Title

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CLINICAL VIGNETTE

If You Don't Take a Picture, You Can't Find a Clot: Left Ventricular Thrombus Formation after an Anterior Myocardial Infarct due to Spontaneous Coronary Artery Dissection

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Case

A 62-year-old female with hypertension, hypercholesterolemia, and chronic headaches, presented to the Emergency Department with acute onset of pressure-like chest pain and nausea. EKG showed normal sinus rhythm with a heart rate of 69 beats per minute and marked diffuse ST elevations in leads V3-V6, I, II, III, and aVF. Blood pressure was initially hypertensive at 180/107 mmHg and her oxygen saturation was 95% on room air. She was rapidly taken to the cardiac catheterization lab where access was obtained in the right femoral artery. The right coronary artery showed no visible atherosclerotic disease. The left coronary artery was then selectively engaged with a 6 French extra-backup (EBU) 3.75 guide catheter, angiogram showed no obvious plaque rupture or thrombus, however there was intimal disruption in the proximal left anterior descending (LAD) and abrupt tapering of the mid LAD to a very small caliber vessel that extended well beyond the left ventricular (LV) apex. There was contrast hangup in the walls of the proximal and mid LAD and TIMI I flow to the distal vessel (Figure 1), consistent with Type 2 angiographic spontaneous artery dissection (SCAD). In the setting of ST elevation myocardial infarction with reduced flow to a large myocardial territory and ongoing ischemic symptoms, the decision was made to proceed with intervention on the LAD to attempt to restore coronary flow.

The EBU guide catheter was sub-selective deep in the left circumflex coronary artery, so it was exchanged for a less aggressive 6F JL4 guide catheter. A coronary guidewire was carefully advanced to the distal LAD and intravascular ultrasound (IVUS) was performed which confirmed wire position in the true lumen. There was essentially no flow to the apical LAD so the decision was made to dilate the mid to distal vessel, initially with a 1.5 x 20 mm balloon to 4 atm for several inflations. Next, a 2.0 x 20 mm balloon was used up to 6 atm for several inflations up to 45 seconds in duration. Repeat angiography showed improvement in the SCAD lesion and flow to the distal vessel. The patient's clinical symptoms also improved and the ST elevation on the monitor had resolved. We then decided to stop the procedure rather than committing the patient to multiple long stents in the small distal artery with poor outflow. She was placed on dual antiplatelet therapy with aspirin and ticagrelor, metoprolol succinate, a continuous heparin infusion, and a continuous nitroglycerin infusion. The patient did well clinically with no further chest pain. Serial troponin I levels went from an initial level of 0.23 ng/mL to a peak of 48.3 ng/mL. The following morning, echocardiogram showed akinesis of the LAD territory including the apical septal, apex, and apical inferior segments, with normal motion of the remaining territories and an overall left ventricular ejection fraction (EF) estimated at 44% by Simpson's biplane method. The heparin and nitroglycerin infusions were stopped after 48 hours. The following day a repeat echocardiogram was performed to evaluate for any early recovery of wall motion and a new hyperechoic layering was seen in the dyskinetic region of the apical septum and apex. This was better visualized with contrast echocardiography, confirming the presence of a large LV thrombus (Figure 2). Oral anticoagulation was started with apixaban 5 mg twice daily and ticagrelor was changed to clopidogrel with discontinuation of her aspirin therapy. She was able to be discharged and on follow up one month later had been doing well with no new cardiac or neurologic events.

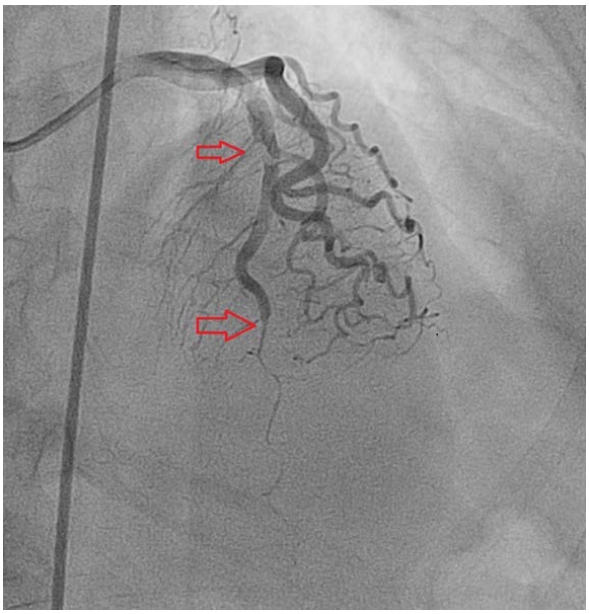

Figure 1. Left coronary angiogram showing intimal disruption in the proximal LAD (top arrow) and an abrupt tapering in the mid LAD (bottom arrow) with severely reduced flow to the distal and apical LAD, consistent with spontaneous coronary artery dissection.

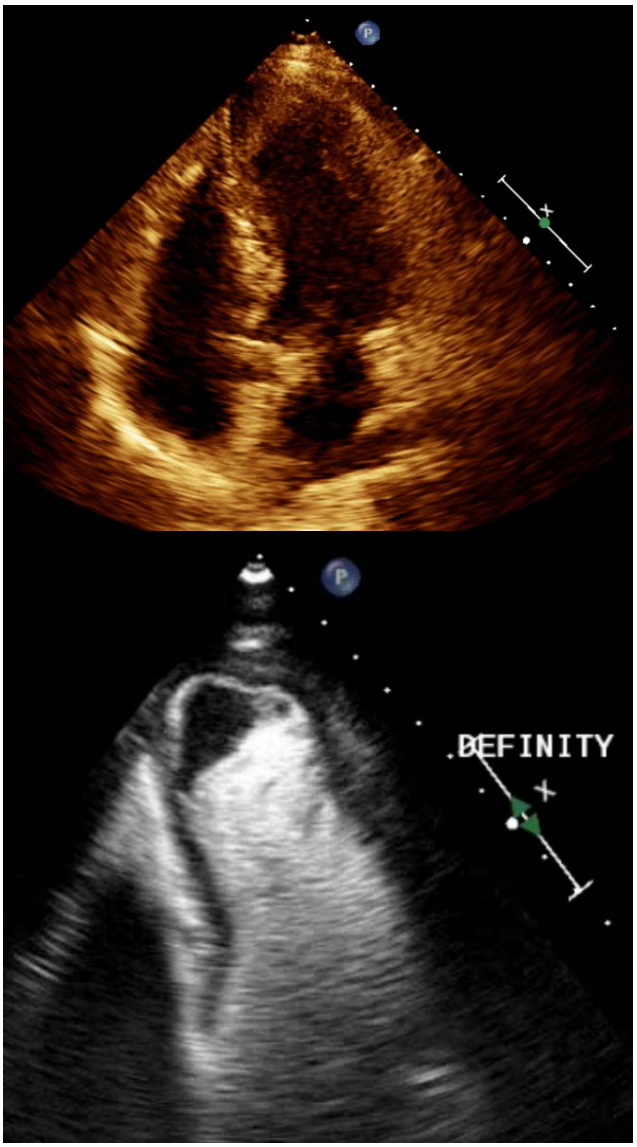

Figure 2: Echocardiograms in the apical 4-chamber view without (left) and with (right) contrast administration, showing a large apical LV thrombus in the dyskinetic region of the apex and apical septal wall segments.

Discussion

LV thrombus is an important complication that can occur following an acute myocardial infarction with the associated increased risk of cardioembolic stroke or systemic embolism. As this case illustrates, it can happen early after the event in the setting of myocardial tissue injury, blood stasis around the akinetic or dyskinetic region, and the pro-inflammatory state of acute coronary syndrome. Patients are at higher risk in large myocardial infarctions, prolonged chest pain symptoms or delayed revascularization, anterior MI, and those with a higher number of wall segments that are akinetic or dyskinetic. The overall incidence of LV thrombus varies among studies, but recent data suggests that the incidence may be as high as 26% of patients with anterior myocardial infarctions with reduced EF <45%.[1] Despite this, there are no specific guidelines regarding the type or timing of imaging to evaluate for this. Echocardiogram is the most widely available and cheapest modality, but can have limited sensitivity depending on acoustic windows and imaging quality. The use of echocardiographic contrast agents can significantly improve visualization at the apex, as in our case. One study specifically stated that LV thrombus assessment is the clinical indication that markedly increased the sensitivity and positive predictive value from 33% and 29% to 60% and 75%.[2] Cardiac magnetic resonance imaging (CMR) with delayed gadolinium enhancement is the modality with the highest sensitivity and specificity, as there is no uptake of gadolinium by the thrombus making it easily differentiated from surrounding myocardial tissue. Limited availability and high cost makes this less useful on a large scale especially for serial examinations. Computed tomography (CT) has potential use in the detection of LV thrombus but has not been validated, although it is already in clinical use for evaluating left atrial thrombus in patients with atrial fibrillation. Initial echocardiography is usually performed soon after the event, but it has been proposed that repeat studies with echo contrast should be considered in high risk individuals around 72 hours and 14 days later.[3] As hospitals push for early 24-48 hour discharge after STEMI, care should be taken that adequate follow-up including imaging can be arranged when indicated.

While the incidence of LV thrombus after myocardial infarction is high, the role of prophylactic anticoagulation to prevent formation and cardioembolic events is not clear. Much of the literature is from the pre-stent era as well as prior to the widespread use of direct oral anticoagulants (DOACs) rather than warfarin, and have shown inconsistent benefit or even harm with adding anticoagulation in these patients.[4] This is in part due to the increased bleeding risk as most patients are on concurrent single or dual anti-platelet therapy. The 2013 ACC/AHA guidelines for management of STEMI have a IIb recommendation that anticoagulant therapy may be considered in post-STEMI patients with anterior apical akinesis or dyskinesis (Level of Evidence: C).[5] Once a thrombus is identified, anticoagulation should be initiated with guidelines recommending warfarin for 3 months with consideration of a lower INR goal of 2-2.5 with antiplatelet therapy. Only one randomized controlled (but non-blinded) trial has been performed with DOACs, comparing rivaroxaban (n=39) to warfarin (n=40) with favorable results.[6] Thrombus resolution was more frequent in the rivaroxaban group with no strokes and numerically less bleeding than the warfarin group. This study, along with the prior observational studies, have been hypothesis-generating and no specific recommendation has been made regarding DOAC use in the guidelines at this time.

Conclusion

LV thrombus formation after anterior myocardial infarctions still happens frequently in the revascularization era and may be unrecognized depending on the timing and type of imaging that is performed. Thrombi can form quickly after an event, within 2-3 days, even in the setting of dual antiplatelet therapy. Close monitoring and follow up with imaging, including echocardio-

graphy with contrast or CMR, should be performed in patients at high risk. Practice guidelines do not have a strong recommendation for prophylactic anticoagulation in these patients, and currently still recommend warfarin pending more data on the newer oral anticoagulants.

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