Colonic amoebic abscess mimicking carcinoma of the colon

Amoebiasis is an uncommon disease in developed countries. Its clinical presentation can be variable and non-specific, and the diagnosis can be easily overlooked. We report a case of colonic amoebic abscess mimicking advanced colonic cancer with acute intestinal obstruction and liver metastasis. The presentation, diagnosis, and treatment of amoebiasis are also reviewed.

Introduction

Amoebiasis is uncommon in developed countries. In Hong Kong, where amoebiasis is a notifiable disease, a total of only 71 cases have been reported from 1997 to 2004.1 Although the disease is rare in many localities including Hong Kong, it is of particular note in that its presentation can mimic more common conditions, such as gastro-intestinal (GI) malignancy and idiopathic inflammatory bowel disease. We report a case of colonic amoebiasis in which the presenting symptoms and radiological findings closely resembled colonic carcinoma with liver involvement.

Case report

A 57-year-old man presented to our hospital with right-lower-quadrant abdominal pain of 10 days’ duration, associated with abdominal distension and constipation. There was no preceding diarrhoea or bleeding per rectum. He gave a history of recent travel to Macau 1 month before presentation. His medical history was otherwise unremarkable. Physical examination revealed a mildly tender, right-sided abdominal mass. Digital rectal examination was normal. Abdominal X-ray revealed dilated small bowel loops, while blood tests showed marked leukocytosis (white cell count, 27.2 x 10⁹/L) and mildly deranged liver function tests. Computed tomographic (CT) scanning of the abdomen confirmed an ascending colon mass with inflammatory changes (Fig 1a); two hypodense lesions with irregular edges in the liver were also identified (Fig 1b). The clinical picture was compatible with an obstructing right-sided colonic carcinoma, with liver metastases.

Emergency surgery was arranged. During the operation, a pericolic abscess was found on the ascending colon. This was walled off by small bowel, mesentery, and the anterior abdominal wall. Enlarged lymph nodes were also found along the ileocolic and right colic vessels. Right hemicolectomy and primary anastomosis were performed. On cutting open the specimen, thickened bowel wall was seen and the colonic mucosa was covered by necrotic material (Fig 2 inset). There was no obvious ulcerative growth however. The diagnosis of colonic amoebiasis was made on subsequent histopathological examination—the pathognomonic feature of protozoa with ingested red blood cells was seen in the specimen (Fig 2). Further serological testing revealed an elevated titre of Entamoeba histolytica immunoglobulin G antibody. The patient commenced treatment on metronidazole and made an uneventful recovery. The liver lesions,
presumably due to early abscess formation, were found to have resolved on a subsequent CT scan of the abdomen 6 weeks later.

**Discussion**

*Entamoeba histolytica* is a major cause of diarrhoea in developing countries. It infects millions of people worldwide each year, and approximately 40 000 to 100 000 people die annually from the disease. The infestation starts with ingestion of the cyst of *E histolytica* from faecally contaminated food or water. The cysts are digested in the intestinal lumen releasing trophozoites. The trophozoites reproduce by clonal expansion and subsequently form cysts which are excreted in the faeces to start a new cycle.

The two main organ systems affected by the parasite are the GI tract and the liver. The presentation of intestinal amoebiasis ranges from an asymptomatic carrier state, colitis, through abscess formation to perforation. Most patients with symptomatic infection present with a history of bloody or watery diarrhoea, abdominal pain, and weight loss for several weeks. In Hong Kong, a travel history to a developing country is common. Amoebiasis needs to be differentiated from bacterial causes of dysentery. The present case is atypical in that the patient had no preceding diarrhoea and a definite travel history was lacking.

Uncommon presentations of GI involvement include fulminant colitis, with a mortality rate exceeding 40%, toxic megacolon which is usually associated with the use of corticosteroids, and formation of an inflammatory phlegmon which mimics a colonic tumour, as seen in the current patient.

An amoebic liver abscess resulting from haematogenous spread from the GI tract is the most common extra-intestinal manifestation. This condition usually presents with fever and right-upper-quadrant abdominal pain. Concomitant GI symptoms may be present. Complications may arise as a result of rupture into body cavities, resulting in peritonitis, pericarditis, pleural effusion, or pneumonia. On an abdominal CT scan, an amoebic liver abscess usually appears as a rounded, well-defined, low-density lesion, with a homogeneous septated cavity, often containing considerable fluid. Even in hindsight, the liver lesions seen in the present case were not typical of an amoebic abscess, presumably because the inflammatory process was still at an early stage.

The conventional method for diagnosis of intestinal amoebiasis is examination of stool by microscopy. The reported sensitivity of this method in identifying amoebic protozoa ranges from 25% to 60% and is operator-dependent. Moreover, false positive results can occur as *E histolytica* is morphologically identical to non-pathological species, such as *Entamoeba dispar* and *Entamoeba moshkovskii*. Recent advances have introduced more sensitive and specific
methods for diagnosis, which include antigen detection both in the patient’s stool and serum.  

The principal treatment for amoebic colitis is with nitroimidazole therapy; metronidazole is the most commonly available drug. Affected patients are given metronidazole for 5 to 10 days; they should also be isolated from other patients to prevent cross-infection, with frequent handwashing and careful waste handling imperative. Treatment with metronidazole is followed by a luminal agent for 5 to 20 days to eradicate colonisation—paromomycin, diloxanide furoate, and iodoquinol are the commonly used agents. Even in the case of fulminant colitis with contained perforation, most patients can be successfully managed by broad-spectrum antibiotics to cover the bowel flora. Surgery is rarely required, and is indicated only in cases of diagnostic uncertainty or when toxic megacolon occurs. In the present case, the patient could have been spared surgery had a preoperative diagnosis of amoebiasis been made. It is likely that the pericolic abscess and the liver lesions would have responded to antibiotics, and the obstructive symptoms would have resolved with conservative therapy.

Metronidazole remains the mainstay of treatment for an amoebic liver abscess. In most patients the response is rapid and dramatic. Surgical drainage is considered only if there is no response to drug therapy, or when the diagnosis is uncertain. Drainage should be avoided in untreated cases, as it may result in systemic dissemination of the parasite.

Although amoebiasis is a rare disease in many parts of the world, it should be included as one of the differential diagnoses of acute abdomen and colonic mass. This is especially so when the patient gives a recent history of dysentery or has a travel history to other countries. Similarly, in the management of liver lesions, the possibility of an amoebic liver abscess must not be overlooked, and a history of bowel symptoms and travel history should be sought. A high index of suspicion is crucial for diagnosis, and is essential to avoid unnecessary surgery.

References