Case Report

PLEURO-PULMONARY LESIONS IN AMEBIC LIVER ABSCESS

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Abstract

Two cases of amebic liver abscess with unusual features are described. Both the cases at the initial stage of the illness had presented as pyrexia of unknown origin with right basal patchy consolidation followed by pleural effusion. In both the cases pleural effusion was creamy colour and sterile. Case-1 had massive total empyema due to rupture of the abscess in the pleural cavity and at the later stage of the disease the creamy coloured pleural pus changed to anchovy Sauce a classical feature of liver abscess. Case-2 showed signs and symptoms of liver involvement apart from signs at the right base of the lung and pleural effusion. Both the cases responded remarkably to antiamebic treatment. Serial liver scanning in Case-2 revealed that the unruptured liver abscess took ten months to resolve completely as revealed by gradual lessening of the cold area on liver scan and persistent elevated right diaphragm which descended to its original level and coincided with the disappearance of the cold area in the upper part of the right lobe of the liver on radioisotope study.

Introduction

Pleuro-Pulmonary lesions are often seen in amebic liver abscess. These are: right basal pleurisy with or without effusion or rupture of the liver abscess—in the right pleural cavity giving rise to sterile empyema and pulmonary crepitations with signs of consolidation at the right base. Amebic liver abscess is endemic in many tropical countries and also parts of southern Africa such as Durban (Wilmot 1962). It is sporadically seen in Pakistan. Khan and Khakwani (1977) reported one case of pleural effusion in a series of 36 cases of amebic liver abscess and in another series of 51 amebic liver abscess, reported by Haider and associates (1974) one case had pleural rupture.

Recently Lamont and Wicks (1976) reported 88 cases of amebic liver abscess from Rhodesia, 13 cases had pleural effusion with one massive pleural effusion and 19 cases had signs of pulmonary consolidation of the right base. Four cases had pleural rupture.

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Patients

In the present communication two cases of amebic liver abscess are described. Both the cases initially presented with involvement of the right base of the lung. One developed massive empyema due to rupture of the abscess in the pleural cavity. Another case showed signs of pleural effusion. These cases, at the initial stage, showed signs and symptoms of inflammatory lesion at the right base of the lung without any clinical signs and symptoms of the liver abscess. Both the cases responded excellently and promptly to metronidazole and dehydroemetine combined therapy, inspite of initiation of delayed treatment in Case-1, after onset of the liver abscess and its rupture in the pleural cavity.

Case-1

Female aged 25 was seen at the PSPC Medical Centre on 8-4-1974 with complaints of sharp pain at the right lower chest with pyrexia and flu like illness since past few days. The onset was sudden.

On examination there were signs of consolidation at the right base of the lung confirmed by x-ray chest (Fig 1). Temperature was 102° F. Full blood count, sputum, urine and other systemic examination revealed no significant abnormality. As there was no leucocytosis therefore it was considered that the illness was of viral origin (Viral Pneumonia). She was put on Oxytetracycline 250 mg every six hourly with supportive vitamines and analgesic. She defaulted for 2 weeks and was having treatment by private practitioner. She reappeared on 23-4-1974, re-examination revealed that now she was more breathless at rest with toxic symptoms. Pulse rate was 110/minute and temperature 102° F. Respiration rate was 50/minute with shallow breathing. Examination of the chest revealed dullness on whole of the right chest on percussion and absent breath sound on auscultation with clinical evidence of mediastinal shift to the left suggestive of massive pleural effusion. X-Ray Chest (Fig-2) revealed massive pleural effusion, with mediastinal shift to the left and there was no evidence of any active lesion in the left lung.

Blood Count: WBC: 5900/Cu.mm, Poly morphs 71% Lymphocytes 23%, Eosinophil: 4%, ESR 35/mm/1st hour.

Diagnostic aspiration revealed creamy coloured purulent fluid which was sent to the laboratory for bacteriological and cytological examination. During investigation she defaulted again and was admitted to other private hospital, where she treated as tubercular empyema because the pus was sterile and it was of creamy colour.

Fig. 1: Case-1 (8-4-1974). X-ray chest P.A. view showing patchy consolidation at right base of the lung.

Fig. 2: Case-1 (25-4-1978). X-Ray chest P.A. view showing massive effusion (empyema) on the right with mediastinal shift to the left.

In the private hospital her empyema was drained repeatedly and was put on oral rifampicin/300 mg+INH 300 mg daily with vitamines. On 3-6-1974 culture revealed no evidence of tuberculous infection. Antituberculous treatment was discontinued and empyema was continuously drained under water seal drainage tube, pending decortication of the lung as no aetiology was established.

She reappeared on 18-6-1974 with an under-water drainage tube in a bottle for second opinion and treatment at this Centre. X-Ray chest (Fig-3) revealed lessened empyema and other fields of both the lungs, except right basal empyema, were clear. Now the pus was of anchovy Sauce colour, a characteristic of amebic abscess clinically. She was still pyrexial, weak and emaciated. She was diagnosed as ruptured amebic liver abscess in the pleural cavity and was put on metronidazole 400 mg t.d.s. for 10 days, dehydroemetine 60 mg I.M. daily for 10 days and tetracycline 250 mg every 6 hourly. She responded dramatically within three days, purulent discharge via drainage tube ceased completely. The drainage tube was removed on 4-7-1974, review examination showed normal temperature and remarkable clinical improvement. X-Ray chest (Fig-4) showed residual signs of subacute pleuritis as the chest aspiration was dry and right diaphragm was not raised. Review examination on 5-10-1974 showed normal x-ray chest (Fig-5) and without any complaint.

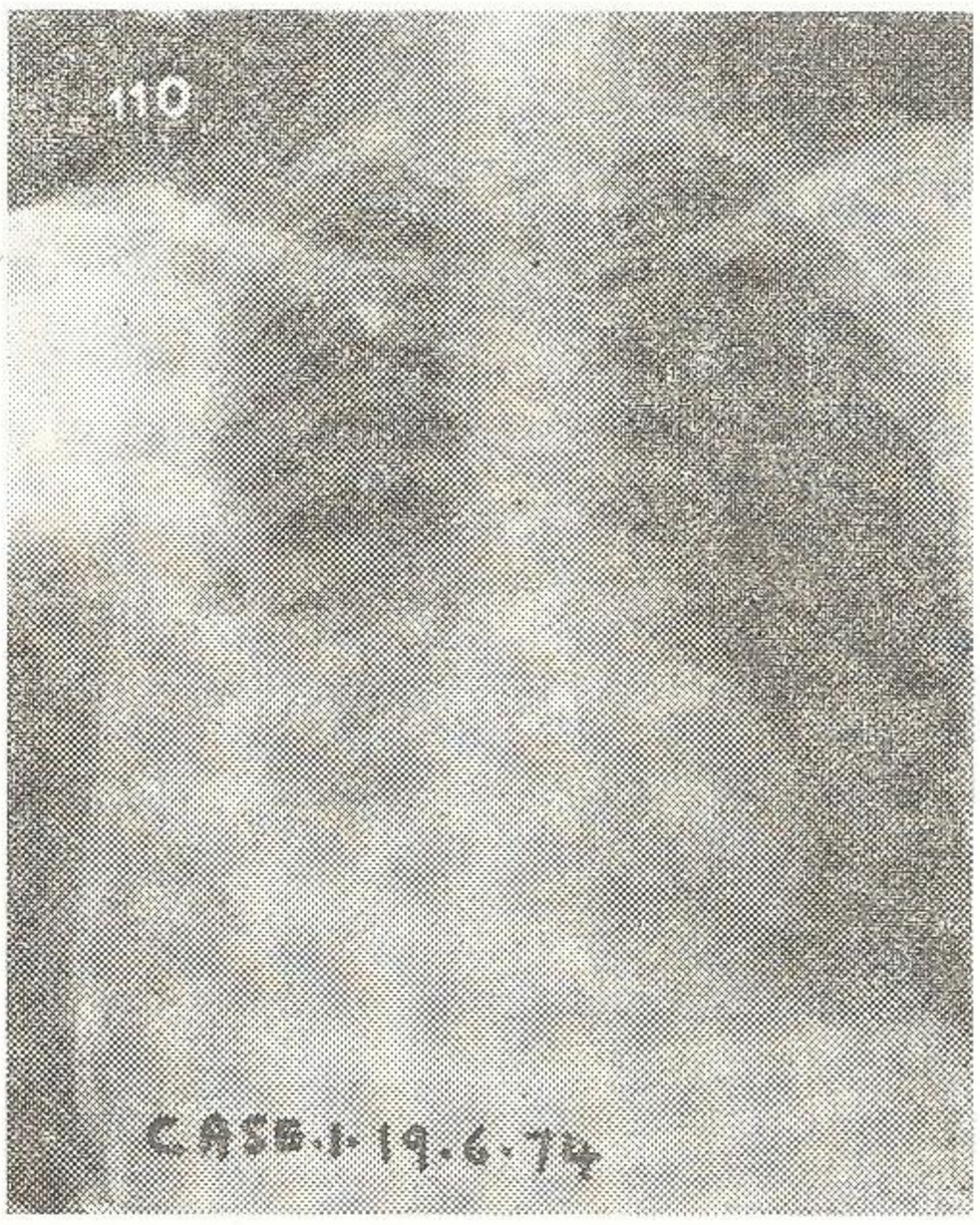

Fig. 3: Case-1 (19-6-1978). X-Ray chest after drainage of empyema with drainage tube. The empyema is leassened with localised abscesses at the drainage points.

Case-2

Male Pharmacist of this Centre aged 30 years was seen at this Centre on 21-6-1976 with a complaint of pain at the right base of the chest and intermittent pyrexia of unknown origin. On examination he was running a temperature of 100° F. Systemic examination revealed no signi-

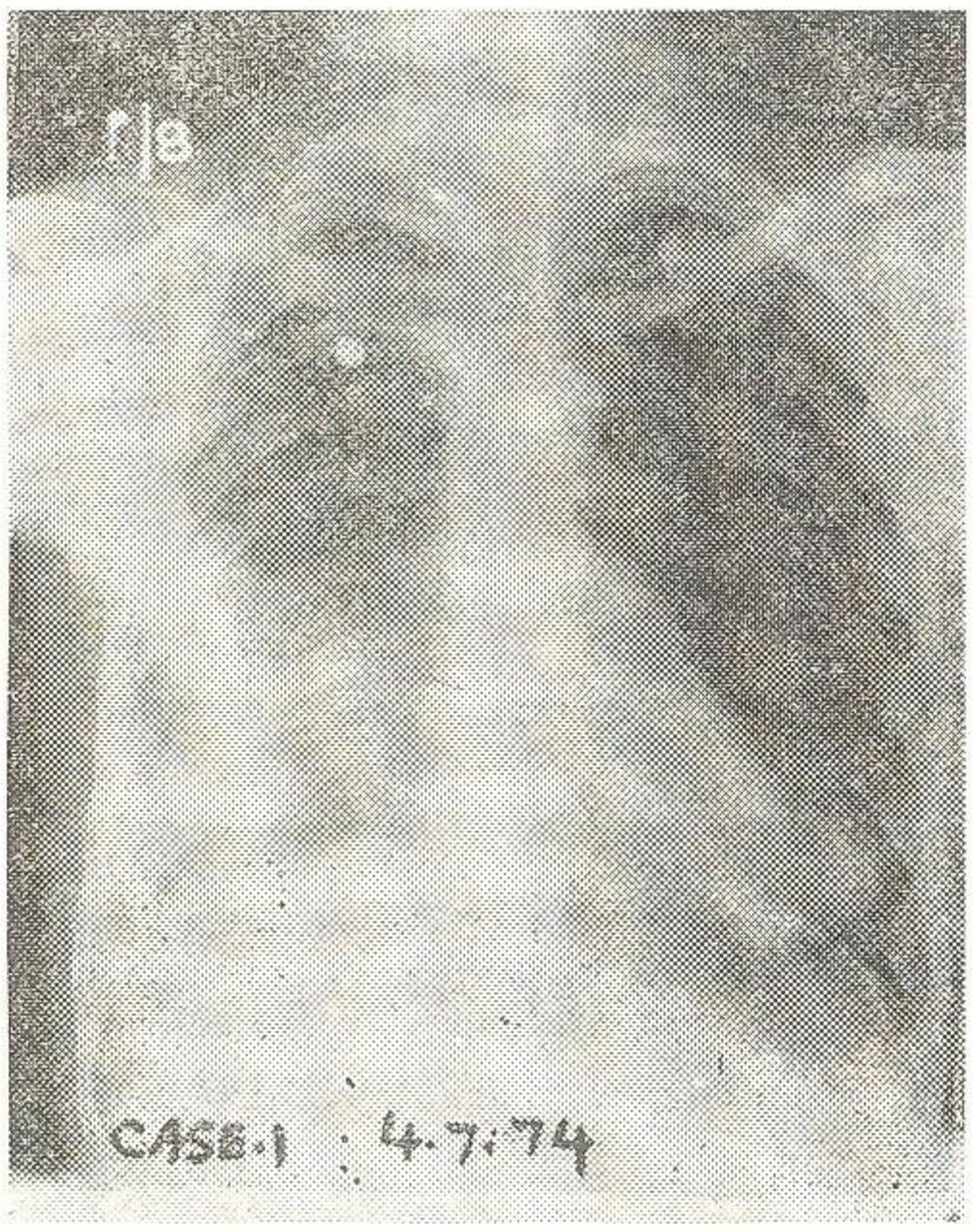

Fig. 4: Case 1 (4-7-1974). X-Ray chest after antiamebic treatment and a dry tap with clearance of the empyema and residual pleural reaction.

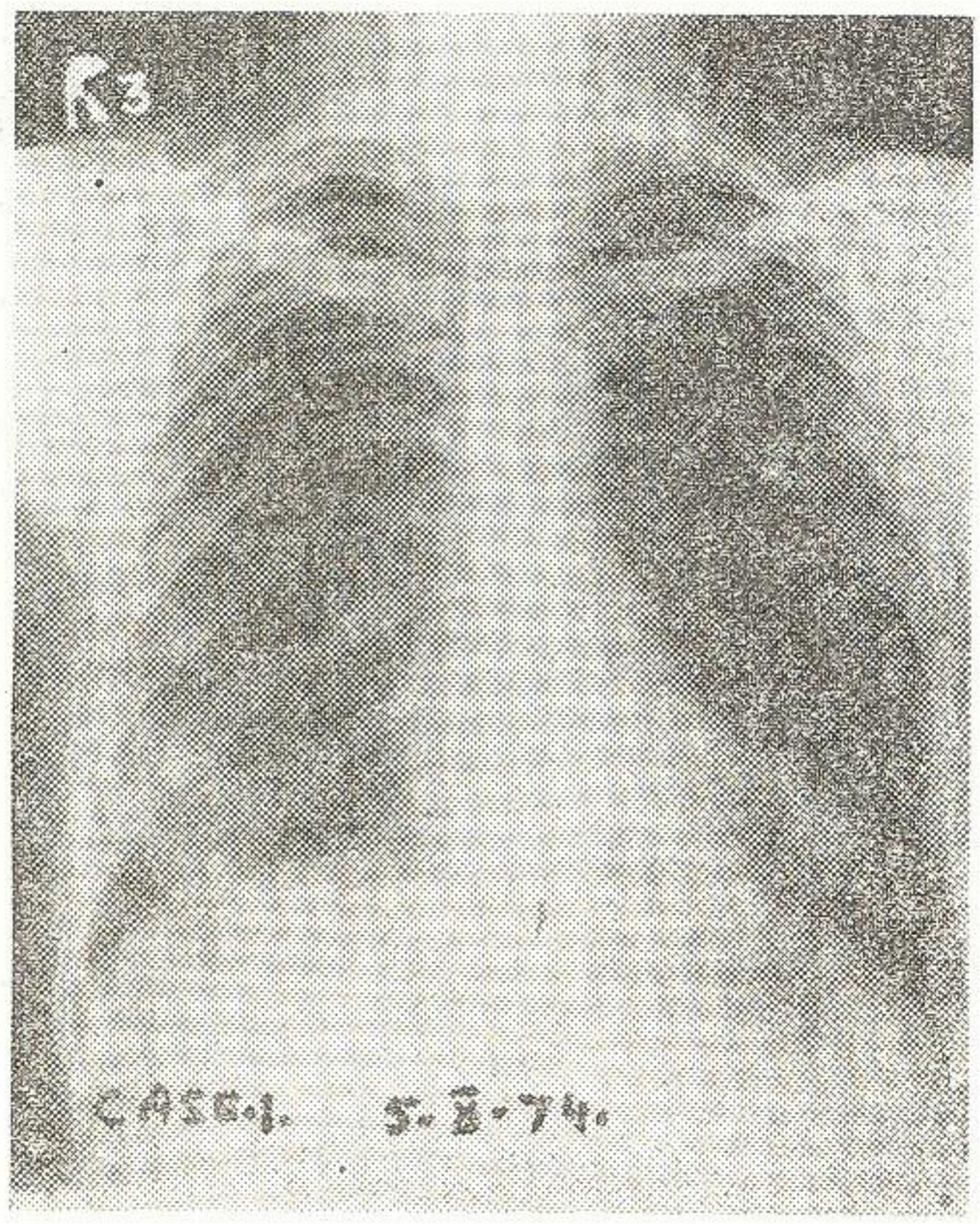

Fig. 5: Case 1 (5-10-1974). X-Ray chest showing complete clearance of the amebic empyema without any residual signs. The right diaphragm is at normal level.

ficant abnormality. Blood counts, x-ray chest (Fig-3), stool and urine were within normal limits. He was first considered to be suffering from some viral illness and was prescribed oxytetracycline capsules 250 mg 6 hourly.

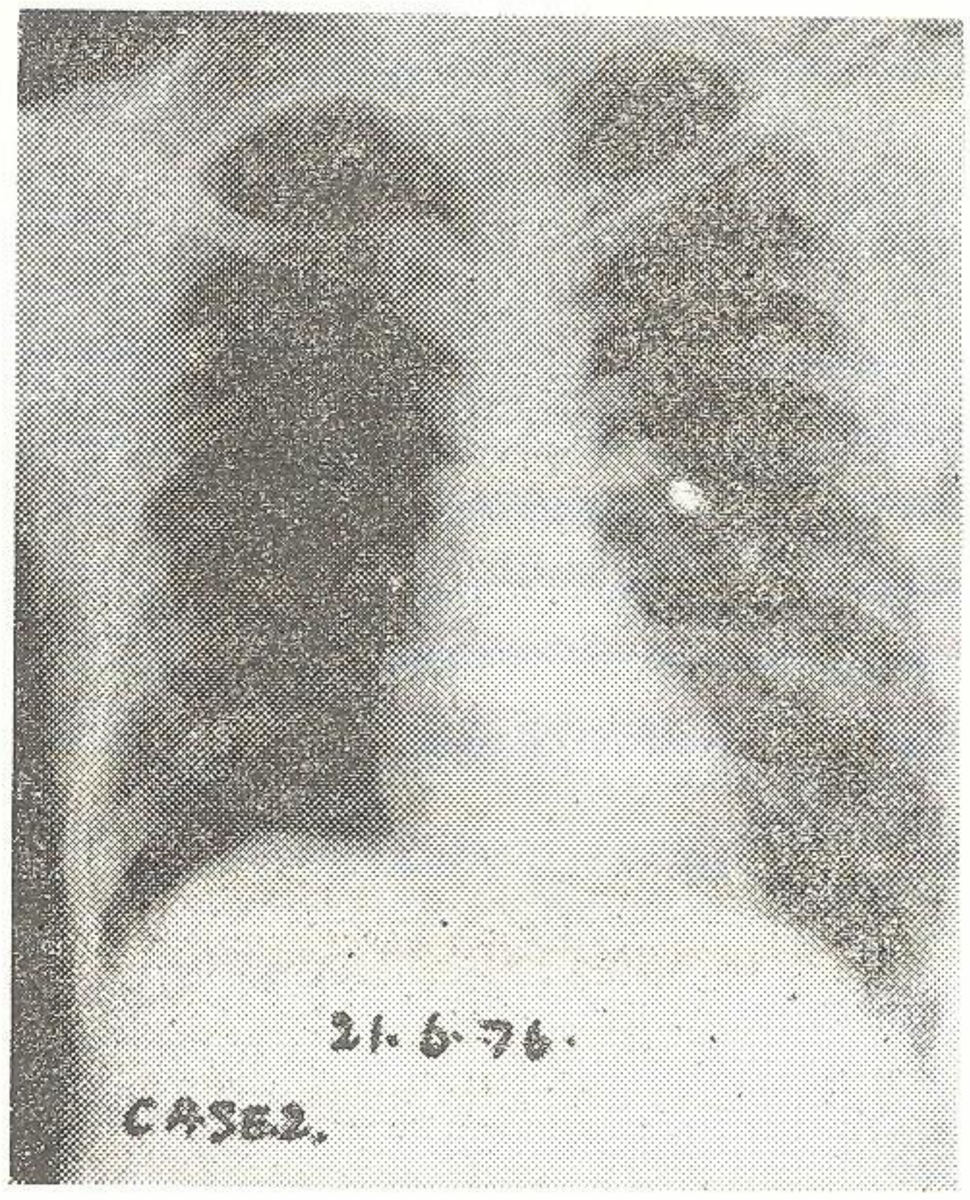

Fig. 6: Case-2 (21-6-1976). X-Ray chest P.A. view showing no significant abnormality with minor tenting of right diaphragm.

On 29-6-1978 he complained sharp pain on the right lower chest on deep breathing. X-Ray chest revealed right basal pleural effusion (Fig-7). Chest aspiration showed light creamy coloured fluid. Bacteriology of the fluid showed no growth of any organism on culture.

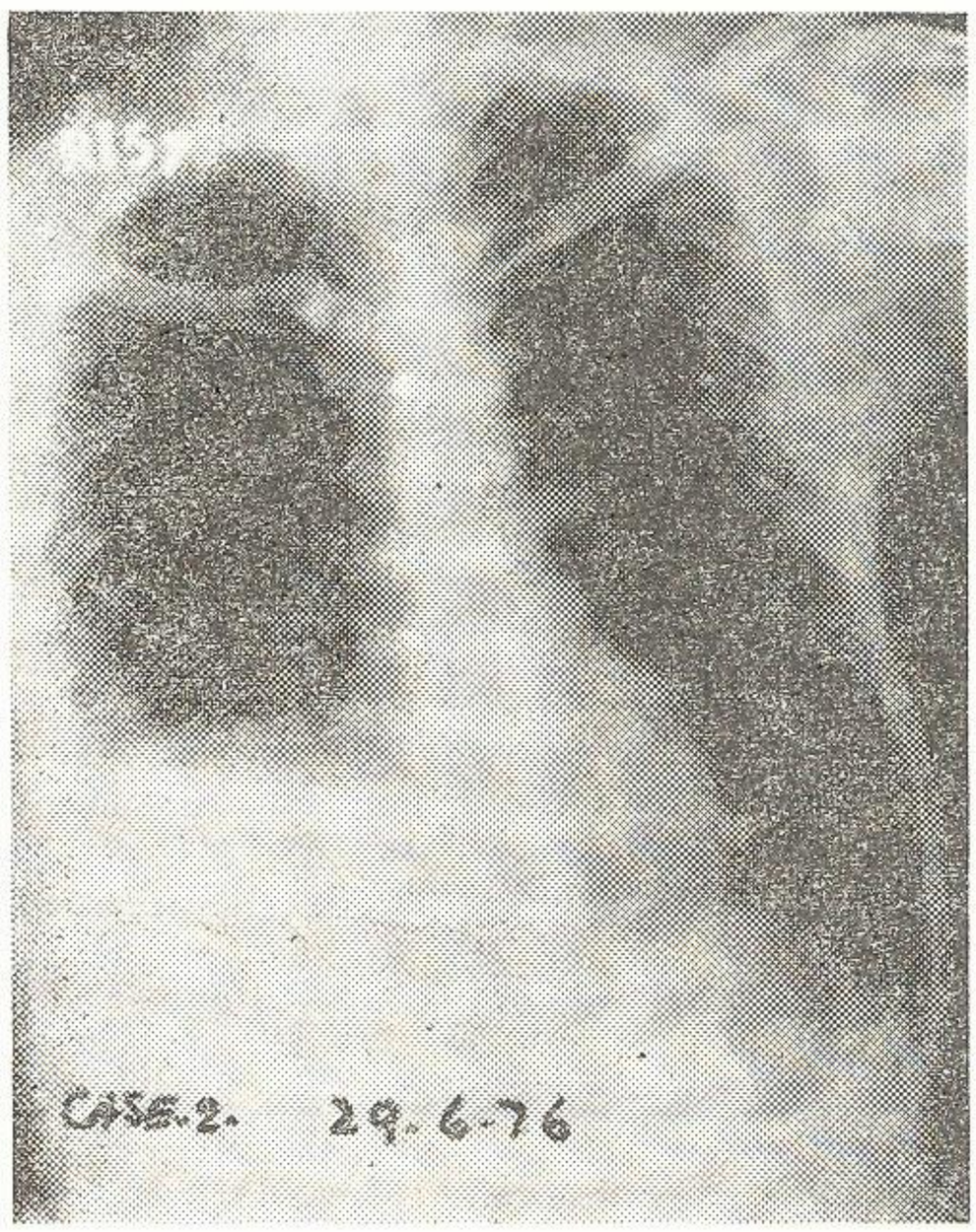

Fig. 7: Case-2 (29-6-1976). X-Ray chest P.A. view showing pleural effusion at the right base of the lung.

Cytology revealed plenty of polymorph, leucocytes and lymphocytes. No malignant cells were seen.

Blood count showed: Hb 65%, R.B.C. 3 million/cu. mm, W.B.C. 9200/cu. mm, Polymorphs 70%, Lymphocytes-22%, Eosinophils 5%, ASOT:Normal limit, Agglutination, Blood culture, Urine and Stool revealed no abnormality.

Next day he complained of pain at the right upper quadrant of the abdomen and examination revealed enlarged tender and hard-smooth liver. Liver function tests on 30-6-1976 showed serum bilirubin 0.4 mg/100 ml, SGOT 42 units, Alkaline phosphatase 122 units and thymol turbidity 4 units.

In view of unresponsive PUO and sterile creamy coloured pleural fluid with enlarged and tender liver and right basal pulmonary lesions the diagnosis of the liver abscess of amebic origin was considered. He was giver dehydroemetine 60 mg I.M. daily for 10 days combined with metronidazole 400 mg t.d.s. for 10 days and tetracycline 250 mg six hourly plus vitamins.

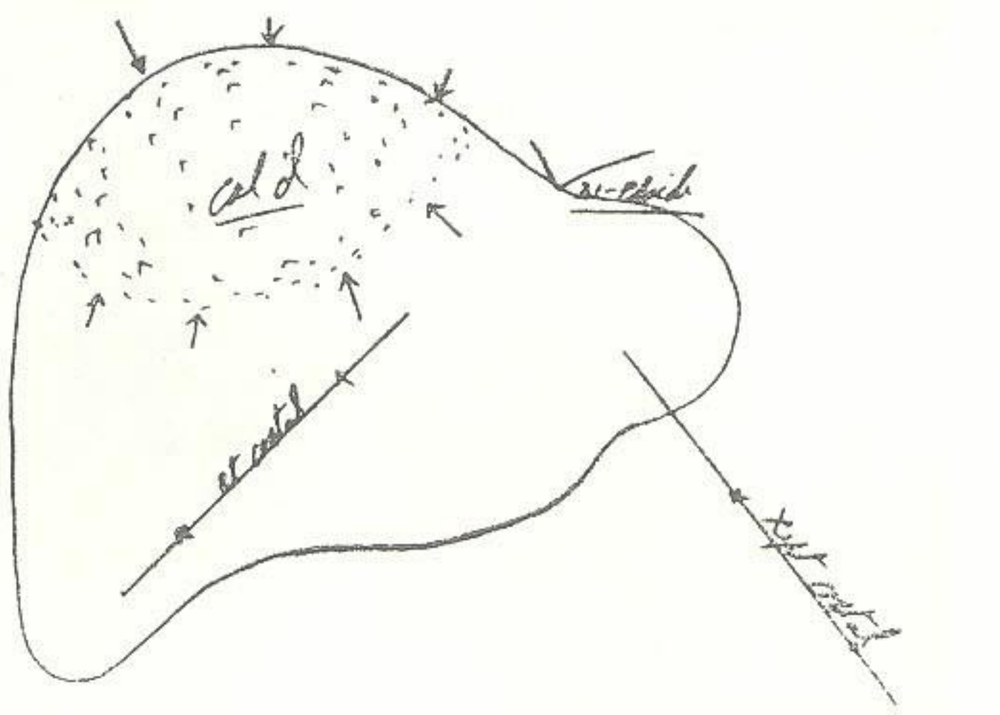

Fig 9: Case-2 (5-8-1976). Liver Scan showing cold at the upper part of right lobe of liver.

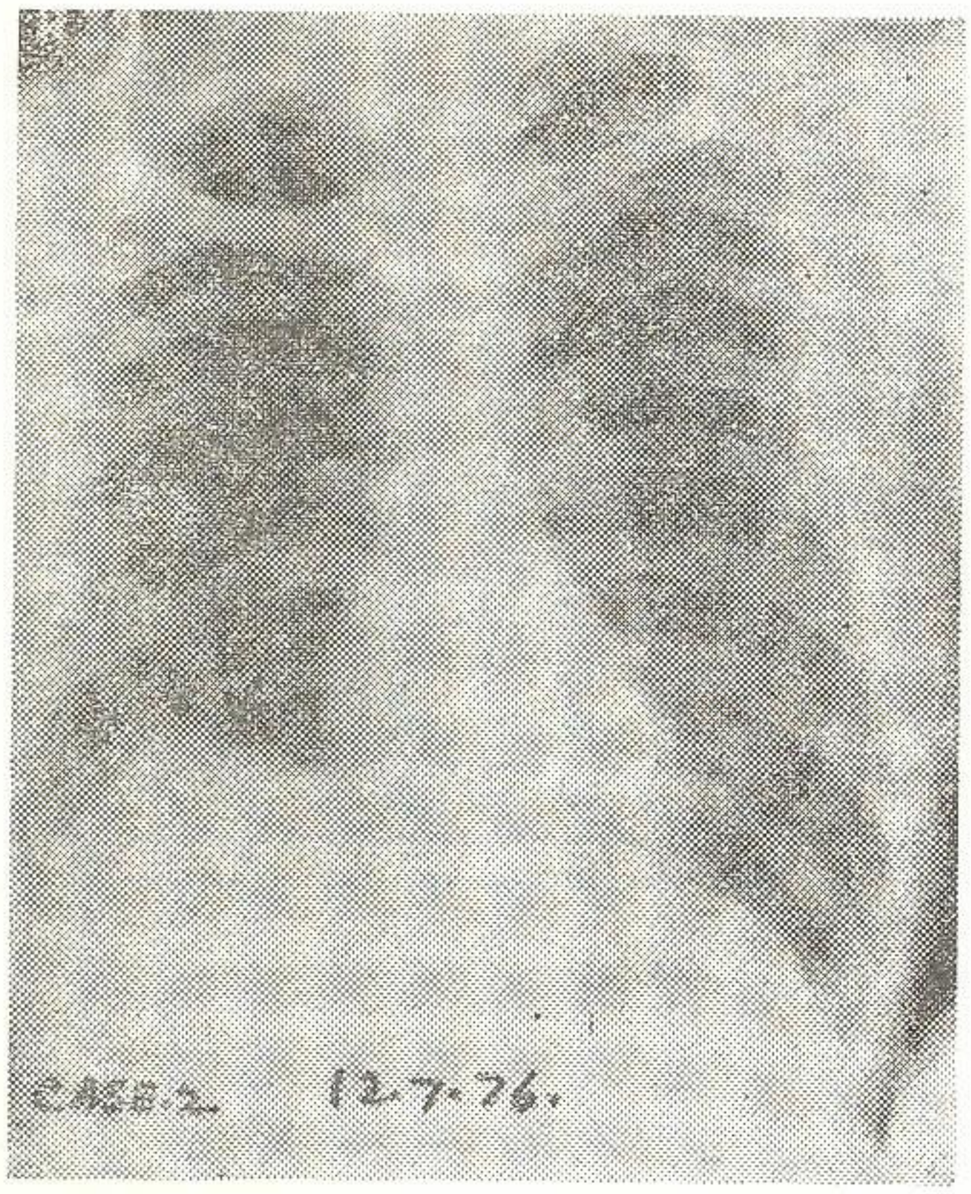

Fig. 8: Case-2 (12-7-1976). X-Ray chest after antiamebic therapy. Right diaphragm is raised with pointed centre suggestive of underlying liver abscess.

Fig. 10: Case-2 X-Ray chest P.A. view showing normal level of the right diaphragm without any pulmonary lesion.

The clinical response to antiamebic treatment was excellent, temperature settled down and pleural fluid cleared rapidly as revealed by radiology of the chest on 12-7-1976 (Fig-8). Right diaphragm was now elevated and it was tenting upward and pointed suggestive of unruptured liver abscess. Liver scan was arranged on 5-8-1976 at Jinnah Post-Graduate Medical Centre Radioisotope Unit which showed "cold area" in the upper part of the right lobe of the liver (Fig-9). Review of x-ray chest on 13-12-1976 showed raised right diaphragm and the liver scan showed a smaller cold area suggestive of further resolution of the unruptured liver abscess. Review x-ray chest on 25-4-1977 showed normal lungs and the level of the diaphragm on the right side descended to its normal level as now abscess has resolved completely, which took 10 months for this process of gradual healing. The abscess was not aspirated and showed no relapse after the 10 days course of antiamebic treatment.

Discussion

Both the presented cases of amebic liver abscess at the early stage of the illness presented as pulmonary lesions, patchy consolidation of the right base of the lung with signs of dullness on percussion and crepitations on auscultation followed by pleural effusion associated with pyrexia of unknown origin and pain on the right lower part of the chest. Aspirated pleural effusion was of light creamy colour in the Case-2 and was of creamy purulent nature in Case-1. In both the cases pleural fluid and the pus was sterile on culture. No parasite or fungus was found on examination of the pus.

In tuberculosis the effusion is usually straw colour. In amebic liver abscess the purulent material is classically anchovy Sauce colour which was obvious in the late stage of the illness in Case-1 suggesting amebic empyema which was confirmed by therapeutic test and excellent response to antiamebic treatment. Case-1 had not presented with signs and symptoms of liver abscess such as pain, localised swelling in the right upper abdominal quadrant and painful enlargement of the liver during her whole course of the illness, whereas Case-2 presented with signs and symptoms of the liver involvement with pleural effusion during second week of the illness, and this was a great help in the diagnosis of liver abscess. The case-2 liver abscess was on the verge of rupture in the pleural cavity as shown by followup radiology of the chest, whereas Case-1 had already ruptured in the pleural cavity when she came back after two weeks of second default with massive empyema.

The liver in Case-2 was enlarged, hard and tender without any localised swelling. Right diaphragm was raised, even after clearance of the pleural effusion and persisted for ten months suggestive of gradual resolution of the unaspirated liver abscess. The case-2 presented first as viral right basal pneumonia followed by pleural effusion and then signs and symptoms of the liver involvement. These sequence of events suggest the presence of amebic liver abscess.

In tropical areas physician should suspect amebic abscess in any case of pyrexia of unknown origin with sterile purulent pleural effusion and signs at the right lung base unless proved otherwise. Therapeutic trial with antiamebic treatment: metronidazole or dehydroemetine should be given at the earliest after preliminary investigations such as x-ray, blood count especially when sophisticated investigation facilities such as bacteriology and liver scan are not available in the rural areas of the tropical countries.

Rapid massive pleural effusion and sterile empyema as in Case-1 was due to rupture of amebic liver abscess in the pleural cavity which did not respond to anti-tuberculous treatment for eight weeks but responded promptly to antiamebic treatment: metronidazole 400 mg t.d.s. for 10 days and dehydroemetine 60 mg I.M. daily for 100 days. The clinical signs and symptoms cleared promptly but the residual pleural signs of subacute pleuritis in Case-1 and persistent raised right diaphragm in case- 2 due to gradual resolution of the liver abscess took 4 to 10 months respectively to attain normality. Liver Scan was normal at ten months in Case-2. Case-2 was treated at the earliest before rupture of the liver abscess in the pleural cavity, while in case-1 due to delay in diagnosis and treatment the abscess had ruptured in the pleural cavity and hence there was no persistent raised diaphragm as in Case-2.

Liver function tests are not specific in amebic liver abscess. Serological tests are not needed for routine cases but may be helpful in those cases with atypical features. In the case-2 alkaline phosphatase was raised to 122 K.A. units. Haider and associates (1974) reported liver function tests carried out in 60 cases of amebic liver abscess from Pakistan. Alkaline phosphatase was raised in 36 cases (60%) with a range of 16-35 K.A. Units, raised bilirubin in 7 cases with a range of 1.5-15 mg%, raised S.G.P.T. in 16 cases and S.G.O.T. in 20 cases (range: 35-60 Units).

Recently Lamont and Wicks (1976) reported 88 African patients with amebic liver abscess from Rhodesia. The diagnosis was made in pyrexial cases who had right sided upper abdominal pain and signs at the right base of the lung. In only half of the aspirations the classical anchovy Sauce appearance was seen. Ameba was found in 14% pus specimen, while biopsy of the abscess edge yielded 40%. In their 69 cases radiology showed raised right diaphragm in 45 cases (66%) pleural effusion in 13 cases (18%), and consolidation of the right base in 19 cases (27%). Liver Scan in 31 cases showed single abscess in 25 cases (80%), right lobe abscess in 18 and left lobe in 7 cases and multiple abscess in 5 cases. One case had massive pleural effusion. Latex amebic test was positive in 82% of sera examined. Five cases did not complain of any abdominal pain and nine were apyrexial, in such cases diagnosis is considerably delayed. Kean and associates (1956) from U.S.A. had reported most of the similar clinical findings.

In some cases it is difficult to differentiate amebic liver abscess from hepatocellular carcinoma and Hodgkin disease. One case was seen at this Centre, who had pyrexia of unknown origin with tender enlarged liver for 6 months. All investigations were negative till the appearance of enlarged cervical glands. The biopsy of

the gland was positive for Hodgkin granuloma. Amebic latex test is useful in such cases.

Radiology of the chest, showing raised right diaphragm and signs at the right base are helpful for diag nosis in 70% cases. Liver Scan is valuable aid in locating the site of the abscess for aspiration and needle biopsy. Biopsy of the edge of the abscess is an important diagnostic aid because ameba is present in 40% cases according to the experience of Lamont and Wicks (1974) and Wicks, Keeley and associates (1962). Ameba was not found in aspirated pus.

Metronidazole is a very good therapeutic agent. The most satisfactory regime appears to be 800 mg t.d.s. for 5 days and needle aspiration in those with large abscess. In the presented cases 400 mg t.d.s. for 10 days combined with dehydroemetine 60 mg I.M. for 10 days gave excellent result. Pleural effusion cleared within one week in Case-2 and pus drainage, which was continued for 2 months in Case-1, was dried up within one week but clearance of the pleural residual lesions (pleural thickening) in Case-1 took some weeks as revealed by radiology. The "cold area" shown on the liver scan in Case-2, took 10 months for resolution.

More recently therapeutic liver abscess aspiration has been advocated less frequently unless the abscess is very large. In Case-1 pleural drainage of the pus has to be done because it was a total and massive empyema due to rupture of the abscess in the pleural cavity. The liver abscess was not aspirated in Case-2 which subsided in 10 months without aspiration as revealed by serial liver scan. It was found that single dose of 2.4G metronidazole was more efficacious than the other dosage schedule in amebic liver abscess (Khan and Khakwani, 1971). Two out of 11 patients treated with such a loading dose reported back after four weeks with relapse (Khan and Khakwani, 1971). There was no relapse in the presented cases treated with metronidazole 400 mg t.d.s. and dehydroemetine 60 mg I.M. daily for 10 days.

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