A 45-year-old male patient, normotensive, non-diabetic, married with children, auto parts dealer by profession, presented to us with complaints of cough with expectoration, low grade fever and breathlessness from past 15 days. He gave no history of previous diseases or surgical intervention. General examination was unremarkable. On respiratory system examination, the left sided shoulder was drooping; trachea deviated to left and apex beat palpable in fifth intercostal space at left mid axillary line. On percussion, impaired to dull note was observed all over the left side. On auscultation, breath sound were absent in the left axillary and infra axillary areas; decreased intensity breath sounds could be heard in the left inter scapular and infra scapular areas. Occasional crepitations were heard over the right hemithorax. CVS (Cardio-vascular system), CNS (Central nervous system) and per-abdominal examinations revealed no abnormality. TLC (Total leukocyte count) was 12,400/mm$^3$, kidney and liver function tests were normal and the Mantoux test was negative. Chest radiograph (PA view) demonstrated opaque left hemithorax with volume loss, ipsilateral mediastinal shift, right sided compensatory hyperinflation and scoliosis of dorsal spine with concavity to the left side [Figure 1]. USG of abdomen was normal. Contrast enhanced CT scans of chest lung window [Figure 2].

**QUESTION**

What is the diagnosis?

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ANSWER

Pulmonary hypoplasia (left).

DISCUSSION

On CECT chest, trachea and both the bronchi are normal. There is volume loss in the left hemithorax with ipsilateral mediastinal shift, raised left hemidiaphragm and herniation of right lung parenchyma. Only a very small area/volume of the left lung is aerated. Mediastinal window showed displaced mediastinum heart and mediastinal structures and the rudimentary left pulmonary trunk.

Pulmonary agenesis/aplasia/hypoplasia has been classified by Schneider[1] and later modified by Boyden.[2] It differentiates between them on the basis of presence or absence of the lung tissue, bronchi or the pulmonary artery [Table 1].

Pulmonary hypoplasia is incomplete development of the lungs, resulting in an abnormally low number or size of bronchopulmonary segments or alveoli. Incidence of pulmonary hypoplasia ranges from 9-11 per 10,000 live births.[3] The developmental anomalies of the lung between the 4th and 24th gestational weeks may cause functional damage which is usually discovered in newborns and infants, but it can rarely present in adulthood.[4] Practically, earlier the anomaly is present, proportionately, the branching of the tracheal-bronchial tree is reduced.[5]

Contrast enhanced computed tomography (CECT) of the chest revealed that only a very small area/volume of the left lung was aerated [Figure 2]. In 70% of cases, the left lung is affected,[6] as was seen in the present patient. Mediastinal window showed displaced mediastinum, heart and mediastinal structures and the rudimentary left pulmonary trunk [Figure 3].

On bronchoscopy, vocal cords were moving normally. Carina was central and sharp, right bronchial tree was well within the normal limits. The left main bronchus was normal up to the first inch, but had a slit like appearance distal to it; bronchoscope could not be negotiated further. Few mucopurulent secretions were seen in the left bronchial stump which were collected and sent for different stains and cultures.

Differential diagnosis of a completely opaque hemithorax is massive pleural effusion, lobar consolidation and complete collapse of the lung, followed by agenesis/aplasia/hypoplasia of the lung, post-pneumonectomy and large space occupying the lesions (SOL). As the mediastinum was shifted towards the same side and there were evidences of volume loss; effusion, consolidation and SOL were ruled out. CECT chest showed rudimentary left pulmonary artery [Figure 3] and small area of lung parenchyma [Figure 2] in the left lung. As per classification by Boyden[2] [Table 1], all the 3 components (lung tissue, artery and the bronchus) were present, but in an under-developed form, thus categorizing the present case into pulmonary hypoplasia.

Pulmonary hypoplasia is usually diagnosed during infancy or early childhood, but many patients remain asymptomatic and the diagnosis may not be made until adulthood.[7] It is a common cause of neonatal death.[8] Wigglesworth and Desai reported an incidence of 14.5% in a large series of perinatal necropsies.[9] Hypoplastic lung, when present, is prone for atelectasis and pulmonary infection and associated congenital anomalies.[7]

The patient was empirically treated for LRTI and discharged in a stable condition. The present case is being reported as pulmonary hypoplasia in itself is rare, it was diagnosed during the adulthood and it is an important element of differential diagnosis of unilateral opaque hemithorax, so the case would be interesting for postgraduate as a radiologic quiz.

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