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Case Report

Gastric mucormycosis in an immunocompetent host: A case report

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ABSTRACT

Mucormycosis is rare but emerging fungal infection. In the past decade, it has emerged as an important lethal infection in diabetics and immunocompromised patients. Rhinosinusitis, pansinusitis, rhino-orbital and rhino cerebral forms constitute the classic manifestations of this fungus. Gastrointestinal mucormycosis is an uncommon disease with high mortality most often affecting patients with immunocompromised state. A very few cases of this disease have been described among patients with no risk factors and no known comorbidites. Our study attributes to one such case of invasive gastric mucormycosis diagnosed incidentally on histopathologic examination.

1. Introduction

The fungus mucormycosis belongs to family Mucoraceae, class Eumycetes. It is widely distributed in nature and is the only opportunistic pathogen to humans. They produce either superficial infection, affecting skin, nails, external ear; or deep mycotic infections, mostly rhino-orbito-cerebral. Rarely, it affects gastrointestinal system, stomach, followed by ileum and colon. Although a relatively rare fungal disease, there has been a substantial increase in number of cases of gastric and gastrointestinal mucormycosis during the past few years. In majority, invasive gastric mucormycosis affects patients with major risk factors, that being, immunocompromised status, diabetes, malignancy, on steroid therapy, organ or stem cell transplantation, or higher levels of available unbound serum iron. A very few cases of the disease have been described among patients with no risk factors and no known comorbidites. Our study attributes to one such case of invasive gastric mucormycosis diagnosed incidentally on histopathologic examination.

2. Case Report

A 73 years old male patient, presented at gastroenterology OPD with complaints of pain in epigastrium and right upper quadrant abdomen for 15- 20 days, along with loss of appetite and generalized weakness. He had been a case of recurrent anemia. There was negative history of vomiting, jaundice, cough, melena, fever. He neither had any known allergies nor any existing comorbidity such as diabetes, hypertension, chronic liver disease, coronary artery disease, tuberculosis, chronic obstructive pulmonary disease, cerebro-vascular accident or drug intake. On examination, he was hemodynamically stable, afebrile, conscious and well oriented to time, place and person, systemic examination being within normal limits. On palpation, abdomen was soft and non-tender. He was then admitted for further evaluation. All relevant

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investigations were done. Peripheral blood revealed anemia (Hb – 8gm/dl/) with neutrophilic leucocytosis. Iron profile was deranged with decreased S.iron = 13.88ug/dL, Total iron binding capacity = 146ug/dL, transferrin saturation = 7.0%, while ferritin being within normal range = 202ng/mL. He also had hyponatremia ($S.Na^{+}$=128.7mEqL), and Metabolic acidosis (pH = 7.05, S.lactate = 15mmol/L). The patient was managed conservatively with correction of the abnormalities aforementioned and best supportive care.

Upper abdomen CECT triple phase was done which showed thick walled necrotic mass lesion in upper abdomen communicating with body of the stomach (Gastro Intestinal Stromal Tumor) with mildly enlarged locoregional lymph nodes, thrombus in main portal vein and branches of right portal vein, and small hypodense lesions in left lobe of liver cysts. Upper gastrointestinal endoscopy was done which was reported as- Esophagus: normal; Stomach: Fundus- normal, Body- large diverticula at lesser curvature with internal necrotic slough likely malignant growth with Diverticula. Gastrointestinal stromal tumor, Lymphoma, Antrum- normal, Pylorus- normal. Biopsy of the gastric growth was taken and sent for histopathologic examination, which revealed aseptate, broad, easily foldable fungal hyphae alongwith necrotic slough. Fungus was confirmed by special stains- Periodic acid Schiff (PAS) and Gomori's methanamine silver (GMS) and diagnosis of mucormycosis was made on morphology. The diagnosis of invasive gastric mucormycosis was confirmed on fungal culture. Antifungals were immediately added to the ongoing treatment. Delayed presentation of the patient along with rapid progression of disease with invasion and thrombosis resulted in case fatality despite best critical care services.

3. Discussion

Mucormycosis is a rare type of fungal infection, incidence of which is increasing in the past decade, especially after the Covid pandemic. It has emerged as an important lethal infection in diabetics and immunocompromised patients. The classical manifestations include rhinosinusitis, pansinusitis, rhino-orbital and rhino cerebral forms. Paultauf et al reported first case of mucormycosis in 1985.[1]

Gastrointestinal mucormycosis is a rare presentation with high mortality most often affecting patients with predisposing conditions mentioned earlier. In a meta analysis done by Roden et al. in the year 2005, 929 cases of mucormycosis were reported, out of which 7% (66) were gastrointestinal mucormycosis with 85% (56) mortality rate.[2]

Another study conducted in 2011 by Chaya et al reported a case of mucormycosis in a 53 years old female. She was a known case of ulcerative colitis with drug history of oral prednisolone, landed up in hospital with urosepsis and was given inotropic agents and intravenous steroids. After investigations, she was found to be anaemic. Endoscopy and histology revealed giant gastric ulcers caused by mucormycosis.[3] The varied clinical manifestations and clinical presentations of mucormycosis often delay the diagnosis, resulting in poor outcomes. Only about 25% of cases of mucormycosis are being diagnosed antemortem.

Gastrointestinal mucormycosis is acquired by ingestion of pathogens in food such as fermented milk and dried bread products. Spore contaminated herbal and homeopathic remedies also serve as significant source of infection. Maravipoma et al. reported a series of case of gastrointestinal mucormycosis presumably transmitted orally by use of sporangiophore contaminated wooden applicators that were used to mix drugs for patients with nasogastric feeding tubes and also used for oropharyngeal examination in an hemato-oncology clinic.[4] A lot of cases of mucormycosis came up during the Covid pandemic, in the immunocompromised patients. Rampant use of steroids for the treatment also served as a cause for the increase in the incidence of mucormycosis (black fungus) cases.

In 2020, Haider et al reported a case of gastric mucormycosis complicated by mucosal invasion in a patient with poorly controlled diabetes and chronic renal disease, confirmed by histopathologic examination, PCR and culture of the biopsy specimens detecting Rhizopusmicrosporus DNA.[5] Chow et al. reported a case of gastric angioinvasive mucormycosis in 2017, as a deadly complication in immunocompromised patient after penetrating trauma.[6] A rare case of gastric mucormycosis complicated with gastropleural fistula was reported by Tomohisa et al in a 82 years old female on immunosuppressives for adult onset Still's disease.[7]

Very few cases of primary mucormycosis in immunocompetent patient have been reported in literature. One such case of pericardium involvement has been reported by Vaideshwar et al in 2007.[8] Jain et al studied 18 cases of mucormycosis presenting as necrotising fasciitis in immunocompetent people in 2006.[9] Sharma et al also reported isolated GI mucormycosis in eight patients of which two were middle-aged without predisposing factors.[10] A case of gastric perforation secondary to fungal gastritis in a 23 years old immunocompetent female was reported in 2021 by Ankit et al. Patient presented with features of peritonitis and underwent emergency laparotomy following which fungal etiology was confirmed on perforation edge biopsy. She was timely treated on antifungals and recovered.[11]

Gastrointestinal mucormycosis can also involve liver, spleen, pancreas, and invade bowel walls and blood vessels. It is categorised into three forms: colonization, infiltration and vascular invasion. The most common presentation is perforation, bleeding or epigastric distention. Mucormycosis in one organ can spread hematogenously to other organs as well. Patients with iron overload (especially those receiving deferoxamine), profound

immunosuppression (eg, recipients of allogeneic stem cell transplants having graft-versus-host disease treated with corticosteroids), or profound neutropenia and active leukemia are the classic groups at risk for disseminated mucormycosis.[10,11]

Diagnosis depends on demonstration of the organism in the tissue. Newer diagnostic modalities include serology and multiplex PCR system. Systemic amphotericin B is the mainstay of treatment. Reversal of underlying medical disease and surgical debridement is necessary for successful management. Newer therapies for mucormycosis include posaconazole and deferiprone and adjunctive hyperbaric oxygen therapy.[5,6]

4. Conclusion

To conclude, this was a case of fungal invasion to blood vessels causing thrombosis secondary to GI mucormycosis which was diagnosed very late due to unsuspected fungal infection in an immunocompetent person complicated by its rapid progressive course. We believe that the primary site of infection was the stomach. Earlier histopathologic confirmation could have clinched the diagnosis. In view of growing incidence of fungal infection with high case fatality, it should be kept as a differential when common causes have been ruled out, for timely management of the patient.

5. Source of Funding

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6. Conflict of Interest

None.

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