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Case Report

Internal Medicine Section

Gastric Mucormycosis in an Immunocompetent Patient Masquerading as Peptic Ulcer Disease: A Case Report

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ABSTRACT

The present case describes a 42-year-old previously healthy male who presented with two weeks of dull epigastric pain, nausea, and melena. On examination, he was pale but haemodynamically stable. Laboratory evaluation revealed iron deficiency anaemia, while other haematological, renal, and liver parameters were within normal limits. Upper gastrointestinal endoscopy demonstrated two large antral ulcers with irregular margins, necrotic bases, and surrounding mucosal oedema. Histopathology confirmed gastric mucormycosis, showing broad aseptate fungal hyphae with right-angle branching, highlighted by Periodic Acid-Schiff (PAS) and Grocott's Methenamine Silver (GMS) staining. The patient was treated with intravenous liposomal amphotericin B (5 mg/kg/day) for 21 days, followed by oral posaconazole (300 mg daily) for six weeks. Clinical improvement occurred within 10 days. Follow-up endoscopy at three months showed complete ulcer healing, and at six months, the patient remained asymptomatic with no evidence of recurrence. The present case emphasises that gastric mucormycosis, although rare, can occur in immunocompetent individuals. Early endoscopic biopsy and prompt initiation of antifungal therapy are crucial, and selected cases may achieve successful outcomes with medical management alone, without surgical intervention.

Keywords: Gastrointestinal diseases, Periodic acid-Schiff, Stomach ulcer, Zygomycetes

CASE REPORT

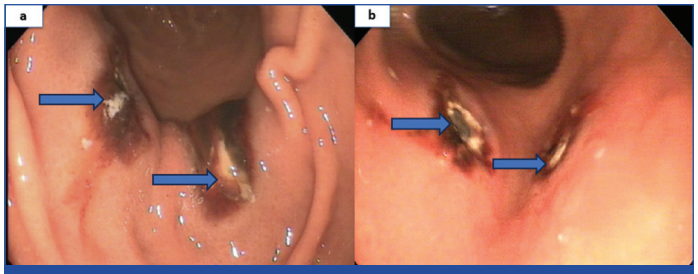
A 42-year-old male, previously healthy with no comorbidities, presented with two weeks of dull epigastric pain associated with nausea and passage of black tarry stools. There was no history of hematemesis, haematochezia, weight loss, fever, or loss of appetite. He denied alcohol use, smoking, or intake of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). There was no history of corrosive ingestion, prior surgery, tuberculosis, Coronavirus Disease (COVID-19) or chronic medication use. Family history was unremarkable. On clinical examination, the patient appeared pale but was haemodynamically stable with a pulse rate of 92/min and blood pressure of 110/70 mmHg. No postural hypotension was noted. His baseline weight was 72 kg. There was no evidence of icterus, lymphadenopathy, pedal oedema, or peripheral stigmata of chronic liver disease. Abdominal examination revealed mild epigastric tenderness without guarding, rigidity, or palpable organomegaly. Other systemic examinations, including cardiovascular, respiratory, and neurological assessments, were unremarkable. Initial management included stabilisation with intravenous access, monitoring of vital parameters, and assessment of haemodynamic status. The patient was administered intravenous pantoprazole 80 mg stat, followed by a continuous infusion at 8 mg/hour. The patient was kept Nil Per Oral (NPO) initially, started on intravenous fluids for hydration, and monitored closely for ongoing blood loss.

Laboratory investigations showed haemoglobin 9.8 g/dL with microcytic hypochromic indices, leukocyte count 7,600/µL, and platelet count 2.1×10^5/µL. Renal and liver function tests were normal. Fasting blood glucose was 92 mg/dL and Glycosylated Haemoglobin (HbA1c) 5.2%. HIV, hepatitis B, and hepatitis C serologies were negative. Iron studies confirmed iron deficiency anaemia [Table/Fig-1].

Following informed consent, an upper gastrointestinal endoscopy was performed, which demonstrated two large antral ulcers with irregular margins, necrotic bases, and surrounding mucosal oedema, with active bleeding noted [Table/Fig-2].

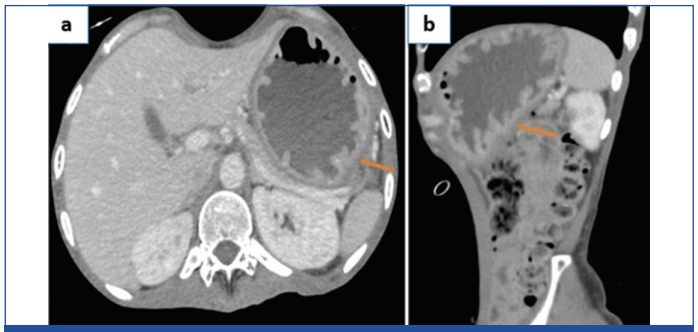
Parameters	Result	Reference Range
Haemoglobin (g/dL)	9.8	13-17 (male)
RBC indices (MCV, MCH, MCHC)	MCV 72 fL, MCH 24 pg, MCHC 29 g/dL	MCV: 80-96 fL, MCH: 27-33 pg, MCHC: 32-36 g/dL
Total leukocyte count (/µL)	7,600	4,000-11,000
Platelet count (/µL)	2.1×10^5	$1.5\text{-}4.5 \times 10^5$
Serum creatinine (mg/dL)	0.9	0.7-1.3
Blood urea nitrogen (mg/dL)	14	7-20
Sodium (mmol/L)	138	135-145
Potassium (mmol/L)	4.1	3.5-5.1
Total bilirubin (mg/dL)	0.8	0.2-1.2
AST (SGOT) (U/L)	28	<40
ALT (SGPT) (U/L)	32	<41
ALP (U/L)	86	44-147
Serum albumin (g/dL)	4.2	3.5-5.0
Fasting blood glucose (mg/dL)	92	70-100
HbA1c	5.2%	<5.7%
HIV 1 & 2	Negative	Negative
HBsAg	Negative	Negative
Anti-HCV antibody	Negative	Negative
Serum iron (µg/dL)	28	60-170
Total Iron Binding Capacity (TIBC) (µg/dL)	420	240-450
Transferrin saturation	7%	20-50%
Ferritin (ng/mL)	8	24-336 (male)

[Table/Fig-1]: Laboratory investigations.
MCV: Mean corpuscular volume; MCH: Mean corpuscular hamoglobin; MCHC: Mean corpuscular haemoglobin concentration; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; HIV: Human immunodeficiency virus; HCV:Hepatitis C virus



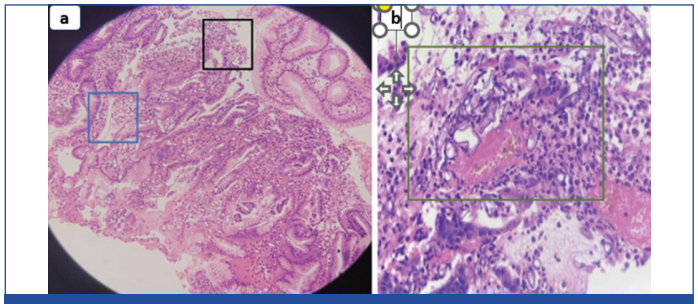
[Table/Fig-2]: Upper gastrointestinal endoscopy: a) and b) demonstrating two large antral ulcers with irregular margins and necrotic base (blue arrows).

Multiple biopsies were obtained from the ulcer edges and bases for histopathological evaluation. Contrast-Enhanced Computed Tomography (CECT) abdomen showed localised thickening of the gastric antrum without perigastric fat stranding, perforation, or lymphadenopathy. No other gastrointestinal involvement was noted [Table/Fig-3].

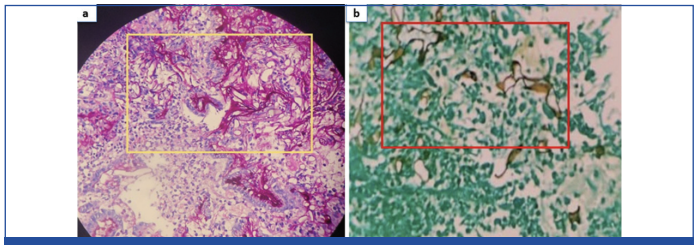


[Table/Fig-3]: CECT abdomen: a) axial; and b) sagittal section showing gastric wall thickening.

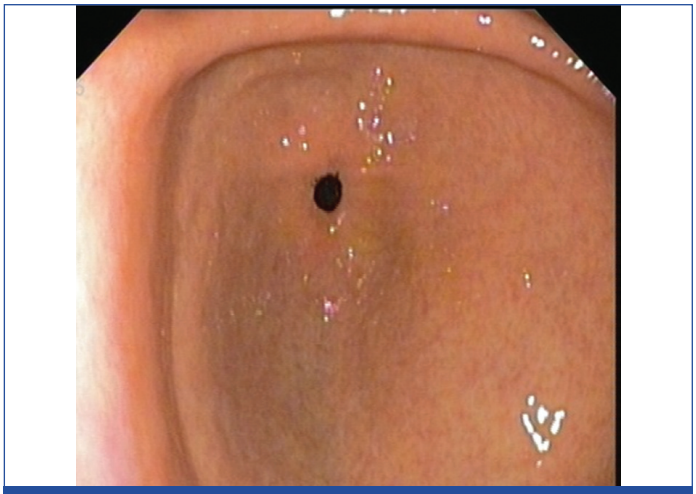
Fungal culture is the gold standard for confirming mucormycosis. However, in this case, culture could not be performed due to limited tissue availability and the need for urgent management. The diagnosis was established based on characteristic histopathological findings. Histopathology demonstrated extensive necrosis containing broad, ribbon-like, aseptate fungal hyphae with right-angle branching, consistent with Mucorales. Periodic Acid-Schiff (PAS) and Grocott Methenamine Silver (GMS) stains confirmed the diagnosis of gastric mucormycosis [Table/Fig-4,5].



[Table/Fig-4a,b]: Haematoxylin and Eosin (H&E) staining shows gastric epithelium with subepithelium showing dense mixed inflammation with areas of ulceration (black box) and necrosis (blue box) along with broad aseptate fungal hyphae with obtuse-angled branching with angioinvasion (green box) consistent with mucormycosis. Image A: 10× magnification; Image B: 40× magnification.



[Table/Fig-5]: Histopathology: a) Periodic Acid-Schiff staining showing broad ribbon-like aseptate magenta-stained hyphae consistent with mucormycosis; b) Grocott Methenamine Silver (GMS) staining showing fungal hyphae (red box) appearing brown on a counterstained green background. Image a: 20× magnification; Image b: 40× magnification.



[Table/Fig-6]: Re-examination of upper gastrointestinal endoscopy at three months revealed complete resolution of ulcers.

The patient was started on intravenous liposomal amphotericin B (5 mg/kg/day), with close monitoring of renal function and electrolytes. Concurrently, he received intravenous proton pump inhibitors pantoprazole 40 mg BD and other supportive measures, including hydration and nutritional optimisation. Within 10 days, his abdominal pain improved and his melena resolved. He completed 21 days of intravenous liposomal amphotericin B, after which he was transitioned to oral posaconazole (300 mg daily) for six weeks.

At the 3-month follow-up, endoscopy demonstrated complete healing of both antral ulcers. At six months, the patient remained asymptomatic, with normalisation of haemoglobin to 13.4 g/dL, stable renal and hepatic parameters, and a weight gain of 3 kg. No clinical or endoscopic evidence of recurrence was observed, and the patient continues to remain under periodic surveillance [Table/Fig-6].

DISCUSSION

Mucormycosis is a severe, angio-invasive fungal infection caused by members of the order Mucorales, predominantly Rhizopus, Mucor and Lichtheimia species [1]. It is characterised by rapid tissue invasion, thrombosis, and necrosis, and carries a high mortality despite advances in antifungal therapy and surgery. It can present in various clinical forms, including rhino-orbito-cerebral, pulmonary, gastrointestinal, cutaneous, disseminated, and rarely renal types, depending on the site of infection and host risk factors.

Gastrointestinal mucormycosis is relatively uncommon, accounting for less than 10% of all cases [2]. The stomach is the most frequently involved site, followed by the colon and ileum [3]. It may occur either as a primary or secondary infection. Primary disease typically results from ingestion of food or materials contaminated with fungal spores, which subsequently adhere to areas of injured gastric mucosa, leading to angioinvasion, tissue ischaemia, and necrosis. In contrast, secondary gastrointestinal involvement arises from haematogenous dissemination of the fungus in the setting of systemic mucormycosis [4]. Clinical manifestations are nonspecific and may range from vague abdominal pain and melena to catastrophic events such as perforation and massive gastrointestinal haemorrhage, which complicates timely diagnosis [3]. Traditionally, gastrointestinal mucormycosis was considered to occur almost exclusively in patients with significant immunocompromise, including uncontrolled diabetes mellitus, haematological malignancies, or prolonged corticosteroid use [5-7]. However, recent evidence suggests that even immunocompetent individuals may be affected, albeit rarely. Although mucormycosis typically occurs in immunocompromised individuals, several factors may explain its presence in patients without obvious risk factors. Local trauma to the gastric mucosa- caused by ulceration or even minor injury- can create an entry point for fungal spores to invade the tissue. Iron availability plays

a crucial role, as even minimal mucosal damage or bleeding can release enough iron to support fungal growth. Additionally, acid suppression and changes in gastric pH may enhance the survival of fungal elements. Ingestion of contaminated food or water containing Mucorales spores has been proposed as a potential route of infection in endemic regions. Subtle impairments in host defences, such as reduced phagocytic activity at the mucosal level or alterations in the gastric microbiota, may also allow fungal invasion in individuals with otherwise intact immune systems. Nutritional deficiencies or acute metabolic stress can further weaken resistance, creating conditions favourable for infection [3,6,8].

The diagnosis of gastrointestinal mucormycosis is often challenging and delayed because of its nonspecific clinical manifestations. Timely histopathological confirmation through endoscopic biopsy remains the cornerstone for establishing the diagnosis. On endoscopy, characteristic findings may include edematous and hemorrhagic mucosa that can resemble ischaemic changes, the presence of ulcers with well-defined margins, areas of necrosis, vascular thrombosis in the surrounding tissue and occasionally ulcers overlaid by black eschar [9] mimicking peptic ulcer disease, ischaemic ulcers, or malignancy. Histopathology demonstrating broad, aseptate hyphae with right-angle branching remains the diagnostic gold standard [10].

Optimal management of gastrointestinal mucormycosis requires a multidisciplinary approach. Conventional strategies favoured surgical debridement or resection combined with systemic antifungal therapy. Surgery remains lifesaving in cases complicated by perforation or uncontrolled bleeding [11]. Nonetheless, emerging evidence suggests that medical management alone may be successful in selected cases diagnosed early, without acute complications [12].

Liposomal amphotericin B continues to be the first-line antifungal agent, administered at 5-10 mg/kg/day due to its favourable efficacy and reduced nephrotoxicity compared to conventional formulations [11]. Step-down therapy with oral azoles, particularly posaconazole, has become increasingly important for consolidation therapy. Cornely OA et al., highlighted that posaconazole provides comparable outcomes when used as monotherapy or as step-down treatment following amphotericin B, reinforcing its role in long-term management [11]. Isavuconazole has also emerged as a potential alternative with improved tolerability, although its role in gastrointestinal mucormycosis remains less well established [13].

Despite advances in diagnostic modalities and antifungal therapy, outcomes in mucormycosis remain suboptimal. In a review of 929 cases of zygomycosis, survival was only 61% with amphotericin B deoxycholate alone, 57% with surgery alone, and improved to 70% when antifungal therapy was combined with surgical intervention [2].

Our case is significant for two reasons: The patient was immunocompetent with no identifiable risk factors, and he achieved complete resolution with medical therapy alone, without surgical intervention. Such outcomes are rarely reported in the literature and underscore the importance of early recognition, timely biopsy, and aggressive antifungal therapy

CONCLUSION(S)

Gastric mucormycosis, though rare, remains a highly lethal infection due to its angio-invasive course and delayed recognition. This case highlights that early endoscopic biopsy, prompt initiation of liposomal amphotericin B, and appropriate step-down azole therapy can achieve complete recovery even in immunocompetent individuals. Heightened clinical suspicion and timely intervention are crucial for improving outcomes and reducing mortality.

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