Pericarditis, Pericardial Tamponade, and Myocarditis

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usually deadly, it can be painfully disabling if not treated appropriately.

Pericardial tamponade is a potentially deadly result of pericardial effusion. Such an effusion can develop from pericardial inflammation or cardiac trauma. Unfortunately, the early features of tamponade can mimic those of other diseases and lead to initial misdiagnosis.

Acute myocarditis is relatively uncommon, but it is a devastating condition that can occur and progress quickly, with little warning. The initial findings can vary from mild, viral-type symptoms to fulminant cardiogenic shock.

PERICARDITIS

PATHOPHYSIOLOGY

The pericardium is the layer of tissue surrounding the heart. It consists of two layers, a serous inner layer (visceral pericardium) and a fibrocollagenous outer layer (parietal pericardium). The pericardium completely encloses the ventricles and the right atrium; a portion of the left atrium remains outside the sac. A thin layer of plasma fluid (usually 15 to 30 mL) separates the visceral and parietal pericardial layers and acts as a lubricant. The main function of the pericardium appears to be provision of ligamentous stability to withstand forces against the heart. It also provides some shielding for the heart. Despite these apparent functions, however, the majority of patients who undergo pericardiectomy do not appear to suffer any decrease in cardiac performance or other ill effects.

Pericarditis refers to inflammation of the layers of the pericardium. It has many possible causes (Box 60.1), but in most cases the cause is unknown. In the majority of these idiopathic cases the presumed cause is viral, although most attempts to prove a viral cause have low yield. The most common viral cause is coxsackievirus B. The most common cause of pericarditis worldwide is tuberculosis.

PRESENTING SIGNS AND SYMPTOMS

CLASSIC

Chest pain is the typical complaint of patients with acute pericarditis. Classically, the chest pain is sharp, retrosternal, and pleuritic, and it radiates to one or both trapezius ridges
because the phrenic nerve, which traverses the pericardium, innervates these muscles. Typically, the pain also changes with body position: it improves when the patient sits up and leans forward and worsens when the patient lies supine.

The physical examination of patients with acute pericarditis is usually nondiagnostic. Although some researchers report the presence of a friction rub in up to 85% of patients at some point in the course of the disease, the presence of a friction rub at the time of initial evaluation is unreliable. When a friction rub is present, it is best heard at the left sternal border at end-expiration with the patient leaning forward. The rub is usually described as consisting of three components that correspond to atrial systole, ventricular systole, and rapid diastolic filling. A friction rub is thought to be caused by rubbing of the inflamed layers of the pericardium against each other.

**TYPICAL VARIATIONS**
The typical findings in patients with acute pericarditis may not always be present. Although chest pain is the most common symptom, patients sometimes have dyspnea as the primary complaint. When chest pain is present, it is not always positional or pleuritic, and it may not always radiate to the trapezius ridge. Patients may also complain of cough, upper respiratory symptoms, nausea, or vomiting, which may mislead the physician to an alternative diagnosis. Patients with bacterial infections are likely to have complaints of fever, and patients with tuberculous pericarditis are likely to report a chronic cough, weight loss, and night sweats.

Although the presence of a triphasic friction rub is classic, it actually occurs in only half of patients with pericarditis. The physical findings may also be notable for hypotension and jugular venous distention in the presence of pericardial tamponade (discussed later).

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**
A thorough differential diagnosis of a patient with chest pain is described in Chapter 54. However, the most important initial consideration should always be the deadly causes of chest pain: pericarditis, acute coronary ischemia or infarction, thoracic aortic dissection, and pulmonary embolism. Table 60.1 lists some historical and physical examination features that are helpful in distinguishing among these conditions. Distinction among these causes of chest pain is critical in terms of treatment; patients with pulmonary embolism or myocardial infarction often require treatment with anticoagulants and thrombolytics, medications that can be deadly in patients with acute pericarditis or aortic dissection.

The diagnosis of acute pericarditis is based primarily on the clinical findings. In many cases an electrocardiogram (ECG) is helpful in confirming the diagnosis. Sound knowledge of the ECG findings in patients with acute pericarditis is critical, as well as some findings that help in diagnosing pericarditis versus myocardial infarction (see Table 60.1). Classically, pericarditis evolves through four ECG stages as described in Table 60.2. The first stage of acute pericarditis, characterized by diffuse ST-segment elevation and PR-segment depression or downsloping (Fig. 60.1), is the most common stage encountered in the emergency department (ED). The ST segments should be concave upward; a convex upward ("tombstone")
### Table 60.1 Differential Diagnosis of Acute Pericarditis

<table>
<thead>
<tr>
<th>Patient Presentation</th>
<th>ACUTE PERICARDITIS</th>
<th>ACUTE MYOCARDIAL ISCHEMIA OR INFARCTION</th>
<th>AORTIC DISSECTION</th>
<th>PULMONARY EMBOLISM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain description</td>
<td>Sharp, pleuritic, positional</td>
<td>Pressure, squeezing, tightness</td>
<td>Sharp, maximal intensity at onset</td>
<td>Sharp, pleuritic, abrupt onset</td>
</tr>
<tr>
<td>Chest pain radiation</td>
<td>Trapezius ridge</td>
<td>Usually to the left arm, jaw, neck, or shoulder; may also radiate to the right side</td>
<td>Straight to midscapular area of the back</td>
<td>Not typical</td>
</tr>
<tr>
<td>Response to nitroglycerin</td>
<td>Not typical</td>
<td>Improves</td>
<td>Not typical</td>
<td>Not typical</td>
</tr>
<tr>
<td>Vital signs</td>
<td>Tachycardia and fever common</td>
<td>Fever not typical; blood pressure and heart rate are variable</td>
<td>Hypertension common</td>
<td>Occasionally low-grade fever, tachycardia and hypoxia common</td>
</tr>
<tr>
<td>Other physical examination findings</td>
<td>Friction rub common during course, though less common on initial evaluation</td>
<td>Fourth heart sound is “classic” in cases of cardiac ischemia; third heart sound if heart failure present</td>
<td>Occasional pulse deficits</td>
<td>Occasional leg swelling or tenderness if embolus originated in the legs</td>
</tr>
<tr>
<td>Electrocardiographic findings</td>
<td>(See Table 60.2) Early: diffuse ST-segment elevation and PR-segment depression</td>
<td>ST-segment elevation or depression typically in an anatomic distribution corresponding to the involved coronary vessel</td>
<td>Left ventricular hypertrophy if chronic</td>
<td>Sinus tachycardia common; bradycardia and AV blocks uncommon</td>
</tr>
<tr>
<td></td>
<td>Absence of reciprocal ST-segment depression</td>
<td>Tachycardia or bradycardia and AV blocks not uncommon</td>
<td>Hypertension present</td>
<td>Large emboli often associated with T-wave inversions, most commonly in right precordial leads and less commonly in inferior leads</td>
</tr>
<tr>
<td></td>
<td>Sinus tachycardia common; bradycardia and AV blocks uncommon</td>
<td></td>
<td>Variable ST-segment or T-wave changes</td>
<td>ST-segment elevation possible but uncommon</td>
</tr>
<tr>
<td>Chest radiography findings</td>
<td>Usually normal; cardiomegaly if large pericardial effusion present</td>
<td>Cardiomegaly if chronic left ventricular hypertrophy present; evidence of heart failure may be present</td>
<td>Cardiomegaly common if chronic left ventricular hypertrophy present; wide mediastinum common</td>
<td>Usually normal; most common abnormalities are elevated hemidiaphragm, atelectasis, small pleural effusion</td>
</tr>
<tr>
<td>Cardiac biomarkers</td>
<td>Levels usually normal; mild elevations not uncommon</td>
<td>Elevations typical in myocardial infarction</td>
<td>Levels normal</td>
<td>Large emboli occasionally associated with mild elevations in troponin or brain natriuretic peptide</td>
</tr>
</tbody>
</table>

**Table 60.2 Electrocardiographic Stages of Acute Pericarditis**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>Diffuse ST-segment elevation and PR-segment depression (except in leads V₁ and aVR)</td>
</tr>
<tr>
<td>Stage II</td>
<td>Resolution of the ST-segment and PR-segment changes T-wave flattening in the same leads</td>
</tr>
<tr>
<td>Stage III</td>
<td>T-wave inversions in the same leads</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Normalization of electrographic abnormalities</td>
</tr>
</tbody>
</table>

morphology virtually excludes the diagnosis of pericarditis and rules in acute myocardial infarction. ST-segment depression may be present in leads aVR and V₁ in a patient with pericarditis but is unlikely to be present in any of the other 10 leads. In fact, the presence of ST-segment depression in any of the other 10 leads should be considered the “reciprocal” changes of acute myocardial infarction. These four stages generally progress over the course of days to weeks. The ECG changes in the second and third stages usually occur in the same leads in which abnormalities in the initial stage occurred.

Although the abnormalities noted on an ECG are typically described as “classic” for acute pericarditis, physicians should...
Pericarditis, Pericardial Tamponade, and Myocarditis

are effectively treated with high-dose aspirin (2 to 4 g daily) or nonsteroidal antiinflammatory drugs (NSAIDs). Ibuprofen is effective in most cases and has fewer side effects than other NSAIDs; the pain usually improves significantly within days with ibuprofen therapy. If the patient’s symptoms persist, an alternative NSAID is indicated. Indomethacin is often used for severe cases because of its stronger antiinflammatory effect, although it should be avoided in patients with a history of ischemic heart disease because it may decrease coronary blood flow.

Evidence now suggests that addition of colchicine (1.0 to 2.0 mg for the first day and then 0.5 to 1.0 mg/day for 3 months) to the standard regimen is effective in hastening the resolution of acute symptoms and also preventing recurrence rates, regardless of the cause of the pericarditis. Colchicine is also effective in cases of recurrent pericarditis. The use of corticosteroids is generally reserved for recurrent cases of pericarditis that are unresponsive to aspirin or NSAIDs plus colchicine. Initiation of steroids early in the course of first-time pericarditis may actually be an independent risk factor for recurrence.

Patients with bacterial or other nonviral infectious causes of pericarditis should be treated aggressively with antimicrobial therapy. Large infected pericardial effusions require drainage as well. Management of neoplastic causes of pericarditis should be targeted at treating the underlying malignancy. Patients with uremic pericarditis require urgent hemodialysis.

**FOLLOW-UP AND NEXT STEPS IN CARE**

The majority of patients with acute pericarditis can be treated as outpatients, with the symptoms generally resolving within 2 weeks. Outpatient management is suitable for patients with
mild symptoms, hemodynamic stability, and ability to tolerate oral medications. Reasonable indications for admission include fever or suspicion of a bacterial cause of the pericarditis, immunosuppression, pericarditis associated with trauma, presence of a moderate to large pericardial effusion, and hemodynamic instability. A history of active anticoagulant use is generally considered a poor prognostic factor and warrants hospital admission as well.4

PERICARDIAL TAMPOONADE

PATHOPHYSIOLOGY

Trauma or inflammation of the pericardium can cause fluid to accumulate within the intrapericardial space. Normally, the pericardium is capable of stretching and accommodating 2 L of fluid or more when the fluid accumulates very slowly.1 However, if the fluid accumulates more rapidly than can be accommodated by the distensibility of the pericardium, especially in the case of trauma, in the presence of fibrotic pericardium, or if the volume of pericardial fluid is excessive, significant intrapericardial pressure results and can produce pericardial tamponade.

Pericardial tamponade develops when intrapericardial fluid produces sufficient pressure to compress the cardiac chambers. Compression of the chambers impairs ventricular diastolic filling and stroke volume. Initial compensatory mechanisms, especially tachycardia, may temporarily sustain cardiac output. However, as pericardial fluid continues to increase, the compensatory mechanisms begin to fail, and diminished cardiac output, hypotension, and full cardiovascular collapse ensue.

The typical causes of pericardial tamponade include any disorder that results in acute or chronic pericardial inflammation, as well as conditions in which a patient sustains penetrating trauma to the heart or cardiac surgery. The conditions previously noted to cause pericarditis are common precipitants of pericardial tamponade. The composition of the intrapericardial effusion varies according to the precipitating cause; in bacterial pericarditis, for example, the effusion is often pus, and in cardiac trauma or cardiac surgery, it is often blood and clots. Regardless of the composition of the effusion, the physiology that leads to pericardial tamponade and the immediate treatment are similar.

PRESENTING SIGNS AND SYMPTOMS

CLASSIC

The typical symptoms associated with a large pericardial effusion are nonspecific. Malaise, generalized weakness, ascites, and edema are common in patients with subacute or chronic effusions as a result of poor cardiac function. Many other symptoms are related to compression of adjacent mediastinal structures by the effusion. Dyspnea and cough are common and may be due to displacement or compression of bronchial structures or lung tissue by the effusion. Dyspnea on exertion is common as well and results from impairment of venous return and cardiac output. Patients often report a sense of dysphagia, which is due to esophageal compression. Hiccups may occur as a result of esophageal compression and involvement of the phrenic and vagus nerves. Hoarseness may result from compression of the recurrent laryngeal nerve.12

Physical findings are also often nonspecific. Tachycardia and tachypnea are common. Despite the presence of tachycardia and dyspnea, however, oxygen saturation levels are usually normal because the effusion itself does not impair alveolar air exchange. Lung sounds are generally normal as well. Findings of hypoxia or focal abnormalities on lung examination should suggest a superimposed pulmonary condition or an alternative diagnosis. Fever is common if the underlying cause is infectious. Pericardial friction rubs are reportedly common if the underlying cause is inflammatory, although diminished heart sounds are also frequent because of reduced cardiac function and attenuation of their transmission by the effusion.

When a pericardial effusion produces pericardial tamponade, additional findings are notable. Decreased cardiac function produces hypotension and shock. Jugular venous distention is typically present because of impaired venous return. Pulsus paradoxus (drop in systolic blood pressure of greater than 10 mm Hg during normal inspiration) is also typical, although its presence has limited specificity for pericardial tamponade. Several other conditions that are associated with hypotension or dyspnea (or both) can also produce pulsus paradoxus, including massive pulmonary embolism, hemorrhagic shock, and obstructive lung disease.12 Death is usually preceded by pulseless electrical activity.13

TYPICAL VARIATIONS

Although the symptoms and physical findings already noted are common, certain conditions may produce unexpected findings in the presence of pericardial tamponade. Patients who have severe hypothyroidism or uremia or who take atroventricular nodal blocking agents (e.g., calcium channel blockers, beta-blockers, digoxin) may have a relative bradycardia. Jugular venous distention is typical in pericardial tamponade as well, but it is often absent in patients who are hypovolemic or in whom the pericardial tamponade developed very quickly (e.g., after cardiac trauma). Overt hypotension may be absent as well in patients with a history of severe antecedent hypertension.12,15

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

A complete differential diagnosis of hypotension and shock is beyond the scope of this chapter. However, the emergency physician (EP) should always consider other typical causes of hypotension, especially those that similarly produce jugular venous distension: massive pulmonary embolism, large left ventricular myocardial infarction with cardiogenic shock, right ventricular myocardial infarction, acute aortic or mitral valve insufficiency, and superior vena cava syndrome.

The primary means of diagnosing pericardial tamponade is via two-dimensional echocardiography. A pericardial effusion is easily seen in most patients on subcostal or parasternal views (Fig. 60.2). An echo-free space should be visible throughout the cardiac cycle when the pericardial effusion is at least 25 mL.1 The presence of a pericardial effusion in combination with hypotension and echocardiographic
evidence of early diastolic right ventricular collapse and late diastolic right atrial collapse is diagnostic of pericardial tamponade. In approximately 25% of cases, the left atrium also demonstrates collapse, a very specific sign of tamponade. The left ventricle rarely demonstrates collapse except in specific conditions such as localized postoperative tamponade. Other echocardiographic findings that may be found in patients with pericardial tamponade are a dilated inferior vena cava without inspiratory collapse and beat-to-beat swinging of the heart within the pericardial fluid (when the effusion is large).

Other imaging studies can be helpful in evaluating these patients as well. Computed tomography (CT) and magnetic resonance imaging are very accurate in detecting pericardial effusions, in addition to diagnosing alternative conditions. However, they should not be used in patients with borderline or overt hemodynamic instability because of the need to remove such patients from the ED for the procedures. Chest radiography is primarily used to evaluate the patient for alternative diagnoses as well, such as pneumonia or pulmonary edema. In patients with a large pericardial effusion, cardiomegaly is a nearly universal finding (Fig. 60.3). The chest radiograph is particularly helpful in this setting if previous radiographs are available that demonstrate a normal-sized heart. Previous radiographs that show the massive cardiomegaly to be new are highly suggestive of a large pericardial effusion.

The ECG can be helpful in the diagnosis of large pericardial effusions. The most common abnormality is tachycardia, especially in the presence of tamponade. Low voltage is common as well and is caused by attenuation of the electrical impulse as it passes through the effusion before reaching the ECG electrodes. Although low voltage is nonspecific, the presence of new low voltage (in comparison with previous ECGs) is much more specific for large pericardial effusions.

The third “classic” ECG abnormality is electrical alternans. Electrical alternans refers to beat-to-beat variation in amplitudes of the ECG complexes (Fig. 60.4) and is attributed to “swinging” of the heart back and forth within the pericardial fluid. Electrical alternans is present in less than one third of cases of pericardial tamponade. Although none of these three findings individually is completely diagnostic of large pericardial effusions, the combination of all three is very highly specific.

**TREATMENT**

Initial management of patients with pericardial tamponade should focus on the typical ABCs of resuscitation (airway, breathing, circulation). Certain caveats should be made, however. Although the primary pathophysiologic abnormality underlying hemodynamic compromise in patients with pericardial tamponade is ventricular filling, the benefit of volume infusion to improve filling is controversial. Animal studies have shown variable results with respect to the hemodynamic benefit of volume infusion, which may improve systemic perfusion only in patients with hypovolemia. Strong supporting data in humans are lacking, however. In patients with traumatic pericardial tamponade, large-volume infusions may actually precipitate further deterioration. Strong evidence supporting specific inotropic agents is lacking as well, although theoretically agents that reduce the elevated vascular resistance, such as dobutamine and milrinone, would seem ideal. Mechanical ventilation should generally be avoided except in patients with respiratory failure; the positive airway pressure associated with mechanical ventilation decreases venous return and cardiac output.

Treatment of pericardial tamponade is drainage of the intrapericardial fluid (pericardiocentesis). Drainage is best...
performed via needle aspiration under echocardiographic, CT, or fluoroscopic guidance. In patients who exhibit rapid cardiac decompensation or are in cardiac arrest, the EP should perform emergency pericardiocentesis without waiting for imaging guidance. The procedure is performed with a 16- or 18-gauge needle, most commonly inserted into the left paraxiphoid area of the chest. The needle is pointed downward at an approximately 30-degree angle to the chest to bypass the left costal margin and is aimed toward the left shoulder. The needle should be inserted and advanced slowly until the pericardium is penetrated and fluid is aspirated. Use of a sheathed needle can facilitate the process—once the pericardium has been penetrated, the core of the needle can be removed with the sheath left in the pericardial space to assist in further removal of fluid. Some researchers have advocated attaching ECG leads to the hub of the needle so that when the pericardium is penetrated, an injury pattern (i.e., ST-segment elevation) will be noted. However, a recent review recommends against this practice because of the likelihood of misleading results. In patients with acute cardiac decompensation, aspiration of even 10 to 20 mL of blood should be sufficient to produce some hemodynamic improvement. However, once full cardiac arrest has occurred, pericardiocentesis has a limited success rate. Potential complications of pericardiocentesis include puncture or laceration of the cardiac chambers, injury to coronary vessels, pneumothorax, ventricular dysrhythmias, pneumopericardium, and delayed infection. These complications are more common in the setting of emergency pericardiocentesis, which is sometimes performed without imaging guidance.

Because pericardiocentesis is less likely to be successful in patients with clotted hemopericardium, surgical drainage will be necessary. Surgical drainage is also required in patients in whom intrapericardial bleeding is present (e.g., postoperative pericardial tamponade, traumatic pericardial tamponade, pericardial tamponade with aortic dissection). In these patients, pericardiocentesis is temporizing at best; in conjunction with surgical drainage, definitive repair of the bleeding sites is essential. Additional therapy should focus on treating the underlying cause of the pericardial inflammation that led to pericardial tamponade.

**FOLLOW-UP AND NEXT STEPS IN CARE**

Patients with pericardial tamponade should be admitted to an intensive care setting for definitive therapy and close hemodynamic monitoring. If surgical therapy is indicated, emergency surgical consultation is mandatory. When surgical therapy is not planned, cardiology consultation is most appropriate for the performance of pericardiocentesis under echocardiographic guidance. Nephrology consultation is also appropriate for urgent hemodialysis in patients with uremia.

**MYOCARDITIS**

**PATHOPHYSIOLOGY**

Myocarditis is an inflammatory condition that causes myocardial damage, usually because of infectious, immunologic, or toxin-mediated conditions (Box 60.2). Myocarditis can be manifested as mild constitutional symptoms, moderate cardiopulmonary symptoms, or fulminant cardiopulmonary decompensation leading to death. In the majority of adult cases, the myocardial damage is believed to be caused by autoimmune processes that are often triggered by viral or other infections, whereas in neonates and infants, injury to myocytes is believed to occur more often because of direct injury by the pathogen itself.

Approximately 10% of postmortem examinations demonstrate some degree of histologic evidence of myocarditis. However, most of the patients were not clinically symptomatic.
before death. Conversely, in patients in whom myocarditis is diagnosed clinically, only one third have histologic findings consistent with the disease. Even in cases that progress to dilated cardiomyopathy, only 40% of patients have microscopic evidence of myocarditis. Consequently, the actual incidence of myocarditis is unknown.

PRESENTING SIGNS AND SYMPTOMS

CLASSIC

The clinical manifestations of myocarditis usually begin days to weeks after the acute infection, especially when viruses are implicated as the cause. However, only 50% of patients report a recent upper respiratory or gastrointestinal viral type of infection. The initial symptoms are nonspecific and constitutional in nature: low fever, fatigue, malaise, myalgia, and arthralgia. These mild symptoms are often the reason for initial misdiagnosis or delays in proper diagnosis of this condition. Cardiopulmonary symptoms, especially mild dyspnea and chest pain, are common as well. The chest pain can be pleuritic, sharp, and positional, much like pericarditis, or substernal and squeezing, much like typical ischemic pain. Patients with congestive heart failure may report more significant dyspnea, as well as cough, orthopnea, paroxysmal nocturnal dyspnea, and edema. Less commonly, patients may initially be seen after a syncopal episode, usually the result of a bradycardia.

The physical findings are often notable for low-grade fever, tachycardia, and tachypnea. Classically, the patient has “tachycardia out of proportion to the fever”—extreme tachycardia without obvious hypovolemia or high fever (Fig. 60.5). Patients may also experience bradycardia if a high-grade atrioventricular block develops (Fig. 60.6). Evidence of congestive heart failure is frequently present as well: jugular venous distention, bibasilar rales on lung examination, a third heart sound (S3) on cardiac examination, and peripheral edema. Patients with advanced or fulminant myocarditis may arrive at the ED in full cardiogenic shock.

TYPICAL VARIATIONS

Pediatric patients tend to have early, nonspecific symptoms, including fever, viral upper respiratory symptoms, and poor feeding. In more severe cases, the infant or neonate may exhibit severe tachycardia, tachypnea, sweating while feeding, respiratory distress, and cyanosis or other signs of poor perfusion. Tachydysrhythmias and bradycardia, especially those involving a high-grade atrioventricular block, are
common as well. Severe myocarditis in the very young is often also manifested as pulmonary edema, cardiogenic shock, and multiorgan hypoperfusion.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis of myocarditis is extremely broad because of the nonspecific nature of the initial findings. Patients who are initially seen with cardiopulmonary symptoms have a slightly more limited differential diagnosis, but initial consideration should always be focused on the most deadly diseases that produce chest pain or dyspnea: acute coronary syndrome, aortic dissection, pulmonary embolism, acute pericarditis or pericardial tamponade, esophageal rupture, cardiogenic pulmonary edema, and pneumonia.

The initial diagnostic evaluation should be prompted by clinical suspicion. The finding of cardiopulmonary complaints, unexplained tachycardia, or evidence of congestive
heart failure on examination in young patients should prompt consideration of myocarditis. An ECG and chest radiograph are appropriate initial tests in these patients. Despite the absence of a specific or single diagnostic ECG abnormality, the majority of patients with myocarditis have at least some abnormality noted on the ECG. The most common abnormalities are sinus tachycardia (see Fig. 60.5) and nonspecific ST-segment or T-wave changes. Other abnormalities that may occur in patients with myocarditis are conduction abnormalities (new fascicular blocks, new bundle branch blocks, and atrioventricular blocks), atrial or ventricular tachydysrhythmias and bradydysrhythmias, overt ischemic changes (T-wave inversions, ST-segment changes), and Q-wave formation. The ischemic changes are often indistinguishable from those seen with true cardiac ischemia or myocardial infarction. If the pericardium is also involved (myopericarditis), PR-segment depression with concurrent ST-segment elevation may be found. If a pericardial effusion develops, low voltage may occur. The ECG findings are rarely completely normal.

Findings on plain chest radiographs are often frequently in mild or early cases of myocarditis. However, most patients with cardiopulmonary complaints have some radiographic signs of congestive heart failure: cardiomegaly, pulmonary vascular redistribution, interstitial edema, and frank pulmonary edema. Pleural effusions may be present as well.

Laboratory studies typically ordered in the ED include evaluation of markers of inflammation and cardiac biomarkers. Inflammatory markers, such as the white blood cell count, erythrocyte sedimentation rate, and C-reactive protein value, are expected to be elevated, but unfortunately, such elevations are nonspecific. Measurements of markers are sometimes used to monitor the course of the disease after the diagnosis is made. Cardiac biomarkers, including troponin and creatinine phosphokinase values, are also sometimes elevated. The initial elevations in cardiac biomarkers are frequently indistinguishable from those expected with acute myocardial infarction. Serial measurements of cardiac biomarkers can be helpful, however, because the levels do not tend to rise and fall as quickly with myocarditis as with myocardial infarction.

Echocardiography may also be helpful in the ED, especially when there is confusion in distinguishing between myocarditis and acute coronary syndrome. Echocardiography in patients with acute coronary syndrome (with active ischemia) usually demonstrates focal wall hypokinesis. Patients with myocarditis can also have focal hypokinesis, but they are more likely to have diffuse hypokinesis and multichamber dilation as well. In addition, the echocardiogram can demonstrate evidence of complications, such as pericardial effusion or intracardiac thrombus.

Endomyocardial biopsy is usually considered the “gold standard” for the diagnosis of myocarditis. Evidence of microscopic myocardial inflammation with various levels of necrosis is generally used to diagnose and categorize stages of myocarditis. However, biopsy is not feasible in the ED. Additionally, recent reports have questioned the sensitivity and specificity of the test.

Other tests that can be ordered by the EP are viral cultures, blood cultures, and other immunologic studies. These tests are rarely helpful in the acute setting but may of use to inpatient physicians during the hospital course.

**TREATMENT**

Close attention should be paid to the ABCs of resuscitation because patients with fulminant myocarditis can decompensate rapidly. The mainstay of treatment of myocarditis is primarily supportive with a focus on hemodynamic support and management of complications. Congestive heart failure and hypoxia should be treated as usual, with high-flow oxygen, preload and afterload reduction, and diuretics. The patient’s fluid status must also be monitored closely because of the risk for congestive heart failure. Bradydysrhythmias should be managed as usual. Patients with high-degree atrioventricular blocks often require pacemaker placement. Tachydysrhythmias should generally be managed as usual, although preference should be given to titratable medications because drugs with negative inotropic effects can precipitate unpredictable hemodynamic collapse in these patients with myocarditis, whose status is tenuous. Intramural thrombi noted on echocardiography should be treated with anticoagulation. The benefit of prophylactic anticoagulation is uncertain, especially given the risk that these patients have for hemopericardium. Additional myocardial workload reduction can be accomplished through fever reduction and correction of anemia.

Patients in shock should be treated aggressively. Adequate perfusion should be achieved in hypotensive patients through the use of vasopressors (e.g., dopamine, norepinephrine) and positive inotropic agents (e.g., dobutamine). Ventricular assist devices, intraaortic balloon counterpulsation, and cardiopulmonary bypass have been used successfully in some patients during the wait for clinical improvement or cardiac transplantation.

Antiviral therapy may be helpful during the patient’s hospital course if a viral cause of the myocarditis has been determined. Other therapies should be focused on the determined underlying condition (e.g., Lyme carditis, toxin-induced myocarditis). Immunosuppressive medications have been used successfully in some patients in the later stages of illness, but this experience appears to be anecdotal only. The use of corticosteroids lacks good evidence of benefit as well. High-dose intravenous gamma globulin has been used successfully in some pediatric patients, with improvement in ventricular function at 1 year.

**FOLLOW-UP AND NEXT STEPS IN CARE**

Patients in whom myocarditis is suspected should be admitted to the hospital. Cardiac monitoring should be started to assess for the development of dysrhythmias, and close hemodynamic monitoring is indicated as well. Critical care, infectious disease, and cardiology consultants should be involved in the care plans for these patients. Because patients with severely decompensated cardiac conditions may require cardiac transplantation, appropriate surgical consultants should also be involved in the care of patients with severe myocarditis.

**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
CHAPTER 60  PERICARDITIS, PERICARDIAL TAMPOONADE, AND MYOCARDITIS

REFERENCES