NSTEMI in a 28-year-old female with recent myocarditis
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Introduction
Myocarditis and acute coronary syndrome (ACS) have similar clinical presentations and can be hard to differentiate.¹ Myocarditis is an inflammatory disease of cardiac muscle and has a variety of noninfectious and infectious etiologies. The cause of acute coronary syndrome is a mismatch between the myocardial oxygen demand and myocardial oxygen consumption. While the source of this mismatch in ST-segment elevation myocardial infarction (STEMI) is almost always coronary plaque rupture, there are several potential causes of this mismatch in non-ST-segment elevation myocardial infarction (NSTEMI). Non-coronary injury to the heart such as cardiac contusion, presence of cardiotoxic substances, and even myocarditis can produce NSTEMI.² Also, conditions such as hypotension, hypertension, tachycardia, aortic stenosis, and pulmonary embolism, though seemingly unrelated to the coronary arteries or myocardium, can lead to NSTEMI because the increased oxygen demand of the heart cannot be met.³,⁴ Unsurprisingly, acute myocarditis is more common than ACS in younger patients aged 18–29 years, but the risk of myocardial infarction (MI) subsequently increases.² Additionally, risk factors such as hypercholesterolemia, diabetes, and hypertension predict MI regardless of age and gender. Although many factors go into diagnosing myocarditis and MI, distinguishing between the two is crucial because the short and long term management and prognosis is largely different.

Case Report
A 28-year-old female with history of type 1 von Willebrand disease (VWD) and recent hysterectomy presented with chest pain. Nine days earlier, she was diagnosed with viral myocarditis at an outside hospital after presenting with chest pain and left hand and foot numbness in the setting of a urinary tract infection. At that time, her troponin was elevated to 1ng/mL and a trans-thoracic echocardiogram (TTE) demonstrated mild global systolic dysfunction with an ejection fraction (EF) of 40–45%. She was subsequently discharged on a beta blocker and ACE inhibitor. However, due to low blood pressures, the medications were discontinued. The patient presented again to the ED with 8/10 chest pain, palpitations, diaphoresis, nausea, and dizziness. The patient described her chest pain as sternal with radiation to her back and neck that increased with exertion. However, her symptoms were the same as when she was first suspected to have myocarditis. She denied infectious symptoms including vomiting, fever, and cough.

Despite minor bleeding complications from the VWD, which prompted the patient
to get a hysterectomy the month prior, the patient had not had any serious medical problems or hospitalizations. The patient’s mother, father, and sister also have VWD. The patient denied alcohol, drug, or tobacco use since 2017. Of note, the patient had an extensive family history of cardiac problems all starting around age 30 (Figure 1).

The second time the patient presented to the ED, she was afebrile and hemodynamically stable. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were within normal limits. Initial troponin was 0.03ng/mL and EKG showed T wave inversions in leads III and aVF (Figure 2). The patient continued to have mild non-pleuritic chest pain with movement, which she described as pressure. On cardiovascular exam, there were no murmurs or pericardial rub appreciated and all other components of the physical exam were unremarkable. The patient’s troponins peaked at 0.052ng/mL and she was placed on aspirin and low dose captopril 6.25mg BID. Based upon the patient’s age, presentation, and initial diagnosis of viral myocarditis, there was low suspicion for ACS and the patient was started on colchicine for suspected viral myocarditis. Repeat TTE was within normal limits and bubble study showed no evidence of a right-to-left shunt. Autoimmune panel (ANA, RF, C3, C4, quant immunoglobulins) and D-dimer were negative. The patient’s lipid profile (LDL 54, Tot 117, TG 158, HDL 44), TSH, and BNP were also unremarkable. The patient’s chest pressure completely resolved after initiating the colchicine and, with no abnormal lab results, the plan was to send her home after cardiac MRI the next day.

Cardiac MRI demonstrated normal biventricular function but, interestingly, a localized abnormal T2 signal and mild subendocardial late gadolinium enhancement, a scar pattern, and presence of edema suggestive of an acute MI in the distal left anterior descending artery territory. This favored a diagnosis of NSTEMI over myocarditis because of the distribution of injury, and the decision was made to take the patient for left heart catheterization. Cardiac catheterization revealed no evidence of coronary disease that would require stenting and normal left ventricular end-diastolic pressure with hypokinesis on the infero-apical wall. With her VWD and no evidence for stent placement, the patient was not treated with anti-platelet therapy on discharge. She went home on metoprolol tartrate 12.5mg BID and lisinopril 5mg daily with no residual symptoms or deficits from her NSTEMI. Although the patient had hypotension when discharged on a beta blocker and ACE inhibitor the first time, she was tolerating them well in the hospital so they were continued as part of her home regimen.

**Discussion**

The differential diagnosis of chest pain in a young female includes a wide variety of cardiac, pulmonary, and musculoskeletal etiologies. While ACS should always be a consideration even in a young healthy patient, it is usually not high on the differential. Although the etiology of our patient’s NSTEMI is idiopathic, spontaneous coronary artery dissection (SCAD), a non-traumatic and non-iatrogenic separation of the coronary arterial wall, is at the top of our differential. A patient this young should not have any appreciable plaque buildup yet, so a plaque rupture, although plausible, would be less likely. Additionally, distal SCAD would appear completely normal on coronary angiography.
uncommon, SCAD should be considered in any young patient, especially a young woman, without coronary heart disease or risk factors who presents with an acute myocardial infarction or cardiac arrest. Given this patient’s demographics and her family history, SCAD seems most likely for this patient.

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is the term used to describe patients presenting with clinical features of an acute MI, but without evidence of obstructive coronary artery disease on angiography. A review of the MINOCA literature reported a prevalence of 1-14% (mean=6%, 95% confidence interval: 5%, 7%) in patients with acute MI. Presentation of myocarditis may mimic an acute MI, resulting in a diagnosis of MINOCA. Routine cardiac MRI in patients with MINOCA has demonstrated that as many as one-third have evidence of myocarditis, making it the most common non-coronary cause of MINOCA.

Furthermore, even in patients in whom ACS is unlikely, it is important not to discount the role family history plays in the pathogenesis of ACS. There are several inherited disorders with coronary artery disease or myocardial infarction as part of the phenotypic expression. These include, but are not limited to, autosomal dominant and recessive familial hypercholesterolemia, antiphospholipid antibody syndrome, partial lipodystrophy, fibromuscular dysplasia, and homocystinuria. Additionally, family history is an independent risk factor for coronary heart disease, particularly among young individuals with a family history of premature disease. Using data from the 2011 to 2014 NHANES survey, the 2017 American Heart Association heart disease and stroke statistics report that 12.2% of adults have a parent or sibling with heart attack or angina before age 50.

**Conclusion**

This patient’s objective data (negative ESR/CRP, absence of a pericardial rub, EKG with isolated T wave inversions) pointed more to an ACS than myocarditis. Given her age, however, it seems the physicians prematurely closed on the myocarditis diagnosis. It is imperative to consider ACS in younger patients and complete a thorough work-up if their symptoms point toward ACS. Also, physicians should take a detailed family history and determine whether their patients have significant family history of heart disease. A careful review of objective data and physical exam findings will help avoid cognitive bias so a more guided approach to diagnosis and treatment can be initiated.

**Disclosures**

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**References**