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CLINICAL VIGNETTE

Acute Anterior Myocardial Infarction in 28-Year-Old Male Secondary to Thrombembolic Phenomenon of Unclear Etiology

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Introduction

Atherosclerosis is usually the primary etiology for most acute coronary syndromes. Plaque rupture and erosion results in coronary thrombosis, which then leads to obstruction of an atherosclerotic coronary artery.[1] Myocardial infarctions usually present in patients over age 45 and thus when patients present with myocardial infarction at much younger ages, alternate etiologies must be considered. We present a 28 year old male with an anterior myocardial infarction requiring emergent cardiac catheterization and coronary intervention.

Case Report

A 28-year-old male with no significant past medical history had just finished playing hockey and after taking a shower developed sudden, severe mid-sternal anterior chest pain that was non radiating. He had associated shortness of breath but no additional symptoms. Patient felt no symptoms while playing hockey or any prior similar symptoms. His immediate family members did not have coronary artery disease and he had no prior tobacco or substance abuse. Paramedics responded and initial ECG revealed ST elevation initially most prominent in V3-4 but upon arrival to ER, ST elevations throughout leads V1-4 with ST depression in II, III, AVF (Figure 1). He had mild elevation of cardiac enzymes and mild renal insufficiency. Emergent CT pulmonary angiogram which did not reveal any pulmonary embolus or aortic dissection. He was given aspirin 325mg and heparin 5000 unit IV bolus and transferred to the catheterization laboratory for further evaluation. Coronary angiogram revealed a large filling defect along the proximal to mid portion of his left anterior descending coronary artery consistent with a thrombus without any significant atherosclerosis angiographically throughout the epicardial coronary arteries (Figure 2). Patient was given ticagrelor 180mg loading dose with intermittent dosing of IV heparin and underwent emergent coronary intervention with insertion of a coronary balloon that was inflated along the area of occlusion. Due to high thrombus burden and lack of improvement, thrombectomy was performed with a coronary thrombectomy system with use of a temporary transvenous pacer wire. Repeat angiogram did not reveal any significant obstructive coronary artery disease requiring a stent and this was confirmed through intravascular ultrasound (Figure 3). Left ventriculogram revealed decrease in left ventricular systolic function to an EF of 40-45%. He remained on maintenance IV heparin with dual antiplatelet therapy. Low dose beta blocker was started but could not be titrated due to baseline low pressures. Repeat echocardiogram 24 hours later showed improvement in left ventricular systolic function to 50-55% and no evidence of any intracardiac shunts per saline bubble study. Hematology consultation obtained hypercoagulable testing which returned unremarkable. Patient had frequent episodes of non-sustained ventricular tachycardia and premature ventricular contractions requiring amiodarone. Patient was transitioned to apixaban 5mg twice a day with dual antiplatelet therapy with daily aspirin 81mg and clopidogrel 75mg on discharge. Patient underwent cardiac rehabilitation program with clinical improvement and discontinued clopidogrel at 1 month and remained on aspirin 81mg and apixaban 5mg twice a day chronically. Amiodarone was tapered and eventually discontinued after two months and he has remained clinically stable on outpatient follow up.

Discussion

Myocardial infarction usually occurs in patients that are greater than 45 years of age in men and 55 years of age in women. When very young patients present with chest pain, an abnormal ECG, and positive cardiac biomarkers, coronary heart disease is not always suspected as the primary etiology despite the prevalence. Most patients that present with acute myocardial infarction at a young age have at least one cardiovascular risk factor.[2] As in older patients, smoking is usually the most important risk factor associated with cardiac risk.[3] Coronary artery atherosclerosis is the usual etiology of acute myocardial infarction in both younger and older patients, but myocardial infarction can occur with normal coronary arteries in some patients.[4,5] Alternate and overlapping mechanisms and important in understanding overall pathophysiology. These include hypercoagulable disorders[6], coronary artery spasm, substance abuse[7], supplements (dietary, anabolic steroids)[8], myocardial bridging, coronary embolism (atrial fibrillation, vegetations), accelerated atherosclerosis (familial hypercholesterolemia, diabetes), spontaneous coronary artery dissections (postpartum, hormone therapy), stress induced (Takotsubo syndrome), coronary artery aneurysms, coronary artery ectasia, and anomalous coronary arteries.[9] Management strategies are similar to those in older patients with guideline directed medical therapy involving antiplatelet agents such as aspirin and P2Y12 inhibitors along with use of beta blockers and statins. Therapies must be adjusted based on suspected etiology and can involve use of long term anticoagulation, angiotensin converting

enzyme inhibitors, and vasodilators such as nitrates and calcium channel blockers. Diagnosis usually will require coronary angiography and in some instances percutaneous coronary intervention. Secondary prevention strategies are important in optimizing long term prognosis for these patients, with young patients having more favorable prognosis.[10]

Conclusion

Our patient presented with an acute myocardial infarction at a very young age. Coronary angiography revealed a very high thrombus burden in an epicardial coronary artery suspected of embolic origin. The exact etiology remains unknown and thus the patient remains on an antiplatelet agent in addition to anticoagulation. Bleeding risks must always be considered with use of antiplatelet agents in addition to anticoagulation. Determining the etiology of acute myocardial infarction in younger patients can remain challenging as in our patient, who presented without any significant identifiable risk factors. Clinical data and standardized treatment approaches for such patients remains limited.

Figures

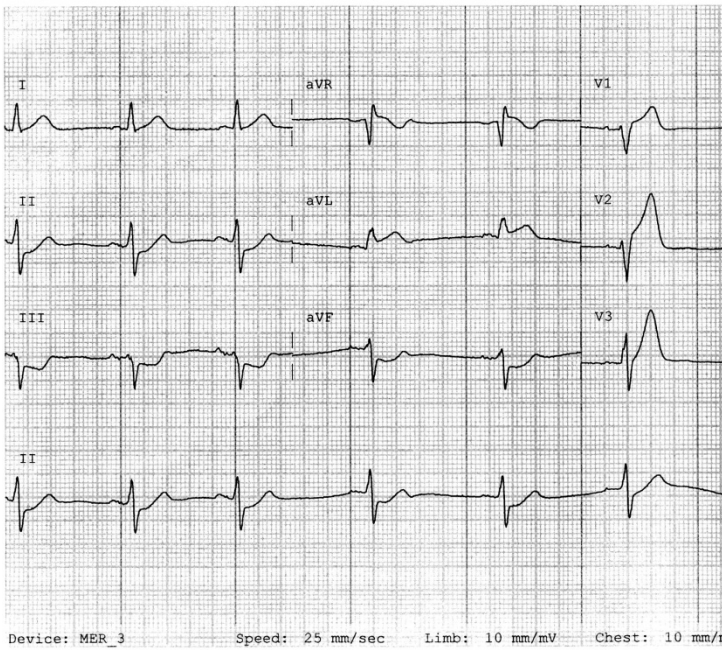


Figure 1: ECG revealing anterior lead ST elevation and inferior lead ST depression.

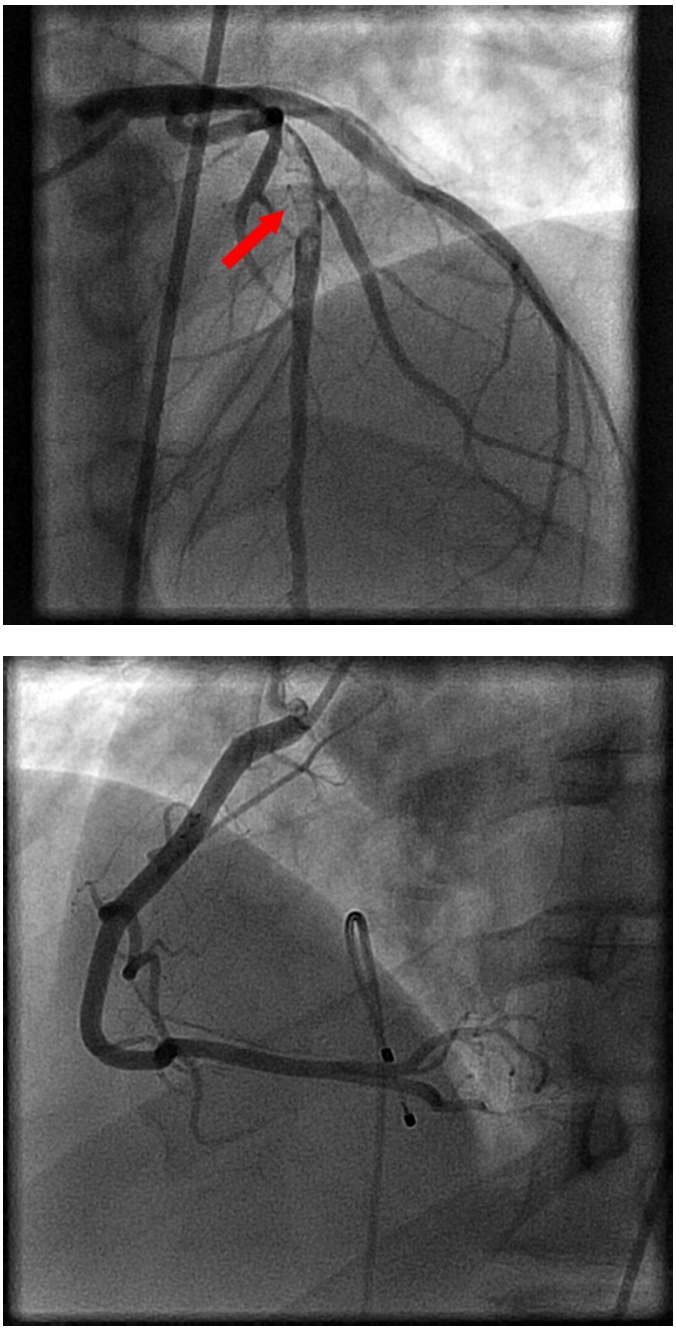
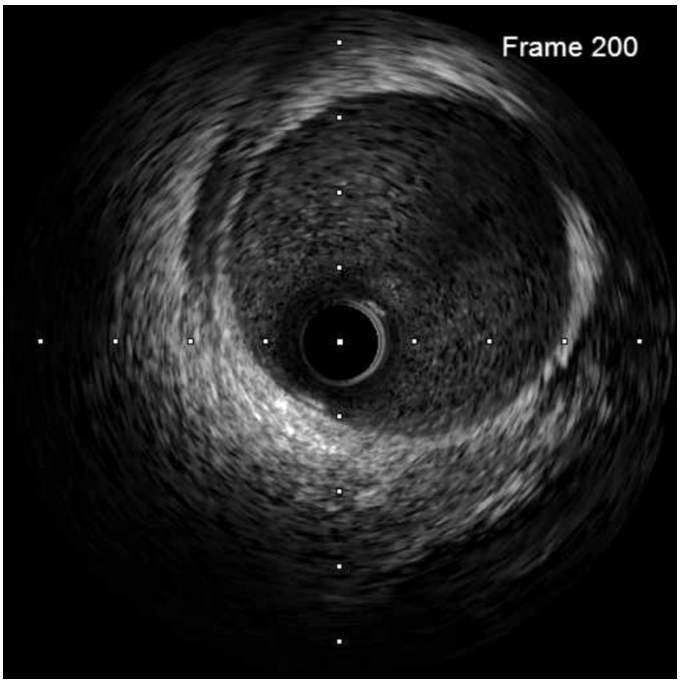


Figure 2: Coronary angiograms revealing large filling defect consistent with thrombus in proximal to mid left anterior descending artery (red arrow) and no significant coronary artery disease throughout rest of epicardial coronary arteries.



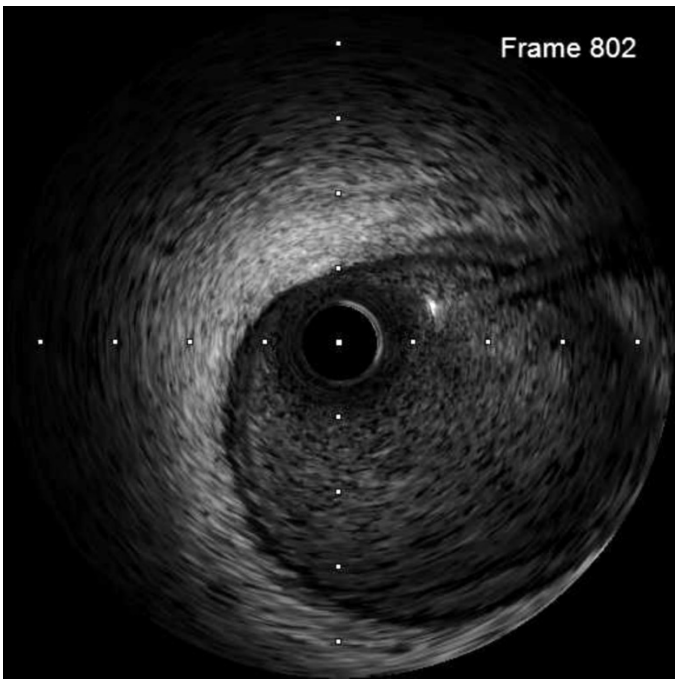


Figure 3: Intravascular ultrasound of left anterior descending artery post thrombectomy revealing no evidence of any coronary atherosclerosis with image of distal (frame 200) and proximal (802) portion of vessel.

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