



CASE REPORT

# Gastric Mucormycosis Simulated as Phytobezoar in a Patient with Diabetes Mellitus and Chronic Kidney Disease: A Diagnostic Challenge

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## Abstract

*Gastrointestinal mucormycosis is a rare yet life-threatening fungal infection that mostly affects immunocompromised individuals. Gastric involvement is uncommon and usually misinterpreted due to ambiguous clinical presentations and endoscopic findings. A 55-year-old man with diabetes, hypertension, chronic kidney disease, and a history of alternative medication use for prolonged period presented with persistent vomiting, unexplained anemia, and abdominal distension during sepsis and acute illness. Initial imaging indicated stomach wall thickening, and upper gastrointestinal endoscopy revealed an ulceroproliferative growth in stomach but biopsy revealed just chronic gastritis. A big phytobezoar was discovered during a repeat endoscopy following proper stomach preparation. The resected bezoar was histopathologically examined and found wide, aseptate fungal hyphae consistent with mucormycosis. Intravenous Liposomal Amphotericin B (5mg/kg/day) was initiated after histological confirmation. Despite six doses of Liposomal Amphotericin B, the patient developed massive gastrointestinal bleeding (hematemesis and hematochezia), progressive hemodynamic instability and subsequently he died.*

**Keywords:** Phytobezoar; Histopathology; Gastric Mucormycosis; Liposomal Amphotericin B

## Introduction

Mucormycosis is an invasive fungal infection caused by fungus from the Mucorales order<sup>1</sup>. It primarily affects people with uncontrolled diabetes, chronic kidney disease, malignancy, or those on immunosuppressive medication<sup>1-3</sup>. Gastrointestinal mucormycosis accounts for a small fraction of cases but has a significant death rate due to late diagnosis and rapid development<sup>4</sup>. Gastric mucormycosis can mimic cancer, peptic ulcer disease, or bezoar, causing diagnostic delays<sup>2,4,5</sup>. We describe a unique case of gastric mucormycosis that was discovered only after removing a phytobezoar.

## Case presentation

A 55-year-old male school teacher presented with a 12-year history of type 2 diabetes mellitus, 5-year history of hypertension, renal impairment for about 6 months, and a traumatic non-healing ulcer on his left great toe for approximately 2-3 years. He has history of taking homeopathic medications, different tree leaves, concentrated alcohol with medication, different powder sachets for control of diabetes over 2-3 years. About one and half months ago, he developed fever, respiratory distress and was admitted to ICU in district hospital where he received four sessions of hemodialysis (HD) with three

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units red cell concentrate (RCC) transfusion. He was then referred to Dhaka Medical College Hospital (DMCH) for better management and admitted to ICU on 12.12.2025 and treated as a case of Sepsis with ARDS due to bilateral pneumonia, receiving three sessions HD while in the ICU. At ICU, his blood C/S revealed *Cryptococcus* growth, urine C/S showed growth of *Klebsiella* and *Candida*, in sputum gram staining there was plenty of pus cells but C/S showed no growth of organism and he was treated with broad spectrum antimicrobials including Aztreonam, Ceftazidime-Avibactam, and Fluconazole. After improvement of his condition, he was stepped down to ward under department of Nephrology. As his renal function became stable and urine output improved, no further HD was needed. Despite optimum fluid management

in Nephrology ward, he remained clinically dehydrated and again developed anorexia, weakness and progressive anemia. He denied any history of hematemesis, melaena, hematochezia, or hemoptysis. A few days later, he complained persistent vomiting about 5 to 6 episodes per day, more marked after taking food, non-projectile, muddy-brown in color, contained partially digested food particles, associated with constipation for 3-4 days with gradual abdominal distension, and diffuse, dull aching abdominal pain, not associated with morning headache. There was no jaundice, lymphadenopathy, or organomegaly. Bowel sounds were present. For evaluation of vomiting and unexplained anemia, initially plain x-ray abdomen was done and he was kept nothing per oral (NPO) due to suspicion of intestinal obstruction but vomiting persisted despite this.

## Investigations

**Table-I**  
*Summary of Laboratory and Radiological Investigations*

| Investigations           | 21.12.25                                   | 30.12.25        | 16.1.26         |                  |
|--------------------------|--------------------------------------------|-----------------|-----------------|------------------|
| CBC                      | Hemoglobin                                 | 11.8 gm/dl      | 8.4 gm/dl       | 8.3 gm/dl        |
|                          | WBC Count                                  | 21.6 K/ $\mu$ L | 7.69 K/ $\mu$ L | 13.01 K/ $\mu$ L |
|                          | Neutrophil                                 | 95%             | 87%             | 87%              |
|                          | Platelet Count                             | 90 k/ $\mu$ L   | 150 k/ $\mu$ L  | 115 K/ $\mu$ L   |
| CRP (mg/L)               | 226.2                                      | 57.3            | 126             |                  |
| Urine R/M/E              | Albumin                                    | 1+              | Nil             | 1+               |
|                          | WBC                                        | Plenty          | 4-5/hpf         | 10-15/hpf        |
|                          | RBC                                        | 1-2/hpf         | Nil             | Nil              |
| S. Creatinine (mg/dl)    | 4.38                                       | 3.88            | 3.19            |                  |
| S. Electrolytes (mmol/L) | Na                                         | 141             | 138             | 139              |
|                          | K                                          | 4.49            | 4.47            | 3.8              |
|                          | CL                                         | 98              | 103             | 109              |
|                          | HCO <sub>3</sub>                           | 22              | 21              | 18               |
| S. IronProfile           | Iron                                       | 22 $\mu$ g/dl   |                 |                  |
|                          | TIBC                                       | 78 $\mu$ g/dl   |                 |                  |
|                          | TSAT                                       | 28.2%           |                 |                  |
|                          | Ferritin                                   | 3764.67 ng/ml   |                 |                  |
| ECG                      | No abnormality                             |                 |                 |                  |
| Chest X-Ray              | Features suggestive of bilateral pneumonia |                 |                 |                  |

During evaluation, his blood tests revealed anti-hepatitis B core (anti-HBc) total positivity with ALT: 49 U/L, AST: 38 U/L, ALP: 282 U/L, GGT: 177 U/L, increasing the possibility of underlying or past hepatitis B infection with hepatitis. HBsAg, HBeAg were negative, HBV-DNA was undetected, anti-HCV and anti-HIV 1 & 2 were negative, serum bilirubin: 0.6 mg/dl, serum albumin: 2.02 gm/dl. In plain x-ray abdomen, no features suggestive of intestinal obstruction or perforation (Figure 1). USG of whole abdomen was done and it revealed mildly enlarged liver (14cm) with non-uniform echotexture of hepatic parenchyma, dilated intrahepatic biliary channels, stomach wall was diffusely thickened. Non contrast CT scan of abdomen was done and it showed circumferential wall thickening of stomach, swelling of pancreatic tail and adhesion to stomach wall, and mild ascites was present. CEA: 4.7 ng/ml, CA 19.9: 98.4 U/ml. Initial upper GI endoscopy revealed ulceroproliferative growth at body of the stomach along lesser curvature (Figure 2). Biopsy was taken & histopathology showed chronic gastritis, no malignancy. We took consultation of department of gastroenterology and surgery. Repeat Upper GI

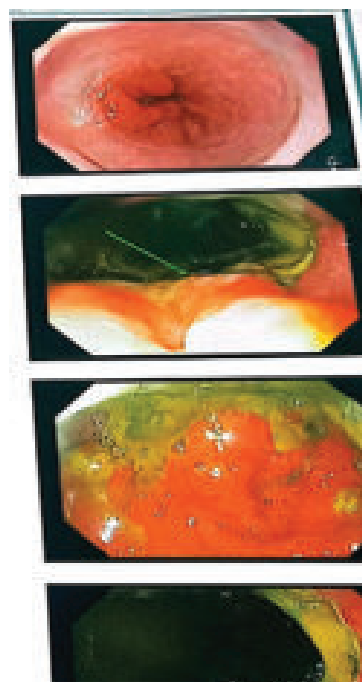


**Figure 1:** Plain X-ray abdomen

endoscopy was suggested by department of gastroenterology due to poor delineation of gastric mucosa. After proper gastric preparation, upper GI endoscopy was done and it revealed phytobezoar (Figure 3), which was removed and sent for histopathology and it showed fungal infection (mucormycosis) (Figure 4 & 5).

### Clinical course and outcome

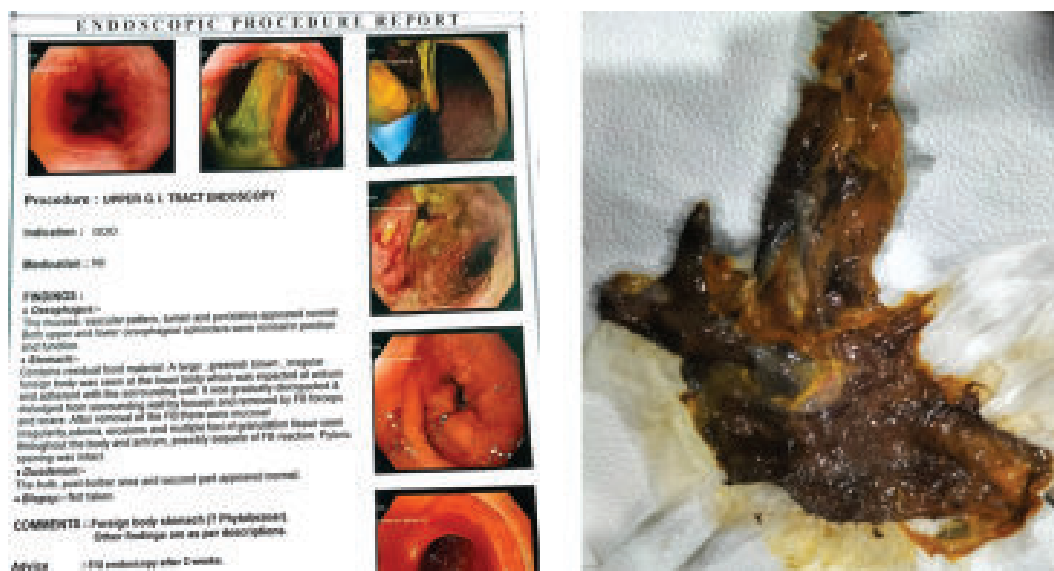
After removal of gastric bezoar, he developed per rectal bleeding, initially fresh then became clotted and features of volume overload including ascites and peripheral edema. The patient was managed conservatively with multiple blood transfusions and human albumin infusion. Following histological confirmation of gastric mucormycosis, the patient was treated with intravenous Liposomal Amphotericin B (5mg/kg/day), started from 20.1.2026 (6). As his clinical state worsened over time, he was shifted to the intensive care unit (ICU). Despite getting six dosages of Liposomal Amphotericin B, his condition deteriorated progressively, and he experienced severe gastrointestinal bleeding, in the form of hematemesis and hematochezia with significant hemodynamic instability and then the patient passed away.



### Findings:

There is a ulceroproliferative growth seen at body of the stomach along lesser curvature  
Comment: ?Carcinoma Stomach

**Figure 2.** Initial upper GI endoscopy report



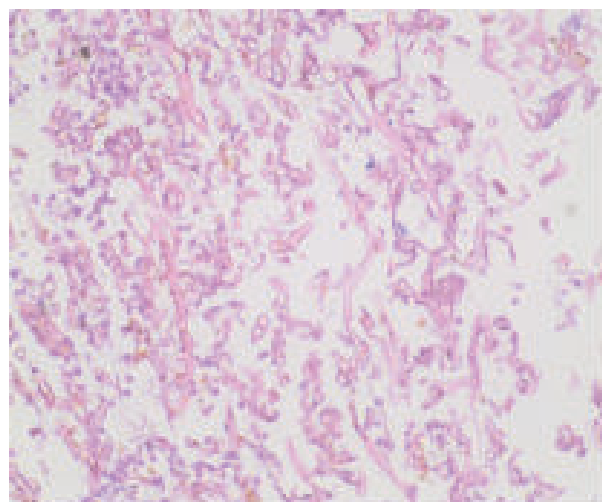
**Figure 3:** *a. Repeat upper GI endoscopy report b. Bezoar*

|                                 |                                                                                                                                                                                                                                                   |
|---------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Clinical information:</b>    | <b>? Stomach bezoar</b>                                                                                                                                                                                                                           |
| <b>Specimen:</b>                | <b>Tissue from stomach</b>                                                                                                                                                                                                                        |
| <b>Gross examination:</b>       | Specimen received in formalin consists of a tan-brown flat piece of tissue, measuring 14x9.5x0.2 cm.<br>One surface is smooth and another surface is tan-yellowish as well as lacerated.                                                          |
| <b>Representative blocks:</b>   | Submitted in three blocks; A1-A3 x6                                                                                                                                                                                                               |
| <b>Microscopic appearances:</b> | Sections show degenerated fibrocollagenous material.<br>These show plenty of fungal hyphae.<br>These hyphae are broad, having no segmentation.<br>There are also some spores.<br><br>No evidence of malignancy is seen in the available material. |
| <b>Diagnosis:</b>               | <b>Tissue from stomach, endoscopic biopsy:<br/>Fungal infection (Mucormycosis).</b>                                                                                                                                                               |

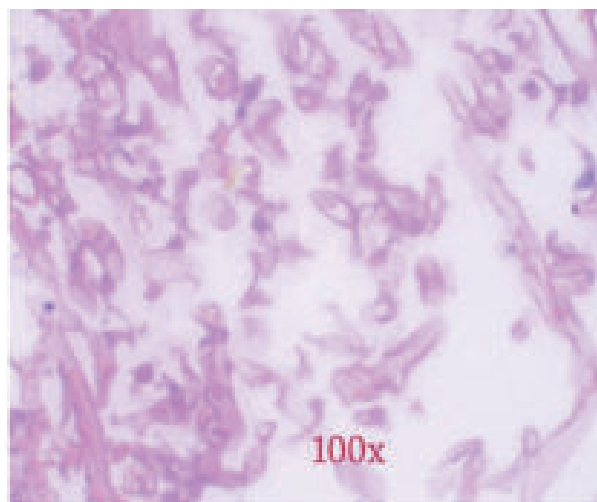
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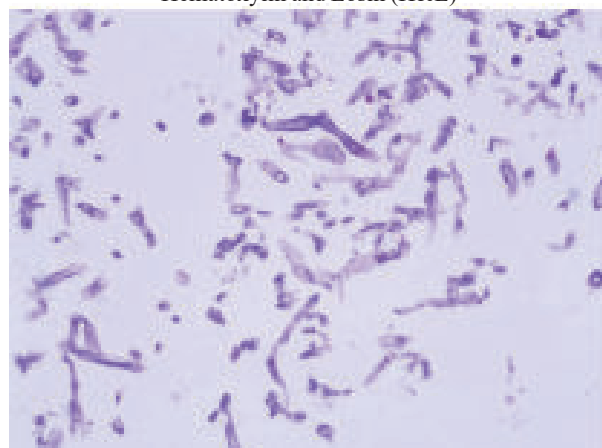
**Figure 4:** *Histopathology report of bezoar*



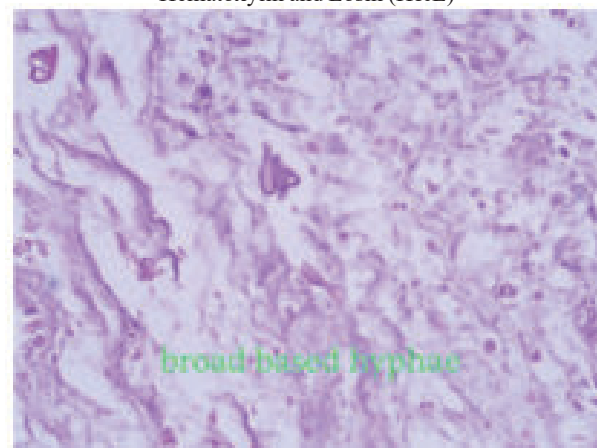
Hematoxylin and Eosin (H&amp;E)



Hematoxylin and Eosin (H&amp;E)



Periodic Acid Schiff (PAS): Weak Positive Hyphae



Periodic Acid Schiff (PAS): Broad Based Hyphae

**Figure 5:** *Light Microscopic Images of Fungal Hyphae*

### Discussions

Gastrointestinal mucormycosis is a rare but highly aggressive form of invasive fungal infection, accounting for only a small proportion of mucormycosis patients but with fatality rates of 50-70%. The stomach is the most commonly affected site in the gastrointestinal tract, followed by the colon and ileum. Diagnosis is usually delayed because clinical symptoms are ambiguous and may resemble more common illnesses such as peptic ulcer disease, gastric cancer, or gastric outlet obstruction.<sup>1,2,5</sup> Patients with diabetes and chronic kidney disease are more susceptible to mucormycosis due to reduced neutrophil activity, altered cellular immunity, metabolic acidosis, and increased availability of free iron, all of which favour fungal growth<sup>3,5,7</sup>. Multiple risk factors were present in this patient, including long-standing diabetes, CKD,

potential mucosal irritation from alternative medicines and ingested plant materials, critical illness with sepsis, prolonged hospitalization, and broad-spectrum antibiotic exposure.

Radiological signs in gastric mucormycosis are typically nonspecific, with stomach wall thickening, mass-like lesions, or characteristics indicative of malignancy or pancreatitis<sup>4,8</sup>. The presence of circumferential stomach wall thickening, swollen pancreatic tail with adhesion to stomach wall, and ascites in our patient raised the possibility of an infiltrative or malignant disease, a pattern that has been observed in published series<sup>9</sup>.

Endoscopic findings vary and may include ulceration, necrotic tissue, black eschar, mass-like lesions, or bezoars. Importantly, initial endoscopic biopsies may be mistakenly

negative because fungal invasion might be patchy, submucosal, or obscured by necrotic debris<sup>1,7</sup>. Despite the severe stage of the disease, the first biopsy merely revealed chronic gastritis. Repeat endoscopy following sufficient stomach preparation was critical in finding a large phytobezoar, which eventually led to histological diagnosis.

The relationship between bezoar formation and mucormycosis is uncommon but clinically significant. Bezoars may provide as a nidus for fungal growth, especially in patients with decreased stomach motility, altered digestion, or the consumption of indigestible plant material. In contrast, invasive fungal infection may decrease stomach motility and encourage bezoar development, resulting in a vicious cycle that delays detection<sup>8,10</sup>.

Histopathology remains the gold standard for diagnosis, with broad, ribbon-like aseptate hyphae penetrating tissue and right-angle branching. Culture yields are generally low, and negative cultures do not rule out disease<sup>1,2</sup>. Therefore, clinical suspicion accompanied with histological confirmation is crucial.

Management of gastrointestinal mucormycosis necessitates the immediate commencement of antifungal medication, usually with Liposomal Amphotericin B, as well as rigorous surgical debridement where possible. Delays in diagnosis and treatment are substantially linked to poor outcomes<sup>7,11</sup>. Unfortunately, in many cases, including ours, diagnosis occurs late in the disease course, when patients are critically ill and therapeutic options are restricted.

This case emphasizes the need of keeping a high level of suspicion for invasive fungal infections in high-risk patients who present with chronic gastrointestinal symptoms, unexplained anemia, or obstructive characteristics, especially if early investigations are equivocal. Repeat endoscopy, deeper tissue collection, and multidisciplinary teamwork are critical for increasing diagnostic yield and patient outcomes<sup>1,5,7</sup>.

#### Learning points

- Patients with diabetes mellitus and chronic kidney disease with persistent vomiting, unexplained anemia, or gastric outlet-obstruction like symptoms should be screened for stomach mucormycosis, a rare but often fatal illness.
- Radiological and endoscopic signs may mimic cancer or bezoar, causing diagnostic delays.
- Repeating endoscopic biopsies with proper preparation and removal of blocking debris is crucial to avoid false-negative results.
- Histopathological identification of wide, aseptate fungal hyphae is the gold standard for diagnosis.

- Early detection is crucial for a better prognosis.

#### Conclusion

This case demonstrates gastric mucormycosis presenting as a phytobezoar with false-negative first biopsies. A high level of suspicion, repeat endoscopy, and histological confirmation are critical for prompt diagnosis, particularly in individuals with diabetes and renal disease.

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