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Internal Medicine Section

Navigating Complex Pulmonary Hypertension: A Case of Post-tubercular Lung Disease and Atrial Septal Defect

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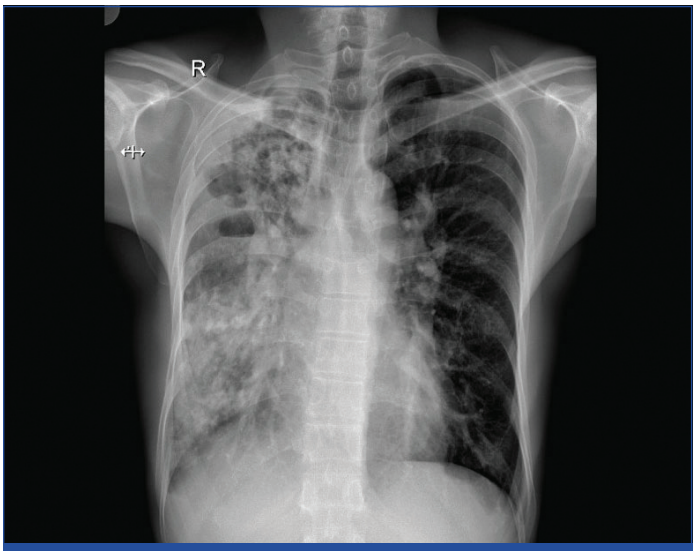
Authors present a case involving a 47-year-old male farmer who sought medical attention at the Outpatient Department (OPD) due to a persistent cough and progressive dyspnoea over the past year. Additionally, he reported experiencing intermittent low-grade fever for the last 10 days, managed with 500 mg of paracetamol prescribed by a local doctor. The patient had a history of being diagnosed with drug-sensitive, sputum-positive pulmonary tuberculosis four years ago and had undergone treatment with HRZE (Isoniazid-300 mg, Rifampicin-450 mg, Pyrazinamide-1500 mg, Ethambutol-1200 mg) for a duration of six months.

Upon admission, the patient was afebrile but had tachycardia (120/min), tachypnoea (28/min), a blood pressure reading of 80/50 mmHg, and an oxygen saturation level of 88% on room air.

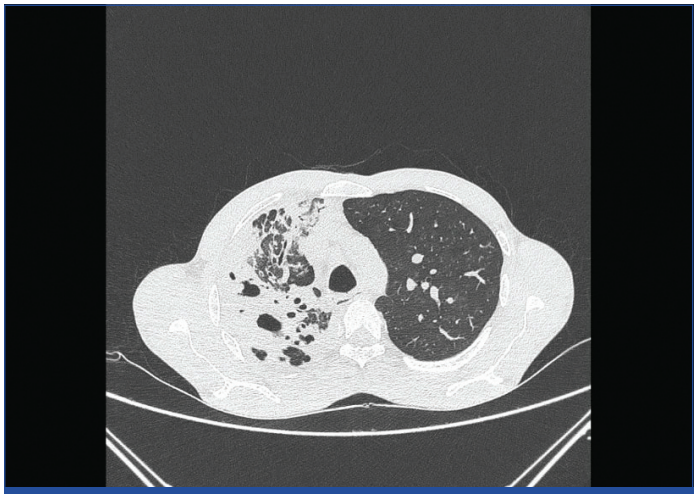
Clinical examination revealed tracheal deviation to the right and drooping of the right shoulder. Impaired percussion notes were observed in the right supra and infra-scapular, infra-axillary, and mammary areas. Auscultation revealed bronchial breath sounds with coarse crepitations in these areas. The cardiovascular system revealed a wider fixed splitting of S2 and a systolic murmur at the upper left sternal border.

Chest radiography revealed scarring and cavities in the right lung [Table/Fig-1]. Contrast-enhanced Computed Tomography (CECT) confirmed these findings and additionally identified areas of bronchiectasis. Multiple acinar nodules with alveolar consolidation were noted in the basal segment of the right lower lobe, alongside dilatation (32 mm) of the main pulmonary artery [Table/Fig-2].



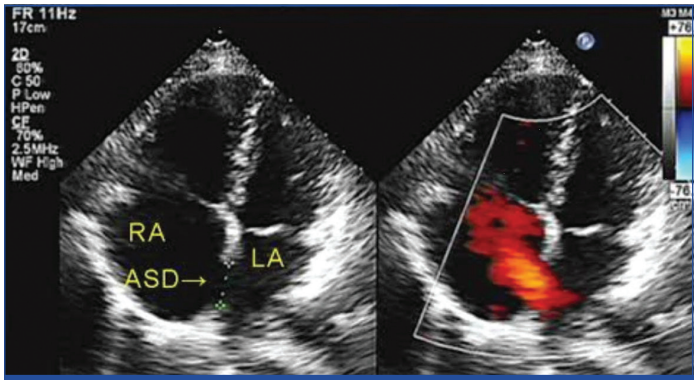
[Table/Fig-1]: Chest radiograph Posteroanterior (PA) view-Cicatrised right lung showing scarring, nodular opacities and cavitation with air fluid levels.

Staining, culture studies, and Gene Xpert testing for Mycobacterium tuberculosis in sputum and bronchoalveolar lavage yielded negative results. Mild leucocytosis was managed with parenteral antibiotics.



[Table/Fig-2]: CT chest, axial view-scarring, cavitation, bronchiectasis and volume loss of right lung. Left lung shows dilated pulmonary arteries.

A 2D echocardiogram provided insights into the patient's cardiac status, revealing an ejection fraction of 50%. It also highlighted a large ostium secundum Atrial Septal Defect (ASD) with a left-to-right shunt, dilated right atrium, right ventricle, mild tricuspid regurgitation, and moderate pulmonary artery hypertension, with a right ventricular systolic pressure of 55 mmHg [Table/Fig-3].



[Table/Fig-3]: A 2D Echo- A large ostium secundum Atrial Septal Defect (ASD) with a left-to-right shunt, dilated right atrium, right ventricle, mild tricuspid regurgitation, and moderate pulmonary artery hypertension, with a right ventricular systolic pressure of 55 mmHg.

Transoesophageal echocardiography confirmed the size of the ASD (30 mm) and rim measurements, thus solidifying the diagnosis of Pulmonary Hypertension (PH) secondary to Post-tubercular Lung Disease (PTLD) and ASD, classifying it under both World Health Organisation (WHO) Groups 2 and 3.

Navigating complex PH involves a multidisciplinary approach with specialists like pulmonologists and cardiologists. Treatment includes medications, lifestyle changes, and in severe cases, surgery or lung transplantation. The World Symposium on Pulmonary Hypertension (WSPH) defines PH as a mean Pulmonary Artery Pressure (mPAP)

greater than 20 mmHg measured by Right Heart Catheterisation (RHC). Respiratory causes of PH are obstructive lung disease, restrictive lung disease, other lung diseases with a mixed restrictive/obstructive pattern, hypoxia without lung disease, and developmental lung disorders [1]. Group 2 PH includes PH due to left heart failure. Cardiac causes of PH are heart failure with preserved left ventricular ejection fraction, heart failure with reduced left ventricular ejection fraction, valvular heart disease, and congenital/acquired cardiovascular conditions [2]. Group 3 PH is due to lung disease and/or hypoxia. Causes include obstructive lung disease, restrictive lung disease, other lung diseases with a mixed restrictive/obstructive pattern, hypoxia without lung disease, and developmental lung disorders [3]. PTLD encompasses a range of disorders affecting the airways, lung parenchyma, pulmonary vasculature, and pleura. It significantly reduces life expectancy and increases the risk of recurrent tuberculosis [4]. Managing present complex case necessitated consideration of early ASD closure and medical therapy. Current guidelines recommend ASD closure when there is still a net left-to-right shunt and the Pulmonary Vascular Resistance (PVR) is less than two-thirds of systemic vascular resistance, to prevent sustained PH and right heart failure [5].

To address the patient's condition, authors devised a therapeutic plan that included Endothelin Receptor Antagonists (ERA), Phosphodiesterase type 5 Inhibitors (PDE5), and home oxygen therapy. These interventions aimed to improve haemodynamics, enhance exercise capacity, boost functional status, elevate overall quality of life, and potentially extend survival [6]. The elevated occurrence of PH in post-tuberculosis pulmonary sequelae underscores the imperative for early detection, appropriate treatment, and strict adherence to pulmonary tuberculosis treatment regimens. National and international tuberculosis programs should incorporate guidelines for long-term follow-up to mitigate the morbidity and mortality linked to PH [7].

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