

TUBERCULOUS PERICARDITIS: A CASE REPORT ON AUTOPSY

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ABSTRACT

Pericarditis is a rare manifestation of tuberculosis. Despite availability of effective treatment, tuberculosis is still a major cause of mortality in India. Some cases of tuberculosis are not identified until patient has died and autopsy has been done. A case of 40 year old female who died due to chronic illness and tuberculosis has not been diagnosed antemortem is reported here with. A definitive or proven diagnosis is based on demonstration of tubercle bacilli in pericardial fluid or on histological section of pericardium. Early diagnosis and prompt treatment are necessary to prevent mortality.

Key words:

Tuberculosis, autopsy, pericarditis, millary, granuloma, mortality

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INTRODUCTION

Tuberculosis is one of the most common infectious diseases in India.¹ Pericarditis is a rare manifestation of tuberculosis that can be fatal even with proper diagnosis and treatment.² Tuberculous pericarditis caused by Mycobacterium Tuberculosis is found in approximately 1% of all autopsy cases of TB and in 1-2% of pulmonary TB.³ Tuberculosis is responsible for approximately 70% of cases of large pericardial effusions and most cases of constrictive pericarditis in developing countries.⁴ Some of the patients are undiagnosed until after the patient has died and autopsy has been done. Absence of suspicion and delayed diagnosis cause complications and sequelae, the reason being non-specific symptomatology, unawareness towards the disease and illiteracy especially in developing and under-developed nations.

Case Report

We present a case report of TB pericarditis diagnosed on post mortem histopathological examination of heart. The deceased was a 40 year old female who died due to chronic illness according to her relatives. She complained of weakness, significant weight loss and on and off fever since 4-5 months before her death. On autopsy, heart, lung, liver, spleen and piece of kidney were sent for histopathological examination. The heart weighed 370 gms and measuring 10x9x4.5 cm. External surface was gray white and exudate covered (Figure 1). On cutting open, heart was unremarkable. On microscopic examination, sections from heart revealed thickening of pericardium, numerous langhan's giant cells, necrotizing

epithelioid cell granulomas and mononuclear cell infiltrate (Figure 2). Ziehl-Neelsen staining for acid fast bacilli using 20% H₂SO₄ was positive. Other organs sent for histopathological examination which included lung, spleen and kidney also revealed necrotizing epithelioid cell granulomas and lymphocytic infiltration (Figure 3). Sections examined from liver were unremarkable. Tuberculosis was not diagnosed antemortem in this case. As other organs also revealed caseating epithelioid cell granulomas, pericardium could be involved as a result of dissemination following a primary infection in lung.



Figure 1 External surface is gray white and exudate covered on gross examination of heart

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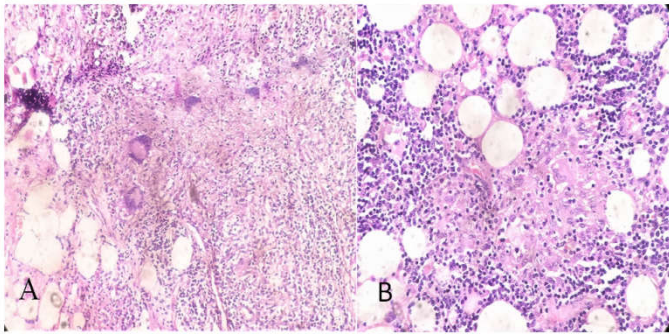


Figure 2 (A) Microphotograph shows Langerhans' giant cells and widespread caseous necrosis in pericardium (H and E, $\times 100$), (B) microphotograph shows epithelioid cell granuloma in the pericardium (H and E, $\times 400$)

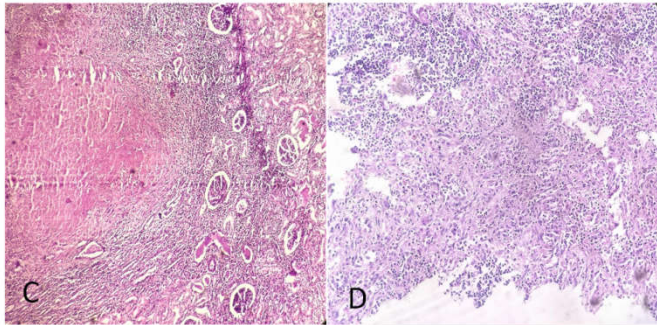


Figure 3 (C) Microphotograph shows widespread caseous necrosis and mononuclear inflammatory infiltrate in sections from kidney (H and E, $\times 100$), (D) microphotograph shows epithelioid cells, necrosis and mononuclear inflammatory infiltrate in sections from lung (H and E, $\times 100$)

DISCUSSION

Tuberculosis is increasing in prevalence in many countries and is now the leading infectious cause of death worldwide.⁵ Tuberculous pericarditis is a dangerous disease with a mortality of 17% to 40%, constriction occurs in a similar proportion of cases after tuberculous pericarditis.⁴ It is a chronic granulomatous disease caused due to infection by *Mycobacterium Tuberculosis*. Pericardial involvement usually develops by retrograde spread of mycobacterium tuberculosis from peritracheal, peribronchial or mediastinal lymph nodes or by hematogenous spread from primary TB infection.^{6,7} Tubercular pericarditis presents clinically as pericardial effusion, constrictive pericarditis or a combination of both.³

The immune response to the *M. tuberculosis* bacilli penetrating the pericardium is responsible for pathological changes of tuberculous pericarditis. Protein antigens of bacillus induce delayed hypersensitivity responses, stimulating lymphocytes to release lymphokines that activate macrophages and induce granuloma formation.⁸

Four pathological stages of TB pericarditis are recognized: (1) fibrinous exudation with initial polymorphonuclear leucocytosis, relatively abundant mycobacteria, and early granuloma formation with loose organization of macrophages and T-cells; (2) serosanguinous effusion with a predominantly lymphocytic exudate with monocytes and foam cells; (3) absorption of effusion with organization of granulomatous caseation and pericardial thickening caused by fibrin, collagenosis, and ultimately, fibrosis; and (4) constrictive scarring: the fibrosing visceral and parietal pericardium contracts on the cardiac chambers and may become calcified, encasing the heart in a fibrocalcific skin that impedes diastolic filling and causes the classic syndrome of constrictive pericarditis.⁹

The criteria for diagnosis were as follows: (1) culture of either pericardial tissue or fluid specimens that was positive for *Mycobacterium tuberculosis*, (2) granulomas and acid-fast bacilli (AFB) seen on histopathological examination of pericardial tissue, or (3) granulomas in the pericardial tissue and a positive culture for *M. tuberculosis* from another site.²

There are numerous reasons because of which the diagnosis is missed or delayed. These include ranging from symptomatic patients not reporting to health care providers, to doctors not investigating despite suspicious symptomatology due to concurrent illness that can produce symptoms indistinguishable from tuberculosis.

One of the many lethal forms of this disease is miliary tuberculosis. In the present case, lung spleen, kidney were involved along with pericarditis. Miliary tuberculosis is caused due to lymphohematogenous spread of the infection from primary focus. It results due to dehiscence of mycobacterium rich inflammatory focus into blood vessels with dissemination of bacilli into blood stream and seeding into non-pulmonary sites. High perfused organs are usually infested with pathognomic tubercles. In the present case lung spleen, kidney were involved along with pericarditis. The non specific symptomatology including fever, night sweats, fatigue, weight loss, cachexia, cough, chest pain, breathlessness results in delayed or missed diagnosis. Predisposing factors for the development of this condition include advanced age, immunosuppression in cases of HIV, chronic renal failure, organ transplantation or corticosteroid therapy.¹⁰⁻¹³

Recent data suggest that histological pattern is affected by the immune status of patient, with fewer granulomas being observed in HIV-infected patients with severely depleted CD4 lymphocytes.¹⁴

The upsurge of tuberculosis has been attributed to association with HIV infection and immunodeficiency. It is therefore necessary to suspect and screen all tuberculosis patients for possible HIV positivity and vice versa.

Diagnosis of this disease becomes important due to the fact that it is curable if diagnosed but highly morbid and fatal if undiagnosed. An undiagnosed infective person is hazard to the society as he/ she may infect contact persons which are not immunised to the disease.

CONCLUSION

Tuberculosis is one of the curable major health challenges faced by developing and under developed nations. There is a need for prompt diagnosis and thorough treatment for the disease. Unrecognised tuberculosis not only extends risk to public but also to the health care professionals.

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