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A dramatic example of pseudodyskinesis of the left ventricle

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A 62-year-old woman was evaluated for worsening dyspnoea. She had previously undergone coronary artery bypass surgery and had a history of a chronic loculated left pleural effusion (PE), which had previously been treated with repeated thoracentesis and talc pleurodesis. An echocardiogram was obtained, which was of sub-optimal image quality, but it did demonstrate a small left ventricular (LV) chamber and normal LV ejection fraction. Pulsed wave Doppler of mitral valve inflow revealed significant respirophasic changes of the E-wave velocity (>25%), a short deceleration time (<160 ms), and an 'L' wave. There was also apparent ventricular interaction. Together these findings raised suspicion for constrictive pericarditis (CP), with the thought being that this condition resulted in dyspnoea due to impaired LV compliance, elevated filling pressures, and an inability to augment stroke volume with exertion.

Cardiac magnetic resonance (CMR) imaging was performed to evaluate the possibility of CP. Cine CMR was performed at a 1.5-Tesla MR scanner using an electrocardiogram gated steady-state free precession pulse sequence. As shown in a short-axis orientation of the heart, there is dyssynchronous contraction of the LV posterior wall but normal wall thickening (Videos 1–3). During systole, there is pronounced posterior motion of the LV posterior wall followed by flattening and compression of the same wall during diastole (*Figures 1* and *2* and Supplementary material online, *Figure S1*). This phenomenon has been described in two-dimensional echocardiography as pseudodyskinesis and is commonly associated with liver or parenchymal lung disease.[1]

Video 1 Basal segment. Cardiac magnetic resonance steady-state free precession imaging of a short-axis view of the left ventricle which is surrounded by a large pleural effusion. During systole, there is pronounced outward motion of the posterior wall of the left ventricle; in diastole, there is inward motion of the same wall.

The abnormal systolic motion of the posterior wall has previously been attributed to diaphragmatic elevation resulting from intrathoracic or intrabdominal pathology. Pleural effusions, likewise, can have a significant impact on cardiac haemodynamics. A study of 47 subjects with large PE by Wang *et al.*[2] demonstrated that drainage of the PE resulted in significant increases in LV end-diastolic volume index, LV ejection fraction, and stroke volume. In rare instances, large left-sided PEs have even been shown to result in cardiac tamponade.[3]

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Video 2 Mid segment. Cardiac magnetic resonance steady-state free precession imaging of a short-axis view of the left ventricle which is surrounded by a large pleural effusion. During systole, there is pronounced outward motion of the posterior wall of the left ventricle; in diastole, there is inward motion of the same wall.

Video 3 Apical segment. Cardiac magnetic resonance steady-state free precession imaging of a short-axis view of the left ventricle which is surrounded by a large pleural effusion. During systole, there is pronounced outward motion of the posterior wall of the left ventricle; in diastole, there is inward motion of the same wall.

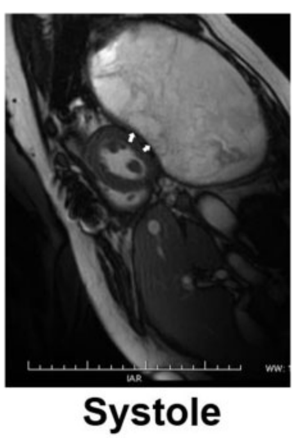

Figure 1 Cardiac magnetic resonance systolic frame showing normal wall thickening and protrusion of the posterior wall of the left ventricular towards a large left pleural effusion (see white arrows).

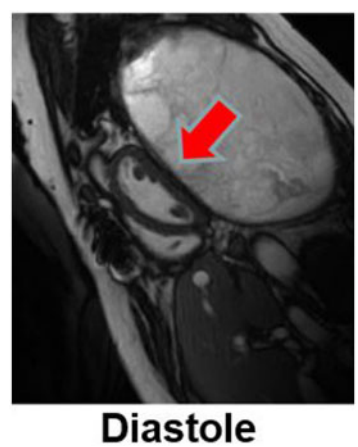

Figure 2 Cardiac magnetic resonance diastolic frame showing flattening and compression of the posterior wall of the left ventricular due to increased pleural pressures secondary to the loculated effusion (see red arrow).

In our case, we hypothesize that the patient's left-sided PE is causing compression of the LV posterior wall in diastole impairing LV filling and reducing preload; in systole, the process is reversed by the higher intracardiac pressure (Supplementary material online, *Figure S2*). The patient was taken to the operating room and underwent a left thoracotomy and decortication for a large haemothorax and treatment of suspected'trapped lung' syndrome. The patient's dyspnoea resolved status-post-thoracotomy and decortication.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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