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Post-Tuberculous Fibrosing Mediastinitis Complicated by Superior Vena Cava Syndrome and Chronic Pulmonary Embolism

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Abstract **Case Report**

Fibrosing mediastinitis (FM) is an unusual fibro-inflammatory disorder of the mediastinum that often leads to obstruction of major mediastinal structures. While histoplasmosis is the most common cause in some regions, tuberculosis remains an important etiology in endemic areas. We present a 48-year-old man with a history of superior vena cava (SVC) syndrome who presented with progressive dyspnea. CT angiography demonstrated extensive mediastinal fibrous invasion with thrombosis of the SVC, azygos vein, and left brachiocephalic vein, accompanied by an extensive collateral venous network. Additionally, there was chronic pulmonary embolism involving the right pulmonary artery and bilateral interstitial lung abnormalities. Microbiological investigation confirmed tuberculosis by GeneXpert. This case underscores the role of CT angiography in diagnosing FM and identifying complications and highlights the importance of considering tuberculosis as an etiology in endemic settings.

Keywords: Fibrosing mediastinitis; Tuberculosis; Superior vena cava syndrome; CT angiography; Chronic pulmonary embolism.

INTRODUCTION

Fibrosing mediastinitis (FM) is a rare pathological condition characterized by fibroproliferative tissue within the mediastinum that may compress or obstruct mediastinal structures, including vessels and airways. Although histoplasmosis is frequently implicated in Western literature, Mycobacterium tuberculosis is a recognized cause, especially in endemic regions such as Africa and Asia [1,2]. FM may present years after the primary infectious insult as a delayed immune-mediated fibroinflammatory response. The clinical manifestations are determined by the structures involved, with superior vena cava syndrome (SVCS) being among the most common vascular complications [3]. Imaging, particularly contrast-enhanced CT, is essential to define disease extent, identify complications, and aid in etiological assessment, but optimal management remains challenging [1,4].

CASE PRESENTATION

A 48-year-old man with a prior diagnosis of superior vena cava syndrome was referred for evaluation of progressive dyspnea. He had no known history of malignancy. Physical examination revealed marked thoracoabdominal collateral venous circulation, characterized by dilated and tortuous superficial veins over the anterior chest and abdominal wall (figure 1), reflecting chronic superior vena cava obstruction. Given the suspicion of vascular compromise, thoracic CT angiography was performed.

Imaging Findings

Contrast-enhanced thoracic CT demonstrated extensive infiltrative soft-tissue density within the mediastinum, corresponding to dense fibroinflammatory infiltration encasing mediastinal vessels and tracheobronchial structures (figure 2), typical of fibrosing mediastinitis.

There was complete thrombosis of the superior vena cava, extending into the left brachiocephalic trunk and the azygos vein, all with parietal calcifications and minimal enhancement after contrast (figure 3) consistent with chronic fibrotic occlusion. Secondary to SVC obstruction, prominent collateral venous circulation was observed (figure 6), including dilation of the hemiazygos vein, lumbar veins, and superior right intercostal veins. An extensive thoracoabdominal anterior venous

collateral network was also noted (figure 6), draining into the external iliac veins and effectively communicating the superior and inferior vena cava systems. Additional dilation involved the common iliac veins, renal veins, and the infrarenal inferior vena cava.

Evaluation of the pulmonary arteries revealed narrowing of the right pulmonary artery with parietal calcifications and endoluminal thrombosis, compatible with chronic pulmonary embolism (figure 5).

On lung parenchymal windows, bilateral alveolo-interstitial involvement was identified, more pronounced on the right, along with multiple bilateral pulmonary nodules (figure 4) suggestive of an infectious process, particularly tuberculosis. A large, loculated right pleural effusion was also noted (figure 6(b))**.**

Subsequent microbiological analysis using GeneXpert confirmed *Mycobacterium tuberculosis* infection.

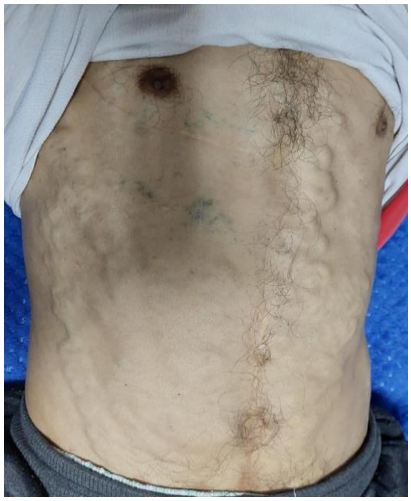

Figure 1: Clinical image demonstrating marked thoracoabdominal collateral venous circulation, characterized by dilated and tortuous superficial veins extending over the anterior chest and abdominal wall, reflecting chronic superior vena cava obstruction

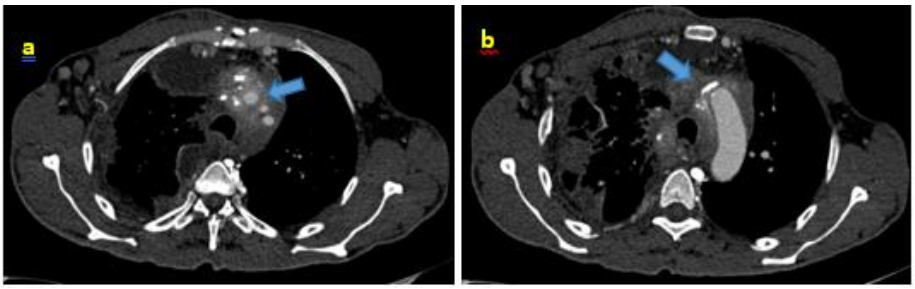

Figure 2: Contrast-enhanced thoracic CT demonstrating extensive infiltrative soft-tissue density within the mediastinum, encasing mediastinal vessels and tracheobronchial structures

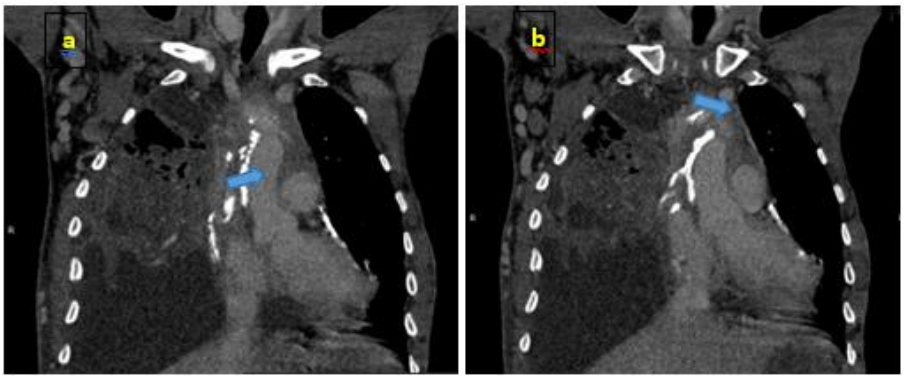

Figure 3: Contrast-enhanced thoracic CT (coronal) showing a complete thrombosis of the superior vena cava (a), extending into the left brachiocephalic trunk (b) all with parietal calcifications and minimal enhancement after contrast

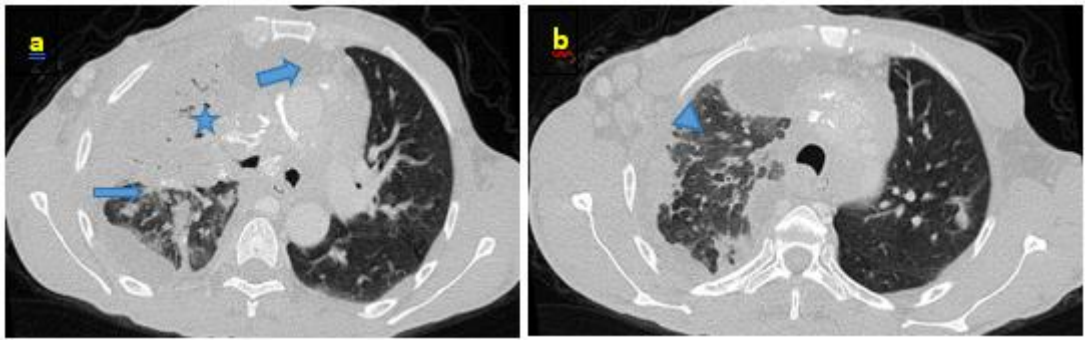

Figure 4: On lung parenchymal window we note a bilateral alveolo-interstitial involvement, more pronounced on the right (pulmonary consolidation (asterisk (a)); ground-glass opacity (arrowhead (b))..), along with multiple bilateral pulmonary nodules (arrow (a)) suggestive of an infectious process

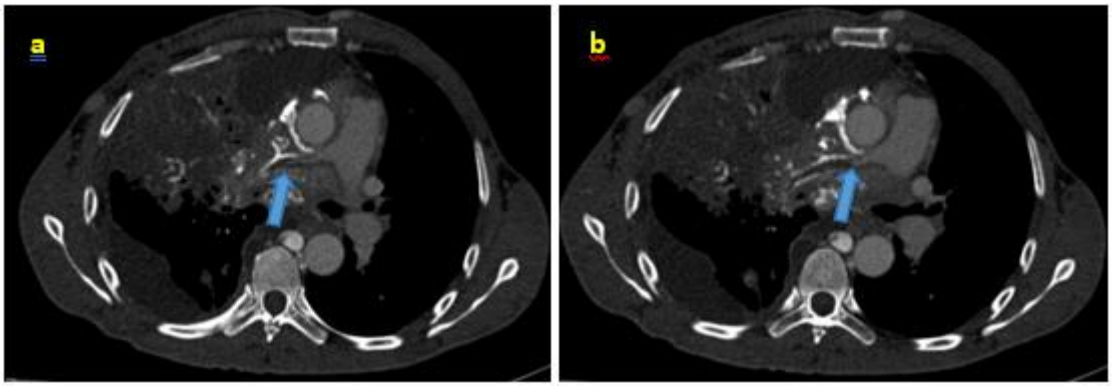

Figure 5 (a, b): Contrast-enhanced thoracic CT showing a narrowing of the right pulmonary artery with parietal calcifications and endoluminal thrombosis (arrow (a, b)), compatible with chronic pulmonary embolism

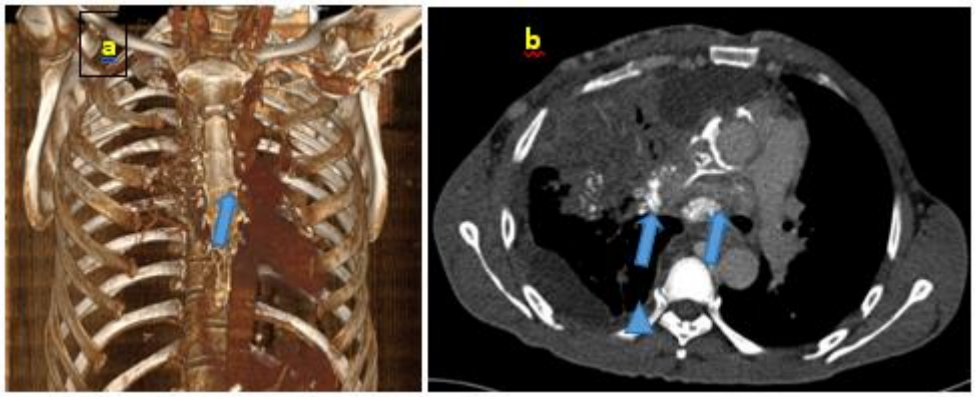

Figure 6: Contrast-enhanced thoracic CT showing a collateral venous circulation and a large (arrow (a, b)), loculated right pleural effusion was also noted (arrowhead (b))

DISCUSSION

Fibrosing mediastinitis represents a rare fibroinflammatory process that can result from a variety of etiologies, including infections (most commonly histoplasmosis and tuberculosis), autoimmune disorders, and idiopathic processes [1,5]. In tuberculosis-endemic regions, post-tuberculous FM should be considered in patients with unexplained mediastinal fibrosis and vascular complications.

The clinical presentation of FM varies widely and depends on the anatomy involved. SVCS is a frequent manifestation due to progressive venous obstruction, and extensive collateral circulation reflects chronic adaptation to long-standing venous blockage [3,6]. Pulmonary vascular involvement, as seen in this case with chronic pulmonary embolism and arterial narrowing, can be associated with significant morbidity and may contribute to pulmonary hypertension [7,8]. While FM may not always be reversible, its complications often determine clinical outcomes.

Contrast-enhanced CT angiography is the cornerstone for diagnosing FM, enabling detailed visualization of fibrotic infiltration, vascular encasement, and collateral pathways. The presence of dense fibrous tissue with calcifications on imaging can aid differentiation from malignant mediastinal processes

[4,9]. Additionally, lung parenchymal findings such as nodules and interstitial changes may raise suspicion for infection, thereby guiding further microbiological testing.

Management of FM is multifaceted and often supportive. Antituberculous therapy is required in cases with active infection, although it may not reverse established fibrosis. Anticoagulation may be indicated for associated thrombosis, and interventional or surgical procedures may be considered for selected complications, such as stenotic lesions amenable to stenting [10]. Pulmonary hypertension management is critical when pulmonary vascular involvement is significant, although evidence for specific therapies remains limited.

CONCLUSION

Post-tuberculous fibrosing mediastinitis is a rare but potentially debilitating condition that can lead to major vascular complications including superior vena cava syndrome and chronic pulmonary embolism. Contrast-enhanced CT angiography is essential for accurate diagnosis, assessment of disease extent, and identification of complications. In regions with a high prevalence of tuberculosis, clinicians should maintain a high index of suspicion for post-tuberculous FM. Early recognition and multidisciplinary management may improve outcomes, though therapeutic options remain limited.

Conflict of Interest: The authors declare no conflict of interest.

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