A case of biventricular thrombi with complications

Mahima K Zandu [1], Pooja S Jagadish [2], Andrew R Williams,[2] Rajesh Janardhanan[2]

[1]Internal Medicine, The University of Arizona College of Medicine, Tucson, Arizona, USA
[2]Division of Cardiology, The University of Arizona College of Medicine, Tucson, Arizona, USA

Correspondence to
Dr Mahima K Zandu; mahimazandu@email.arizona.edu

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DESCRIPTION

Biventricular thrombi are a rare phenomenon, occurring in patients with hypercoagulable states including antiphospholipid antibody syndrome,[1] hypereosinophilic syndrome,[2] cardiomyopathies,[3 4] myocarditis,[5] myocardial infarction (MI)[6] and HIV infection.[6] Transthoracic echocardiography is a low-cost, widely available and reproducible tool that can rapidly detect intracardiac thrombi.[7–10] By using ultrasound contrast, the sensitivity of detecting a thrombus increases from 35% to 64%.[8 11] Cardiac MRI is the gold standard for detecting intracardiac thrombus (sensitivity of 88%) and is helpful when the diagnosis is inconclusive on other modalities.[10 12] Transoesophageal echocardiography poorly visualises the left ventricular (LV) apex and is not recommended.[10]

A man in his mid-50s with tobacco dependence presented with pain, swelling and blue-purple discolouration of the legs that had worsened over 2 days. Two weeks prior, a respiratory tract infection left him bedbound with progressive pain during ambulation. The patient denied the history of thromboembolic phenomena, clotting disorders, stroke or MI. His only symptoms were fatigue, poor appetite and leg pain. He specifically denied chest pain, palpitations, dyspnoea, weight loss and bleeding.

On presentation, he was tachycardic. His blood pressure was 150/70 mm Hg. Physical examination of the lower extremities revealed grossly intact sensory and motor function bilaterally but absent dorsalis pedis on the right and posterior tibial pulses bilaterally with impending gangrene of all toes. ECG revealed non-specific anterolateral ST elevations. This prompted point-of-care echocardiogram which demonstrated biventricular thrombi and an LV ejection fraction estimated at <20%. These findings were confirmed by a formal echocardiogram (figures 1 and 2). Contrast-enhanced chest CT was performed which confirmed pulmonary embolism along with large-volume biventricular thrombi (figure 3A,B).

Laboratory studies revealed leucocytosis (13 100 K/µL) without eosinophilia, thrombocytopaenia (80 K/µL), elevated C reactive protein (173 mg/L) with normal sedimentation rate, a high creatine kinase (5688 mg/L), elevated D-dimer (7289 ng/mL) and negative COVID-19 PCR. High-sensitivity troponin rose from 29 to 33 ng/L. HIV and coagulopathy testing, including lupus anticoagulant, protein C, protein S and cardiolipin all returned negative.

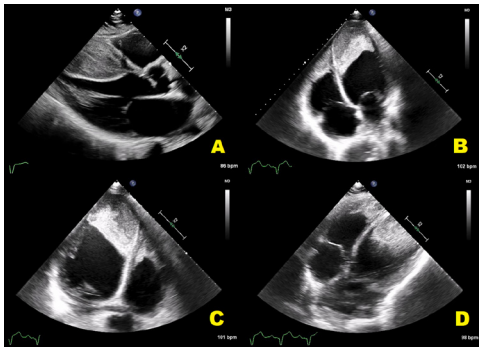

Figure 1 Transthoracic echocardiography without contrast demonstrating large-volume biventricular thrombi. (A) Parasternal long-axis view demonstrating thrombus in the left ventricle; (B) standard four-chamber view demonstrating thrombi in both ventricular apices; (C) off-axis/non-standard four-chamber view demonstrating thrombi in both ventricular apices and (D) subcostal view demonstrating large-volume biventricular thrombi. bpm, beats per minute.

For subacute bilateral limb ischaemia without Doppler-detectable pulses below the right posterior tibial artery, the vascular surgery team attempted catheter-directed tissue plasminogen

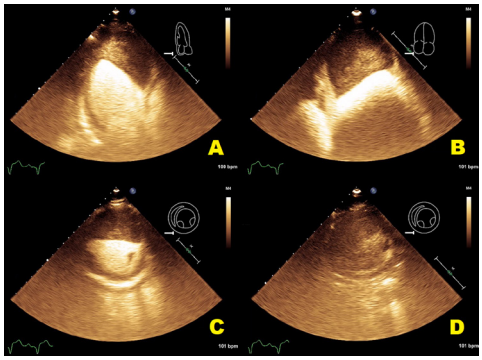

Figure 2 Transthoracic echocardiography with contrast highlighting the size and location of the biventricular thrombi. The contrast somewhat penetrates the left ventricular thrombus, indicating that it is not solid and is evolving. (A) Three-chamber view demonstrating the size and shape of the left ventricular thrombus; (B) standard four-chamber view demonstrating the defined border between the thrombus and the contrast-filled cavity and (C) short-axis view at the level of the mitral valve highlighting the partial opacification of the chamber at this level. The right ventricle is wholly opacified at this level and (D) short-axis view more apically displaced than (C), demonstrating total opacification of both chambers by thrombus. bpm, beats per minute.

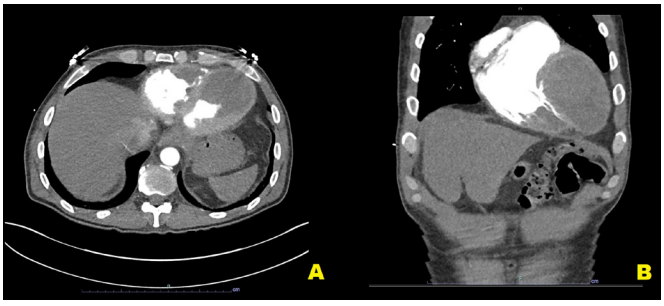

Figure 3 CT of the (A) chest and (B) abdomen noting biventricular thrombi obscuring most of the ventricles.

activator (tPA) that ultimately proved unsuccessful. Cardiac surgery to remove the ventricular thrombi was determined to be risk-prohibitive in the setting of evolving heart failure.

The patient subsequently developed an acute anterior ST-elevation MI complicated by ventricular tachycardia (VT) arrest. Cardiac catheterisation revealed an organised thrombus in the left anterior descending artery, indicating a chronic occlusion of days to weeks. Following shared decision-making with the patient's family, he was made comfort care. He expired 7 days after the initial presentation from VT.

The aetiology of biventricular thrombi is possibly myocarditis from an infection leading to cardiomyopathy and stasis of ventricular blood. Silent MI from the LV thrombus caused the ST elevations on presentation, leading to progressive myocardial ischaemia and cardiac arrest. Further decompensation resulted from peripheral arterial emboli, multiterritory cerebral infarcts and pulmonary embolism. This case highlights the role of point-of-care echocardiography as a quick tool to detect biventricular thrombi. The case also demonstrates multiple devastating complications in the same patient.

Learning points

- Point-of-care transthoracic echocardiography is a quick and easy modality for ventricular thrombus detection that can guide management and expedite treatment in acute settings.
- Ventricular thrombi are associated with a wide spectrum of complications including myocardial infarction, peripheral arterial embolism, stroke and pulmonary embolism.
- The differential diagnosis for the aetiology of biventricular thrombi includes hypereosinophilic syndromes, restrictive cardiomyopathy, viral infections, myocardial infarction and endomyocardial fibroelastosis.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

ORCID iDs
Mahima K Zandu http://orcid.org/0000-0003-2724-6417
Pooja S Jagadish http://orcid.org/0000-0002-6167-0820

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