Silent Infiltration, Sudden Occlusion: LV Thrombus embolization to the LAD

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Abstract

Infiltrative cardiomyopathies are characterized by the accumulation of abnormal substances within the heart muscle, potentially leading to both systolic and diastolic dysfunction. These conditions can progress to heart failure and, if left untreated, may result in premature death. Common forms of infiltrative cardiomyopathy include cardiac amyloidosis, cardiac sarcoidosis, and Anderson-Fabry disease (1).

The formation of a left ventricular thrombus (LVT) is a recognized complication in various types of cardiomyopathies, encompassing both ischemic and non-ischemic origins. LVT is frequently associated with a reduced left ventricular ejection fraction (LVEF) and can also occur in non-ischemic cardiomyopathies, including infiltrative forms (2).

According to Virchow's triad, the development of LVT involves endothelial injury, blood stasis, and a hypercoagulable state. The presence of LVT significantly increases the risk of stroke or systemic embolism, thereby elevating morbidity and mortality in patients with both ischemic and non-ischemic cardiomyopathies. LVT thrombus are at risk of systemic embolization but can also embolize to coronary arteries leading to acute coronary syndrome.

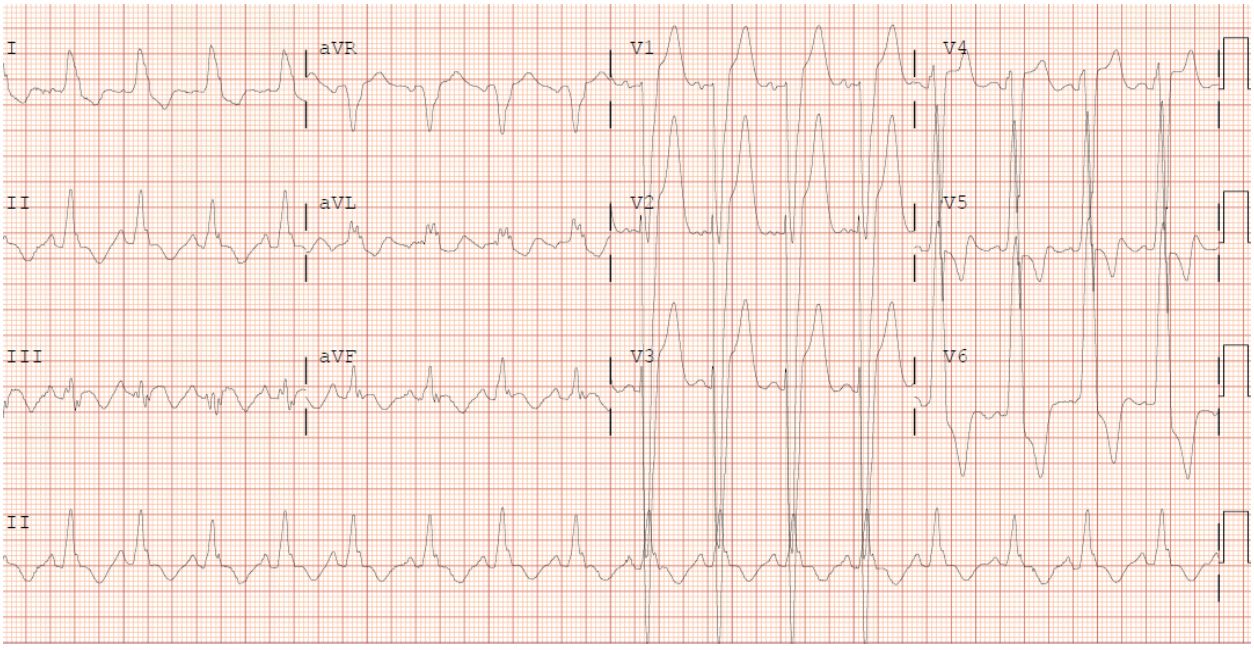
We present the case of a patient with a thrombus from the left ventricle embolized into the LAD artery, causing a 100% occlusion presenting as NSTEMI. Found to have severe systolic dysfunction and ground glass infiltrates in LV raising suspicion for infiltrative disease.

Case Presentation

A 61-year-old woman with a medical history including hypertension, chronic obstructive pulmonary disease (COPD), a lung abscess complicated by empyema treated with video-assisted thoracoscopic surgery (VATS) decortication, and tobacco use presented to our facility with sudden-onset shortness of breath. Emergency medical services (EMS) noted her blood pressure was in the 200s systolic, and her oxygen saturation was in the 80s. She was administered sublingual nitroglycerin and placed on a non-rebreather mask.

Upon arrival in the emergency department (ED), she received additional nitroglycerin and intravenous labetalol, resulting in a reduction of her blood pressure to the 160s systolic. Her troponin levels were significantly elevated at 5000 ng/L, rising to 6500 ng/L, and her pro-brain natriuretic peptide (proBNP) was greater than 35,000 pg/mL. Electrocardiogram (EKG) findings included sinus rhythm with severe left

ventricular hypertrophy, left bundle branch block, and concurrent ST-segment changes. Chest X-ray revealed vascular congestion, prompting the administration of furosemide (Lasix).



Point-of-care ultrasound (POCUS) demonstrated severe left ventricular dysfunction, left ventricular hypertrophy, trivial pericardial effusion, and no gross wall motion abnormalities. She was initiated on a heparin drip, aspirin, and a statin.

A subsequent echocardiogram revealed a left ventricular ejection fraction (LVEF) of less than 10%, elevated E/E' ratio consistent with elevated left atrial pressures, a ground-glass appearance of the left ventricular wall raising concern for infiltrative heart disease, prominent trabeculations in the lateral and inferior posterior walls, moderate concentric left ventricular hypertrophy, moderate left ventricular cavity enlargement, moderate mitral regurgitation, and small pericardial effusions.

Cardiac catheterization identified a 100% distal lesion in the left anterior descending artery (LAD), which was thrombotic in nature. Percutaneous transluminal coronary angioplasty (PTCA) was performed, followed by manual aspiration thrombectomy with removal of a large clot. Post-thrombectomy angiography showed no evidence of ruptured plaque or residual obstructive coronary artery disease, but elevated left and right-sided filling pressures and severely reduced left ventricular systolic function persisted. The Fick cardiac index was measured at 2.07 L/min/m².

Given the concern that the LAD thrombus may have originated from a left ventricular thrombus due to the severely reduced ejection fraction, anticoagulation with warfarin was initiated, targeting an international normalized ratio (INR) of 2–3, and bridged with low-molecular-weight heparin (Lovenox). Dual antiplatelet therapy (DAPT) continued with aspirin for 14 days and clopidogrel (Plavix) for one year, following interventional cardiology recommendations.

A repeat limited echocardiogram showed severe global hypokinesis with an LVEF of less than 20%, a speckled pattern of the interventricular septum, and 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. Immunofixation did not reveal any monoclonal proteins.

Due to the echocardiographic findings concerning infiltrative cardiomyopathy, further evaluation with cardiac magnetic resonance imaging (MRI) was recommended to assess for amyloidosis or other infiltrative diseases.

Discussion

Left ventricular (LV) thrombus is a known complication of severe systolic dysfunction of the left ventricle. It occurs in approximately 15% of cases following ST-elevation myocardial infarction (STEMI), whereas in non-ischemic cardiomyopathy, its occurrence ranges between 2% and 36% (2). The development of LV thrombus is attributed to endothelial injury, a prothrombotic state, and blood stasis resulting from conditions such as myocardial infarction, chronic heart failure, and various cardiomyopathies (3). LV apical thrombus is commonly linked to reduced ejection fraction (EF below 35%) and the presence of apical aneurysms (4).

LV apical thrombi carry a risk of systemic embolization, and in some cases, they may embolize into the coronary arteries, causing blockages and leading to secondary myocardial infarctions. Coronary embolism, though rare, is responsible for about 3% of myocardial infarctions and often goes unrecognized (5). It can result from dislodged thrombi from the left heart chambers or from paradoxical embolization via a patent foramen ovale, allowing venous thrombi to reach the arterial system (6).

Coronary angiography remains the key diagnostic tool, often revealing abrupt blockages in the small, distal coronary vessels, typically without underlying atherosclerosis (5). In patients presenting with myocardial infarction and non-obstructive 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 acute coronary syndrome (ACS) until atherosclerotic causes are excluded. Thrombectomy may be indicated in cases with significant thrombus burden. Anticoagulation therapy is the cornerstone of treatment for coronary embolism (6).

LV thrombus tends to form in areas of the ventricle with akinesis or dyskinesis, due to impaired blood flow (7). Patients with systolic dysfunction and low EF, even from non-ischemic cardiomyopathies (such as infiltrative types), are at increased risk of developing LV thrombus, which may embolize and mimic ACS. If infiltrative (nonischemic) cardiomyopathy is suspected, additional diagnostic evaluation—such as cardiac MRI—is required to help determine the underlying cause.

Upon diagnosis of LV thrombus, prompt initiation of anticoagulation is recommended. Warfarin (Coumadin) is the preferred agent due to extensive clinical use, although its use is limited by the need for frequent INR monitoring, drug interactions, and difficulty maintaining therapeutic levels. The target INR is 2.0–3.0, which remains the standard (7).

The optimal duration of anticoagulation is not well-established, but a minimum of 3 months is advised, with follow-up imaging to assess thrombus resolution. If repeat echocardiograms show no evidence of thrombus, oral anticoagulation can be stopped, and dual antiplatelet therapy (DAPT) may be continued for up to one year. The exact duration of anticoagulation should be individualized based on clinical context (7).

Conclusion

Left ventricular thrombus is a significant and sometimes overlooked complication of both ischemic and non-ischemic cardiomyopathies, especially in patients with reduced ejection fraction and apical wall motion abnormalities. Because these thrombi can embolize, including coronary arteries, early identification and initiation of anticoagulation are critical to prevent serious outcomes such as systemic embolism or secondary myocardial infarction. In cases of myocardial infarction without obstructive coronary disease, coronary embolism should be considered—particularly when LV thrombus is present. Comprehensive evaluation, including cardiac MRI when infiltrative cardiomyopathy is suspected, is important to determine the underlying cause and guide treatment. Anticoagulation is the mainstay of therapy, with the duration of treatment based on follow-up imaging and the patient's overall risk profile. Early recognition and a tailored treatment approach are key to improving clinical outcomes in these patients.

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