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Clinical Image

Extensive Left Ventricular Thrombosis in a Young Patient: An Unexpected Threat

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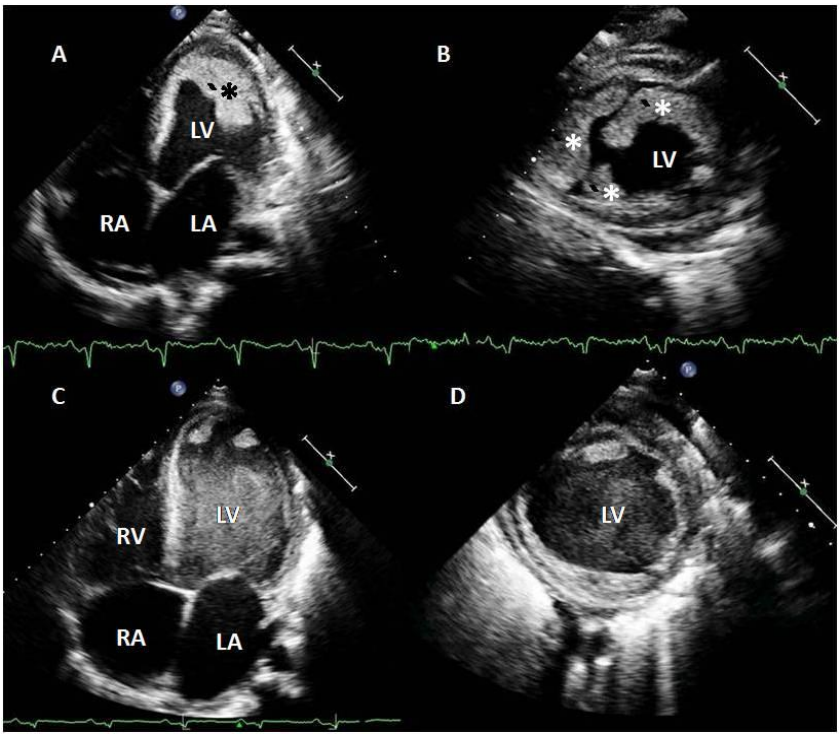

Figure 1: A. Apical 4-chamber view showing a huge parietal thrombus, covering the anterior, lateral and posterior walls (asterisk). B. Short axis showing a huge thrombus (asterisks). C. Apical 4-chamber after 3 weeks of treatment showing a drastic reduction in the thrombus and the presence of marked self-contrast. D. Short axis showing a reduction in the thrombus and the presence of self-contrast. RV – right ventricle; LV – left ventricle; RA – right atrium; LA – left atrium.

A 28-year-old Black man presented to the cardiology clinic with a 7-month history of progressive dyspnea on minimal exertion, orthopnea, paroxysmal nocturnal dyspnea, and lower limb edema. He reported moderate alcohol intake and denied any history of infection or use of pro-thrombotic substances. Transthoracic echocardiography revealed biventricular dilation, severe global hypokinesia with an ejection fraction of 18%, biatrial enlargement, and a large echogenic mass suggestive of thrombus. The mass exhibited well-defined contours, some protruding segments, no signs of pedunculation, and no apparent mobility. It involved the entire apical endocardium and extended along the anterior, lateral, and posterior walls, measuring about 13 mm in thickness in the apical segments and 6.5 mm in the mid segments. A prominent thrombus protrusion (12 mm) was noted at the mid-to-apical transition of the anterolateral Wall (Figure 1A and 1B).

Based on these findings, the patient was diagnosed with advanced congestive heart failure (NYHA class IV) due to idiopathic dilated cardiomyopathy. Cardiac magnetic resonance imaging and genetic testing were not performed due to their unavailability in the country at the time. Ventricular thrombus formation is explained by Virchow's triad: blood stasis, endothelial injury, and hypercoagulability. In patients with severe LV dysfunction, such as dilated cardiomyopathy or post-myocardial infarction, thrombus formation is common and carries a high risk of systemic embolism and stroke [1].

Left ventricular thrombus (LVT) is a potentially life-threatening complication of both ischemic and non-ischemic cardiomyopathies. Patients with an LVEF <20% are particularly at risk and are often considered for prophylactic anticoagulation. In confirmed LVT, anticoagulation is recommended for at least 3 to 6 months, with repeat imaging to assess resolution. Long-term management should be individualized, considering thrombus persistence, degree of ventricular dysfunction, embolic risk, bleeding risk, and response to heart failure therapy [2, 3]. Treatment options include vitamin K antagonists (VKAs), direct oral anticoagulants (DOACs), low-molecular-weight heparin, or intravenous unfractionated heparin. While recent studies have explored the use of DOACs, current guidelines still recommend warfarin as the preferred first-line agent [2, 4].

In this case, warfarin was chosen due to its accessibility, affordability, and ease of monitoring. The target INR was set between 3.0 and 3.5. After three weeks of therapy, the thrombus showed marked regression and was nearly resolved. Spontaneous echo contrast in the LV suggested ongoing thrombus lysis (Figure 1C and 1D). No embolic events were observed. The patient was simultaneously managed for Heart failure with enalapril, carvedilol, spironolactone, dapagliflozin, and furosemide, leading to significant clinical improvement. The patient was discharged in NYHA class II.

This case highlights the importance of early recognition and treatment of LVT in dilated cardiomyopathy. Prompt anticoagulation and optimized heart failure therapy are essential to reduce morbidity, prevent embolic events, and improve clinical outcomes.

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Conflicts of Interest: None.

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