



Large Intracardiac Thrombus in Advanced Heart Failure with Multiorgan Dysfunction

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Introduction

Intracardiac thrombus formation in patients with advanced heart failure remains a critical and complex issue, particularly when accompanied by multiorgan dysfunction. Large thrombi are associated with increased morbidity and mortality, posing significant therapeutic challenges [1]. We present the case of a 68-year-old male with chronic heart failure with reduced ejection fraction (HFrEF), chronic coronary syndrome, and multiple prior percutaneous coronary interventions (PCI), who developed a large left ventricular thrombus alongside acute hepatic and renal failure.

The patient was admitted in critical condition due to worsening heart failure, reporting progressive dyspnea and a decline in exercise tolerance over two weeks. Four days before admission, he developed petechial skin lesions. On examination, he presented with cachexia, jaundice, confusion, and hypotension. Laboratory tests indicated severe hepatic dysfunction (total bilirubin 5.2 mg/dL, ALT 311 U/L, AST 322 U/L), renal impairment (creatinine 3.9 mg/dL, eGFR 15 mL/min), thrombocytopenia (platelet count $140 \times 10^9/L$), and an elevated INR of 4.33 despite no vitamin K antagonist therapy. Transthoracic echocardiography revealed severe left ventricular systolic dysfunction (EF 20%) and a massive thrombus (59×29 mm) occupying half of the ventricular cavity. (Figure A, B,)

Given the hemodynamic instability and hepatic dysfunction, standard heart failure medications were discontinued, and anticoagulation with enoxaparin was initiated at 40 mg twice daily, later increased to 60 mg after an improvement in renal function. Supportive measures, including hepatoprotective therapy and volume resuscitation, were implemented. Follow-up echocardiography after four days demonstrated partial thrombus resolution, with a fragmented mass reduced to 17 mm. (Fig.D) Cardiac magnetic resonance (CMR) confirmed the presence of an apical thrombus and excluded left ventricular wall rupture.

Despite transient hepatic and renal improvement, the patient's condition deteriorated due to refractory hypotension that required norepinephrine and oliguria that was unresponsive to

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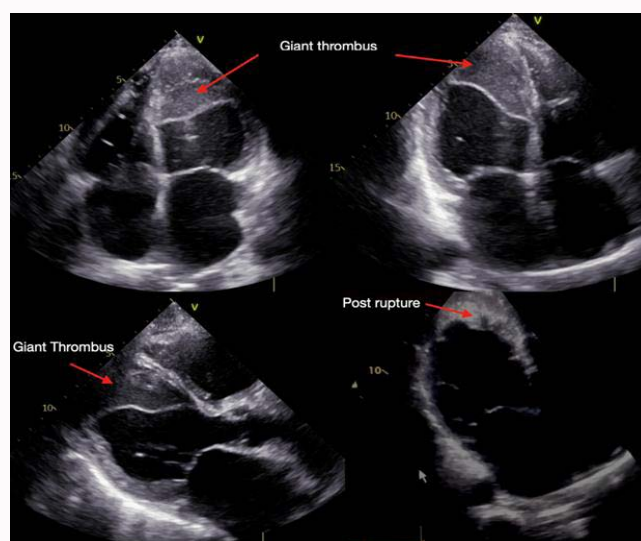


Figure 1:

diuretics. He experienced multiple ventricular tachycardia episodes necessitating implantable cardioverter-defibrillator shocks. Due to the thrombus, mechanical circulatory support was contraindicated. Ultimately, the patient succumbed to cardiogenic shock ten days after admission.

This case highlights the interplay of systemic dysfunction in advanced heart failure and the challenges of managing large intracardiac thrombi. The primary mechanisms of left ventricular thrombus formation include blood stasis due to poor contractility, hypercoagulability from systemic inflammation or liver failure, and extensive myocardial scarring post-infarction [2]. In this patient, ischemic heart disease contributed to ventricular dysfunction, but the predominant issue was heart failure exacerbation rather than an acute infarct.

While anticoagulation with enoxaparin facilitated partial thrombus resolution without embolic complications, progressive

hemodynamic instability and ventricular arrhythmias precluded definitive intervention. This case underscores the need for individualized management that integrates anticoagulation, heart failure therapy, and multiorgan support. The prognosis for patients with advanced heart failure and large intracardiac thrombi remains poor, emphasizing the importance of early detection and a multidisciplinary approach to optimizing outcomes.

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