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Clinical Signs of Acute Pericarditis and Its Complications

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Acute pericarditis, or inflammation of the pericardial sac, is a disorder that can be caused by many underlying conditions. Making the diagnosis requires a high index of suspicion because pericarditis may occur alone or in association with a systemic disease.[1] In many cases, the etiology is not identified. The most commonly identified causes of acute pericarditis are infection, autoimmune disorders, inflammatory diseases, neoplastic disorders, iatrogenic mechanisms, metabolic disorders, and trauma (**Table 1**). Most cases of acute pericarditis, however, can be attributed to a viral infection.[2] Acute pericarditis may be complicated by pericardial effusion, cardiac tamponade, recurrent pericarditis, and chronic constrictive pericarditis.[1,3] Thus, it is important to not only recognize pericarditis, but also to assess for its complications.

SIGNS AND SYMPTOMS OF ACUTE PERICARDITIS

- The classic presentation of acute pericarditis is chest pain that is sharp, pleuritic in nature, sudden in onset, and retrosternal or left-sided in location.
- The most specific sign is a pericardial friction rub.
- Typical electrocardiographic findings include concave ST-segment elevations and PR-segment depression.
- Cardiac tamponade from a pericardial effusion is a serious acute complication of pericarditis.
- Signs and symptoms suggestive of a significant effusion include tachycardia, hypotension, jugular venous distension, muffled heart sounds, dyspnea, orthopnea, postural dizziness, and pulsus paradoxus.
- Constrictive pericarditis is a serious long-term complication of pericarditis. In advanced disease, signs and symptoms are consistent with right-sided heart failure, such as dyspnea, edema, and elevated jugular venous pressure.

EVALUATION

Clinical Presentation and History

The classic presentation of acute pericarditis, regardless of its etiology, is a patient with chest pain that is sharp, pleuritic in nature, sudden in onset, and retrosternal or left-sided in location. The pain is often exacerbated by lying down and is relieved by sitting up or leaning forward. The pain may radiate to the neck, arms, or left shoulder, making it difficult to differentiate from the pain of myocardial infarction. However, pain that radiates to one or both trapezius muscle ridges suggests pericarditis because the phrenic nerve innervates these muscles and crosses the pericardium as well.[1,4] Nonspecific symptoms include malaise, fever, chills, dyspnea, and cough. A concurrent pericardial effusion may manifest with the latter 2 symptoms. Mental status changes resulting from uremia may also be present in patients with end-stage renal disease (ESRD).[3,5]

Physical Examination

The most specific sign of acute pericarditis is a pericardial friction rub, although it is intermittently present and often varies in intensity. It is characterized as a high-pitched scratchy sound and is heard best in end expiration and along the left sternal border with the patient leaning forward.[6] A triple cadence is classically described, which coincides with atrial systole, ventricular systole, and rapid ventricular filling during early diastole. However, the triphasic rub occurs in only about half of patients. In a prospective study of 100 patients

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Table 1. Causes of Acute Pericarditis

Idiopathic
Many cases have no identifiable cause, although most are presumed to have a viral etiology
Infections
Bacterial
Viral
Mycoplasma
Fungal
Parasitic
Autoimmune
Rheumatoid arthritis
Rheumatic fever
Systemic lupus erythematosus
Scleroderma
Vasculitis
Wegener's granulomatosis
Metabolic disorders
Uremia
Dialysis-associated
Hypothyroidism
Neoplastic disorders
Primary: mesothelioma, sarcoma, fibroma, lipoma
Secondary: metastatic or direct spread
Trauma/iatrogenic
Blunt or nonpenetrating chest injury
Pericardial perforation
Radiation
Catheter and pacemaker perforations
Postthoracic surgery
Association with other syndromes
Postmyocardial and pericardial injury syndromes
Inflammatory bowel disease
Löffler's syndrome
Stevens-Johnson syndrome

Adapted from Troughton RW, Asher CR, Klein AL. Pericarditis. Lancet 2004;363:717–27. Copyright 2004, with permission from Elsevier.

with pericardial friction rub, 52 had a triphasic rub, 33 had a biphasic rub, and 15 had a monophasic rub.[7] The friction rub must be distinguished from a pleural rub, which ceases when the patient holds his or her breath. The origin of the sound has been attributed to the visceral and parietal layers of the pericardial sac rubbing together, but the fact that the friction rub occurs in the presence of an effusion between the

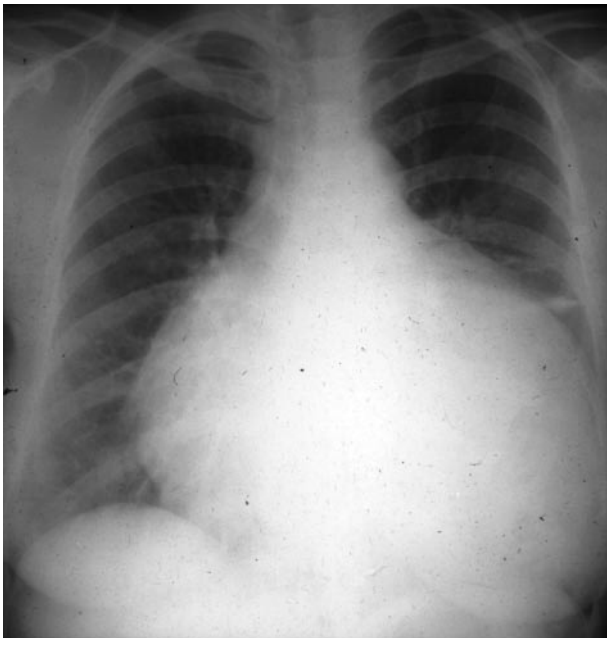

Figure 1. Pericardial effusion enlarging the cardiac silhouette on chest radiograph. (Radiograph courtesy of Dr. Melvin H. Schreiber, Department of Radiology, University of Texas Medical Branch at Galveston.)

2 layers and disappears when the effusion is removed makes this explanation less likely.[1] Other significant physical signs associated with complications of pericarditis such as tamponade and constrictive pericarditis are discussed in the Complications of Pericarditis section.

Laboratory Findings

Laboratory testing for acute pericarditis is fairly nonspecific and provides little guidance in determining a cause. White blood cell count, erythrocyte sedimentation rate, and serum C-reactive protein level are commonly elevated in acute pericarditis no matter what the cause. Patients with idiopathic pericarditis likely have a viral infection, but viral cultures and antibody titers are not clinically useful and would not alter management.[1] Patients with uremic pericarditis almost always have a blood urea nitrogen level over 60 mg/dL,[8] and leukocytosis is often mild in dialysis-associated pericarditis.[3] If another explanation for pericarditis is suspected, the clinical presentation should direct decisions for further laboratory studies, such as rheumatoid factor, cardiac enzymes, antinuclear antibodies, or sputum samples to assess for mycobacteria.[1,9]

Chest Radiograph

A chest radiograph is obtained to rule out abnormalities in the mediastinum or lung fields that may be

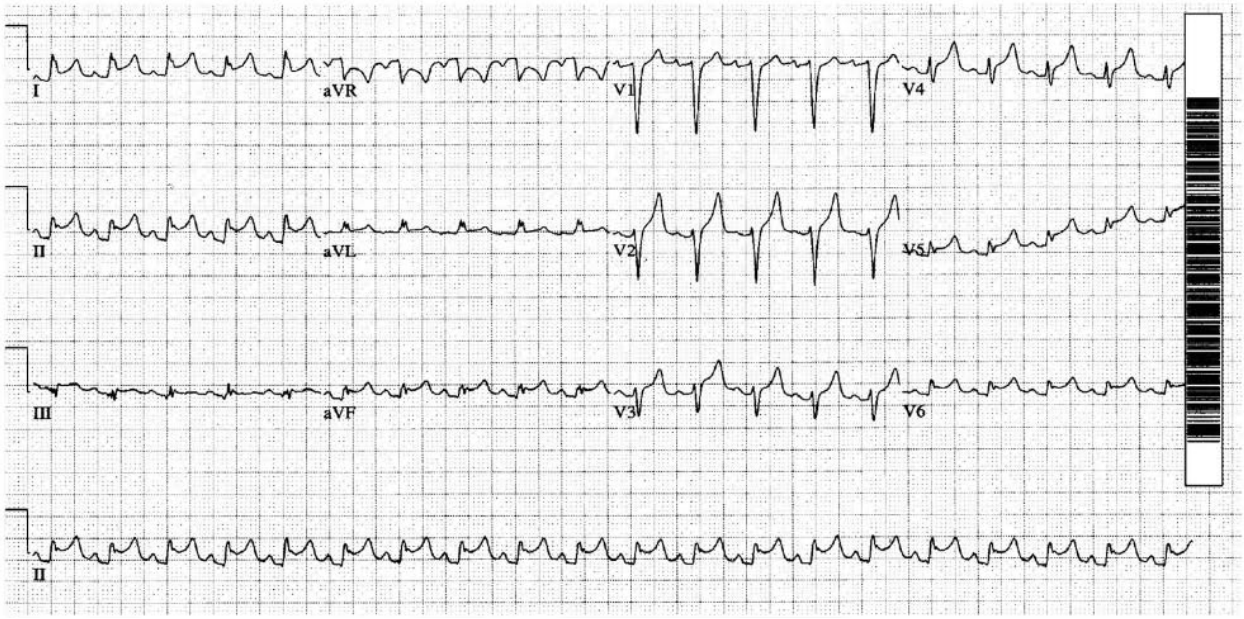

Figure 2. Electrocardiogram showing diffuse ST-segment elevations and PR-segment depression (best seen in lead II) in a patient with end-stage renal disease and acute pericarditis.

the cause of pericarditis, such as malignancy and infection, and is helpful in assessing for possible pericardial sequelae, such as effusion or constriction. Cardiomegaly on chest radiography is nonspecific and may represent left ventricular hypertrophy or a pericardial effusion. More than 250 mL of fluid, the amount required to enlarge the cardiac outline, must be present for a pericardial effusion to appear on a chest radiograph (**Figure 1**).[1,4] The chest radiograph is typically abnormal in patients with ESRD who have pericardial involvement, with cardiomegaly and an abnormal cardiac silhouette being frequently reported.[3]

Table 2. Classic 4-Stage Electrocardiography Changes in Acute Pericarditis

Stage*	ST segment	T wave	PR segment
I	Elevated	Upright	Depressed or isoelectric
II	Isoelectric	Upright to flat	Isoelectric or depressed
III	Isoelectric	Inverted	Isoelectric or depressed
IV	Isoelectric	Upright	Isoelectric

*Stages I and II develop within days, while stages III and IV take weeks to develop.

Electrocardiography

The classic electrocardiographic findings in a patient with acute pericarditis consist of diffuse upright, concave (saddle-shaped) ST-segment elevation and PR-segment depression (**Figure 2**). Four phases of electrocardiographic abnormalities have been described: ST-segment elevation, upright T waves, and PR-segment depression (stage I); normalization of these changes (stage II); widespread T-wave inversions (stage III); and normalization of the T waves (stage IV) (**Table 2**).[8]

The electrocardiogram (ECG) of patients with a myocardial infarction also may demonstrate ST-segment elevations, but several features differentiate the 2 conditions (**Table 3**). In myocardial infarction, the ST-segment elevations are localized rather than diffuse; they are often convex (dome-shaped) rather than concave; Q waves and loss of R-wave voltage frequently occur; T-wave inversions appear before ST segments normalize; PR-segment depression is rare; and atrioventricular block or ventricular arrhythmias are common.[1] The most reliable method for distinguishing between pericarditis and infarction, however, is by calculating the ratio of the height of the ST-segment elevation (in mm) to the height of the T-wave amplitude (in mm) in lead V_6. A ratio greater than 0.25 strongly suggests acute pericarditis.[1,4]

Although most ESRD patients with pericarditis have an abnormal ECG, few of them are reported to have the classic ECG changes, particularly those with uremic pericarditis.[3,5,8] Instead, nonspecific repolarization abnormalities are frequently observed (44%–69%).[3] In contrast, more than 80% of patients with acute

Table 3. Comparison of Electrocardiography Changes Associated with Acute Pericarditis, Myocardial Infarction, and Early Repolarization

ECG Finding	Acute Pericarditis	Myocardial Infarction	Early Repolarization
ST-segment shape	Concave upward	Convex upward	Concave upward
Q waves	Absent	Present	Absent
Reciprocal ST-segment changes	Absent	Present	Absent
Location of ST-segment elevation	Limb and precordial leads	Area of involved artery	Precordial leads
ST/T ratio in lead V_6	> 0.25	N/A	< 0.25
Loss of R-wave voltage	Absent	Present	Absent
PR-segment depression	Present	Absent	Absent

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ECG = electrocardiogram; N/A = not applicable.

pericarditis will have the classic stage I ECG findings. The reason for this discrepancy is that ST-segment elevations reflect subepicardial myocarditis. In uremic pericarditis without infection, inflammatory cells do not penetrate the myocardium and therefore do not produce the characteristic ST-segment elevations. Thus, when the typical ECG changes are seen in a uremic patient, an alternative cause for pericarditis, such as infection, should be investigated.[8]

Echocardiography

Transthoracic echocardiography (TTE) is useful for detecting a pericardial effusion in patients with suspected pericarditis. TTE is also essential in evaluating for cardiac tamponade, which would indicate the need for either surgical intervention or a pericardiocentesis. However, it is not required for every patient; TTE is not recommended in patients with definitive evidence of pericarditis or in patients who have no poor prognostic factors.[1] Poor prognostic factors include fever, subacute onset over several weeks, immunocompromised state, trauma-associated pericarditis, elevation of cardiac enzymes greater than 2 weeks, and signs of tamponade.[1]

COMPLICATIONS OF PERICARDITIS

Cardiac Tamponade

Cardiac tamponade resulting from a large pericardial effusion is a serious complication of pericarditis. It can lead to hemodynamic compromise and even death, especially in ESRD patients who tend to be more refractory to treatment (**Figure 3**).[3,5,10] Clinical signs and symptoms suggestive of a significant effusion include tachycardia, hypotension, jugular venous distension, muffled heart sounds, dyspnea, orthopnea, and postural dizziness.[1,3] Pulsus paradoxus is another physical sign commonly associated with cardiac tamponade. However, it can be seen occasionally in other conditions, such as constrictive pericarditis, bronchial asthma, acute pulmonary hypertension, and acute myocardial infarction.[11,12] Pulsus paradoxus is an exaggeration of the normal decrease in systolic blood pressure during inspiration and is formally defined as an inspiratory decrease in systolic blood pressure greater than 10 mm Hg. Normally, the systolic blood pressure varies with the respiratory cycle, but not to the extent seen in pulsus paradoxus. The proposed mechanism of pulsus paradoxus is described as follows: During inspiration, the right ventricle distends due to increased venous return. The right ventricular distention causes the interventricular septum to bulge into the left ventricle, decreasing the capacity for left ventricular filling and causing a pooling of blood into the pulmonary vessels, which in turn results in a decrease in the left ventricular stroke volume. This fall in stroke volume manifests as an exaggerated decrease in the systolic blood pressure.[11,12]

To measure pulsus paradoxus, the sphygmomanometer cuff should be inflated to 20 mm Hg above the systolic blood pressure and then deflated until the first Korotkoff sound is heard, which initially should be heard only in expiration. The cuff is then deflated until Korotkoff sounds are heard during both inspiration and expiration. The pressure at which Korotkoff sounds are heard throughout the respiratory cycle should be subtracted from the pressure at which the first Korotkoff sound is heard. A difference exceeding 10 mm Hg indicates that the patient has pulsus paradoxus.

Constrictive Pericarditis

Constrictive pericarditis, defined as chronic fibrous thickening and/or calcification of the pericardial sac, is another possible complication of pericarditis (**Figure 4**).

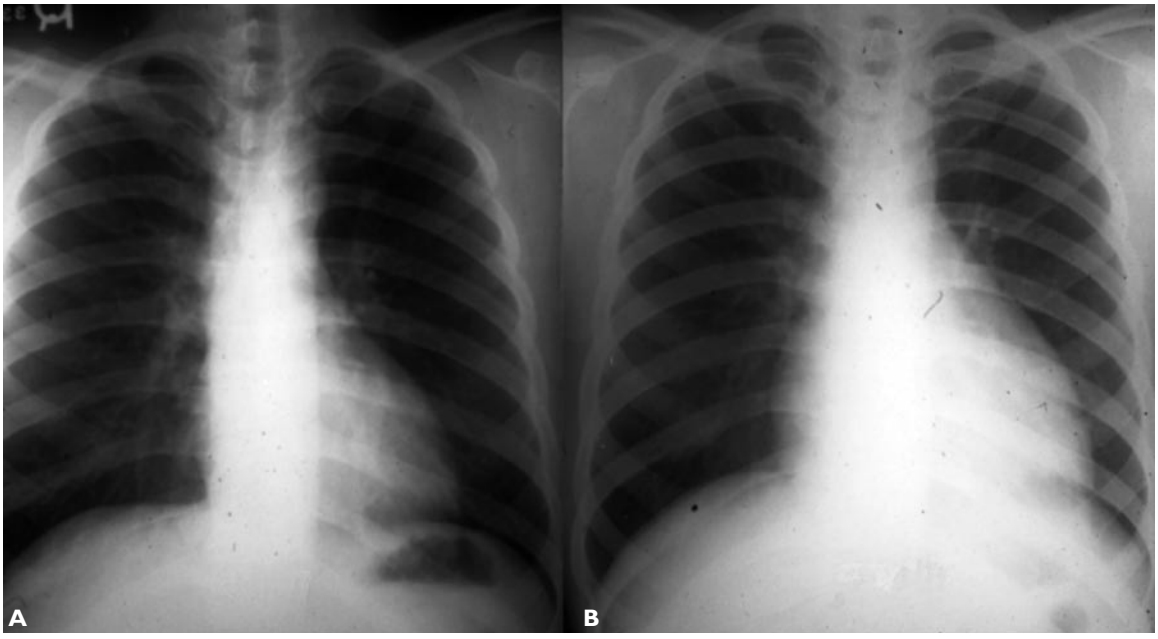

Figure 3. (**A**) Normal chest radiograph of a patient with end-stage renal disease. (**B**) Chest radiograph of the same patient with acute pericarditis and pericardial effusion. (Chest radiographs courtesy of Dr. Melvin H. Schreiber, Department of Radiology, University of Texas Medical Branch at Galveston.)

It results in impaired diastolic filling due to a reduction in the pericardium's compliance. In its early stages, constrictive pericarditis can present with vague clinical manifestations (eg, fatigue, decreased exercise tolerance), which makes diagnosis based on history alone difficult.[13] However, in the advanced stages of disease, the classic presentation includes signs and symptoms consistent with right-sided heart failure, such as dyspnea, edema, and elevated jugular venous pressure.[13]

Kussmaul's sign, distention of the jugular veins on inspiration, also may be present, but it is nonspecific for constrictive pericarditis and may be seen in patients with right ventricular failure, right ventricular infarction, tricuspid stenosis, and restrictive cardiomyopathy.[11,12] This sign reflects an elevation of jugular venous pressure (JVP) on inspiration rather than the expected decrease in JVP. The increased JVP is caused by the decreased compliance of the right ventricle, which causes a rise in right atrial pressure that is greater than the fall in pleural pressure, ultimately leading to distended neck veins during inspiration.[12]

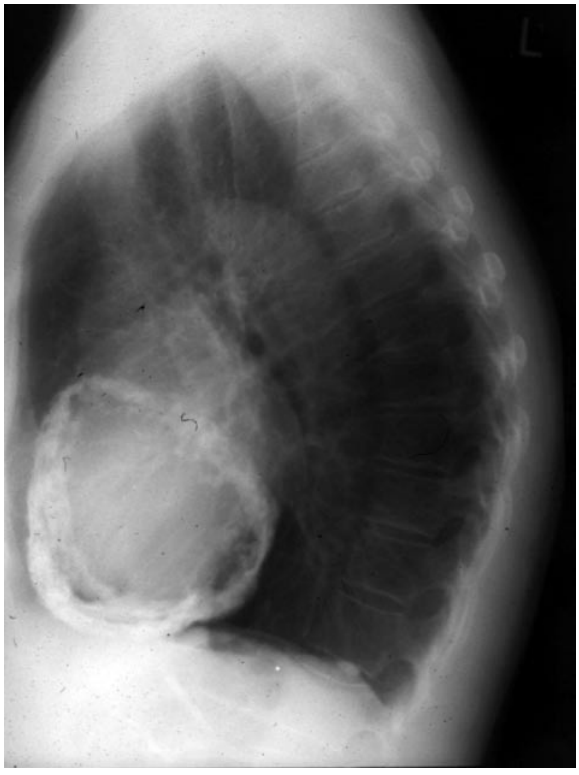

Figure 4. Lateral chest radiograph of a patient with pericardial calcifications, resulting in constrictive pericarditis. (Radiograph courtesy of Dr. Melvin H. Schreiber, Department of Radiology, University of Texas Medical Branch at Galveston.)

TREATMENT

In cases where the etiology of pericarditis has been identified, treatment should be focused on the underlying cause.[1] For patients with idiopathic pericarditis, nonsteroidal anti-inflammatory drugs (NSAIDs) should be used with the goal of relieving chest pain,

inflammation, and fever.[1,4] Aspirin, ibuprofen, and indomethacin are the most commonly prescribed NSAIDs, although ibuprofen is preferred by some experts because it has a lower incidence of side effects than the other medications.[4] Indomethacin is an acceptable alternative, but it should be avoided in patients with coronary artery disease because it reduces coronary blood flow. Aspirin is favored in patients with a recent history of myocardial infarction since other NSAIDs tend to impede scar formation.[1]

CONCLUSION

Acute pericarditis can be caused by many underlying conditions. The most specific sign of acute pericarditis is a pericardial friction rub. Electrocardiographic changes consist of diffuse upright, concave ST-segment elevation and PR-segment depression. Laboratory testing for acute pericarditis is fairly nonspecific as to the etiology. Treatment should be appropriately focused on the underlying cause. For patients with idiopathic pericarditis, NSAIDs are typically effective. Short-term complications include cardiac tamponade, while long-term complications include constrictive pericarditis. **HP**

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