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# Modern Solutions for the Rational Treatment of Pericarditis

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**Abstract:** Pericarditis is inflammation of the pericardium, often with fluid accumulation in the pericardial cavity. Pericarditis can occur for a variety of reasons (e.g., infectious diseases, myocardial infarction, trauma, tumors, metabolic disorders), but is most often idiopathic. Symptoms of pericarditis include chest pain or tightness, often worsened by deep breathing. If cardiac tamponade or constrictive pericarditis develops, cardiac output may be significantly reduced. Diagnosis is based on symptoms, auscultatory findings (pericardial friction rub), electrocardiographic changes, and the presence of fluid accumulation in the pericardial cavity on radiography or echocardiography. Further investigation is required to determine the cause. Treatment depends on the cause, but usually includes analgesics, anti-inflammatory drugs, colchicine, and occasionally surgery.

**Keywords:** Anatomy, Pathophysiology, Etiology, Clinical Manifestations, Diagnostics, Treatment.

**Introduction:** The pericardium consists of 2 layers ( 1 ). The visceral pericardium is a layer of mesothelial cells adjacent to the myocardium and is connected to the parietal pericardium, a dense fibrous layer that extends to the bases of the great vessels and surrounds the heart. The space formed by these sheets contains a small amount of fluid ( < 25-50 ml), consisting mainly of plasma ultrafiltrate. The pericardium limits the acute distension of the heart chambers and enhances the mechanical interaction of the heart chambers.

The pericardium is richly innervated by sympathetic and somatic afferent fibers. Stretch-sensitive mechanoreceptors sense changes in cardiac volume and pressure and may be responsible for the transmission of pericardial pain. The phrenic nerves run in the parietal pericardium and may be damaged during pericardial surgery.

Acute pericarditis develops rapidly, causing inflammation of the pericardial sac and often pericardial effusion. The inflammation may extend to the epicardial myocardium (myopericarditis). Adverse effects on hemodynamics and arrhythmias are rare, but cardiac tamponade may occur.

The acute disease may be completely remitting, relapsing and remitting (up to 30% of acute cases), or subacute or chronic ( 1 ). These forms develop more slowly.

Subacute pericarditis occurs within weeks or months of the triggering event and may resolve spontaneously or with drug therapy.

Chronic pericarditis is defined as pericarditis that lasts longer than 6 months.

Pericardial effusion is the accumulation of fluid in the pericardial cavity. The fluid may be serous (sometimes with fibrin strands), serous-hemorrhagic, hemorrhagic, purulent, or chylous.

Cardiac tamponade occurs when a moderate to large pericardial effusion impairs the filling of the heart, leading to decreased cardiac output and sometimes shock and death. If fluid (usually blood) accumulates rapidly, even a small amount (such as 150 mL) can cause tamponade because the pericardium cannot stretch quickly. In contrast, a slow accumulation of fluid of up to 1500 mL may not cause tamponade. Focal effusions can cause localized tamponade on the right or left side of the heart and may be difficult to diagnose.

Sometimes pericarditis can cause significant thickening and hardening of the pericardium (constrictive pericarditis).

Constrictive pericarditis, now much less common than in the past, is caused by severe inflammation and fibrous thickening of the pericardium. Sometimes there is adhesion of the visceral and parietal layers to each other or to the myocardium. The fibrous tissue often contains calcium deposits. The stiff, thickened pericardium significantly impairs ventricular filling, reducing stroke volume and cardiac output. Significant accumulation of pericardial fluid is rare. Rhythm disturbances, especially atrial fibrillation, are often observed. The diastolic pressure in the ventricles, atria, and venous bed is almost the same. Systemic venous congestion leads to significant transudation of fluid from the systemic capillaries with the development of edema and ascites. Prolonged elevation of systemic and hepatic venous pressure can lead to scarring of the liver tissue, which is called cardiac cirrhosis, in which case patients may initially be diagnosed with cirrhosis. Compression of the left atrium, left ventricle, or both can lead to increased pressure in the pulmonary veins. Sometimes a pleural effusion forms.

a. There are several types of constrictive pericarditis:

b. Chronic constrictive pericarditis usually requires pericardiectomy as definitive treatment

c. Subacute (early stage) constrictive pericarditis develops weeks to months after the inciting injury and is initially treated conservatively.

d. Transient constrictive pericarditis (usually subacute) that resolves spontaneously or after conservative treatment.

e. Effusional constrictive pericarditis, characterized by compression of the heart by the visceral pericardium and the presence of significant pericardial effusion, sometimes requires the same treatment as cardiac tamponade.

**Research methods and materials:** Acute pericarditis can be caused by infection, autoimmune or inflammatory diseases, uremia, trauma, myocardial infarction (MI), cancer, radiation therapy, or certain drugs (see table of causes of acute pericarditis).

Infectious pericarditis is often viral or idiopathic (often presumed to be viral). Bacterial pericarditis caused by pyogenic organisms is rare but may occur as a result of infective endocarditis, pneumonia, septicemia, penetrating trauma, or cardiac surgery. Often the cause of the disease cannot be determined (nonspecific or idiopathic pericarditis), but in most cases it is probably a viral pathology.

Acute myocardial infarction accounts for 7–12% of acute pericarditis cases ( 1 ). Post-myocardial infarction syndrome (Dressler syndrome) is a less common cause, occurring primarily in patients with transmural infarction who have failed percutaneous transluminal coronary angioplasty (PTCA) or reperfusion with thrombolytic drugs ( 2 ). Pericarditis develops after pericardiotomy (termed postpericardiotomy syndrome) in 5–30% of cardiac surgeries ( 3 ). Postpericardiotomy syndrome, postinfarction syndrome, and traumatic pericarditis (which includes iatrogenic pericarditis, such as after percutaneous transluminal cardiac intervention, pacemaker implantation, and ablation) constitute the “postcardiac injury syndrome.”

**Conclusions** : Subacute pericarditis is a sequela of acute pericarditis and therefore has the same causes. Some patients develop a relapse days or weeks after recovery from acute pericarditis.

Chronic pericarditis, or chronic constrictive pericarditis, can occur after acute pericarditis of almost any etiology. In more resource-rich settings, the most common precursors to constrictive pericarditis are idiopathic/viral diseases, previous cardiac surgery, and a history of radiation therapy; the most common cause worldwide is tuberculous pericarditis ( 4 ). In addition, in some cases, it precedes acute pericarditis.

Chronic pericarditis with a large amount of effusion (serous, serosanguinous, or bloody) is often caused by tumor metastases ( 5 ), most commonly lung carcinoma, breast cancer, sarcoma, melanoma, leukemia, or lymphoma.

Hypothyroidism can cause pericardial effusion and cholesterol pericarditis. Cholesterol pericarditis is a rare condition that may be associated with myxedema, in which there is chronic pericardial effusion with high cholesterol levels, which causes inflammation and pericarditis.

Transient constrictive pericarditis often results from infection or inflammation following pericardiotomy, or is idiopathic.

Pericardial fibrosis, sometimes leading to chronic constrictive pericarditis, may develop after purulent pericarditis or as a result of systemic rheumatic disease. Common causes in elderly patients include malignant neoplasms, myocardial infarction, and tuberculosis. Hemopericardium (accumulation of blood in the pericardial cavity) may lead to pericarditis or pericardial fibrosis; common causes include chest trauma, iatrogenic injury (eg, due to cardiac catheterization, pacemaker implantation, central venous catheter placement), or ruptured thoracic aortic aneurysm.

Some patients have signs and symptoms of inflammation (acute pericarditis), while others have fluid accumulation (pleural effusion) or signs of compression.

Symptoms vary depending on the severity of the inflammation and the amount of pericardial fluid. Even large amounts of pericardial fluid may be asymptomatic if they accumulate slowly (for example, over months).

Acute pericarditis can cause chest pain, fever, and pericardial effusion, and sometimes shortness of breath. Sometimes the first manifestation may be hypotension, shock, or tamponade with the development of pulmonary edema.

Because the pericardium and myocardium share the same innervation, chest pain in pericarditis can be similar to that in myocardial inflammation or ischemia: dull or sharp precordial or retrosternal pain may radiate to the neck, trapezius muscles (especially on the left), or shoulders. The pain varies from mild to severe. The pain may be relieved by sitting and leaning forward. Unlike ischemic pain, pericarditis pain is usually aggravated by chest movement, coughing, breathing, or swallowing.

Tachypnea and a nonproductive cough may be present, often accompanied by fever, chills, and weakness. In 15–25% of patients with idiopathic pericarditis, symptoms may recur periodically over months or years (recurrent pericarditis).

The most important finding on physical examination is a triphasic or systolic and diastolic precordial pericardial rub. This murmur is often intermittent and disappears quickly; it may be heard only during systole or, less commonly, only during diastole. A pleural rub is sometimes heard, which is caused by inflammation of the area adjacent to the pericardium.

**Discussion** : Pericardial effusion is often painless, but may be painful in acute pericarditis. Large amounts of pericardial fluid may muffle heart sounds, increase the area of cardiac dullness, and change the size and shape of the cardiac silhouette. Pericardial rubs may be heard. With large effusions, compression of the lower lobe of the left lung may cause shortness of breath (heard near the left scapula) and wheezing. Arterial pulse, jugular venous pulse, and arterial pressure are within normal limits, except in cases where intrapericardial pressure is significantly elevated, leading to tamponade.

In post-infarction syndrome, pericardial effusion may be accompanied by fever, friction rub, pleurisy, pleural effusion, and joint pain. This syndrome usually occurs 10 days to 2 months after myocardial infarction. It is usually mild but can be severe. Rarely, usually between 1 and 10 days after the heart attack, more often in women, rupture of the heart muscle can develop, leading to hemopericardium and tamponade.

The clinical manifestations of cardiac tamponade are similar to cardiogenic shock: decreased cardiac output, low systemic arterial pressure, tachycardia, and shortness of breath. The jugular veins are markedly dilated. Severe cardiac tamponade is almost always accompanied by a decrease of > 10 mm Hg. Art. systolic blood pressure during inspiration ( pulsus paradoxus ). In severe cases, the heart rate may disappear during inspiration. However, a paradoxical pulsus can also be detected in chronic obstructive pulmonary disease (COPD), bronchial asthma, pulmonary embolism, right ventricular infarction, and shock. Heart sounds are muffled until the effusion stops. Encapsulated effusion and eccentric or local hematoma can lead to local tamponade, in which only some chambers of the heart are compressed. In such cases, physical, hemodynamic, and some echocardiographic signs may be absent.

Fibrosis or calcification rarely causes symptoms until constrictive pericarditis develops. The only early sign may be an increase in ventricular diastolic pressure, atrial pressure, pulmonary, and systemic venous pressure. The clinical picture of peripheral venous congestion (peripheral edema, dilated jugular veins, hepatomegaly) may be accompanied by an early diastolic extra sound (pericardial click), which is most pronounced during inspiration. This sound is caused by a sharp slowdown in diastolic filling of the ventricles due to a stiff pericardium. Ventricular systolic function is usually preserved. Prolonged increased pressure in the pulmonary veins leads to shortness of breath (especially during physical exertion) and orthopnea. Fatigue is expressed. There is dilatation of the jugular veins (Kussmaul's sign) with increased venous pressure during respiration, which is absent in tamponade. Pulsus paradoxus is rare and usually less pronounced than in tamponade. There is no pulmonary congestion until severe left ventricular dysfunction develops.

**Conclusion** : ECG and chest X-ray are performed. Echocardiography is performed to detect cardiac filling abnormalities that may indicate effusion, cardiac tamponade, and wall motion abnormalities that are characteristic of myocardial injury. Blood tests may reveal leukocytosis and elevated inflammatory markers (e.g., C-reactive protein, erythrocyte sedimentation rate), which can be used to guide the duration of therapy.

Repeated ECG recordings are necessary to detect changes. In acute pericarditis, ECG changes may be limited to the ST and PR segments and the T wave, usually in most leads. (ECG changes in lead aVR are usually opposite to changes in other leads.) Unlike myocardial infarction, acute pericarditis does not cause reciprocal depression of the ST segments (except in leads aVR and V1), and pathological Q wave changes in the ECG in pericarditis may, although not always, occur in phase 4.

Stage 1: ST segments are elevated, concave, and high; PR segment depression may be present (see Acute Pericarditis: Stage 1 ECG).

Phase 2: ST segments return to their original position; T wave flattens.

Stage 3: T wave inversion in all directions; T wave inversion occurs after the ST segment has returned to baseline and is therefore different from the changes observed in acute myocardial ischemia or infarction.

Stage 4: T wave returns to normal

The typical diagnostic feature of acute pericarditis is effusion, except in patients with purely fibrinous pericarditis, in whom echocardiography is often normal. Findings suggestive of myocardial injury include new focal or diffuse signs of left ventricular dysfunction.

Cardiac MRI can determine the presence, severity, and extent of pericardial inflammation but is not usually necessary to diagnose acute pericarditis. MRI can detect extension of inflammation into the underlying myocardium (myopericarditis), which occurs in 15–24% of cases. Unlike perimyocarditis (with isolated or predominant myocardial involvement), there is no known long-term adverse outcome ( 2 ). Because the pain of pericarditis can mimic that of myocardial infarction or pulmonary infarction, additional investigations (eg, signs of myocardial injury, lung scan) may be required if the history and ECG are atypical for pericarditis. Troponin levels are often elevated in acute pericarditis due to epicardial inflammation and therefore cannot differentiate between pericarditis, acute infarction, and pulmonary embolism. Very high troponin levels may indicate myopericarditis.

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