SILENT INFILTRATION, SUDDEN OCCLUSION: LV THROMBUS EMBOLIZATION TO THE LAD

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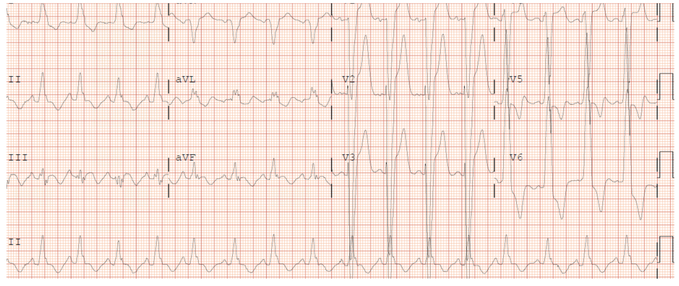
BACKGROUND

Infiltrative cardiomyopathies are characterized by the accumulation of normal substances within the heart muscles, potentially leading to both systolic and diastolic dysfunction. The formation of a left ventricular thrombus is a recognized complication in various types of cardiomyopathies, encompassing both ischemic and non-ischemic origin. The presence of LVT significantly increases the risk of stroke or systemic embolism but can also embolize to coronary arteries leading to ACS. We present a case of a patient with emboli zed thrombus from LV to the LAD artery and presentation as ACS. Later found to have systolic dysfunction and ground glass infiltrates in the LV raising suspicion for infiltrative disease.

CASE PRESENTATION

A 61 year old female with past medical history of HTN, COPD, a lung abscess treated with VATS decortication, tobacco use presented with sudden onset shortness of breath. Her troponin were noted to be elevated at 5000 ng/L, rising to 6500 ng/L, proBNP >35000 pg/mL. EKG revealed sinus rhythm with severe left ventricular hypertrophy, LBBB, concurrent ST segment changes. POCUS demonstrated severe left ventricular dysfunction, left ventricular hypertrophy, trivial pericardial effusion.



EKG

INVESTIGATIONS

Echo revealed LVEF <10%, elevated E/e' ratio consistent with elevated left atrial pressures, a ground-glass appearance of left ventricular wall raising concerns for infiltrative heart disease, prominent trabeculations in the lateral and inferior posterior walls, moderate concentric LVH, moderate left ventricular cavity enlargement, moderate mitral regurgitation, small pericardial effusions. Cardiac catheterization identified a 100% distal lesion in the LAD, which was thrombotic in nature, PTCA was performed followed by manual aspiration thrombectomy with removal of a large clot. Post-thrombectomy angiography showed no evidence of ruptured plague or residual coronary artery disease, but elevated left and right sided filling pressures and severely reduced left ventricular systolic function. The Fick cardiac index was 2.07L/min/m2.

When the heart sheds a clot, coronaries can pay the price.

Severe LV dysfunction especially from infiltrative disease — can generate ventricular thrombi that embolize to coronary arteries —mimicking ACS and demanding revascularization and anticoagulation.

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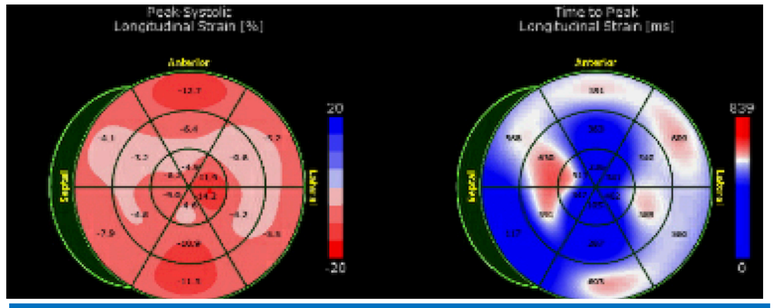
MANAGEMENT

Given the concern that LAD thrombus may have originated from LV thrombus due to severely reduced ejection fraction, anticoagulation with warfarin was initiated, targeting INR of 2-3 with bridging with low molecular weight heparin. A repeat limited echo showed severe global hypokinesis with LVEF of less than 20%, a speckled pattern of interventricular septum, findings suggestive of amyloidosis. Serum free light chain analysis revealed elevated free kappa light chains at 51.2 mg/L and free lambda light chains at 37.1 mg/L, with a kappa/lambda ratio of 1.38. Due to concerns of infiltrative cardiomyopathy on echocardiogram, further evaluation with MRI was recommended to assess for amyloidosis or other infiltrative diseases.

DISCUSSION

LV thrombosis is a known complication of severe systolic dysfunction of the left ventricle. The development of LV thrombus is attributed to endothelial injury, a prothrombotic state and blood stasis resulting from conditions like myocardial infarction, chronic heart failure and various cardiomyopathies. LV apical thrombi is commonly linked to reduced ejection fraction (below 35%) and the presence of apical aneurysm. They carry a risk for systemic embolization, in some cases, they may embolize into the coronary arteries, causing blockages and leading to secondary MI. Coronary angiography remain a key diagnostic tool, often revealed abrupt blockages in the small, distal coronary vessels, typically without atherosclerosis. In patients presenting with myocardial infarction and non-obstructing coronary arteries (MINOCA), intracoronary imaging is essential to rule out plaque rupture or erosion. The initial management of coronary embolism aligns with treatment protocols for ACS until atherosclerosis is excluded. Thrombectomy may be needed depending upon thrombus burden. Anticoagulation remains the main treatment modality.



CONCLUSION

LV thrombus is a significant complication of both ischemic and non ischemic cardiomyopathy especially with reduced ejection fraction and apical wall motion abnormalities. These thrombi can embolize including into coronary arteries, early identification and initiation of anticoagulation are essential to prevent secondary MI or systemic embolism. In cases of MINOCA, coronary embolism should be considered.

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