Introduction

Acute myopericarditis refers to the inflammation of the pericardium and myocardium. Many times pericarditis and myocarditis present individual; however, it is not uncommon to see involvement of both structures due to their close anatomic proximity. The etiologies of myocarditis and acute pericarditis have much overlap, with most cases having an infectious origin. Other etiologies include: autoimmune, cardiac (post MI), metabolic, trauma, drugs and radiation. NSAIDs appear to be the mainstay therapy, with a good prognosis.

Pericarditis is an important differential in the emergency department for patients presenting with a chief complaint of chest pain. Recurrent idiopathic pericarditis occurs in as many as 30% of cases of acute pericarditis. Overall, such cases have a good long-term prognosis with little risk of major complications. In many cases, the definitive etiology cannot be identified. The mainstay medical therapy is aspirin or NSAID plus colchicine; second-line therapy consists of low to moderate doses of corticosteroids plus colchicine. Typically, the diagnosis can be established with a history and physical exam, accompanied by testing such as ECG, troponins, Echo, chest x-ray.

A 19-year-old male presented to the emergency department with chest pain, shortness of breath and nausea. He reported feeling ill 2 days prior to the onset of pain. History was negative for any cardiac condition. Family history was notable for a father that died of an MI at age 45. Vital signs were: T 100.7 F, BP 116/67, HR 115. On physical exam, the patient was diaphoretic. Lung and cardiac exam were benign.

Methods/Procedure

Patient was given a diagnosis of myopericarditis and was discharged with colchicine, prednisone and Naproxen with outpatient cardiology follow up. The patient re-presented to the emergency department 9 days later with the same symptoms. EKG showed ST elevations across several leads and troponins were elevated at 456 ng/L. The patient was taken to the cath lab; coronary angiography showed no narrowing or signs of occlusion in any of the coronary arteries.

An ECG revealed diffuse ST elevations. Troponins were elevated at 573 ng/L. His ECG (see Figure 1) and clinical picture were consistent with pericarditis rather than a STEMI. The ED consulted internal medicine, infectious disease and cardiology for advice on how to proceed. The patient was placed on Naproxen, colchicine, prednisone and was discharged on Naproxen with outpatient cardiology follow up. The patient re-presented 2 days later with the same symptoms. EKG showed ST elevations across several leads and troponins were elevated at 456 ng/L. The patient was taken to the cath lab; coronary angiography showed no narrowing or signs of occlusion in any of the coronary arteries.

Other lab tests performed at re-presentation (all negative):
- Viral Panel
- Lyme disease titer
- Autoimmune disease work up
- Bacterial and fungal blood cultures
- PPĐ skin test
- Urine drug screen

Clinical Discussion

Various specialists recommended different anti-inflammatory agents, leading to the patient being started and discharged on steroids and 2 anti-inflammatory agents; the combination of the two is known to cause kidney damage. A more thorough workup wasn’t necessary since the presentation was not severe and a more ominous etiology was not suspected.

Conclusions

The case demonstrates how a multi-specialty intervention can lead to an excessive diagnostic work up in terms of costs, healthcare utilization and patient safety. Additionally, consultation from multiple services can lead to utilizing too many medications that can have potentially synergistic adverse effects.

Differential Diagnosis

- Cardiac
  - Congenital heart diseases
  - Various forms of Angina
  - Myocardial Infarction
  - Peri/Myocarditis
  - Arrhythmias
  - Aortic dissection
  - Substance use

- Non-Cardiac
  - Spontaneous pneumothorax
  - Pulmonary embolus
  - Tumor
  - GERD
  - Esophageal rupture
  - MSK pain
  - Trauma
  - Foreign body

References