Introduction
Mucormycosis is caused by environmental fungi of the class Zygomycetes, subphylum Mucoromycotina and order Mucorales and mainly by species of genera Rhizopus.[1] It mostly affects patients with uncontrolled diabetes, defects in phagocytes and organ or hematopoietic stem cell transplantation[2,3] Gastrointestinal mucormycosis is an uncommon entity associated with high mortality. Diagnosis is established by biopsy. Within the gastrointestinal tract, stomach is the most common site of involvement followed by that of colon.[4]

Case Report
Gastrointestinal (GI) mucormycosis is uncommon but has high mortality despite aggressive treatment.

A 65-year-old male patient, with chronic obstructive airway disease on inhalation steroid treatment, nondiabetic, nonhypertensive, presented with the complaints of 8–10 episode of painless fresh bleeding per rectum. On admission, the general condition of the patient was poor with systolic blood pressure of 70 mm of Hg. His systemic, examination was normal except per rectum examination, that showed rectum full with blood clots. Laboratory examination revealed his Hb-9.1, total lymphocyte count of 2380/mm³, platelet count 1.50 lakhs/mm³, peripheral smear showed mild lymphocytosis, serum creatinine-2.9 mg/dl, Na-137 mEq/L, K-2.8 mEq/L, serum bilirubin-0.8 mg/dl, serum AST 132 IU/L, ALT 99 IU/L and INR 1.14, random blood sugar-130. He was started on intravenous antibiotics, fluid, and other supportive measures. His ultrasonography abdomen was done suggestive of fatty liver with cholelithiasis. His upper GI endoscopy was normal, colonoscopy showed circumferential deep ulcers in cecum, ascending, and sigmoid colon with active oozing that was controlled with argon plasma coagulation [Figure 1]. Multiple biopsies were taken from the colonic ulcers to ascertain the etiology. Histopathology of ulcer biopsy was suggestive of angioinvasive mucormycosis [Figures 2 and 3] so he was started on amphotericin-B and posaconazole. Despite ongoing antifungal and antibiotic treatment, the patient had worsening clinical course. Later on, he required ventilator support and multiple dialyses and finally he succumbed.

Discussion
Mucormycosis mostly affects patients with uncontrolled diabetes, defects in phagocytosis and organ or hematopoietic stem cell transplantation.[2,3] Incidence of mucormycosis has also increased because of frequent use of antibiotics and steroids.

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Histopathological examination remains the most sensitive and specific modality for definitive diagnosis. Biopsy reveals characteristic wide (6–30 µ), thick-walled, ribbon-like, aseptate hyphae that branch at the right angles.

Based on the involvement of a particular anatomic site mucormycosis is divided into five categories (1) rhinocerebral, (2) pulmonary, (3) cutaneous, (4) GI, and (5) disseminated. Gastrointestinal mucormycosis comprise 7% of the reported cases.[5] Invasive GI mucormycosis has a mortality rate of over 90%;[6] whereas in disseminated disease, it can be as high as 100%.[3]

Diagnosis of mucormycosis can be based on either growth of the mold on culture or histopathologic demonstration on biopsy of aseptate, wide, ribbon-like hyphae that branch at the right angles.[3,7] However, due to the difficulty of growing organisms from tissue culture, biopsy with histologic identification remains the preferred mode of diagnosis.[7]

Treatment for mucormycosis consists of both surgical debridements of necrotic material and administration of liposomal or lipid-based formulations of amphotericin-B.[3] Depending on the site of infection, surgical debridement may not be possible or plausible as in cases of disseminated disease. Adjunctive treatment options such as the use of nonsiderophore iron chelators, hyperbaric oxygen, or cytokine therapy with either gamma interferon or colony-stimulating factor are being investigated, but these methods have not yet proven to be more beneficial than standard treatment with surgery and amphotericin B.[8,9]

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Conflicts of interest
There are no conflicts of interest.

References