

Fulminant Amoebic colitis: A Rarity in the Pediatric Population

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Abstract

Although amoebiasis is a common parasitic infection, fulminant amoebic colitis remains a very rare complication, especially in the paediatric age group. It is necessary to diagnose this entity as it is associated with high morbidity and mortality and timely intervention may be proved to be life-saving.

Keywords: Amoeba; Perforation; Colitis

Introduction

Amoebiasis is a common parasitic infection affecting upto 10% of the world's population. Despite it being a common condition, a very small percentage of individuals become symptomatic and less than 0.5% of them develop invasive disease [1]. We report a case of 5 year boy with fulminant amoebic colitis which is an extremely rare manifestation of amoebiasis, especially in pediatric population.

Case Report

A 5 year boy, previously well had come with an acute onset of fever, dysentery, abdominal pain followed by distension for 5 days prior to admission to our institution. On examination, he had abdominal distension, guarding, rigidity and sluggish bowel sounds. X ray abdomen was suggestive of air under the diaphragm and ultrasound abdomen revealed a sealed off caecal perforation with collection of fluids and typhlitis. In view of high grade fever and abdominal pain, a diagnostic possibility of enteric fever was considered and the Widal test was also positive with H titres >1:320 though the major oddity was the perforation in the first week of illness. The per-operative findings were suggestive of multiple perforating ulcers in the ascending and transverse colon with a large caecal perforation and fecal peritonitis. A right hemicolectomy was done and the ileal end was brought out as a stoma in the right iliac region with a distal mucosal fistula for decompression.

Grossly, a right hemicolectomy specimen was received in 3 fragments separating the terminal ileum along with caecum and appendix, ascending colon and part of transverse colon. The respective measurements of terminal ileum, appendix and caecum were 4 cm, 3 cm and 4 cm respectively. The ascending colon measured 8 cm and the transverse colon measured 7 cm in length. Grossly, there was diffuse ulceration involving whole of the terminal ileum and large bowel with only occasional area of preserved mucosa standing out as pseudopolypi (Figure 1A). The ulcer base was shaggy and necrotic and a few showed undermined edges. These ulcers were deep and showed perforation at a few foci concealed and sealed off by the serosa. In most of the places the serosa was dull and also showed exudates apart from a few patches where congestion was also noted in addition. A perforation measuring 1.5 cm in diameter was noted in the ileocaecal region involving the anti-mesenteric border surrounded by erythematous mucosa. None of the segments showed any gross luminal dilatation. It is noteworthy to mention the ulcers in this index case was diffuse, not delimited by the ileocaecal valve and was circumferential thereby not pertaining to the typical morphology of an ulcer of enteric fever or tuberculosis which typically show longitudinal or transverse ulcers respectively. Moreover, unlike Cröhn's disease, no skip area, creeping fat or fistula was noted.

Ulcerative colitis is rare to cause transmural involvement and in the index case the rectosigmoid was spared.

On microscopy, diffuse deep ulcers were noted involving the whole of the resected bowel with dense inflammatory granulation tissue at the ulcer base, at places extending transmurally with myonecrosis and myolysis and causing severe acute serositis (Figure 1B). Peyer's patches were seen in the terminal ileum and the appendix though these ulcers were not really sitting at the top of these Peyer's patches. The inflammatory reaction at the top and base of these ulcers were paucipolymorphonuclear and predominantly composed of lymphocytes and plasma cells along with nuclear debris entrapped within fibrin-rich exudate. On higher magnification numerous singly scattered amoebae were seen ranging in size from 30-60 μ with centrally to eccentrically placed nuclei, prominent karyosome and abundant vacuolated cytoplasm some of which contained ingested erythrocytes (Figure 1C). These protozoan structures were highlighted on performing PAS (per-iodic acid Schiff) and Masson trichrome stains (Figure 1D). PAS stain also highlighted the protozoan profiles traversing through the muscularis propria and reaching in the serosa. Candidal colonization was noted over few of these ulcers. No evidence of activity or chronicity was noted in the adjacent mucosa. Taking into account the gross and microscopic findings a diagnosis of amoebiasis causing perforation and serositis was rendered.

The child developed encephalopathy on day 2 of the post-operative period and a possibility of septic encephalopathy was thought after ruling out other metabolic causes. The blood culture at this point of time showed growth of *Klebsiella pneumonia* that was negative pre-operatively. The CT scan of the brain was normal and the patient recovered from the encephalopathy following antibiotic treatment. The child was initially on Ceftriaxone at 50 mg/kg/dose twice a day. Post surgery he was also started on Metronidazole at 30 mg/kg/day in three divided doses which he had received prior to the collection of the biopsy report following which he was given a higher dose of metronidazole at 45 mg/kg/day in three divided doses for 10 days. Amoebic serology

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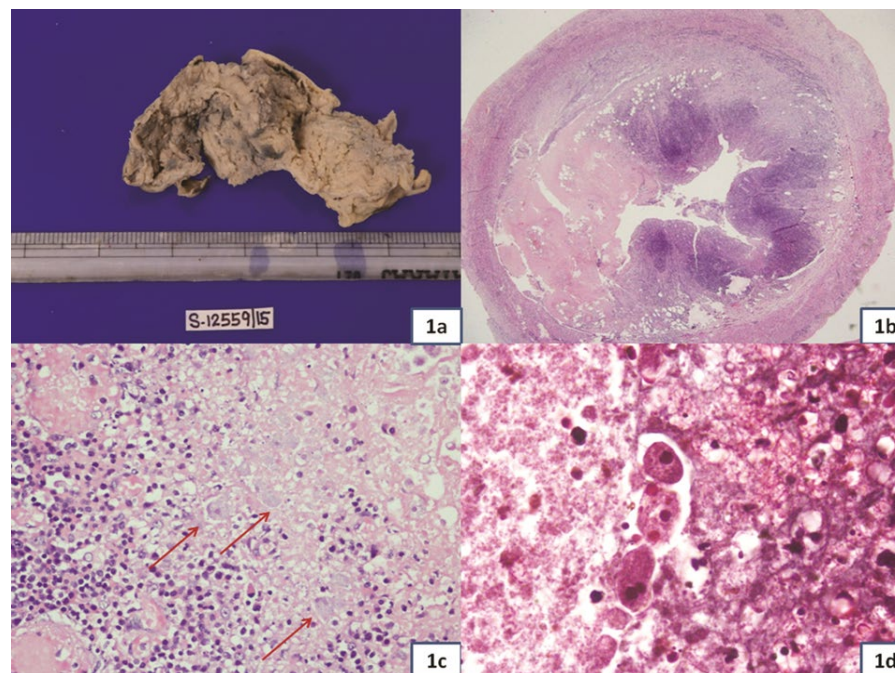


Figure 1 A: The gross photograph of the terminal ileum and colon shows diffusely ulcerated mucosa with shaggy and necrotic ulcer base.
B: Scanner view photomicrograph of the appendix shows deep ulceration of the appendix (left side) with necrotic ulcer base sitting over the muscularis propria (20x)(Hematoxylin and eosin stain).
C: Numerous amoebae are seen in the necrotic material that are larger than the macrophages and show prominent karyosome (400x)(Hematoxylin and eosin stain).
D: The amoebae are highlighted by the Masson trichrome stain with prominent karyosomes and ingested erythrocytes (1000x)(Masson trichrome stain).

(IgG for amoebiasis) was strongly positive. Microscopy examination of the ileostomy output was negative for cysts, which was expected as he had already received one week metronidazole and the sample was from the unaffected portion of the gut.

Discussion

Amoebiasis is the second most common cause for mortality among parasitic infections caused by *Entamoeba histolytica* [2]. The word “histolytica” stands for eating up the tissue which was vividly demonstrated in the index case. 90% of the individuals are asymptomatic and only 0.5% of them have a risk of developing invasive disease and fulminant colitis. It is usually common in immunocompromised individuals, pregnant women and malnourished children [3]. Most common symptoms involve the gastrointestinal tract like nausea, vomiting, abdominal distension and pain in about 10-35%. Amoebic liver abscess is rare and even rarer is cerebral abscess or pericarditis. The child in the index case was doing well prior to this illness. The major challenge in this case was a positive Widal test with high titres which could explain both the perforation and encephalopathy however caecal perforation in the first week multiple colonic perforations were the significant oddities. It is noteworthy that elevated H titres in Widal are not specific for an active infection. The on-table findings were multiple perforations that may be seen in inflammatory bowel disease. However the age of the child, with no history of bowel disturbances in the past were the odd points for IBD. A study of 739 cases of inflammatory bowel disease from Great Britain showed that only 4% of the study population less than 5 years of age became symptomatic and the mean age of diagnosis was 12.6 years [4]. A completely asymptomatic presentation of an inflammatory bowel disease is unheard of. A recent

North Indian study showed around 8% of the patients undergoing emergency exploratory laparotomy was diagnosed of amoebic colonic perforation [5], though none of the patients in this cohort was a child, thereby demonstrating the rarity of the index case scenario.

Diffuse ulceration of the ileum and colon with perforation and serositis can occur due to inflammatory bowel disease, ischaemic colitis, vasculitides and infective causes. In this context, gross and microscopic examination of the vessels is an extremely important adjunct to a proper diagnosis. The index case did not show any vascular alteration either in gross or in microscopy. Moreover, a diffuse ulcer with shaggy necrotic base suggests an infective/ inflammatory origin rather than a vascular insult. Ulcerative colitis is a disease that mostly involves the rectosigmoid although ileocaecal region can be involved, however complete sparing of the descending colon and rectosigmoid should arouse the suspicion of an alternative diagnosis. Moreover, a transmural involvement is rare in the setting of ulcerative colitis, though it can happen in toxic megacolon. The index case however did not show any luminal dilatation or thinning of the wall precluding a gross diagnosis of toxic megacolon. Crohn’s disease involves the small and large intestine in patches with typical skip areas and transmural involvement is also common. In the index case, no skip area, creeping fat or fistula was noted. Microscopically, there were no features of activity or chronicity to suggest a possibility of inflammatory bowel disease.

It is important to rule out an infectious colitis in the form of coinfection or superinfection prior to the beginning of steroid therapy in a suspected case of inflammatory bowel disease to prevent its devastating effects. The definitive diagnosis is hence based on the

histopathological examination of the surgical specimen. This again highlights the importance of having a suspicion of amoebic colitis even in such rare presentations as an early surgical intervention and appropriate amoebicidal agent can be life saving in these children. Fulminant colitis, toxic megacolon complicate less than 0.5% of cases, whereas multiple colonic perforations have been reported in upto 75% of cases described by Chen et al. [6]. Fulminant amoebic colitis had been reported by different authors in different time periods and the incidence had naturally reduced in the era of newer drugs. Different large case series had mentioned the occurrence of fulminant amoebic colitis in adults as well as occasional case reports of infantile occurrence had also been reported. However, the literature is mostly silent about fulminant amoebic colitis in paediatric population except for occasional case reported in the series of Eggleston et al. and Nisheena et al. [7,8]. Moreover in pediatric age group presenting with fulminant colitis and perforation, with no previous history suggestive of an ongoing infection has not been reported so far to the best of our knowledge.

Amoebiasis, as mentioned can be associated with rarer complications such as amoebic brain abscess. The child continued to be afebrile from day 5 of hospital stay with no signs of raised ICP or mass effect which made any evolving abscess less likely. Encephalopathy was in the improving trend and hence septic encephalopathy was considered.

Surgical management is warranted in cases of fulminant colitis, peritonitis and abscess formation. Male gender, peritonitis, hypoalbuminaemia and electrolyte imbalance are some poor prognostic factors in fulminant colitis. Early surgical intervention in such cases have been found to decrease the mortality which is 55-87.5% on conservative management [9]. A Cochrane study in 2009 highlights the advantage of additional luminal amoebicide treatment for clearance of cysts [3].

We would mainly like to emphasize the importance of considering amoebiasis in any child with intestinal perforation, as early surgical intervention and anti amoebicidal agents can be lifesaving in such children. Despite its rarity, especially so in the era of newer antibiotics, the possibility should always be considered in a child with symptoms and signs not really fitting into the commoner causes. It is also essential to screen and administer metronidazole in the siblings and parents and to educate them regarding hygiene measures.

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