PART I

Systems

1. Infective Endocarditis

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Outline

INTRODUCTION

Cardiac infections are classified by the affected site: endocardium, myocardium, or pericardium. Although the terms *pericarditis*, *myocarditis*, and *endocarditis* refer to inflammation in general, most cases are secondary to infectious disease.

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Infective endocarditis (IE) affects the endocardium, though inflammation may damage the cardiac valves themselves, as well as the underlying myocardium. IE more commonly affects the left side of the heart, more commonly affects males (2:1), and increases in incidence with age. The pathogenic agent is usually bacterial but may also be fungal, rickettsial, or protozoan, particularly in immunocompromised patients.

Infective endocarditis occurs when circulating pathogens adhere to the endocardium in areas of turbulent flow, particularly around cardiac valves. Host susceptibility is an integral part of the pathophysiology. Several decades ago, rheumatic fever was the most common cause of valvular lesions, and bacterial adherence to these damaged valves could occur in any age group. Now, congenital heart disease and degenerative valvular disease are the most common predisposing factors to IE, in children and the elderly, respectively. An increasing percentage of cases arise from prosthetic heart valves, which have enhanced susceptibility to infection.

When bacteremia is frequent, adherence to the endocardium may occur even in the absence of a valvular lesion, and intravenous drug users, immunocompromised patients, and those with indwelling vascular catheters or poor dental hygiene are at greater risk for IE.

The most common pathogens found in IE are gram-positive cocci, such as *Staphylococcus* species, both coagulase positive (e.g., *S. aureus*) and negative (e.g., *Staphylococcus epidermidis*), and the viridans group streptococci (*Streptococcus sanguis*, *bovis*, and *mutans*). Enterococci are also becoming increasingly common causes of IE. The clinical scenario may suggest the pathogen involved: *S. aureus* is common in intravenous drug users, viridans streptococci in patients with recent dental procedures, and gram-negative bacilli in patients following invasive genitourinary procedures.

Pathogens that are much less commonly implicated in IE include the HACEK (*Haemophilus aphrophilus*, *Haemophilus paraphrophilus*, *Haemophilus parainfluenzae*, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella kingae*) group of slow-growing gram-negative bacteria, *Bartonella*, and atypical organisms such as *Chlamydia*, *Legionella*, and fungi. Infections with these organisms may be especially difficult to detect in the acute care setting because they do not always cause fever or grow in routine blood cultures.

Once bacteria adhere to the endocardium, infection spreads toward the valves, resulting in stenotic and/or regurgitant function, and toward the myocardium, resulting in mural endocarditis, which may result in septic emboli.

CLINICAL FEATURES

The presentation of IE (Table 1.1, Figure 1.1) ranges from the well-appearing patient with nonspecific symptoms to the toxic patient in severe septic shock with multiorgan failure.

Patients with mild symptoms are often misdiagnosed with viral syndromes. Symptoms may include low-grade fever, headache, malaise, and anorexia. The presence of a new murmur may be helpful, especially in a young person, but its importance in making the diagnosis is often overemphasized. The high prevalence of a baseline murmur in older adults makes this finding rather nonspecific. Patients with a more indolent or subacute presentation may display physical findings that result from the deposition of immune complexes in end-vessels throughout the body: hematuria (due to glomerulonephritis), subungual splinter hemorrhages, or petechiae of the palate and conjunctiva. They also include the so-called classic stigmata of IE: *Roth spots* (exudative lesions on the retina), *Janeway lesions* (painless erythematous lesions on the palms and soles), and *Osler nodes* (painful violet lesions on the fingers or toes). These signs are present in the minority of patients with IE; they should be sought on examination, but their absence does not rule out the diagnosis.

As the clinical presentation becomes more severe, it is characterized by the septic and mechanical complications of endocarditis. In left-sided endocarditis, this may include signs of systemic embolization, which may occur in any organ system. Infections that initially appear to be focal or localized may, in fact, be a result of septic emboli. Examples include stroke and spinal cord syndromes, mycotic aneurysms, osteomyelitis,

Table 1.1 Clinical Features: Infective Endocarditis

Organisms	• *Staphylococcus aureus* • *Streptococcus viridans* • *Enterococcus* • *Staphylococcus epidermidis* • *Streptococcus bovis* • HACEK
Signs and Symptoms	Fever, malaise, chest/back pain, cough, dyspnea, arthralgias, myalgia, neurologic sypmtoms, weight loss, night sweats
Laboratory and Radiologic Findings	**Duke Clinical Criteria** 2 Major *or* 1 Major + 3 Minor *or* 5 Minor **Major (Microbiology)**: • Typical organisms × 2 blood cultures (*S. viridans*, *S. bovis*, HACEK, *S. aureus*, or enterococcus) with no primary • Persistent bacteremia (≥12 hours) 3/3 or 3/4 positive blood cultures **Major (Valve)**: • Positive echocardiogram • New valve regurgitation **Minor**: • Predisposing heart condition or IDU • Fever ≥ 38°C (100.4°F) • Vascular phenomenon (arterial embolism, mycotic aneurysm, intracerebral bleed, conjunctival hemorrhage, Janeway lesions) • Immune phenomenon (glomerulonephritis, Osler node, Roth spot, rheumatoid factor) • Positive blood culture not meeting above criteria • Echocardiogram – abnormal but not diagnostic

IDU, injection drug use.

epidural abscesses, septic arthropathies, necrotic skin lesions, and cold, pulseless extremities. Mycotic aneurysms most often occur in the middle cerebral artery and may cause meningitis, headaches, or focal neurological deficits. When significant mechanical failure of the mitral or aortic valve occurs, signs and symptoms of severe acute left-sided heart failure, such as pulmonary edema and hypotension, may occur.

Right-sided endocarditis is frequently associated with septic pulmonary emboli. These may cause respiratory symptoms that mimic the presentation of pneumonia or pulmonary embolism. Mechanical failure of the valves usually results in regurgitant disease with signs and symptoms of acute right-sided heart failure.

Other serious sequelae of endocarditis include intravascular hemolysis, which results in hemoglobinemia, hemoglobinuria, and jaundice. In patients with mural endocarditis, abscesses around the annulae of the cardiac valves may result in conduction blocks and bradydysrhythmias. Finally, ventricular wall rupture may lead to cardiac tamponade or hemorrhagic shock.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of IE is vast, especially in its more indolent presentations. It includes both acute and chronic infections, malignancies, and a wide spectrum of inflammatory and autoimmune disorders. IE should be suspected in any febrile patient with a history of:

- injection drug use
- rheumatic heart disease
- valvular insufficiency
- indwelling catheters
- pacemakers
- prosthetic heart valves
- congenital heart disease
- prior endocarditis

In patients with more severe signs and symptoms, the differential diagnosis includes other life-threatening causes of:

- severe sepsis with end-organ dysfunction (pneumonia, urinary tract infection, peritonitis, or soft-tissue infections)
- left- and right-sided heart failure (myocardial infarction, valvular incompetence or stenosis, pulmonary embolism, or aortic dissection)
- systemic embolization (carotid stenosis, vascular dissection, or cardiac dysrhythmias)

When these complications occur in febrile patients, the diagnosis of IE should be suspected, particularly in those patients at risk. Alternatively, when these complications occur in the absence of fever or risk factors, the underlying diagnosis of IE is highly unlikely.

LABORATORY AND RADIOGRAPHIC FINDINGS

The majority of tests available in an acute care setting are insufficient to confirm or eliminate a suspected diagnosis of IE. Results of routine blood tests, including inflammatory markers (complete blood count [CBC], erythrocyte sedimentation rate [ESR], C-reactive protein [CRP]) lack specificity. The definitive diagnosis is made, in large part, by blood culture. It is important that blood cultures be drawn with good technique and sufficient volume (10 mL) and at multiple sites to enhance diagnostic sensitivity.

The administration of empiric antibiotics in ill-appearing patients with suspected IE should not be unduly delayed, though it is essential to obtain blood cultures prior to giving antibiotics. Special cultures are necessary for the following organisms: HACEK, *Legionella*, *Mycoplasma*, nutritionally variant strep (*Abiotrophia*), *Bartonella*, *Coxiella*, *Brucella*, gonococci, *Listeria*, *Nocardia*, corynebacteria, and mycobacteria.

Many of the positive findings in diagnostic evaluation may mislead clinicians toward a focal process, rather than direct them toward a unifying diagnosis. For example, an abnormal urinalysis may lead to a diagnosis of cystitis or glomerulonephritis, infiltrates on a chest x-ray may be interpreted as consistent with pneumonia, or findings on a lumbar puncture may lead to a diagnosis of meningitis.

Electrocardiography is seldom helpful in establishing the diagnosis of IE. The most common electrocardiogram abnormality in IE is sinus tachycardia. Signs of acute right heart strain, such as right bundle branch block and rightward axis, may accompany right-sided endocarditis and pulmonary emboli. Severe heart blocks may represent an infection that has moved into the myocardium and around the valvular annulae.

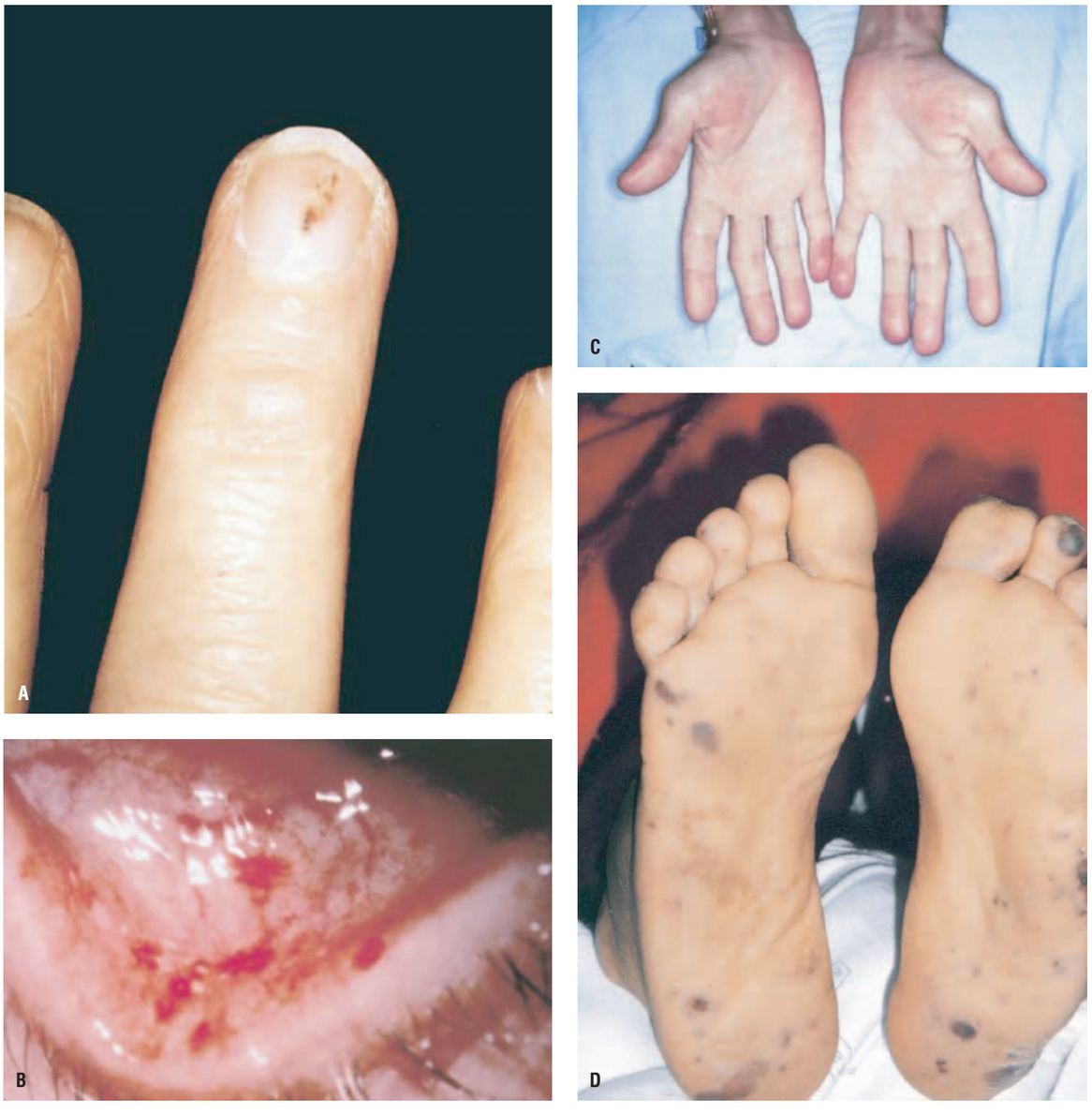

Figure 1.1 Classic physical examination findings in IE. (A) Splinter hemorrhages. (B) Conjunctival petechiae. (C) Osler's nodes. (D) Janeway lesions. Images from Mylonakis E and Calderwood SB. Infective endocarditis in adults. N Engl J Med, 2001;345(18):1318–30. Copyright © 2008 Massachusetts Medical Society. All rights reserved.

Echocardiography is essential in establishing the definitive diagnosis of IE; however, its utility in the emergency department (ED) is more related to its ability to detect life-threatening complications such as pericardial effusion, cardiac tamponade, and valvular rupture.

TREATMENT AND PROPHYLAXIS

Empiric therapy toward likely bacterial pathogens is indicated when the diagnosis of endocarditis is strongly suspected, and antibiotic selection should occur with current and local patterns of sensitivity in mind (Table 1.2). The duration of therapy is typically long, up to and, in some cases, exceeding 6 weeks. It may be appropriate to withhold antibiotics pending culture results in patients with chronic, intermittent fevers who otherwise appear well, provided that close follow-up is available.

Antibiotic prophylaxis should be administered to patients at risk for IE prior to certain invasive procedures (Table 1.3). Fortunately, most procedures routinely performed in the ED do not require prophylaxis, except for emergent upper endoscopy (only if sclerotherapy of esophageal varices is performed), incision and drainage of gingival abscesses, and urethral catheterization in the setting of urinary tract infections. In these situations, patients with known valvular

Table 1.2 Empiric Treatment for Infective Endocarditis

Patient Category	Therapy Recommendation
Adults	nafcillin 2 g IV q4h *or* vancomycin 1 g IV q12h (if high prevalence of resistant staph or PCN allergy) *and* gentamicin 1–1.7 mg/kg IV q8h
Children	nafcillin 50 mg/kg IV q6h *or* vancomycin 10–15 mg/kg IV q12h (if high prevalence of resistant staph or PCN allergy) *and* gentamicin 1.5–2.5 mg/kg IV q8h
Pregnant Women	nafcillin 2 g IV q4h *or* vancomycin 1 g IV q12h (if high prevalence of resistant staph or PCN allergy) *and* ceftriaxone 2 g IV q12h
Immunocompromised	As above, depending on age and pregnancy status

PCN, penicillin.

Table 1.3 Antibiotic Prophylaxis for Invasive Procedures in High Risk Patients

Patient Category	Recommended Antibiotics Prophylaxis
Adults	ampicillin 2 g IV/IM × 1 *or* vancomycin (if PCN allergic) 1 g IV × 1 *and* gentamicin 1.5 mg/kg IM/IV × 1
Children	ampicillin 50 mg/kg IV/IM × 1 *or* vancomycin (if PCN allergic) 20 mg/kg IV × 1 *and* gentamicin 1.5 mg/kg IM/IV × 1
Pregnant Women	ampicillin 2 g IV/IM ×1 *or* vancomycin (if PCN allergic) 1 g IV × 1 *and* ceftriaxone 2 g IV × 1
Immunocompromised	As above, depending on age and pregnancy status

PCN, penicillin.

disease or a prior history of IE should be given prophylaxis tailored to the typical pathogens associated with the organ system involved.

COMPLICATIONS AND ADMISSION CRITERIA

The treatment of septic and mechanical complications of endocarditis can be challenging. Cardiac dysrhythmias can be treated according to advanced cardiovascular life support (ACLS) guidelines. In cases of suspected acute valvular dysfunction, emergent echocardiography and consultation with a cardiothoracic surgeon and cardiologist are indicated. In cases of septic emboli, anticoagulation with heparin is not recommended because it has no effect on decreasing the rate of subsequent embolization and because the risk of hemorrhagic transformation is particularly high in these patients. Limb-threatening emboli (e.g., a cold, pulseless extremity) may require revascularization with interventional or surgical techniques, such as the administration of local fibrinolytics.

Patients for whom the diagnosis of IE is being considered should generally be admitted for further evaluation and parenteral antibiotics. In cases in which suspicion for IE is lower, it may be appropriate to discharge certain febrile but otherwise well-appearing patients home with blood cultures pending, provided that follow-up is available within 48 hours. Patients with septic or mechanical complications of IE should be managed in a monitored setting, preferably one in which cardiothoracic surgical intervention is readily available.

PEARLS AND PITFALLS

1. Echocardiography is recommended prior to discharge in all cases of suspected myocarditis, pericarditis, endocarditis, and acute rheumatic fever.
2. Endocarditis is important to consider in any febrile patient with a preexisting valvular abnormality.
3. Mechanical complications of IE may require emergent surgical intervention. Diagnosis of such complications should be made by echocardiography.

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2. Myocarditis and Pericarditis

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Outline

INTRODUCTION

Cardiac infections are classified by the affected site: endocardium, myocardium, or pericardium. As the pathophysiology, clinical presentation, differential diagnosis, and treatment of myocarditis and pericarditis overlap significantly, these will be discussed together.

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Myocarditis is an inflammation of the myocardium; the term myopericarditis describes the frequent additional involvement of the pericardium. Pericarditis involves only the pericardium. Isolated myocarditis is often relatively asymptomatic and therefore frequently misdiagnosed. Thus, the true incidence is unknown, although autopsy studies have demonstrated occult myocarditis in up to 1% of the general population. For unclear reasons, young men more frequently develop myocarditis as well as pericarditis.

The pericardium provides a protective barrier and is composed of two layers: visceral and parietal. The visceral layer is firmly attached to the epicardium, whereas the parietal layer moves freely within the mediastinum. Approximately 20 mL of fluid is normally present within the pericardial sac. Fluid accumulation within the pericardial sac may result in cardiac tamponade if the pericardium does not have sufficient time to stretch, as compliance increases slowly over time. Thus, the rate rather than the absolute amount of fluid accumulation in the pericardial sac is the most important determinant of tamponade physiology.

Cardiac infections may spread directly from one intracardiac region to another (from endocardium toward pericardium or vice versa). Alternatively, pleural or mediastinal infections can extend into the pericardium and cause cardiac infections.

Infectious Causes of Myocarditis

Infectious causes of myocarditis include viruses, bacteria, fungi, rickettsia, spirochetes, and parasites. In all types of infection, myocardial damage may result from destructive effects of the invasive pathogen or from immune-mediated lysis of infected cells. In developed nations, viruses represent the most common infectious cause. Several viruses cause myocarditis, with coxsackieviruses representing more than 50% of confirmed cases of viral myocarditis. Other common agents include influenza virus, echovirus, herpes simplex virus (HSV), varicella-zoster virus (VZV), Epstein-Barr virus (EBV), cytomegalovirus (CMV), and the hepatitis viruses. Human immunodeficiency virus (HIV) infection may also cause myocarditis, either directly from HIV-induced cytotoxicity during any phase of the infection, or indirectly as a result of other opportunistic infections. Most cases of viral myocarditis are preceded by an upper respiratory infection by 1–2 weeks.

Bacterial myocarditis is often caused by direct extension from infected endocardial or pericardial tissue. The most common causative organisms in these cases mirror those most commonly causing bacterial endocarditis or pericarditis. Certain exotoxin-mediated bacterial illnesses, such as diphtheria, may also cause myocarditis.

Other pathogenic organisms associated with myocarditis include rickettsia, spirochetes, and parasites. Tick-borne illnesses caused by rickettsia (Rocky Mountain spotted fever, Q fever, and scrub typhus) and spirochetes (Lyme disease) have all been associated with myocarditis. Immunocompromised patients may develop myocarditis secondary to toxoplasmosis. Parasitic causes of myocarditis in immigrant populations include Chagas disease and trichinosis.

Noninfectious Causes of Myocarditis

There are a variety of noninfectious causes of myocarditis, including autoimmune disorders, medications, and environmental toxins. Autoimmune causes include systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), sarcoidosis, and various vasculitides (Kawasaki disease and giant cell arteritis). A variety of drugs and chemotherapeutics can directly induce myocardial inflammation, including cocaine, amphetamines, lithium, phenothiazines, zidovudine (AZT), chloroquine, and doxorubicin. Hypersensitivity reactions to penicillin and sulfonamides may trigger inflammatory changes in the myocardium, resulting in myocarditis. Environmental toxins such as carbon monoxide, lead, and arsenic, as well as stings from spiders, scorpions, and wasps, can also result in myocardial inflammation.

Table 2.1 Important Causes of Myocarditis and Pericarditis

Idiopathic Viral Infections	**Mycobacterial Infections**	**Malignancy Medications**
Coxsackievirus A and B	*Mycobacterium tuberculosis*	Penicillin
Echoviruses	**Parasitic Infections**	Sulfa drugs
Adenoviruses	Chagas disease	Procainamide
HIV	Trichinosis	Hydralazine
Bacterial Infections	Toxoplasmosis	Isoniazid
Gram-positive species	**Fungal Infections**	Phenytoin
Gram-negative species	*Histoplasma capsulatum*	Chemotherapeutic agents
Anaerobes	*Aspergillus* species	**Environmental/Toxins**
Mycoplasma	**Autoimmune Mediated**	Cocaine
Rickettsial Infections	Acute rheumatic fever	Amphetamines
RMSF	Dressler's syndrome	Carbon monoxide
Q fever	Systemic lupus erythematosus	Lead
Scrub typhus	Rheumatoid arthritis	Stings/bites
Spirochetes	Vasculitis (e.g., Kawasaki)	**Radiation Exposure Metabolic Disorders**
Lyme disease	Sarcoidosis	Hypothyroidism
Syphilis	Postvaccination	Uremia (dialysis-related)
	Trauma or Surgery	
	Postpericardiotomy syndrome	

Adapted from Ross AM, Grauer SE. Acute pericarditis. Evaluation and treatment of infectious and other causes. Postgrad Med. 2004 Mar;115(3):67–75.
RMSF, Rocky Mountain spotted fever.

Table 2.2 Clinical Features: Myocarditis and Pericarditis

Signs and Symptoms: Adults	• Nonspecific – fever, malaise, night sweats • Chest pain (substernal, sharp, stabbing, squeezing, or pleuritic) • Friction rub (only if pericarditis) • Congestive heart failure L-sided: DOE, near syncope, rales R-sided: JVD, HSM, peripheral edema • Tamponade: Syncope, Beck's triad, pulsus paradoxus • Dysrhythmia or conduction disturbance – palpitations, light-headedness, or syncope • Bacterial: Pneumonia – cough, dyspnea, hemoptysis Mediastinitis – odyno/dysphagia, sepsis Endocarditis – murmur, septic emboli, rash • Tuberculous – TB exposure, cachexia, pleurisy • Lyme/Rickettsial – tick exposure, rash, arthritis
Signs and Symptoms: Infants	• As above • Nonspecific – lethargy, poor feeding, cyanosis
Laboratory and ECG Findings	• Leukocytosis, elevated C-reactive protein level, ESR, and cardiac enzymes • ECG findings include: Sinus tachycardia and nonspecific ST-T changes Diffuse ST-segment elevation Decreased QRS amplitude and Q waves Ventricular ectopy • Occasional conduction disturbances, BBB, or tachydysrhythmias

DOE, dyspnea on exertion; HSM, hepatosplenomegaly; TB, tuberculosis.

Infectious Causes of Pericarditis

Acute pericarditis, like myocarditis, is most frequently caused by viruses, including coxsackieviruses, echoviruses, influenza, EBV, VZV, HIV, mumps, and hepatitis (Table 2.1). Again, upper respiratory infection generally precedes pericardial involvement, and males older than 50 years are at highest risk.

Tuberculous pericarditis is prevalent in developing nations and in immigrant populations. It is caused by hematogenous or lymphatic spread of mycobacteria.

Bacterial pericarditis is fortunately rare. It most often results from direct extension of adjacent pulmonary, mediastinal, or endocardial infection, or iatrogenic inoculation following cardiac surgery. These patients usually appear toxic, unlike most patients with viral pericarditis.

Noninfectious Causes of Pericarditis

Noninfectious causes of acute pericarditis include uremia, trauma, malignancy (lymphoma or cancers of the breast, lung, or kidney), radiation, chemotherapy, drug reactions (penicillin or minoxidil), and autoimmune disorders (SLE, RA, or Dressler's syndrome after myocardial infarction or cardiac surgery). See Table 2.1.

CLINICAL FEATURES

The clinical presentation of infectious myocarditis and pericarditis varies depending on the virulence of the infective agent and the severity of the host immune response (Table 2.2). Most patients with acute myocarditis or pericarditis have mild symptoms, which include low-grade fever, malaise, and substernal chest pain. The pain is often described as sharp, stabbing, squeezing, or pleuritic. The pain is commonly postural: lying supine exacerbates the pain, whereas sitting relieves it. Patients may complain of dyspnea on exertion, particularly when presenting in heart failure or with cardiac tamponade. Patients with pericarditis may also complain of cough, odynophagia, or dysphagia, presumably secondary to the spread of the inflammatory process to adjacent structures. In the event of associated dysrhythmia, patients may complain of palpitations, light-headedness, or syncope. Neonates and infants frequently present with nonspecific symptoms, such as fever, respiratory distress, cyanosis, or poor feeding.

Physical exam findings in myocarditis and pericarditis depend on the severity of illness and presence of complications. The classic finding in acute pericarditis is a pericardial friction rub, which is best heard at the apex while the patient leans forward or lies prone. Insensitive findings for cardiac tamponade include pulsus paradoxus (>10 mm Hg decline in systolic blood pressure with inspiration) and Beck's triad (jugular-venous distension [JVD], distant heart sounds, and hypotension). In cases of myocarditis, signs of left-sided heart failure may include tachypnea, hypoxia, and pulmonary rales. Right-sided heart failure may present with JVD, hepatosplenomegaly, and peripheral edema. Occasionally, patients with acute myocarditis present in acute heart failure without associated fever or chest pain.

Bacterial Myopericarditis

Patients with bacterial myopericarditis generally appear toxic and frequently have evidence of lower respiratory, endocardial, or mediastinal infection. The diagnosis should be

suspected whenever septic-appearing patients have a history of cardiac surgery, or present with chest pain, congestive heart failure (CHF), or tamponade.

In contrast, tuberculous pericarditis generally presents as an indolent illness with nonspecific symptoms such as fever, night sweats, weight loss, and fatigue. However, these patients occasionally appear toxic. This diagnosis should be suspected in all cases of pericarditis associated with possible exposure to tuberculosis.

Spirochete and Rickettsial Myopericarditis

Lyme or rickettsial myopericarditis should be considered in all symptomatic patients living in endemic regions, as most patients do not recall a history of tick exposure. Any cardiac inflammation associated with rash and arthritis should heighten the suspicion of tick-borne illness.

Clinical Course of Myocarditis and Pericarditis

Most cases of acute myocarditis resolve spontaneously without sequelae. Recurrent or chronic myocarditis, however, can progressively damage the myocardium, leading to dilated cardiomyopathy and chronic congestive heart failure in up to 30% of patients. If symptomatic heart failure results from myocardial infection, the 5-year mortality exceeds 50%.

The prognosis of acute pericarditis depends on the etiology: viral pericarditis is frequently benign and transient, whereas malignant pericardial effusions carry an exceedingly poor prognosis. In cases of recurrent pericarditis, prognosis is worsened by chronic fibrosis and thickening, which cause constrictive pericarditis and diminished cardiac output.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a patient complaining of chest pain or dyspnea in an emergent or urgent setting should include:

- aortic dissection
- pulmonary embolism
- pneumothorax and tension pneumothorax
- acute coronary syndrome
- cardiac dysrhythmias
- esophageal perforation
- myopericarditis
- mediastinitis
- pneumonia
- bronchitis
- gastroesophageal reflux disease
- costochondritis
- panic attack
- herpes zoster
- pleurisy

The diagnosis of myopericarditis should be strongly considered whenever chest pain, dyspnea, dysrhythmias, heart failure, or cardiac tamponade accompanies recent upper respiratory symptoms or in association with risk factors for myopericarditis (autoimmune disorders, malignancy, renal failure, recent cardiac surgery, or exposure to toxins, tuberculosis, or ticks).

Misdiagnosis of acute myopericarditis as ST-segment elevation myocardial infarction may result in inappropriate administration of fibrinolytic agents, beta-blockers, and heparin. The differentiation of myopericarditis and acute coronary syndrome should not be made on historical features alone, as fever, cough, and pleuritic chest pain may be seen in both conditions. Electrocardiographic findings are more reliable because myopericarditis generally occurs diffusely, unlike acute coronary syndrome, which involves the territory of a specific coronary artery.

Differentiating myopericarditis from adjacent infectious or inflammatory processes is frequently difficult. In fact, bacterial myopericarditis frequently occurs in conjunction with endocarditis, pneumonia, empyema, or mediastinitis. Furthermore, inflammatory conditions (such as SLE, RA, or vasculitis) and toxins (including medications and environmental exposures) may cause cardiac, pulmonary, or aortic disease. Electrocardiography, echocardiography, and advanced imaging are frequently necessary to differentiate these conditions.

LABORATORY AND RADIOGRAPHIC FINDINGS

In the acute care setting, routine studies in patients presenting with chest pain or dyspnea include pulse oximetry, chest x-ray, and electrocardiography. Unfortunately, these tests are insensitive and nonspecific in acute myocarditis. Electrocardiography (ECG) is useful in detecting early cases of pericarditis.

Laboratory findings in acute myocarditis and pericarditis may include leukocytosis, elevated C-reactive protein levels, and increased erythrocyte sedimentation rate (ESR). Elevated cardiac markers may be seen in severe cases of myocarditis; this may cause difficulty in distinguishing cases of acute myocarditis from acute coronary syndrome. Fortunately, ECG findings are usually distinct in each entity. Although viral serology may reveal a causative agent, these results will rarely be available acutely (with the possible exception of rapid influenza or mononucleosis tests). Skin testing and acid-fast bacilli testing of the sputum should be performed in suspected tuberculous pericarditis, and blood cultures should be obtained in all toxic-appearing patients.

Common ECG findings in myocarditis include sinus tachycardia and nonspecific ST-T changes (Figure 2.1). When present, ST-segment elevation is frequently diffuse. Other characteristic findings include decreased QRS amplitude and the development of Q waves. Ventricular ectopy is common. Occasionally, conduction system disturbances, bundle branch blocks, or tachydysrhythmias may develop as well.

Electrocardiography findings can be diagnostic of pericarditis (Figure 2.2). Acute pericarditis causes a characteristic progression of ECG findings through four distinct phases. The first stage may last for days and is characterized by diffuse ST elevation in all leads except avR and V1. PR segment depression is another common finding during the first stage and may precede the ST elevation. The second stage, which occurs days to weeks after the first, involves normalization of the ST segment with T wave flattening. The third stage involves diffuse T wave inversion without Q wave formation, and the fourth stage is characterized by ECG normalization. Electrical alternans, characterized by alternating voltage of the P wave, QRS segment, and T wave, is a rare but pathognomonic finding of cardiac tamponade. Other ECG findings in tamponade