Intrathoracic complications of amoebic liver abscess[1]

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Summary: Amoebiasis is a world-wide parasitic infestation but is more prevalent and virulent in warm countries. Of 33 patients with amoebic liver abscess (ALA) seen over a 5-year period, 24 had intrathoracic complications. Management consisted mainly of the administration of the antiamoebic agent metronidazole (Flagyl), repeated needle aspiration of ALA and surgical drainage when aspiration failed. Closed tube thoracostomy was the preferred management of amoebic empyema. Associated cerebral involvement in 4 cases improved with parenteral metronidazole. Pericardial amoebic abscess claimed 100% mortality in our series.

Introduction

Infection with *Entamoeba histolytica* occurs world-wide but is most prevalent in warm climates, especially in the tropics and subtropics. In its usual form, amoebiasis is a common cause of recurrent diarrhoea, bloody mucoid stool and infrequently liver abscess. It occasionally presents as hepatopulmonary fistula, empyema or cardiac tamponade when it involves the cardiopulmonary systems. The present study reviews 24 intrathoracic as well as cerebral complications of amoebic liver abscess.

Methods

From 1978 to 1982, 33 cases with amoebic liver abscess (ALA) were treated at Ife University Teaching Hospitals Complex, Ile-Ife, Nigeria. Twenty-four of these had intrathoracic complications, of which 4 had concurrent associated central nervous system manifestations. Criteria for diagnosis included combinations of all or some of the following: (1) good history and physical examination as to previous or concurrent diagnosis of amoebiasis; (2) chest and abdominal X-rays showing elevated cupola of diaphragm (Figure 1), pleural effusion and massive hepatomegaly (Figure 2); (3) expectoration and or aspiration of anchovy-sauce fluid from both the involved pleural space and the liver; (4) recovery of trophozoites or cysts of *Entamoeba histolytica* from stools or aspirates of affected patients; and (5) marked improvement of patients on intravenous or oral metronidazole.

Results

The ages of the patients ranged from 9 to 53 years, the peak incidence occurring in the age group 19–45 years. Only one patient presented before the age of 19 years. There were 21 males and 3 females. Seven patients (29%) had moderate to severe elevation of the cupola of the right diaphragm; 7 (29%) had right-sided pleural empyema; 5 (20.5%) had serous pleural effusion; 3 (12.5%) had hepatopulmonary fistula due to ALA rupture into the right pulmonary parenchyma; and amoebic pyopericardium resulting in fatal pericardial tamponade occurred in 2 patients (8.3%).

The clinical presentation was variable (Table 1). Nineteen cases presented with varying respiratory complaints including chest pain, dyspnoea and cough. Three of these cases expectorated anchovy-sauce material with an associated haemoptysis; 2 of them had an uneventful recovery whilst the third had a fatal haemoptysis. The predominant clinical

[1]Accepted 10 June 1983

0141–0768/84/010017–05/$01.00/0

Table 1. Clinical features of 24 cases with intrathoracic complications of amoebic liver abscess

	No. of cases		No. of cases
Fever	21	Pyrexia	21
Recent or previous history of diarrhoea with bloody mucoid stool	17	Abnormal chest findings	19
Right upper abdominal discomfort	14	Tender swelling of intercostal spaces and upper abdominal wall	7
Dyspnoea, cough and chest pain	12	Hyperreflexia with varying features of	4
Confusion	4	meningeal irritation (confusion and coma)	
Haemoptysis	3	Pedal-oedema	3
Lethargy, coma	2	Jaundice	2
Hepatomegaly	21	Prolapsed haemorrhoid	1

features in those with pleuropulmonary complications included a previous history of diarrhoea and associated bloody mucoid stool, fever, cough, and dyspnoea of varying intensity according to the severity of the predisposing empyema; right-sided chest pain; epigastric fullness; warm, tender and bulging intercostal spaces and right upper quadrant fullness with massive tender hepatomegaly. Haemoptysis featured in only the 3 cases with hepatopulmonary fistula, and only 2 patients developed deep jaundice. Four cases (16.7%) had concurrent central nervous system involvement; the dominant presenting features were hyperreflexia, confusion, lethargy and coma.

The parasitologic study consisted of identification of amoebic cysts or trophozoites in the stools and aspirates of the patients. Amoebic cysts were identified in the stools of 24 patients, whilst trophozoites were recovered in the watery-mucoid stools of 6 patients who had concurrent diarrhoea. Sputum specimens from 2 of the 3 patients who had hepatopulmonary fistula yielded trophozoites. In 11 cases amoebic trophozoites were

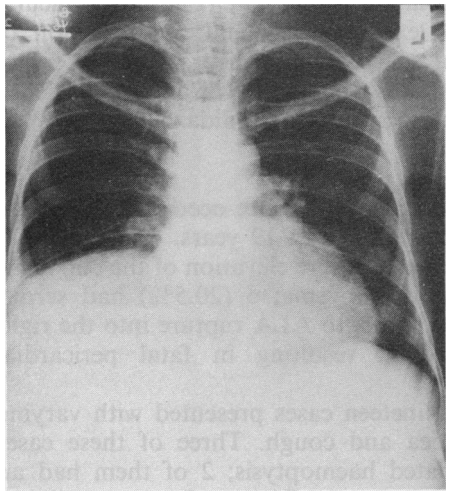

Figure 1. Posteroanterior chest X-ray showing marked elevation of right hemidiaphragm

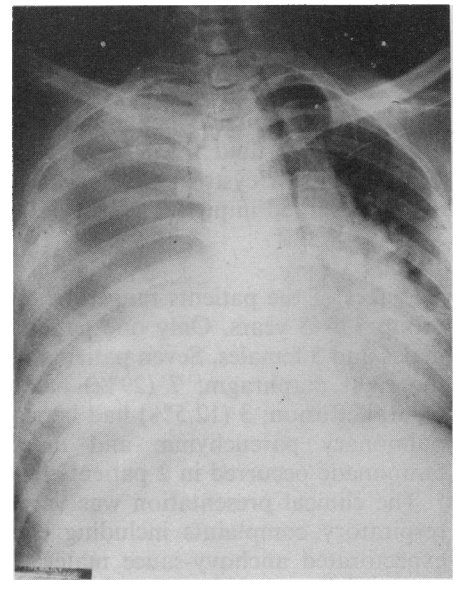

Figure 2. Posteroanterior chest X-ray showing amoebic pleural empyema in continuity with fluid space under right hemidiaphragm

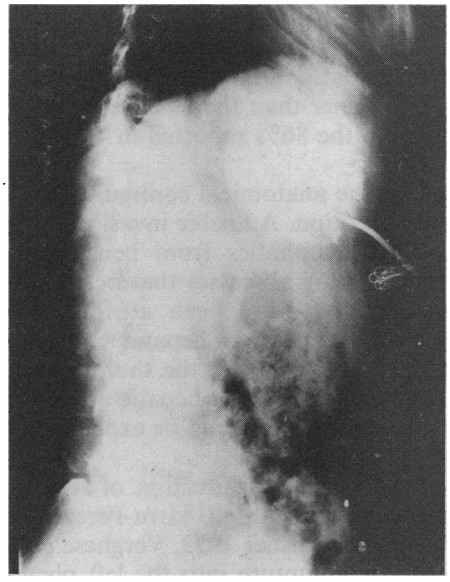

Figure 3. Intracavity barium sulphate study of abscess cavity

identified in the pus aspirated from the liver and the resultant amoebic empyema thoracis. There was no bacterial growth in the remaining anchovy-sauce material nor in the serous effusion, in which no trophozoites were identified. In all, 28 of the 33 patients had cysts or trophozoites or both in their stools or aspirates.

The radiological findings included moderate to marked elevation of the cupola of the right diaphragm, atelectasis with pneumonitis and massive pleural effusion (Figures 1 and 2). In some of the patients who had simultaneous drainage of the liver abscess extraperitoneally, dye contrast studies of the abscess cavity were carried out (Figure 3) using the modified technique of Harding *et al.* (1970).

Management consisted mainly of the administration of the antiamoebic agent metronidazole (Cameron 1978), repeated needle aspiration of ALA and surgical drainage. Metronidazole 500 mg in 100 ml was given intravenously every 6 hours for the first few days, and then orally, 200–400 mg thrice daily, for an average of 4–5 weeks. Concurrent surgical drainage was carried out when repeated aspiration failed. The point of surgical drainage was at times over the point of maximal tenderness and occasionally in an area of fluctuation when present. In all cases a self-retaining Foley catheter was inserted into the abscess cavity for continuous drainage (Figure 3). Three patients had posterior extraperitoneal drainage of the liver abscess because the site of maximal induration and tenderness was located posterolaterally. All the cases with amoebic empyema thoracis had closed tube thoracostomy for continuous drainage with periodic pleural instillation of parenteral metronidazole and intravenous administration of metronidazole plus 4–6 g daily of Ampiclox (ampicillin + cloxacillin).

Patients with serous pleural effusion and pneumonitis improved on repeated thoracentesis and chemotherapy (Ampiclox and metronidazole). Patients with hepatopulmonary fistula benefited from parenteral metronidazole 500 mg intravenously every six hours for an average of 8 days, and parenteral Ampiclox (1 g every 4 hours) to combat concurrent bacterial invasion. Oral metronidazole was then given daily for an average of 5 weeks. These patients all had postural drainage and repeated bronchoscopy for bronchial toilet. The 2 cases with amoebic pericarditis had diagnostic pericardiocentesis during resuscitation. The 4 patients with concurrent cerebral manifestations had a gradual resolution of their clinical features within 24–48 hours of parenteral administration of metronidazole.

Discussion

Of the 33 patients with amoebic liver abscess, 24 (73%) developed intrathoracic (pleuropulmonary cum pericardial) complications of amoebiasis, and 4 of these also had cerebral amoebiasis. This rate of complication of ALA is higher than the 20% reported by Debakey & Ochsner (1951) from the USA, but is similar to the 86% reported in Nigerians by Nwafo & Egbue (1981).

Rupture of ALA into the pleural cavity is facilitated by the anatomical contiguity of the liver to the thoracic cavity, the diaphragm being the only partition. Amoebic invasion of the thorax has also been reported to occur by way of the lymphatics from beneath the diaphragm (Takaro & Bond 1958). The occasional amoebic lung abscesses that occur with or without associated demonstrable hepatic amoebic abscess have been attributed to embolization from a diseased liver or colon via the portal system or hepatic veins, the valveless paravertebral veins of Batson, inferior vena cava, and through the thoracic duct and subclavian vein (Takaro & Bond 1958). We were able to demonstrate hepatic abscess in all our cases. Cerebral involvement in the 4 cases in the present series could be explained by the blood-borne embolization theory (Takaro & Bond 1958).

The right lobe of the liver is the commonest site of ALA and the direction of rupture, according to most reported series, is into the right pleural space or lung (Ibarra-Perez *et al.* 1972, Lamont & Pooler 1958, Ragheb *et al.* 1976, Ribaudo & Ochsner 1973, Verghese *et al.* 1979). Similarly, an abscess in the left lobe of the liver could rupture into the left pleural space, lung or the pericardial sac (Ibarra-Perez *et al.* 1972, Lamont & Pooler 1958, Macleod *et al.* 1966). In our series, 22 (66.6%) of the 33 cases had right hepatic involvement, while in 2 cases left hepatic abscess was indicated. The symptomatology in this series, as in other reported studies, was a function of the site of pathology, pulmonary symptoms dominating in those with pleuropulmonary complications and cardiac symptoms manifesting when ALA ruptured into the pericardium.

The 7 patients who had amoebic empyema thoracis presented with toxic features, cough, chest pain and marked dyspnoea. Expectoration of anchovy-sauce (reddish-brown) material in 3 cases was pathognomonic of hepatopulmonary fistula. Haemoptysis of varying intensity was another prominent feature in these patients. Diagnosis of amoebiasis was made only when trophozoites or cysts of amoebae were demonstrated in the stool, in the aspirate or on biopsy of the abscess wall, and when the aspirated material contained no bacteria on culture. The frequency of identification on this basis has varied from 13% (Ribaudo & Ochsner 1973) to 80% (Harding *et al.* 1970). Eggleston *et al.* (1978) recorded positive results in 39%. In our series, 28 (85%) of 33 patients had cysts or trophozoites of amoeba isolated from either their stools or aspirates. Ragheb *et al.* (1976), on the other hand, have argued that the diagnosis could be made when clinical features of ALA are present in association with a sterile aspirate. The serologic tests used in the diagnosis of amoebiasis are indirect haemagglutination, cellulose acetate membrane precipitin, agar-gel precipitation and counter-current electrophoresis, but we have no facilities for these tests.

Radiography plays a major role in the diagnosis of intrathoracic amoebiasis (Harding *et al.* 1970, Macleod *et al.* 1966, Rasaretnam *et al.* 1974, Ribaudo & Ochsner 1973). In 29% of our cases there were varying degrees of diaphragmatic elevation (Figure 1), 29% had massive amoebic empyema thoracis (Figure 2) and 20% had blunting of the right costophrenic angle and serous pleural effusion. Ribaudo & Ochsner (1973) from the USA reported diaphragmatic elevation in 71% of their series of 21 cases, while Nwafo & Egbue (1981) from Nigeria reported a 31.8% incidence. The lower rate of diaphragmatic elevation in the latter and our own report from Nigeria is probably due to the fact that most of the abscesses had ruptured into the pleural cavity before presentation at hospital. Intracavity contrast studies were carried out to determine the size of the ALA cavity and also to follow its progressive healing (Figure 3), as previously described by Harding *et al.* (1970). The 2 cases with pericardial amoebiasis discovered at autopsy died from amoebic pericarditis due to ALA in the left lobe of the liver presenting with marked toxicity and acute cardiac tamponade. According to Takaro & Bond (1958) the clinical picture may be variable, but

toxic signs and varying features of acute or chronic tamponade are dominant. X-rays may reveal a large cardiac shadow, left pleural effusion and left diaphragmatic elevation. The hallmark of diagnosis in this situation is the isolation of *E. histolytica* in anchovy-sauce pus aspirated from the pericardium (Macleod *et al.* 1966, Takaro & Bond 1958).

Four of the 24 patients with intrathoracic amoebiasis had concurrent cerebral amoebiasis. They were toxic, drowsy, delirious or comatose but all improved with intravenous metronidazole. Cerebral amoebiasis usually follows hepatic or pulmonary lesions and is invariably due to amoebic embolization from diseased colon (Takaro & Bond 1958). The clinical features of these 4 patients could not have been due to toxaemia because they were not severely dehydrated and had only moderate temperature elevation compared with the other patients. In addition, their blood cultures were negative for bacterial growth, blood film showed no malarial parasitaemia and the Widal agglutination titre was not increased to incriminate *Salmonella typhi* infection, which may have a similar presentation in our environment.

Management of these complications of ALA consisted in most cases of the administration of intravenous fluids and parenteral metronidazole (Cameron 1978), followed by surgical drainage as soon as the patient was fit for surgery. Metronidazole is active against the hepatic and intestinal phase of amoebiasis. The parenteral form of the drug has been found to be very useful in our hands in the treatment of pleuropulmonary and hepatic amoebiasis. We do not recommend the use of emetine hydrochloride because of its myocardial toxicity. Only one of the 3 patients who developed hepatopulmonary fistula had fatal haemoptysis, whilst the 2 cases with suppurative amoebic pericarditis were only discovered at autopsy. This finding is in agreement with the earlier report of Debakey & Ochsner (1951) that pleuropulmonary amoebiasis is associated with a low mortality; but the mortality rate when amoebic liver abscess ruptures into the pericardium has been reported at between 63 and 96% (Eggleston *et al.* 1978, Ibarra-Perez *et al.* 1972, Takaro & Bond 1958).

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