-axis orientation with the following parameters: echo time (TE)=1.4 ms; repetition time (TR)=3.2 ms; field of view (FOV)=40 × 40 cm; slice thickness, 8 mm; flip angle (FL)=45°. Pericardial wall thickness was assessed utilising double inversion-recovery (IR) T_2 weighted images in the 4-chamber view, obtained with the following parameters: TE=84 ms; TR=1481 ms; FOV=32 × 32 cm; slice thickness, 8 mm; FL=90°.

Double IR T_2 weighted images showed diffuse pericardial thickening, measuring approximately 7 mm. The pericardium anterior to the focal right ventricular free wall outpouching was measured as normal and was 1 mm. Pericardial thickening was causing some mass effect on the heart, which was manifested by dilation of the inferior vena cava and hepatic veins and relative atrial enlargement. Dyskinetic systolic motion of the right ventricle was observed on SSFP cine images, causing aneurysm-like outpouching during systole in the right ventricle free wall, which was covered by normal pericardium (Figure 1a,b). Paradoxical septal motion was also noted, compatible with CP. There was no evidence of pericardial contrast enhancement. Delayed enhancement or thrombus within the right ventricular aneurysm was not observed (Figures 1–3). Left ventricular function, wall thickness and enhancement were normal.

In order to evaluate pericardial calcification, non-contrast chest CT was performed using a 64-channel multidetector CT scanner (VCT; GE Healthcare, Milwaukee, WI), with the following parameters: detector collimation, 0.625; FOV=32.8 cm; pitch, 1.3; tube voltage,

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Figure 1. Four-chamber fast imaging employing steady-state acquisition (FIESTA) cine images (a, b) show focal aneurysm-like outpouching of the right ventricular free wall (arrows).

Figure 2. Double inversion-recovery T_2 weighted images show diffuse thickening of the (a) pericardium (white arrow) and (b) focal area of the normal thickness pericardium (yellow arrow).

Figure 3. Axial T_2 weighted fast imaging employing steady-state acquisition (FIESTA) images show intra-abdominal ascites and a dilated inferior vena cava and hepatic veins, indicative of constrictive physiology.

120 kV; tube current range, 155–332 mAs. Images were reconstructed in 5.0 mm-thick axial soft tissue and lung algorithms and were reviewed on a PACS (Philips Medical Systems, Bothell, WA) station. Non-contrast CT study showed similar findings to the MRI, including a thickened pericardium, measuring up to 9 mm. At least one area of pericardial calcification was noted (Figure 4). Dilated hepatic veins and inferior vena cava again demonstrated changes consistent with a right ventricular overload. Additionally, a small amount of abdominal ascites and passive hepatic congestion were seen. A right ventricular aneurysm-like outpouching, measuring 2×1.5 cm, was noted (Figure 4).

The imaging findings were consistent with CP with hepatic congestion and resultant liver dysfunction. It was unclear why the right ventricle free wall had an aneurysm. The patient was referred for surgical exploration and pericardiectomy. Intra-operatively, the focal right ventricle aneurysm was a morphologically normal right ventricle wall with the overlying area of normal pericardium, the only area of pericardium that was normal. The remainder of the pericardium was diffusely thickened and fibrotic, constricting the remainder of the right ventricle and the other chambers (Figure 5). Pericardiectomy was successfully performed. The post-operative period was uneventful and the patient was discharged; he remained symptom-free during the 4 month follow-up.

Figure 4. Axial non-contrast CT images in the soft-tissue window (a, b) show thickened pericardium, tiny areas of calcification and aneurysm-like outpouching arising from the right ventricular free wall (arrow).

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 [5]. 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 a focal region of sparing. The part of the right ventricle free wall that was covered with normal pericardium was not constricted and the adjacent thickened pericardium was stiff enough that this normal region protruded in between the edges of thickened pericardium, simulating an aneurysm. The region of right ventricle that appeared abnormal and aneurysmal was the only normal functioning portion of the right ventricle free wall, with the only overlying normal pericardium.

Conclusion

Diffuse CP with a focal area 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.

References

1. Sengupta PP, Eleid MF, Khandheria BK. Constrictive pericarditis. *Circ J* 2008;72:1555–62.

Figure 5. Gross pathology shows dissected thickened and fibrotic pericardium.

Case report: Constrictive pericarditis presenting with an outpouching

2. Baccouche H, Ursulescu A, Yilmaz A, Ott G, Klingel K, Zehender M, et al. Images in cardiovascular medicine. Right ventricular false aneurysm after unrecognized myocardial infarction 28 years previously. *Circulation* 2008;118:2111–14.
3. Teixeira Filho GF, Schwartzmann P, Kersten RN. Right ventricular aneurysm following right ventricular infarction. *Heart* 2004;90:472.
4. Bogaert J, Francone M. Cardiovascular magnetic resonance in pericardial diseases. *J Cardiovasc Magn Reson* 2009;11:14.
5. Wang ZJ, Reddy GP, Gotway MB, Yeh BM, Hetts SW, Higgins CB. CT and MR imaging of pericardial disease. *Radiographics* 2003;23:S167–80.
6. Kayser HW, van der Wall EE, Sivananthan MU, Plein S, Bloomer TN, de Roos A. Diagnosis of arrhythmogenic right ventricular dysplasia: a review. *Radiographics* 2002;22: 639–48; discussion 649–50.
7. Inoue S, Murakami Y, Shimada T, Inoue A, Maruyama R. Right ventricular aneurysm caused by acute myocarditis. *Can J Cardiol* 2000;16:1025–8.
8. Hasuda T, Satoh T, Yamada N, Sakamaki F, Kyotani S, Nakanishi N, et al. A case of constrictive pericarditis with local thickening of the pericardium without manifest ventricular interdependence. *Cardiology* 1999;92: 214–16.