CARDIOLOGY CASEBOOK
A regular feature of the American Journal of Critical Care, Cardiology Casebook is intended to enhance practitioners’ knowledge and critical thinking. Stylized case studies are accompanied by self-assessment quizzes. We welcome letters to the Editors regarding this feature.

PERICARDITIS

Laurie G. Futterman, ARNP, MSN, CCRN, and Louis Lemberg, MD. From the Division of Cardiology, Department of Medicine, University of Miami Miller School of Medicine, Miami, Fla.

A 23-year-old ski lift attendant in Telluride, a skiing village in southern Colorado, was awakened one morning with chest pains that lessened when he sat up. The symptoms persisted and the young man decided to see an internist in Durango, Colo, a larger town several miles south of Telluride, because medical facilities in the village were limited to treatment of skiing injuries. On detailed questioning by the internist, the patient admitted to having had a mild “cold” the day before and that the chest pains were precordial with radiation to the area of the trapezia. On auscultation over the precordium with the stethoscopic diaphragm, which is preferred when auscultating high-frequency sounds, a biphasic or “to and fro” precordial friction rub was heard and was most apparent along the left mid to lower sternal edge. The electrocardiogram revealed the classic features of acute pericarditis (Figure 1). The patient was hospitalized for 48 hours to help ascertain the etiology.

QUESTIONS

1. Clinical signs and symptoms of acute pericarditis include which of the following?
   a. midsternal pressure or pain that lessens when the patient sits and leans forward
   b. midsternal pressure or pain that is enhanced when the patient sits
   c. chest pains that are relieved by sublingual nitroglycerin
   d. pericardial friction rubs, which are best heard with the bell of the stethoscope
   e. atrial and ventricular premature beats
   f. prolonged QT interval

3. What factors in acute pericarditis determine the level of risk?
   a. gradual onset of symptoms
   b. fever (body temperature >38ºC)
   c. failure to respond to nonsteroidal anti-inflammatory drugs (NSAIDs)
   d. a large pericardial effusion or tamponade
   e. traumatic pericarditis
   f. any of the above

4. Pericardial disease can be difficult to diagnose because of which of the following?
   a. pericardial disease may be silent
   b. pericarditis complicating acute myocardial infarction (MI) is uncommon
   c. steroid use in pericarditis complicating acute MI

5. The echocardiogram in acute pericarditis and tamponade is useful in which of these functions?
   a. to quantify the size of a pericardial effusion
   b. to determine the presence and location of a pericardial effusion
   c. to document the hemodynamic impact of effusion on cardiac chambers
   d. to provide follow-up surveillance for resolution or progression of an effusion
   e. all of the above

6. A diagnostic pericardiocentesis is indicated in which of the following circumstances?
   a. the history is noncontributory
   b. to determine the infective agent in acute bacterial infection
   c. to rule out malignant neoplasms
   d. to rule out an opportunistic infection
   e. after cardiac surgery

Reprint requests: Louis Lemberg, MD, University of Miami Miller School of Medicine, Division of Cardiology (D-39), P.O. Box 016960, Miami, FL 33101.
7. Treatment of acute viral or idiopathic pericarditis includes which of the following?
   a. aspirin and NSAIDs
   b. aspirin and steroids
   c. colchicine and rest
   d. steroids and rest

8. The disappearance of Dressler’s syndrome (ie, acute pericarditis after MI) is attributed to which of the following?
   a. the lower incidence of large transmural MIs
   b. the early use of thrombolytics in acute MI
   c. increased use of antibiotics in the general population
   d. drug-eluting coronary stents

ANSWERS
1. a. midsternal pressure or pain that lessens when the patient sits and leans forward.

   The pain in acute pericarditis is centralized and radiates to the trapezial or scapular areas as a consequence of phrenic nerve irritation. The pain is accentuated with inspiration and in recumbency, and it is promptly lessened or relieved by sitting and leaning forward. Anginal pain is also lessened in the sitting position, but requires a few moments to improve in contrast to the immediate relief seen when the chest pain is due to acute pericarditis. The improvement in anginal pain when changing from recumbency to a sitting position occurs after a brief time that allows a reduction in blood volume return to the heart as a consequence of gravitational forces. The salutary effect is a decrease in cardiac work.

   A high-frequency vibratory sound is characteristic of a pericardial friction rub, heard best when using the diaphragm of the stethoscope, rather than the bell, and applied with slight pressure on the precordium. Pericardial friction rubs, although heard throughout the precordium, are best heard along the middle to lower sternal borders.

   b. “J” point elevation in all leads except aVR and frequently V1.

   Figure 1 In acute pericarditis, “J” point elevation in standard lead II is greater than the elevation in any of the other standard or unipolar leads, except in aVR and often in chest lead V1, where “J” point depression is characteristic.
The diagnostic electrocardiographic feature of acute pericarditis is elevation of the ST segment (often referred to as “J” point, indicative of the junction when the QRS joins the ST segment) in all leads that reflect the potential electrical variations of the epicardial surfaces of the ventricles. Included are all leads with the exception of aVR and frequently chest lead V1, which record the potential electrical variations of the endocardial surfaces of the ventricles as “J” point depression. Equally diagnostic in acute pericarditis is that the “J” point elevation in standard lead II is greater than that in standard lead III. This is because standard lead II is a bipolar lead that records the difference in electrical potentials between 2 sites: aVR with a negative “J” point subtracted from a positive “J” point in aVF (Figure 2).

Figure 2 In acute pericarditis, “J” point depression in aVR is characteristic, whereas in acute anterior or inferior myocardial infarctions the “J” point in aVR is isoelectric.

The diagnostic electrocardiographic feature of acute pericarditis is elevation of the ST segment (often referred to as “J” point, indicative of the junction when the QRS joins the ST segment) in all leads that reflect the potential electrical variations of the epicardial surfaces of the ventricles. Included are all leads with the exception of aVR and frequently chest lead V1, which record the potential electrical variations of the endocardial surfaces of the ventricles as “J” point depression. Equally diagnostic in acute pericarditis is that the “J” point elevation in standard lead II is greater than that in standard lead III. This is because standard lead II is a bipolar lead that records the difference in electrical potentials between 2 sites: aVR with a negative “J” point subtracted from a positive “J” point in aVF (Figure 2).

3. f. any of the above
Acute pericarditis is usually self-limited. The course is severe when the patient’s body temperature is greater than 38°C, or the patient is immunocompromised, or when symptoms present gradually over weeks. Additional problems may occur in patients who are anticoagulated or fail to respond to NSAIDs, or in trauma patients, and in patients with large pericardial effusion or tamponade.

4. c. steroid use in pericarditis complicating acute MI
Pericardial disease is often silent and detected only after investigating other related complaints. The spectrum of pericardial disorders include congenital lesions, pericarditis, and its complications: tamponade and constriction. The pericardium is secondarily affected by many diseases and vulnerable to infections, malignant neoplasms, trauma, metabolic disorders, and iatrogenic factors. Improvement in thrombolytic protocols in acute MI and specific anti-infective therapy
have all but eradicated Dressler’s syndrome (pericarditis after MI) and infective pericarditis. Cardiovascular surgery, radiation therapy, and hemodialysis add to the prevalence of pericardial complications.

Acute pericarditis with or without effusion may be primary or a consequence of systemic disease. Consequently, an underlying cause should be considered when initiating treatment. However, the majority of causes of acute pericarditis are viral or idiopathic. As a result of progress in medicine, the occurrence of an infective and post-MI pericarditis (bacterial and tuberculous) has become uncommon. The pericarditis that complicates the immunocompromised patient is an exception.4

5. e. all of the above

In acute pericarditis with tamponade, echocardiography is used to characterize and locate pericardial effusions and to evaluate the hemodynamic impact on the cardiac chambers. A pericardial effusion is revealed as an echo-free space surrounding the cardiac chambers. Hemorrhagic tamponade may result in a pericardial thrombus, in which case the echocardiogram will reveal an echo-dense mass in the pericardial space. Small effusions are usually located posteriorly, whereas large effusions are circumferential.4 Echocardiographic findings in hemodynamically significant tamponade include free wall collapse of the right atrium and right ventricle, distention of the inferior vena cava during inspiration, and changes in mitral and tricuspid flow velocities. Collapse of right-sided chambers is a sensitive finding in tamponade, whereas abnormalities in cardiac filling are specific for tamponade. Follow-up echocardiographic studies are useful in evaluating the resolution or progression of effusion.

6. b. to determine the infective agent in acute bacterial infection
c. to rule out malignant neoplasms
d. to rule out an opportunistic infection

Pericardial disorder, as well as cardiac surgery, can trigger the accumulation of pericardial transudates, exudates, or blood. Pericardial effusion in the cardiac transplant recipient is often associated with acute rejection. When pericardial fluid accumulates slowly, a large effusion (>1 L) can result without tamponade. Although pericardiocentesis is usually not diagnostic in acute pericarditis, when opportunistic infection or neoplastic disease is suspected as a complication, the procedure can be diagnostic. Therapeutic pericardiocentesis is indicated when acute pericarditis is complicated by a large pericardial effusion shown by echocardiography (>20 mm), regardless of the presence or absence of tamponade. The risk of progression of tamponade is greatest when the onset is recent, the effusion is large, and diastolic pressure on the right side collapses.

7. a. aspirin and NSAIDs
c. colchicine and rest

Initially an underlying systemic disease should be ruled out. Hospitalization helps to determine etiology and to assess for complications and high-risk factors. Follow-up in the uncomplicated case can be continued on an outpatient basis. Excessive activity and exercise are not permitted until the chest pain is resolved to prevent superficial myocarditis.1 Viral or idiopathic pericarditis is self-limiting and responds to aspirin or ibuprofen. Colchicine, an old standby anti-inflammatory agent effective in acute gouty arthritis, is also effective in acute viral or idiopathic pericarditis.2,3 In chronic use, colchicine prevents recurrent pericarditis.1,4 Gastrointestinal side effects (eg, diarrhea) require temporary dose reduction. Relapse in acute pericarditis requires continued use of colchicine, aspirin, and other NSAIDs for 3 additional months.4 A proton pump inhibitor can be helpful in preventing aspirin gastritis. Indomethacin, which potentially reduces coronary flow, is avoided when there is a history of coronary artery disease.1,4

Use of corticosteroids in acute pericarditis is dramatically effective, but is associated with an increased incidence of recurring pericarditis following steroid taper, even when combined with colchicine. Systemic steroid therapy is reserved for patients who are unresponsive to or intolerant of aspirin, colchicine, or NSAIDs, and when an infectious etiology has been excluded, or when steroids are required to treat underly ing inflammatory states.2,3 Relapse and recurrence of pericarditis with pain and leukocytosis can recur over several years.3 These can be spontaneous (autoimmune response or reinfection) or follow tapering or discontinuation of steroid therapy. Colchicine and reduction in the use of steroids have prevented recurrent episodes of pericarditis.6

8. a. the lower incidence of large transmural MIs
b. the early use of thrombolytics in acute MI

Post-MI pericarditis occurs within 1 to 3 days following an acute extensive transmural MI. An estimated 7% are detected clinically; however, 40% of patients with fatal MI had complicating pericarditis. Rapid interventional protocols have reduced the incidence of extensive transmural MIs and, as a result, dramatically reduced the incidence of complicating pericarditis. Acute post-MI pericarditis was initially reported in 1956 by cardiologist William Dressler and bears his name, “Dressler’s syndrome.” The clinical syndrome occurred
in 4% of patients, 2 to 10 weeks following an acute MI, and includes fever, leukocytosis, pleuritic pain, pericardial friction rub, and effusion. The clinical characteristics were initially attributed to an allergic response to myocardial necrosis or to latent viral pericarditis. Dressler’s syndrome has been eradicated as a result of routine thrombolytic therapy.

SUMMARY

The combined use of colchicine and aspirin has all but eliminated recurrent attacks of pericarditis and is equally effective in the treatment of the acute state. Corticosteroids are avoided since they increase the incidence of recurrent attacks. Dressler’s syndrome, a designation for the pericarditis that complicates an acute MI, has been eliminated with the advent of routine thrombolytic therapy. Medical therapeutics continue to thrive and flourish!

ACKNOWLEDGMENTS

Supported in part by a grant from the Applebaum Foundation, in loving memory of Joseph Applebaum.

REFERENCES


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Pericarditis
Laurie G. Futterman and Louis Lemberg

Am J Crit Care 2006;15 626-630
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