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PERICARTIDIS IS THE MOST COMMON TYPE OF ETHYLOGIC AND MEASURES TO TREAT IT

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Annotation: Pericarditis is inflammation of the pericardium, often with fluid accumulation in the pericardial space. Pericarditis may be caused by many disorders (eg, infection, myocardial infarction, trauma, tumors, metabolic disorders) but is often idiopathic. Symptoms include chest pain or tightness, often worsened by deep breathing. Cardiac output may be greatly reduced if cardiac tamponade or constrictive pericarditis develops. Diagnosis is based on symptoms, a friction rub, electrocardiographic changes, and evidence of pericardial fluid accumulation on x-ray or echocardiogram. Finding the cause requires further evaluation. Treatment depends on the cause, but general measures include analgesics, anti-inflammatory drugs, colchicine, and rarely surgery.

Key words: Pericardium, pericarditis, myocardium, endocardium, blood, heart.

The pericardium has 2 layers. The visceral pericardium is a single layer of mesothelial cells that is attached to the myocardium, folds back (reflects) on itself over the origin of the great vessels, and joins with a tough, fibrous layer to envelop the heart as the parietal pericardium. The sac created by these layers contains a small amount of fluid (< 25 to 50 mL), composed mostly of an ultrafiltrate of plasma. The pericardium limits distention of the cardiac chambers and increases the heart's efficiency.

The pericardium is richly innervated with sympathetic and somatic afferents. Stretch-sensitive mechanoreceptors sense changes in cardiac volume and tension and may be responsible for transmitting pericardial pain. The phrenic nerves are embedded in the parietal pericardium and are vulnerable to injury during surgery on the pericardium.

Pericarditis may be

- Acute
- Subacute
- Chronic

Acute pericarditis develops quickly, causing inflammation of the pericardial sac and often a pericardial effusion. Inflammation can extend to the epicardial myocardium (myopericarditis). Adverse hemodynamic effects and rhythm disturbance are rare, although cardiac tamponade is possible.

Acute disease may resolve completely, resolve and reoccur (up to 30% of acute cases) or become subacute or chronic. These forms develop more slowly; their prominent feature is effusion.

Subacute pericarditis occurs within weeks to months of an inciting event.

Chronic pericarditis is defined as pericarditis persisting > 6 months.

Pericardial effusion is accumulation of fluid in the pericardium. The fluid may be serous fluid (sometimes with fibrin strands), serosanguineous fluid, blood, pus, or chyle.
Cardiac tamponade occurs when a large pericardial effusion impairs cardiac filling, leading to low cardiac output and sometimes shock and death. If fluid (usually blood) accumulates rapidly, even small amounts (eg, 150 mL) may cause tamponade because the pericardium cannot stretch quickly enough to accommodate it. Slow accumulation of up to 1500 mL may not cause tamponade. Loculated effusion may cause localized tamponade on the right or left side of the heart.
Occasionally, pericarditis causes a marked thickening and stiffening of the pericardium (constrictive pericarditis).

Constrictive pericarditis, which is now less common than in the past, results from marked inflammatory, fibrotic thickening of the pericardium. Sometimes the visceral and parietal layers adhere to each other or to the myocardium. The fibrotic tissue often contains calcium deposits. The stiff, thickened pericardium markedly impairs ventricular filling, decreasing stroke volume and cardiac output. Significant pericardial fluid accumulation is rare. Rhythm disturbance is common. The diastolic pressures in the ventricles, atria, and venous beds become virtually the same. Systemic venous congestion occurs, causing considerable transudation of fluid from systemic capillaries, with dependent edema and, later, ascites. Chronic elevation of systemic venous pressure and hepatic venous pressure may lead to liver scarring, called cardiac cirrhosis, in which case, patients may initially present for evaluation of cirrhosis. Constriction of the left atrium, the left ventricle, or both may elevate pulmonary venous pressure. Occasionally, pleural effusion develops.
There are several variants of constrictive pericarditis:

- Chronic constrictive pericarditis, usually requiring pericardiectomy as definitive treatment
- Subacute (early stage) constrictive pericarditis,developing weeks to months after an inciting injury and managed initially with medical therapy
- Transient constrictive pericarditis (typically subacute), is that which resolves spontaneously or after medical therapy
- Effusive-constrictive pericarditis characterized by pericardial constriction involving the visceral pericardium with significant pericardial effusion sometimes requiring treatment for cardiac tamponade

Acute pericarditis may result from infection, autoimmune or inflammatory disorders, uremia, trauma, myocardial infarction (MI), cancer, radiation therapy, or certain drugs (see table Causes of Acute Pericarditis).
Infectious pericarditis is most often viral or idiopathic. Purulent bacterial pericarditis is uncommon but may follow infective endocarditis, pneumonia, septicemia, penetrating trauma, or cardiac surgery. Often, the cause cannot be identified (called nonspecific or idiopathic pericarditis), but many of these cases are probably viral.
Acute myocardial infarction causes 10 to 15% of cases of acute pericarditis. Post-myocardial infarction syndrome (Dressler syndrome) is a less common cause now, occurring mainly when reperfusion with percutaneous transluminal coronary angioplasty (PTCA) or thrombolytic

drugs is ineffective in patients with transmural infarction. Pericarditis occurs after pericardiotomy (called postpericardiotomy syndrome) in 5 to 30% of cardiac operations. Postpericardiotomy syndrome, post-MI syndrome, and traumatic pericarditis comprise the post-cardiac injury syndrome.

Subacute pericarditis is a prolongation of acute pericarditis and thus has the same causes. Some patients have transient constriction occurring days to weeks after recovery from acute pericarditis.
Chronic pericarditis with pericardial effusion or chronic constrictive pericarditis may follow acute pericarditis of almost any etiology. In addition, some cases occur without antecedent acute pericarditis.
Chronic pericarditis with large effusion (serous, serosanguineous, or bloody) is most commonly caused by metastatic tumors, most often by lung carcinoma, breast carcinoma, sarcoma, melanoma, leukemia, or lymphoma.
Hypothyroidism may cause pericardial effusion and cholesterol pericarditis. Cholesterol pericarditis is a rare disorder that may be associated with myxedema, in which a chronic pericardial effusion has a high level of cholesterol that triggers inflammation and pericarditis.
Sometimes no cause of chronic pericarditis is identified.

Transient constrictive pericarditis is most commonly caused by infection or postpericardiotomy inflammation or is idiopathic.
Fibrosis of the pericardium, sometimes leading to chronic constrictive pericarditis, may follow purulent pericarditis or accompany a connective tissue disorder. In older patients, common causes are malignant tumors, MI, and tuberculosis. Hemopericardium (accumulation of blood within the pericardium) may lead to pericarditis or pericardial fibrosis; common causes include chest trauma, iatrogenic injury (eg, resulting from cardiac catheterization, pacemaker insertion, central venous line placement), and rupture of a thoracic aortic aneurysm.
Some patients present with symptoms and signs of inflammation (acute pericarditis); others present with those of fluid accumulation (pericardial effusion) or constriction. Symptoms and signs vary depending on the severity of inflammation and the amount and rate of fluid accumulation. Even a large amount of pericardial fluid may be asymptomatic if it develops slowly (eg, over months).

Acute pericarditis
Acute pericarditis tends to cause chest pain, fever, and a pericardial rub, sometimes with dyspnea. The first evidence can be tamponade, with hypotension, shock, or pulmonary edema.

Because the innervation of the pericardium and myocardium is the same, the chest pain of pericarditis is sometimes similar to that of myocardial inflammation or ischemia: Dull or sharp precordial or substernal pain may radiate to the neck, trapezius ridge (especially the left), or shoulders. Pain ranges from mild to severe. Unlike ischemic chest pain, pain due to pericarditis is usually aggravated by thoracic motion, cough, breathing, or swallowing food; it may be relieved by sitting up and leaning forward.

Tachypnea and nonproductive cough may be present; fever, chills, and weakness are common. In 15 to 25% of patients with idiopathic pericarditis, symptoms recur intermittently for months or years (recurrent pericarditis).

The most important physical finding is a triphasic or a systolic and diastolic precordial friction rub. However, the rub is often intermittent and evanescent; it may be present only during systole or, less frequently, only during diastole. If no rub is heard with the patient seated and leaning forward, auscultation may be attempted by listening with the diaphragm of the stethoscope while with the patient is on all fours. Sometimes, a pleural component to the rub is noted during breathing, which is due to inflammation of the pleura adjacent to the pericardium.

Pericardial effusion

Pericardial effusion is often painless, but when it occurs with acute pericarditis, pain may be present. Considerable amounts of pericardial fluid may muffle heart sounds, increase the area of cardiac dullness, and change the size and shape of the cardiac silhouette. A pericardial rub may be heard. With large effusions, compression of the base of the left lung can decrease breath sounds (heard near the left scapula) and cause crackles. Arterial pulse, jugular venous pulse, and blood pressure are normal unless intrapericardial pressure increases substantially, causing tamponade.

In the post-MI syndrome, pericardial effusion can occur with fever, friction rub, pleurisy, pleural effusions, and joint pain. This syndrome usually occurs within 10 days to 2 months after MI. It is usually mild but may be severe. Occasionally, the heart ruptures post-MI, causing hemopericardium and tamponade, usually 1 to 10 days post-MI and more commonly in women.

Cardiac tamponade

(See also Cardiac tamponade due to trauma.)

The clinical findings are similar to those of cardiogenic shock: decreased cardiac output, low systemic arterial pressure, tachycardia, and dyspnea. Neck veins are markedly dilated. Severe cardiac tamponade is nearly always accompanied by a fall of > 10 mm Hg in systolic blood pressure during inspiration (pulsus paradoxus). In advanced cases, pulse may disappear during inspiration. (However, pulsus paradoxus can also occur in chronic obstructive pulmonary disease [COPD], bronchial asthma, pulmonary embolism, right ventricular infarction, and noncardiogenic shock.) Heart sounds are muffled unless the effusion is small. Loculated effusions and eccentric or localized hematoma may cause localized tamponade, in which only selected cardiac chambers are compressed. In these cases, physical, hemodynamic, and some echocardiographic signs may be absent.

Constrictive pericarditis

Fibrosis or calcification rarely causes symptoms unless constrictive pericarditis develops. The only early abnormalities may be elevated ventricular diastolic, atrial, pulmonary, and systemic venous pressures. Symptoms and signs of peripheral venous congestion (eg, peripheral edema, neck vein distention, hepatomegaly) may appear with an early diastolic sound (pericardial knock), often best heard during inspiration. This sound is due to abrupt slowing of diastolic ventricular filling by the rigid pericardium.

- Ventricular systolic function (based on ejection fraction) is usually preserved. Prolonged elevation of pulmonary venous pressure results in dyspnea (particularly during exertion) and orthopnea. Fatigue may be severe. Distention of neck veins with a rise in venous pressure during inspiration (Kussmaul sign) is present; it is absent in tamponade. Pulsus paradoxus is rare and is usually less severe than in tamponade. Lungs are not congested

unless severe left ventricular constriction develops. Electrocardiography (ECG) and chest x-ray

- Echocardiography
- Tests to identify cause (eg, pericardial fluid aspiration, pericardial biopsy)

ECG and chest x-ray are done. Echocardiography is done to check for effusion, cardiac filling abnormalities that may suggest cardiac tamponade, and wall motion abnormalities characteristic of myocardial involvement. Blood tests may detect leukocytosis and elevated markers of inflammation (eg, C-reactive protein, erythrocyte sedimentation rate), which may be used to guide duration of therapy.

Acute pericarditis

The diagnosis is based on the presence of the following clinical findings and ECG abnormalities, which are not always present in all cases.

- Characteristic chest pain
- Pericardial rub
- ECG abnormalities
- Pericardial effusion

Serial ECGs may be needed to show abnormalities. The ECG in acute pericarditis may show abnormalities confined to ST and PR segments and T waves, usually in most leads. (ECG changes in lead aVR are generally in the opposite direction of other leads.) Unlike MI, acute pericarditis does not cause reciprocal depression in ST segments (except in leads aVR and V1), and there are no pathologic Q waves. ECG changes in pericarditis can occur in 4 stages although not all stages are present in all cases.

- Stage 1: ST segments show upward concave elevation; the PR segments may be depressed (see figure Acute pericarditis: Stage 1 ECG).
- Stage 2: ST segments return to baseline; T waves flatten.
- Stage 3: T waves are inverted throughout the ECG; T wave–inversion occurs after the ST segment has returned to baseline and thus differs from the pattern of acute ischemia or MI.
- Stage 4: T wave changes resolve.

Echocardiography in acute pericarditis typically shows an effusion, which helps confirm the diagnosis, except in patients with purely fibrinous acute pericarditis in whom echocardiography is often normal. Findings indicating myocardial involvement include new focal or diffuse left ventricular dysfunction.

MRI can detect the presence, severity, and acuity of pericardial inflammation but is generally not required to diagnose acute pericarditis.

Because the pain of pericarditis may resemble that of acute MI or pulmonary infarction, additional tests (eg, serum cardiac marker measurement, lung scan) may be required if the history and ECG findings are atypical for pericarditis. Troponin is often elevated in acute pericarditis due to epicardial inflammation, so it cannot discriminate between pericarditis, acute infarction, and pulmonary embolism. Very high levels of troponin may indicate myopericarditis. The CK-MB (creatine kinase muscle band isoenzyme) level, which is less sensitive than the troponin level, is usually normal in acute pericarditis unless myocarditis is also present.

Postpericardiotomy and post-MI syndromes may be difficult to identify and must be distinguished from recent MI, pulmonary embolism, and pericardial infection after surgery. Pain, friction rub, and fever appearing 2 weeks to several months after surgery and a rapid response to aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, or corticosteroids aids diagnosis.

Pericardial effusion

Diagnosis is suggested by clinical findings but often is suspected only after finding an enlarged cardiac silhouette on chest x-ray. On ECG, QRS voltage is often decreased, and sinus rhythm remains in about 90% of patients. With large, chronic effusions, the ECG may show electrical alternans (ie, P, QRS, or T wave amplitude increases and decreases on alternate beats). Electrical alternans is associated with variation in cardiac position (swinging heart).

Echocardiography estimates the volume of pericardial fluid; identifies cardiac tamponade, sometimes acute myocarditis, and/or heart failure; and may suggest the cause of pericarditis.

Although CT can detect a pericardial effusion (often incidentally on a scan done for other conditions), it may overestimate its size and is not a first-line test to evaluate possible pericardial effusion.

Patients with a normal ECG, small (< 50 mL) effusion, and no suspicious findings from the history and examination may be observed with serial examination and echocardiography. Other patients must be evaluated further to determine etiology.

Constrictive pericarditis

Diagnosis may be suspected based on clinical, ECG, chest x-ray, and Doppler echocardiography findings, but cardiac catheterization and CT (or MRI) are usually required. Rarely, right heart biopsy is needed to exclude restrictive cardiomyopathy.

ECG changes are nonspecific. QRS voltage is usually low. T waves are usually nonspecifically abnormal. Atrial fibrillation occurs in about one third of patients; atrial flutter is less common.

Lateral chest x-ray often shows pericardial calcification best, but the finding is nonspecific.

Echocardiography also is nonspecific. When the right and left ventricular filling pressures are equally elevated, Doppler echocardiography helps distinguish constrictive pericarditis from restrictive cardiomyopathy.

- During inspiration, mitral diastolic flow velocity usually falls > 25% in constrictive pericarditis but < 15% in restrictive cardiomyopathy.

- In constrictive pericarditis, inspiratory tricuspid flow velocity increases more than it normally does, but it does not do so in restrictive cardiomyopathy.

Determining tissue velocities at the mitral annulus may be helpful when excessively high left atrial pressure blunts respiratory variation in transvalvular velocities. Mitral annular velocities (especially at the septal location) increase in constrictive pericarditis; they decrease in restrictive cardiomyopathy.

Presence of a septal bounce (shift of the interventricular septum towards the left ventricle during inspiration and away from the left ventricle during expiration) and hepatic vein expiratory diastolic flow reversal (which occurs due to reduced filling of the right ventricle) can also be visible in constrictive pericarditis.

Respiration-related ventricular septal shift, preserved or increased medial annular velocity, and hepatic vein expiratory diastolic flow reversal collectively are referred to as the Mayo criteria, but each factor is independently associated with constrictive pericarditis (1).
Cardiac catheterization, right and left sided, is done if clinical and echocardiographic findings suggest constrictive pericarditis. Cardiac catheterization helps confirm and quantify the abnormal hemodynamics that define constrictive pericarditis:

- Mean pulmonary artery occlusion pressure (pulmonary capillary wedge pressure), pulmonary artery diastolic pressure, right ventricular end-diastolic pressure, and mean right atrial pressure are roughly equal, all at about 10 to 30 mm Hg.
- The pulmonary artery and right ventricular systolic pressures are normal or modestly elevated, so that pulse pressures are small.
- In the atrial pressure curve, *x* and *y* descents are typically accentuated.
- In the ventricular pressure curve, a diastolic dip occurs at the time of rapid ventricular filling.
- During peak inspiration, right ventricular pressure increases when left ventricular pressure is lowest (sometimes called mirror-image discordance, suggesting increased ventricular interdependence).
- Because ventricular filling is restricted, ventricular pressure tracings show a sudden dip followed by a plateau (resembling a square root sign) in early diastole.

Measuring these changes requires simultaneous right and left heart cardiac catheterization, using separate transducers. These hemodynamic changes almost always occur with significant constrictive pericarditis but may be masked during hypovolemia.

Right ventricular systolic pressure of > 50 mm Hg often occurs in restrictive cardiomyopathy but less often in constrictive pericarditis. When the pulmonary artery occlusion pressure equals the right atrial mean pressure and an early diastolic dip in the ventricular pressure curve occurs with large *x* and *y* waves in the right atrial curve, either disorder may be present.

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