

Case Report Vascular Interventional

Pulmonary venous outflow obstruction secondary to fibrosing mediastinitis triggered by histoplasmosis: A case report

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ABSTRACT

We report a case of long-standing fibrosing mediastinitis (FM) in a 63-year-old female with prior *Histoplasma capsulatum* infection, complicated by complete obstruction of the left pulmonary venous outflow and marked hypoperfusion of the left lung. Remarkably, the patient remained largely asymptomatic despite extensive anatomic compromise, offering important insights for imaging-based evaluation and management. A ventilation-perfusion (V/Q) scan demonstrated left lung hypoperfusion, and computerized tomography revealed calcified nodules suggestive of fibrosis. Digital subtraction pulmonary angiography confirmed elevated pressures across the main, right, and left pulmonary arteries and absence of outflow through the left superior and inferior pulmonary veins with cavernous transformation. While recanalization was considered, intervention was deferred given severe obstruction and minimal symptoms. This case underscores the role of multimodality imaging in diagnosing venous outflow obstruction and highlights compensatory vascular remodeling in chronic FM. The educational value lies in balancing intervention versus observation in asymptomatic patients with significant structural disease.

Keywords: Angioplasty, Fibrosing mediastinitis, Histoplasmosis, Pulmonary venous obstruction, Stenting

INTRODUCTION

Fibrosing mediastinitis (FM) is a rare condition which is characterized by the replacement of mediastinal fatty tissue with dense, fibrous tissue, often in response to an inflammatory or immunologic trigger.^[1] FM can be triggered by respiratory infections with the fungus *Histoplasma capsulatum*, which can create mediastinal granulomas that ultimately rupture and release fungal antigens that propagate the fibroinflammatory reaction, which is characteristic of FM's presentation.^[2]

Although FM is traditionally characterized as a benign condition, it still has significant impacts on the functions of the cardiovascular and respiratory systems. At present, FM has been shown to be associated with pulmonary hypertension and subsequent cor pulmonale, superior vena cava syndrome secondary to lateral extension of fibrosis, chylothorax, and symptoms such as dysphagia or neurologic deficits due to thoracic nerve impingement.^[3] However, there is still a paucity of literature regarding its associations with the pulmonary vasculature, with a majority of clinical knowledge on the subject being derived from case presentations.

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In this report, we present a case of long-standing FM in a 63-year-old female with a previous history of *H. capsulatum* infection complicated by pulmonary vein obstruction and hypoperfusion of the left lung, and discuss the resulting compensatory changes made to her cardiopulmonary circulation as seen on digital subtraction pulmonary angiography, as well as the utility of endovascular treatment using pulmonary vein angioplasty and stenting.

CASE PRESENTATION

A 63-year-old female with a medical history of FM secondary to *Histoplasmosis* diagnosed in 1997 presented to our department for evaluation of suspected pulmonary vein outflow obstruction using pulmonary angiography with hemodynamics. Her referral to interventional radiology was prompted by her previous evaluation with ventilation-perfusion (V/Q) scintigraphy 2 months prior, which showed the left lung receiving 3% of total pulmonary perfusion and only contributing to 44% of total pulmonary ventilation [Figures 1 and 2]. At the time of visit,

she was in no apparent distress, with her oxygen saturation measured at 98% and respirations of 16 breaths/min. On review of systems, she had no presentations of dyspnea, fever, or chest pain. Physical examination revealed no wheezing or rales, accessory muscle use, or uneven chest expansion. Her most recent pulmonary function testing 1 month prior revealed a forced vital capacity (FVC) of 2.81 (85% of predicted), forced expiratory volume (FEV) in 1 s of 1.95 (76% of predicted), and an FEV/FVC of 70%.

Her prior medical care consisted of long-term evaluations by an in-house pulmonologist for her obstructive sleep apnea, recurrent left-sided pneumonia, and history of asthma. Her most recent assessment with pulmonology was 1 month prior for an asthma exacerbation, which revealed a resolving respiratory infection that was managed with amoxicillin-clavulanate. Her asthma was managed long-term with tiotropium twice daily and budesonide/formoterol as needed and was shown to be well controlled at the time of visit with a fractional exhaled nitric oxide of 11 ppb and an asthma

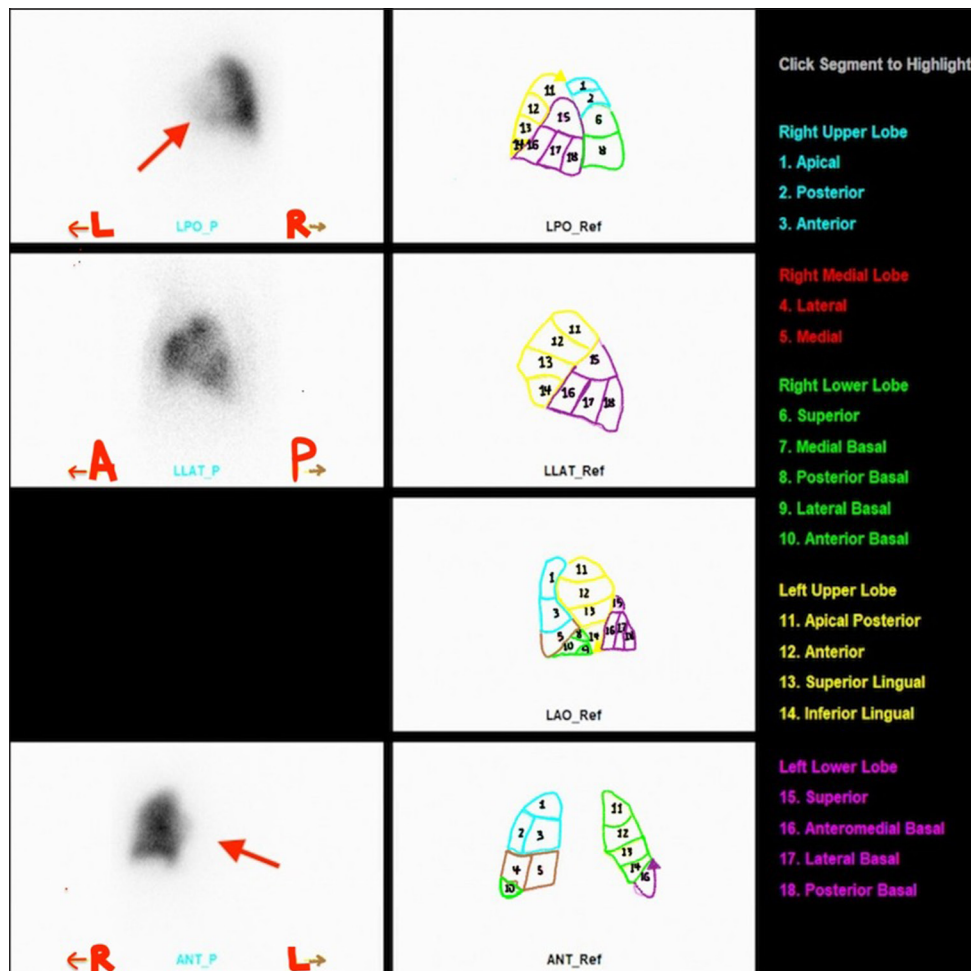


Figure 1: 63-year-old woman with fibrosing mediastinitis presenting with minimal symptomatology. V/Q perfusion images demonstrating no measurable perfusion to the left lung (red arrows) after intravenous administration of 4.2 mCi Tc-99m macroaggregated albumin. V/Q: Ventilation-perfusion.

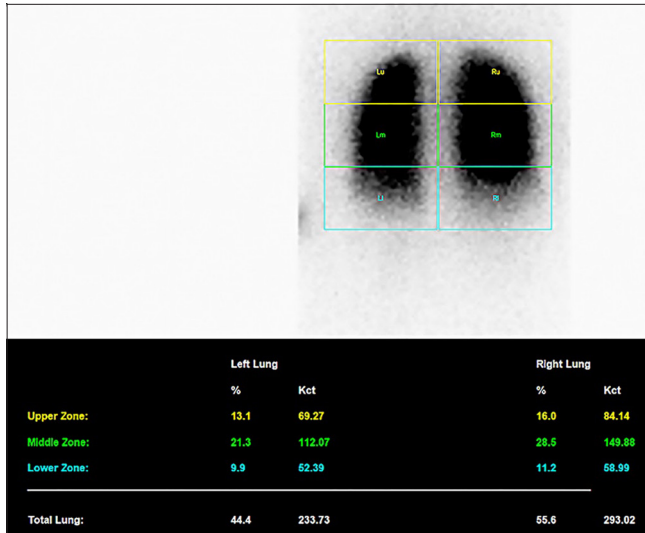


Figure 2: 63-year-old woman with fibrosing mediastinitis presenting with minimal symptomatology. V/Q ventilation images after inhalation of 21 mCi Xe-133 gas demonstrate the right lung contributing 56% and the left lung contributing 44% to total pulmonary ventilation. There is a whole lung mismatch present involving the left lung. V/Q: Ventilation-perfusion.

control test score of 20. Her obstructive sleep apnea was managed with a continuous positive airway pressure (CPAP) machine, but she was non-compliant in using it. She did not have a history of active smoking and had not smoked in over 40 years at the time of visit. When asked about her exercise tolerance, she was capable of participating in Zumba classes with minimal fatigue and walking with her friends.

Other pertinent medical history included renal carcinoma of the left kidney treated with a partial nephrectomy, and type 2 diabetes mellitus with a hemoglobin A1c of 6.1% that is managed with empagliflozin.

In our technique, the right common femoral vein was accessed for diagnostic assessment and for possible intervention. A 6 French sheath was placed, and a 0.035" wire and 6 French angled pigtail catheter were advanced into the main pulmonary artery. Digital subtraction angiograms were performed in anteroposterior and left anterior oblique projections [Figure 3a and b]. These demonstrated a near absence of perfusion to the left lung, and a narrowing of the left pulmonary artery with lack of contrast flow distally and shunting of flow to the right pulmonary artery. The right lung demonstrated physiologically normal perfusion. In the delayed phase, there was an absence of discernable outflow through the left superior and inferior pulmonary veins.

To prepare for pulmonary artery pressure measurements, an exchange was made for an angled multi-port balloon catheter (MPB) catheter with side holes over a Bentson wire. The catheter was advanced past the area of caliber change in the

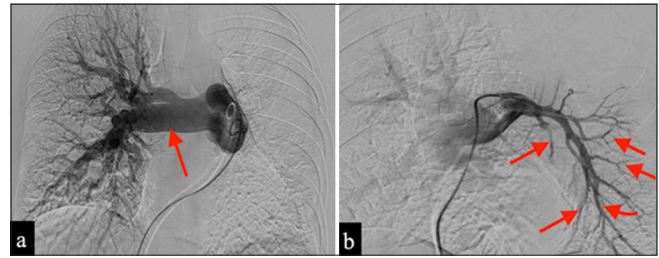


Figure 3: (a) 63-year-old woman with fibrosing mediastinitis presenting with minimal symptomatology. Digitally subtracted angiogram of the main pulmonary artery demonstrating no opacification of the left pulmonary artery (red arrow) and preferential flow of contrast into the right pulmonary arterial system. (b) Selective digitally subtracted angiogram of the left main pulmonary artery showing focal narrowing of the left lobar pulmonary artery with poor opacification of the distal branches (red arrows) and no significant enhancement of the pulmonary parenchyma on the left.

left main pulmonary artery. A selective digital subtraction angiogram was performed, demonstrating atrophy of the left pulmonary arterial vasculature, persistent absence of perfusion of the left lung, and suspected shunting of blood to the right pulmonary artery. The configuration of the cavernous transformation did not allow for a reliable point of catheterization through retrograde approach from the systemic arterial side. Measurements directly in the left atrium would not allow for assessment of the pressurized circuit in the capillary bed. Therefore, a pulmonary capillary wedge pressure using a Swan-Ganz catheter was used as a surrogate for capillary bed and pulmonary venous pressures. Left pulmonary artery pressure distal to the area of luminal narrowing was measured to be 39/12 (25) mmHg. Left pulmonary artery pressure proximal to the area of luminal narrowing was measured to be 39/16 (26) mmHg. The right pulmonary artery was then selected, and a pressure of 39/18 (27) mmHg was measured. The catheter was then positioned within the main pulmonary artery, and a pressure of 38/19 (27) was measured. Considering the procedural findings, stenting was deemed futile. All wires and catheters were removed, hemostasis was achieved with manual compression, and a sterile dressing was applied to the catheterization site. The patient was discharged after 2 h of post-procedure observation.

DISCUSSION

FM has been shown to present either from acquired causes or idiopathically. FM from acquired causes can be characterized as granulomatous, often arising from infections with *Mycobacterium tuberculosis* and *H. capsulatum*, or non-granulomatous, typically resulting from autoimmune diseases such as Behcet syndrome, radiation exposure, and cabergoline or methysergide therapy.^[4,5]

The most reliable imaging modality for confirming the diagnosis of FM is computerized tomography (CT) with contrast, which can enhance the fibrotic tissue and calcifications, as well as visualizing the extent of visceral encroachment [Figure 4a and b].^[6] For characterizing vascular changes secondary to granulomatous FM, pulmonary angiography remains a strong reference standard and can also be used to deliver interventions such as vascular stenting.^[6,7]

Pulmonary vascular remodeling is a physiologic consequence of obstruction or stenosis distal to the area being remodeled, serving as a compensatory mechanism to enable blood flow past areas with elevated resistance and prevent congestion of the cardiopulmonary circuit.^[8] Vascular remodeling can take many forms, often manifesting as hypertrophy of the tunica media or intimal and adventitial fibrosis. In addition to the modification of preexisting pulmonary vasculature, new arteriovenous malformations can arise to accommodate elevated arterial pressures through vascular endothelial growth factor and fibroblast growth factor secretion, resulting in direct artery-to-vein connections which reduce resistance in the circuit at the expense of proper alveolar gas exchange.^[9]

In the case of our patient, her initial infection with *H. capsulatum* resulted in antigenic irritation of the mediastinum and subsequent development of fibrotic scar tissue capable of externally compressing critical mediastinal structures and causing pulmonary venous outflow obstruction. Prior CT scans revealed calcified nodules in the lower lobe of her left lung, which matched the expected presentation of pulmonary histoplasmosis.

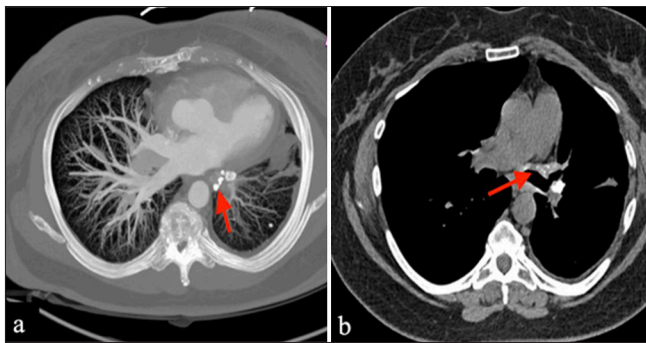


Figure 4: (a) 63-year-old woman with fibrosing mediastinitis presenting with minimal symptomatology. Axial maximum intensity projection of this computed tomography angiography chest through the level of the left atrium demonstrates no pulmonary venous drainage on the left (red arrow). (b) 63-year-old woman with fibrosing mediastinitis presenting with minimal symptomatology. Axial non-contrast view of the chest at the level of the main pulmonary artery shows fibrotic thickening and coarse calcification (red arrow) of the posterior mediastinum encasing the left hilum.

Our patient's pressure measurements indicated that there were diffusely elevated pressures across both branches of the pulmonary artery and the main pulmonary trunk. The V/Q scan showed a nearly complete lack of perfusion to the left lung, matching the luminal narrowing of the left pulmonary artery seen on pulmonary angiography. CT of the chest demonstrated volume loss of left lung parenchyma, most likely due to decreased perfusion to the lung. Prior echocardiography showed mild left ventricular concentric hypertrophy with preserved ejection fraction and filling, but there was no evidence of right heart strain or structural changes indicative of cor pulmonale.

Interventional management for this patient's pulmonary venous outflow obstruction involved consideration for percutaneous pulmonary vein angioplasty with stent placement to restore blood flow through the outflow tract.^[10] However, in the setting of no discernable left pulmonary veins on a delayed phase angiography series, and the presence of cavernous transformation, she would not be a candidate for recanalization. Moreover, previous studies indicated high rates of restenosis following pulmonary vein stenting, with evidence of little in mortality with transient symptomatic improvement. In addition, the patient's health had minimally deteriorated throughout the years she spent following up with her pulmonologist, and she remained relatively asymptomatic with adequate tolerance for exercise and activities of daily living. The patient was satisfied with her care and understood why she was not a good candidate for revascularization, and the decision was made to discharge the patient after 2 h of post-procedure observation, with follow-up visits scheduled with pulmonology for additional pulmonary function testing and monitoring.

CONCLUSION

FM can result from prior infection with *H. capsulatum*, triggering permanent calcific changes to the mediastinal structures. Pulmonary venous outflow obstruction is a potential consequence of these calcifications and can cause compensatory vascular remodeling with elevated arterial pressures to alleviate congestion of the pulmonary circulation. Although treatment options such as percutaneous stenting exist to alleviate symptoms, there are high rates of long-term restenosis with minimal improvement to mortality. In patients with mild to minimal symptoms, the condition is best managed with symptomatic treatment and routine follow-up.

Availability of data and material

Data sharing is not applicable to this article as no datasets were generated or analyzed during the present study.

Authors' contributions: AB wrote the manuscript, AK was responsible for the editing and image acquisition, SS was responsible for the initial patient consultation and associated notes, and SS and SAB performed the procedure. All authors read and approved the final manuscript.

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