CASE REPORT

Branch portal vein pyaemia secondary to amoebic liver abscess

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SUMMARY

We describe a case of a young returning traveller who contracted amoebic dysentery while visiting India. She presented to a major London Hospital several months later with features suggestive of amoebic liver abscesses, a known sequelae of amoebiasis. MRI with intravenous contrast demonstrated an area of likely occlusion of the portal vein. The patient was treated with intravenous metronidazole for 10 days followed by diloxanide furoate, an intraluminal agent. The largest abscess was drained acutely under ultrasound guidance. The portal vein occlusion was treated medically without the use of anticoagulation. A repeat ultrasound at 6 weeks post-treatment confirmed patency of the portal vein indicating spontaneous recanalisation with antimicrobial therapy alone.

BACKGROUND

Entamoeba histolytica is an anaerobic parasitic protozoan that is endemic in many developing countries. It is estimated to infect about 50 million people worldwide and causes up to 100 000 deaths per year.¹ It is transmitted via the faecal-oral route, and symptoms can vary from mild diarrhoea to dysentery with blood and mucus. Transmission through the faecal-oral route usually occurs through the ingestion of faecally contaminated food or water containing the parasitic cysts. The cysts excystate inside the small bowel releasing trophozoites, which persist in the colon for weeks to months while being excreted in the faeces, and can cause an amoebic colitis.^{2 3}

E. histolytica infection can be intraintestinal or extraintestinal. This case report focuses on the diagnosis and management of extraintestinal infection. It is worth noting, however, that in colonic infection, stool microscopy alone is poor at differentiating the non-pathogenic forms of *Entamoeba* (including *E. dispar* and *E. moshkovskii*). Combined colonoscopy with biopsy and microscopy is required to differentiate between species.^{4 5}

Liver abscesses are the most common extraintestinal manifestation of *E. histolytica* infection. These are well defined areas of necrotic hepatocytes and cellular material surrounded by a thin rim of connective tissue and trophozoites, which develop through spread via portal vein radicles.⁶ Patients commonly present with upper abdominal pain and fever, and right upper quadrant tenderness and hepatomegaly on clinical examination.⁷ A leucocytosis, with raised alkaline phosphatase and a high erythrocyte sedimentation rate, are often found.^{3 6} Imaging modalities such as ultrasonography, CT

and MRI can characterise the lesions and identify the extent of the infection, including involvement of the portal circulation. Fine-needle aspiration and microscopy can be used to confirm diagnosis.⁸

While liver abscesses are well-documented sequelae of *E. histolytica* infection, there is a lack of published literature relating to the specific complication of portal pyaemia in the context of amoebic liver abscess.² In this report, we present one such case and discuss the challenging issues surrounding appropriate management. We highlight our finding that antibiotic and amoebicide therapy without anticoagulation successfully treated the portal vein congestion.

CASE PRESENTATION

A 20-year-old female patient, a university student, presented to hospital with a 2-day history of headaches, myalgia and right upper quadrant pain exacerbated by movement. This was on a background of intermittent 2-week episodes of bloody diarrhoea over the previous 4 months. She also reported increased urinary frequency and nocturia.

Two months prior to the admission, the patient had returned to the UK following a 3-month stay in India. Her travelling companions also reported similar symptoms. She had attended a clinic in India for her gastrointestinal symptoms and was given empirical treatment for 'travellers' diarrhoea', with oral ciprofloxacin. She continued to use this when symptoms returned several weeks later.

She had no significant medical history, took no regular medications and had no known allergies.

On physical examination, her blood pressure was 110/71 mm Hg, respiratory rate 18 breaths/min, saturations of oxygen 100% on air, heart rate 94 bpm and temperature 38.3°C. She was alert but appeared unwell. Examination of her abdomen revealed tenderness in the right upper quadrant and significant right-sided renal angle tenderness. The rest of the abdomen was soft and non-tender. Examination of the cardiovascular, respiratory and neurological systems was unremarkable. She had no peripheral stigmata of inflammatory bowel disease.

INVESTIGATIONS

Initial laboratory investigations found significantly raised white cells count and C reactive protein. Liver function and hepatitis screens were unremarkable. Malaria was not detected on blood film (table 1). Urine dip and culture were negative. Chest and abdominal radiographs were unremarkable, as was kidney ultrasound.

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Table 1 Table of results

Source	Variable	Value	Unit	Ref range
Blood	WCC count	19.42	10 ⁹ /L	4.0–11.0×10 ⁹ /L
	Haemoglobin	130	g/L	115–155 g/L
	Platelet count	333	10 ⁹ /L	150–400×10 ⁹ /L
	CRP	56.3	mg/L	<5 mg/L
Blood film				
Malaria	No malarial parasites seen			
WCC	Marked toxic neutrophilia			
Cultures				
Stool	<i>Entamoeba histolytica</i> /E. dispar seen			
Urine	Pus cells seen			
Blood	No growth after 5 days incubation			

CRP, C reactive protein; WCC, white cell count.

Liver ultrasound found at least three focal hypoechoic lesions in the right lobe of the liver measuring 5.4 cm, 2.5 cm and 2.3 cm in diameter. A subsequent MRI showed three liver abscesses within the right liver lobe and associated branch portal vein partial occlusion, indicative of portal pyaemia (figures 1 and 2). Stool samples subsequently tested positive for amoeba. Further plasma analysis tested positive for amoebic serology. Serological testing for *Echinococcus* was negative.

DIFFERENTIAL DIAGNOSIS

On the basis of the patient's history and examination findings, initially, the mostly likely source of her infection was a urinary tract infection, which was ruled out by negative urine dipstick and culture, and normal ultrasound imaging. Subsequent differential diagnoses included infective diarrhoea or gastrointestinal abscess. With her significant travel history and risk of parasitic infection, liver abscesses were a likely differential, therefore the patient underwent a liver ultrasound.

TREATMENT

Initially, the patient was treated with empirical intravenous broad spectrum co-amoxiclav. Metronidazole 800 mg three times a day was started to cover for amoebiasis. This was continued for 10 days to treat the disseminated parasitaemia with the

Figure 1 T1-weighted MR coronal section showing the loculated liver abscess in the right lobe.

Figure 2 T1-weighted venous phase contrast MRI demonstrating partial occlusion of the posterior division of the right portal vein.

initiation of the intraluminal agent, diloxanide furoate, being used subsequently to clear remaining ova from the gastrointestinal tract.

In addition to antibiotic treatment, ultrasound-guided drainage of the patient's largest liver abscess was undertaken. Anticoagulation options for portal pyaemia were discussed with the haematology department who, in liaison with the liver team, advised that anticoagulation was not indicated. A percutaneous drain was inserted on day 5 of admission and the liver abscess was drained—this was complicated by a small pneumothorax. The patient also underwent a full coagulopathy screen. The percutaneous drain was removed after 1 day. The patient remained clinically well and was discharged on the ninth day of admission to complete the remaining course of oral metronidazole and diloxanide furoate in the community.

OUTCOME AND FOLLOW-UP

The patient responded well to treatment and was discharged home after a 9-day hospital admission with arranged follow-up with hepatology and infectious diseases teams in 1 month. She was given advice for her travelling companions who were suffering from similar symptoms to present to hospital.

On the advice of the microbiology department, the intraluminal agent diloxanide furoate was prescribed to start after the complete course of metronidazole. This agent destroys the trophozoites of *E. histolytica* that eventually form into cysts. The cysts are then excreted in faeces.

A follow-up liver ultrasound scan at 6 weeks post-treatment revealed a 16 mm subcapsular focal lesion of slightly reduced reflectivity posteriorly in the right lobe, in keeping with a virtually resolved abscess. The portal vein was patent with normal velocity antegrade flow. The liver was of normal size and shape.

DISCUSSION

Documented complications of amoebic liver abscesses include rupture into peritoneal, pleural and pericardial cavities, and disturbances of portal circulation.^{9–11} This case is a unique example of the rare complication of portal vein congestion.

While there are numerous reports of vascular involvement in *postmortem* cases of hepatic amoebiasis, this is extremely rare in living patients. A *postmortem* study observed that 27.5% of portal veins, 29.5% of hepatic veins and 4% of inferior vena cava had evidence of thrombosis.¹⁰ However, there are only a small number of case reports identifying portal vein and inferior vena cava thrombosis in living individuals. Two recent reports

identified thrombosis of the right suprahepatic and inferior vena cava.^{12 13} Another report found hepatic vein thrombosis as a complication of extraintestinal amoebiasis, also known as Budd-Chiari syndrome.¹⁴ A recent review of three cases found inferior vena cava obstruction secondary to amoebic liver infection.¹⁵

The pathophysiology underlying vascular complications in amoebic infection is thought to be related to prolonged contact between vascular endothelial cells and amoeba, stimulating adhesion and procoagulant molecules. In the liver, hyperactivation of endothelial cells and exposure of subendothelial cells occurs, with later development of necrotic areas.¹⁶

No previous case reports have been identified involving portal vein congestion due to pyaemia secondary to hepatic amoebiasis. Throughout this case, the aetiology of the observed congestion generated much discussion. Unlike portal vein thrombosis, which is managed with anticoagulation, the underlying pathology of this portal congestion was proposed to be around parasite clumping and pyaemia, rather than clot formation. For this reason, anticoagulation was deemed unsuitable in this individual's case. The patient responded well to treatment of the *E. histolytica* infection and the portal vein congestion had cleared on repeat ultrasound imaging.

Routine treatment of amoebic liver abscesses commonly includes a course of 7–10 days of metronidazole 750 mg three times a day.¹⁷ Diloxanide furoate, an intraluminal amoebicide,

is often used in combination with metronidazole.¹⁸ A recent Cochrane review examined the evidence for percutaneous drainage and metronidazole in the treatment of amoebic liver abscess. Seven low quality randomised trials were included. The authors concluded that therapeutic aspiration in addition to metronidazole could not be supported or refuted by the present evidence. They concluded that further randomised trials are needed.¹⁹ The authors of this report observed that clinical and biochemical improvement were seen following percutaneous aspiration of 25 mL of pus from the largest of the patient's three liver abscess.

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Learning points

- Amoebic intestinal infection is an epidemic in the developing world and an important differential diagnosis for fever in the returning traveller, particularly in the context of gastrointestinal symptoms.
- When confronted with amoebiasis, clinicians should consider and exclude the possibility of extraintestinal infection, particularly liver abscess, as this is the most common manifestation.
- Portal vein congestion is a rare complication of amoebic liver abscess and may be due to pyaemia and parasite clumping rather than thrombosis. In this particular case, treatment of the infection alone, without anticoagulation, was found to relieve the congestion. There is a need for more research into the aetiology of this complication.
- There is a lack of conclusive evidence regarding percutaneous drainage as a treatment for amoebic liver abscess; however, in this particular case there appears to have been a benefit in draining the largest abscess.

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