

Fulminant necrotising amoebic colitis: A diagnostic conundrum

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SUMMARY

Fulminant necrotising amoebic colitis is a complication of untreated amoebiasis. This is seen in mainly low-income countries. It has a high mortality rate and is difficult to diagnose. We present an extremely rare case of fulminant necrotising amoebic colitis that caused diagnostic confusion in mimicking an acute abdomen, presumably caused by a perforated duodenal ulcer.

KEY WORDS:

Fulminant, amoebic colitis, Entamoeba histolytica, acute abdomen, haematochezia

INTRODUCTION

Entamoeba histolytica is one of the commonest parasitic infections worldwide, infecting about 50 million people.¹ The World Health Organization suggests that this pathogen is responsible for 70,000 deaths annually, second only to malaria as the leading cause of death from a parasitic infection worldwide.²

In Malaysia, where there is a huge population of migrant workers it will become increasingly likely that this disease may present mimicking various other illnesses, such as our patient who presented with an acute abdomen. Therefore it is of paramount importance to approach such cases with a broad mindset of differential diagnosis.

CASE REPORT

A 41-year old male Burmese national, living in Malaysia for five months, working as a construction worker presented to the Emergency Department at Hospital Pulau Pinang with septic shock. He had no prior medical illness. At presentation he was intubated for airway protection in the emergency department.

Initial investigations in the intensive care unit diagnosed him as decompensated heart failure with atrial fibrillation and underlying pneumonia which was managed by the cardiology unit. Echocardiography demonstrated severe mitral stenosis with moderate mitral and tricuspid regurgitation. His ejection fraction was forty percent. In the intensive care he was started on intravenous hydrocortisone along with intravenous Vancomycin and Tazosin. Ten days later he was referred to the surgical team for an acute abdomen associated with haematochezia.

Clinically he appeared dehydrated and cachexic, his vitals were stable but temperature was 38.4°C. His abdomen was in generalised peritonitis. His white blood cell count was 14.5 10⁹/L, sodium 129 mmol/L, potassium 2.9 mmol/L and albumin was 19g/dL. Proctoscopy showed no haemorrhoids but evidence of blood mixed with faeces. A colonoscopy revealed severe colitis of rectosigmoid with slough.

Subsequently he underwent a contrast enhanced CT scan of the abdomen and pelvis, which showed pneumoperitoneum with dilated bowel loops and a liver cyst. No other lesions seen in the other solid organs. An emergency laparotomy revealed multiple perforations along the colon with gross necrotic contamination of the abdominal cavity. There was no duodenum perforation seen intraoperatively after the duodenum was Kocherised and examined. The free air seen in the retroperitoneum likely arose from the perforated colon. There was no liver abscess seen. He underwent a subtotal colectomy and end ileostomy.

Post operatively he was started on enteral nutrition feeds and treated with Meropenam and antifungal intravenous antibiotics for two weeks. He was nursed in the intensive care unit initially then transferred to the surgical ward for further rehabilitation.

Histopathological examination diagnosed fulminant necrotic amoebic colitis. Microscopically the specimen showed submucosal ulcers. He was discharged well post op day 18. Prior to discharge he was counselled for surgery of his underlying cardiac condition but requested for it to be done in Myanmar.

DISCUSSION

Amoebiasis is a parasitic infection caused by the protozoa *Entamoeba histolytica* and is the leading cause of death from parasitic disease worldwide.³ This infection is seen in under developed countries and communities with low socio-economic status living in areas of poor sanitation,⁴ as seen in our patient who had recently arrived from Myanmar and gave history of poor sanitation at his place of residence.

The protozoa most commonly affect the gastrointestinal tract and liver usually presenting with initial symptoms of abdominal pain and diarrhoea with bloody diarrhoea indicating progression to caecum, ascending or sigmoid colon.⁵ Our patient presented with symptoms of an acute

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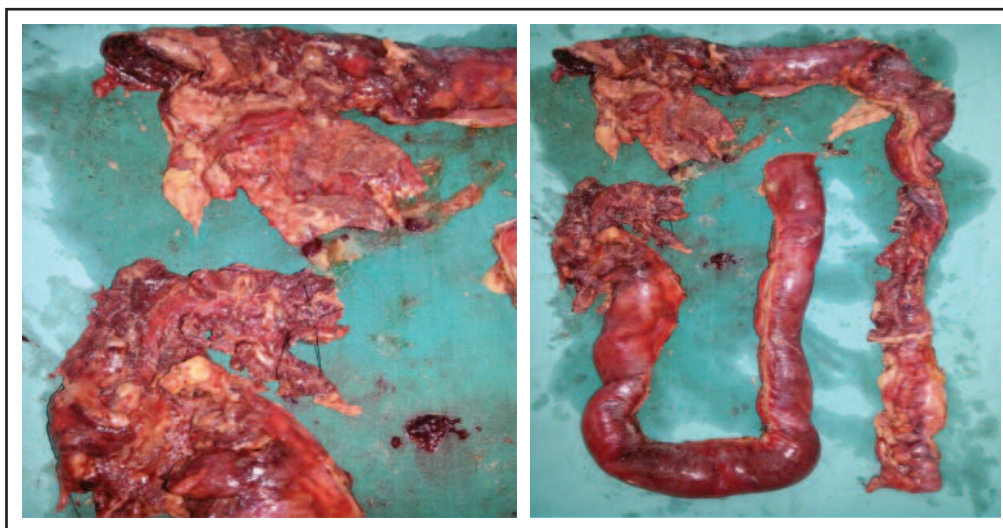


Fig. 1: Subtotal colectomy specimen with evidence of necrosis

abdomen coupled with bloody diarrhoea. On occasion a palpable mass in the right iliac fossa associated with pain may indicate an amoeboma.⁵ Extraintestinal manifestation is seen once the parasite gains entry to the portal circulation causing weight loss, right upper quadrant pain and fever.⁵ Upon review, this patient appeared cachexic and in sepsis, and taking into account his social history, a differential diagnosis of amoebiasis should be considered.

Most individuals who harbour the protozoa are asymptomatic throughout their lifetime. Approximately 6-11% patients will develop symptomatic infection which lead to a fulminant reaction causing necrotising colitis with perforation, peritonitis either due to a frank perforation, as seen in our patient, or slow leak through diseased bowel and possible death.⁵ Fulminant disease is associated with various factors including male gender, associated liver abscess, signs of peritonitis, leucocytosis, hyponatremia, hypokalaemia and hypoalbuminemia,² most of which was demonstrated at the point of referral. Such form of intestinal amoebiasis, termed as fulminant necrotising amoebic colitis, has only a few cases reported in literature. It is a rare complication and carries a high chance of mortality ranging from 55-87.5%.^{3,5}

Colonoscopy may provide some role for achieving the diagnosis of amoebiasis, provided a good biopsy is obtained as compared to other forms of colitis.¹ On initial review, a colonoscope was performed and a biopsy which subsequently was reported as colitis with evidence of amoebiasis. Computed tomography scan is the established gold standard in assessing the presence and complications of amoebic colitis and solid organ involvement.⁵ Extended submucosal ulcer with intramural dissection is characteristic of fulminant amoebic colitis and may also be seen with a flask-shaped ulcer.²

Amoebic colitis may be managed medically with metronidazole and diloxanide furoate for 10 days to eliminate luminal cysts.⁵ There is no role for conservative

treatment in fulminant necrotising amoebic colitis. In this case since the patient developed fulminant necrotising colitis the conservative option could no longer be applied. However, this patient was treated with metronidazole post operatively and diloxanide furoate was not given as this patient presented in a disseminated state. Early diagnosis and extensive surgical treatment is essential in reducing morbidity and mortality.³ Several authors have described various procedures including resection, diverting stoma and primary closure of perforation. In amoebiasis, primary anastomosis is contraindicated even in the absence of contamination.⁵ Complications that can occur after surgery are fistulae, including rectovaginal, colcutaneous, enterohepatic and cholecystocolonic.

CONCLUSION

Fulminant necrotising amoebic colitis is a life threatening condition and requires immediate intervention. One should always keep in mind this diagnosis after factoring in the social history and clinical presentation of patients. In Malaysia, with such a large number of migrant workforce, diseases not previously seen will eventually present itself after mimicking a variety of other illnesses. It is important to ensure appropriate history taking to aid in the diagnosis and prompt intervention to reduce morbidity and mortality of such illnesses.

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