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CASE REPORT
Cardiac imaging

Extensive intracardiac thrombi in a patient with heart failure and hepatic congestion: a case report

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Background Left ventricular (LV) thrombi is a complication associated with anterior ST-elevation myocardial infarction, dilated cardiomyopathies, or LV aneurysms. Right sided intracardiac thrombi may be associated with other prothrombotic causes.

Case summary A 66-year-old man admitted with congestive heart failure was found to have extensive intracardiac masses on transthoracic echocardiography and cardiac magnetic resonance imaging (MRI). This occurred in the absence of a recent myocardial infarction. During his hospital stay, he was found to have deranged liver enzymes and coagulation profile due to hepatic congestion. The patient was presumed to have intracardiac thrombi and was treated with warfarin therapy. There was complete resolution of the masses on repeat cardiac MRI after 4 weeks of treatment, confirming the diagnosis.

Discussion Cardiac MRI is useful in the diagnosis of intracardiac thrombi. Clinicians should appreciate the prothrombotic risks associated with liver disease, despite the inability of standard coagulation tests to quantify this risk.

Keywords Intracardiac thrombi • Cardiac magnetic resonance imaging • Liver disease • Congestive cardiac failure • Case report

Learning points

- Right sided intracardiac thrombi should prompt the clinician to search for causes of hypercoagulability.
- T1- and T2-weighted sequences on cardiac magnetic resonance imaging may be useful to determine the age of intracardiac thrombi.
- Standard coagulation profiles do not measure the prothrombotic risk of patients with liver disease.

Introduction

Left ventricular (LV) thrombi are a complication associated with anterior ST-elevation myocardial infarction, dilated cardiomyopathies, or LV aneurysms.[1] The incidence of LV thrombi has decreased in recent years with the improvement in primary reperfusion strategies, which decrease infarct size and the development of LV aneurysms.[2–4] Unlike LV thrombi, the development of right heart thrombi is often precipitated by non-infarct causes, and they are morphologically heterogeneous. Type A thrombi are mobile, serpiginous and have a

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high association with deep vein thrombosis. Type B thrombi have a non-specific shape, are less mobile, and often resemble LV thrombi.[5] Right heart thrombi have been reported in the presence of right ventricular (RV) pacemaker leads, right sided mechanical valves, and ventricular or atrial septal closure devices.[6,7]

Timeline

Date	Events
22 May 2018	Presented with symptoms of biventricular heart failure. Transthoracic echocardiography showed biventricular dilatation with severe systolic dysfunction and multiple masses in both apices and the right atrium
24 May 2018	Cardiac magnetic resonance imaging (MRI) showed ischaemic scars and confirmed intracardiac masses
01 June 2018	Cardiac catheterization showed occluded left circumflex and right coronary arteries, and severe stenosis of the mid-left anterior descending artery
11 June 2018	Discharged on warfarin and medical therapy for heart failure
28 June 2018	Repeat cardiac MRI showed complete resolution of intracardiac masses
01 July 2018	Out of hospital cardiac arrest, admitted to intensive care unit
05 July 2018	Percutaneous coronary intervention to left anterior descending artery
06 July 2018	Implantable cardioverter defibrillator inserted
11 July 2018	Patient discharged home

Case presentation

A 66-year-old man without any prior cardiac history presented to the hospital with a 1-month history of dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, and 4 kg unintentional weight loss. On admission, he had a blood pressure of 103/81 mmHg, heart rate 90 b.p.m., respiratory rate 20 b.p.m., and an oxygen saturation of 98% on room air. Jugular venous pressure was elevated at +4 cm, and he had bilateral peripheral oedema to the knees. Auscultation revealed a pansystolic murmur at the apex with a third heart sound, and bilateral crackles at the lung bases. Initial laboratory tests showed an elevated N-terminal pro-brain natriuretic peptide [2056 pmol/L (reference range <35)] and normal troponin I [21 ng/L (reference range <40)]. Electrocardiogram showed sinus rhythm, left axis deviation, inferior Q-waves, and no ST changes. He was referred for transthoracic echocardiography which revealed dilated left and right ventricles. The LV internal diameter end diastole and systole were 64 mm and 61 mm, respectively, with a LV ejection fraction (LVEF) calculated by Simpson's biplane method of 17%. There were moderate mitral and tricuspid regurgitation secondary to the dilated ventricles. The pulmonary artery systolic pressure was 34 mmHg, and the estimated mean right atrial pressure was 10–15 mmHg, as evidenced by an inferior vena cava (IVC) diameter of 17 mm which collapses less than 50% on inspiration. There were masses in both apices and two further masses in the right atrium suspicious for type B thrombi (*Figure 1* and Supplementary material online, *Video S1*). The cardiomyopathy and intracardiac masses were further analysed with cardiac magnetic resonance imaging (MRI) (Supplementary material online, *Video S2*), which showed the intracardiac masses had high signal intensity on T1- and T2-weighted sequences (*Figure 2*). They were low-signal on first pass imaging following gadolinium administration. The LV and RV end-diastolic volumes were 385 mL and 305 mL, respectively. The calculated LVEF was 19% and RV ejection fraction (RVEF) 20% (*Table 1*). Additionally, there was evidence of fluid overload with small bilateral pleural effusions and an IVC diameter of 29 mm. Delayed enhancement sequences showed near full thickness scars in the lateral and inferior walls, consistent with prior myocardial infarctions (*Figure 3*). Coronary angiography revealed severe triple vessel disease, including occlusion of the left circumflex and right coronary arteries. The presence of suspected right heart thrombi led to the investigation of hypercoagulable states. His weight loss raised the suspicion of underlying malignancy, but a subsequent computed tomography of his body and pulmonary angiogram was negative for malignancy or pulmonary embolism. His weight loss was therefore attributed to cardiac cachexia. Review of the admission blood work identified elevated liver enzymes; aspartate aminotransferase 106 U/L (reference range <45), alanine aminotransferase 406 U/L (reference range <45), gamma-glutamyl transferase 64 U/L (reference range <60), and hyperbilirubinemia 60 μmol/L (reference range <25). His coagulation profile revealed a prolonged prothrombin time [24 s (reference range 10–14)], prothrombin ratio [2.0 (reference range 0.8–1.2)], and activated partial thromboplastin time [43 s (reference range 25–38); which fully corrected with the addition of normal plasma]. It was likely that these derangements were due to hepatic congestion from his RV failure, as his abnormal liver enzymes and coagulation profile normalized following diuresis. The patient was started on warfarin with bridging enoxaparin, for the likely diagnosis of multiple intracardiac thrombi. Revascularization was postponed until resolution of the thrombi to avoid increased bleeding risk from triple antithrombotic therapy, due to risk of further hepatic congestion and coagulopathy. The patient was discharged home on warfarin, lisinopril 10 mg daily, frusemide 40 mg daily, and carvedilol 12.5 mg twice daily. He returned 1 month later for a follow-up MRI, which showed the LVEF was still significantly reduced at 22%, but the RVEF had improved to 46%. There was complete resolution of the all the intracardiac masses, therefore confirming the diagnosis of thrombi (*Figure 4*). Unfortunately, the patient had an aborted out of hospital cardiac arrest shortly thereafter, which led to percutaneous coronary intervention to his left anterior descending

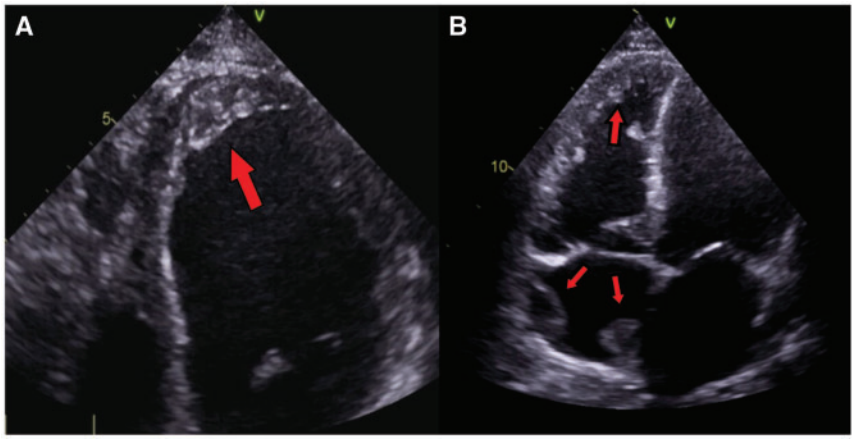

Figure 1 Transthoracic echocardiography showing a mass in the apex of the left ventricle (*A*) and multiple masses (red arrows) in the right atrium and ventricle (*B*).

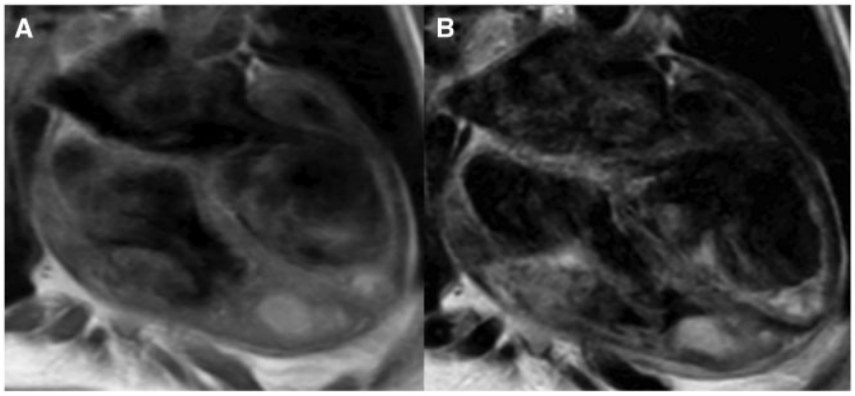

Figure 2 Cardiac magnetic resonance imaging in four chamber views showing high intensity signal for the intracardiac masses on T1 (*A*) and T2-weighted (*B*) black blood sequences, suggestive of acute thrombi.

artery, and subsequent insertion of an implantable cardiac defibrillator.

Discussion

Intracardiac thrombi are associated with morbidity and mortality related to embolization. Empiric anticoagulation in heart failure without other indications such as atrial fibrillation and prosthetic valves to reduce thromboembolism has been well studied. The WARCEF and COMMANDER HF trials showed no benefit for warfarin and rivaroxaban, respectively.[8,9] T1 and T2 sequences on cardiac MRI may be helpful in differentiating the age of thrombi. Different signal intensities of thrombi on cardiac MRI has been linked with the age of haemoglobin degradation, with progressive loss of T1 and T2 signals as the thrombi becomes more organized.[10] The presence of right heart thrombi, particularly in the right atrium, should prompt the clinician to search for prothrombotic conditions. These may include liver disease, sepsis, disseminated intravascular coagulation, or hereditary thrombophilia in younger patients. Liver disease is commonly associated with increased bleeding risk due to its effects on platelets and clotting factors. However, it is also associated with prothrombotic

Table 1 Volume measurements of left and right ventricles on cardiac magnetic resonance imaging

	Left ventricle	Right ventricle
End diastolic volume	385 mL (190 mL/m^2)	302 mL (149 mL/m^2)
End systolic volume	312 mL (154 mL/m^2)	242 mL (119 mL/m^2)
Stroke volume	73 mL (36 mL/m^2)	60 mL (30 mL/m^2)
Ejection fraction	19%	20%

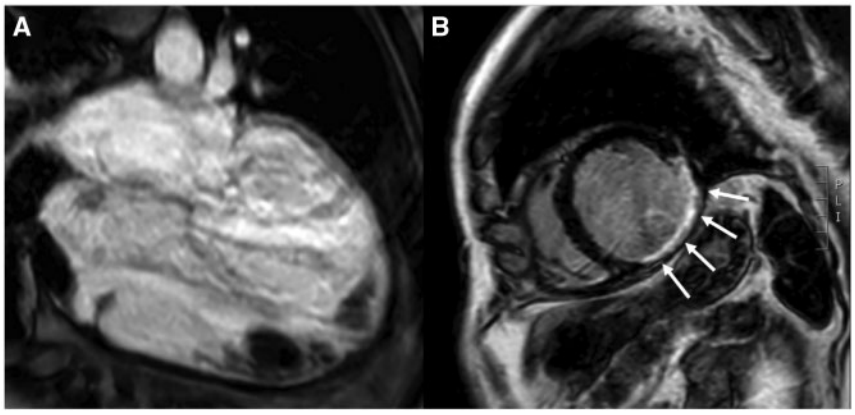

Figure 3 First pass imaging following gadolinium administration showing lack of perfusion in the intracardiac masses (*A*). Delayed enhancement sequence in the short-axis view showing near full thickness scar in the lateral and inferior walls (*B*).

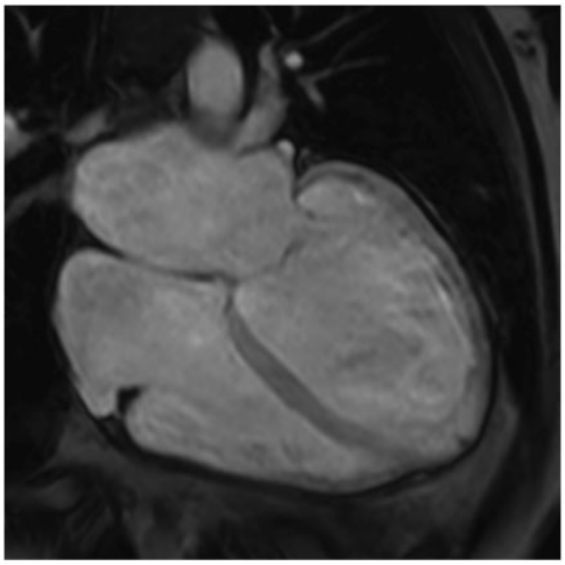

Figure 4 First pass imaging following gadolinium administration after 1 month of warfarin therapy showing complete resolution of all intracardiac masses.

changes due to reduced production of endogenous inhibitors of coagulation such as protein C and antithrombin, while levels of factor VIII are elevated.[11] Standard coagulation tests do not measure this prothrombotic risk, though it should be appreciated by clinicians when liver disease is present. Although the intracardiac thrombi cannot be linked directly to hepatic congestion in our patient, it occurred despite the impaired coagulation system associated with hepatic dysfunction, as supported by prolonged coagulation tests. This suggests that the combination of dilated cardiomyopathy and prothrombotic changes associated with hepatic dysfunction, resulted in a hypercoagulable state, which precipitated intracardiac thrombi formation.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

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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