

PRIMARY PULMONARY AMOEBIASIS - AN UNUSUAL CAUSE OF PULMONARY CONSOLIDATION

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Salahuddin Afsar, Ali Nawaz Choudhri (Department of Medicine, Dow Medical College and Civil Hospital, Karachi.)

Juzar Ali (Department of Medicine, Liaquat National Hospital, Karachi.)

INTRODUCTION

While colonic and hepatic amoebiasis occur commonly in tropical countries, primary pulmonary amoebiasis remains a rare condition¹⁻⁴. Pulmonary involvement, when it occurs, is usually secondary to amoebic liver abscess⁵. Primary pulmonary amoebiasis is thought to occur via haematogenous spread. We report a case of primary pulmonary amoebiasis confirmed by visualizing the trophozoites of *Entamoeba histolytica*.

CASE REPORT

A 78 years old man with a 60 pack year smoking history presented with one week history of cough productive of mucoid sputum and high grade fever. There was no history of haemoptysis or weight loss. On examination temperature was 39°C, respiratory rate 28 per minute and he was not clubbed, cyanosed or wasted. Chest examination revealed harsh vesicular breath sounds throughout with crepitations over the left fourth and fifth intercostal spaces anteriorly. On abdominal examination liver was not enlarged or tender. Rest of the examination was normal. Investigation revealed WBC 17,000/cmm (neutrophil leucocytosis). ESR 96 mm 1st hour (Westergreen) and normal liver function tests. Chest x-ray revealed a large opacity in the left mid zone with irregular margins. Sputum culture showed normal bacterial flora. Sputum was negative for AFB, fungi and malignant cells. He was treated with amoxycillin and ofloxacin for two weeks but did not improve. Repeat x-ray chest showed cavitation (Figure 1).

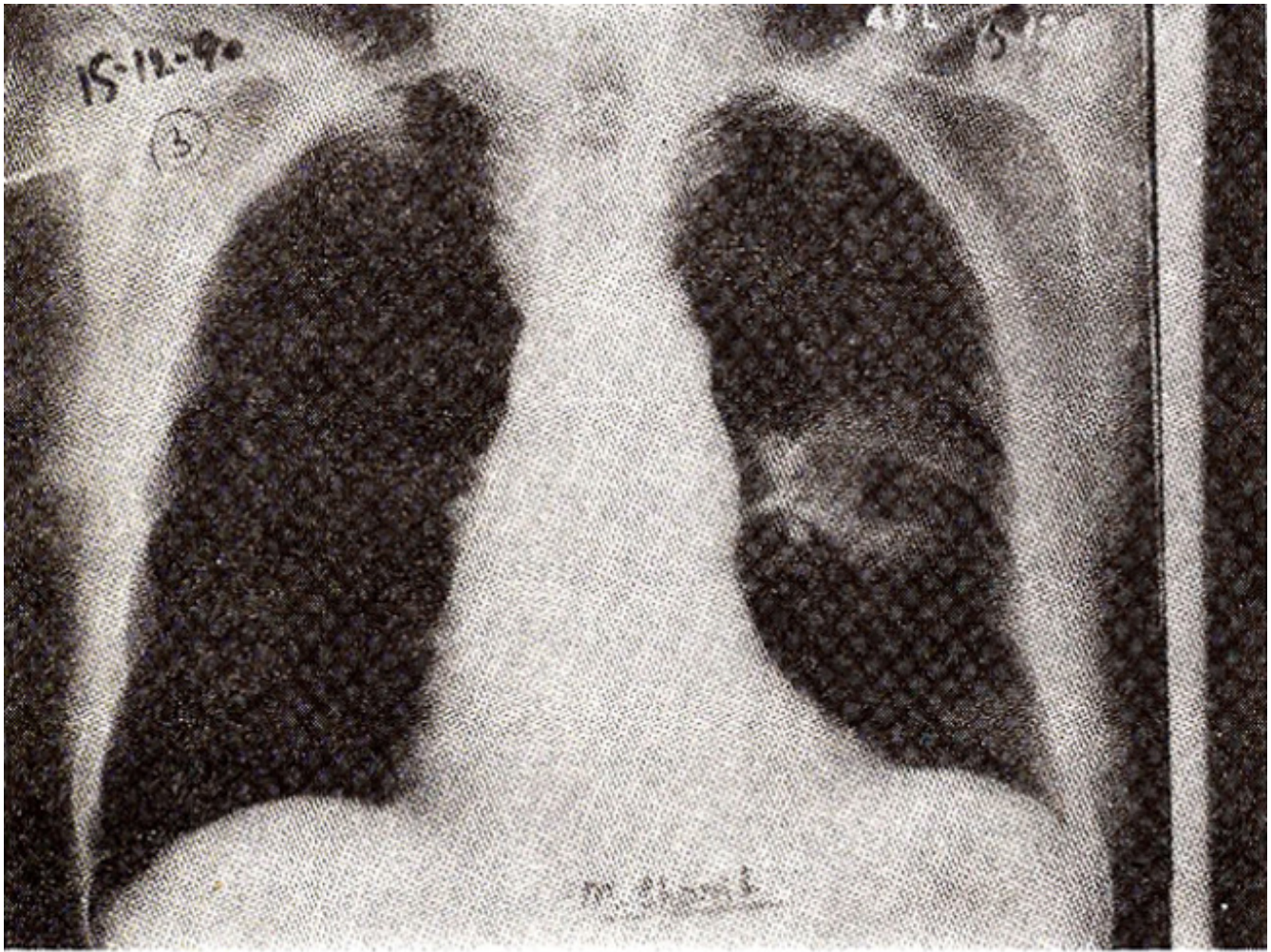


Figure 1. X-ray chest, showing cavitating lesion in the left midzone.

CT scan showed collapse with consolidation of the left upper zone peripherally and enlargement of para-oesophageal and para- tracheal lymph nodes (Figure 2).

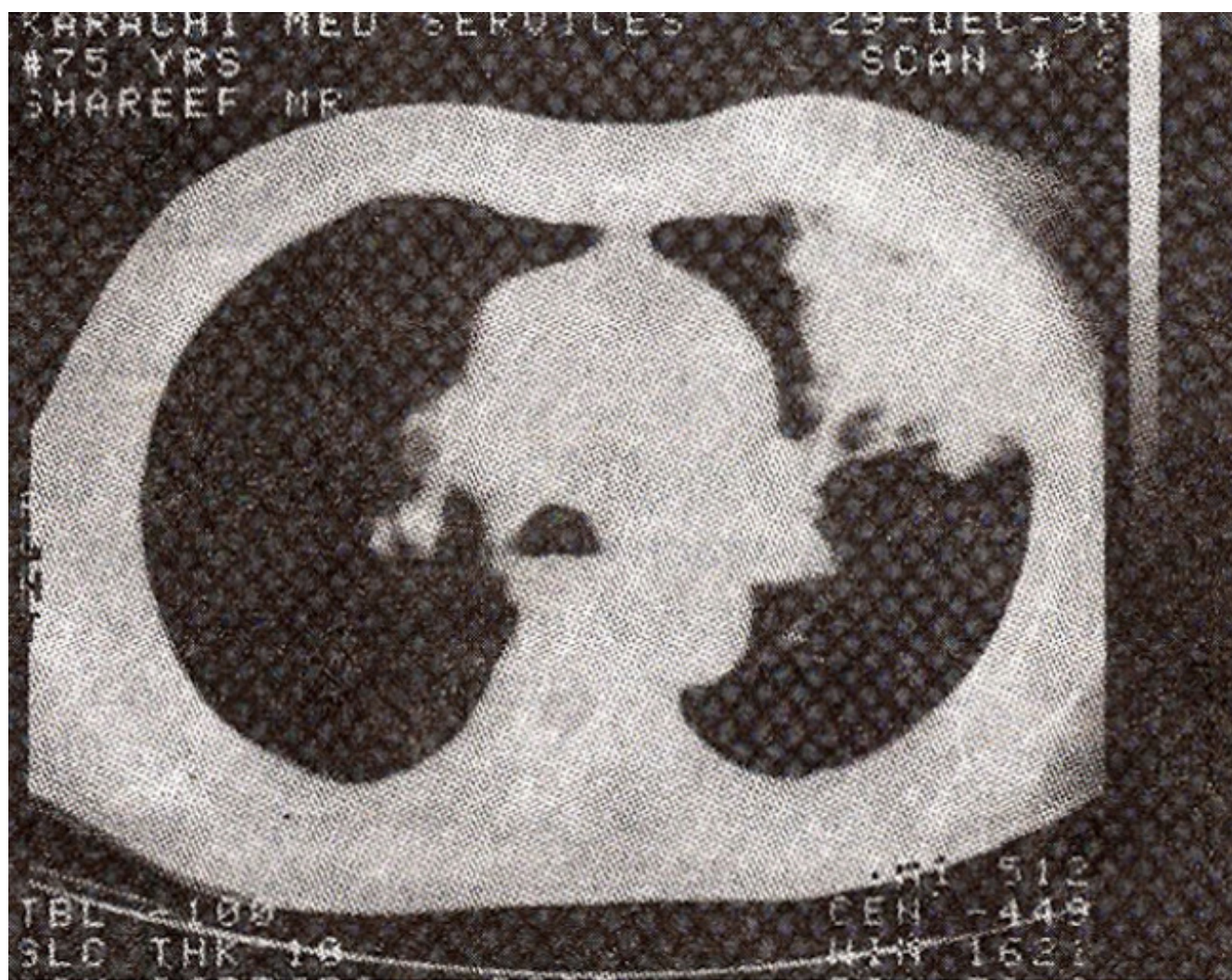


Figure 2. CT scan thorax, showing consolidation in left lung.

Bronchoscopy revealed congestion of the mucosa of left upper lobe bronchus. Bronchial washing and biopsy showed infiltration by acute and chronic inflammatory cells but were negative for AFB, malignancy and micro-organism. Fine needle aspiration under fluoroscopy, revealed trophozoites of *entamoeba histolytica* seen both in aspiration cytology and histopathology samples. Indirect haemagglutination titres for amoebiasis were positive in 1:5 12 dilution. Ultrasound of the liver subsequently performed was normal. The patient was treated with metronidazole 800 mg 8 hourly and showed complete clinical response and disappearance of the opacity on chest x-ray in three weeks time.

DISCUSSION

In tropical countries pulmonary amoebiasis usually presents as basal pneumonia or abscess secondary to direct extension from liver^{1,2,5}. Chest radiographs show absence of healthy lung tissue between the lesion and diaphragm. Various routes of spread, without involving the liver, have been suggested. The most accepted view is that the trophozoites enter through branches of middle and inferior haemorrhoidal or vertebral system of veins into the inferior vena cava and then reaching the pulmonary circulation^{1,4,5}. It may also result from inhalation of dust containing amoebic cysts¹. In primary pulmonary amoebiasis history of dysentery may be missing just as in the case of amoebic liver abscess. Cavitation on chest x-ray has also been reported in other cases of primary pulmonary amoebiasis¹. The

diagnosis of primary pulmonary amoebic lung abscess can only be made with certainty by demonstrating trophozoites of *Entamoeba histolytica* in sputum⁶ or on fine needle aspiration as in our case. However, the isolation of the organism may be difficult and indirect proof of the aetiology can be obtained by serology and dramatic therapeutic response both clinically and radiographically by metronidazole. Primary pulmonary amoebiasis forms an unusual cause of a cavitating lung lesion but should be borne in mind as resolution with treatment is very satisfactory.

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