

CASE REPORTS

Massive Lower Gastrointestinal Bleeding Due to Fulminant Necrotizing Amebic Colitis: A Diagnostic and Therapeutic Challenge

Sanjay Chandnani¹, Suhas Udgirkar¹, Samit S Jain², Nikhil Sonthalia³, Qais Contractor², Pravin M Rathi⁴, Anirudh Chapekar⁵

Abstract

Acute fulminant necrotizing amebic colitis rarely presents with massive life-threatening lower gastrointestinal bleeding without diarrhea. Diagnosis is difficult as colonoscopy is suboptimal due to active bleeding, stool testing is often negative and a positive serology cannot confirm the diagnosis. We herein report a case of a 39-year-old male who presented with profuse bleeding per rectum, without associated significant antecedent history of fever or diarrhea. Colonoscopy was inconclusive as active bleeding obscured the vision. Computed tomography of abdomen revealed non-specific thickening of the caecum. Emergency laparotomy with right hemicolectomy and temporary ileostomy was performed. Microscopic examination of colonic mucosa revealed *Entamoeba histolytica* trophozoites with erythrophagocytosis suggestive of fulminant amebic colitis. Intravenous metronidazole was given subsequently and patient recovered completely. Ileocolonic anastomosis was done after closing the ileostomy three months later. This case highlights this exceedingly rare presentation of fulminant amebic colitis which poses a diagnostic challenge and can be life threatening without early surgical intervention.

Introduction

Amebic colitis caused by *Entamoeba histolytica* is a common cause of diarrhea in the tropics.¹ Fulminant necrotizing amebic colitis (FNAC) is an uncommon but life-threatening complication which can lead to perforation, peritonitis, toxic megacolon, bloody diarrhea and a high mortality (40-89%) if not recognized early.² It is common in endemic areas, among travelers visiting endemic areas and is often misdiagnosed as Inflammatory Bowel disease initially. Even after appropriate antibiotics mortality rate remains high because of delayed diagnosis and systemic complications. We here report a rare case of FNAC presenting with acute massive life threatening lower gastrointestinal bleeding treated with emergency colectomy and antibiotics.

Case Report

A 39-year-old male presented with acute onset massive bleeding per

rectum for one day, associated with dizziness and altered sensorium. For two days prior to presentation he had history of low grade fever with increased stool frequency (3-4 per day). There was spontaneous passage of only fresh blood mixed with clots as well as passage of blood mixed stools on the day of presentation. There was no history of abdominal pain, peri-anal pain, straining during defecation, finger evacuation, increased defecation time, mass coming out of per rectum, weight loss, anorexia, urgency or tenesmus. There was no previous history of diarrhea, constipation, abdominal distension, or feeling of lump. Patient had pulmonary tuberculosis 3 years ago, which was treated with anti-tubercular drugs. He was a chronic alcoholic taking 30 gm alcohol/day since last 10 years. On examination

patient was drowsy with pulse rate 132/min, blood pressure 90/60 mm of Hg and had peripheral cool extremities. He had pallor with mild generalized abdominal tenderness with no evidence of any lump or organomegaly. On per rectal examination external skin tag was present with normal anal tone and finger was stained with blood. Proctoscopy revealed blood clots in the rectum. Patient was initially resuscitated with intravenous colloids, inotropes and moist oxygen inhalation. Laboratory investigation revealed hemoglobin of 5.2 gm%, leucocyte count of 22,500/cumm, serum Alanine transaminase of 65 IU/L, Aspartate transaminase of 60 U/L, Alkaline Phosphatase 280 IU/L and Serum creatinine 1.7 mg%. Serum anti-HIV antibodies were negative. Rest of the laboratory tests were unremarkable. After initial resuscitation, blood transfusion and hemodynamic stabilization, urgent colonoscopy was performed. There was poor visibility due to large amount of fresh and altered blood from caecum till rectum. Subsequently contrast enhanced computed tomography (CECT) of abdomen with angiography was done which revealed concentric, segmental, intramural wall thickening of ileocecal (IC) junction, caecum and proximal ascending colon (approximately 1.3 cm wall thickness) and inflamed appendix with pericolonic and peri appendiceal fat stranding (Figure 1 A, B). There were also discrete heterogeneously enhancing lymph nodes, about 10.9 x 11 mm in size in the right iliac fossa. There was no evidence of active contrast blush. There was no evidence

¹Senior Registrar, ²Associate Professor, ³Assistant Professor, ⁴Professor and Head of the Department, Gastroenterology, ⁵Associate Professor, Department of General Surgery, Topiwala National Medical College and BYL Nair Ch. Hospital, Mumbai, Maharashtra
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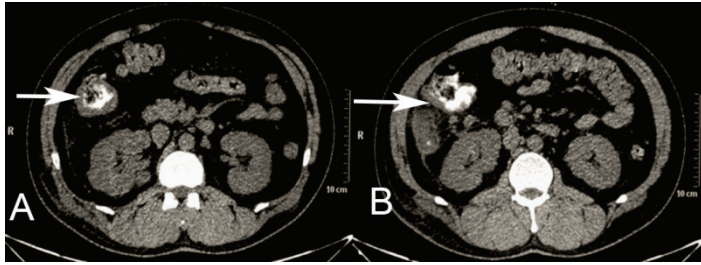


Fig. 1: (A, B) Computed tomography of abdomen showing caecal and ascending colon thickening (white arrow in A) with thickened appendix and surrounding fat stranding (white arrow in B)

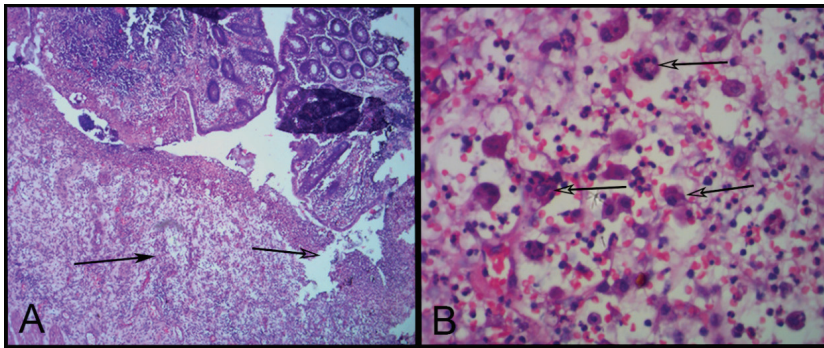


Fig. 3: (A, B) Microscopic examination of caecal ulcer edge biopsy under low power magnification (40x) using hematoxylin and eosin stain shows irregular ulcerated mucosa (open arrow in A) with submucosal inflammatory infiltrate (solid arrow in A). High power magnification (100x) shows trophozoites of *Entamoeba histolytica* with few of them showing ingested erythrocytes suggestive of characteristic erythrophagocytosis

of air under the diaphragm, perinephric air or paraspinal air suggestive of luminal perforation. Stool microscopic examination did not show evidence of ova, cyst or parasite. Serum ELISA for *Entamoeba histolytica* antibody was positive. In view of colonic thickening, persistent bleeding per rectum, hemodynamic instability and requirement of multiple transfusions (6 units in total), exploratory laparotomy was performed. Intra-operative findings revealed thickened IC junction with perforated retrocecal appendix and multiple ulcers in the caecum which was the site of bleed. Right hemicolectomy with ileostomy was done. Macroscopic examination of the resected colonic specimen showed multiple ulcers, largest of size 2.5 cm with necrotic base surrounded by edematous mucosa involving IC junction and caecum (Figure 2). There was an associated appendicular lump. Microscopic examination revealed caecal mucosa with a flask shaped ulcer with undermined edges. Mucosa was infiltrated with neutrophils and fibrin and there was a submucosal infiltrate of lymphocytes, plasma

cells, histiocytes and proliferating capillaries (Figure 3 A, B). Muscularis propria was normal and serosa showed dense fibrinous exudate. Occasional trophozoites of *Entamoeba histolytica* were seen with ingested erythrocytes suggestive of erythrophagocytosis (Figure 3 B). Subsequently he was treated with intravenous metronidazole (800 mg thrice daily) for 10 days. Post-operatively patient recovered gradually and oral feeds were started after 7 days. There was no further episodes of bleeding per rectum or drop in hemoglobin and he was discharged in a stable condition. After 3 months stoma was closed and ileocolonic anastomosis was done. He is doing well at 6 months follow up.

Discussion

Amebiasis is a water and food borne protozoal disease infecting as much as 10% of the world's population and is responsible for 40,000-100,000 deaths annually.² It mainly affects the colon and the liver. It has predilection for both sexes in childhood but males are affected more than females in adults. There is a bimodal age of distribution

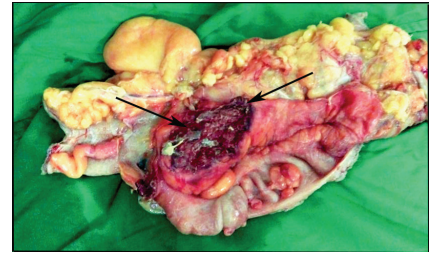


Fig. 2: Macroscopic examination of resected colonic specimen shows a large ulcer with necrotic base in caecum and adjacent ascending colon (black arrows)

with peaks at 2-3 years and 40 years.² Ameboma occurs in 1.5% of infected patients and carries a mortality of 0.5%.^{3,4} Stool routine microscopy is the most commonly employed diagnostic test which has a poor sensitivity of 25-60%. Antigen detection in stool is more sensitive but not widely available.² Fulminant necrotizing amoebic colitis affects 6-11% of symptomatic patients.² It can be complicated by microscopic sealed off perforation or can cause macroscopic perforation resulting in generalized peritonitis.² Mortality is high if not treated urgently.⁵ Ameboma is a mass of granulation tissue with peripheral fibrosis and a core of inflammation related to chronic amoebic infection usually found in the cecum and ascending colon. Colonization with other bacterium like *Clostridium* species, malnourishment, chronic alcohol intake, chronic corticosteroid use, male sex, age >60 yrs., associated liver abscess, abdominal pain, leukocytosis, hyponatremia, hypokalemia, hypoalbuminemia are factors associated with FNAC.⁶ All layers of the colon can be involved which can lead to toxic megacolon and perforation. Close differential diagnosis includes severe attack of IBD, carcinoma colon, other infectious colitis and ischemic colitis. Misdiagnosing and treating it as IBD with steroids is common in both non-endemic and endemic regions, which can be fatal.⁷ Diagnosis is often difficult and is usually made by microscopic identification of amoeba on colonoscopic biopsies or surgically resected specimens. Macroscopically they form large geographic ulcers with yellow green pseudo-membranes. On microscopy, muscularis externa may become attenuated and necrotic resulting in wet blotting paper consistency. Chen et al in their study of 15 patients of

FNAC found favorable results and survival in the surgical group.² All patients (6/6) survived after surgery, whereas only 3 of 9 patients survived in the conservative management group in their study.² Stein and Bank suggested some indications for surgery which included no improvement on intravenous antibiotic therapy, severe abdominal tenderness, abdominal distension, perforation with or without abscess, toxic megacolon with hypoproteinemia and anemia, bowel obstruction due to ameboma, ileocecal intussusception secondary to amoeboma.⁸ These observations require further validation. In our case refractory lower GI bleeding was the indication for surgery and subsequently FNAC was diagnosed. Abdominal CT scan is instrumental to rule out perforation. However, in our case abdominal CT revealed concentric intramural thickening of terminal ileum, cecum, proximal ascending colon with inflamed appendix. There was no evidence of air under the diaphragm, retroperitoneal air or paraspinal air pocket on CECT abdomen suggestive of perforation. Perforated retrocecal appendix was found intraoperatively.

This highlights the need for high index of suspicion and low threshold for surgical exploration in cases of FNAC. The treatment of ameboma includes antibiotics and agents for eliminating intestinal cysts. Early surgery is life saving and it is to be performed as two staged procedure. Primary resection anastomosis is contraindicated because of high risk of suture breakdown.^{2,9,10} There is high incidence of gangrene with anastomotic leaks if resection anastomosis is done primarily.¹⁰ Early surgery includes aggressive resection of bowel and exteriorizing the bowel ends i.e., right hemicolectomy with ileostomy and mucus fistula. Hartmann's procedure has been advocated by some in elderly toxic patients.²

In conclusion, acute FNAC presenting as massive lower gastrointestinal bleed without significant antecedent diarrhea is an exceedingly rare complication of intestinal amebiasis. Pre-operative diagnosis is often difficult as stool testing is often negative and colonoscopy is suboptimal in view of active bleeding. High degree of clinical suspicion and timely operative

intervention is critical in managing this life threatening condition.

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