

Case Report

Disseminated tuberculosis presenting as pneumomediastinum: a case report

Aditya Dhananjay Phadte^{1*}, Chitralkha Anilkumar Nayak¹, Ramona Menezes²,
Akshay Surendra Naik³

¹Department of Medicine, ²Department of Radiology, ³Department of Surgery, Goa Medical College, Goa, India

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*Correspondence:

Dr. Aditya Dhananjay Phadte,
E-mail: phadteaditya@gmail.com

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ABSTRACT

This study describes a rare case of a 19-year-old girl who presented with features of subcutaneous emphysema and pneumomediastinum to the emergency department in whom further investigation revealed pulmonary and neurotuberculosis. Tuberculosis presenting as pneumomediastinum and subcutaneous emphysema is a rare, but an important entity and a better knowledge of this condition can help in the early diagnosis and adequate management of tuberculosis.

Keywords: Miliary, Spontaneous pneumomediastinum, Subcutaneous emphysema, Tuberculosis

INTRODUCTION

Pneumomediastinum, also known as mediastinal emphysema was first described by Laënnec in 1819 and is defined as the presence of free air in the mediastinal cavity.¹ It is classified into two types, one is secondary pneumomediastinum as a result of trauma or iatrogenic due to endoscopic or other therapeutic procedures. The second is the presence of free air within the mediastinum without a clear etiology, also known as spontaneous pneumomediastinum. Pneumomediastinum occurring in tuberculosis is a rare presentation and can cause dilemma in the diagnosis and management of the patient.² Here, we present a case of disseminated tuberculosis in a 19-year-old girl who presented with features of pneumomediastinum in the emergency department at admission.

CASE REPORT

A 19-year-old female with no prior illness presented to the emergency department with breathlessness for 1

month, associated with cough and moderate grade fever for 15 days.

The patient had developed worsening of dyspnea 2 days prior to the admission. The cough was associated with minimal mucoid expectoration and the patient denied any history of haemoptysis. There was no history of chest trauma, weight loss, skin rash, vomiting, abdominal pain, loose motions. Patient denied any history of Koch's contact.

On examination, the patient's pulse rate was 120beats/min, BP was 140/80mm Hg and respiratory rate was 30/min. General examination revealed pallor. There was no icterus, lymph node enlargement, clubbing, cyanosis, pedal oedema.

There was subcutaneous emphysema on local chest examination. Respiratory system examination revealed bilateral coarse crepitations in all areas with findings much more pronounced in the right lung.

The patient was drowsy, yet arousable. Associated neck stiffness was present. She had no other cranial deficits. Rest of the systemic examination was normal.

Laboratory investigations revealed a total leukocyte count of $7200/\text{mm}^3$ with neutrophilic predominance of 89%. The haemoglobin was 10.78gm% and platelet count were $1.4 \text{ lakh}/\text{mm}^3$. Renal function tests were normal and liver function tests revealed hypoalbuminemia.

Chest X-ray showed pneumomediastinum with subcutaneous emphysema in the right axillary region. Numerous miliary nodules and patchy areas of consolidation were noted in both lungs with blunting of the left costophrenic angle (Figure 1).

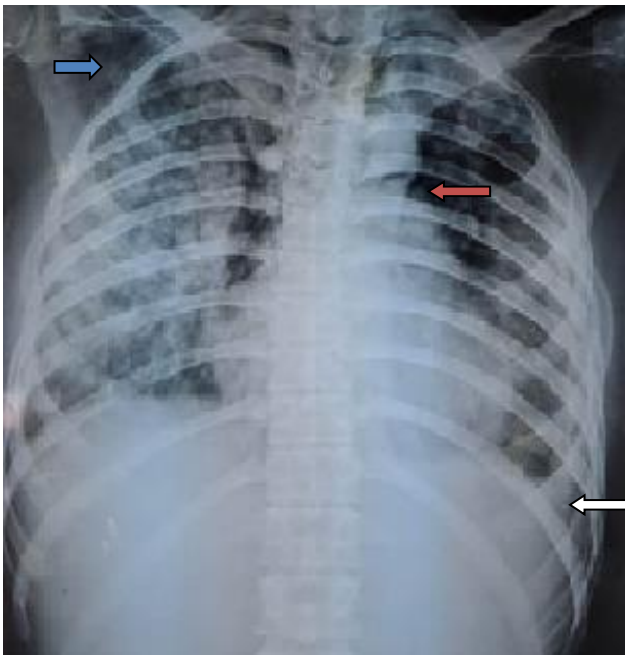


Figure 1: Chest x ray showing lucency outlining the mediastinal contours consistent with pneumomediastinum (red arrow). Streaky areas of lucency are noted in the right axilla consistent with subcutaneous emphysema (blue arrow). Numerous miliary nodules and patchy areas of consolidation are noted in both lungs. there is blunting of the left costophrenic angle (white arrow).

CT thorax revealed miliary nodules in both lungs, with areas of consolidation predominantly in right lower lobe, with pneumomediastinum and left pleural thickening (Figure 2).

Sputum examination was normal. Tests for malaria, leptospirosis, enteric fever, blood culture, urine culture, HIV ELISA were normal. CT Brain showed acute infarcts in the both frontal lobes, in parafalcine location and in the right caudate nucleus with mild hydrocephalus (Figure 3). CSF examination showed lymphocytic predominance with low glucose and high protein of 170mg/dl and was positive for TB PCR.

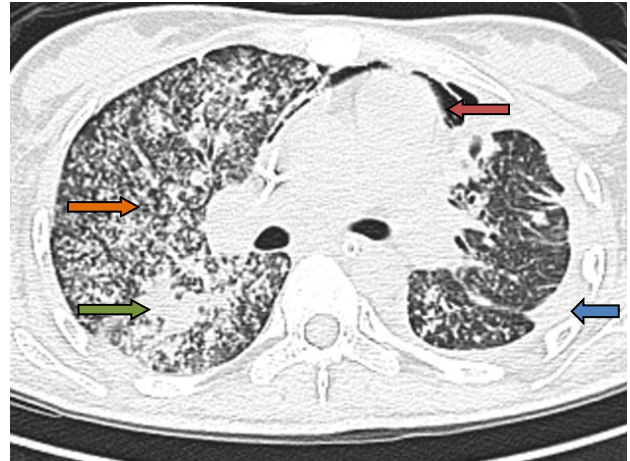


Figure 2: HRCT thorax of the patient showing pneumomediastinum (red arrow), miliary nodules (orange arrow), focal areas of consolidation (green arrow) and left pleural thickening (blue arrow).

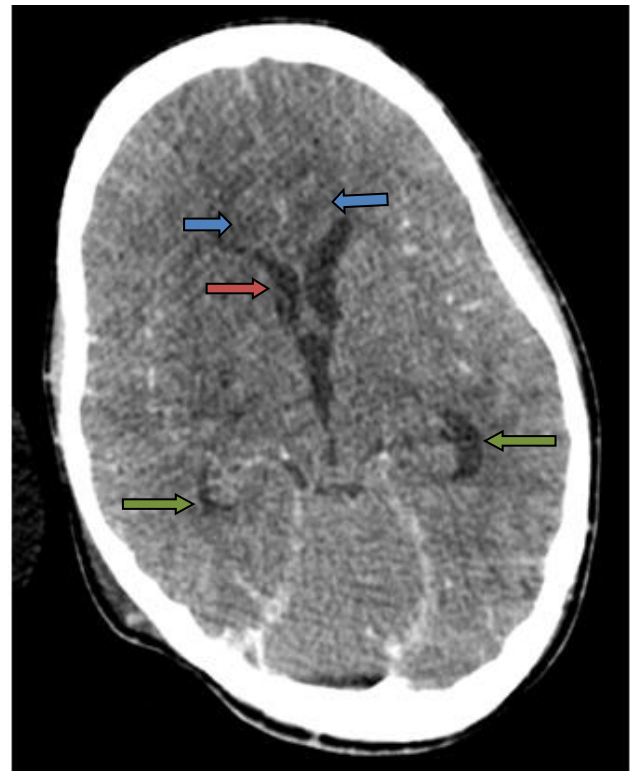


Figure 3: Post contrast CT brain showing acute infarcts in both frontal lobes in parafalcine location (blue arrows) and in the right caudate (red arrow). There is prominence of the temporal horns of the lateral ventricles consistent with early hydrocephalus (green arrows).

The patient was shifted to ICU for ventilatory support and started on antituberculous drug therapy and steroids. She was afebrile within 72 hours of starting antituberculous therapy. Repeat CXR showed significant resolution of the pneumomediastinum as well as the consolidation. She was gradually weaned from ventilator

support. However, there was neuroworsening on the 20th day of admission and repeat CT brain showed worsening of hydrocephalus. Patient suffered cardiorespiratory arrest and could not be revived in spite of rigorous resuscitative efforts.

DISCUSSION

Subcutaneous emphysema and pneumomediastinum are relatively common in critically ill patients of blunt or penetrating trauma, soft-tissue infection, or any condition that creates a gradient between intra-alveolar and perivascular interstitial pressures (as in the case of inappropriate mechanical ventilation or foreign body inhalation).³ Most cases of pneumomediastinum probably are the result of alveolar rupture into the bronchovascular sheath, from shearing force due to sudden pressure discrepancy between them, mainly in the presence of alveolar overdistension.³ Alveolar rupture may occur as a result of marked increase in intra-alveolar pressure secondary to conditions like violent cough especially with glottis closed, asthmatic paroxysms, strenuous vomiting which allows gas to leak along the bronchovascular bundle towards the mediastinum.⁴ It has also been reported in pulmonary tuberculosis, pneumonia, bronchogenic carcinoma, chronic obstructive pulmonary disease, interstitial lung disease.^{5,6} The pressure gradient between the intra-alveolar air and the contiguous blood vessels lining the alveoli favors their rupture. The air so escaped enters the perivascular sheath, courses either towards the visceral pleura or towards the lung hilum and mediastinum. If the former is ruptured a condition of homolateral pneumothorax occurs while a sequel of the dissection to the lung hilum results in mediastinal emphysema. Subcutaneous emphysema may be noted in association with pneumothorax, or pneumomediastinum.⁷ Spontaneous pneumomediastinum usually follows a benign course and management is, in most cases conservative.

Spontaneous pneumomediastinum in tuberculosis has been reported in miliary as well as nonmiliary and the cavitary forms of pulmonary lesions.⁸ Adhesions between visceral and parietal pleura are common over the areas of lung affected by tuberculosis. A tear of such pleural adhesions into subcutaneous tissue may produce emphysema giving rise to a typical crepitus.⁷ Local airway obstruction and distal airway trapping can also contribute to alveolar rupture in tuberculosis. Subcutaneous emphysema secondary to tuberculosis may develop due to associated pneumothorax, pneumomediastinum, or following the chest tube insertion.⁷ Subcutaneous emphysema without pneumothorax or pneumomediastinum is an extremely rare occurrence in cavitary pulmonary tuberculosis.² A case of pneumomediastinum with subcutaneous emphysema in a patient of silicotuberculosis was reported

where the patient had bilateral reticulonodular lung opacities with areas of confluent opacities in upper and midzones.⁸

CONCLUSION

Spontaneous pneumomediastinum in tuberculosis is a rare entity but can be seen in miliary and non miliary forms of tuberculosis due to damage to alveoli. The diagnosis of spontaneous pneumomediastinum is often challenging as chest pain and dyspnea, two of the most common presenting symptoms, have a broad differential diagnosis. Therefore, a high index of clinical suspicion of tuberculosis should always be kept in mind in a young patient of acute spontaneous pneumomediastinum without trauma.

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