CASE REPORT

Invasive Mucormycosis: An Unusual Cause of Intestinal Gangrene in an Immunocompetent Adult

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Abstract

Invasive mucormycosis is an opportunistic fungal infection that commonly involves the rhinocerebral and pulmonary system. We are presenting a rare case of small intestine invasive mucormycosis causing multiple perforations and gangrene in the small intestine. Mucormycosis infection occurred after traumatic perforation peritonitis. Mucormycosis usually occurs in the presence of predisposing conditions like uncontrolled diabetes mellitus, neutropenia, underlying malignancy, or those receiving immunosuppressive agents. Gastrointestinal mucormycosis is uncommon and seldom diagnosed in living patients. In these cases, diagnosis is delayed and the mortality rate is high.

Keywords: Gangrene, Ileum, Mucorales, Mucormycosis, Perforation.

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Introduction

Mucormycosis is a rare infection caused by fungi of the order Mucorales. The infection occurs in the rhinocerebral, respiratory, gastrointestinal, or cutaneous regions depending on the portal of entry. It is found more commonly in immunocompromised adults.[1] We present a rare case of the small intestine gangrene due to invasive mucormycosis following traumatic jejunal perforation peritonitis. The patient was not having diabetes, any malignancy, and other immunocompromised state which are commonly associated with fatal mucormycosis infection.

Case Description

A 35-year-old male had a road traffic accident and sustained blunt abdominal injury. The patient was initially evaluated and resuscitated at the Primary Health Center and referred to our institute after 2 days of the injury. He had a pulse rate of 110/minute, BP 100/76 mm Hg, respiratory rate 22/minute, and was febrile on presentation. His abdomen was distended, and diffuse guarding was present. He was resuscitated with intravenous fluids, and broad spectrum antibiotics were started empirically covering gram-negative and anaerobic organisms. On investigations, he was found to have a low leukocyte count, acidosis on blood gas analysis, and deranged renal function. X-ray of the abdomen showed pneumoperitoneum. The patient was resuscitated and ionotropic support was given because blood pressure did not improve after fluid resuscitation and underwent exploratory laparotomy. Intraoperative findings revealed gross contamination with 3 L of bilious fluid in peritoneal cavity and 2×2 cm perforation on anti-mesenteric border of the small intestine and 110 cm distal to the duodenojejunal flexure. The rest of the abdominal viscera were normal. A thorough peritoneal lavage was done and a loop stoma was made through the same perforation. In the postoperative period, the patient was kept on mechanical ventilator and inotropic support. The patient gradually improved and inotrope was stopped on the postoperative day 4. The patient was extubated on postoperative day 7. The patient remained febrile in the postoperative period. Repeated bacterial blood cultures were sterile. The patient had a bilious discharge from the main wound on day 8, which increased on the next day and the stoma stopped functioning. The patient was reexplored. Laparotomy revealed 2 L of biliopurulent fluid in the peritoneal cavity with perforations and gangrenous segments in the proximal part of the small bowel (Fig. 1). The pulsation of the superior mesenteric artery and the mesenteric vessels was normal. Starting approximately 60 cm distal to duodenojejunal flexure, 120 cm of gangrenous and multiple perforation bearing segment of jejunum and proximal ileum was resected. End jejunostomy and distal mucus fistula were constructed. The resected bowel wall was thinned out and serosa showed multiple fibrinous exudates. On cutting open, the mucosa was found to be congested and the bowel contained blood mixed with fecal content. The patient died on the second postoperative day. Microscopic examination of sections from the perforation margin showed transmural hemorrhagic necrosis bordered by acute necrotizing inflammation. The base of the ulcer as well as the vessels in the lamina propria showed invasive aseptate fungal hyphae consistent with the morphology of *Mucor* (Fig. 2). The serosa showed evidence of acute serositis with the presence of vegetable matter and fungal hyphae. The mesentery also showed acute necrotizing inflammation along with the fungal hyphae consistent with *Mucor*. A few areas showed fibroblastic proliferation. Resection from both the margins showed viable mucosa but had serositis.

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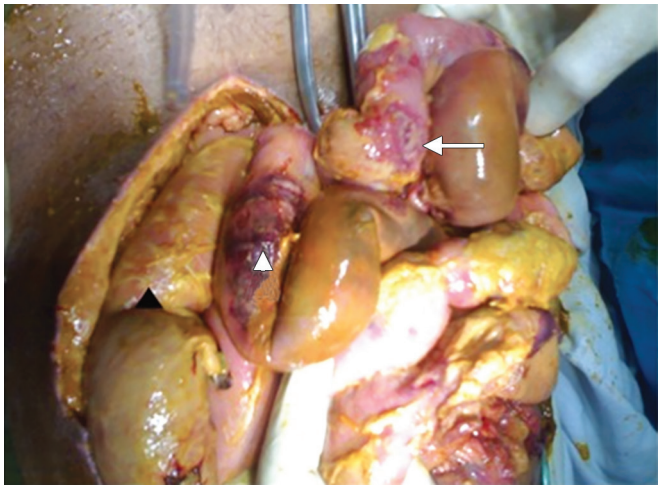

Fig. 1: Laparotomy showing perforation (arrow) and areas of gangrenous small bowel (white arrow head). The bowel is covered with fibrinous flakes (black arrowhead)

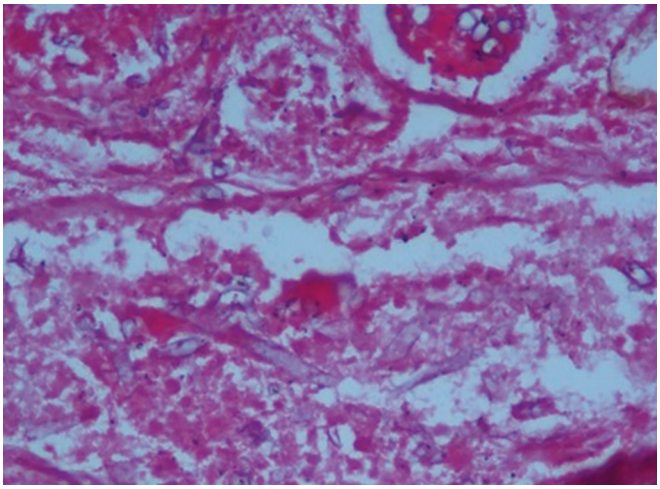

Fig. 2: High-power photomicrograph showing necrotic tissue with large easily foldable fungal profiles, some of which are also visible within the blood vessel leading to angioinvasion (H and E)

Discussion

Mucormycosis is a life-threatening fungal infection caused by a ubiquitous fungus belonging to the order Mucorales of the class Zygomycetes. These organisms are found in the soil and decaying organic matter. The genera causing disease in humans include *Mucor*, *Rhizopus,* and *Absidia*.[1] Mucormycosis is more common in immunocompromised adults particularly in case of diabetic ketoacidosis (especially associated with rhinocerebral mucormycosis), lymphoma, leukemia (especially associated with disseminated disease), and renal failure on peritoneal dialysis, allogeneic bone marrow transplantation, and solid organ transplantation (associated with pulmonary, gastrointestinal and disseminated disease).[1] Mucormycosis of the gastrointestinal tract is rare and possibly accounts for around 8% of all cases of mucormycosis and is associated with a mortality of around 50%.[2] It is difficult to diagnose. In particular, gastrointestinal mucormycosis has been seen in premature neonates, often in association with widespread disseminated disease. Rare cases of gastrointestinal mucormycosis have been described in association with other immune-compromising conditions, including AIDS, systemic lupus erythematosus, and organ transplantation.[2] In a recent systematic review on gastrointestinal mucormycosis, most of the reported cases were from Asia. Although gastric mucormycosis may be easy to diagnose, a recent review suggests that the stomach is the commonest organ involved in adults, whereas intestinal involvement is more frequent in children.[2] Though rare, gastrointestinal mucormycosis has been also reported in posttraumatic peritonitis.[3] In the present case, the patient also had peritonitis due to jejunal perforation following the blunt trauma abdomen. The diagnosis of intestinal mucormycosis was a histological surprise from intestinal biopsy. Unfortunately, there are no serologic tests for rapid diagnosis, and autopsy series have reported that up to half the cases of mucormycosis are diagnosed after death.[4] Gastrointestinal mucormycosis is uncommon and seldom diagnosed in living patients. In such cases, diagnosis is delayed and the mortality rate is as high as 85%.

The key to improved outcomes is early recognition of the infection which is difficult in these patients. The presence of mucormycosis must be considered especially in unexplained gastrointestinal ulcers, masses, and gangrene. The key to successful therapy is treatment with a combination of amphotercin B and surgical debridement.[2] Since the diagnosis of gastrointestinal mucormycosis seems to be increasing even in the immunocompetent individuals, the index of suspicion must remain high.

References

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