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Case Report

Working Backwards: Splenic Infarcts from Left Ventricular Thrombus

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INTRODUCTION

Splenic infarction is a rare but significant complication often linked to embolic events, with an incidence of 8.9 per 100,000 person-years.[1,2] It is frequently overlooked as a cause of abdominal pain, though improved imaging modalities now aid in its recognition, even as up to 30% of splenic infarcts remain asymptomatic.[3] A primary source of emboli is thrombus formation within the left ventricle (LV) in patients with heart failure, particularly heart failure with reduced ejection fraction (HFrEF). HFrEF, defined by a left ventricular ejection fraction (LVEF) ≤ 40%, predisposes patients to intracardiac thrombi formation due to blood stasis.[4] Here, we present a rare case of splenic infarction secondary to a LV thrombus in a patient with HFrEF.

CASE REPORT

A 59-year-old male with a history of HFrEF presented to the emergency department with sudden, sharp left upper quadrant abdominal pain. Diagnosed with heart failure 12 years prior, his condition had been managed with medications, though his LVEF remained at 30-35%. Four days before presentation, he had a fall, resulting in a bruise and pain unresponsive to pain medications.

On arrival, the patient's vital signs were stable except for elevated blood pressure. Physical examination revealed tenderness in the left upper quadrant without signs of an acute abdomen. Cardiovascular examination showed signs consistent with chronic heart failure, including a displaced apical impulse and a third heart sound (S3).

Laboratory results were unremarkable. An electrocardiogram showed normal sinus rhythm, and a chest X-ray indicated cardiomegaly without pulmonary congestion. Given his history, an echocardiogram was performed, revealing severe LV dysfunction and a large, mobile thrombus measuring 2.2 cm within the LV (Figure 1), with a new LVEF of 20-25%. An abdominal computerized tomography (CT) scan (Figure 2) confirmed splenic infarction, identifying two regions of infarction in the spleen and occlusion of the superior mesenteric artery (SMA) with distal reconstitution, likely due to embolization from the LV thrombus. The chronic nature of the SMA occlusion was suggested by reconstituted flow beyond the occlusion.

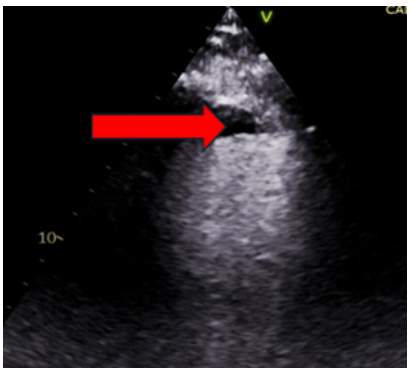

Figure 1. Left ventricular (LV) apical thrombus.

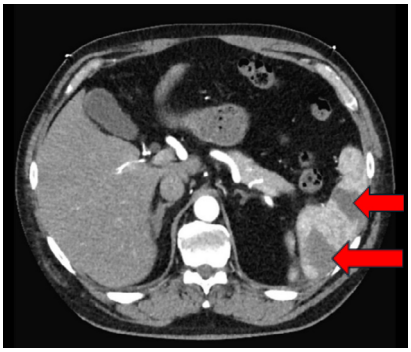

Figure 2. Splenic infracts on abdominal computerized tomography scan.

The patient was diagnosed with splenic infarction secondary to embolization from a LV thrombus, a complication of HFrEF. He was initiated on anticoagulation therapy with intravenous heparin, later transitioning to apixaban. His heart failure management also was optimized, including adjustments to his beta-blocker dose and the addition of spironolactone.

Additional workup for hypercoagulability and splenic infarction included: lactate dehydrogenase (LDH) <140 U/L (indicating no excessive cell turnover), erythrocyte sedimentation rate (ESR) 31 mm/hr (slightly elevated), anticardiolipin antibodies (IgG, IgM, IgA) <2 (arguing against anti-phospholipid syndrome), protein C activity 160%, protein S activity 97% (indicating absence of hereditary hypercoagulable states), ANA negative (arguing against autoimmune disease), QuantiFERON negative (indicating no tuberculosis), and *Histoplasma* antigen negative (indicating no histoplasmosis).

The patient's symptoms gradually improved, and follow-up imaging showed partial resolution of the LV thrombus. He was discharged on long-term anticoagulation therapy to reduce future thromboembolic risk, with ongoing monitoring of heart function and the thrombus.

This case underscores the risk of embolic complications in patients with HFrEF, especially with LV thrombi. Splenic infarction, though rare, should be considered in HFrEF patients with acute abdominal pain. Early imaging diagnosis and prompt anticoagulation initiation are crucial to prevent complications and improve patient outcomes.

DISCUSSION

Splenic infarction, a rare cause of abdominal pain, is more likely to occur in patients with splenomegaly, often due to hematologic malignancies. In fact, between 50-72% of patients with chronic myelogenous leukemia or myelofibrosis may experience splenic infarction.[5] Cardioembolic phenomena, such as those from atrial fibrillation, valvular disorders, and atrial septal defects (ASDs), also increase the risk of

infarcts. In immunocompromised patients, septic emboli should be considered, and up to 40% of patients may be asymptomatic.[6] Less common causes include autoimmune diseases, connective tissue diseases, surgical complications, post-transplant conditions (pancreas and liver), or infections like Brucella.[7] Clinically, splenic infarction usually presents as left upper quadrant pain, often accompanied by fever, nausea, or vomiting.[8]

A thorough investigation is essential to determine the cause of splenic infarction. Initial workup should evaluate splenic size and potential hematologic causes, including malignancies. While no specific laboratory tests confirm splenic infarction, up to 50% of patients may show leukocytosis with a white blood cell count >12,000/mm.[3,9] Although a study has linked elevated D-dimer levels to splenic infarcts,[6] this association has not been consistently validated. CT scans are the preferred imaging modality in acute cases, typically revealing a characteristic pyramidal wedge shape.[10] For more established infarcts, ultrasound can reveal a "bright band sign" (highly hyperechoic linear bands within lesions of varying ages).[11] However, ultrasound is limited in diagnostic value due to challenges in visualizing splenic parenchyma and high inter-operator variability, with only 18% sensitivity, though color Doppler may enhance this by detecting areas without blood flow.[12,13]

Cardioembolic causes, including atrial fibrillation and valvular vegetations, should be evaluated via electrocardiogram and echocardiogram. In the absence of arrhythmias like atrial fibrillation, LV thrombus formation due to reduced ejection fraction (EF) occurs at an annual rate of 0.9 to 5.5%.[13] While reduced EF can result in LV thrombus,[14,15] it has not been clearly linked to splenic infarction, and management protocols are not well-defined. Current guidelines do not recommend prophylactic anticoagulation for heart failure with reduced EF, as indicated in the COMMANDER HF trial.[16]

This case highlights the importance of a comprehensive workup in patients with splenic infarction and underscores the need for further evidence to clarify any potential association between HFrEF and splenic infarction.

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