Chapter 1

Infective Endocarditis

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Outline

Introduction

Infectious endocarditis (IE) is a difficult diagnosis to make in the emergency setting. Early diagnosis and management requires an understanding of endocarditis risk factors, typical and atypical clinical presentations, and current diagnostic and empiric treatment strategies.

Epidemiology and Microbiology

In developed countries, the incidence of IE is roughly 5 cases per 100,000 persons per year. It more commonly affects males (2:1). Well-recognized risk factors for IE include presence of a prosthetic heart valve (which carry an annual incidence of approximately 1%), congenital heart disease, endocardiac devices, injection drug use (see Chapter 61), and a prior history of endocarditis. Rheumatic heart disease is now an uncommon predisposing risk factor in the United States. However, in modern series, there is no easily identifiable risk factor for underlying valve damage in approximately 50% of endocarditis cases. Such cases are believed to be due to age-related degenerative valve disease and subtle immunosuppresion from diabetic endocarditis and other factors. Health-care associated cases, often in the elderly, account for a growing proportion of endocarditis in the United States.

Infective endocarditis occurs when circulating pathogens adhere to damaged endothelium and form a vegetation, usually on or around a cardiac valve. Abnormal turbulent flow and damaged endothelium lead to fibrin and platelete deposition which presents a nidus for bacterial infection during bacteremia. In the setting of frequent bacteremia, such as intravenous drug use and dental infection, IE may occur even without an identifiable pathologic valvular lesion. Growth of the infected vegetation eventually leads to valve destruction and impaired function, typically regurgitation, and eventually heart failure. Invasion of the myocardium can lead to paravalvular abscess and heart block. Large, mobile vegetations are associated with embolization and metastatic infection (see below).

The list of pathogens that have been reported to cause IE is enormous and includes fungi and protozoa. The most common etiolgies, however, are gram-positive cocci, including *Staphylococcus* species, both *S. aureus* and coagulase negative *Staphylococcus*, and Streptococcal species, particularly viridans Streptococci and group D *Streptococcus*. *S. aureus* is both the most common etiology and the pathogen most often associated with metastatic complications. *Enterococcus* is common in the elderly. The clinical setting may suggest the pathogen involved: *S. aureus* is the most common in injection drug users, viridans Streptococci in patients with recent dental procedures, and gram-negative bacilli in patients that have undergone invasive genitourinary procedures.

Pathogens that are 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 fastidious bacteria, *Bartonella*, chlamydia, *Legionella*, and fungi. Infections with these organisms may be difficult to detect because they do not always grow in routine blood cultures.

Clinical Features

The presentation of IE (see Table 1.1 and Figure 1.1) ranges from the well-appearing patient with non-specific symptoms to the toxic patient in severe septic shock with multi-organ failure. Symptoms are often frustratingly non-specific, and may include low-grade fever, malaise, myalgias, headache, and anorexia. Patients with mild symptoms are often misdiagnosed as having a viral syndrome. Approximately 80% of patients with IE will have a fever during their initial emergency department stay. The presence of a new murmur may be helpful;

Table 1.1 Clinical Features: Infective Endocarditis

Pathogens	*Staphylococcus aureus* *Staphylococcus epidermidis* Viridans *Streptococcus bovis* *Enterococcus* spp. HACEK Immuno-compromised: fungal, rickettsial, protozoan
Signs and symptoms	Fever, malaise, weight loss, night sweats, myalgias, headache, chest/neck/back pain, cough, dyspnea, hepatosplenomegaly, hematuria, arthritis, edema, neurologic symptoms, jaundice, rash.
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*) 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 – intravenous drug use.

however, the high prevalence of a baseline murmur in older adults makes this finding non-specific.

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. These findings include the 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), as well as hematuria (due to glomerulonephritis), subungual splinter hemorrhages, or petechiae of the palate and conjunctiva. These subtle signs of IE should be sought on examination; however, they are actually quite uncommon and their absence does not rule out IE.

In left-sided endocarditis, arterial embolization may occur in any organ system. The central nervous system is the most common location. Infections that initially appear to be focal or localized, particularly when due to *S. aureus*, may actually be the result of septic emboli from IE. Examples include stroke and spinal cord syndromes, mycotic aneurysms, osteomyelitis, epidural abscesses, septic arthropathies, necrotic skin lesions, and cold, pulseless extremities. Mycotic aneurysms may cause meningitis, headaches, or focal neurological deficits. Destruction of the mitral or aortic valve can cause acute respiratory failure and cardiogenic shock. Right-sided endocarditis may present with septic pulmonary emboli, which cause respiratory symptoms that may be mistaken for pneumonia or pulmonary embolism. Mechanical failure of the pulmonic or tricuspid valves can cause signs and symptoms of acute right-sided heart failure.

Other serious sequelae of endocarditis include intravascular hemolysis, and disseminated intravascular coagulation. Abscesses around the annulae of the cardiac valves may result in conduction blocks and bradydysrhythmias. Ventricular wall rupture may lead to cardiac tamponade or hemorrhagic shock, and extension into the coronary arteries may cause acute coronary syndrome.

Differential Diagnosis

The differential diagnosis of IE includes both acute and chronic infections, malignancies, and a wide spectrum of inflammatory and autoimmune disorders. However, IE should be suspected in any febrile patient with the following risk factors:

- injection drug use
- rheumatic heart disease
- valvular insufficiency
- indwelling catheter
- pacemaker

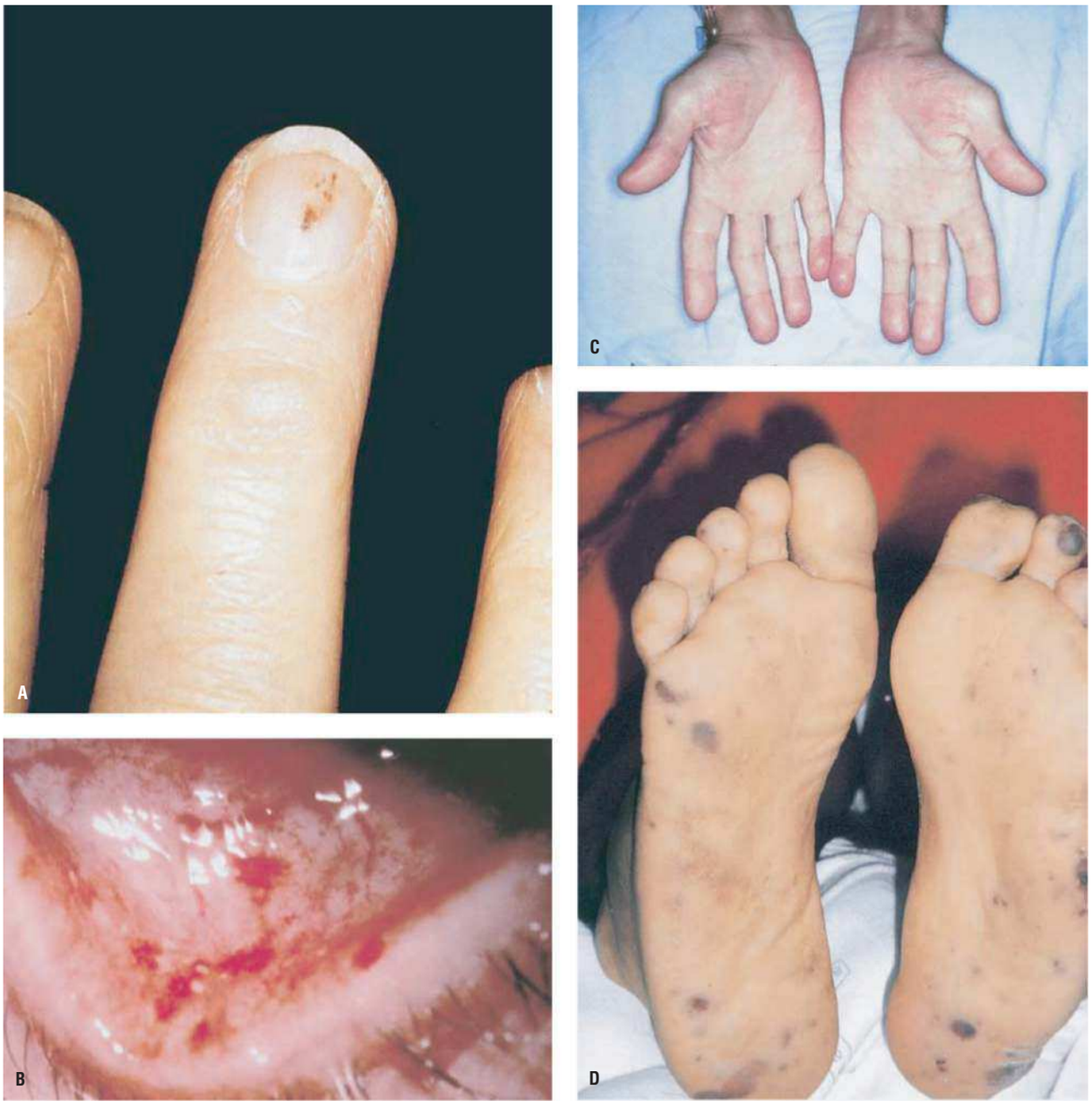

Figure 1.1 Classic physical examination findings in IE. Splinter hemorrhages (A); conjunctival petechiae (B); Osler nodes (C); and Janeway lesions (D).
Images from E. Mylonakis and S. B. Calderwood, Infective endocarditis in adults. *N. Engl. J. Med.*, 2001; 345(18): 1318–30.

- prosthetic heart valve
- congenital heart disease
- prior endocarditis

In more severe cases, the differential diagnosis will depend on the presenting signs and symptoms:

- severe sepsis with end-organ dysfunction: pneumonia, urinary tract infection, peritonitis, soft-tissue infections, and meningitis
- left- or right-sided heart failure: myocardial infarction, acute myocarditis, decompensated valvular disease, pulmonary embolism, or aortic dissection
- systemic embolization: carotid stenosis, vascular dissection, or cardiac dysrhythmias
- altered mental status with fever: meningitis, encephalitis, brain abscess

Laboratory and Radiographic Findings

Blood cultures are a crucial basis for the definitive diagnosis of IE. Thus, it is important for emergency providers to obtain blood cultures prior to giving antibiotics whenever IE is suspected. At least two and preferably three sets of blood cultures should be drawn with aseptic technique, be of

sufficient volume (10 mL), and be drawn at multiple sites. The sensitivity of three sets of blood cultures approaches 90% in patients who have not received antibiotics. Serologies for Bartonella, Brucella, and Coxiella Burnetii (Q fever) may be indicated if standard cultures are negative. Other routine blood tests such as inflammatory markers (complete blood count [CBC], erythrocyte sedimentation rate [ESR], C-reactive protein [CRP]) lack specificity.

Endocarditis produces abnormal findings on standard diagnostic tests that can lead the clinician to an incorrect initial 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 pneumonia, or abnormalities on a lumbar puncture may lead to a diagnosis of primary meningitis.

Electrocardiography (ECG) is seldom helpful in establishing the diagnosis of IE. The most common ECG abnormality in IE is sinus tachycardia. A valve ring abscess can produce heart block, particularly an elongating PR interval. Cardiac ischemia may result if IE extends into a coronary artery lumen.

Like blood cultures, echocardiography is an essential test in establishing the definitive diagnosis of IE. However, its main utility in the emergency setting is in the detection of life-threatening complications such as pericardial effusion, cardiac tamponade, and valvular rupture. Transthoracic echocardiography is useful if positive for a clear-cut vegetation; however, transesophageal echocardiography has higher sensitivity and is generally required in suspected IE if the transthoracic echocardiogram is negative.

The Duke Criteria (see Table 1.1) are a widely accepted, structured diagnostic tool for assisting in the often challenging diagnosis of IE. However, these criteria have limited utility in the emergency setting. Emergency providers must maintain constant vigilance for IE, have a low threshold for obtaining blood cultures and echocardiography in suspicious cases, and must exercise judgment in when to admit patients for empiric therapy.

Treatment and Prophylaxis

Empiric therapy targeting common IE bacterial pathogens is indicated when the diagnosis is strongly suspected. The empiric regimen should be tailored to whether or not there is a prosthetic valve, and, when possible, to the current hospital antibiogram (see Table 1.2). The duration of therapy is typically 4 to 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 was previously recommended to all patients at risk from IE prior to certain invasive dental, gastrointestinal, and genitourinary procedures; however, this practice has now become controversial, with conflicting guidelines in the United States and Europe. While most procedures routinely performed in the emergency department do not require prophylaxis, prophylaxis should be strongly considered for dental or skin abscess incision and drainage (see Table 1.3) or skin infections (with vancomycin 20mg/kg IV × 1) in very high risk patients: those with a prior history of IE; prosthetic valve; heart transplant with abnormal valve function; repaired congenital heart disease.

Table 1.2 Empiric Treatment for Infective Endocarditis

Patient Category	Empiric Therapy Recommendation*
Adults	**Native valve:** Vancomycin 15–20 mg/kg/dose IV every 8–12 hours *and* Ceftriaxone 2 g IV every 24 hours (alternate: ciprofloxacin 400 mg IV every 12 hours) **Prosthetic valve:** Vancomycin 15–20 mg/kg/dose IV every 8–12 hours *and* Gentamicin 1 mg/kg IV every 8 hours *and* Rifampin 300 mg PO/IV every 8 hours
Children	Vancomycin 15–20 mg/kg/dose IV every 6 hours *and* Gentamicin 1.5–2.5 mg/kg IV every 8 hours
Pregnant women	Vancomycin 15–20 mg/kg/dose IV every 8–12 hours *and* Ceftriaxone 2 g IV every 24 hours *and* Rifampin 300 mg PO/IV every 8 hours (if prosthetic heart valve)
Immunocompromised	As above, depending on age and pregnancy status

* Vancomycin and gentamicin dosing may need to be adjusted based on renal function and ideal body weight. Trough monitoring with both agents is strongly recommended. Rifampin has many clinically important drug–drug interactions and may require other drug-level monitoring.

IV – intravenous.

Complications and Admission Criteria

The treatment of septic and mechanical complications of endocarditis can be challenging. In cases of suspected acute valvular dysfunction with pump failure, emergent echocardiography and consultation with a cardiothoracic surgeon and cardiologist are indicated. Anticoagulation with heparin is not recommended for septic emboli because it does not reduce further embolization and the risk of hemorrhagic transformation is very high. 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 suspected should generally be admitted for further work-up and empiric intravenous antibiotics. In selected cases, it may be appropriate to discharge febrile but otherwise well-appearing patients home with blood cultures pending, provided that reliable, urgent

Table 1.3 Antibiotic Prophylaxis for Invasive Procedures in Highest Risk Patients

Patient Category	Recommended Antibiotic for ED Dental Procedures
Adults	Amoxicillin 2 g PO × 1 *if PCN allergy* Clindamycin 600 mg PO × 1 *Unable to take oral medications:* Ceftriaxone 1 g IV/IM × 1 *if PCN allergy* Clindamycin 600 mg IV/IM × 1
Children	Amoxicillin 50 mg/kg PO × 1 (max. 2 g/dose) *if PCN allergy* Clindamycin 20 mg/kg PO × 1 (max. 600 mg/dose) *Unable to take oral medications:* Ceftriaxone 50 mg/kg IV/IM × 1 (max. 1 g/dose) *if PCN allergy* Clindamycin 20 mg/kg IV/IM × 1 (max. 600 mg/dose)
Pregnant women	As above
Immunocompromised	As above

IM – intramuscular; IV – intravenous; PCN – penicillin; PO – by mouth.

follow-up is available. Patients with septic or mechanical complications of IE should be managed in a closely monitored setting, preferably one in which cardiothoracic surgical intervention is readily available.

Pearls and Pitfalls

1. Endocarditis is important to consider in any febrile patient with a predisposing valve disease or other risk factors.
2. Emergency providers can play an essential role in IE diagnosis by obtaining blood cultures prior to empiric antibiotics.
3. Mechanical complications of IE may require emergent cardiovascular surgery.
4. Do not heparinize patients with septic emboli and endocarditis.

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Chapter 2

Pericarditis and Myocarditis

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Outline

Introduction

Cardiac infections are classified by the affected site: pericardium, myocardium, or endocardium. Since pericarditis and myocarditis often coexist, and the infectious etiologies are very similar, these will be discussed together here. Endocarditis is a fundamentally different type of infection that is covered in Chapter 1. Pericarditis is a common cause of chest pain that has the potential to result in significant morbidity and mortality. Acute care providers should be well versed in the identification, risk stratification, and evidence-based management of this common condition.

Pericarditis

The pericardium is composed of two layers of fibrous tissue, the visceral and parietal, which envelop and protect the heart. The visceral layer is firmly attached to the epicardium, whereas the parietal layer moves freely within the mediastinum. Approximately 15 to 50 mL of fluid is normally present within the pericardial sac.

Pericarditis is defined as inflammation of the pericardium. It frequently causes a small pathologic pericardial effusion and may be associated with adjacent myocardial inflammation or infection, termed myopericarditis. Large pericardial fluid accumulations may occur in pericarditis, which can result in cardiac tamponade, if they develop rapidly.

The majority of infectious pericarditis and myocarditis are due to direct viral infection or less commonly bacterial seeding of the pericardium. Contiguous spread to the pericardium from pleural, pulmonary, or mediastinal infections, or from endocarditis, can also occur. There are also numerous noninfectious causes of both pericarditis and myocarditis.

Epidemiology and Microbiology

While the epidemiology of pericarditis is not well described, it is clearly a common condition, estimated to account for 5% of non-ischemic chest pain cases seen in emergency departments (EDs). Pericarditis commonly affects young men, for reasons that are not well understood.

Acute pericarditis is often idiopathic, in that routine evaluation reveals no definite cause; the majority of such cases are presumed to be viral. When a pathogen is identified, viruses predominate, including coxsackieviruses, echoviruses, influenza, EBV, VZV, mumps, and hepatitis. Human immunodeficiency virus (HIV) can cause pericarditis and myocarditis and remains a common cause of pericardial disease in developing countries where HIV is prevalent.

Bacterial pericarditis, termed purulent pericarditis, is fortunately rare. It can result from hematogenous seeding or direct spread, usually from pneumonia. Myriad bacteria have been reported to cause pericarditis, with the most common pathogens being *Staphyloccus aureus* and *Streptococcus pneumoniae*. Pneumococcal pneumonia and empyema and *S. aureus* endocarditis (via endomyocardial abscess) are the infections that classically spread directly to the pericardium. Mediastinitis, penetrating trauma, and thoracic surgery can also lead to purulent pericarditis. *S. aureus* is the predominant pathogen in hematogenous cases.

Mycobacterium tuberculosis is considered to be the most uncommon etiology of infectious endocarditis in developing countries. Fungi are a relatively uncommon cause of

Table 2.1 Important Causes of Pericarditis and Myocarditis

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

Adapted from A. M. Ross and S. E. Grauer, Acute pericarditis. Evaluation and treatment of infectious and other causes. *Postgrad Med.* 2004 March; 115(3): 67–75.

RMSF – Rocky Mountain spotted fever.

pericarditis. Histoplasomosi pericarditis is seen in endemic regions of the United States and *Candida* species are a common etiology in nosocomial cases.

The list of non-infectious causes of acute pericarditis is very long (see Table 2.1). These include uremia, trauma, malignancy (lymphoma, cancers of the breast, lung, and kidney), radiation, chemotherapy, drug reactions (penicillin, minoxidil), post-cardiotomy or thoracic surgery, and autoimmune disorders (systemic lupus erythematosus [SLE], rheumatoid arthritis [RA], Dressler's syndrome after myocardial infarction postpericardiotomy syndrome).

Clinical Features

The clinical presentation of infectious pericarditis varies depending on the pathogen and the the host immune response (see Table 2.2). Most patients with acute viral (or ideopathic) pericarditis have mild symptoms, which include low-grade fever, malaise, and substernal chest pain. There may be a history of a preceeding viral respiratory or gastrointestinal illness. The pain is typically described as sharp or stabbing, but may be squeezing. It usually has a pleuritic quality – worsened by inspiration and cough. The pain is commonly postural: lying supine exacerbates the pain, whereas sitting upright or leaning slightly forward relieves it. The phrenic nerve traverses the pericardium, so the pain of pericarditis is often described as radiating to the trapezial ridges. Patients with pericarditis may also complain of cough, odynophagia, or dysphagia, presumably secondary to the spread of the inflammatory process to adjacent structures.

Patients with slowly accumulating effusions, such as in uremic or autoimmune pericarditis, may have no chest pain and limited hemodynamics signs. Those with rapidly accumulating effusions may present with tamponade and shock. This classically occurs from malignancy, in patients on anticoagulants and in purulent pericarditis. Associated myocarditis can lead to rapid heart failure, cardiogenic shock, and arrythmias.

Patients with purulent pericarditis usually appear toxic with an acute febrile illness and may have evidence of pneumonia, empyema, endocarditis, or mediastinal infection. Tuberculous pericarditis generally presents as an indolent illness with nonspecific symptoms such as fever, night sweats, weight loss, and fatigue.

The classic physical finding in acute pericarditis is a pericardial friction rub, which is typically a three-phase "scratchy" heart sound that comes and goes, best heard while the patient leans forward. Signs of pericardial tamponade are discussed under "Complications and Admission Criteria."

Differential Diagnosis

The differential diagnosis of a patient complaining of chest pain or dyspnea in an emergent or urgent setting includes the following:

- aortic dissection
- pulmonary embolism
- pneumothorax and tension pneumothorax
- acute coronary syndrome
- esophageal perforation
- myopericarditis
- mediastinitis
- pneumonia
- pleurisy

Table 2.2 Clinical Features: Pericarditis and Myocarditis

	Pericarditis	Myocarditis
Signs and symptoms: adults	• Fever, malaise, night sweats • Chest pain (typically sharp, pleuritic) • Pericardial friction rub • Tamponade: tachycardia, Beck's triad, pulsus paradoxus	• Fever, malaise, night sweats • Chest pain uncommon unless associated pericarditis • Dyspnea, orthopnea • Left and right-sided heart failure signs: lung crackles, hypoxemia, hypotension, JVD, HSM, peripheral edema • Dysrhythmia or conduction disturbance
Signs and symptoms: infants	• As above • Non-specific – lethargy, poor feeding, cyanosis	• As above • Non-specific – lethargy, poor feeding, cyanosis
Laboratory and ECG findings	• Elevated WBC, CRP, ESR • ECG findings include: • Sinus tachycardia and non-specific ST-T changes • Diffuse ST-segment elevation • PR depression • T wave inversion without Q wave formation • Ultrasound – pericardial effusion, possible signs of tamponade	• Elevated WBC, CRP, ESR, and cardiac biomarkers • ECG findings non-specific: • Sinus tachycardia and non-specific ST-T changes • ST-segment elevation or depression • Decreased QRS amplitude and Q waves • Atrial or ventricular ectopy • Bundle branch blocks • Ultrasound – decreased left ventricular function

CRP – C-reactive protein; DOE – dyspnea on exertion; ECG – electrocardiography; ESR – erythrocyte sedimentation rate; JVD – jugular venous distention; HSM – hepatosplenomegaly; TB – tuberculosis; WBC – white blood (cell) count.

- gastroesophageal reflux disease
- costochondritis
- panic attack
- herpes zoster
- cholecystitis

The diagnosis of pericarditis and/or myocarditis should be considered when chest pain, dyspnea, dysrhythmias, heart failure, or cardiac tamponade accompanies a recent viral-seeming upper respiratory or gastrointestinal illness, or in the setting of an underlying autoimmune disorder, malignancy, renal failure, recent cardiac surgery, or exposure to tuberculosis.

Acute pericarditis can be mistaken for ST-segment elevation myocardial infarction resulting in inappropriate treatment with fibrinolytic agents and/or anticoagulants. Electrocardiographic findings should distinguish these disorders: ST elevations of pericarditis generally occur diffusely, whereas acute coronary syndrome (ACS) involves a specific coronary artery territory. Likewise, pericarditis can be difficult to distinguish from other pain syndromes associated with underlying immunologic disease, or from pulmonary embolism in a patient with underlying cancer.

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. Echocardiography is recommended in all cases of suspected pericardial disease.

While blood tests may not always be necessary in an otherwise healthy patient presenting with typical findings of acute pericarditis and normal vital signs, most patients require further risk stratification. Laboratory findings in pericarditis may include leukocytosis, elevated CRP, and increased ESR. Negative inflammatory markers argue against pericarditis. A single set of biomarkers is recommended; elevated cardiac biomarkers suggest associated myocarditis (myopericarditis). Blood culture should be drawn in patients with a high fever or signs of toxicity. Skin testing and sputum testing for acid-fast bacilli should be considered in the appropriate setting.

Chest X-ray is useful in excluding pneumonia and pneumothorax, and it may reveal a pleural effusion, lung mass, or infiltrate suggestive of active tuberculosis, which can focus the differential diagnosis. A large pericardial effusion or severe myocarditis with heart failure will cause cardiomegaly (see Figure 2.1).

Electrocardiography is a cornerstone of pericarditis diagnosis. Typical findings are shown in Figure 2.2. Acute pericarditis causes a characteristic progression of ECG findings through four distinct phases. Stage one lasts for days and is characterized by diffuse ST elevation in all leads except avR and V1 and PR segment depression. Stage two is normalization of the ST and PR segments. Stage 3 is characterized by diffuse T wave inversion without Q wave formation, and stage 4 is ECG normalization. In the case of a large effusion, these signs are usually not seen; rather, there may be tachycardia, loss of QRS voltage, and electrical alternans.

Echocardiography is recommended for risk stratification in suspected pericarditis (See Figure 2.4). In typical acute idiopathic pericarditis, a small effusion may or may not be seen. An effusion greater than 20 mm is considered high risk, generally necessitating admission. Echocardiographic evidence of tamponade (discussed below under "Complications and Admission Criteria") or decreased ventricular function, suggesting associated myocarditis, also necessitate admission.

Diagnostic pericardiocentesis should be considered in patients with a significant effusion and fever, to rule out purulent pericarditis, in those with tamponade or impending tamponade, and to work up suspected malignant pericardial effusion.

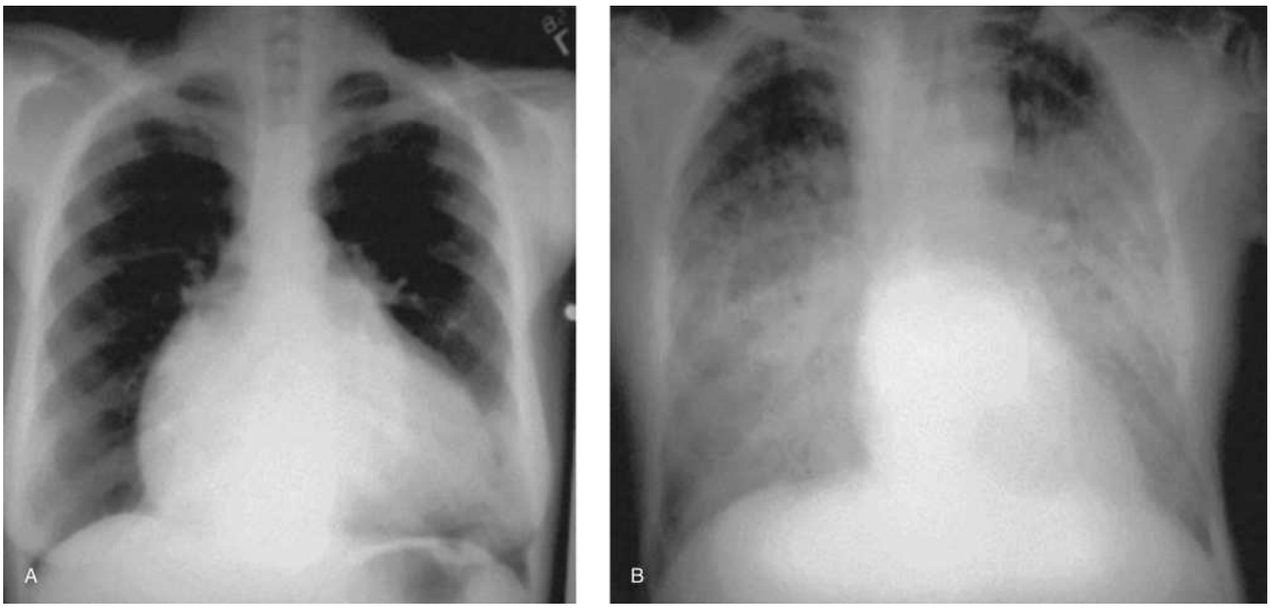

Figure 2.1 Chest X-ray findings in pericarditis and myocarditis. (A) Cardiomegaly from pericardial effusion. (B) Acute pulmonary edema in myocarditis.
Reprinted with permission from W. J. Brady, J. D. Ferguson, E. A. Ullman, and A. D. Perron, Myocarditis: emergency department recognition and management. *Emerg. Med. Clin. North Am.* 2004; 22(4): 865–85.

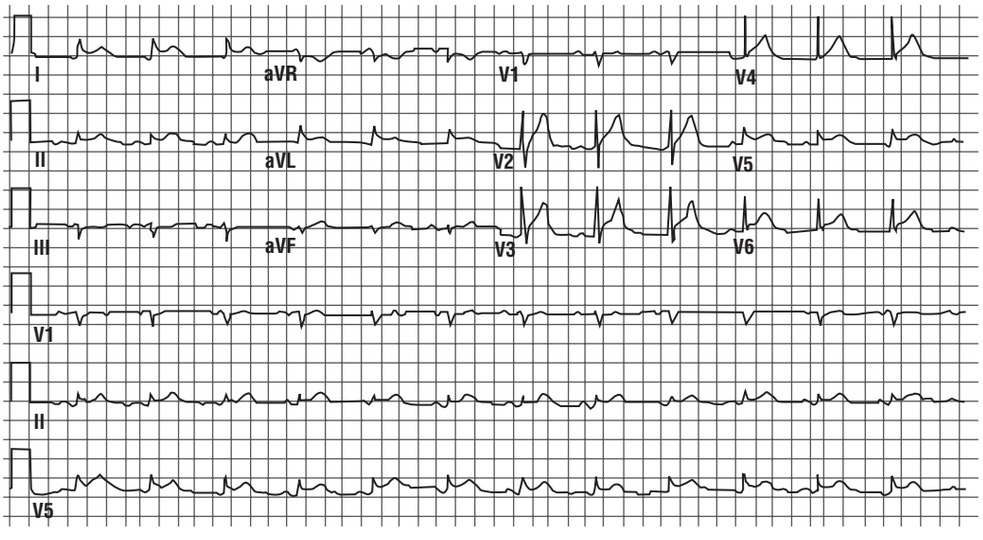

Figure 2.2 Electrocardiography in acute pericarditis. Stage 1, showing diffuse ST segment elevation.
Reprinted with permission from A. M. Ross and S. E. Grauer, Acute pericarditis. Evaluation and treatment of infectious and other causes. *Porstgrad. Med.* 2004; 115(3): 67–75.

Treatment and Prophylaxis

Symptomatic treatment of pericarditis should be undertaken after ruling out other life-threatening causes of chest pain and life-threatening complications of pericarditis (see Table 2.3). Treatment of pain and inflammation with aspirin or non-steroidal agents like ibuprofen is the mainstay of pericarditis treatment. Based on trial data showing a reduction in recurrence, routine addition of colchicine is now recommended for acute uncomplicated pericarditis. No definitive treatment benefit of corticosteroids has been documented, except when there is an underlying collagen vascular disease such as SLE or RA. Additionally, the use of steroids in acute pericarditis appears to increase the risk of recurrent or chronic pericarditis. Exercise restriction until symptom resolution and normalization of inflammatory markers is recommended in young patients with idiopathic or viral pericarditis.

Complications and Admission Criteria

Important complications of pericarditis include myocarditis, tamponade, and recurrence (see Table 2.4). Patients with purulent or tuberculous pericarditis are at risk from progression of the infection itself. Signs of myocarditis should always be sought.

Table 2.3 Initial Treatment for Pericarditis

Patient Category	Therapy Recommendation
Adults	**Non-steroidal anti-inflammatories (avoid if isolated myocarditis):** Aspirin 650–1000 mg PO TID *or* Ibuprofen 600–800 mg PO TID *or* Indomethacin 50 mg PO TID *plus* Colchicine 0.6 mg PO BID
Children	**Non-steroidal anti-inflammatories (avoid if isolated myocarditis):** Ibuprofen 5–10 mg/kg PO QID *or* Naproxen 5–10 mg/kg PO BID *plus* Colchicine 0.3–0.6 mg PO daily
Pregnant women	Acetaminophen 500 mg PO every 6 hours
Immunocompromised	As above, depending on age and pregnancy status

PO – by mouth.

Evaluation of a patient with suspected pericarditis should routinely include assessment for signs of hemodynamic compromise and pericardial tamponade. These signs include pulsus paradoxus, tachycardia, and Beck's triad of hypotension, JVD, and muffled heart sounds. Electrical alternans, characterized by alternating voltage of the P wave, QRS segment, and T wave, is pathognomonic of a large, hemodynamically significant pericardial effusion. Echocardiography is the gold standard test for diagnosis. Diagnostic findings include pericardial effusion, inferior vena cava dilation, diastolic collapse of the right atrial or ventricular, and leftward bowing of the septum with inspiration (see Figure 2.3). Cardiac tamponade requires aggressive fluid resuscitation followed by emergent pericardiocentesis if a patient does not immediately improve with IV fluids.

Recurrence occurs in up to 38% of patients with idiopathic pericarditis who are not treated with colchicine and 17% of those who are. Recurrecnt of pericarditis is thought to be autoimmune and can prove difficult to manage.

In the setting of a normal echocardiogram, patients with acute pericarditis who are well appearing may be safely discharged. Small or moderate effusions can be followed with serial echocardiograms; large effusions may require pericardiocentesis or placement of a pericardial window.

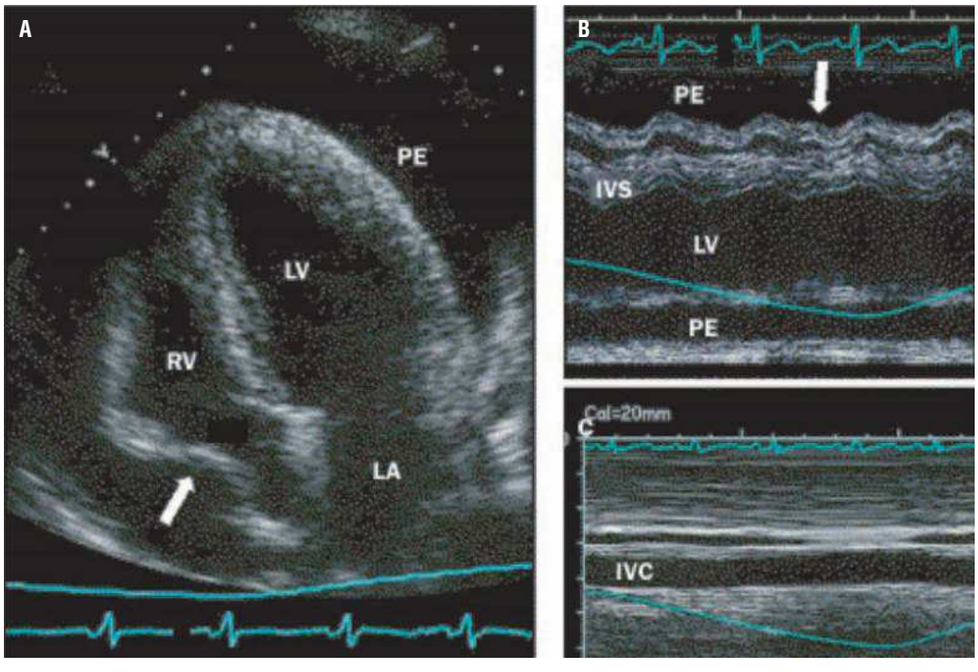

Figure 2.3 Echocardiographic evidence of cardiac tamponade. Echocardiographic images of large pericardial effusion with features of tamponade. (A) Apical four-chamber view of LV, LA, and RV that shows large PE with diastolic right-atrial collapse (arrow). (B) M-mode image with cursor placed through RV, IVS, and LV in parasternal long axis. The view shows circumferential PE with diastolic collapse of RV free wall (arrow) during expiration. (C) M-mode image from subcostal window in same patient that shows IVC plethora without inspiratory collapse. Reprinted with permission from Elsevier (*The Lancet,* 2004, vol. 363, pp. 717–27).

Photo and text from R. W. Troughton, C. R. Asher, and A. L. Klein, Pericarditis. Lancet 2004; 363(9410): 717–27.

IVC – inferior vena cava; IVS – interventricular septum; LA – left atrium; LV – left ventricle; PE – pericardial effusion; RV – right ventricle.