Amoebic lung abscesses

A case report

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Summary

A case of multiple amoebic lung abscesses without indication of direct extension from a subclinical liver abscess, which delayed correct diagnosis, is reported. Severe constitutional symptoms, life-threatening haemoptysis and large pulmonary lesions were the prominent clinical manifestations. The response to metronidazole was dramatic. It is postulated that haematogenous spread was responsible. The rarity of this form of amoebiasis is evident on published reports.

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Pulmonary complications after amoebic liver abscess occur frequently. However, haematogenous spread, especially in the absence of clinical involvement of the liver, is not often seen but since appropriate treatment successfully prevents life-threatening complications its recognition is important. The prominent haemoptysis, although it may be little and have a long history, can easily be misdiagnosed as tuberculosis. Good clinical judgement is necessary since serological tests can support the diagnosis but cannot confirm it.

A case that illustrates some interesting diagnostic characteristics is presented.

Case report

A 23-year-old coloured man was referred from a country area where he had presented to hospital 9 months previously with a cough productive of small amounts of blood-streaked sputum. This was followed 2 months later by pain in both shoulders, more severe on the right. A provisional diagnosis of pulmonary tuberculosis was made on the basis of this history and radiographic evidence of a cavitating lesion in the left upper lobe (Fig. 1). Despite the fact that 4 sputum specimens, stained and cultured for acid- and alcohol-fast bacilli, were negative, treatment was started on a standard 4-drug regimen. After 6 months of compliant treatment the patient's symptoms had not improved. He had lost 15 kg in body mass, developed a grade II dyspnoea and episodes of fever, and the chest radiograph showed progression of the lesion. The patient smoked cigarettes, including cannabis, and abused alcohol. He had no contact with sheep. There was no positive history of diarrhoea or abdominal pain.

On initial examination at Tygerberg Hospital the patient was anaemic (haemoglobin 5 g/dl), had a swinging temperature and clubbing of the fingers. No lymphadenopathy was found and examination of the cardiovascular system revealed no abnormality. The respiratory system showed signs of consolidation in the left upper lung zone and bilateral scattered coarse crepitations. A 2 cm enlarged non-tender liver could be palpated.

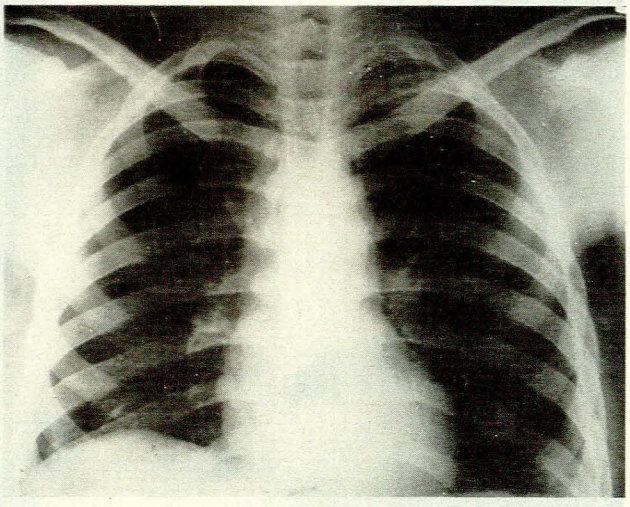

Fig. 1. Cavitating opacification in the left apex.

Laboratory investigations revealed: normochromic, normocytic anaemia with a white cell count of 21,3 x 10^9/l of which 75% were neutrophils; partial oxygen pressure 11,1 kPa; partial carbon dioxide pressure 4,4 kPa; pH 7,48; and CO_2 content 25,4; urea and electrolyte values were normal; liver function tests — serum albumin level 19 g/l, globulin 69 g/l, normal bilirubin and transaminases, alkaline phosphatase 401 U/l (normal 30-85 U/l), γ-glutamyl transferase 74 U/l (normal 0-54 U/l). Apart from many pus cells in the sputum, the cultures of both sputum and blood were negative. Radiography of the chest showed large cavitating opacifications in both upper lung zones and also a well-demarcated round lesion in the right middle lobe and an ill-defined streaky opacification in the left lower lung zone (Fig. 2).

The patient received 4 U packed red cells and was treated as for a lung abscess with gentamicin and high doses of penicillin with no response. On day 4 he developed a life-threatening haemoptysis, which necessitated another 2 U blood. Bronchoscopy revealed haemorrhage from the left upper lobe. Chest radiographs proved that the mass lesions had enlarged and were less well demarcated (Fig. 3). Computed tomography demonstrated that the masses had the density of soft tissue and a percutaneous needle aspiration and biopsy were done. Cytological and histological examination of these specimens showed only inflammatory cells and no malignant cells. The white blood cell count rose to 28,9 x 10^9/l and the differential count indicated neutrophilia and toxic granulation.

Repeated cultures were negative and the fever was unremitting. Despite the radiographic picture, which showed marked deterioration, the blood gases remained nearly normal. Abdominal ultrasonography on day 7 showed a normal size liver with a 7 x 7 cm cystic lesion in the right lobe just anterior and to the right of the inferior vena cava. This strongly suggested

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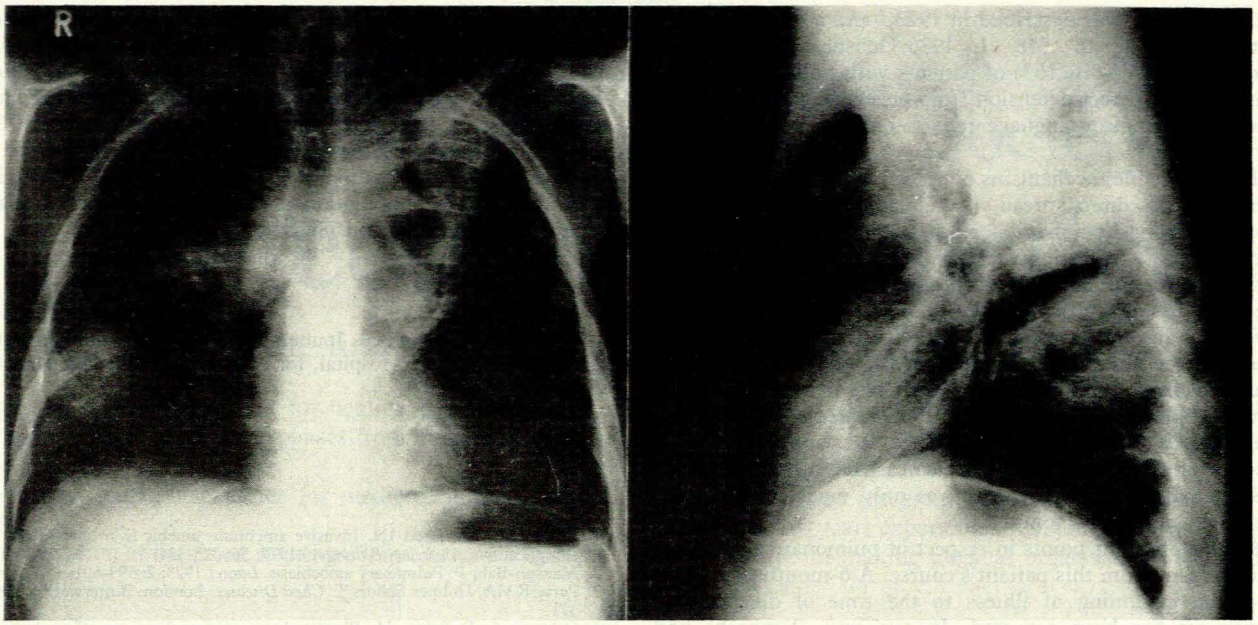

Fig. 2. After 6 months' treatment for tuberculosis 3 cavitating opacifications are apparent.

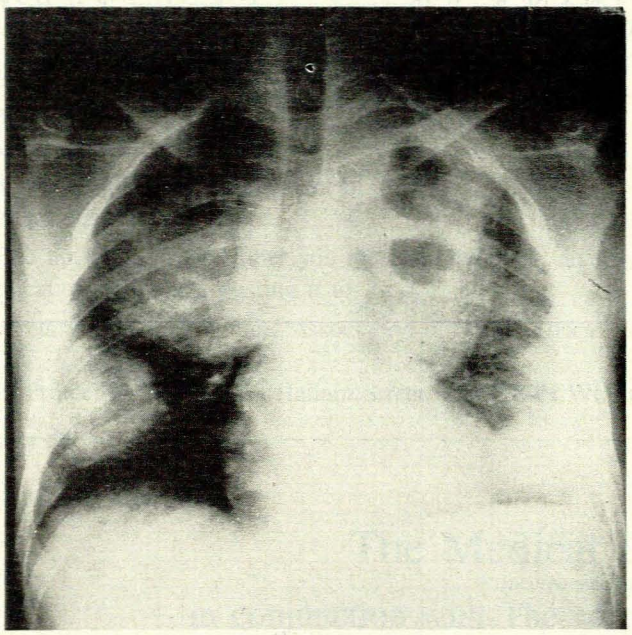

Fig. 3. Extension of lesions after massive haemoptysis.

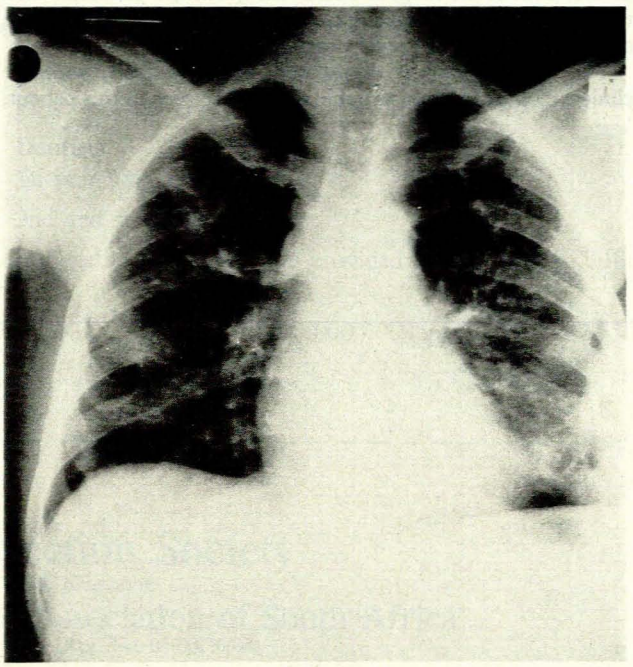

Fig. 4. Three remaining cavities after 10 days' treatment with metronidazole.

an amoebic abscess and was supported by a very high amoebic indirect haemagglutination titre of 1:1 052. Metronidazole 800 mg 8-hourly was administered orally and treatment continued with broad-spectrum antibiotics (amikacin and vancomycin). The response was dramatic. The temperature normalised after 36 hours and within days all signs of toxicity had cleared. The haemoptysis, which had changed from frank blood to a more anchovy-like colour before the metronidazole administration, diminished rapidly, with a complete cessation of all sputum production within 10 days. The radiographic response was equally impressive (Fig. 4).

In spite of the absence of vegetative forms of *Entamoeba histolytica* in the sputum or in the lung aspirate, the diagnosis of amoebiasis was made on the clinical presentation, the very high titre of amoebic indirect haemagglutination test and the dramatic response to metronidazole.

Discussion

Lung involvement in amoebiasis is not uncommon. According to published reports this occurs in 6,2% of cases.[1] In a review article by Adams and MacLeod[1] the following are described: (*i*) pleural effusion and empyema (29%); (*ii*) hepatobronchial fistula (47%); (*iii*) lung abscess by direct spread (14%); and (*iv*) consolidation (10%). A lung abscess, not in direct proximity to

the diaphragm, was described in 1923,[2] and a further 5 cases were reported up to 1948.[3] In 1936 Ochsner and DeBakey[4] found that 10,5% (16/153) of patients with pulmonary amoebiasis had no direct extension from an existing liver abscess. Thereafter, English-language reports only mention that it is very rare.

Three possible mechanisms of spread to the lung are postulated, of which direct spread via the diaphragm is certainly the most common. Lymphatic spread is an alternative,[5] whereas haematogenous spread, which we believe took place in our patient, is rare.[1,6] Factors which support this conclusion are the close proximity of the liver abscess to the inferior vena cava, the absence of any pleural effusion throughout the course of the illness and the fact that the lung abscesses were multiple, bilateral and that none of them was in direct proximity to the diaphragm. A similar hypothesis for a single amoebic lung abscess, which occurred in the left upper lobe, was described by Blyth and Pirie.[7] In that case no liver involvement was evident and the diagnosis was only made on histology after a left pneumonectomy.

A few important points in respect of pulmonary amoebiasis were evident from this patient's course. A 6-month time lapse from the beginning of illness to the time of diagnosis of amoebiasis has been reported. In our case, 9 months had elapsed. The near normal blood gases, reflecting good gas exchange, in the presence of severe clinical and radiographic involvement of the lung, as well as the quick and uncomplicated recovery on metronidazole illustrates the non-destructive nature of this disease and the good prognosis.

The initial diagnosis of pulmonary tuberculosis, with the clinical picture and radiographic appearance seen in this patient, is certainly not strange. The differential diagnosis included lymphoma, lung abscesses, other primary or secondary malignant lesions, sarcoidosis and *Echinococcus* cysts. This case illustrates, however, that other infective causes for cavitating lung lesions must be considered and that amoebiasis is one. The reported sensitivity of the indirect haemagglutination test in invasive amoebiasis is 95 - 100%,[8,9] but the specificity varies depending on the population and may only be 50%,[8] again depending on the titre considered positive.

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