Acute Pericarditis: From Chest Pains to Treatment

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Acute pericarditis can be a bothersome condition for primary care physicians. Although its course is generally benign, the differential diagnosis includes a number of potentially very dangerous conditions that need to be ruled out before discharge. As well, rare complications, such as pericardial effusion, tamponade and constrictive pericarditis, constantly loom in the back of our minds.

Acute pericarditis refers to the inflammation of the fibroelastic pericardium and is a common ED presentation of chest discomfort. Acute pericarditis is most commonly caused by viral infections, neoplasm and autoimmune disorders, but a more complete list can be found in Figure 1. At first presentation the cause is usually not yet determined and is considered idiopathic until further testing is complete.

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Meet Matthew

Matthew is a 37-year-old school teacher.

He presents complaining of central, pleuritic, chest pain following 1 week of nonproductive cough, rhinorrhea and a sore throat.

He denies fever and his pain occurs without anginal equivalent symptoms. His pain is not exertional and is relieved by sitting upright. Matthew does not have risk factors for ischemic heart disease.

He is otherwise healthy, without allergies or regular medications. His vital signs are normal.

His cardiovascular examination demonstrates normal heart sounds with a precordial friction rub, but no murmurs. His jugular veins are flat and his lungs are clear to auscultation. An ECG (similar to Figure 2) demonstrates diffuse ST-segment elevation and PR segment depression.

Presentation

Patients typically present with an abrupt onset of sharp, pleuritic anterior chest pain, although the discomfort is often described as dull, similar to the pain experienced with myocardial ischemia. Sitting forward classically relieves the discomfort. Patients will sometimes describe a viral prodrome. A physical examination may demonstrate a pericardial friction rub best appreciated with the diaphragm of the stethoscope over the left sternal border, but this finding may be difficult to elucidate and often varies with position.

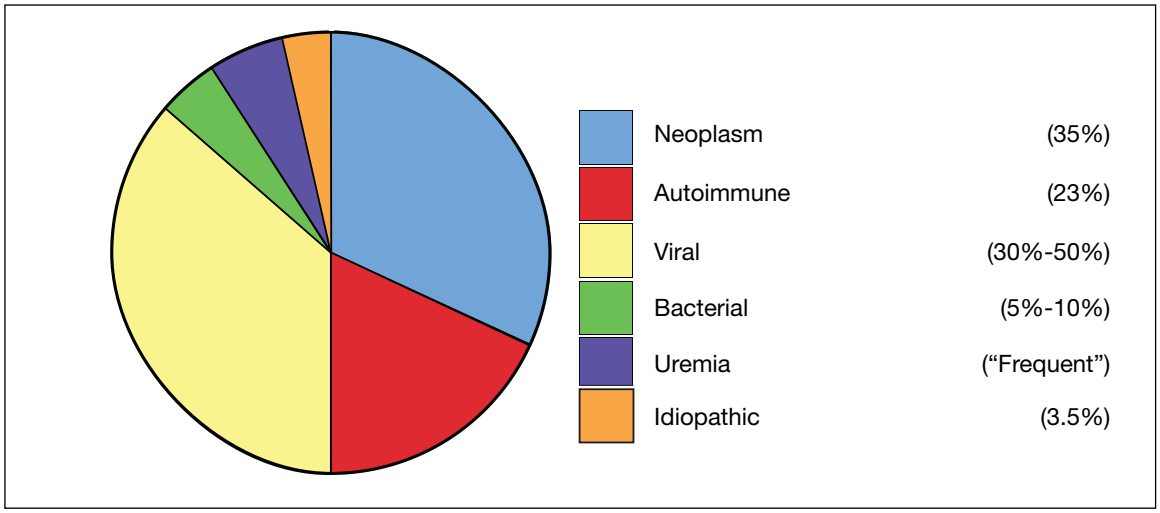

Figure 1. Etiology of acute pericarditis. Data abstracted from Maisch and Ristic (2002). The most common viral causes of acute pericarditis include adenovirus, enterovirus, cytomegalovirus, influenza, herpes simplex virus and Hepatitis B virus.

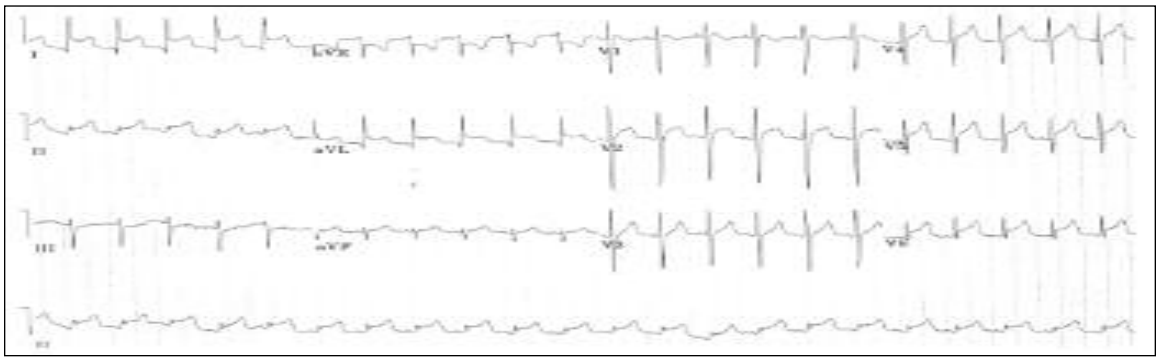

Figure 2. ECG demonstrating acute pericarditis. Note is made of (A) diffuse ST-segment elevation that does not follow an anatomic distribution and has a concave upward morphology, (B) diffuse PR-segment depression and (C) PR-segment elevation in lead aVR.

Making the diagnosis

Pericarditis is diagnosed in the setting of chest pain and pericardial friction rub as described, with ECG findings suggestive of acute pericarditis or a pericardial effusion. Two of these characteristics are necessary to make the diagnosis.

All patients suspected of having acute pericarditis should have blood sampled for a complete blood count, electrolytes, blood urea nitrogen and creatinine, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and cardiac enzymes. Anti-nuclear antibody titre, tuberculin skin testing and HIV serology should be considered in the proper clinical context. Blood cultures should be performed on septic patients. Patients should have an ECG and chest x-ray performed.

NSAIDs are the treatment of choice for viral or idiopathic pericarditis.

ECG findings of acute pericarditis occur in four phases. Patients generally present in the first phase (Figure 2), characterized by diffuse ST-segment elevation with concave upward morphology and PR-segment depression. Reciprocal ST-segment depression is seen in leads V1 and aVR. PR-segment elevation is also seen in lead aVR. The ST-segment findings can be difficult to differentiate from early repolarization, but an elevation > 0.25 of the height of the T wave in I, V5 or V6 makes early repolarization unlikely.

In the second phase, the ST segments normalize. The third stage is characterized by diffuse T wave inversion that is again followed by normalization of the T waves (fourth stage).

Acute pericarditis is an intensely inflammatory process. As a result, it is common to see elevations in serum CRP and ESR. The inflammatory process frequently involves the superficial myocardium that lies in close proximity to the pericardium and as a result, serum troponin and creatine kinase are often elevated.

Depending on the situation, acute pericarditis may need to be differentiated from myocardial ischemia and infarction, pneumonia, pneumothorax, pulmonary embolus, pleuritis and musculoskeletal chest pain. Table 1 summarizes the clinical presentation of acute pericarditis.

Table 1

Clinical indicators of acute pericarditis

Historical data

- Acute onset, sharp pleuritic chest pain
- Relief of pain when sitting forward
- Fever
- Clinical deterioration following cardiac surgery or percutaneous coronary intervention

Physical Findings

- Precordial friction rub

ECG

- Diffuse ST-segment elevation
- Concave upward morphology
- PR-segment depression
- PR-segment elevation in lead aVR

Laboratory

- Elevated erythrocyte sedimentation rate
- Elevated C-reactive protein
- Elevated troponin
- Elevated creatine kinase - muscle and brain (subunits)
- Leukocytosis

Radiological

- New or unexplained cardiomegaly on chest x-ray
- Pleural effusion

Treatment

NSAIDS are the treatment of choice for viral or idiopathic pericarditis, but if patients do not respond to therapy within seven days, other etiologies should be considered. Colchicine (alone or in combination with acetylsalicylic acid) has been shown to reduce the duration of symptoms and recurrence rates, but given its side-effect profile and significant drug interactions, we advocate an initial trial of NSAIDs alone. This is particularly true in patients with multiple medications, renal insufficiency, hepatic dysfunction or bleeding disorders.

Corticosteroids may be considered in idiopathic pericarditis after failure of NSAID and colchicine therapy, or in patients with connective tissue disorders, autoimmune, or uremic pericarditis.

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Table 2
Conditions that suggest admission in acute pericarditis

- Sepsis
- Cardiac tamponade
- Large pericardial effusion
- Compromised immune system
- Trauma (or post-procedure)
- Coagulopathy
- Significant elevation of cardiac biomarkers
- Failure of outpatient management
- Gradual onset of symptoms
- Unable to rule out other significant diagnosis

Specific therapy targeted to other specific causes of acute pericarditis should also be undertaken.

The relationship between pericarditis and pericardial effusions

This is an area that often causes significant confusion. Pericardial effusion is not always present in acute pericarditis and pericarditis is not often the cause of a pericardial effusion. Patients who have a significant pericardial effusion with pericarditis generally present as effusions—the effusion prevents the friction rub and typical pain pattern of pericarditis. Low voltage and electrical alternans are the classic ECG findings. Patients with effusions may present with shortness of breath, hypotension and shock, often with jugular venous distention.

Ideally, patients should have a baseline ECHO performed before discharge from the ED, but in stable, non-septic patients without clinical signs of a significant effusion, the ECHO can be performed in 24 to 48 hours.

Take-home message

1. At first presentation, pericarditis is most often idiopathic
2. For viral or idiopathic pericarditis, the illness is typically brief and benign
3. Oral NSAIDs are the treatment of choice
4. An ECHO can be performed within 24-48 hours in the stable, non-septic patient

Before discharge from the ED, patients should be instructed to return if their symptoms worsen or if they begin to feel unwell.

Disposition

Most cases of acute pericarditis follow a benign course and are treated on an outpatient basis, however the patient should be admitted to hospital if they have any of the conditions found in Table 2. Before discharge from the ED, patients should be instructed to return if their symptoms worsen or if they begin to feel unwell. Arrangements should be made for ECHO (if not already performed) and cardiology follow-up.

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For references, please contact **cme@sta.ca**