# Case Report

# Confluence of Challenges: Massive Tubercular Pericardial Effusion Entangled with Heart Failure and Diabetes Mellitus - A Multidisciplinary Odyssey

Mohammad Abdul Motalib[1], Md Zulfikar Ali[2], Md Raisul Islam[3], Sagar Ahammed[4]

## Abstract

*This case report describes a rare and compelling presentation of Massive Tubercular Pericardial Effusion (MTPE) concomitant with Heart Failure (HF) and Diabetes Mellitus (DM). Tuberculosis, once thought to be a disease of the past, continues to manifest in diverse clinical scenarios, emphasizing the importance of maintaining a high index of suspicion, particularly in endemic regions. The patient, a 60-year-old male with a known history of DM and HF, presented with symptoms of worsening dyspnea, fatigue, and peripheral edema. A diagnostic workup revealed a massive pericardial effusion with characteristics suggestive of tubercular etiology, subsequently confirmed by pericardial fluid analysis. The challenges in managing this triad of conditions: MTPE, HF, and DM were addressed through a multidisciplinary approach involving cardiology, infectious disease, and endocrinology specialists. This case underscores the significance of a comprehensive diagnostic evaluation and collaborative management in optimizing outcomes for patients with complex cardiovascular and infectious comorbidities.*

***Key words:*** *Tuberculosis, Pericardial Effusion, Heart Failure, Diabetes Mellitus, Diagnostic Challenges, Multidisciplinary Approach, Therapeutic Management.*

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## Introduction

Tuberculosis, a disease with ancient roots, continues to wield its clinical complexity in contemporary medicine. Despite advancements in diagnostic modalities and therapeutic interventions, the protean manifestations of tuberculosis persist, posing a diagnostic challenge to clinicians worldwide.[1] Tubercular pericardial effusion, a relatively uncommon extrapulmonary manifestation, further complicates its recognition, especially when intertwined with concurrent cardiovascular and metabolic disorders.[2] This case report highlights the convergence of Massive Tubercular Pericardial Effusion (MTPE) with two prevalent chronic conditions—Heart Failure (HF) and Diabetes Mellitus (DM). While tuberculosis has been traditionally associated with pulmonary involvement, extrapulmonary presentations, such as tubercular pericarditis, demand heightened clinical suspicion, as they often masquerade as other cardiovascular diseases.[3] The interplay between MTPE, HF, and DM poses a unique clinical challenge, as the coexistence of these conditions can amplify the intricacies of management and potentially compromise patient outcomes. In this context, we present the case of a 60 year old male with a history of DM and HF who presented with symptoms indicative of cardiac compromise, ultimately unveiling a massive tubercular pericardial effusion.[4] This report aims to contribute to the expanding body of knowledge surrounding the diverse presentations of tuberculosis and its intersection with prevalent cardiovascular and metabolic disorders. Through an exploration of this unique clinical scenario, we underscore the necessity for a comprehensive approach to diagnosis and management, emphasizing the collaborative efforts of specialists in cardiology, infectious diseases, and endocrinology.

Pathogenesis of tuberculosis of the heart Myocardial tuberculosis, first diagnosed by Maurocordat on autopsy in 1664, is unusual and often goes undiscovered while the patient is still alive. Cardiopulmonary tuberculosis accounts for between 1% and 2% of all tuberculosis cases in immunocompetent persons. In a study of 19 individuals with cardiovascular TB, one case was diagnosed antemortem, 11 had nodular lesions on autopsy, and 7 had miliary lesions; only one patient had acid-fast bacilli.

1. Assistant Professor, Department of Internal Medicine, Khwaja Yunus Ali Medical College and Hospital, Enayetpur, Sirajganj, Bangladesh.
2. Professor, Department of Internal Medicine, Khwaja Yunus Ali Medical College and Hospital, Enayetpur, Sirajganj, Bangladesh.
3. Assistant Professor, Department of Internal Medicine, Khwaja Yunus Ali Medical College and Hospital, Enayetpur, Sirajganj, Bangladesh.
4. MD (Student), Department of Oncology, Khwaja Yunus Ali Medical College and Hospital, Enayetpur, Sirajganj, Bangladesh.

**Corresponding author:** Mohammad Abdul Motalib, Assistant Professor of Internal Medicine, Khwaja Yunus Ali Medical College and Hospital, Enayetpur, Sirajganj, Bangladesh. Cell: +880 1747766347, E-mail: mamtlb@gmail.com

TB infection of the cardiovascular system is transmitted mostly retrogradely from mediastinal lymph nodes, hematogenous from a primary tuberculosis infection, and rarely contiguously. Several studies have found that the right ventricle and right atrium are the most typically impacted, most likely due to the frequent involve¬ment of right mediastinal lymph nodes and subsequent myocardial involvement. Pericardial tuberculosis is more common in immuno-compromised patient, eg diabetic, HIV, organ- tranplanted patients & also stroid user. A Tuberculous pericarditis has four recognized pathological stages: (1) fibrinous exudation with initial polymorphonuclear leukocytosis with the loose organization of macrophages and T cells; (2) serosanguineous effusion with a predominantly lymphocytic exudate composed of monocytes and foam cells; (3) effusion absorption with the organization of granulomatous caseation and pericardial thickening; and (4) constrictive scarringThe most common symptoms of fluid accumulation are broad systemic symptoms or heart failure. When fluid accumulates rapidly, and compensatory mechanisms are unavailable, the patient develops tachycardia and hypotension. With inadequate treatment, up to half of patients may develop cardiac tamponade, with mortality rates as high as 85% at six months.

## Case Presentation

A 60-year-old male presented to the emergency department with complaints of progressively worsening dyspnea, orthopnea, and generalized fatigue & weight loss over the past four weeks. His medical history revealed a longstanding diagnosis of type 2 diabetes mellitus and chronic heart failure (New York Heart Association class II). On physical examination, the patient appeared cachectic evidenced by temporal muscle wasting, prominent zygoma, reduced MUAC and decreased skin fold thickness over triceps muscle. He is moderately anemic, bilateral pitting edema & he had tachycardia, low blood pressure, elevated jugular venous pressure, kussmaul's sign, pulsus paradoxus, increased area of cardiac dullness & muffled heart sounds on auscultation and his lung base revealed fine crepitation with vesicular breath sound. Laboratory investigations showed elevated inflammatory markers, including an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). The patient's glycosylated hemoglobin (HbA1c) levels were above the recommended target for diabetes management. Chest X-ray showed an enlarged cardiac silhouette, and an electrocardiogram revealed low-voltage QRS complexes with electrical alternance. Echocardiography demonstrated a massive pericardial effusion with signs of diastolic collapse, suggesting tamponade physiology. Computed tomography (CT) of the chest confirmed the pericardial effusion and revealed mediastinal lymphadenopathy. A pericardio-centhesis was performed and the fluid study showed that it was exudative with neutrophilic leukocytosis with high ADA level providing indirect evidence of tubercular infection. He has also positive MT test.

## Treatment

The immediate treatment was pericardiotomy with insertion of a drain tube by the cardiothoracic surgeon. The patient began to response with decreasing breathlessness & disappearance leg swelling. Daily collection of pericardial fluid was gradually decreasing in amount & turning its color from serosanguinous to straw color and then serous color ultimately. Upon confirmation of tubercular etiology through pericardial fluid analysis, our patient was promptly initiated on a standard four-drug anti-tubercular regimen comprising isoniazid, rifampicin, ethambutol, and pyrazinamide. The inclusion of corticosteroids, following the recommendations from landmark trials such as the Investigation of the Management of Pericarditis (IMPI) and the Tuberculous Pericarditis Trial 2 (TPT-2), aimed to mitigate inflammation and prevent the development of constrictive pericarditis. Prednisolone, at a dose of 1–2 mg/kg/-day, was initiated and tapered over several weeks under close monitoring for potential side effects. Simultaneously, the management of heart failure involved a meticulous balance between fluid restriction, diuretic therapy. The patient was started on an angiotensin-converting enzyme (ACE) inhibitor, with close attention to titration based on clinical response and tolerability. Given the coexistence of diabetes, the choice of antihyperglycemic agents was tailored to optimize glycemic control without exacerbating heart failure. Metformin was continued, while sulfonylureas and thiazolidinediones were avoided due to their potential for fluid retention. Regular follow-up assessments included clinical evaluation, imaging studies to monitor pericardial effusion resolution, and laboratory investigations to ensure therapeutic efficacy and detect any potential adverse effects. Throughout the course of treatment, the patient demonstrated gradual clinical improvement, with resolution of symptoms and a reduction in pericardial effusion size. A multidisciplinary team approach, involving specialists from various domains, facilitated the seamless integration of therapeutic interventions and optimization of care.

## Discussion

The presented case of Massive Tubercular Pericardial Effusion (MTPE) coexisting with Heart Failure (HF) and Diabetes Mellitus (DM) underscores the intricate interplay between infectious, cardiovascular, and metabolic pathologies. Tubercular pericarditis, though infrequent, requires vigilant consideration in the diagnostic algorithm, especially when entwined with other chronic conditions. The convergence of these entities introduces challenges in both diagnosis and management, necessitating a comprehensive, multidisciplinary approach.[3] The coexistence of MTPE with HF and DM accentuates the complexity of managing patients with overlapping comorbidities. Diabetes, recognized for its pro-inflammatory state and immune dysregulation, may potentiate the progression of tuberculosis, influencing the clinical course and complicating therapeutic decisions.[2] Moreover, the bidirectional relationship between diabetes and heart failure further amplifies the intricacy of this clinical scenario, as the management of one condition may inadvertently impact the other.[3] Guidelines for the treatment of tubercular pericarditis recommend a combination of anti-tubercular drugs and corticosteroids to mitigate inflammation and prevent constriction. Additionally, the management of heart failure in the context of concomitant tuberculosis necessitates a delicate balance to avoid exacerbating the infectious process.[4] Our patient's therapeutic journey involved collaboration between cardiology, infectious disease, and endocrinology specialists, reflecting the necessity of a unified approach to optimize patient outcomes.

## Conclusion

This case highlights the intricate relationship between massive tubercular pericardial effusion, heart failure, and diabetes mellitus. Successful management requires a coordinated effort from a multidisciplinary team to address the unique challenges posed by the intersection of these conditions. Early diagnosis, prompt initiation of appropriate therapies, and ongoing collaboration among specialists are crucial for optimizing outcomes in such complex cases.

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