A Rare Case of Gastrointestinal Mucormycosis

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Abstract

Gastrointestinal Mucormycosis (GIM) is a rare life-threatening angio-invasive infection. The classic risk factors include immunosuppression and metabolic derangement. Usually, there are classical risk factors in patients affected by ileocecal mucormycosis. Few case reports have shown the absence of salient clinical presentation of mucormycosis in prolonged hospitalisation. The presence of association of mucormycosis in patients of typhoid infection is rare. Here, we present a case of invasive ileal mucormycosis occurring as a sequel to typhoid infection which lacked the typical risk factors for mucormycosis.

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The patient was discharged in satisfactory condition on posaconazole and antibiotics.

FIGURE 1: a) Necrotic tissue with fungal hyphae, H&E x400 b) Thrombosed blood vessel, H&E x200 c) Peyer’s patch (star) and thrombosed vessel (arrow), PAS x100 d) Necrotic lymph node, H&E x100

FIGURE 2: a) GMS stain showing fungal hyphae, x400 b) PAS stain showing right-angled branching (arrow), x400 c) Fungal hyphae within necrotic lymph node, PAS x400 d) fungal hyphae invading vessel wall, PAS x400
Discussion
Mucormycosis is a fatal infection caused by fungi belonging to the subphylum Mucoromycotina and order Mucorales [5]. GIM (Gastro-intestinal Mucormycosis) accounts for only 7% of all cases, but the mortality rate can be as high as 85%. The most common site of GI mucormycosis is the stomach followed by the colon and ileum [3,6].

Typical risk factors of mucormycosis are diabetes, defects in host phagocytes resulting in the immunocompromised state, corticosteroid use, immunosuppression for organ or stem cell transplantation, and increased levels of serum iron as a result of acidosis or administration of deferoxamine [2,3,7]. GIM has also been reported in the literature in individuals without the typical risk factors of uncontrolled diabetes mellitus or immunosuppression [2,3,6,9,10]. This is called healthcare-associated gastrointestinal mucormycosis occurring in immunocompetent adults who are admitted to the intensive care unit or after prolonged hospitalization and major surgery [11].

GI mucormycosis with concurrent typhoid fever has been very rarely reported in the literature[4]. Pathophysiology of GI mucormycosis in typhoid includes malnutrition, impaired mucosal integrity following S. typhimurium enteritis facilitates hyphal invasion into the intestinal wall and impaired phagocytic function of macrophages in typhoid infection further facilitates the growth of fungi [12]. Angioinvasion by the fungi, resulting in thrombosis of vessels and local ischemic necrosis, provides the nidus for hematogenous dissemination [13].

Diagnosis of GIM can often be delayed because of non-specific presentation. The most common symptoms are non-specific abdominal pain and distention associated with nausea and vomiting. Fever and haematochezia can be present. The diagnosis is usually during surgery or endoscopy by biopsy of the suspected area [3]. Successful management requires timely diagnosis, reversal of predisposing risk factors, prompt antifungal therapy, and early surgical debridement [7]. Treatment options include amphotericin B, triazole, Posaconazole [14].

Conclusions
Association of GI mucormycosis with enteric virus is very rare. It assumes importance in developing countries where Salmonella infections are very common. The exact mechanism of their association is not well understood but can be attributed to disturbed host immunity in typhoid infections or due to prolonged hospital admission in immunocompetent persons.

Patients not improving on conservative management of typhoid and presenting with melaena warrant exploration. It is important to have a high index of suspicion in these patients so that early identification of such a life-threatening infection can be made.

Additional Information
Disclosures
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