AMOEBIC LIVER ABSCESS CAUSING CAVAL THROMBOSIS, PULMONARY EMBOLIC DISEASE AND COR PULMONALE

THREE CASE REPORTS

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The purpose of this paper is to present the aetiological role of amoebic liver abscess in the production of venous thrombosis and subsequent pulmonary embolic disease leading to cor pulmonale. Cor pulmonale associated with amoebic liver abscess has not been encountered here previously, nor has an extensive search of the literature revealed any published reports.

Three cases of amoebic liver abscess complicated by thrombosis of the inferior vena cava are reported; in 2 of these pulmonary embolic disease leading to right ventricular failure occurred.

CASE REPORTS

Case 1

A previously healthy African male, aged 25 years, was admitted to hospital complaining of pain in the right upper abdomen of 1 week's duration. The pain was aggravated by coughing. He admitted to dyspnoea on exertion, orthopnoea, swelling of the ankles and headache.

On examination oedema of the ankles and sacrum was found, with puffiness of the face. The jugular venous pressure was raised. Tachycardia, cardiomegaly and a presystolic gallop rhythm were present. The blood pressure on admission was 150/108 mm.Hg. Gross ascites and a right-sided pleural effusion were found. The liver was enlarged 3 fingerbreadths below the right costal margin and was tender.

Special investigations. Hb. 12·5 G/100 ml., WBC 16,000/cu.mm. The CSF had xanthochromic supernatant fluid. RBCs scanty, polymorphs +, lymphocytes + +, protein 504 mg./100 ml., globulin + + +, Cl 640 mg./100 ml., sugar 24 mg./100 ml. X-ray of the chest showed elevation of the right hemidiaphragm posteriorly and a small effusion at the right base.

A clinical diagnosis of congestive cardiac failure was made on admission, but the aetiology was undetermined. Treatment consisted of bed rest, digoxin and diuretics. Two days later the patient developed neck stiffness, bilateral extensor plantar reflexes and pin-point pupils. Clinically, meningitis or an amoebic brain abscess was suspected and a lumbar puncture was performed. Emetine hydrochloride was added to the treatment. The patient became confused, lapsed into coma, and died on the 4th day after admission.

Necropsy findings. The body weighed 130 lb. Oedema of the legs and sacrum was noted. The liver weighed 2,700 G and a large amoebic abscess was found in the right lobe posteriorly. The diaphragm was adherent anteriorly and a loculated subphrenic abscess was present. The liver abscess communicated with the inferior vena cava through a small perforation, and a polypoidal thrombus was found adherent at the site of perforation. The lungs (750 and 650 G) showed minimal oedema and numerous emboli adherent to the walls of the arteries. Haemorrhagic infarction was not found. The heart weighed 350 G and gross examination demonstrated hypertrophy and dilatation of the right ventricle and a relatively normal left ventricle. Mural thrombi were present in the right atrial appendage. The brain revealed a large abscess situated in the left frontal lobe. The colon showed no abnormality. The other organs were congested. Bilateral small hydrothoraces and moderate ascites were also present.

Microscopic examination of sections from the liver disclosed an amoebic abscess but no amoebae were seen. The absence of amoebae in sections may have been due to emetine therapy. Sections from both lungs revealed recent and organizing emboli in the pulmonary arteries at all levels. Sections from the brain showed an abscess but again amoebae were not seen.

Case 2

An African male aged 70 years was admitted to the King Edward VIII Hospital acutely ill and unable to give any history of himself. The patient's daughter testified that her father had been admitted to another hospital recently complaining of swelling of the ankles, shortness of breath and upper abdominal pain. He left hospital about 10 days before the present admission and had discontinued his maintenance therapy. Swelling of the ankles and shortness of breath at rest recurred and he was brought to hospital.

On examination he was very ill, dyspnoeic, pale, and had oedema of the ankles and sacrum. The jugular venous pressure was raised. Tachycardia and cardiomegaly were found and the blood pressure was 130/90 mm.Hg. The abdomen was slightly distended and a firm liver was felt in the epigastrium. Free fluid was present in the peritoneal cavity.

A diagnosis of congestive cardiac failure of undetermined aetiology was made. The patient died within hours of admission and no special investigations were done.

Necropsy findings. The body weighed 120 lb. Oedema of the lower limbs was noted. The liver weighed 1,650 G, was siderotic and dense adhesions were present on the diaphragmatic surface. An amoebic abscess 5 cm. in diameter was found in the right lobe of the liver. Thrombosis of the right hepatic vein extending into the inferior vena cava was noted (Fig. 1). Examination of the lungs showed extensive emboli in

Fig. 1. Thrombosis of the right hepatic vein extending into the inferior vena cava.

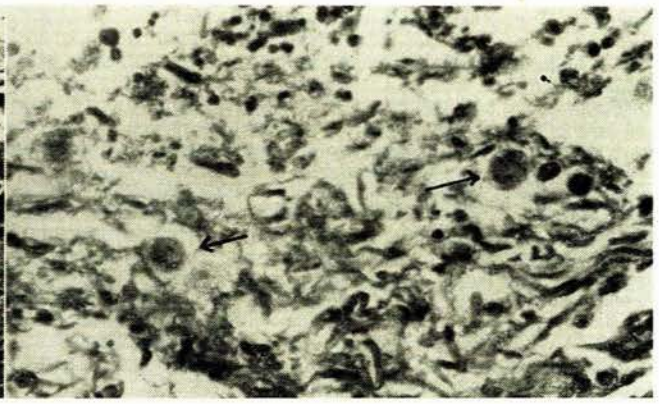

Fig. 2. Section from edge of liver abscess showing amoebae (x 400).

the arteries. These involved the pulmonary arteries at all levels but were particularly numerous in the small arteries. Haemorrhagic infarction was not found. A small abscess was present in the upper lobe of the left lung. The heart weighed 350 G and right ventricular hypertrophy and dilatation were demonstrated. A large thrombus was found adherent to the wall of the right atrium near the foramen ovale. The colon showed smooth areas on the mucosal surface suggesting healing areas of ulceration. Moderate ascites was present. At necropsy, pus was taken from the liver and lung abscesses. *Entamoeba histolytica* was demonstrated from the liver but not from the lung abscess.

Microscopic examination of sections from both the liver and lung abscesses showed amoebae in the liver section (Fig. 2) but not in the lung sections. The sections from the lungs showed extensive embolization with variation in the size, age and location of the emboli (Fig. 3). Various stages of organization of the emboli were present (Fig. 4). Some of the lesions in the small arteries were so well organized that they closely resembled arteriosclerosis (Fig. 5).

Case 3

A 22-year-old African female, 3 months pregnant, was admitted to hospital with pleuritic pain, having been treated for dysentery as an outpatient 2 weeks previously.

On examination she was ill, pyrexial and dyspnoeic. Tachycardia was present but no signs of congestive cardiac failure were found. There was dullness to percussion at the right base with diminished air entry and crepitations. Intercostal tenderness was present on the right side but the liver was not palpable. Tenderness to palpation was present in the epigastrium.

Special investigations. Hb. 9·3 G/100 ml., PCV 31, MCHC 30, ESR 60, WBC 19,000/cu.mm. with 78% polymorphs. X-ray screening of the chest showed elevation of the right hemidiaphragm, with minimal movements on the right side.

A diagnosis of amoebic liver abscess was made and the patient was treated with emetine hydrochloride. Two days later she developed abdominal distension, pallor (Hb. 7·9 G/100 ml.) and a palpable liver. She was transfused with packed red blood cells, and on the 5th day after admission the liver was

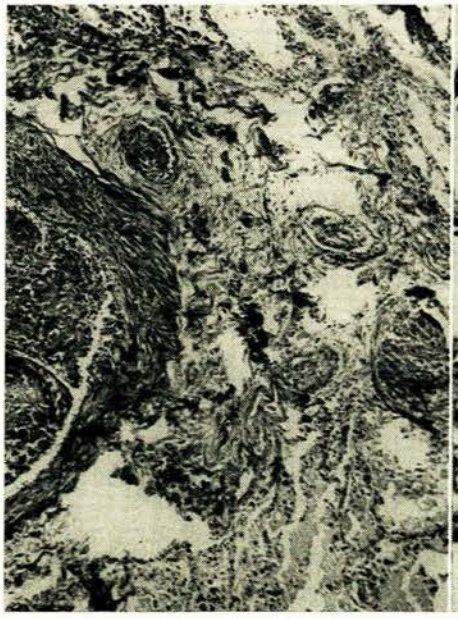

Fig. 3

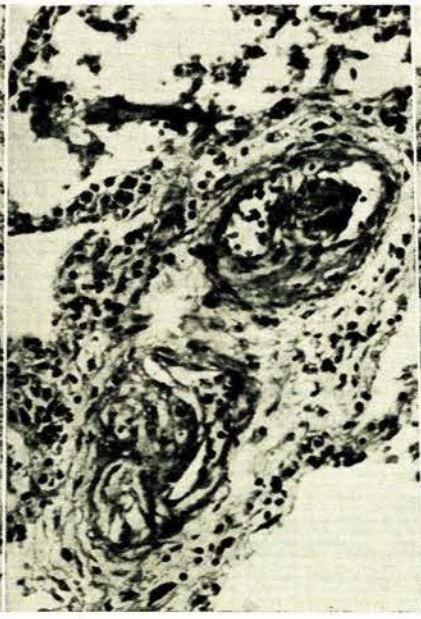

Fig. 4

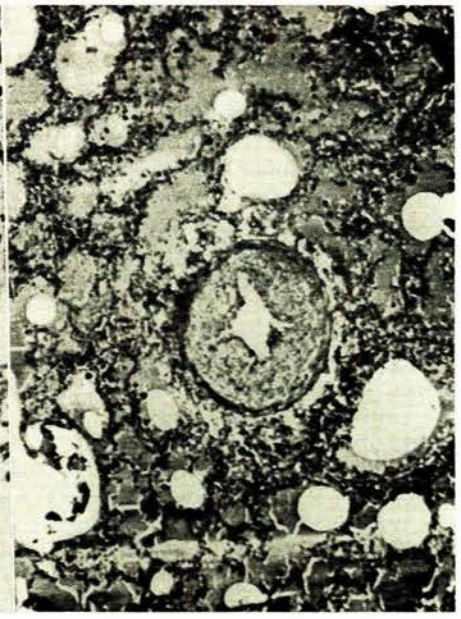

Fig. 5

Fig. 3. Lung section showing variation in size of emboli (x 150). *Fig. 4.* Lung section showing evidence of organization of emboli (x 320). *Fig. 5.* Lung section with well-organized lesion in a small artery, bearing a close resemblance to arteriosclerosis (x 230).

aspirated and 190 ml. of thick anchovy pus removed. Two days later a second aspiration yielded only 2 ml. of thick pus. The patient thereafter developed severe pain and sweated profusely. The liver had now increased in size. Laparotomy was performed and approximately 5 l. of pus were removed. Subsequently the patient became jaundiced, developed an ileus, and aborted. However, she made a satisfactory recovery and was returned to the medical wards. Two weeks later she complained of pain in the right upper abdomen, pleuritic pain in the right lower chest, and abdominal distension. She looked ill, and was pyrexial and pale. There was marked intercostal tenderness associated with tender hepatomegaly and abdominal distension. Recurrence of the abscess was diagnosed and needle aspiration yielded 20 ml. of pus. On the following day the liver edge was palpable at the umbilicus. Marked pallor was noted (Hb. 8·7 G/100 ml.) and a further transfusion was given, but there was little improvement until the patient's sudden death 7 weeks after admission to hospital.

Necropsy findings. Oedema of the legs was noted. The liver was grossly enlarged (2,070 G) and showed a nutmeg pattern. A large amoebic abscess was found in the right lobe of the liver. It was situated posteriorly, and communicated with the inferior vena cava through a ½-inch perforation in the anterior wall of the vessel. This perforation was situated just below the opening of the hepatic veins. Extensive thrombosis of the inferior vena cava was present below the site of perforation and extended as far down as the common iliac veins. Thick anchovy pus was present, above the thrombus, filling the right atrium, right ventricle, and pulmonary arteries. The pulmonary arteries showed no evidence of thrombotic emboli. The heart unfortunately was not weighed, but cardiac hypertrophy was not suspected at necropsy. Moderate ascites was present.

Microscopic examination of sections from the liver revealed part of the abscess cavity lined by dense fibrous tissue and containing a few inflammatory cells. Amoebae were not seen. Sections from the lung revealed no evidence of thrombo-emboli in the pulmonary arteries.

DISCUSSION

Necropsy in all 3 cases revealed an amoebic liver abscess associated with thrombosis in the inferior vena cava. In 2 cases (1 and 3) the initial site of thrombus formation was the inferior vena cava. In one (case 2) the primary site of thrombosis was in the right hepatic vein, and from here thrombosis extended into the inferior vena cava. In 2 cases (1 and 3) communication between the abscess and the inferior vena cava, indicating that rupture of the abscess had occurred, was established at necropsy. Thrombo-embolic disease of the lungs with right ventricular hypertrophy and dilatation was evident in the 2 patients who presented with congestive cardiac failure. In case 3, despite extensive thrombosis of the inferior vena cava, thrombotic pulmonary emboli were not found. Death in this patient was due to the emptying of a large part of the contents of the liver abscess into the inferior vena cava.

When an amoebic liver abscess ruptures into a hollow viscus, efficient drainage of the pus is often provided and the fistulous opening may close with conservative treatment.[1] If, however, rupture occurs in the form of a leak into a vascular channel such as the inferior vena cava, the dangers are 2-fold. Firstly there is the possibility of thrombus formation locally within the lumen with the danger of repeated showers of pulmonary emboli, and secondly, blood-borne infection of the lung and brain may occur.

Despite the failure to demonstrate amoebae, it seems probable that the brain abscess in case 1 and the lung abscess in case 2 were amoebic in origin. Other possible causes of these abscesses were considered and rejected.

Thrombosis of small veins often accounts for the nutmeg appearance seen around an amoebic liver abscess, but major veins are rarely involved.[1] Gordin[2] reported a case of amoebic liver abscess with thrombosis of the inferior vena cava, and thrombosis of the portal and hepatic veins was described by Hare and Ritchey.[3] Pulmonary embolism was not mentioned.

The 2 forms of pulmonary embolic disease best known are, firstly, where a large embolus occludes the pulmonary artery trunk and major branches causing sudden death, and secondly, the type characterized by small emboli settling in the peripheral branches of the pulmonary arteries producing haemorrhagic infarction. A third form recognized both clinically and at postmortem examination occurs when embolic disease is succeeded by right ventricular failure (cor pulmonale) following extensive obstruction to the pulmonary arteries after a succession of emboli. In this type haemorrhagic infarction may be present, but it is not a prominent feature.[4]

In recent years many published reports describing the third type of pulmonary embolic disease have appeared.[5,6] The clinical picture in such cases is one of increasingly severe congestive heart failure, but the signs of pulmonary hypertension may be absent. This, in addition to the silent nature of the pulmonary embolism, accounts for the difficulties in diagnosis. In the 2 cases described (1 and 2) which presented with congestive heart failure, the aetiology was undetermined during life.

The common sources of pulmonary emboli are the veins of the legs, and less commonly they originate in the right side of the heart and the pelvic veins. Thrombosis of the inferior vena cava is not common, and its association with amoebic liver abscess is rare. A posteriorly placed amoebic liver abscess is more likely to lead to thrombosis of the inferior vena cava and, when this occurs, the cause may be due either to extension from or rupture of the abscess into the inferior vena cava.

While the incidence of pulmonary thrombo-embolic disease with right ventricular failure is difficult to assess,[6] the condition does not appear to be common.

The prognosis of pulmonary embolic disease is grave.[6] Early recognition is essential to prevent irreversible damage to the pulmonary vasculature. The only therapy that can be offered is anticoagulants, unless a causative factor can be found which must be treated.

SUMMARY

Three cases of amoebic liver abscess with associated venous thrombosis, 2 with extensive pulmonary thrombo-embolic disease and subsequent cor pulmonale, are described.

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