A Case of Seasonal Recurrent Myopericarditis

Laura Divoky, MD
Rex D. Wilford, DO

From the Department of Internal Medicine at Summa Health System in Akron, Ohio, where Dr Divoky is in her third year of residency.

Financial Disclosures: None reported.

Address correspondence to Laura Divoky, MD, Department of Internal Medicine, Summa Health System, 75 Arch St, Suite #501, Akron, OH 44304-1424.

E-mail: ldivoky@neomed.edu

Submitted March 11, 2013; final revision received August 2, 2013; accepted August 16, 2013.

Myopericarditis occurs in 15% of patients with pericarditis. Recurrent myopericarditis occurs in 15% to 30% of patients after partial or complete recovery from acute myopericarditis. Relapse often occurs within 1 month of an initial episode. The standard of care for pericarditis or myopericarditis is initial treatment with nonsteroidal anti-inflammatory drugs for 10 to 14 days. Colchicine is often administered for 3 to 6 months for residual chest pain due to myopericarditis. The authors present a case of seasonal recurrent myopericarditis in a 32-year-old man who presented with severe chest pain in nearly the same month for 4 consecutive years. The authors conducted an extensive review of the literature but found no other case reports of seasonal recurrent myopericarditis. If a patient presents with severe chest pain requiring hospitalization, physicians should consider prescribing nonsteroidal anti-inflammatory drugs or colchicine before seasonal symptom recurrence.

J Am Osteopath Assoc. 2014;114(1):52-55
doi:10.7556/jaoa.2014.007

Myopericarditis occurs in up to 15% of patient with pericarditis.[1] Recurrent pericarditis occurs in 15% to 30% of patients after partial or complete recovery from acute pericarditis.[1] Relapse may occur in the form of recurrent chest pain, pericardial effusion, or both.[1] Myopericarditis has been linked to autoimmune disorders or their treatments, as well as many viral and bacterial infections.[2-6] We report what is, to our knowledge, the first case of seasonal recurrent myopericarditis.

Report of Case

First Presentation

A 32-year-old African American man presented to the emergency department at a northern Ohio hospital with chest pain in November 2010.

He said that for 3 days, he had experienced a dull, aching left-sided chest pain that was exacerbated by laying supine. The chest pain radiated to the center of his chest and down his right arm. Associated symptoms were diaphoresis, dyspnea, nausea, lightheadedness, cough, myalgias, arthralgias, and 1 episode of vomiting. He denied contact with any person who was ill.

Patient history included herpes labialis and chronic hepatitis B infection. His only medication was acyclovir for herpes labialis suppression; he had no signs of active lesions. He had a 13-pack-year smoking history, drank alcohol socially, and smoked 3 marijuana cigarettes per day. He denied other illicit drug use.

Vital signs at his first presentation were notable for a temperature of 101.3°F and tachycardia at 110 beats per minute. Cardiac rhythm and rate were normal with no murmurs, gallops, or rubs. Laboratory studies disclosed the following elevated values: aspartate aminotransferase, 68 U/L (normal, 12-41 U/L); erythrocyte sedimentation rate, 33 mm/h (normal, 0-10 mm/h); C-reactive protein, 17.20 mg/dL (normal, <0.6 mg/dL); troponin, 10.99 ng/mL (normal, 0.00-0.04 ng/mL), with no remarkable change when checked at 6 hours and 12 hours; creatine kinase, 983 U/L (normal, 24-195 U/L); and creatine kinase myocardial b fraction, 18.6 ng/mL (normal, 0.00-5.0 ng/mL). Hemogram with autodifferential was normal except for an elevated monocyte count of 17.0% (normal, 4%-10%). Group A streptococcal throat culture was negative. Blood cultures taken from 2 peripheral sites were negative for growth at 5 days. Results from the human immunodeficiency virus (HIV) enzyme-linked immunosorbent assay and hepatitis C antibody test were nonreactive. Antinuclear antibody (ANA) was normal (<1:40 titer). Drug screen results were positive for tetrahydrocannabinol. A chest radiograph revealed no abnormalities.

An electrocardiogram (ECG) showed subtle upward sloping T waves in anterior precordial leads and subtle PR depression in the inferior leads (see *Figure*). A left cardiac catheterization showed no evidence of coronary artery disease and an ejection fraction of 55%. An echocardiogram revealed mild concentric left ventricular hypertrophy with normal left ventricular function and no pericardial effusion.

The patient was admitted to the hospital and subsequently given a diagnosis of acute myopericarditis. He started naproxen 500 mg twice daily and colchicine 0.6 mg twice daily, and he showed clinical improvement. He was discharged to home and prescribed prednisone, which was tapered from 60 mg/d to 10 mg/d over a 3-week period.

Second Presentation

In November 2011 the patient presented to the emergency department with a continuous, dull, left-sided chest pain exacerbated by arm movement and chest wall palpation. Vital signs were normal. Cardiac examination was again normal. Complete blood cell count was notable only for mild anemia with hemoglobin level of

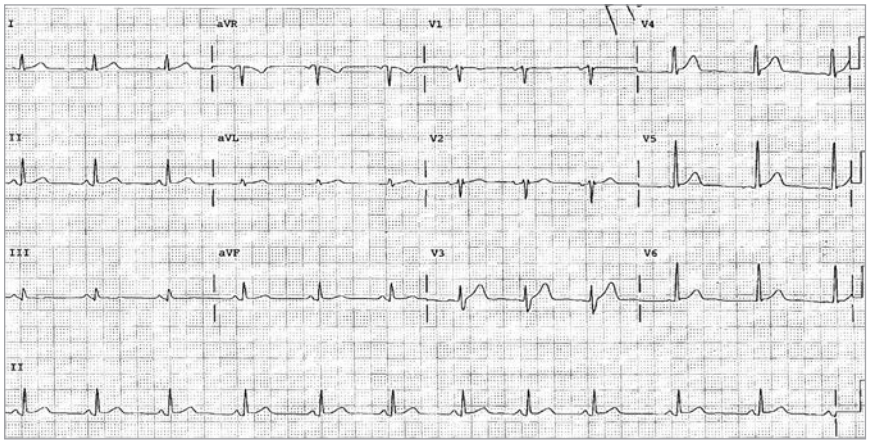

Figure.
An electrocardiogram of a 32-year-old man with left-sided chest pain revealed subtle upward sloping T waves in anterior precordial leads and subtle PR depression in the inferior leads.

Table.
Elevated Laboratory Values for a Patient With Seasonal Recurrent Myopericarditis Between November 2010, November 2011, and November 2012

Levels	2010	2011	2012	Normal Range
Initial troponin, ng/mL	10.99	5.66	8.69	0.00-0.04
Serial troponin, ng/mL	11.66	8.16	8.13	0.00-0.04
Monocyte, %	17	14.2	17.3	4.0-10.0
Erythrocyte sedimentation rate, mm/h	33	27	28	0-10

13.8 g/dL (normal 14.0-17.5 g/dL) and hematocrit level of 40.0% (normal, 42.0%-50.0%), as well as for an elevated monocyte level of 14.2% (normal, 4.0%-10.0%). Hepatic panel results were normal; erythrocyte sedimentation rate was also elevated at 27 mm/h (normal, 0-10 mm/h). Troponin levels were elevated at 5.66 ng/mL (normal, 0.00-0.04 ng/mL) and peaked at 8.16 ng/mL during serial testing. Chest radiograph revealed no abnormalities. Results of the HIV 1,2 antibody/p24 antigen combination assay and for the hepatitis C antibody test were negative. Hepatitis B core antibody test result was positive with a detected hepatitis B surface antigen. Hepatitis B surface antibody was not detected. The ECG showed minimal diffuse ST elevation. Echocardiogram revealed no abnormality, with an ejection fraction of 67%.

He was discharged to home when his chest pain resolved. We diagnosed recurrent myopericarditis, and the patient started naproxen 500 mg twice daily and colchicine 0.6 twice daily. We did not believe the patient's condition merited a prednisone taper.

Third and Fourth Presentations

In November 2012, he presented to the emergency department with a sharp and dull chest pain that was exacerbated by supine positioning, which he had experienced for 1 day. He had an associated cough, tingling in his left hand, and subjective fever and chills. Vital signs were normal. Results from the physical examination were unremarkable. Laboratory results were notable for a low hematocrit level of 41.7% (normal, 42.0%-50.0%) and an elevated monocyte level of 17.3% (normal, 4.0%-10.0%); erythrocyte sedimentation rate was also elevated at 28 mm/h (normal, 0-10 mm/h). Results of the HIV 1,2 antibody/p24 antigen combination assay were negative. Drug screen was positive for tetrahydrocannabinol and opiates, with the latter administered in the emergency department prior to urine collection. Chest radiograph revealed no abnormalities. Troponin level was elevated at 8.69 ng/mL (normal 0.00-0.04 ng/mL) and then 8.13 ng/mL. The *Table* shows a comparison between the laboratory values for the 3 years. The patient's ECG showed no ST- or T-wave abnormalities. Echocardiogram revealed no abnormalities, with an ejection fraction of 55%.

The patient's chest pain was managed with naproxen 500 mg twice daily and colchicine 0.6 mg twice daily, and he was discharged to home.

The patient never followed up in the outpatient clinic. He presented again to the emergency department with chest pain in late October 2013 but left against medical advice before workup or treatment.

Comment

Our extensive review of the literature showed no other case reports of seasonal recurrent myopericarditis. There is an overall excellent prognosis in idiopathic recurrent pericarditis.[7] The etiologic pattern of pericarditis is classified as infectious, noninfectious, or immune mediated.[1] The recurrent findings in this patient were similar pain presentations, elevations in troponin level and creatine kinase myocardial b fraction level, as well as the presence of monocytosis without leukocytosis. Monocytosis could be consistent with a viral cause for his seasonal recurrent myopericarditis. We did not search for a viral etiologic pattern because it would not have changed the course of treatment. Although the patient history included herpes labialis, the patient did not have active lesions during any of the encounters, thus ruling out this condition as the cause. He also had serologic evidence of active hepatitis B, a known cause of myopericarditis.[8] Persistent viral hepatitis, however, would not explain this patient's seasonal presentations. From an immunologic standpoint, the seasonal aspect of this case does not necessarily support a viral etiologic process because immunity would develop after the first illness—unless a different organism was responsible each time.

The cornerstone for the initial treatment of myopericarditis is a nonsteroidal anti-inflammatory drug for 10 to 14 days.[1] Colchicine for 3 to 6 months can be administered for residual chest pain.[1] Although the benefits of corticosteroids for patients with myopericarditis are a subject of debate, it is generally understood that if nonsteroidal anti-inflammatory drugs and colchicine are ineffective, low dose steroids are recommended.[1] More aggressive therapies—such as azathioprine, cyclophosphamide, and anakinra—have been used for recurrent myopericarditis that is unresponsive to conventional treatment.[1] Whereas pericardiectomy is a last treatment option for pericarditis, this procedure would be ineffective in treating patients with myopericarditis.[1]

The present case raises the question of whether seasonal prophylactic treatment would benefit a patient. A literature search yielded no recommendations because we found no previous cases of seasonal recurrent myopericarditis. We regarded yearlong prophylactic treatment to be excessive because the patient experienced pain only at the beginning of the winter months. To avoid recurrent pain for a patient with this condition, physicians may consider starting patients on prophylactic naproxen before seasonal symptom recurrence. This practice would prevent unnecessary pain to the patient and may also preempt emergency department visits and hospitalization.

Conclusion

The present case underscores the possibility that myoperidcarditis could be seasonal in nature. Physicians should recognize seasonal recurrent myopericarditis in patients who present with recurrent chest pain at similar times each year.

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