

The pericardium provides a physical barrier for neoplastic or infectious processes, limits acute myocardial distension, and likely aids diastolic coupling of the ventricles. Normally lubricated with a small amount of lymphatic fluid, the pericardial space includes the cardiac mass and extends to enclose the proximal great vessels (ascending aorta to transverse aortic arch and PAs just beyond the bifurcation), the SVC proximal to the azygous vein, the proximal aspect of the IVC, and the proximal pulmonary veins. The pericardium is susceptible to infectious and autoimmune-mediated inflammation from a wide variety of etiologies and there can also be associated myocardial irritation and/or inflammation. Pericardial inflammation can result in fluid accumulating in the pericardial space, resulting in a pericardial effusion.

Accumulation of pericardial fluid can increase the pressure within the pericardial sac, which is insignificant in normal conditions. The pericardial sac can slowly distend over prolonged periods of time to accommodate chronic fluid buildup, but with acute accumulation, the pericardial sac may act as a noncompliant structure resulting in rapid rise of the pericardial pressures. In the early stages, rising pericardial pressure can intermittently exceed intracardiac pressures during cardiac-chamber pressure nadirs of the cardiac cycle, resulting in transient chamber collapse. In true tamponade, elevated pericardial pressures ultimately overcome and equalize all intracardiac chamber pressures, precipitating cardiovascular collapse.

Clinical Presentation

Pericarditis typically presents with insidious onset of substernal chest pain, which can radiate to the left upper back. Classically, the pain is worsened by lying flat and can be alleviated with elevation in bed or leaning forward. Fever and sinus tachycardia are common on presentation; fever severity and patterns are often related to etiology (see below) and can aid in differential diagnosis for the underlying process. A friction rub can be present on physical examination and distant heart sounds should raise suspicion for pericardial effusion.

Diagnosis

- **ECG.** Diffuse ST-segment elevations with associated PR depressions are often seen. Typically, the ST-segment changes are not segmental, which, if present, should raise concern for coronary artery disease, especially if they follow a coronary distribution pattern and there is an underlying risk factor for early coronary artery disease.
- **Labs.** Cardiac enzymes can be mildly elevated due to direct irritation or they can be markedly elevated if there is associated myocarditis.
- **CXR.** Cardiomegaly may or may not be present depending if there is associated pericardial effusion.
- **Echocardiography.** Echocardiography can identify an associated pericardial

effusion, but assessment for pericardial inflammation or “echo-brightness” is unreliable.

Pericardial Effusion and Tamponade

Depending on the etiology, pericarditis can be associated with a pericardial effusion, which may or may not be hemodynamically significant. Patients may present with tachycardia and dyspnea in cases of tamponade or impending tamponade, but an abnormal CXR with enlargement of the cardiac silhouette may be the first concerning clinical sign.

Even though tamponade is a clinical diagnosis, risk stratification for tamponade or impending hemodynamic compromise can be aided with careful transthoracic 2D and Doppler imaging. One should evaluate for cardiac chamber collapse during specific periods of the cardiac cycle (i.e., RA collapse during ventricular systole and/or RV collapse during ventricular diastole). Doppler imaging can identify excessive respiratory variability of cardiac stroke volume, which can be seen in early tamponade. The most consistently available flow pattern for interrogation of this phenomenon is the mitral valve inflow velocity. A >30% inspiratory decrease of mitral valve A-wave inflow velocity is suggestive of impaired LV filling and early tamponade. Excessive variability of the aortic valve outflow velocity (>10% inspiratory decrease) and tricuspid valve inflow (>50-70% inspiratory increase) can also be used to support the diagnosis. Importantly, however, diagnosis of early tamponade must be made at the bedside based upon clinical factors including history, heart rate trends, respiratory status, BP measurements (preferably via an arterial line), and physical examination. Unexplained tachycardia is usually one of the most sensitive clinical signs of impending tamponade physiology. However, caution should be employed using this sign in isolation, particularly when pathologic sinus node dysfunction could be present, either intrinsic or medication-induced.

Differential Diagnosis

Identification of etiology is important to help determine medical and interventional management strategies. *Idiopathic* pericarditis (and associated pericardial effusion) is believed to be the most common etiology in the developed world with a presumed viral cause and, generally, a benign self-limiting course. Clinical history of low-grade fever, nontoxic appearance, and a benign course resolving over several days are supportive of an idiopathic or virally mediated etiology; preceding or concurrent respiratory or GI illness may also be present. If idiopathic or virally mediated pericarditis is clinically suspected, only limited diagnostic evaluation is typically needed beyond basic labs (i.e., CBC, electrolytes, renal function, and inflammatory markers). Echocardiography should be obtained to assess for associated pericardial effusion.

High fevers, markedly elevated WBC, and/or toxic presentation should raise suspicions for other specific etiologies, such as *bacterial pericarditis*, *autoimmune disorder* or *associated malignancy*. Diagnostic evaluation for systemic processes should be broadened in these situations and be based on clinical history and physical exam findings.

Bacterial pericarditis is secondary to either direct extension of thoracic and/or pulmonary infections or due to bacteremia. It is often associated with septic shock.

A bacterial etiology for pericarditis should be considered in the setting of high fevers, ill presentation, or markedly elevated WBC. *S. aureus* is the most common etiology.

Prolonged or recurrent episodes of pericarditis should prompt evaluation for systemic autoimmune disease. Pericardial fluid sampling for diagnostic purposes, in the absence of other indications, may be helpful in persistent or recurrent cases, but is often unrevealing. A history of malignancy should also prompt pericardial fluid sampling to rule out malignant pericarditis.

Delayed presentation of pericarditis and/or pericardial effusion following recent pericardial injury, such as cardiac surgery, lead placement, or chest wall trauma, should raise clinical suspicion for *postcardiotomy syndrome*.

Treatment

Management of pericarditis is largely dictated by underlying mechanism and treatment of systemic autoimmune or infectious disease, if present. For most cases of presumed viral or idiopathic pericarditis, nonsteroidal anti-inflammatory drugs (NSAIDs), and possibly colchicine, help decrease the intensity and duration of symptoms. Systemic steroids for the initial bout of idiopathic pericarditis should be avoided as they may be associated with increased risk of recurrence. Management of recurrent pericarditis can be challenging and rheumatology consultation can be beneficial. Reevaluation for an identifiable underlying etiology should also be performed. Again, steroids should be avoided as best possible during initial recurrences, but they may be eventually required for symptom control in recalcitrant cases. Newer immunomodulators, such as Anakinra (Interleukin-1 receptor antagonist), have shown possible utility in early studies treating recurrent idiopathic pericarditis or cases which are steroid resistant. Associated pericardial effusions may resolve with appropriate medical therapy but may persist despite it. Indications to intervene procedurally, via percutaneous drainage with or without drain placement, or via surgical approach, include clinical tamponade and the need to obtain a sample of the effusion for diagnostic purposes.

Intubation and positive pressure ventilation are generally to be avoided in the setting of clinical tamponade, as the resultant increase in intrathoracic pressure can further hinder cardiac filling and thus precipitate cardiovascular collapse. Aggressive diuresis is likewise to be avoided in the setting of tamponade. Administration of intravenous fluid may be necessary in order to ensure adequate preload.

Antibiotics are the mainstay of treatment for bacterial pericarditis. Infectious disease consultation can help narrow antibiotic treatment, which is typically prolonged over several weeks. Bacterial pericarditis is often associated with hemodynamically active pericardial effusion or tamponade prompting intervention. However, all pericardial effusions should be drained if bacterial etiology is confirmed or strongly suspected in order to reduce the long-term risks of constrictive pericarditis. Purulent pericardial fluid can often occlude minimally invasive tubing and surgical drainage may be needed.

Constrictive Pericarditis

Constrictive pericarditis is a delayed serious complication of pericarditis, usually from an infectious etiology. Globally, tuberculosis is most often associated with constrictive pericarditis, but any infectious, usually bacterial, etiology can result in the pericardial thickening and scarring which leads to the development of constrictive physiology. Primarily, diastole is affected as the stiff pericardium impairs cardiac filling, but intrinsic myocardial diastolic and systolic function remain intact. Patients can present with exercise intolerance initially and then progress to signs of right-heart failure with hepatosplenomegaly, ascites, and protein-losing enteropathy.

Constrictive pericarditis can be detected by TTE, which can show marked respiratory variation of mitral and tricuspid inflow in the absence of a pericardial effusion. Additionally, the presence of septal bounce where the interventricular septum quickly shifts leftward with diastolic RV filling, usually during inspiration, should raise clinical suspicion for constrictive pericardial physiology when it accompanies the typical clinical scenario. Again, ventricular systolic and diastolic echocardiographic parameters are usually normal. CT scan or MRI can reveal pericardial thickening or calcifications. Cardiac catheterization typically shows near-equalization of atrial pressures and the ventricular end-diastolic pressures. This near-equalization should persist with a fluid challenge. The presence of constrictive pericardial physiology, when associated with symptoms, should prompt surgical pericardial stripping, as it can be an effective treatment.