

Acute Perimyocarditis Masquerading as Acute Coronary Syndrome with Spontaneous Resolution of Increased Left Ventricular Wall Thickness

Dear Editor,

A 20-year-old normotensive male presented with sudden onset of near syncope, chest discomfort and dyspnoea. The patient has no known cardiovascular risk factors, including history of smoking, diabetes mellitus, dyslipidaemia and positive family history of ischaemic heart disease. Electrocardiogram (ECG) showed ST-elevation in the anterior and inferior leads (Panel A). Total white blood cell count was elevated at $16.9 \times 10^9/L$ with predominance of neutrophils ($13 \times 10^9/L$). Serum cardiac markers were also elevated: creatine kinase, 342 U/L; MB fraction, 24.3 ug/L; troponin T, 1.01 ug/L. Initially, myocardial infarction (MI) was suspected. Emergent coronary angiography was performed and it was normal. Echocardiography revealed increased left ventricular (LV) wall thickness, hypokinesia in the inferior and inferolateral walls, preserved ejection fraction (EF) and mild pericardial effusion (Panel B). The patient was treated with anti-platelet therapies and investigated for possible causes of young MI. In addition, he was questioned for the possible use of cocaine which he denied. Serum levels of Lp(a), fibrinogen, anti-cardiolipin antibodies, anti-thrombin III, lupus anticoagulant and screen, protein C and S activities and activated protein C resistant test were normal.

Four days later, cardiovascular magnetic resonance (CMR) showed an undilated LV with increased myocardial thickness of 12 mm at the inferolateral wall at end-diastole (Panel D). LV mass index was elevated at 109 g/m^2 and LVEF was preserved (74%). Late gadolinium enhancement was documented in the subepicardial lateral wall and mid interventricular septum (Panel E). There was moderate pericardial effusion. These findings were consistent with acute perimyocarditis. There was no sub-endocardial late gadolinium enhancement.

The patient was discharged without any medication. Investigations for infective aetiologies including mycoplasma serology, urine for legionella and stool for enterovirus were negative. At 8 weeks, with spontaneous clinical improvement, CMR documented normalisation of wall thickness to 8 mm and LV mass index of 61 g/m^2 . There was no residual myocardial late gadolinium enhancement. Echocardiography (Panel C) and ECG also normalised. The patient then resumed vigorous activity.

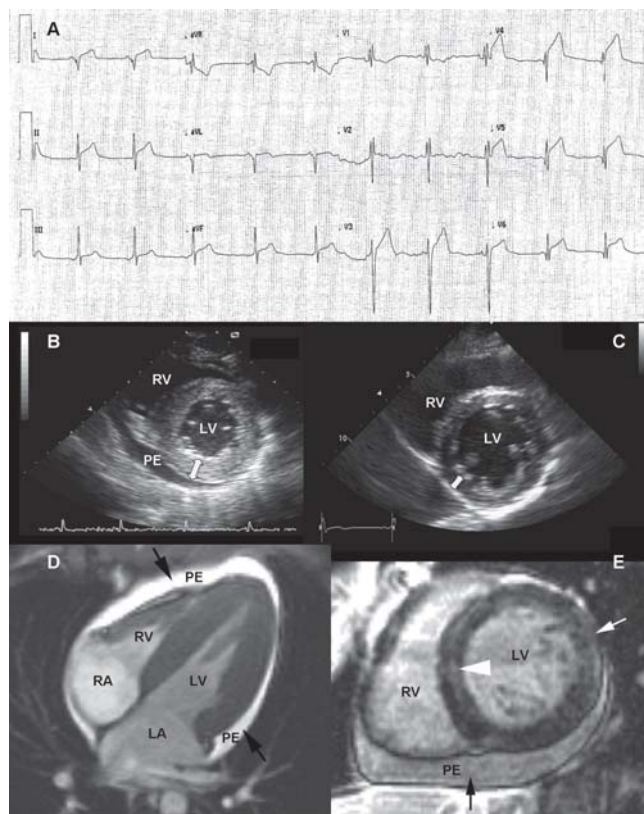


Fig. 1. 12-lead ECG showed mildly concave ST elevations in multiple leads, right bundle branch block morphology and Q waves in I, aVL, V5-6 (Panel A). Echocardiography performed on admission, demonstrated increased wall thickness (arrows) and presence of pericardial effusion (PE) at short axis window (Panel B). Follow-up echocardiography at 10 weeks showed resolution of increased wall thickness and PE (Panel C). Cardiac magnetic resonance imaging at 4 days after admission showed steady state free precession gradient echo images in the 4-chamber (end-systole) view of the heart (Panel D). Note the PE and thickened myocardium consistent with findings on echocardiography. T1-weighted contrast-enhanced gradient echo image acquired about 10 minutes after gadolinium administration, showed subepicardial late gadolinium enhancement was present in the lateral wall (white arrow) (Panel E). Mid-wall late enhancement in the ventricular septum was indicated by the white arrowhead (Panel E).

LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle

Discussion

Clinical presentation of chest discomfort associated with ST-elevation on ECG, elevated cardiac enzymes and regional wall motion abnormalities on echocardiogram often connote myocardial injury. Acute ST-elevation MI

has to be ruled out. Though the urgent coronary angiogram was normal, it does not exclude transient coronary occlusion with spontaneous clot lysis or coronary spasm. The patient was managed medically in the coronary care unit. The presenting ECG was unusual: multi-lead concave upward ST-elevation and Q waves in leads I, aVL, V5-6 suggested pericardial inflammation and transmural injury. However the diagnosis of perimyocarditis is notoriously difficult, often mimicking MI.¹ It is important to exclude causes of young MI including tobacco or cocaine abuse, though the latter can also result in myopericarditis.²

Evidence-based controlled clinical trials on treatment modalities are lacking. Although aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) are the mainstay of therapy for viral or idiopathic pericarditis, it should be prescribed with caution in the presence of predominantly myocarditis. In animal models of myocarditis, NSAIDs are not only ineffective but may enhance the myocarditic process and increase mortality.² In clinical practice, lower anti-inflammatory doses may be considered to control symptoms. Corticosteroids use should also be restricted as they have been shown to be risk factors for recurrences. In this case, initial anti-platelet therapy prescribed for presumed MI may paradoxically be harmful if continued at high dose without subsequently identifying correct aetiology. There appears to be no specific therapy for perimyocarditis. However, several non-specific measures may be important. These include medical support and after the acute phase of perimyocarditis, rest, restriction of strenuous exercise and alcohol intake.² Our patient, on discharge, was not prescribed any medication as he was asymptomatic. Instead he was given extended medical leave.

This case also demonstrates reversible increase in myocardial thickness and mass that sometimes accompanies acute myocarditis. This rare finding is most likely due to myocardial oedema. At first presentation, the increased myocardial thickness was surprising as the patient has no history of hypertension and no evidence of LV outflow tract obstruction.

Contrast-enhanced cardiovascular magnetic resonance is able to differentiate acute myocarditis from MI. In acute myocarditis, myocardial late gadolinium enhancement is present in up to 88% of cases.^{3,4} The distribution of gadolinium uptake is characteristically patchy, does not conform to any particular coronary territory, usually in the sub-epicardial or mid-wall layers of the myocardium, and never in the sub-endocardium alone.⁵ The lateral wall and ventricular septum are often involved. In patients who undergo endomyocardial biopsy, sampling of the regions of gadolinium uptake can increase the accuracy of diagnosis

of acute myocarditis. In contrast, in acute MI, gadolinium uptake is always sub-endocardial extending to the sub-epicardium. The late enhancement region corresponds to the territory of the infarct-related artery. In some patients with acute MI, a dark hypointense sub-endocardial layer may be seen within the bright infarct zone. This represents an area of microvascular obstruction, and is an independent predictor of worse prognosis.⁶

Echocardiography and CMR are useful non-invasive diagnostic imaging modalities, able to document changes in LV wall thickness and mass in the evolution of the disease process. Echocardiography is easily accessible and provides information rapidly. Complementary use of CMR allows further myocardial characterisation.³ Perimyocarditis may be difficult to recognise initially resulting in uncertain treatment strategies.² Imaging can provide insights into the underlying aetiologies, guiding further clinical management.

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