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Constrictive Pericarditis Secondary to Tuberculosis Infection: Literature Review

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Abstract

Tuberculous pericarditis is produced by Mycobacterium tuberculosis, accounting for 1% of all forms of tuberculosis. Its prevalence varies according to coinfection with HIV. Mortality varies between 17 and 40%. In the US, the prevalence is low compared to developing countries. This article aims to review the literature on pericarditis caused by tuberculosis (TBP), its prevalence in the US, clinical manifestations, diagnosis, and treatment. Among the most frequent clinical manifestations are dyspnea, fever, chest pain, and cough. TBP should be suspected in patients at high risk of exposure to tuberculosis. There are multiple lab tests for diagnosis, and its primary treatment is triple therapy with isoniazid, rifampin, and streptomycin or ethambutol. In case of persistent elevation of systemic venous pressure, surgical intervention is indicated. The clinical presentation was found to be variable.

Keywords: Tuberculous pericarditis; Mycobacterium tuberculosis; Constrictive pericarditis; Tuberculosis; TB diagnosis; TB treatment

Abbreviations: TB: Tuberculosis, Mtb: Mycobacterium Tuberculosis, CDC: Centers for Disease Control and Prevention, TBP: Tuberculous Pericarditis, HIV: Human Immunodeficiency Virus, TST: Tuberculin Skin Test, IGRA: Interferon-Gamma Release Assay, AFB: Acid-Fast Bacilli, ADA: Adenosine Deaminase, NSAIDs: Nonsteroidal Anti-Inflammatory Drugs, LGE: Late Gadolinium Enhancement

Introduction

Tuberculosis (TB) is a potentially dangerous infectious disease caused by Mycobacterium tuberculosis (Mtb) which primarily affects the lungs but can also attack other tissues and form caseating granulomas. According to the CDC, in 2021, 7,860 TB cases were provisionally reported in the US [1]. People contract tuberculosis from one another by coughing and sneezing, which

release microscopic droplets of germs into the air. One of the organs that can be affected is the heart, mostly the pericardium, causing pericarditis (inflammation of the pericardial sac), and the scarring and the subsequent loss of the pericardial sac's typical flexibility can lead to constrictive pericarditis. The pericardium can become infected with Mtb bacilli through retrograde lymphatic spread, hematogenous dissemination, or, less frequently, direct contiguous spread from nearby infected tissues such as the lungs, pleura, and spine [2]. Heart failure is the most common clinical presentation of tuberculosis pericarditis (TBP). However, some patients with tamponade may also experience hemodynamic compromise with hypotension, tachycardia, and shock [3]. The most common diagnostic test for TBP is pericardial fluid culture, which has a sensitivity of between 53 and 75% [4]. The treatment of TBP consists in eradicating the Mtb with rifampicin, isoniazid, ethambutol, and pyrazinamide, in combination, for a minimum of six months. The preferred procedure to drain compressive pericardial fluid from the pericardium is needle pericardiocentesis guided by fluoroscopic or echocardiographic imaging. Also, oral or intrapericardial corticosteroids can prevent constriction progression. Finally, the goal of the treatment is to avoid pericardial fibrosis [3]. The purpose of this review article is to provide a general understanding of TBP to enable the correct diagnosis and treatment of the condition to prevent the development of complications, such as pericardial fibrosis.

Etiology & Pathogenesis

Tuberculous pericarditis is caused when infection of Mycobacterium Tuberculosis reaches the pericardium. It is the leading cause of pericarditis in countries where the prevalence of tuberculosis is still high, and it is found in 1% of autopsies performed in patients with the infection [5]. Pericardial involvement develops through three mechanisms dependent on the immune competency of the host. The first mechanism involves retrograde spread from mediastinal, peritracheal, and parabronchial lymph nodes; the second is the hematogenous route during primary tuberculosis infection; the third is by direct spread from the lung parenchyma or pleura. Immune-competent hosts favor lymphatic spread, whereas hosts with HIV-related immune suppression will favor the hematogenous route [6]. Among the first, TB pericarditis is frequently a paucibacillary condition in which the morbidity is related to the immune response and not the pathogen's virulence. In contrast, immune-deficient hosts suffer from disseminated disease associated with TB bacteremia, which may significantly impact morbidity and mortality. This has been supported by histopathological and immunologic analyses demonstrating diminished granuloma formation and aberrant CD4+ function in patients with HIV.

Four pathological stages have been described in TB pericarditis. In the first stage, protein antigens induce a hypersensitivity reaction where CD4+ lymphocytes trigger the release of cytokines that activate macrophages to induce granuloma formation and induce the production of complement-fixing anti-myolemmal and anti-myosin antibodies. These are responsible for the cytolysis that characterizes the fibrinous exudate of this phase. The second stage is the formation of a serosanguineous effusion with abundant foam cells. Finally, the third stage constitutes the absorption of the effusion and organization of granulomas with pericardial thickening that will eventually lead to constrictive scarring and calcification of the pericardium [6].

Epidemiology

In a 2019 report of annual trends of patients admitted to US hospitals with constrictive pericarditis from 2005 to 2014, investigators found a stable prevalence of 9-10 cases per million [7]. Tuberculous pericarditis accounts for 1% of all forms of tuberculosis and 1-2% of extrapulmonary tuberculosis [8]. Most data regarding TBP come from developing countries with a high tuberculosis burden and frequent coinfection with M. tuberculosis and HIV. In these geographic regions, TBP accounts for 50-70% of effusive pericarditis in HIV-negative patients and more than 90% in HIV-positive patients [8]. In industrialized countries, tuberculosis accounts for only 4% of cases of pericardial effusion and an even smaller proportion of constrictive pericarditis. Tuberculous pericarditis is a dangerous disease with a mortality of 17% to 40%. Constriction occurs in a similar proportion of cases after tuberculous pericardial effusion [7].

Tuberculous pericarditis has a low prevalence in the USA. Less than 1% of the patients with a diagnosis of TB had an associated pericarditis diagnosis. While only 1% of TB patients have pericarditis in the US, in developing countries with a high prevalence of TB, 70% of cases of significant pericardial effusion are attributable to TB, and it is an essential etiology of constrictive pericarditis [9].

Clinical Manifestations

The clinical manifestations of tuberculosis are pretty variable and depend on several factors. Host and microbe-related characteristics, as well as their interactions, influence the disease's clinical features. Before the beginning of the epidemic of infection with HIV, approximately 85% of reported tuberculosis cases were limited to the lungs, with the remaining 15% involving only non-pulmonary or both pulmonary and non-pulmonary sites. This proportional distribution is substantially different among persons with HIV infection. Although there are no national data that describe the locations of involvement in HIV-infected persons with tuberculosis, one large retrospective study of tuberculosis in patients with advanced HIV infection reported that 38% had only pulmonary involvement, 30% had only extrapulmonary sites, and 32% had both pulmonary and non-pulmonary involvement. Moreover, extrapulmonary involvement tends to increase in frequency with worsening immune compromise [10].

Two broad mechanisms drive the clinical presentation of pericarditis: fluid accumulation within the pericardium, which compresses the heart chambers throughout the cardiac cycle

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impeding both cardiac filling and cardiac contraction (tamponade) or thickening of the pericardium with minimum or absent effusion resulting in impairment of cardiac filling in diastole (constriction). The predominant clinical manifestation of TBP is heart failure syndrome, regardless of the mechanism. However, a relatively small proportion of patients with tamponade may also have hemodynamic compromise with hypotension, tachycardia, and shock [11]. However, the frequency of tuberculous pericarditis is very different depending on the overall prevalence of tuberculosis in the region and in particular categories of patients (for example, HIV-infected). For example, among patients with constrictive pericarditis in a South African Hospital, tuberculosis was diagnosed in 29.8% of patients [6].

Diagnosis

The diagnosis of tuberculous pericarditis should be considered in patients with pericarditis that does not have a self-limited course with a high-risk factor of exposure to TB [12]. The initial evaluation of a patient with tuberculous pericarditis includes tuberculin skin test (TST) and interferon-gamma release assay (IGRA) that supports the diagnosis of TB, and more specific test as chest radiography, echocardiography showing pericardial effusion, electrocardiography with non-specific ST-T wave changes and micro voltages, sputum, and pericardial fluid evaluation for acid-fast bacilli (AFB) smear and culture [13]. The pericardial fluid should also be evaluated for cell count, protein concentration, lactate dehydrogenase concentration, adenosine deaminase (ADA) concentration, and cytology [12]. The tuberculous pericarditis effusion is usually exudative with lymphocytes, monocytes, and high protein concentration. ADA levels above 30 units/L in the pericardial fluid also indicate tuberculous pericarditis. In cases when the pericardial fluid and smear are not conclusive is recommended to obtain a right scalene lymph node and/or pericardial biopsy for AFB stain and examination for granulomatous disease [11,12]. It is to remark that the sensitivity of the biopsy is 10-64%, so a negative biopsy does not exclude the diagnosis [11]. The diagnosis of tuberculous pericarditis is made when there is evidence of tubercle bacilli in the pericardial fluid either by smear or culture or by the presence of caseating granulomas or bacilli in the histological examination of the pericardium [9,11].

Treatment

After the bacteriological diagnosis is established, the treatment is based on triple-drug therapy for at least nine months with isoniazid, rifampin, streptomycin, or ethambutol. For the first two months, pyrazinamide can be used, and the total therapeutic period can then be shortened to 6 months after culture conversion. Also, it is reasonable to add treatment with three months of corticosteroid therapy in patients with pericardial effusion that persists or recurs despite the use of anti-tuberculous drugs [14]. In patients with extrapulmonary TB, the following treatment has been shown to be highly effective: at least two months of rifampicin, isoniazid, pyrazinamide, and ethambutol, then a regimen followed by isoniazid and rifampicin, for a full six months of therapy. If the treatment is for nine months or longer, it does not provide better results and has disadvantages such as increasing cost and poor compliance. For recurrent or life-threatening tamponade or in cases when there is persistent elevation of systemic venous pressure unrelieved by pericardiocentesis, surgical resection of the pericardium is indicated [12,14].

It has been demonstrated that those patients with tuberculosis pericarditis receiving antituberculosis medications increase their survival dramatically. Because the management of pericardial diseases is mainly empiric (because of the relative lack of randomized trials), to prevent recurrences, the mainstay of empiric anti-inflammatory therapy (NSAIDs) and some cases adding colchicine can be the best course of action [15].

Prognosis

Patients with a compromised immune system uncommonly develop constrictive pericarditis. However, they do have higher mortality [16]. In contrast, a study published in 2020, consisting of 50 patients with different types of TB pericarditis in immunocompetent patients, showed that only five patients exhibited constrictive pericarditis after six months, one out of 28 patients with effusion developed constrictive pericarditis [17].

Constriction leads to impaired diastolic filling, which in time, causes differences in intrathoracic and intracardiac pressures, diminishing the left-sided diastolic filling and transmitral flow. This can also cause an increase in interventricular dependence; the right heart ventricular filling rises, hepatic vein diastolic forward velocity, and cardiac filling pressure. Other findings in constrictive pericarditis include fibrosis, inflammation edema, and pericardial thickening. However, these are not seen in 100% of cases. Late gadolinium enhancement (LGE) can discover pericardial inflammation, chronic inflammation, neovascularization, and fibroblastic proliferation in tuberculous constrictive pericarditis. This finding stands out because it denotes the possible benefit of NSAIDs and that constrictive pericarditis can be reverted [16]. In addition, echocardiography, CT, cardiac MRI, and 18F-FDG PET/CT are used to assert a prognosis and estimate if patients will improve with anti-inflammatory treatment [18]. Constrictive pericarditis is the most severe sequel of tuberculous pericarditis, which makes it imperative that the anti-tuberculous therapy begins quickly. A pericardiectomy could be performed if the patient has failed conservative treatment [16,18].

Conclusion

Tuberculosis pericarditis is the leading cause of pericarditis in the world. It is caused by Mycobacterium tuberculosis and can be found in up to 1% of the autopsied cases of patients with TB and up to 2% of the cases of pulmonary TB. Tuberculous pericarditis has a

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variable clinic presentation and should be considered in evaluating all cases of pericarditis without a rapidly self-limited course. The most common manifestations are dyspnea, fever, chest pain, and cough. Without treatment, mortality exceeds 90%, whereas anti-tuberculous therapy, usually rifampicin, isoniazid, pyrazinamide, and ethambutol for at least two months, followed by isoniazid and rifampicin for another four months. Therefore, reaching the correct diagnosis is crucial in initiating appropriate therapy. The diagnosis of TBP is made when there is evidence of tubercle bacilli in the pericardial fluid by smear or culture. Tuberculous pericarditis with constrictive physiology at initial diagnosis can be reversible in 80% of cases if the appropriate steroid treatment is given. Assessment of echocardiographic features in tuberculous pericarditis can provide prognostic information and help predict treatment response. Constrictive pericarditis is the most severe sequel of tuberculous pericarditis. Management involves prompt initiation of anti-tuberculous therapy and appropriate timing of pericardiectomy for selected patients who have failed to respond to medical treatment. Despite the advances in medical therapy for TBP, further research is still needed in order to gain a deeper understanding of this complex condition.

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