Pericarditis

Pericarditis is a common but frequently subclinical entity. There are a number of causes, including infection, systemic illness, cardiac disease, trauma, and neoplasm. Iatrogenic causes include surgery, cardiac instrumentation, irradiation, and medications. The clinical presentation varies, depending on the cause. Chest pain and dyspnea are characteristic complaints. A typical progression of ECG changes occurs during the course of acute pericarditis. These changes occasionally require differentiation from those of acute myocardial infarction or normal variant ST segment elevation. Echocardiography is the most sensitive technique for detecting the presence of pericardial effusion. In addition, a number of echocardiographic findings are characteristic of larger effusions and cardiac tamponade. Any form of pericarditis may lead to the development of cardiac tamponade. Malignant effusion is probably the most common single cause. [Sternbach GL: Pericarditis. Ann Emerg Med March 1988;17:214-220.]

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INTRODUCTION

The normal pericardium consists of two layers of fibrous tissue ordinarily containing up to 50 mL of fluid with a chemical composition similar to that of serum. Pericarditis is a common but often subclinical entity, observed more frequently at autopsy than during life.[1] Its true incidence is uncertain, because the course is often self-limited and because pericardial inflammation may be an unrecognized part of a systemic illness or infection. Pericarditis may be the consequence of a number of pathologic processes.

ETIOLOGY

Common causes are listed (Figure 1). The relative incidence of specific types varies from one institution to another, depending on patient population. The most common form is probably idiopathic pericarditis.[2] This has been postulated to be of viral origin in most cases, but efforts to isolate a virus from pericardial fluid in cases of idiopathic pericarditis usually have been unsuccessful.[2]

A number of viruses are known to be highly cardiotropic. The Coxsackie B group is especially likely to produce pericarditis or myocarditis.[3] Other viruses known to cause pericarditis include echovirus type 8, mumps, influenza, Epstein-Barr, and varicella-zoster viruses.[4]

Pericarditis may be the consequence of bacterial infection, but the incidence of purulent pericarditis has declined in the antibiotic era.[5] *Staphylococcus aureus*, once the most common causative agent by far, remains the single most frequent etiologic organism.[6] However, there has been a progressive "widening" of the bacterial spectrum over the past three decades, with a large increase in pericarditis caused by Gram-negative aerobic bacilli.[5] In children, purulent pericarditis most frequently occurs before the age of 2, with *S aureus, Haemophilus influenzae, Neisseria meningiditis,* and *Streptococcus pneumoniae* the most common pathogens, in descending order of frequency.[7]

The pericardium is rarely a primary site of bacterial infection, and purulent pericarditis almost always occurs as a complication of another illness.[5,6] Spread of infection to the pericardium occurs from one of the following pathways: direct pulmonary extension; hematogenous spread; septic embolization due to endocarditis or rupture of a myocardial abscess; perfora-

tion of the chest wall, either traumatic or surgical; and extension of a suppurative subdiaphragmatic focus. The last is much less common.[5] Patients predisposed to the development of purulent pericarditis are those who are debilitated or immunocompromised; those who have undergone extensive thoracic or cardiac surgery; those with uncontrolled endocardial or myocardial infection; and those with preexisting nonpyogenic pericarditis.[6]

Tuberculous infection, once the most common cause of pericarditis, is now rare, occurring in 1% to 2% of patients with pulmonary tuberculosis.[8] Fungal and parasitic infections are also uncommon causes in this country.

Pericarditis may be the consequence of noninfectious systemic illness, such as rheumatoid arthritis, rheumatic fever, systemic lupus erythematosus, scleroderma, and sarcoidosis. Its course is frequently subclinical in these diseases. Evidence of pericarditis has been discovered in up to 50% of rheumatoid arthritis patients at autopsy, but the condition is seldom diagnosed in these patients during life.[9] Similarly, post-mortem findings of pericarditis are seen in 60% to 80% of patients with SLE, but only about 50% display signs while alive.[10]

Patients with severe rheumatoid pericarditis as evidenced by cardiac tamponade or constrictive pericarditis are likely to be those whose rheumatoid disease is moderate to severe. Subcutaneous nodules are present in the majority of these patients. However, the duration of rheumatoid arthritis appears to have no bearing on the development of clinically severe pericarditis.[9]

Pericarditis may accompany myocardial infarction or follow its onset, usually by one to three days. The incidence of such early postmyocardial infarction pericarditis has been reported as 7% to 16%.[11] The course is generally benign, lasting one to two days.

In 1956, Dressler described a syndrome (subsequently named for him) of fever, pericarditis, and pleuritis appearing weeks to months after myocardial infarction.[12] He estimated that this syndrome occurred in 3% to 4% of myocardial infarction patients.[13] Dressler's syndrome has been assumed to be a product of an autoimmune reaction. However, this mechanism has been challenged, and the very existence of the syndrome as a distinct entity questioned.[11,14] Dressler's syndrome may actually represent a continuing inflammation in patients who develop early post-myocardial infarction pericarditis.[11]

Both penetrating and blunt thoracic trauma may produce pericarditis, as may any rupture or leakage of an aortic aneurysm into the pericardial space. There are a variety of iatrogenic causes of pericarditis. These include perforation of the heart during cardiac catheterization, central venipuncture, pacemaker insertion, and pericardiocentesis.[15-18] Complications of cardiac surgery include purulent pericarditis and the post-pericardiotomy syndrome. The latter is a febrile illness with pleural and pericardial reaction that appears beyond the first postoperative week of surgery involving pericardial entry. An immune mechanism has been postulated.[19] There is a 10% to 40% incidence of this syndrome, which usually runs a benign course.[19]

A number of medications are known to cause pericarditis (Figure 2).[20-29] Pericarditis may constitute a part of the drug-induced SLE syndrome produced by procainamide,[20] hydralazine,[24] methyldopa,[21] isoniazid,[26] and reserpine.[26] Pericarditis is caused by penicillin[25] and cromolyn sodium[27] on the basis of a hypersensitivity reaction. Constrictive pericarditis may be a part of a generalized mediastinal fibrosis induced by methysergide.[23] The antineoplastic drug doxorubicin may produce pericarditis in addition to cardiomyopathy.[30]

High-dose mediastinal irradiation, used in the 1960s primarily for the treatment of Hodgkin's lymphoma, may produce pericarditis, pericardial effusion, or constrictive pericarditis. Symptoms may occur during the course of radiation therapy or weeks to years later. The incidence of these forms of pericardial disease has been markedly diminished since the early 1970s owing to the limitation of cardiac radiation exposure and the increasing use of chemotherapy in the treatment of Hodgkin's disease and breast carcinoma.[30]

FIGURE 1. *Common causes of pericarditis.*

FIGURE 2. *Drugs known to produce pericarditis.*

Infection
- Pyogenic
- Tuberculosis
- Viral
- Fungal
- Parasitic

Systemic Illness
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Scleroderma
- Rheumatic fever
- Sarcoidosis
- Renal failure
- Hypothyroidism

Cardiac Disease
- Myocardial infarction
- Aortic dissection

Trauma

Iatrogenic
- Cardiac instrumentation
- Postsurgical
- Irradiation
- Drugs

Neoplasm

Idiopathic

1

- Procainamide
- Hydralazine
- Reserpine
- Methyldopa
- Isoniazid
- Phenytoin
- Penicillin
- Cromolyn sodium
- Dantrolene
- Minoxidil
- Methysergide
- Doxorubicin

2

Pericardial disease is the most prominent clinical aspect of neoplasm-related cardiac disease. The heart is involved in approximately 10% of cases of malignant disease, but such involvement is most commonly asymptomatic, being discovered at autopsy.[1] Primary pericardial neoplasms are rare.[1] Although pericardial metastases may occur with virtually any neoplasm, about 80% occur in patients with lung or breast carcinoma, lymphoma, and leukemia. Malignant melanoma accounts for another 5%.[30] Neoplastic pericardial invasion often leads to pericardial effusion, and malignancy is probably currently the

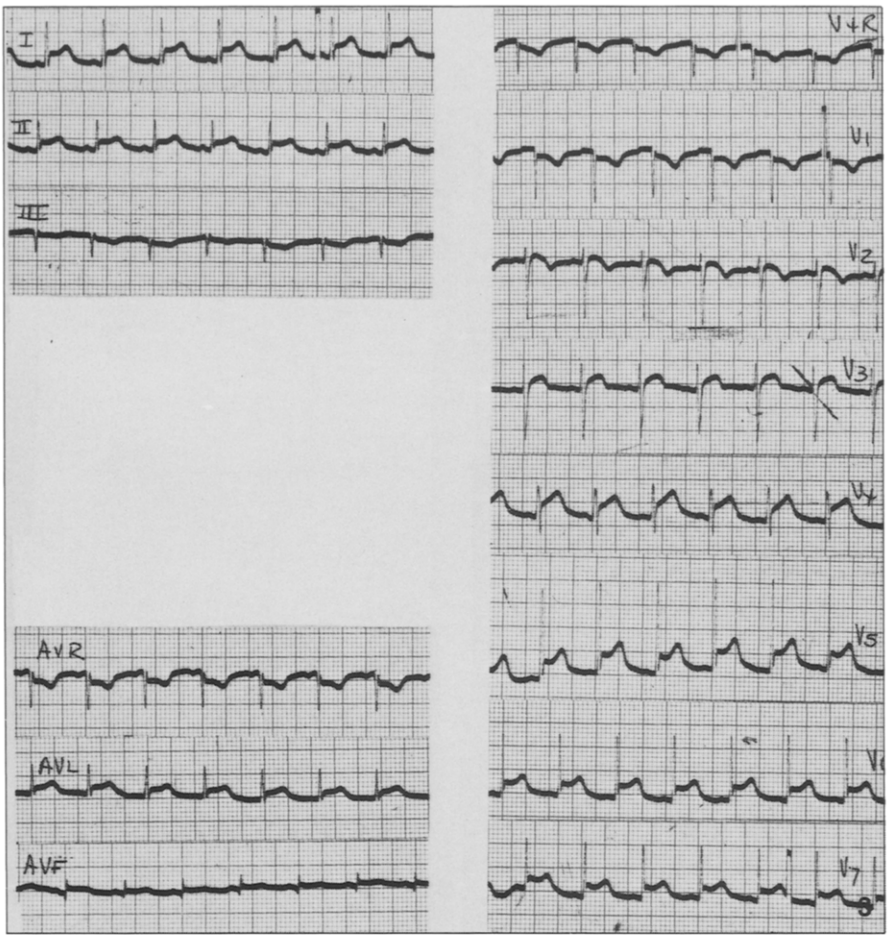

FIGURE 3. *Acute pericarditis with marked ST segment elevation.*

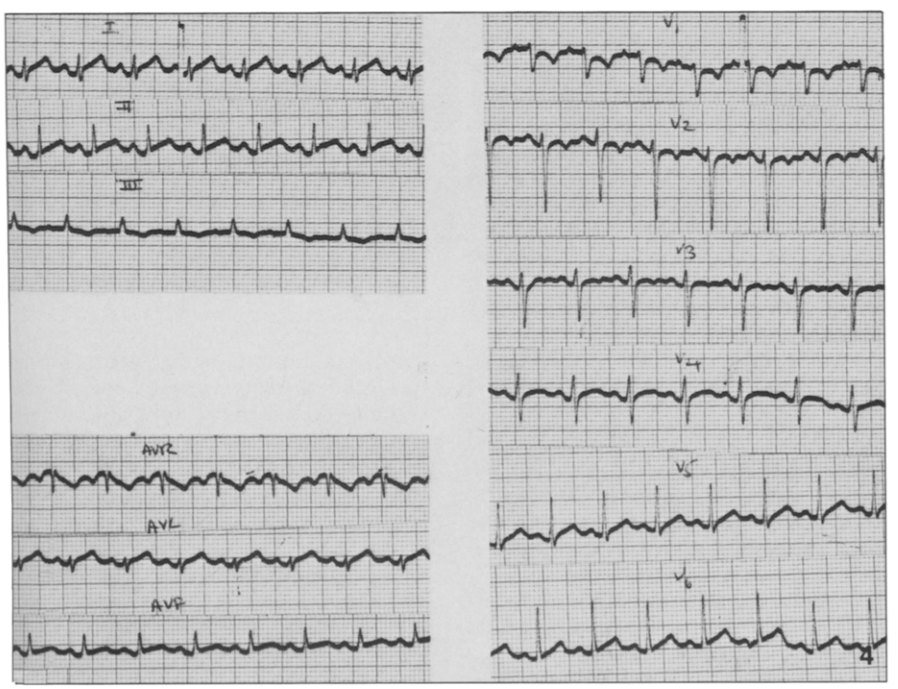

FIGURE 4. *ECG revealing PR segment depression and T wave inversion.*

most frequent cause of nontraumatic cardiac tamponade in most centers.[30]

CLINICAL PRESENTATION

The nature of the onset of pericarditis varies depending on the etiology. Purulent pericarditis usually presents abruptly as a fulminant illness. The clinical manifestations of viral pericarditis may follow an upper respiratory infection, and include chest pain, dyspnea, and fever. Pain is typically a prominent feature of viral pericarditis, and its absence in that setting is unusual. Tuberculous pericarditis, on the other hand, frequently has an insidious onset, presenting with the gradual development of such nonspecific symptoms as fever, malaise, anorexia, and weakness.[8] Presentation of other forms of pericarditis varies and may be gradual or abrupt, with symptoms referable to pericardial inflammation or effusion.

Pain is one of the most important features of pericarditis. It is usually retrosternal or precordial, sharp and almost always pleuritic, becoming dramatically worse with cough or inspiration. The pain is characteristically aggravated by recumbency and eased by sitting upright and leaning forward. Occasionally it is felt at the cardiac apex synchronously with each heart beat. There may be a wide area of radiation, with pain felt in the neck, back, left shoulder, and occasionally the left arm or epigastrium.

Dyspnea is frequently present, its pathogenesis being incompletely understood. Other signs and symptoms that occur with variable frequency include cough, fever, hiccups, nausea, vomiting, dizziness, malaise, and palpitations. The pericardial friction rub is the pathognomonic physical finding of pericarditis. However, it is not present in all cases, may be audible intermittently, or may change in quality from one examination to the next. It may be heard even in the presence of large effusions.

Symptoms of pericardial effusion may include chest pain, dyspnea, cough, hoarseness, nausea, and abdominal pain. A precordial discomfort

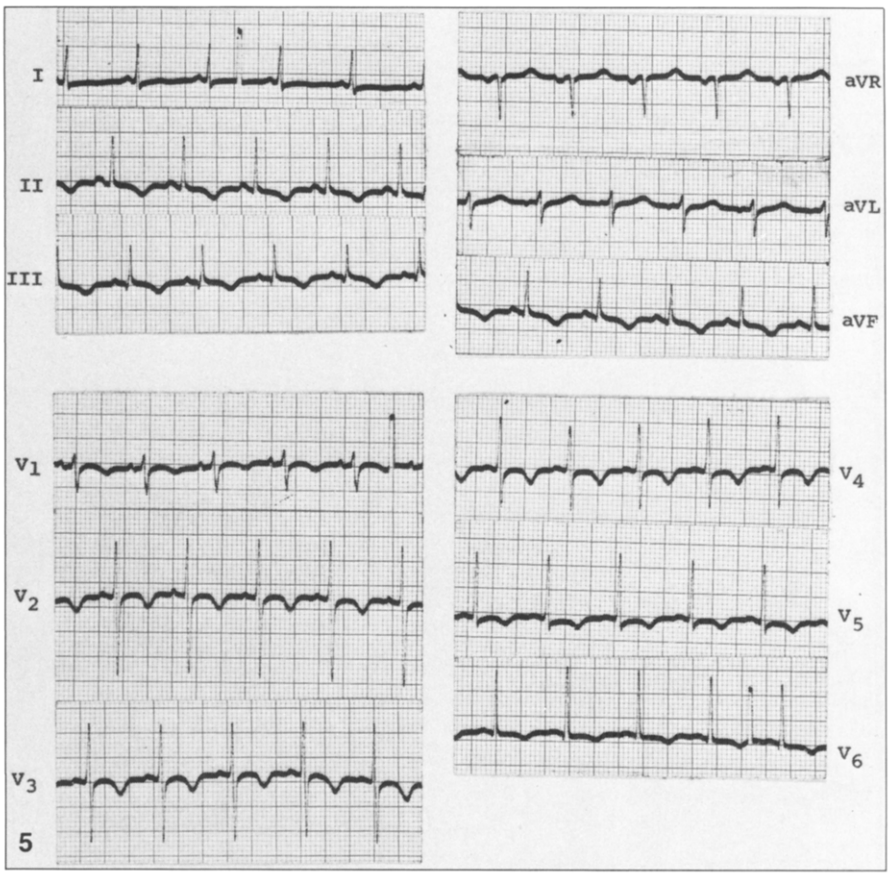

FIGURE 5. *Resolving stage of pericarditis, in which ST segment elevation has normalized, but T wave inversion persists.*

FIGURE 6. *Major causes of pulsus paradoxus other than cardiac tamponade.*

distinct from the characteristic pain of pericarditis may be present, and is probably caused by stretching of the pericardium by fluid. Tachypnea is the earliest and most common physical finding of effusion.

DIAGNOSTIC TESTS

ECG

A typical progression of ECG changes occurs during the course of acute pericarditis. This sequence has been divided into four stages. Stage 1 consists of ST segment elevation (Figure 3). This is concave upward and is usually present in all leads except aVR and V1. The height of ST segment elevation is seldom more than 5 mm above the baseline.[20] T waves are usually upright in leads in which ST segment elevation is present. ST segment depression is not seen, except in aVR. Stage 1 changes are generally present at the time of onset of pain, and may last several days.

In Stage 2, the ST segments return to the baseline, and the T waves flatten. The PR segment may become depressed. T wave inversion (Figure 4) occurs in Stage 3. Normalization of the ECG is referred to as Stage 4. However, T wave inversion (Figure 5) may persist for several months or be permanent.[10]

ECG changes of pericarditis may require distinction from those of acute myocardial infarction. The ST segment elevation of Stage 1 pericarditis is typically concave upward, whereas that of myocardial infarction is oppositely oriented. ST segment changes of pericarditis are unaccompanied by reciprocal patterns of ST depression. The T wave inversion seen in Stage 2 occurs after the ST segment returns to the baseline. In myocardial infarction, T wave inversion often accompanies ST elevation. Furthermore, T wave inversion in pericarditis is not associated with loss of R wave voltage or the appearance of Q waves, either of which may appear in myocardial infarction.

Differentiation of the ECG changes of pericarditis from those of normal variant ST segment elevation (early repolarization) may be exceedingly difficult. Complex criteria have been suggested, but some believe that these may be indistinguishable on isolated tracings.[31] Among findings that mediate against pericarditis are the absence of simultaneous PR segment changes in the limb and precordial leads, and presence of ST elevation only in the precordial leads.[32] The best discriminator may be the ratio of ST segment amplitude to T wave amplitude in lead V6. This ratio was found to be $\geq$ 0.25 only in tracings of pericarditis patients.[33]

Arrhythmias occur in the setting of pericarditis, with sinus tachycardia being the most common. Paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation also occur. However, serious and potentially serious rhythm disturbances appear to occur only in patients whose pericarditis accompanies cardiac disease, especially acute myocardial infarction.[34]

Chest Radiograph

Enlargement and alteration of the cardiac silhouette occur as a result of pericardial effusion. However, these usually do not occur until at least 250 mL of fluid has accumulated.[10] A normal chest radiograph, therefore, does not exclude the presence of a significant pericardial effusion.

Pulmonary Disease
- Chronic obstructive pulmonary disease
- Asthma
- Tracheal compression
- Tension pneumothorax
- Massive pulmonary embolus

Cardiac Disease
- Constrictive pericarditis
- Restrictive cardiomyopathy
- Right ventricular infarction

Shock

6

Echocardiogram

Echocardiography is the most sensitive technique for the detection of pericardial effusion. Effusions as small as 15 mL can be identified.[35] Pericar-

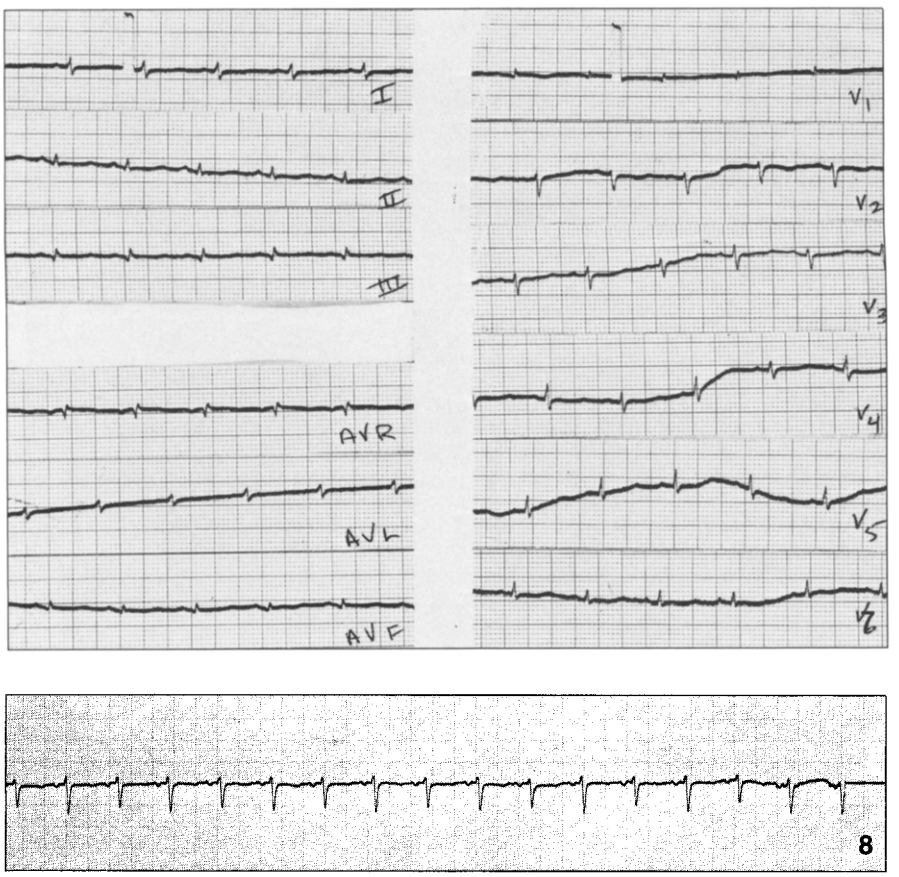

FIGURE 7. *Low voltage in a patient with pericardial effusion.*

FIGURE 8. *Electrical alternans. In this case a subtle variation of S wave amplitude can be seen in each alternate complex.*

dial fluid appears as an echo-free space between the anterior wall of the right ventricle and the chest wall and the posterior left ventricular wall and the posterior parietal pericardium. The technique has the advantages of being portable and noninvasive. Computerized tomography has been shown to be a sensitive technique for evaluating pericardial thickening and effusion.[36] It may be useful in patients in whom the echocardiogram is technically unsatisfactory. However, echocardiography remains the primary diagnostic modality in pericardial disease.

PERICARDIAL EFFUSION

Pericardial effusion may be generated by pericarditis of any cause. It may be clinically silent, produce chest discomfort by stretching the pericardium, or produce a variety of symptoms on the basis of compression of mediastinal structures. Dysphagia (due to compression of the esophagus), cough (bronchi), dyspnea (lung parenchyma), hiccups (phrenic nerve), and hoarseness (recurrent laryngeal nerve) may be produced in this way.

The development of increased intrapericardial pressure is dependent on the absolute volume of fluid, its rate of accumulation, and the physical characteristics of the pericardium. The normal unstretched pericardium can accommodate the rapid addition of only 80 to 200 mL of fluid before pressure rises precipitously.[37] Slowly developing effusions are tolerated more readily. Effusions of more than 2 L may result from hypothyroidism, for example, without producing cardiac tamponade.[2] A pericardium that is stiff because of fibrosis or tumor infiltration may accommodate less fluid than a normal, more supple pericardium.

CARDIAC TAMPONADE

Accumulation of sufficient fluid within the pericardial space may produce cardiac tamponade, characterized by elevation of intracardiac pressures, progressive limitation of left ventricular filling, and reduction of stroke volume. Tamponade may result from pericarditis of any cause. In one study detailing causes in medical patients over 17 years, the following were the leading types of pericarditis producing tamponade: malignant disease (32%), idiopathic (14%), uremia (9%), and acute myocardial infarction (9%).[38]

The classic findings of cardiac tamponade are those described by Beck in 1935, and consist of decreased systemic arterial pressure, increased venous pressure, and muffled heart sounds.[39] These findings are typical in patients in whom tamponade is due to intrapericardial hemorrhage, such as that caused by trauma, aortic dissection, or rupture of an aortic or ventricular aneurysm. In the majority of patients in whom tamponade develops more gradually, a quiet heart and severe hypotension are not seen. These patients' most common complaint is dyspnea, and the most frequent physical findings are tachypnea, jugular venous distention, and pulsus paradoxus.[38]

Pulsus paradoxus is the product of complex mechanisms, and constitutes the fall of aortic systolic pressure during inspiration. It actually represents an exaggeration of the normal inspiratory decline of left ventricular stroke volume by 7% and systemic blood pressure by 3%.[40] A paradox of up to 10 mm Hg is considered normal, but this figure, when determined by sphygmomanometric measurement, has occasionally been found to correspond to 20 to 25 mm Hg as ascertained by direct intra-arterial recording.[2] Cuff pressure, therefore, may underestimate the degree of paradox, and a 10 mm Hg paradox determined by cuff pressure should be considered significant in the appropriate clinical setting.

The recommended method of determination of pulsus paradoxus is as follows: with the patient breathing normally, the cuff should be inflated, then deflated very slowly to the point that the Korotkoff sounds are first audible intermittently. This represents the systolic pressure during expiration. The cuff then is deflated slowly until all beats are heard. This is the pressure during inspiration. The difference between the two levels is the degree of paradox. Causes of pulsus paradoxus

other than cardiac tamponade[41-46] are listed (Figure 6).

The ECG in cardiac tamponade may reveal low QRS voltage (Figure 7) and T wave flattening. Electrical alternans (Figure 8), the alternation of QRS complex amplitude from beat to beat, is a phenomenon characteristically associated with larger pericardial effusions. It is produced through the more extensive mobility of the heart in a pericardial sac filled with fluid. The presence of electrical alternans is not diagnostic of tamponade, but tamponade is more likely to develop in the setting of effusions large enough to produce this ECG entity.[2]

The echocardiogram is useful in demonstrating the presence and quantity of pericardial fluid. In addition, several types of abnormal heart wall motion and other echocardiographic findings characteristic of larger effusions and tamponade have been described.[47] However, the diagnosis of cardiac tamponade should, in general, be made clinically.

TREATMENT

Treatment should be aimed at the underlying cause whenever appropriate. Many forms of pericarditis run a self-limited course and have good prognoses. Analgesic and anti-inflammatory agents are frequently administered. Aspirin may be begun, 650 mg every three to four hours. If there is no response, indomethacin 25 to 75 mg four times a day should be given, and the dose tapered after the patient has been asymptomatic for five to seven days.

The use of corticosteroids in pericarditis is controversial. Corticosteroids are effective anti-inflammatory agents in the treatment of pericarditis. However, 10% to 20% of patients receiving such treatment develop relapsing pericarditis when steroid dosage is tapered to less than 10 to 15 mg of prednisone per day, and are difficult to withdraw from treatment.[2]

Fluid producing cardiac tamponade may be removed using percutaneous pericardiocentesis. If this cannot be done immediately, volume expansion should be undertaken with infusion of IV fluids. Volume expansion is beneficial in increasing arterial pressure, cardiac output, and renal and myocardial perfusion.[48] Administration of isoproternol may also help increase cardiac output.[10] Isoproternol administration in cardiac tamponade was found to increase cardiac output principally by increasing stroke volume, and to a lesser degree by increasing heart rate.[49] Positive pressure ventilation has been shown to depress cardiac output in experimental animals with cardiac tamponade, and should be avoided if possible.[50]

The likelihood that pericardiocentesis can safely be performed and fluid obtained is largely related to effusion size. In a series of 123 patients at Stanford in whom pericardiocentesis was performed, the procedure was successful in 93% of patients with large effusions (as estimated by echocardiogram), but only in 58% with small or moderate-sized effusions. Pericardiocentesis was likely to be either unsuccessful or complicated in patients with effusions smaller than 200 mL, those with acute traumatic hemopericardium, and those who did not demonstrate an anterior effusion on echocardiogram.[51]

SUMMARY

Pericarditis is a common entity produced by a variety of processes. The clinical examination, ECG, and echocardiogram are most useful in diagnosis. Cardiac tamponade is the most serious complication. This may also be suspected on the basis of clinical and ECG findings and confirmed by echocardiography.

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