CASE REPORT

Diffuse perforated necrotising amoebic colitis with histoplasmosis in an immunocompetent individual presenting as an acute abdomen

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SUMMARY

Perforated necrotising amoebic colitis associated with intestinal histoplasmosis has rarely been reported in an immunocompetent individual. Radiology and preoperative features are non-specific and requires histopathological examination for a definitive diagnosis. Hence, this condition needs to be considered in the differential diagnosis of complicated infective colitis.

BACKGROUND

Diagnosis of complicated and perforated amoebic colitis could be a challenging task in routine clinical practise. Here, we report a rare case of diffuse necrotising perforated amoebic colitis with atypical clinical presentation and unusual association with histoplasma infection.

CASE PRESENTATION

A 62-year-old man, Asian Indian, a known case of hypertension, hypothyroidism and prostatomegaly, presented with abdominal distension, urinary retention and obstipation of 3 days. He gave a history of urinary retention 3 days previously for which he was catheterised. Following this, he developed abdominal distension. There was a history of altered bowel habit, constipation and crampy abdominal pain. There was no history of nausea, vomiting, diarrhoea, fever or weight loss. He was an ex-alcoholic, non-smoker, non-diabetic and he denied any high-risk behaviour. On physical examination, he was normothermic with normal haemodynamics. Abdominal examination revealed tense abdomen, with diffuse tenderness more on the right half of the abdomen and no appreciable

organomegaly. Personnel and family histories were unremarkable in context to the present illness. He consumes a mixed diet comprising of both vegetarian and occasional non-vegetarian.

INVESTIGATIONS

Laboratory results showed haemoglobin of 12.4 g/dL, total leukocyte count of $15.5 \times 10^9/L$ with the differential of 88 polymorphs and 12 lymphocytes. Erythrocyte sedimentation rate of 52 mm at the end of first hour. Liver and kidney function tests were within normal range. HIV, hepatitis B and C virus serologies were non-reactive. Stool analysis was negative for occult blood and parasite. Routine urine examination was within normal limit. His plain chest X-ray was within normal limit. Abdominal X-ray showed dilated bowel loops. Ultrasonography of abdomen showed minimal free fluid with no organomegaly; dilated bowel loops were identified with no exact localisation. Contrast-enhanced CT of the abdomen showed dilated small intestinal loops, intramural oedema of terminal ileum and diffuse hypodense mural thickening of caecum, ascending colon and hepatic flexure (figure 1).

DIFFERENTIAL DIAGNOSIS

A clinical diagnosis of colonic malignancy or colonic involvement by an infective pathology was considered.

TREATMENT

As the patient had presented with acute problem, he was taken up for emergency laparotomy. He was found to have evidence of hollow organ

Figure 1 Contrast-enhanced CT abdomen showing intramural oedema of terminal ileum, diffuse hypodense mural thickening of caecum and ascending colon.

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Figure 2 Gross photograph of the resected specimen showing thinned out pale caecal and colonic mucosa covered diffusely by greyish necrotic slough. Terminal ileum and ileocaecal valves are also seen.

perforation diffusely inflamed and thickened colon, a right hemicolectomy with end ileostomy was carried out.

OUTCOME AND FOLLOW-UP

Following the histopathology report, the patient was treated with intravenous tazocin 4.5 g and metronidazole 500 mg 8 hourly. He was also given intravenous amphotericin B (500 mg) and itraconazole (200 mg) once daily for 15 days. Possibility of any other focus of possible histoplasmosis had been excluded. Patient had full recovery in a month's time and he was discharged from the hospital. On follow-up visits in subsequent months (October, November, December 2012), he was doing well and asymptomatic.

Pathology of the resected specimen: gross examination showed diffusely dilated colon with thinning involving caecum, proximal ascending colon, ileocaecal valve and terminal ileum. Mucosa in these areas was pale and friable. Three perforations measuring between 5 and 12 mm sizes were identified in caecum, covered by fibrinopurulent exudate over the pericaecal fat. Remaining colonic

Figure 3 Photomicrograph showing deep flask-shaped ulcer with overhanging margin containing necrotic inflammatory cell exudate. The submucosal is expanded by fibrosis, inflammatory cell and fat infiltration. A part of the adjoining normal colonic mucosa could also be seen (H&E, ×10).

Figure 4 High-power photomicrograph of the exudates to show an entamoeba histolytica trophozoite with engulfed red blood cells (H&E, ×100).

mucosa showed oedema and congestion (figure 2). Microscopy: there were multiple deep mucosal ulcers having overhanging margins, giving flask-shaped appearances along with transmural necrotising inflammation (figure 3), extending and involving the pericolic fat with pericolic abscesses. The deep ulcers contained fibrin rich-inflammatory cell exudate consisting of degenerated cells, nuclear debris, macrophages, neutrophils, lymphocytes and eosinophils. Ulcer beds showed inflammatory granulation tissue. The exudates contained many round to oval bodies measuring between 20 and 80µm, conforming to the morphology of trophozoites of *Entamoeba histolytica* with engulfed red blood cells (figure 4). These bodies were brightly peroxidase acid Schiff positive, occasional ones had vacuolated intracytoplasmic spaces (figure 5A,B). The deeper abscess cavity also contained several much smaller encapsulated round bodies measuring 4–5 microns conforming to yeast form of histoplasma capsulatum (figure 6A,B). Regional lymph nodes showed reactive hyperplasia. The appendix was grossly and microscopically within normal limit. A morphological diagnosis of diffuse necrotising amoebic colitis with multiple caecal perforations and colonic histoplasmosis was made.

DISCUSSION

Hippocrates noticed that dysenteries were especially bad when they present with fever, intestinal discharge of mixed character or with inflammation of the liver.¹ Infective colitis like amoebic colitis can be severe and fatal, the clinical spectrum can range

Figure 5 High-power photomicrograph to show many peroxidase acid Schiff (PAS) positive trophozoites of *Entamoeba histolytica* intermixed with the inflammatory cell exudates (PAS, ×40).

Figure 6 High-power photomicrograph to show the brightly peroxidase acid Schiff (PAS) positive trophozoites highlighted by the red arrows. Some of them also have vacuolated cytoplasm (PAS, ×40).

Figure 8 High-power photomicrograph of the abscess to show many encapsulated budding yeast cells and many intracellular forms could be identified (Grocott-Gomori methamine silver stain, ×100).

from asymptomatic state to mild form with abdominal pain, fever or diarrhoea to severe state where the patient may present with gastrointestinal bleed or colonic perforation.^{2–4} Necrotising form of amoebic colitis complicated by perforation is rare and associates with high mortality.⁵ Peritonitis in the background of perforated amoebic colitis tends to be associated with fatal outcome.^{6–7} Upto now, more than 100 such cases have been reported in the English literature (PubMed search). Gastrointestinal primary histoplasmosis has been reported both in immunocompetent and immunocompromised individuals, but more frequently in AIDS.⁸ In a recent publication in series of 77 patients, three quarters of the patients were immunocompetent.⁹ Our index patient was tested negative for HIV, and also not associated with malignancy, chemotherapeutic treatment, diabetes or use of immune suppressive drugs.

Intestinal histoplasmosis per se, in an immunocompetent individual is a rare condition (figure 7). The incidence is still unknown, as there are only few isolated case reports in patients with undetermined immune status.^{10–12} In *in vitro* studies, amoebic parasites have been observed to increase the virulence or pathogenic property of histoplasma.¹⁰

To conclude, we report a rare case of complicated amoebic colitis associated with histoplasmosis (figure 8). Our patient is the second reported case in the world literature. This unusual condition needs to be considered in differential of severe colitis in tropical countries like India, Bangladesh and parts of Africa.

Figure 7 High-power photomicrograph of the content of the abscess showing many round encapsulated yeast form of histoplasma capsulatum highlighted with red arrows (peroxidase acid Schiff (PAS), ×100).

Learning points

- ▶ Diffuse necrotising amoebic colitis complicated by perforation though rare, needs to be excluded in all cases.
- ▶ Superadded or associated infection by any other organism needs to be ruled out in all cases of amoebic colitis.
- ▶ Intestinal histoplasmosis may be observed in any situation including immunocompetent patients.

Contributors KV and RKB were involved in working up the case for the final diagnosis. RG performed the clinical work-up and operated on the patient. He also had monitored the patient postoperatively; and follow-up of the patient at out patient door was carried out. Manuscript writing was equally contributed by all the authors.

Competing interests None.

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