

CLINICAL VIGNETTE

Late Presenting Anterior Myocardial Infarction Complicated by Left Ventricular Apical Thrombus with Embolization in Setting of Severe Bleeding from Uterine Leiomyoma

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Introduction

Left ventricular (LV) apical thrombus is a complication that continues to occur frequently after myocardial infarction. Increased number of available antithrombotic agents and anticoagulants have complicated anticoagulation strategies. We present a 57-year-old female with a late presenting anterior myocardial infarction complicated with a LV apical thrombus with embolization to lower extremity in setting of severe vaginal bleeding from uterine leiomyoma.

Case Report

A 57-year-old female with longstanding tobacco use, presented more than three days after onset of severe substernal chest pain radiating to the left arm and shoulder. On arrival, she had significant elevation of cardiac enzymes and ECG with anterior lead q waves with residual anterior and lateral lead ST elevations (Figure 1). Her chest pain had reduced to 1/10 in severity upon arrival but she had difficulty breathing and reported recent episodes of heavy vaginal bleeding for which she was treated with hormone supplementation. Urgent echocardiogram revealed severe left ventricular systolic dysfunction with left ventricular ejection fraction of 30-35% with segmental wall motion abnormalities and a large apical echodensity consistent with a mobile thrombus that was 3.0X1.9cm in size (Figure 2). Chest x ray revealed pulmonary congestion consistent with acute systolic congestive heart failure due to the late presenting anterior myocardial infarction. She was given low dose aspirin and therapeutic dosing of low molecular weight heparin with standard medical therapy including beta blocker, ACE inhibitor, high dose statin. After being diuresed, she was taken for coronary angiography and bilateral heart catheterization revealing a mid 100% thrombotic total occlusion of the mid left anterior descending artery with elevated left sided filling pressures with mean pulmonary capillary wedge pressure of 37mmHg and mean pulmonary artery pressure of 35mmHg (Figure 3). Intra-aortic balloon pump (IABP) was placed. She was considered a suboptimal candidate for percutaneous coronary intervention in light of potential need for dual antiplatelet therapy in addition to systemic anticoagulation and ongoing bleeding. After optimization of volume status, IABP was removed. Hospital course was further complicated with decreased perfusion to right foot with imaging revealing right mid popliteal artery thrombotic occlusion. Vascular surgery felt the right popliteal artery occlusion was secondary to embolization from the left ventri-

cular apical thrombus (Figure 4). Distal perfusion to foot was preserved without any threatening signs of limb ischemia with continued anticoagulation with observation. She then developed worsening vaginal bleeding with hemoglobin rapidly dropping from baseline of 15 to 6.0g/dL requiring transfusions over several days. Gynecology service felt the bleeding was due to large uterine leiomyomas and patient was considered too high risk for a hysterectomy. She subsequently underwent uterine embolization per interventional radiology which helped control bleeding. She was advised to avoid hormone therapy and was transitioned to oral anticoagulation with apixaban and low dose aspirin and discharged from hospital only to return several weeks later with recurrent vaginal bleeding requiring transfusions and repeat uterine embolization. After the second procedure bleeding did not recur after resuming anticoagulation. After three months of anticoagulation, a cardiac PET scan was performed detected viable and hibernating myocardium. She underwent one vessel coronary artery bypass with LIMA graft to left anterior descending artery without complication. Due to persistent left ventricular systolic dysfunction, she also received a defibrillator. Serial echocardiograms revealed resolution of left ventricular apical thrombus as well as right popliteal artery thrombus. She was managed with low dose aspirin and apixaban post cardiac surgery.

Discussion

Left ventricular thrombus formation is an important complication that usually occurs in the setting of anterior ST-elevation myocardial infarction and associated with anteroapical aneurysm formation but can also occur in other areas of the left ventricle. Left ventricular systolic dysfunction leads to stasis, which along with increasing inflammation and platelet aggregation during myocardial infarction and activation of the clotting cascade leads to hypercoagulability which can extend up to 6 months following myocardial infarction.¹ With the introduction of percutaneous coronary intervention in the 1980's the overall incidence of post myocardial infarction complications has decreased, specifically for left ventricular thrombus formation.²⁻⁴ The incidence of left ventricular thrombus following myocardial infarction estimated in some studies at 3%, is still felt to be underestimated due to limitations of standard thoracic echocardiography compared to newer modalities such as cardiac magnetic resonance imaging.⁵

Left ventricular thrombus usually resolves or endothelializes, but with high risk of embolization. Studies have suggested embolization rates up to 10-20%, prompting systemic parenteral anticoagulation, usually with unfractionated heparin or low molecular weight heparin, transitioned to warfarin, with both single and dual antiplatelet therapy.⁶⁻⁷ Randomized trials are not available to guide duration and efficacy for long term anticoagulation in such situations but observational data has guided management strategies.^{6,8} Triple antithrombotic therapy has not been directly compared to dual antiplatelet therapy in the setting of myocardial infarction with LV thrombus.

Conclusion

We encountered a challenging situation in which our patient was actively bleeding upon presentation and demonstrating embolization. Strategies to control bleeding while initiating warranted anticoagulation involved constantly weighing the risks and benefits of treatment strategies. The decision was made to avoid immediate coronary revascularization due to the patient's clinical stability after optimization with medical therapy.⁹ She was managed with single antiplatelet therapy with off label use of a direct oral anticoagulant. Current data is limited to case reports regarding use of direct oral anticoagulants in this setting. This case demonstrates the complexity of individualized decisions involved in managing a complication post myocardial infarction with LV thrombus in a patient with active bleeding. Data and standardized treatment approaches for such patients continues to remain limited.¹⁰

Figures

Figure 1: ECG revealing anterior q waves and ST elevation in anterior and lateral leads.

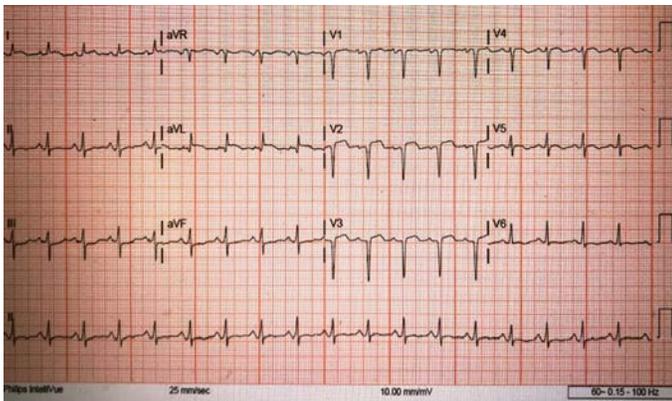


Figure 2: Echocardiogram of left ventricle with apical thrombus without and with contrast.

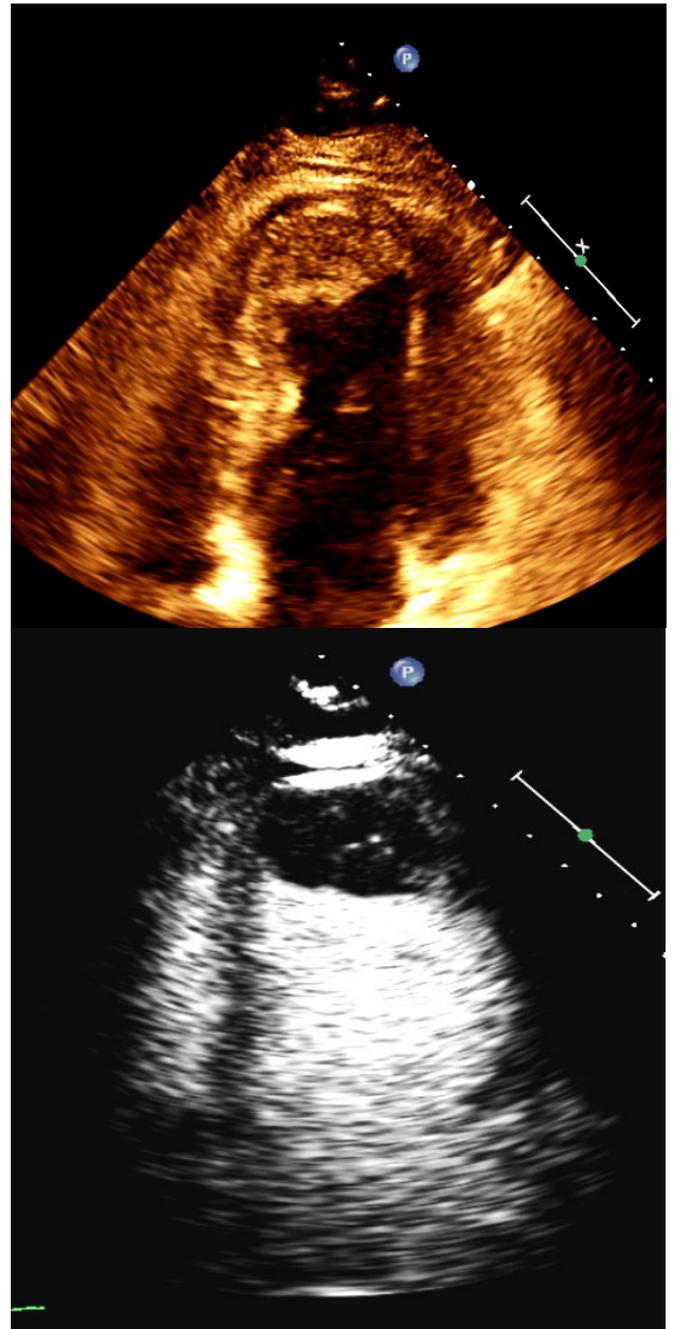


Figure 3: Coronary angiogram revealing 100% thrombotic mid occlusion of left anterior descending artery.

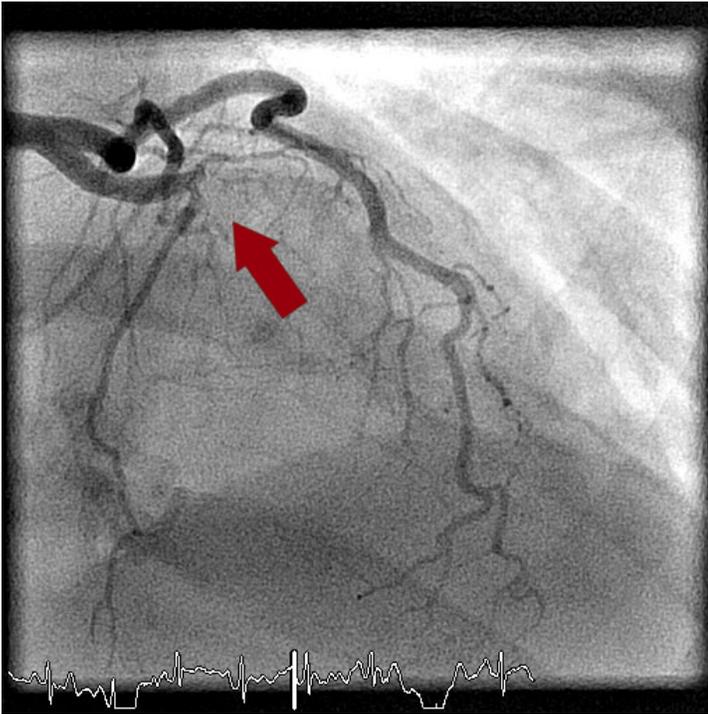


Figure 4: CT angiogram of lower extremities revealing occlusion of right mid popliteal artery.



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