Case Report
An 83 year old male with known coronary disease, hypertension, and dyslipidemia presented to the local emergency department with several days of substernal chest discomfort. He described his symptoms as a pressure in his upper chest, radiating to his back, worsened with deep inspiration. He denied an exertional component to his symptoms. He also noted lightheadedness and dizziness, as well as questionable chills for the past day. He had no other complaints.
In the emergency department, the patient was noted to be afebrile, hypotensive with a blood pressure of 79/66 mmHg, and tachycardic with an irregular heart rate of 105. He showed minimal improvement with IV fluid resuscitation. Initial EKG showed atrial fibrillation with a rapid ventricular response, left anterior fascicular block, and isolated ST elevation of less than 1mm in V2 (figure 1).

Initial laboratory evaluation was notable for a mildly elevated troponin I to 0.2, creatinine elevated to 2.2, and mild thrombocytopenia with 80,000 platelets. A non-contrast chest CT scan showed a mildly dilated aortic root and diffuse aortic calcifications. A bedside transthoracic echocardiogram showed normal left ventricular wall motion with an ejection fraction of 65-70% with normal right ventricular size and function. There was a trace pericardial effusion. An MR angiogram of the chest was then performed, showing no evidence of aortic dissection or
pulmonary embolism, and bilateral lower extremity Doppler examination showed no evidence of deep venous thrombosis.

The patient continued to have chest pain with hypotension refractory to IV fluid resuscitation, and was admitted to the intensive care unit. He was started on empiric broad-spectrum antibiotics, and repeat EKGs showed evolving diffuse ST segment elevations and PR depressions, consistent with acute pericarditis (figure 2).

Blood cultures showed no growth and antibiotics were discontinued, and as the patient’s renal function normalized he was started on empiric NSAID therapy with minimal relief. He was then treated empirically with colchicine and prednisone, with improvement in his symptoms and hemodynamics. The decision was made to withhold anticoagulation for atrial fibrillation given concern for hemorrhagic pericardial effusion. By hospital day 5, the patient was symptomatically improved and hemodynamically stable, back in normal sinus rhythm. He was restarted on his cardiac medications and discharged home to continue a 2-week course of colchicine and indomethacin and complete a rapid steroid taper, with recommendations to consider anticoagulation for atrial fibrillation once the pericarditis had resolved.

Background:
Pericarditis is the inflammation of the fibroelastic sac surrounding the heart. The exact prevalence of pericarditis is unknown, but one study found evidence of pericarditis in up to 1 in 1,000 hospitalized patients and up to 5% of patients presenting to the emergency department with chest pain. The spectrum of pericarditis can range from mild inflammation isolated to the pericardium presenting with chest pain, to severe inflammation involving both the pericardium and myocardium (myopericarditis) with or without hemodynamic compromise that can require hospitalization. Patients typically present with typical chest pain, often proceeded several days by non-specific viral symptoms. Typical findings include chest pain, pericardial friction rub,
diffuse ST elevations on EKG with or without PR depressions, and pericardial effusion. In order to diagnose pericarditis, at least 2/4 of the above findings should be present\(^2\). Other common findings include elevated acute phase reactants such as elevated CRP or ESR, and elevation of cardiac markers such as CK-MB or troponin when the myocardium is involved, and hemodynamic effects such as hypotension and tachycardia.

Etiology:
There are multiple potential causes of pericarditis. In approximately 2/3 of cases, pericarditis is secondary to infection. This is most often viral, but can also be fungal, bacterial, or tuberculous. The remaining cases are secondary to a non-infectious process such as neoplastic, rheumatologic, vascular, or iatrogenic (such as post-procedural or post-surgical)\(^2\). In developed countries, the cause is most often presumed to be viral or idiopathic, and often an exact cause cannot be determined. Regardless, more serious potential etiologies such as underlying tuberculosis, HIV infection, malignancy, or systemic inflammatory disease should be ruled out.

Evaluation:
All patients with a suspicion for pericardial disease should have a detailed history and physical exam with close attention to travel and exposure history and high-risk behaviors, and careful cardiac auscultation for friction rub, evaluation of pulsus paradoxus for evidence of tamponade, and examination of jugular venous pressures. All patients should further be evaluated by EKG, transthoracic echocardiography, chest x-ray, and basic blood tests including blood counts, serum chemistries, renal function, and cardiac and systemic inflammatory markers. Additional testing, such as blood cultures, screening for tuberculosis, HIV, malignancy, or rheumatologic diseases should be performed on a case-by-case basis depending on patient-specific findings\(^3\).

Risk Stratification:
Although many patients with acute pericarditis are admitted for in-patient management, patients with uncomplicated pericarditis without high-risk features can be managed as an outpatient. High-risk features that confer a higher rate of complications and warrant admission include: fever with leukocytosis, large pericardial effusion or any effusion with concern for tamponade, history of immunosuppression, systemic anticoagulation, trauma, elevated cardiac markers, or failure to respond to initial first-line therapy\(^4,5\).

Treatment:
Treatment of acute pericarditis should be directed at the underlying etiology; however, in the developed world most cases are assumed to be viral or idiopathic in nature. Therefore, once more concerning etiologies have been ruled-out, uncomplicated pericarditis without high-risk features can often be managed as an outpatient with a two-week course of non-steroidal anti-inflammatory drugs (NSAIDs). Multiple studies have demonstrated that for idiopathic or non-specific viral causes, NSAIDs alone are affective in 70-80% of cases\(^4,6\). However, several studies have demonstrated that the addition of colchicine to standard therapy increases the response rate and decreases the rate of recurrence, and therefore most experts recommend addition of colchicine to NSAIDs as first-line treatment for uncomplicated viral or idiopathic pericarditis\(^7-9\). Corticosteroids have been found to confer an increased risk of recurrent pericarditis, and should therefore be reserved for those patients who have failed standard therapy with NSAIDs and colchicine, and in whom an alternate etiology has been ruled-out\(^8,10\).
Additional therapy may be warranted based on individual circumstances, such as pericardiocentesis or pericardial window for patients with large effusions or evidence of tamponade, HAART for patients with HIV/AIDS, or immunosuppression for patients with systemic inflammatory diseases.

Prognosis:
The majority of patients with idiopathic or viral acute pericarditis respond well to initial therapy. However, approximately 15-30% of those not treated with colchicine will have recurrent or persistent disease. Complications are rare but can be serious, and can include pericardial tamponade, hemorrhagic effusion, arrhythmias, and pericardial constriction with hemodynamic compromise.

Conclusion:
Pericarditis is a relatively common finding in the emergency or hospital setting, and can often present similarly to other cardiac and non-cardiac diseases. With appropriate clinical suspicion and diagnostic testing, it can usually be readily diagnosed and managed in the outpatient setting. However, serious complications can arise, and one must be aware of potential complications and predisposing risk factors that would warrant hospitalization and in-patient observation and management.

REFERENCES