INTRODUCTION

Tuberculous (TB) pericarditis, caused by Mycobacterium tuberculosis, is found in approximately 1% of all autopsied cases of TB and in 1-2% of pulmonary TB [1]. Although there has been a significant decline in TB in developed countries over the past decades, Asia, Africa and Latin America, with 86% of the World’s population are home to 95% of all cases of active TB but also the world over, since the introduction of HIV infection. A case of 82-year-old female who died after a long history of dyspnea, weakness and fever is reported herewith. She was never investigated for TB. This case report highlights that the diagnosed cases of TB could actually represent just the tip of the iceberg. There is, therefore, a need for increased awareness and to include post-mortem data in the annual statistics of TB for precise assessment and reporting of the magnitude of the TB burden in the country.

DISCUSSION

Pericardial involvement usually develops by retrograde lymphatic spread of M. tuberculosis from peritracheal, peribronchial or mediastinal lymph nodes or by hematogenous spread from primary TB infection [8]. The pericardium is rarely involved by contiguous spread from a TB lesion in the lung or by hematogenous spread from distal skeletal or genitourinary infection. TB pericarditis presents clinically as pericardial effusion, constrictive pericarditis or a combination of effusion and constriction [1]. Signs and symptoms of TB pericarditis are often nonspecific and vague, and the diagnosis may not be established on clinical grounds alone. The case we report below highlights the importance of a high index of suspicion of TB pericarditis in autopsy cases where TB is a common cause of death in the area.

CASE REPORT

We present a case report of TB pericarditis diagnosed on post-mortem histopathological examination of the heart. The deceased was an 82-year-old female who, according to her relatives, was maintaining poor health since past few months. She complained of weakness, shortness of breath and on and off fever. Two days before her death, she had high-grade fever. Only heart was received for histopathological examination. The heart weighed 250 g and measured 12 cm × 10 cm × 6 cm. External surface was rough and dull [Figure 1]. On cutting open, heart was unremarkable. On microscopic examination, the pericardium was thickened and showed numerous caseating epithelioid cell granulomas, Langhan’s giant cells and mononuclear inflammatory infiltrate [Figure 2a-d]. The coronary arteries did not reveal atherosclerotic changes. TB was not diagnosed antemortem, in this case. As other visceras were not submitted for histopathological examination, it could not be established whether the pericarditis was the primary manifestation of TB or pericardium was involved as a result of dissemination following a primary infection elsewhere in the body.

ABSTRACT

The primary aims of tuberculosis (TB) control program are early diagnosis and prompt treatment of infectious cases to limit transmission. Failure to diagnose and adequately treat TB could lead to premature death and unrecognized transmission of Mycobacterium tuberculosis. The diagnosis of TB etiology in pericarditis is important since the prognosis is excellent with specific treatment. The clinical features may not be distinctive, and the diagnosis is often missed. The incidence of TB pericarditis is increasing not only in Afro-Asian countries but also the world over, since the introduction of HIV infection. A case of 82-year-old female who died after a long history of dyspnea, weakness and fever is reported herewith. She was never investigated for TB. This case report highlights that the diagnosed cases of TB could actually represent just the tip of the iceberg. There is, therefore, a need for increased awareness and to include post-mortem data in the annual statistics of TB for precise assessment and reporting of the magnitude of the TB burden in the country.
patient, with fewer granulomas being observed in HIV-infected patients with severely depleted CD4 lymphocytes [12]. The immune response to the viable acid-fast bacilli penetrating the pericardium is responsible for the morbidity associated with TB pericarditis. Protein antigens of the bacillus induce delayed hypersensitivity responses, stimulating lymphocytes to release lymphokines that activate macrophages and influence granuloma formation. The cytokine profile suggests that TB pericardial effusions arise as a result of a hypersensitivity reaction orchestrated by the TH-1 lymphocytes [13]. The demonstration of complement fixing anti-myomlemmal and anti-myosin type antibodies in 75% of patients with acute TB pericardial effusion has been cited as possible evidence that cytolysis mediated by anti-myomlemmal antibodies may contribute to the development of exudative TB pericarditis [14]. CT scan of the chest shows typical changes in mediastinal lymph nodes (i.e., enlargement >10 mm with matting and hypodense centers and sparing of hilar lymph nodes) in almost 100% of cases in addition to the features of pericardial disease i.e., pericardial effusion and thickening of pericardium [15].

Mayosi et al. [1] proposed diagnostic criteria for TB pericarditis for countries and communities in which TB is endemic. According to this criteria, a definite diagnosis of TB pericarditis is based on demonstration of tubercle bacilli in pericardial fluid or on histological section of the pericardium or caseating granulomata on histological examination of pericardium and a probable diagnosis is made when there is proof of TB elsewhere in a patient with unexplained pericarditis, a lymphocytic pericardial exudate with elevated adenosine deaminase levels, and/or a good response to anti TB therapy.

Despite prompt anti TB treatment and use of corticosteroids, constrictive pericarditis is one of the most serious sequelae of TB pericarditis and occurs in 30% to 60% of the patients [11]. TB as an etiology of constrictive pericarditis is more common than other causes in Africa and Asia [1]. According to Das et al. [5], out of 61 patients with constrictive pericarditis, the etiology was TB in 37 (61%) and non TB in 24 (39%) on pericardiectomy.

CONCLUSION

The present case highlights that TB pericarditis can be missed antemortem. Early diagnosis and management of TB pericarditis is imperative in areas endemic for TB.

REFERENCES

Rana et al.: Tuberculous pericarditis on autopsy


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Source of Support: Nil, Conflict of Interest: None declared.