Learning points from a case of severe amoebic colitis

Christina Petridou1, Adnan Al-Badri2, Anjana Dua1, Matthew Dryden1, Kordo Saeed1
1Microbiology Department, Hampshire Hospitals NHS Foundation Trust, Royal Hampshire County Hospital, Winchester, United Kingdom;
2Pathology Department, Hampshire Hospitals NHS Foundation Trust, Royal Hampshire County Hospital, United Kingdom

SUMMARY

A case of amoebic colitis and liver abscess is described in a previously fit 59-year old man who had been given the incorrect diagnosis of ulcerative colitis. His symptoms were so severe that a colectomy was being considered. The patient had a significant travel history including trips to Morocco, the Gambia and Cape Verde, putting him at risk of acquiring amoebic disease. However, this history was not ascertained until much later on in the disease process. The case highlighted crucial learning points including the importance of taking a lifelong travel history, the difficulties in telling ulcerative colitis and amoebic colitis apart both clinically and histopathologically, and the importance of sending multiple stool samples for parasitological microscopy analysis in patients being investigated for inflammatory bowel disease.

Keywords: amoebic colitis, liver abscess.

CASE REPORT

A 59-year-old man was admitted to Royal Hampshire County Hospital, a district general hospital in the United Kingdom, directly from the Inflammatory Bowel Disease (IBD) clinic on 15 January 2017 with worsening of his long-standing bloody diarrhoea, abdominal pain and weight loss. He had previously been diagnosed with ulcerative colitis (UC) in September 2016 following a flexible sigmoidoscopy and biopsy showing active chronic colitis. His symptoms at the time included gradually worsening bloody diarrhoea and abdominal pain and he was started on mesalazine in December 2016. His symptoms continued and on 7 January 2017 he began a reducing course of steroids. He had no additional medical history and was on no other medication.

On admission he was passing loose, bloody stools 8 times a day, his inflammatory markers were markedly raised and he had deranged liver function tests with a C-reactive protein of 263 mg/L, a white cell count of 25.6 10^9/L, an ALT of 102 U/L and an ALP of 336 U/L. He was tachycardic but other bedside observations were normal. He was diagnosed as having a flare-up of UC and started on intravenous hydrocortisone with a view to repeat sigmoidoscopy and starting infliximab. A stool sample sent on admission was culture negative, polymerase chain reaction (PCR) negative for Campylobacter, Salmonella, Shigella and Escherichia coli 0157 and negative for intestinal parasites on direct wet film microscopy and ethyl-acetate concentration.

His condition deteriorated with ongoing bloody diarrhoea and 5 days after admission a CT scan was performed due to his lack of improvement and deranged liver function. Imaging showed a large, septated liver abscess measuring 15 cm x 15 cm in his right lobe with pancolitis, and he was started on intravenous co-amoxiclav (Figure 1). A drain was inserted under ultrasound and no organisms were seen on microscopy of the pus and it was culture negative and pan-bacterial 16S PCR
negative. Due to the septated nature of his abscess only 250 mls was drained prompting a further attempt to re-site the drain on 24 January. He was transferred to the Intensive Care Unit 2 days later for on-going monitoring and total parenteral nutrition where he remained symptomatic with raised inflammatory markers. Repeat stool samples and hydatid and amoebic serology were advised by the Microbiology team and intravenous metronidazole added. By 25 January, the patient became agitated and confused and was intubated and ventilated and antibiotic therapy broadened to meropenem. A CT brain and lumbar puncture were unremarkable.

A liver biopsy was planned to assess whether there was an underlying malignancy and he was under consideration for a colectomy as he was not responding to mesalazine and steroids. Immunosuppressive therapy was not recommended due to his liver abscess.

16 days after admission the amoebic indirect immunofluorescent antibody test was reported as strongly positive. Hydatid serology was negative. Following the serology results, *Entamoeba* cysts were seen on a third stool sample using ethyl-acetate faecal concentration and PCR for *Entamoeba histolytica* was positive on the BD MAX Enteric Parasite Panel which is able to detect nuclei acids from *Giardia lamblia*, *Cryptosporidium* and *E. histolytica*. Although not validated PCR was also performed on his pus sample and was strongly positive for *E. histolytica*. The metronidazole was increased to 800 mg TDS and was followed by paromomycin. He continued on co-amoxiclav for 2 weeks as there was concern of super-added bacterial infection.

On repeat imaging 2 weeks after diagnosis the size of the liver abscess had improved significantly (Figure 2). He was transferred to the ward after 9 days in ICU and his drain removed 2 weeks later. His mesalazine was stopped following the diagnosis of amoebic colitis and his steroids weaned. His symptoms slowly improved and he was discharged after 9 weeks in hospital.

**Figure 1** - CT scan shows a large, septated liver abscess measuring 15 cm x 15 cm in his right lobe with pancolitis.

**Figure 2** - CT scan two weeks after diagnosis. The size of the liver abscess had improved significantly.
DISCUSSION

Amoebiasis is common in tropical countries and is caused by the parasitic protozoan *E. histolytica*. Humans become infected through the oro-faecal route. Presentations vary from asymptomatic to severe invasive amoebic colitis. Liver abscesses are the most common extra-intestinal complication and a delay in diagnosis may be life-threatening [1].

This case highlighted many important learning points. Firstly, due to similarities in clinical presentations, amoebiasis needs to be considered as a differential for IBD, especially if not responding to conventional therapy. This patient had been treated with steroids and mesalazine and had not improved, with immunosuppression and a life changing colectomy being considered. This case highlighted how severe amoebic colitis can be and how difficult it can be to tell the conditions apart clinically. Secondly, it is crucial to obtain a travel history. In industrialised countries *E. histolytica* is mainly restricted to returning travellers, immigrants, institutionalised settings where sanitary conditions may be poor and men who have sex with men [1]. *Entamoeba* infections accounted for only 1% of laboratory-confirmed cases of travel-associated gastrointestinal disease in the UK in recent years, with 14 confirmed cases in 2008. The main countries of travel were India and Pakistan however there was one case imported from Europe (Greece) [2]. Once the patient was extubated, a full travel history was taken and he reported visiting Cape Verde in November 2016, Morocco in 2015 and Gambia in 2014. He stated that his symptoms started after his return from Gambia although they were mild and he developed coping strategies. They progressed over the years becoming significantly worse in January, possibly coinciding with the introduction of steroids, which are known to be associated with more severe disease, culminating in his admission. An initial travel history may have prompted amoebiasis to be considered and appropriate microbiology samples to be sent. When amoebiasis is suspected clinicians need to be mindful that the patient’s life long travel history is explored.

Thirdly, although the sensitivity of stool microscopy is poor for the diagnosis of amoebiasis, the yield increases if multiple samples are sent due to the variable shedding of organisms. For patients with presumed IBD three stool samples should ideally be collected on alternate days to exclude the diagnosis of parasitic disease such as amoebiasis. Hydatid and amoebic serology should also be performed. The first stool sample ever sent for this patient was following his hospital admission and only one of three samples was positive for *E. histolytica* cysts. For complex patients such as these the early involvement of infection specialists is important to help guide appropriate microbiological investigations.

PCR for *E. histolytica* is not performed routinely in many diagnostic laboratories however if available, in-house testing for such patients could be an invaluable tool allowing rapid diagnosis in patients where amoebic disease is suspected or warrants excluding.

Apart from the clinical similarities between amoebic colitis and IBD, they can also be difficult to distinguish histologically. This patient had biopsies taken from the colon and rectum. These showed non-specific patchy active chronic colitis. There was no ulceration, granuloma formation or neoplasia seen. The histological changes in amoebiasis are non-specific and can be difficult to differentiate from other types of active colitis including IBD, drug-related inflammation, ischaemic colitis and other types of infections. The diagnosis of amoebiasis relies on the identification of the trophozoites, which may or may not be present in the surface exudate as round globules with a foamy cytoplasm but are difficult to differentiate from other surface debris like macrophages and sloughed epithelial cells. Amoebiasis is not a common cause of colitis in industrialised countries and if the number of trophozoites is small they can easily be missed. The histological detection of the infection depends on a high degree of clinical suspicion and on being given a thorough clinical and travel history.

In uncomplicated amoebic liver abscesses of less than 10 cm diameter in the right lobe, amoebicidal drugs alone should be adequate treatment [3-5]. This differs from pyogenic liver abscesses where surgery/drainage alongside antibiotics is optimal, however it can be difficult to distinguish them clinically. For larger abscesses however the criteria for medication alone versus medication plus percutaneous drainage, aspiration or surgery is unclear with some studies showing a shorter...
length of hospital stay and quicker resolution of symptoms in early aspiration groups and others showing that percutaneous catheter drainage is more effective [6, 7]. A systematic review from the Cochrane database is unclear on the advantages of aspiration (8). Therapeutic aspiration or drainage is generally considered if symptoms fail to resolve on medical therapy alone, when there is pyogenic superinfection and abscesses in the left lobe where there is high risk of rupture [5, 9, 10]. In this case the patient already had a drain in when the diagnosis was made. As his abscess was greater than 10 cm and a pyogenic superinfection was suspected, aspiration or catheter drainage would most likely have been required.

In conclusion, amoebiasis should be considered as a differential diagnosis in IBD due to the similarities in clinical presentation. Although rare in the UK and other industrialised countries, the rise in international travel and immigration means clinicians are increasingly likely to see this condition and patients may present to their general practitioner, medical or surgical hospital teams. In these complex cases a multidisciplinary approach with close liaison between the gastroenterology, microbiology, parasitology and laboratory teams is vital.

Conflict of interest
The authors declare that there is no conflict of interest. Patient consent was obtained for publication.

ACKNOWLEDGEMENTS
We would like to acknowledge Dr Murphy and his clinical team for managing this patient’s care.

REFERENCES