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Case Report

Huge Amoebic Liver Abscess Presented with Massive Right Empyema: A Case Report

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Abstract
Amoebic liver abscess is a complication of amoebiasis that needs early diagnosis and proper treatment before further complications occur. We report a-35 year old female presented by fever and dyspnea due to huge liver abscess complicated by massive right side empyema. The patient was effectively treated by percutaneous drainage for both the right lobe abscess and empyema together with pharmacologic agents.

Introduction

Entamoeba histolytica is an intestinal protozoan that is highly prevalent in developing countries. Infection to humans occurs through the oral–fecal route. The infection by this protozoan is called amoebiasis; a primary intestinal infection which may be complicated by extra-intestinal infection (1). This protozoan organism is the third leading parasitic cause of death in the developing world and is an important health risk to travelers in endemic areas (2). Amoebic liver abscess (ALA) is the commonest extra-intestinal manifestations of amoebiasis. The incidence of ALA has been reported to vary between 3% and 9% of all cases of amoebiasis

and usually occurs in the right lobe of liver and is solitary (30–70%) (1).

Case Report

A 35-year old female presented by fever and dyspnea. On detailed history taking she gave no history of any chronic medical disease. She is a farmer of rural residence; married and had one offspring. The condition started one month ago by gradual onset, progressive course of dull aching right hypochondrial pain that was continuous, referred to the right shoulder and to the back. It was associated with continous fever, more at night and subsided by the use of antipyretics. There was anorexia, vomiting, dyspepsia to fatty meals, altered bowel habits in the form of (constipation alternating with diarrhea) with no dysentery or tenesmus.

She sought medical advice one week later in a primary health care unit and was diagnosed as acute cholecystitis and she was prescribed ciprofloxacin 500 mg/twice daily, paracetamol and i.v. fluids. Ten days ago the patient developed dyspnea on exertion associated with orthopnea with no paroxismal nocturnal dyspnea, no lower limb edema, associated also with right sided dull aching chest pain increased by respiration, relieved by holding breath with cough and expectoration of whitish sputum. Few days later with progressive increase in her body temperature, cough and progressive dyspnea she presented to our outpatient clinic.

On examination she looked ill and toxic with temperature 39.4 ºC, blood pressure 110/70, pulse 86/minute and was regular, abdominal and lower chest tenderness that hindered local abdominal palpation, and diminished air entry over the right lung and stony dull percussion note.

Her laboratory investigations were hemoglobin 8.7 gm/dl, white blood cells 16 600/mm^3 (mainly neutrophils), total bilirubin 0.4 mg/dl, total serum proteins 7.2 gm/dl, creatinine 0.9 mg/dl, ALT 35 IU/L, AST 41 IU/L, serum albumin 2.9 gm/dl, ESR 102/120, INR 1.17, Chest X ray showed homogenous opacity obliterating the right zone (Fig. 1). Abdominal ultrasonography showed huge abscess in the right liver lobe (Fig. 2). Abdominal CT (Fig. 3) showed cystic mass in right lobe of the liver. Pleural fluid aspiration showed total leucocyte count of 180580 cell/mm^3 (mainly neutrophils), LDH 10981 IU/L, Protein 2.2 gm/dl. The fluid in the abscess cavity was aspirated under US guidance and was chocolate colored “anchovy sauce" and subjected to culture & sensitivity which showed no bacterial growth, while the diagnosis of an Amoebic abscess was made by identification of *E. histolytica* trophozoites.

The case was then treated by hydroxychloroquine 250 mg two tablets twice daily for 2 days then once daily for 21 days. Metronidazole was prescribed i.v. drip twice daily for one week then orally 500 mg three times daily for another two weeks. Intravenous third generation cephalosporin cefotaixme in a dose of 1 gm/twice daily for two weeks was given plus other supportive therapies including antipyretics and i.v. fluids. The empyema was treated by fixation of an intercostal tube with good response (Fig. 4), while the liver abscess was drained through fixation of a pig-tail catheter with daily wash and aspiration. On discharge most of her laboratory parameters were normalized. Abdominal ultrasonography showed collapsed abscess cavity and the pig-tail catheter was removed and also good lung inflation and intercostal tube extraction was done.

Discussion

Entamoeba histolytica protozoan is a cosmopolitan infection that is frequent in tropical and subtropical areas including Egypt. Various factors such as poor hygiene, diabetes, steroid overuse, immunosuppressants have been known to complicated amoebiasis and predispose to the development of ALA (3).

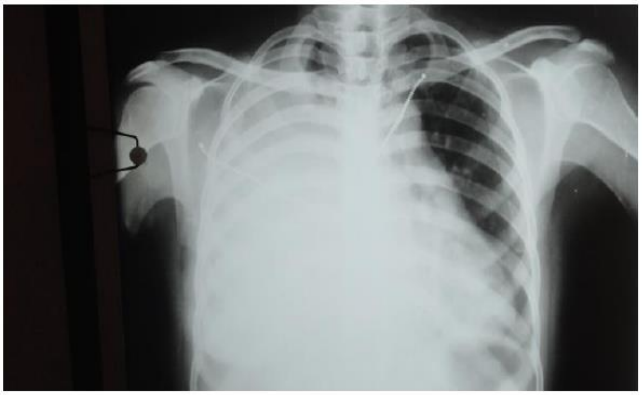

Fig. 1: Chest x ray showing homogenous opacity involving the right lung

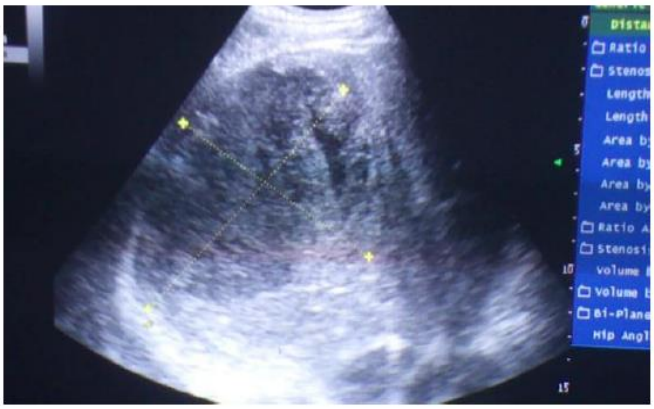

Fig. 2: Ultrasound picture showing huge abscess in the right liver lobe

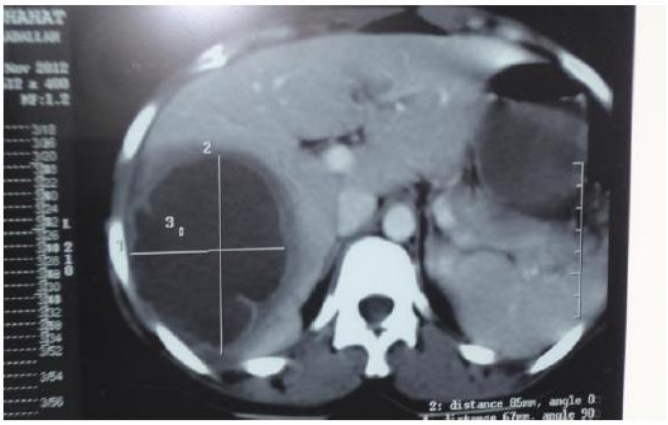

Fig. 3: CT scan showing cystic abscess cavity

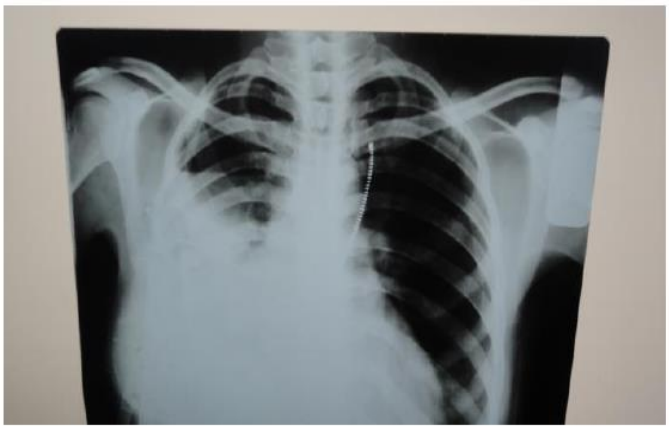

Fig. 4: Chest x ray showing improving right chest opacity

Development of ALA in this case per se is not surprising because she lives in a subtropical community where this infection is frequent and she is a farmer vulnerable to faeco-oral contamination.

ALA occurs most commonly in the age group of 20–45 years and has been noted infrequently in the extremes of ages (4), our case is a middle age and her framer job would expose her to the risk of infection.

Entamoeba histolytica exists in the cyst and trophozoite forms. Cysts are infective and are ingested through contaminated water or soil, and once reaching the alkaline medium of the small intestine, releases immature trophozoites. These trophozoites colonize the large intestine, predominantly the cecum. Liver abscesses have been attributed to invasion of the portal venous system by the amoebae. When the Amoebic infection reaches the liver parenchyma, microscopic sites of thrombosis, cytolysis, and liquefaction develop, causing hepatic necrosis. Coalescence of these areas of necrosis forms the abscess. Hepatic Amoebic abscesses are not true abscesses but pockets of necrotic debris. This material often contains mobile trophozoites, which can be demonstrated on a warmed microscope stage (diagnosis of our case was achieved by detection of this trophozoite on microscopic examination). The reddish brown color of the abscess content is due to the digestion of liver tissue and

red blood cells. The frequent location in the right lobe is most likely due to its larger volume. Further, it receives a major part of the venous drainage from the cecum and ascending colon, the portions of bowel most frequently affected by amoebiasis (5).

Liver abscess can be presented by a variety of manifestations that may mimic abdominal and systemic diseases (4, 5). This was occurred in our case, she was initially diagnosed as acute cholecystitis. At this moment the patient was not investigated by abdominal ultrasonography, a simple and bedside effective diagnostic modality, an investigation that if performed early would prevent further complications of liver abscess particularly ruptures. This simple diagnostic modality should be available in primary health care units.

Amoebiasis is a treatable disease but delay in the diagnosis may predispose the patient to serious complications and even death (3) and that was reinforced by this case, she was presented more than two weeks after her initial complaint and the lack of diagnostic facilities in the primary health care contributed to this delay in diagnosis and development of the complication empyema.

About 2 to 7% of Amoebic liver abscesses are complicated by perforation. Perforation sites mostly include pleuropulmonary structures (72%), the subphrenic space (14%) and the peritoneal cavity (10%) (6). Our case was complicated by right side empyema because her liver abscess was not only huge but also lies in the right lobe near the dome of the liver. The incidence of secondary involvement of the adjoining organs and other complications is higher in cases involving the left lobe rather than the right (4), but the huge size of the abscess and its proximity to the surface would explain the sequale of perforation into the right hemithorax.

The mortality in ruptured ALA is higher than non-ruptured ALA, while the hospital stay also prolonged for ruptured cases (3, 6). Our case had hospital stay of 24 days and she was delivered in a good condition, we assume this success to the rapid intervention to both the empyema by the intercostal tube insertion and drainage of the liver abscess, also the use of anti-Amoebic medications and antibiotics. The patient was also some what lucky because her huge abscess was not ruptured inside the abdominal cavity a complication if occurred would not only complicate the management plan but also potentially threatens her survival.

Conclusion

Amoebic liver abscess is a complication of amoebiasis that needs early diagnosis and proper treatment before further complications occur. Percutaneous drainage and pharmacologic agents are effective in treating complicated ALA.

Acknowledgements

The authors declare that there is no conflict of interests.

References

1. Sharma MP, Ahuja V. Amoebic Liver Abscess. JIACM. 2003;4(2):107–11.
2. Hughe MA, Petri WA Jr. Amoebic liver abscess. Infect Dis Clin North Am. 2000; 14(3):565-82.
3. Papavramidis TS, Sapalidis K, Pappas D, Karagianopoulou G, Trikoupi A, Souleimanis CH, Papavramidis ST. Gigantic hepatic Amoebic abscess presenting as acute abdomen: a case report. J Med Case Rep. 2008;12(2):325.
4. Yin LS, Jayarani K, Kumar G. Left lobe amoebic liver abscess mimicking a perforated gastric tumour. European Journal of Radiology Extra. 2008; 66: e25–e27.
5. Miller Q, Kenney JM, Cotlar AM. Amoebic abscess of the liver presenting as acute cholecystitis. Curr Surg. 2000;57(5):476-479.
6. Meng XY, Wu Jx. Perforated Amoebic liver abscesses: clinical analysis of 110 cases. South Med J. 1994;87(10):985-990.