Case Report

A case of gastrointestinal mucormycosis

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ABSTRACT

We report a case of a 74-year-old asthmatic lady on inhalers who came with acute exacerbation of bronchial asthma with no evidence of hypercarbia or acidosis and during her course in hospital stay, she developed an acute onset of abdominal distension due to intestinal obstruction. She was extensively evaluated and was finally diagnosed with gastrointestinal mucormycosis. Primary gastric mucormycosis is a rare but potentially lethal fungal infection due to the invasion of Mucorales into the gastrointestinal mucosa. Being a rare entity, the diagnosis is most often overlooked. The importance of this case is that a high index of suspicion is required for early diagnosis and management of the disease, and the mortality is very high, particularly in immunocompromised patients.

Key words: Fungi, Gastrointestinal, Infection, Mucormycosis

Mucormycosis is a life-threatening infection caused by fungi of the subphylum Mucoromycotina, order Mucorales. Mucormycosis can affect any organ system, but the most common presentations involve either the nasal sinuses, orbit, brain (rhino-orbital-cerebral), or the lung; however, gastrointestinal mucormycosis is rare [1]. Diagnosis is often histopathological; occasionally, a fungal culture or polymerase chain reaction may also yield results. Treatment comprises antifungal drugs such as liposomal amphotericin B, posaconazole, isavuconazole, or triazoles; or surgical resection of the necrotic area is needed in some cases [2].

CASE PRESENTATION

A 74-year-old hypertensive and asthmatic lady, on regular doses of inhalers, presented with cough for 2 weeks, followed by fever and breathlessness for 2 days.

On examination, she had stable vitals with bilateral polyphonic wheeze. Investigations showed neutrophilic leukocytosis with normal renal and hepatic function, with no evidence of hypercarbia or acidosis. The electrocardiogram was within normal limits, and the chest X-ray was normal except for prominent bronchovascular markings.

She was admitted with a provisional diagnosis of infective exacerbation of bronchial asthma and treated with empirical antibiotics, nebulizations, and other supports. However, she responded poorly and was initiated on non-invasive mechanical ventilation. During her course in the hospital, her blood sugar levels were found to be profoundly high with glucose, random blood sugar 860 mg/dL, with no evidence of ketosis, and she was managed with insulin. She also developed acute kidney injury, which was managed conservatively. Serum creatinine was 2.9 mg/dL, blood urea was 96 mg/dL, serum Na was 120 mmol/L, and serum K+ was 5.6 mmol/L. Sputum analysis grew no significant organism and was negative for acid–fast bacilli or fungal KOH mount.

A few days into her hospital stay, she developed constipation, followed by acute abdominal distension. On examination, the abdomen was distended, gaseous distension, with no guarding or rigidity, and bowel sounds were absent. Computed tomography (CT) abdomen showed features of paralytic ileus with dilated bowel loops of jejunum, ileum, cecum, and ascending colon with fecal loading and collapse of the bowel loops distally to the hepatic flexure, including the transverse colon, sigmoid colon, and the rectum.

Her general condition deteriorated. She became drowsy, anuric with a persistent rise in inflammatory parameters. She was intubated and mechanically ventilated and initiated on hemodialysis. Antibiotics were escalated, and antifungals were added. Prokinetics were added to improve gastrointestinal motility; however, it was in vain. In view of persistent abdominal distension with absent gastrointestinal (GI) motility, she then underwent exploratory laparotomy and small bowel resection with end ileostomy.

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Intraoperatively, the bowel loops showed no peristaltic movements and had multiple ulcerations at the fecal impaction sites. Histopathological examination of the resected bowel showed ischemic changes with serositis and perforation along with fungal organisms stained with PAS (Fig. 1) and GMS stains (Fig. 2), morphologically consistent with Mucormycosis. Thus, a diagnosis of gastrointestinal mucormycosis was made. However, she succumbed to her illness before the diagnosis was made.

DISCUSSION

The Mucorales are ubiquitous environmental fungi to which humans are constantly exposed. Mucormycosis typically occurs in patients with diabetes mellitus, solid-organ transplantation, hematopoietic stem cell transplantation, prolonged neutropenia, corticosteroid use, or malignancy [3]. It can occur as an isolated cutaneous or subcutaneous infection in immunologically normal individuals after traumatic implantation of soil or vegetation or in nosocomial settings through direct access through intravenous catheters, subcutaneous injections, or maceration of the skin by a moist dressing [4].

Hyperglycemia directly contributes to the risk of mucormycosis through various mechanisms:

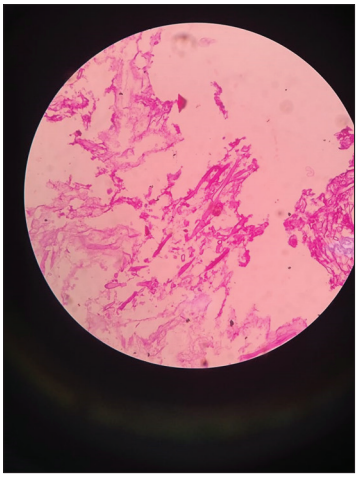

Figure.1: Ribbon-like aseptate hyphae suggestive of *Mucor* fungi using PAS stain

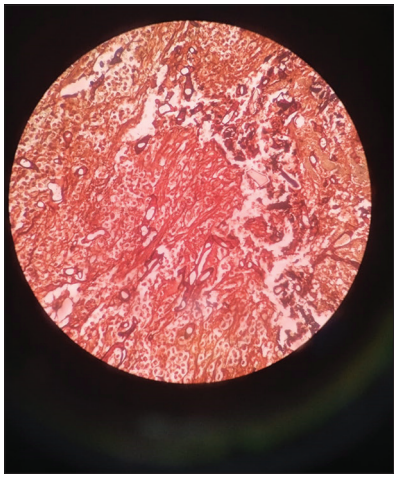

Figure 2: Ribbon-like aseptate hyphae with 90f branching with GMS staining suggestive of *Mucor* species

(a) Hyperglycation of iron-sequestering proteins, disrupting normal iron sequestration; (b) upregulation of a mammalian cell receptor (GRP78) that binds to Mucorales, enabling tissue penetration (due to both a direct effect of hyperglycemia and increasing levels of free iron); (c) induction of poorly characterized defects in phagocytic function; and (d) enhanced expression of CotH, a Mucorales-specific protein that mediates host cell invasion by binding to GRP78 (due to hyperglycemia and the resulting free iron).

Mucormycosis commonly affects the nose, sinuses, and the orbit; gastrointestinal involvement is extremely rare. The stomach is the most common site of gastrointestinal mucormycosis, followed by the colon and ileum. In the past, gastrointestinal mucormycosis was seen primarily in premature neonates, often in association with widespread disseminated disease. Because this infection is acute and rapidly fatal, it is often diagnosed postmortem.

There are very few cases of GI mucormycosis reported worldwide [1,2,5,6]. Our patient is a 74-year-old lady, who was on long-term inhalational steroids with newly diagnosed diabetes, was diagnosed with GI mucormycosis. The involvement was extensive, involving the small and large bowel, including the ascending colon. Although she presented with an acute exacerbation of bronchial asthma, fungal elements could not be isolated from the sputum samples. Morton *et al*. reported a case of a 38-year-old lady who presented with an acute abdomen due to cecal perforation as a complication of gastrointestinal mucormycosis. She was on steroids for Crohn's disease, which probably increased her risk for the infection and overlooked her symptoms. She was treated with liposomal amphotericin B, followed by posaconazole for 1 month, with gradual improvement in her clinical condition [1]. Another case of gastrointestinal mucormycosis presenting as recurrent gastric perforation was reported in a patient with Crohn's disease who was receiving glucocorticoids, 6-mercaptopurine, and infliximab. In this case, because the recurrent perforations were thought to be due to active Crohn's disease, treatment with amphotericin B was delayed, and the patient suffered from multiorgan system failure and subsequent death [2]. A rare case of emphysematous gastritis associated with invasive gastric mucormycosis in a 43-year-old man, suffering from alcoholism and diabetes, has experienced diffuse abdominal pain for 4 days. Abdominal CT scan demonstrated gas within the stomach wall. A histologic examination of the total gastrectomy specimen showed several gas-filled bubbles in the wall, along with numerous fungal hyphae throughout the necrotic stomach wall. He died of multiorgan failure secondary to disseminated mucormycosis, despite the intensive medical therapy [3]. A 42-year-old bisexual man was hospitalized because of fever and severe dyspnea. Pneumocystis carinii bilateral pneumonia was diagnosed, and the patient was treated with co-trimoxazole and steroids

at high doses with satisfactory response. The human immunodeficiency virus serologic result was positive (ELISA and Western blot). The CD4 subset of lymphocytes was 60/mm^3. Kaposi sarcoma lesions were observed in both feet and confirmed by histologic examination. Two weeks after admission, the patient stated that he had epigastric pain and retrosternal discomfort. Oral thrush was seen, and he was started on ketoconazole. Histologic examination of the endoscopic biopsy specimen revealed superficial ulceration with granulomatous reaction in the lamina propria containing giant cells, and the histologic findings were compatible with mucormycosis [5]. Two cases reported by Rivero *et al*. of gastrointestinal mucormycosis by *Mucor indicus:* A 77-year-old woman with a gastric ulcer and a 25-year-old man with liver lesions. Both were treated with surgery and liposomal amphotericin B; only one survived. Recognizing gastrointestinal mucormycosis in the correct clinical context is essential and requires timely surgical and antifungal treatment [6].

Often driven by progression of the underlying predisposing condition and the highly invasive nature of the disease, the morbidity and mortality rates are very high, despite effective antifungal therapies. Optimizing the chances for successful treatment of mucormycosis requires four steps: (1) Early initiation of therapy; (2) surgical debridement, when possible; (3) rapid reversal of underlying predisposing risk factors, if possible; and (4) proceeding to treat underlying malignancy, if present, without waiting to complete antifungal therapy first. Primary therapy for mucormycosis should be based on a polyene antifungal agent.

CONCLUSION

With non-specific symptoms and the rapidly deteriorating nature of the disease, a strong index of suspicion is crucial for early identification and treatment of this disease. Extensive investigations with imaging and histopathologies, along with early initiation of antifungals, may help reduce the burden of the disease.

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