Multiple, extensive intracardiac thrombi in patient with ischaemic cardiomyopathy

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DESCRIPTION
A 52-year-old man with ischaemic cardiomyopathy from severe triple coronary artery disease was referred to our hospital due to recurrent acute decompensated heart failure (ADHF). He has a history of myocardial infarction diagnosed 5 months ago. On admission, the patient had symptoms and signs of lung congestion with poor tissue perfusion (wet and cold). A 12-lead ECG revealed sinus rhythm, left axis deviation and poor R progression. An echocardiogram showed severely dilated left ventricular (LV) chamber and severely impaired systolic function with an ejection fraction of 18%. There were regional myocardial wall thinning and akinesis of basal-to-apical anterior, septal and inferior walls. An apical LV thrombus was noted. A cardiovascular magnetic resonance (CMR) was scheduled to evaluate myocardial viability, which showed that the LV myocardium in the territory of left anterior descending, and right coronary arteries were non-viable on delayed enhancement images (bright areas on figure 1B). The right ventricular systolic function was normal with an ejection fraction of 50%. Moreover, a large right atrial (RA) thrombus and three LV thrombi at apical, anterior and inferior walls were visualised with largest measuring 3.2×2.2 cm (arrows on figure 1). The laboratory workup for hypercoagulable states was unremarkable. The patient was treated with inotropic agents, diuretics and warfarin. Guideline-directed medical therapy was subsequently initiated when the ADHF improved. After a multidisciplinary heart team discussion, the patient has been listed to heart transplantation for the definitive treatment. A 2-month follow-up imaging demonstrated minimal change in size of thrombi.

The LV thrombus is not uncommonly found in patients with ischaemic cardiomyopathy.1 However, multiple sites of LV thrombi with RA appendage thrombus in ischaemic cardiomyopathy with sinus rhythm are rare.2-4 The Virchow’s triad, which consists of blood stasis, endothelial injury and hypercoagulable state, could explain the intracardiac thrombus formation. Multiple sites of left ventricular thrombi with RA appendage thrombus in ischaemic cardiomyopathy with sinus rhythm are rare.2-4 The Virchow’s triad, which consists of blood stasis, endothelial injury and hypercoagulable state, could explain the intracardiac thrombus formation like other vascular thrombi. In our case, the LV thrombi are located at the non-viable and akinetic segments as a result of blood flow stagnation. However, the blood in RA is not stagnant thus the possible explanation for the RA thrombus formation is likely to be the prothrombotic state from chronic liver congestion and synthetic dysfunction.2-4 The CMR is considered as the gold standard modality to detect an intracardiac thrombus with the highest sensitivity, specificity, positive predictive value and negative predictive value.1 An intracardiac thrombus can cause an embolic event leading to catastrophic morbidity and mortality. Early detection with prompt initiation of anticoagulant is the key management to prevent disability and improve survival.

Learning points
- Multiple sites of left ventricular thrombi with right atrial (RA) appendage thrombus in ischaemic cardiomyopathy with sinus rhythm are rare.
- It has been hypothesised that the prothrombotic state from chronic liver congestion and synthetic dysfunction could account for the thrombus formation in area without blood flow stagnation such as RA.

Patient’s perspective
I was frightened when the doctor first told me that I had several blood clots in my heart. I later on was glad to have it detected early and treated before it goes to my brain.

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