Tuberculous and Infectious Pericarditis

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KEYWORDS

• Infectious pericarditis • Viral pericarditis • Bacterial pericarditis • Fungal pericarditis
• Tuberculosis pericarditis

KEY POINTS

- The clinical presentation dictates whether comprehensive assessment for infective pericarditis is necessary. Viral pericarditis presents as acute pericarditis with chest pain but rarely progresses to constrictive pericarditis. The most common clinical manifestations of tuberculous pericarditis are dyspnea and pericardial effusion. Purulent pericarditis is rare but should be suspected if fever or sepsis is present.
- Viral pericarditis is the most common infectious pericarditis and is often classified as idiopathic pericarditis. It can be treated with aspirin or nonsteroidal anti-inflammatories. Steroid therapy should be reserved for refractory cases.
- The presentation of tuberculous pericarditis is variable. It is typically fatal in immunocompromised hosts, if untreated. However, in immunocompetent patients, tuberculous pericarditis often improves spontaneously and progresses to chronic constrictive pericarditis. Steroid therapy along with antituberculosis treatment can rescue these patients.
- Bacterial and fungal pericarditis are fatal if untreated and are rare in the current era. Microbiologic diagnosis and appropriate antibacterial/antifungal treatment should be followed by drainage of the purulent pericardial effusion.

INTRODUCTION

The diagnosis and treatment of pericarditis has been largely empirical because of lack of clinical trials and to the fact that infectious pericarditis may present in a way that is similar to noninfectious pericarditis. However, pericarditis caused by certain infectious pathogens should be managed differently because it can be fatal or result in severe complications if treatment is delayed.

All infectious forms of pericarditis can present as acute pericarditis, pericardial effusion, tamponade, or constrictive pericarditis. Initial management should be based on the severity of the presentation and the clinical scenario (eg, if bacterial pericarditis is suspected). Environmental factors and host factors, including other underlying diseases, should be considered when assessing potential etiologies of infectious pericarditis.

TUBERCULOUS PERICARDITIS

Epidemiology

Tuberculous pericarditis is caused by *Mycobacterium tuberculosis* and is found in 1% to 2% of people who have pulmonary tuberculosis in endemic areas.[1] Tuberculous pericarditis is more prevalent in Africa and East Asia. Although tuberculous pericarditis is rare in Europe and North America (less

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than 5% of all pericardial disease[2]), it should be suspected in patients who have immigrated from those areas that have a higher prevalence. In Africa, human immunodeficiency virus infection/acquired immune deficiency syndrome (HIV/AIDS) have a relatively high prevalence, which, in turn, leads to a relatively high prevalence of tuberculous pericarditis. In fact, tuberculous pericarditis is the cause for up to 70% of cases referred for diagnostic pericardiocentesis.[2] Mortality and morbidity is high for untreated cases, especially in patients with HIV/AIDS (30%–60%), who have a 3-fold higher mortality rate when compared with patients without HIV/AIDS.[3] In Asian countries, most patients with tuberculous pericarditis do not have HIV/AIDS and are immunocompetent. Therefore, spontaneous regression with subsequent fibrosis is not unusual.

Clinical Features

Clinical presentations of tuberculous pericarditis include pericardial effusion, effusive-constrictive pericarditis, or constrictive pericarditis. Symptomatic pericardial effusion is more common among HIV/AIDS patients and its prevalence is reported to be as high as 80% in patients referred for pericardiocentesis.[4] The classic presentation of acute pericarditis with sudden-onset chest pain and typical electrocardiographic changes is rare in tuberculous pericarditis. Instead, systemic symptoms and signs are common, such as cough (94%), dyspnea (88%), chest pain (76%), fever (70%), night sweats (56%), orthopnea (53%), and weight loss (48%).[1,5] Constrictive pericarditis develops after acute infection has resolved and can linger for many years, eventually resulting in heart failure; surgical pericardiectomy should be reserved for these patients. Progression to constrictive pericarditis, even with optimal antituberculosis therapy (without corticosteroid therapy), is reported in up to 30% of cases.

Pathogenesis

Tuberculous pericarditis has various clinical presentations that are associated with 4 pathologic stages[6]: (1) fibrinous exudation with initial polymorphonuclear leukocytosis, relatively abundant mycobacteria, and early granuloma formation with loose organization of macrophages and T cells; (2) serosanguineous effusion with a predominantly lymphocytic exudate with monocytes and foam cells; (3) effusion absorption with organization of granulomatous caseation and pericardial thickening caused by fibrin, collagenosis, and ultimately, fibrosis; and (4) constrictive scarring, which involves fibrosis of the visceral and parietal pericardium. The scarred (and sometimes calcified) pericardium encases the heart in a fibrocalcific skin that impedes diastolic filling and causes the classic constrictive pericarditis syndrome.

In HIV/AIDS patients, fewer granulomas were observed than in non-HIV patients because of severely depleted CD4+ lymphocytes.[7] This finding correlates with clinical findings of fewer effusive-constrictive pericarditis cases in HIV/AIDS patients than in non-HIV patients.

Diagnosis

Diagnosing tuberculous pericarditis is not simple in most cases. In countries in which tuberculosis is not endemic, treatment should be reserved for patients with proven diagnosis or high likelihood of having tuberculous pericarditis. The tuberculin skin test can be a diagnostic clue in countries with low tuberculosis prevalence. However, in areas in which tuberculosis is prevalent, this is of little value, given the high prevalence of primary tuberculosis, mass Bacillus Calmette–Guérin immunization, and the likelihood of cross-sensitization from the environment.[8] Among patients who are at risk for tuberculosis, a presumptive diagnosis of tuberculous pericarditis is enough for initiation of therapy, given the high risk of not treating when tuberculosis is the etiology. The European Society of Cardiology 2015 guidelines suggested that, if pericardial fluid is not accessible in cases from endemic areas, a diagnostic score of $\geq$6, based on the following criteria, indicates tuberculous pericarditis and warrants presumptive treatment: fever (1), night sweats (1), weight loss (2), globulin level greater than 40 g/L (3), and peripheral leukocyte count less than 10×10^9/L.[3,9]

Clinical features and risk factors should be considered at initial evaluation. Acute chest pain with electrocardiographic abnormalities is rarely found in tuberculous pericarditis patients. Risk factors for patients include HIV/AIDS[10] and long-term use of steroids or immunosuppressive agents.[11] Echocardiography is the initial diagnostic tool for most cases, allowing for assessment of pericardial effusion and constrictive physiology. Electrocardiographic results are abnormal in most cases; however, these results are nondiagnostic because most ST-segment and T-wave abnormalities are nonspecific. PR-segment deviation and ST-segment elevation are found in only 10% of cases.[12,13] Pericardial effusion with fibrinous material of porridgelike appearance on echocardiography is a typical finding for tuberculous pericarditis but is not sufficiently specific to make the diagnosis.