

Left Ventricle Pedunculated Thrombi Risks and Outcomes: A Case Report and Systematic Review

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May 30, 2023

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Left Ventricle Pedunculated Thrombi Risks and Outcomes: A Case Report and Systematic Review

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The authors received no funding.

The authors declare no conflict of interest.

Abstract

We report a case of a 42-year-old male with cardiomyopathy and acute bilateral femoral artery embolization. Following bilateral femoral artery embolectomy and fasciotomy, transthoracic echocardiography revealed a pedunculated highly mobile left ventricle (LV) thrombus. Surgical removal of the thrombi was not recommended for the procedural risk; consequently, anticoagulation therapy was recommenced. Unfortunately for the patient, the bleeding risk impeded the continuation of anticoagulation, which resulted in an increase in the thrombus size. The patient shortly developed multiorgan failure and possibly disseminated intravascular

coagulopathy (DIC) and died. Besides this case, we have systematically reviewed the PubMed and Scopus databases for all the previously reported pedunculated thrombus/ thrombi cases.

1. **Introduction** A left ventricle (LV) thrombus is defined as an echo-dense mass near an akinetic or hypokinetic ventricular wall that is visible in at least two different views . LV thrombi formation following acute myocardial infarction (MI) or dilated cardiomyopathy (DCM) is predisposed by Virchow's triad (myocardial infarction-induced endothelial injury and the subsequent elevation of catecholamine levels , inflammation-triggered hypercoagulability, and blood stasis due to segmental wall motion). About 6.3% of ST segment elevation myocardial infarction (STEMI) cases and 19.2% of anterior STEMI patients with LVEF <50% are complicated with LV thrombus formation within two weeks to three months of the onset of myocardial injury. Other risk factors of LV thrombus formation are dilated heart failure, hypercoagulable states, nonischemic cardiomyopathy, and Takotsubo cardiomyopathy . LV thrombi develop in 1.3% to 2.2% of patients with acute Takotsubo cardiomyopathy, and they are significantly associated with the presence of both apical ballooning and high troponin level >10 ng/mL. Pedunculated LV thrombi have a higher embolic potential than mural ones depending on the extent of protrusion into the left ventricle, mobility, and the pedunculated shape. There is no theory explaining the exact mechanism or combination of factors that favor the formation of pedunculated thrombi. Nonetheless, the literature has reported many mural thrombi that partially detached and transformed into pedunculated thrombi during follow-up or hospitalization . Herein, we depict a case of pedunculated LV thrombus that presented with bilateral acute lower limb ischemia and review the literature for similar case presentations highlighting the main risks and outcomes.
2. **Methodology** We searched PubMed using the key search terms “left ventricle OR left ventricular” AND pedunculated AND “thrombus OR thrombi”. All published reports posing cases of pedunculated left ventricular thrombus were included with no restriction on the age or year of publication.
3. **Results**
4. **Presentation of Case**

A man in his 40s with a history of diabetes mellitus, hypertension, and ischemic heart disease with resultant ischemic heart failure presented with bilateral lower limb pain and loss of motor and sensory activity. The patient was evaluated in the emergency department and the evaluation revealed acute bilateral lower limb ischemia that prompted an immediate surgical intervention.

Five years before the current presentation, the patient reportedly had extensive anterior ST elevation myocardial infarction(*Figure I*) that was treated with streptokinase and rescue PCI . Coronary angiography showed 80% stenosis of the proximal LAD, for which a drug-eluting stent was deployed. The patient's transthoracic echocardiography (TTE) showed an ejection fraction (EF) of 45% with an akinetic apical and mid septum, apical and mid anterior, and apical inferior segments; normal LV dimensions, and dilated left atrium. Over four years, the patient had deteriorating heart failure with EF 30%, akinetic anteroseptal and mid to apical septal segments, a restrictive pattern of diastolic function, dilated LV and left atrium, and mild mitral regurgitation.

On examination, the vital parameters recorded systolic blood pressure of 90 mm hg, heart rate of 89/ min, respiratory rate of 18/ min, and temperature of 36. Laboratory investigations were notable for leucocytosis (15.300 cells/ microliter), elevated cardiac enzymes (CK-MB 130 U/L; troponin I 71.6-fold the upper normal limit), and an international normalized ratio (INR) of 1. Other laboratory test results (hemoglobin, platelets, serum creatinine, serum urea, arterial blood gas) were normal. Arterial duplex revealed bilaterally damped monophasic flow across the external iliac artery down to the superficial femoral artery. Furthermore, there was no detectable flow distally down to the infra-popliteal arteries. The patient's acute limb ischemia was managed surgically by bilateral mechanical thrombectomy and fasciotomy.

A TTE was obtained after the operation and elucidated decreased ejection fraction (20%, measured by M-mode) with global hypokinesia and a large pedunculated irregular hypermobile LV thrombus at the LV apex measuring 4.32*2.82 cm (figure II). An electrocardiogram revealed LBBB and prolonged QT interval.

Cardiothoracic surgery consultation refused the surgery for the procedural risks; for this, anticoagulation treatment was decided. Soon after the operation, the patient's condition deteriorated and required endotracheal intubation. The deterioration entailed circulatory collapse and multiorgan failure (acute kidney injury, ischemic/ shocked liver, and disturbed consciousness level). Furthermore, the patient exhibited a bleeding tendency (hematemesis, melena, and nasal bleeding), which necessitated discontinuing the anticoagulation; consequently, the thrombus size increased to measure 4.7*3.9 cm (Figure III). The laboratory test results were then notable for low hemoglobin (7.4 g/dl), low platelets (87×10^3 /UL), elevated renal function test values (serum creatinine 6 mg/dl; urea 267 mg/dl); elevated liver function test values (AST 1145; ALT 1063), and elevated serum potassium (6.5 mmol/dl). The patient eventually died.

Systematic review of literature

Searching PubMed and Scopus databases retrieved 74 and 63 articles, respectively. After removing duplicates, 90 records were eligible for the title and abstract screening, which revealed 66 reports to be eligible for full-text screening. Only cases reporting pedunculated thrombus/ thrombi at the first assessment for any etiology were included; records with irretrievable data were excluded; reports of mural thrombus/ thrombi that transformed into pedunculated thrombi during the follow-up were also excluded for carrying different risks. Only 45 cases from 37 papers met our inclusion and exclusion criteria. The manual search revealed 12 more pertinent cases. Our report was included in the analysis (figure IV)/ flowchart) to eventually have 57 cases analyzed.

Baseline demographic and clinical data, management, and outcome are listed in Table I. Overall, the mean age of all the cases was 50.5 ± 15.6 years, and 66.6% (38/57) were male. Based on etiology, 41.1% of cases (N=24) were attributed to old (N=12) or acute (N=12) myocardial infarction; 17.5% (N=10) to cardiomyopathy (non-ischemic dilated cardiomyopathy, Takotsubo cardiomyopathy, peripartum or idiopathic cardiomyopathy); and 7% (N=4) were due to heart failure. Other etiologies entailed ulcerative colitis (N=2), CoVID-19 infection (N=2), and coagulopathy (N=6). About 17.5% (N=10) developed idiopathic LV thrombi. Combined etiologies were also found .

Twelve (21%) of the cases had a previous coronary artery disease, 11 (19.2%) had hypertension, seven (12.2%) had a smoking history, and three (5.1%) had a history of substance abuse (alcohol, anabolic androgenic steroids, and cocaine). Furthermore, seven (12.2%) had diabetes mellitus, five (8.7%) had coagulopathy (essential thrombocytopenia, polycythemia vera, and cystic fibrosis for example), and four (7%) had hyperlipidemia. Of note, seven cases (12.2%) had no identifiable risk factors. The vast majority of LV thrombi were located at the apex (73.6%; 42/57) followed by the interventricular septum (8.7%; 5/57). However, some cases exhibited more than one thrombus at two or more sites and three cases did not report the thrombus site (table II). About 16 (28%) of cases developed LV thrombus despite having a normal systolic function on presentation. The etiology of the LV thrombus was idiopathic in 10/ 16 of these cases, due to a hypercoagulable state in two cases, and due to ulcerative colitis inflammatory condition in two cases. It is worth stating that hypertension was a risk factor in 4/16 (25%), and the ECG showed abnormal changes in only 3/16 (18.7%) of these cases. Thirty-three patients (57.8%) developed distal emboli at different sites, yet the commonest site was the brain (N=14) followed by the arterial system of the lower limb (N=13). Other sites are displayed in (Table I). The definitive treatment for most cases was surgical removal (75.4%; N=43). Furthermore, three cases responded well to oral anticoagulation with warfarin. While five cases responded well to heparin infusion, four cases did not show any improvement, and the management plan was readjusted to either surgery, tirofiban, recombinant tissue plasminogen activator (RTPA), or streptokinase.

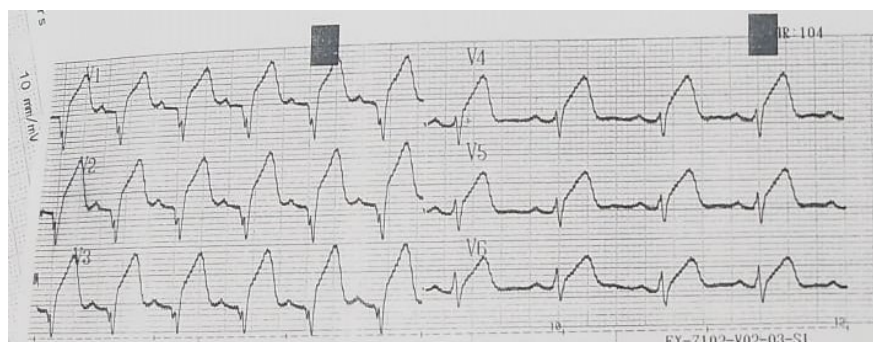
Discussion

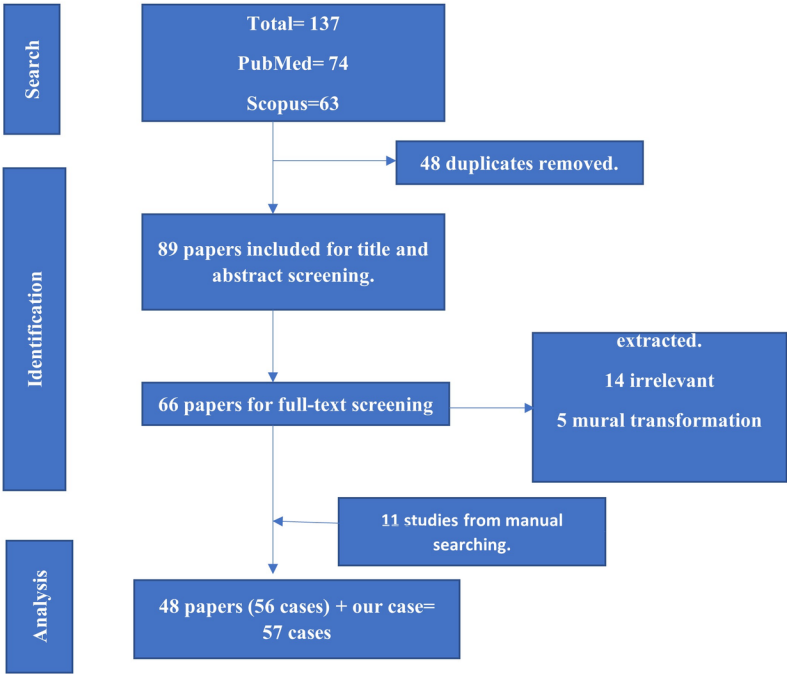
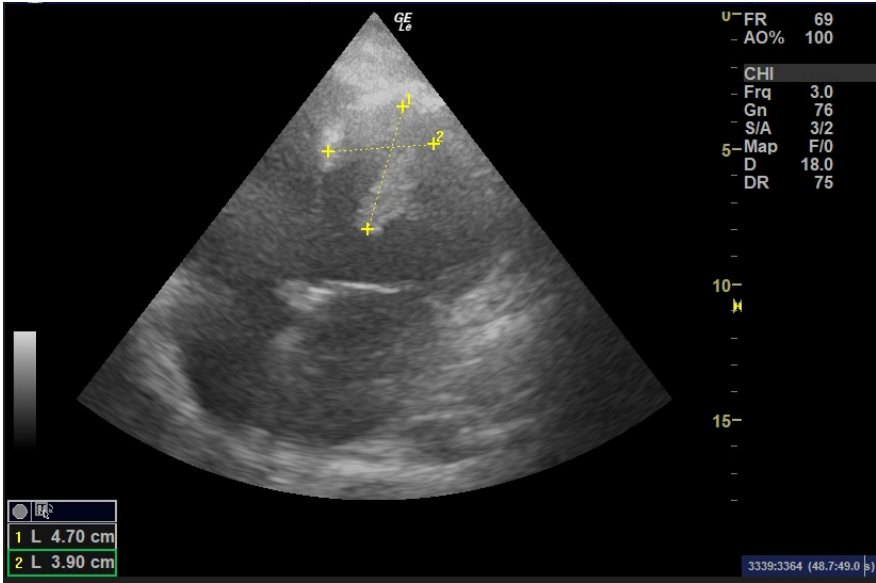
Despite the absence of histopathological confirmation, our patient was diagnosed with LV thrombus for his associating factors (history of anterior STEMI with reduced EF) and the current presentation. Surgical removal is the definitive management of the mobile pedunculated masses for their high embolization risk . Alternatively, for cases that refuse or deem to be unfit for surgery, direct oral anticoagulants were non-inferior to Vit K antagonist (warfarin) in treating LV thrombus . Based on our review, surgical removal was almost

always successful, whereas oral and intravenous anticoagulation was relatively less successful (5/12; 41.6% failure rate). Furthermore, the three cases that died, entailing our case, were managed using anticoagulation and did not proceed to surgery. There are no clinical trials assessing the efficacy of thrombolysis via streptokinase, RTPA, or urokinase. Nonetheless, thrombolysis carries a high embolic and hemorrhagic risk despite the potential of successfully dissolving the LV thrombus. The deterioration of the LVEF in our patient can be explained by the thrombus given the fact that the LVEF improves or even normalizes after the removal or dissolution of LV thrombus in some other patients. This finding has been confirmed earlier where the LAD/ anterior wall infarctions were significantly associated with contractile dysfunction at the apex and with decrements in the peak systolic function. Four-dimensional magnetic resonance imaging (4D MRI) for intracardiac hemodynamic assessment was tested in anterior MI cases, being the most reported risk for LV thrombi. It revealed a reduced peak systolic flow in the mid ventricle and apex and reduced peak diastolic flow in the apex in anterior acute MI. This explains the occurrence of apical thrombi in anterior/ LAD MI. However, there is no reported prophylactic anticoagulation strategy to date. Accordingly, any use of prophylactic anticoagulation should be tailored on a patient-by-patient basis. Notably, prophylactic anticoagulation in LV thrombus was a class IIb recommendation according to the 2013 ACC/AHA STEMI guidelines. Low-dose anticoagulation with rivaroxaban 2.5 mg BID for 30 days, besides the dual antiplatelet therapy (DAPT), has been recently tested. The low-dose rivaroxaban plus DAPT cohort had a lower incidence of LV thrombus formation as opposed to the DAPT alone cohort (0.7% and 8.6%, respectively; hazard ratio 0.08). On the other hand, anticoagulation has no role in preventing LV thrombus formation in dilated cardiomyopathy with sinus rhythm. The Heart Failure Long-Term Antithrombotic Study (HELAS) trial has also compared the incidence of thromboembolism with warfarin, aspirin, or placebo in chronic heart failure. There was no significant difference in the incidence among the three groups. TTE has been the major diagnostic tool for the literature cases, entailing our case. Noting the difficulty of diagnosing mural thrombi, and their potential of transformation into pedicled thrombi, routine assessment of patients following MI (particularly anterior MI with reduced LVEF) or cardiomyopathy diagnosis is advised for early diagnosis and management. Delayed enhancement cardiac magnetic resonance imaging (DE-CMRI) was revealed to be the most sensitive imaging modality for detecting LV thrombi and distinguishing them from the normal myocardium. It had a significantly higher performance than both the standard TTE and cine-CMRI in detecting LV thrombi. DE-CMRI has a 100% negative predictive value and 100% sensitivity. The absence of vascularity in the thrombi prevents the late gadolinium enhancement on CMRI increasing the sensitivity and specificity of such a modality. Nonetheless, DE-CMRI cannot be afforded for all acute MI patients. So, an algorithm entailing routine non-contrast echocardiography for stratifying patients based on apical wall motion score was proposed; the presence of apical wall motion then warrants performing DE-CMRI.

V. Conclusion This report highlights a pedunculated LV thrombus in a case of toxic cardiomyopathy with a previous history of anterior acute myocardial infarction. Multidisciplinary management is a cornerstone in managing similar complicated cases. Early surgical management of pedunculated LV thrombi is the management of choice, and it should be considered to avoid the failure rates of anticoagulation and thrombolytic medications. A clear diagnostic algorithm should be adopted for early diagnosis and for avoiding embolic presentations. Similarly, screening algorithms should also be developed for patients with non-ischemic cardiomyopathies and those liable for LV thrombosis with normal LV function, inflammatory bowel disease, and hypercoagulable states for example. Furthermore, large clinical trials on the efficacy of prophylactic anticoagulation following acute MI, specifically anterior/ LAD MI are needed.

References





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