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CASE REPORT

Coronary Embolism Caused by Massive Intraventricular Thrombus in a Young Patient with Thrombophilia and Drug-Induced Dilated Cardiomyopathy

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ABSTRACT

Introduction: Cardiac thrombi are often incidental findings on cardiac imaging and typically appear as uniform echo-densities. They develop in areas of blood flow stasis, particularly in structurally abnormal chambers such as in dilated cardiomyopathy. Their differential diagnosis includes vegetations and tumors, therefore accurate identification is critical for appropriate management. **Case Presentation:** We present the case of a 27-year-old male patient with a history of smoking and drug use, admitted for dyspnea and fatigue. Echocardiography revealed globally dilated chambers with severe apical hypokinesia and a large echogenic mass in the left ventricle. Laboratory investigations showed elevated hs-cTnI, NT-proBNP, and D-dimer levels. Coronary angiography demonstrated a subocclusive stenosis in the left anterior descending artery, likely from a partially recanalized thrombus, and was treated with angioplasty and drug-eluting stent placement. The thrombophilia panel revealed minor thrombophilia: MTHFR A1298C (homozygous), EPCR (A1/A2 Allele), Factor XIII and PAI-1 (heterozygous). The patient was managed conservatively with anticoagulation, heart failure therapy, and percutaneous coronary intervention. Follow-up showed a two-thirds reduction in thrombus size and stable status. **Conclusions:** This case highlights the importance of considering inherited thrombophilia in young patients presenting with intracardiac thrombus. For favorable outcomes, assessment of underlying thrombophilia, a combination of appropriate anticoagulation and interventional therapies are essential.

Keywords: intracavitary thrombus, dilated cardiomyopathy, thrombophilia

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INTRODUCTION

Thrombus formation in the left ventricle is a potentially life-threatening consequence of impaired ventricular function, often leading to embolic events and increased mortality. Treatment approaches, especially concerning the efficacy and safety of direct oral anticoagulants, remain inadequately defined and require further clinical clarification.[1] Myocardial infarction (MI) in young adults is a very uncommon finding, therefore discovering the underlying cause is of utmost importance.

When classifying the risk factors for MI by their nature, two main categories have to be considered: traditional risk factors and non-traditional risk factors. In younger individuals, the leading cause of acute MI is the rupture of atherosclerotic plaques, typically linked to traditional

cardiovascular risk factors such as dyslipidemia, hypertension, diabetes, smoking, obesity, and a family history of coronary artery disease.[2] Additionally, younger patients may experience more severe metabolic disturbances, such as treatment-resistant hypertension, familial hypercholesterolemia, or significant obesity, that make standard guideline-based treatments less effective.[3] Smoking remains the most prominent and widespread risk factor among young patients with MI, and it not only elevates the risk of an initial event but also significantly increases the chance of recurrent events. Substance abuse is another significant risk factor for MI in younger individuals, while its prevalence remains comparatively low in older populations. It is frequently linked to poor nutritional status, which is in turn associated with increased rates of recurrence and unfavorable clinical outcomes.[4,5] Recreational drug use, including substances such as cocaine and cannabis, has been reported in nearly 25% of young patients with acute coronary syndrome, and is frequently linked to larger infarct sizes and reduced left ventricular function compared to non-users.[6]

Ventricular thrombus formation is frequently associated with dilated cardiomyopathy (DCM), reduced left ventricular ejection fraction (LVEF), and acute coronary syndromes. In line with Virchow's triad, thrombi develop in the presence of blood stasis, endothelial injury, and hypercoagulable states.[7] These elements synergistically activate the coagulation cascade, promoting fibrin deposition and consequently left ventricular thrombus development, even in the presence of sinus rhythm.[8] Conditions such as DCM and acute coronary events can lead to impaired ventricular contractility manifesting as akinesia or dyskinesia, thereby promoting stasis. These pathologies are often accompanied by systemic inflammation, vascular damage, and a prothrombotic state.[7] The majority of DCM cases are idiopathic, while the rest may result from factors such as infiltrative diseases, coronary artery disease, alcohol abuse, certain medications, Takotsubo syndrome, or pregnancy.[9]

Transthoracic echocardiography (TTE) remains a fundamental tool in the assessment of intracardiac masses due to its accessibility and routine use in cardiac examination. For accurate identification, these masses should be visualized consistently across both systolic and diastolic phases and confirmed in multiple echocardiographic views.[10] However, cardiac magnetic resonance imaging and computed tomography (CT) might offer superior sensitivity and specificity for both identifying and monitoring the progression of a left ventricular thrombus.[11]

The aim of this paper is to highlight the clinical presentation, diagnostic challenges, and management of an atypical case involving a large left ventricular thrombus in a young patient with no prior cardiovascular disease.

CASE PRESENTATION

A 27-year-old male with a history of smoking and drug abuse, but no known cardiovascular disease, presented to the emergency department with complaints of progressive dyspnea and fatigue for the past month. He denied chest pain, syncope, or palpitations. Clinical examination showed peripheral edema, tachycardia (heart rate 105 bpm), mild psychomotor agitation, and normal blood pressure values (110/65 mmHg).

INITIAL LABORATORY INVESTIGATIONS

Initial laboratory testing revealed an elevated hs-cTnI level of 560 ng/L, with a CK-MB level of less than 1 ng/mL. D-dimer was markedly raised at 3,279 ng/L, and NT-proBNP was elevated at 7,774 pg/mL, supporting the suspicion of heart failure with reduced ejection fraction (HFrEF).

IMAGING STUDIES

TTE demonstrated significant dilation of all cardiac chambers, with severe hypokinesia of the apical segments and a LVEF of 25%. Notably, an echogenic mass was identified in the apex of the left ventricle, consistent with thrombus formation. These findings were further confirmed by chest CT angiography, which provided clearer delineation of the left ventricular thrombus (Figure 1).

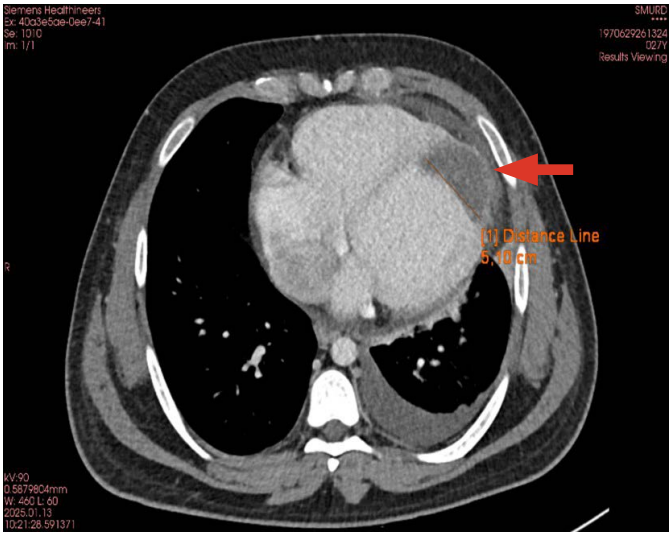

FIGURE 1. Cardiac computed tomography imaging showing a large thrombus at the left ventricular apex (arrow).

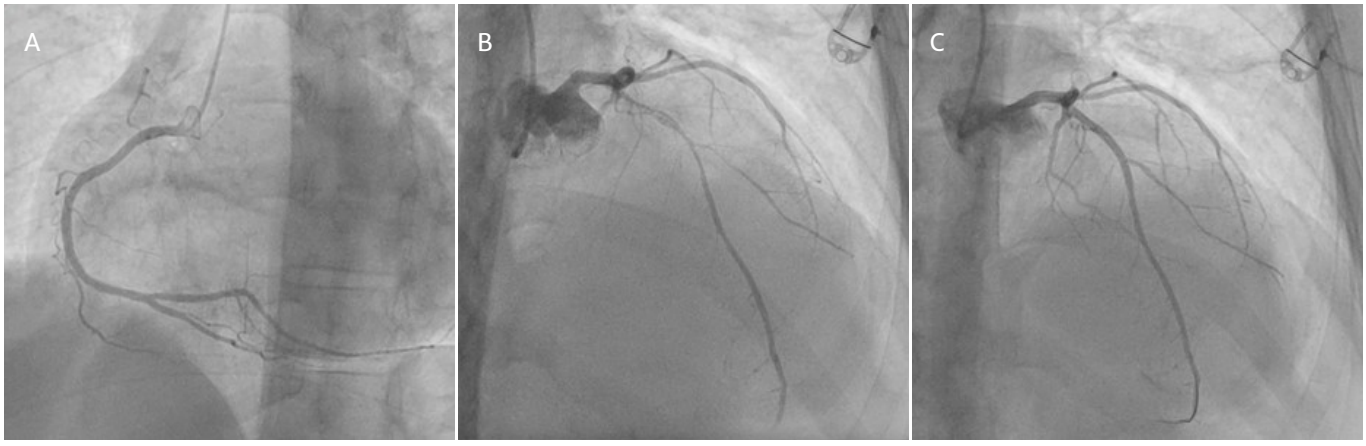

FIGURE 2. Coronary angiography. **A.** Right coronary artery. **B.** Left coronary artery showing subocclusive stenosis in the left anterior descending artery. **C.** Left coronary artery showing the result of the percutaneous coronary intervention.

INITIAL MANAGEMENT

The initial management involved hemodynamic and respiratory support and intravenous diuretics. Anticoagulation was initiated with unfractionated heparin and later transitioned to acenocoumarin with a stable INR value between 2 and 3. Guideline-directed medical therapy for HFrEF was commenced, including sacubitril/valsartan at 49/51 mg twice daily, metoprolol 50 mg twice daily, spironolactone 25 mg twice daily, dapagliflozin 10 mg once daily, and furosemide 40 mg twice daily.

CORONARY ANGIOGRAPHY

To identify the underlying cause of the intracavitary thrombosis, coronary angiography was conducted, which showed a subocclusive stenosis in the left anterior descending artery caused by a partially recanalized thrombus. This lesion was managed with percutaneous angioplasty followed by the placement of a drug-eluting stent. Cardiac optical coherence tomography revealed an extended thrombotic load in segments I/II, involving the origin of the first diagonal branch (D1), with features suggestive of recanalization, preceded by a fibrous plaque without signs of vulnerability. Additionally, after the coronary angiography, antiplatelet therapy with Aspirin 75 mg daily and Clopidogrel 75 mg daily, as well as Rosuvastatin 40 mg daily, was initiated.

THROMBOPHILIA TESTING

In light of the patient's young age and atypical presentation, a thrombophilia workup was performed. This revealed a homozygous mutation in MTHFR A1298C and heterozygous mutations in Factor XIII and plasminogen activator inhibitor-1 (PAI-1). The endothelial protein C receptor (EPCR) genotype was A1/A1, which is associated with a prothrombotic state.

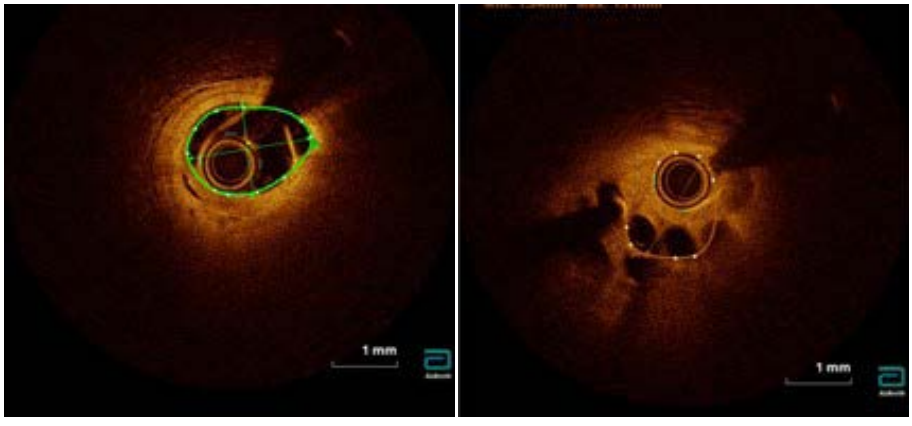

FIGURE 3. Cardiac optical coherence tomography demonstrating extended thrombotic load with recanalization features and adjacent fibrous plaque without vulnerability signs.

ADDITIONAL FINDINGS

Additional findings included a normal eosinophil count and positive IgG for cytomegalovirus (CMV), suggesting past infection. Endomyocardial biopsy excluded active myocarditis.

CLINICAL OUTCOME

Following stabilization, the patient was discharged on anticoagulation and a comprehensive regimen for heart failure. At the 1-month follow-up, TTE revealed a significant reduction in thrombus size, with approximately two-thirds of the original mass resolved. Clinically, the patient remained asymptomatic and free of any signs of congestion.

DISCUSSION

This case highlights the multiple underlying causes of left ventricular thrombus formation in the setting of DCM and severe LV systolic dysfunction in a young patient. The patient presented a reduced LVEF and wall motion abnormalities, which created a prothrombotic environment due to blood stasis and endothelial injury.[7] Additionally, the presence of inherited thrombophilia mutations (MTHFR A1298C homozygous, Factor XIII and PAI-1 heterozygous, EPCR A1/A1 genotype) likely further increased the risk of intracardiac thrombosis by promoting hypercoagulability.[12]

The increasing prevalence of heart failure has shifted clinical focus to thrombus formation in DCM and other cardiomyopathies, where a left ventricular thrombus remains an important cause of thromboembolic complications such as stroke and systemic embolism.[8] Despite this, standardized treatment recommendations for left ventricular thrombi in DCM are lacking, particularly regarding anticoagulation regimens and the role of direct oral anticoagulants.[8]

In this patient, prompt anticoagulation with unfractionated heparin transitioned to acenocoumarin, alongside comprehensive guideline-directed medical therapy for heart failure, resulted in a significant reduction in thrombus size in 1 month. This underscores the importance of early recognition and management. Moreover, coronary angiography revealed subocclusive stenosis due to a partially recanalized thrombus in the left anterior descendent artery, a very uncommon finding considering the patient's age, which was successfully treated with percutaneous coronary intervention. Coronary imaging, ranging from noninvasive procedures such as coronary CT angiography for risk assessment[13] to invasive techniques, has a crucial role even in young patients, as highlighted by the present case.

For follow-up, serial TTE remains essential to monitor thrombus resolution and cardiac function, as well as to guide the duration of anticoagulation therapy. Furthermore, given the patient's thrombophilia profile, long-term hematology follow-up and consideration of extended anticoagulation may be recommended.

The integration of genetic and thrombophilia screening in younger patients with unexplained intracardiac thrombosis could lead to an effective personalized treatment.

CONCLUSION

Left ventricular thrombus in the context of DCM poses a significant clinical challenge due to its multifactorial etiology, which includes impaired ventricular function, blood stasis, and hypercoagulability. This case emphasizes the importance of a thorough diagnostic approach, incorporating advanced imaging techniques and thrombophilia screening, particularly in young patients, to inform effective management strategies. Identifying underlying ischemic factors, even in younger individuals, is critical and can be life-saving. Initiating anticoagulation early, alongside optimized heart failure treatment, can result in substantial thrombus resolution and enhance patient outcomes. A multidisciplinary approach that considers individual risk profiles, along with collaboration among cardiology, hematology, and genetics specialists, is essential for improving prognosis and preventing thromboembolic events in this vulnerable group.

CONFLICT OF INTEREST

Nothing to declare.

ETHICS APPROVAL

The study was conducted in accordance with the guidelines of the Declaration of Helsinki and received ethical approval from the Ethics Committee of the George Emil Palade University of Medicine, Pharmacy, Science and Technology of Târgu Mureș (approval no. 351/12 December 2017).

CONSENT TO PARTICIPATE

The patient provided written informed consent regarding the publication of this case.

FUNDING

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AUTHOR CONTRIBUTIONS

C.G. and I.P.R. led the conceptualization of the study. C.G. and I.H. developed the methodology. D.P. conducted the validation. C.G., I.H., and D.P. performed formal analysis and investigation. C.G. wrote the original draft. I.P.R. reviewed and edited the manuscript. I.H. prepared the visualizations. I.P.R. provided supervision. C.G. handled project administration. All authors have read and agreed to the published version of the manuscript.

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