Amoebic colitis with liver abscess

Mansharan Kaur Chaincel Singh[1,2]

Abstract

Amoebiasis is a parasitic infection caused by the intestinal protozoan Entamoeba histolytica, most prevalent in developing countries. It results in 40,000 to 100,000 deaths each year from amoebic colitis and extra intestinal infections. Amoebic liver abscess (ALA) is the most common extra intestinal site of infection with an incidence of between 3% and 9% of all cases of amoebiasis. Ultrasound which has a sensitivity of more than 90% for detecting ALA is highly recommended as an initial investigation followed by serological demonstration of circulating antibodies specific to *Entamoeba histolytica*.

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Introduction

Amoebiasis is a parasitic infection caused by Entamoeba histolytica due to lack of hygiene and proper sewerage systems, alcoholism, immunosuppression and travel to endemic areas such as Mexico, India and Africa.[1,2] Infection occurs when mature cysts ingested through faecally contaminated water or food reach the small intestine causing excystation, releasing trophozoites which penetrate the colonic mucosa leading to infection of the liver, brain, lungs and pericardium.

Though various organs can be affected by extra-intestinal amebiasis, liver abscess (ALA) is the most frequent manifestation presenting with an acute or subacute history of fever and right upper quadrant pain, although a chronic presentation of diarrhoea, weight loss and abdominal pain may occur.[1] Diagnosis of ALA is based on clinical symptoms, radiological imaging and serological diagnosis by PCR detection of *E. histolytica* DNA.[3,4]

Case History:

A 79-year-old Malay man presented with history of feeling unwell for four days, abdominal pain and vomiting for two days and watery diarrhoea for one day. He had no previous medical illness except for benign prostate hyperplasia and rheumatoid arthritis. On examination, the patient was alert but dehydrated with a temperature of 37.6°C and minimal right lower lobe crepitations. The patient was started on intravenous Augmentin 600 mg 12 hourly with blood, urine and stool specimens sent for biochemical investigation. A day later, the medication was changed to intravenous Rocephin 2.0 gm over 24 hours.

Blood investigations revealed a total white cell count of 40,000 and haemoglobin level of 7.2 gm%, thus a differential diagnosis of lymphoma or underlying malignancy was entertained with arrangements made for computed tomography (CT) scan of the chest, abdomen and pelvis, an oesophagogastroduodenoscopy and colonoscopy.

Amoeba serology came back positive from the blood culture and patient was started on intravenous Flagyl in addition to Rocephin, however, the patient still had fever, diarrhoea on and off and was quite unwell. Colonoscopy revealed a mass distal to the splenic flexure, a biopsy was done and sent for histopathology examination with a provisional diagnosis of carcinoma colon.

CT scan of the neck, thorax and abdomen revealed shotty pretracheal lymph nodes with bilateral pleural thickening and basal atelectasis, a large hypodense lesion in segment seven of the liver (Figure 1) and another smaller lesion in segment two with bowel thickening of the caecum (Figure 2) and splenic flexure. Histopathology of the splenic flexure mass revealed granulation tissue with no evidence of malignancy, while ultrasound guided aspiration of the liver abscess revealed anchovy sauce aspirate and grew amoebae. However, despite 18 days of treatment with Flagyl, the patient still had diarrhoea though the fever had settled. A repeat CT

[1]Institute of Pathology, Laboratory and Forensic Medicine (I-PPerForM), Universiti Teknologi MARA (UiTM) Sungai Buloh, MALAYSIA

[2]Faculty of Medicine, Universiti Teknologi MARA (UiTM) Sungai Buloh, MALAYSIA

Address for Correspondence:

Mansharan Kaur A/P Chainchel Singh, Fellow of Forensic Radiology, Institute of Pathology, Laboratory and Forensic Medicine (I-PPerForM), UiTM Sungai Buloh, Selangor, MALAYSIA.

E-mail address: mansharan70@gmail.com

Phone number: +603-6126 7724 Fax number: +603-6126 7465

scan of the chest, abdomen and pelvis done three weeks later showed that the liver lesions were smaller though the thickened wall of the ascending colon remained the same. The patient was discharged home after 41 days of intravenous Flagyl treatment with the diagnosis of amoebiasis with amoebic liver abscess (ALA) and asked to come back for review in six weeks.

A repeat CT scan of the liver after 6 weeks showed that the liver lesions had resolved (Figure 3) and a repeat colonoscopy showed a benign looking stricture of the sigmoid colon with no evidence of malignancy.

Discussion

E. histolytica the causative organism in amoebiasis, is a substantial risk in countries where the barriers between human faeces and food and water are inadequate. Infection begins with the ingestion of cysts in food or water that has been contaminated by human faeces which travel through the small intestine or colon and excyst to form the trophozoites resulting in colitis. Human and some non-human primates are the only natural hosts in this infection.

The pathological range of amoebic colitis encompasses mucosal thickening, multiple discrete ulcers separated by normal colonic mucosa, diffusely inflamed and oedematous mucosa, necrosis and perforation of the intestinal wall which can resemble those seen in inflammatory bowel disease. The most common extra-intestinal manifestation of amoebiasis is ALA where *E. histolytica* reaching the liver via the portal vein create abscesses which are well circumscribed areas of dead hepatocytes, liquefied cells and cellular debris. A rim of connective tissue with few inflammatory cells and amoebic trophozoites surround the abscess while the surrounding liver parenchyma is often unaffected.[5]

CT scan of liver abscess will show a hypodense lesion with attenuation values of 15 to 35 Hounsefield units. This lesion is usually avascular with well-defined margins and demonstrate alternating hypervascular and hypovascular halos following contrast media enhancement. Ultrasonographic appearances of liver abscess vary, depending on the stage and evolution of the necrotic mass. Initially, the lesion has greater echogenicity when the necrotic centre is solid, eventually converting to an echolucent area with posterior acoustic enhancement.

Patients with amoebic colitis present with bloody diarrhoea, abdominal pain and tenderness, though rectal bleeding without diarrhoea is commonly seen in children. Occasionally, individuals develop fulminant amoebic colitis, with bloody diarrhoea, fever, pronounced leucocytosis and widespread abdominal pain. Mortality with fulminant amoebic colitis is higher in pregnant patients, immunocompromised individuals and those on corticosteroids.[5]

The diagnosis of amoebiasis is based on light microscopy of *E. histolytica* during its various stages of development particularly the identification of amoebic trophozoites in stool and serological tests including latex agglutination that identify circulating amoeba specific antibodies. Plain films of the abdomen are of little value unless a perforation occurs which is seen in 60 to 75% of fulminant colitis while barium enema examinations allow evaluation of mucosal abnormalities as does colonoscopy.

Amoeboma which was seen in this patient and initially mistaken for a colonic malignancy is actually a hyperplastic granuloma of the large bowel caused by bacterial invasion of an amoebic abscess in the bowel wall. It is characterized by marked thickening of the bowel, with dense fibrosis at the periphery and a core of acute and chronic inflammation and necrosis at in the centre. This mass may present as an eccentric filling defect in the bowel lumen, but more commonly it causes a long, tapered stricture encircling the bowel to produce an apple core associated with a palpable mass mimicking malignancy.

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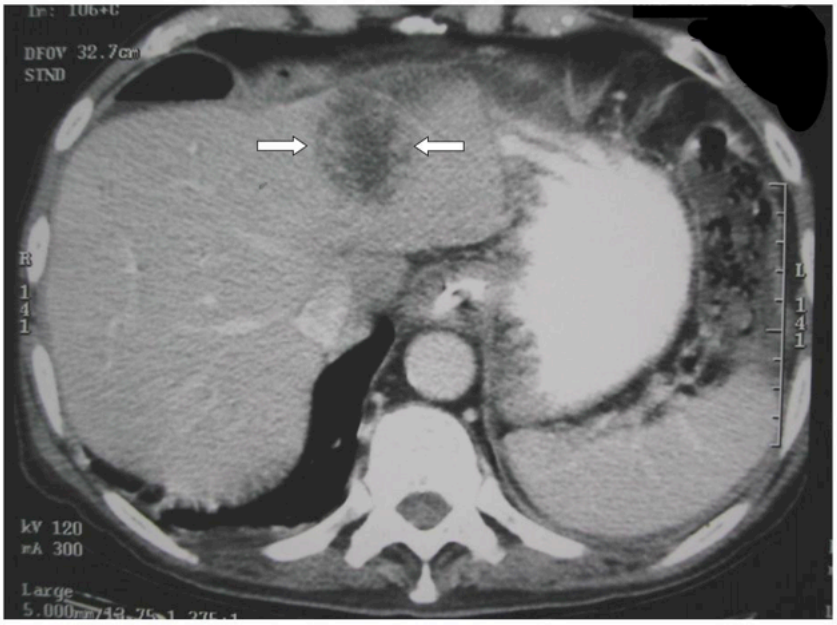

Figure 1: Axial CT scan of the liver showing liver abscess in the left lobe of the liver (arrows).

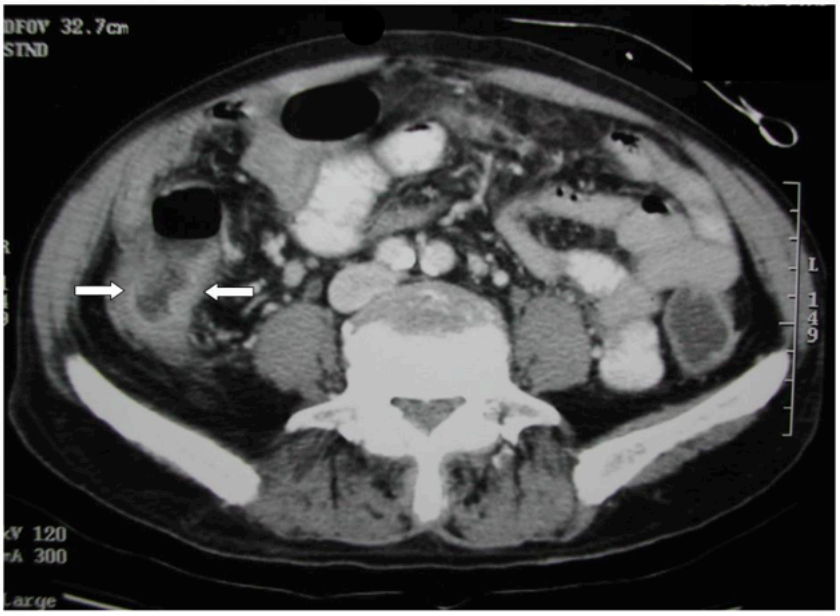

Figure 2: Axial CT scan of the abdomen showing a mass in the ascending colon/caecum (arrows).

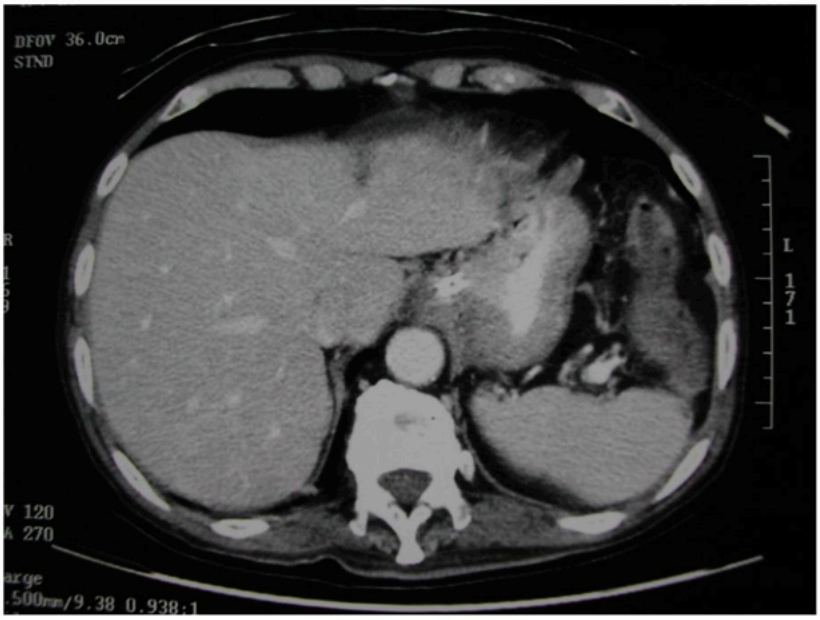

Figure 3: Axial CT scan of liver showing resolution of liver lesion.