CLINICAL VIGNETTE

Viral Myocarditis and Utility of Cardiovascular Magnetic Resonance Imaging

Hana Hazani, MD and Melkon Hacobian, MD

A 57-year-old male with history of hyperlipidemia and non-obstructive coronary artery disease presented to urgent care with one week of intermittent fevers, cough, and chest pain. He was sent to emergency department for elevated troponin (2.2) and ischemic changes on EKG concerning for acute coronary syndrome (ACS).

Patient reported intermittent fevers (up to 102◦F), chills, headache, and dry cough for the past week. He denied rhinorrhea, sore throat, or any sick contacts. His symptoms began after a visit to an amusement park with his teenage daughter. He developed intermittent chest pain approximately 3 days after onset of his cold symptoms. Chest pain was substernal and non-radiating, sharp in quality, mild in severity and not worsened by deep breathing. The chest pain would come on at rest and did not worsen with exertion, lasting a few minutes and resolving spontaneously. The pain was not associated with diaphoresis, lightheadedness or dyspnea. He attributed his symptoms to an upper respiratory infection and took acetaminophen for fevers. Because the fevers were recurrent, he presented to urgent care for further evaluation.

Upon arrival to urgent care, he was afebrile and hemodynamically stable. Physical exam was notable for normal cardiac exam with no murmurs and lung exam with diffuse rhonchi. Rapid influenza and streptococcal antigen tests were negative. Electrocardiogram was notable for new T-wave inversions in leads III and aVF and T-wave flattening in leads V5-6 (in comparison to EKG done 6 months ago). Stat labs were notable for normal CBC and BMP, troponin I 2.2, and BNP 109. Patient was given aspirin and was sent to emergency department for evaluation of possible acute non-ST elevation myocardial infarction.

Upon presentation to emergency department, vital signs remained stable with similar exam findings as above. Electrocardiogram demonstrated more diffuse T-wave inversions including leads II, III, aVF, V4-6. Troponin I remained elevated at 2.2. Chest X-ray showed patchy right upper lobe opacities and borderline cardiomegaly. He was admitted and Cardiology was consulted and recommended initiation of heparin drip per ACS protocol.

He was also started on baby aspirin, atorvastatin, and metoprolol, and heparin drip was continued. Repeat troponin at 6 hours, was down to 1.5. Echocardiogram demonstrated normal ejection fraction with no wall motion abnormalities. He remained free of chest pain free throughout hospitalization.

Of note, a prior CT coronary angiogram in 2013 demonstrated non-obstructive atherosclerosis of left anterior descending artery (LAD). His risk factors for coronary artery disease included BMI of 28 and LDL cholesterol of 140. He did not take any medications at home.

Given the atypical nature of his chest pain and evidence of pneumonia, viral myocarditis felt to be more likely than ACS. The patient was recommended to undergo coronary angiogram given his history of LAD atherosclerosis as well as cardiovascular magnetic resonance imaging (CMR). The patient opted for CMR prior to undergoing cardiac catheterization. Cardiac MR demonstrated patchy mid to sub-endocardial delayed enhancement involving the left ventricle free wall in basal and mid inferior segments consistent with myocarditis. It did not conform to any particular vascular territory. After CMR the patient declined additional cardiac workup. He was discharged on levofloxacin, baby aspirin, metoprolol and atorvastatin with close follow up with cardiology. On outpatient follow up, he remained symptom free and low-dose Lisinopril was added.

Discussion

There is no single clinical or imaging finding to definitively confirm the diagnosis of myocarditis. A combination of a careful history, physical examination and non-invasive testing should be performed to make the diagnosis. Endomyocardial biopsy remains the gold standard for the diagnosis of myocarditis. The invasiveness of endomyocardial biopsy along with sampling errors and false negative results in mild disease are significant limitations.

Echocardiogram has limited value, especially in patients with less severe disease. Nuclear imaging is rarely used to diagnose myocarditis due to low specificity, radiation exposure, and limited radiotracer availability.

Cardiovascular magnetic resonance (CMR) is a safe imaging modality with clear anatomical visualization, inter-observer consistency, and quantitative accuracy and is considered the most accurate cardiovascular imaging modality.

Targets for CMR include; 1) functional and morphological abnormalities, 2) tissue pathology and 3) features of myocardial inflammation.[1]

Since assessment of LV and RV function with CMR is reproducible, it allows identifying, quantifying and detecting mild functional abnormalities. Pericardial effusion is a sign of active inflammation. A transient increase in wall thickness or a decrease of LV mass can also be detected by CMR.

Features of tissue pathology in active myocarditis include intracellular and interstitial edema, capillary leakage, hyperemia, and in severe cases, cellular necrosis and subsequent fibrosis.

Vasodilatation is a characteristic feature of tissue inflammation that leads to increased uptake of contrast agents during the early vascular phase. T1-weighted CMR images can be used to detect increased uptake of gadolinium-based contrast into the interstitial space during the initial minutes after the contrast bolus. This is called myocardial early gadolinium enhancement and is a reflection of myocardial inflammation.

In contrast, late gadolinium enhancement reflects irreversible myocardial injury (i.e., necrosis and fibrosis). In ischemic injury, subendocardium is always involved, however, in non-ischemic injury, such as myocarditis, subendocardium is spared.

In order to have optimal diagnostic accuracy for myocarditis, at least 2 out of the 3 criteria must be present.

A CMR study should only be performed in symptomatic patients (presenting with chest pain) who have elevated troponin, abnormal EKG, normal coronary arteries, and high suspicion for viral etiology, or in patients with possible myocarditis being exposed to strenuous physical exercise, as seen with professional athletes.

Images are obtained in supine position with patients holding their breaths, with ECG-gated image acquisition. Current literature is based on data published utilizing Gadolinium Gadepentetate Dimeglumine, therefore, the recommendations are valid for this agent.

Cardiac magnetic resonance may not be sensitive during the first few days (up to 7 days) of clinical onset of myocarditis due to the focal nature of early stages of the disease. Therefore, in patients with high suspicion and normal CMR, a repeat CMR may be considered. Since tissue inflammation should resolve in 2-3 weeks, a follow-up at least 4 weeks after the onset of disease may be useful to differentiate uncomplicated from complicated cases.

REFERENCES

1. **Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, White JA, Abdel-Aty H, Gutberlet M, Prasad S, Aletras A, Laissy JP, Paterson I, Filipchuk NG, Kumar A, Pauschinger M, Liu P; International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis**. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol*. 2009 Apr 28;53(17):1475-87. doi: 10.1016/j.jacc.2009.02.007. PubMed PMID: 19389557; PubMed Central PMCID: PMC2743893.