

Case Report

A fatal case of gastric mucormycosis: a case report

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ABSTRACT

Mucormycosis is an invasive infection caused by the fungi of the order Mucorales. The members of the family Mucoraceae most commonly isolated are *Rhizopus* and the second most common genus is *Mucor*. It typically affects immunocompromised individuals. Common presenting symptoms are abdominal pain, hematemesis and melena. The infection pathway is airborne through skin or ingestion of the sporangiospores. Primary gastrointestinal mucormycosis is a rare disease with a high mortality rate. We report a case of mucormycosis associated necrotic gastric ulcer with a fatal outcome. A 47-year-old female with history of chronic kidney disease presented with acute gastrointestinal symptoms. An endoscopy was performed and diagnosis of peptic ulcer disease in the setting of hemodialysis was made. Histopathology revealed mucormycosis and she was started on posaconazole. The patient was stabilized and discharged however failed to comply to antifungal treatment; resulting in a fatal outcome. Recognizing gastric mucormycosis early with timely treatment is essential for a good outcome.

Keywords: Mucormycosis, Necrotic gastric ulcer, Posaconazole, Chronic kidney disease

INTRODUCTION

Mucormycosis is a rare life-threatening fungal infection prevalent in those with diabetes mellitus, neutropenia, systemic glucocorticoid therapy, hematological malignancies, and immunocompromised individuals.¹ Gastrointestinal involvement is seen in less than 15% of all reported cases.^{2,3} The organs commonly affected in the gastrointestinal tract include the stomach followed by the colon and ileum. Here, is a case of gastrointestinal mucormycosis (GIM) with acute gastrointestinal bleeding with a fatal outcome most likely due to poor compliance to treatment.

CASE REPORT

The patient, a 47-year-old female, who lived in rural central India, presented the emergency room with acute

abdominal pain and massive hematemesis on day 0. She had a history of chronic kidney disease and was on maintenance hemodialysis. Other comorbidities included hypertension, hypothyroidism and osteoporosis.

She had vomited approximately 300 ml of fresh blood. Her abdominal pain was sudden, severe and progressively increasing. She also reported one episode of lower gastrointestinal bleed with passage of blackened stools. There was no history of ingestion of non-steroidal anti-inflammatory drugs (NSAIDs), anticoagulation drugs, non-smoker and nonalcoholic. There was no history of hematological malignancies and viral serology for hepatitis B, hepatitis C and HIV was negative.

Her general condition was poor; she had marked pallor and extremities were cold. She had tachycardia (110 beats per minute), hypotension (blood pressure; 90/60 mmHg),

tachypneic (respiratory rate; 28/min) however maintained a SpO₂ of 95% on room air. She was afebrile. The chest was clinically clear and abdominal examination revealed diffuse tenderness with guarding. She was resuscitated in ER and a ryle's tube was placed demonstrating fresh blood on aspiration following which patient was shifted to the ICU for stabilization.

Investigations

All routine investigations showed a hemoglobin of 6.1gm/dL, total white count of 5100/mm³, platelet count 3.32 lac/mm³, serum urea 24 mg/dL, serum creatinine 3.21 mg/dL, serum sodium 131meq/L, serum potassium 3.3meq/L, prothrombin time of 15.4 seconds and internationalized ratio (INR) of 1.32. The rest of the laboratory parameters including liver function tests, hepatitis B antigen, hepatitis C antigen and HIV antigen were normal. Chest x-ray and EKG revealed no abnormalities except sinus tachycardia.

Treatment

After resuscitation and transfusion of 2 units of packed cells, an urgent esophagogastroduodenoscopy (EGD) was done on day +1. It showed a necrotic gastric ulcer with black eschar in the fundal region (Figure 1). Multiple biopsies were taken. A colonoscopy showed only melanic stools. Patient was managed in the intensive care unit (ICU) and continued on oxygen administered by bilevel positive airway pressure (BiPAP), intravenous saline, pantoprazole infusion and intravenous ceftriaxone. Post transfusion hemoglobin level was 8.1 gm/dL. Computed tomography (CT) abdomen revealed mild wall thickening in the posterior wall of the stomach with no extravasation of contrast. Patient was dialyzed on day +2 following which her oxygen requirement reduced and was taken on nasal prongs. She had no further episodes of hematemesis or melena and was transitioned to a step-down unit. By day +5, biopsy of the ulcer showed necrotic material with dense inflammatory infiltrate containing neutrophils, plasma cells, eosinophils with broad aseptate, obtuse angled fungal hyphae (Figure 1 and 2). She was started on posaconazole 300 mg daily regimen. A partial gastrectomy was recommended to the patient however she continued with medical treatment. She was discharged on day +7. Patient failed for follow up and due to terminal illness, ultimately succumbed to illness.

DISCUSSION

Mucormycosis is a rare and potentially life-threatening infection that can affect many parts of the body including head and neck, lung, skin, gastrointestinal tract, central nervous system and most commonly the nasal cavity. The incidence of GIM has been reported around 5% to 15%, however, over the course of the past two decades, gastrointestinal mucormycosis has been increasingly reported in immunocompromised and immunocompetent individuals.² Most cases are diagnosed incidentally during

endoscopic or operative procedure, hence assessing the incidence is challenging. A systemic review of 87 cases revealed a mortality of 42% and 87% in adults and infants respectively in GIM.⁴

GIM can involve any part however most commonly affects the stomach (57.5%), followed by colon (32%), small intestine (7%) and esophagus (7%).⁵ Symptoms tend to be non-specific presenting as abdominal pain, dyspepsia, UGIB, lower gastrointestinal bleed (LGIB) and fever, especially in patients that are immunocompromised.⁶ The hallmark of mucormycosis is angioinvasion into the host tissue by fungal hyphae leading to infarction and necrosis. This prompts development of necrotic ulcers with resultant hematemesis, perforation, and peritonitis. Progression is usually rapid hence it carries a poor prognosis of 85% mortality.⁷

Diagnosis of GIM is usually delayed due to its nonspecific presentation and suspicion. Early detection plays a key role in improving prognosis.⁸ Mucorales hyphae exhibit similar characteristics irrespective of species. Hyphae tend to be broad based, thin, ribbon-like, aseptate with right angle branching.⁹ Histopathological examination (HE) has demonstrated a sensitivity of 70% in diagnosis of mucormycosis. A combination of culture and polymerase chain reaction (PCR) coupled with HE helps provide a definitive diagnosis.^{10,11} In our patient, diagnosis was made using HE however culture failed to demonstrate growth. More recently, Imprint cytology has been shown to be an established tool in the diagnosis of gastrointestinal lesions providing a sensitivity and specificity of 95% and 100% respectively.¹²

GI mucormycosis can be treated with different approaches including surgery with antifungal therapy, antifungal therapy alone or surgery alone. Early initiation of appropriate antifungal therapy is crucial for successful treatment. Amphotericin-B (AMB) or Liposomal amphotericin-B (LAMB) is usually recommended as the first line therapy for mucormycosis. Starting dosage schedule for AMB and LAMB 1 mg/kg/day and 5-7.5 mg/kg/day are commonly used. Insufficient data exists whether higher dosing provides additional benefit.^{13,14} LAMB dosing higher than 10mg/kg per day has demonstrated substantial increase in creatinine especially in patients with compromised eGFR<15 ml/min/1.73m².¹⁵ however a retrospective study has demonstrated LAMB to only significantly worsen serum creatinine in patients with eGFR>60ml/min/1.73m².¹⁶ For moderate mucormycosis or salvage therapy, intravenous or delayed-release tablet posaconazole and intravenous isavuconazole has proven to be effective in treatment of mucormycosis.¹⁷ Isavuconazole is typically administered as a loading dose of 200 mg intravenously every 8 hours for the first 48 hours, following which patients are transitioned to a daily dosing regimen either orally or intravenously. Overall, mortality was found to be similar to LAMB therapy alone however LAMB in combination with azoles revealed to significantly reduce mortality in adults. Typical side

effects noticed are hepatotoxicity and QTc shortening hence regular monitoring and potential drug interactions, particularly in patients receiving CYP3A4-metabolized drugs is required.¹⁸ Combination therapy with posaconazole, at starting dosage of (for suspension) 200 mg four times per day and then 400 mg twice a day or (for tablet) 300 mg twice a day and then 100 mg daily should be continued till clinical improvement as demonstrated by imaging is advised.¹⁹ A combination of surgical and medical treatment is strongly advised in general for patients with GIM; because the infection is associated with high rates of ulceration, perforation, and bleeding.²⁰⁻²² Owing to the angioinvasive nature of Mucorales, there is increased propensity for extensive thrombosis and tissue necrosis with black eschar formation, which further impairs penetration of antifungal and leucocytes to the site of infection which renders antifungal therapy ineffective *in vivo*.^{23,24}

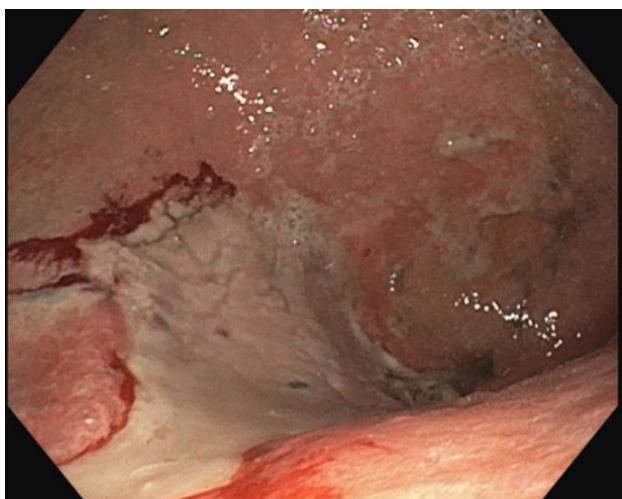


Figure 1: Esophagogastroduodenoscopy demonstrating large superficial necrotic ulcer with mild active oozing.

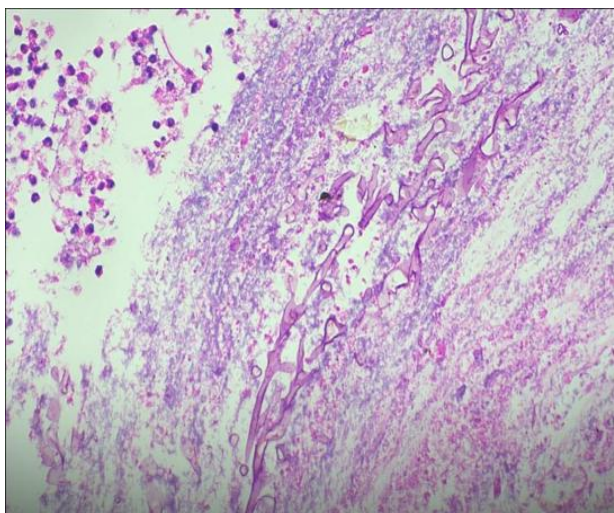


Figure 2: Demonstrating H and E stain for broad aseptate, obtuse angled fungal hyphae.

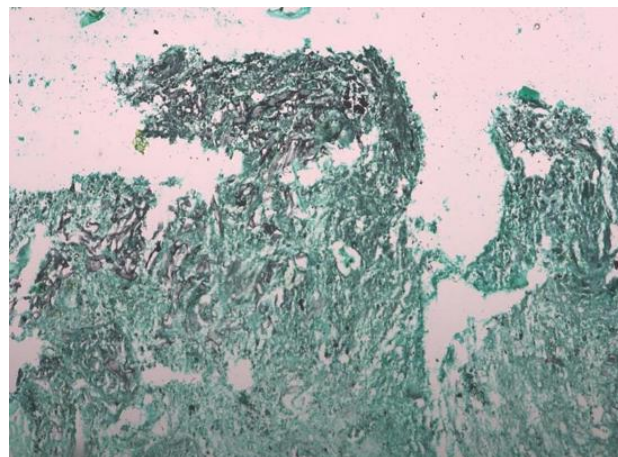


Figure 3: Demonstrating black broad aseptate hyphae on Grocott-gomori Methenamine Silver Stain.

CONCLUSION

Mucormycosis is a fatal opportunistic fungal infection which may result in high mortality in untreated patients. A high index of suspicion and awareness of physicians is required for early diagnosis and management of disease particularly in immunocompromised patients. A typical presentation such as worsening abdominal pain should be investigated with CT scan or MRI and OGD in those with inconclusive finding on radiological imaging.

Biopsy of the suspected mucosal lesions is the diagnostic of gastric mucormycosis. Immediate medical management with antifungal agents such as liposomal amphotericin B or posaconazole is required given the invasive nature of disease and high mortality. Serial OGD may be essential to monitor healing of gastric mucormycosis and determine duration of medical management. A combination of medical and surgical re- section of mucosal lesions is warranted in the case of poor response to antifungal therapy and those with extensive disease burden.

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