Extensive pericardial thickening without constriction

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ABSTRACT

Abstract is not required for Clinical Images
CASE REPORT

A 73-year-old male presented with atypical chest pain. He had no significant past medical history of note and he was otherwise fit and well and not on prescribed medications. He was an ex-smoker and had no family history of cardiovascular diseases. Physical examination was unremarkable and revealed a normal blood pressure, non-elevated jugular venous pressure, normal heart sounds and no other significant abnormal findings. Laboratory investigations showed normal blood count and normal kidney and liver function tests.

In view of his presentation, age and history of smoking he underwent a coronary angiography procedure to exclude coronary artery disease. Unexpectedly, it showed a grey shadow around the heart mainly on the right side (Figure 1). Pericardial thickening and possible constrictive pericarditis was suspected, subsequently, a right heart catheterization was performed and intracardiac pressures were assessed. Intracardiac hemodynamics were entirely normal (right atrial pressure 3 mmHg, right ventricular and pulmonary artery systolic pressures 20 mmHg, mean pulmonary capillary wedge pressure 8 mmHg and left ventricular end diastolic pressure was 12 mmHg).

Transthoracic echocardiography suspected the presence of pericardial thickening. Otherwise there was no evidence of other structural heart abnormalities. A computed tomography scan of the thorax confirmed it to be pericardial thickening. No particular cause for the pericardial thickening was identified (idiopathic). He remained well and is under regular twelve monthly cardiology follow-up.

DISCUSSION

Pericardial constriction happen when a thickened, and frequently calcified pericardium impairs cardiac filling, limiting the total cardiac volume [1, 2, 3].

The most common causes include mediastinal radiation such as for Hodgkin’s lymphoma, chronic...
idiopathic pericarditis, post-cardiac surgery and tuberculous pericarditis [1, 4–6]. These conditions tend to cause chronic pericardial inflammation causing pericardial scarring with thickening, fibrosis, and calcification [3].

The pathophysiological hallmark of pericardial constriction is equalization of the end-diastolic pressures in all four cardiac chambers; as a result systemic congestion is much more marked than pulmonary congestion. Typical presentations are due to elevated systemic venous pressures and low cardiac output [5].

The increased systemic venous pressure lead to marked jugular venous distension, hepatic congestion, ascites, and peripheral oedema, while the lungs remain clear. Low cardiac output causes exercise intolerance that could progress to cardiac cachexia and muscle wasting.

Transthoracic echocardiography is an essential diagnostic test in patients being evaluated for constrictive pericarditis.

Computed tomography scan of the heart is useful in the diagnosis of constrictive pericarditis and can provide additional data to guide perioperative management decisions.

Gated cardiac magnetic resonance imaging provides direct visualization of the normal pericardium, which is composed of fibrous tissue and has a low MRI signal intensity [7]. CMR is advocated by some as the diagnostic procedure of choice for the detection of certain pericardial diseases, including constrictive pericarditis [8–10]. Characteristic CMR features in patients with constrictive pericarditis include increased pericardial thickening and dilatation of the inferior vena cava, an indirect sign of impaired right ventricular diastolic filling.

Pericardiectomy is the only definitive treatment option for patients with chronic symptomatic constrictive pericarditis.

Most patients with pericardial constriction have a thickened pericardium (>2 mm) [1, 3, 11]. It is important to recognize, however, that pericardial constriction can be present without pericardial calcium and, in some cases, even without pericardial thickening. In a series of 143 patients from the Mayo Clinic with surgically proven pericardial constriction, 26 (18%) had a normal pericardial thickness (<2 mm) [12].

So it is possible to have constrictive pericarditis without pericardial thickening, but it is not common to have pericardial thickening without constriction.

Our case demonstrates that in spite of extensive pericardial thickening, our patient was asymptomatic with no hemodynamic evidence of constriction during cardiac catheterization which is an unusual occurrence.

CONCLUSION

Not all patients with pericardial constriction have pericardial thickening and not all patients with pericardial thickening have pericardial constriction.


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