

Case Report

Melena in a Diabetic : Unveiling the Lurking Danger

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Background : Non variceal bleed accounts for 6-8% of admissions in India. Most common cause is peptic ulcer secondary to *Helicobacter pylori*. We report a case of Gastric Mucormycosis with Upper Gastrointestinal Bleed.

Case Report : An elderly female, uncontrolled diabetic with presented with acute onset Nausea, Vomiting and Fatigue. Examination revealed hypotension, hyperglycemia, low Glasgow coma scale requiring intensive care, mechanical ventilation, insulin and vasopressors. On investigation, Leucocytosis, Acute Kidney Injury, Glycated Hemoglobin - 11.6% were noted. Cultures were sterile. On day 7, patient developed 5 episodes of melena. Esophagogastro-duodenoscopy revealed ulcero-proliferative friable growth along the greater curvature of the stomach, suspicious of malignancy. Biopsy revealed broad branching filamentous structures resembling mucor. Despite antifungal therapy, patient succumbed to the disease.

Conclusion : Gastrointestinal Mucormycosis is rare and most commonly involves Stomach (58%), Colon (32%), Ileum, Duodenum and Jejunum. Management involves risk factor control, antifungals and surgery.

[J Indian Med Assoc 2025; 123(2): 49-50]

Key words : Mucor, Melena, Diabetic Ketoacidosis.

At least 5% of all hospital admissions from the Emergency Department in India is due to Upper Gastrointestinal Bleed (UGIB) which can be a variceal or non-variceal. Non-variceal UGIB accounts for 6-8% of hospital admissions in India¹. Peptic ulcer disease caused by *Helicobacter pylori* is the most common cause of non-variceal UGIB followed by erosive gastritis, erosive esophagitis, mucosal tears in the esophagus or fundus-Mallory Weiss tear, Dieulafoy lesion, Gastric Cancer². We hereby report an interesting case of non-variceal UGIB in an elderly female due to a rare infection.

CASE REPORT

A 75 year elderly lady presented to the Emergency Department in a state of altered sensorium with history of multiple episodes of non-projectile vomiting and generalised fatigue since 4 days. Her past history revealed Type 2 Diabetes Mellitus and systemic hypertension. Relatives reported that she was not compliant to therapy and was not on regular follow-up.

Examination revealed a low Glasgow Coma Scale (GCS) of E₁V₂M₂, hypoxia (SpO₂-75%) with signs of respiratory distress, hypotension (70/50 mmHg) and blood glucose of 465mg/dl. She was afebrile. Rest of the examination was unremarkable. Patient was intubated and mechanically ventilated, Initiated on Intravenous (IV) fluids, vasopressor/ionotropic support, insulin infusion, empirical IV antibiotics following admission to Intensive Care Unit.

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Received on : 28/04/2023

Accepted on : 02/08/2023

Editor's Comment :

- Even if the number of COVID-19 cases has significantly decreased, Mucor continues to pose a challenge to the medical community.
- It is paramount that every diabetic, especially those with uncontrolled sugars and those presenting with Diabetic ketoacidosis, be examined for the presence of this treatable yet potentially fatal infection.

A provisional diagnosis of Diabetic Ketoacidosis was made.

Blood parameters revealed a haemoglobin of 15g/dl, Neutrophilic Leucocytosis (20,500/cu mm) with Acute Kidney Injury (AKI) (1.77mg/dl), hyperkalemia of - 8.99mEq/L, high anionic gap metabolic acidosis, hepatocellular pattern of transaminitis (AST/ALT- 2079/783) and glycated hemoglobin (HbA1C)- 11.6%. Viral markers including anti- HIV, HBsAg and anti-HCV were negative. Blood and urine cultures yielded no growth. Chest radiograph was normal. Ultrasonography of abdomen revealed Grade I nephropathy and gall bladder sludge. During Intensive Care Unit (ICU) stay, patient's glycemic control was closely monitored and insulin was titrated accordingly. Anti-hyperkalemic measures were administered. Vasopressors were tapered and stopped. Her general condition gradually improved and she was planned for extubation.

However, on day 7 of hospital stay, patient developed 5 episodes of Melena with no hematochezia or hematemesis. Repeat haemoglobin showed a drop from 15g/dl to 9g/dl. Repeat platelet and coagulation profile were normal. Emergency upper Gastrointestinal (GI) endoscopy (Fig 1) revealed an ulcero-proliferative friable growth at the greater curvature of the stomach suspicious of malignancy, biopsy was obtained to confirm the diagnosis. Patient was treated with proton pump inhibitors.

However, patient continued to have episodes of Melena on and off. Subsequently, gastric histopathology (Fig 2) showed a non-specific ulceration with fungal growth by broad branching filamentous structures resembling Mucormycosis focally eliciting a giant cell response. Contrast enhanced Computed Tomography (CT) of the abdomen and pelvis was planned but deferred in view of recent AKI. Patient was initiated on IV liposomal amphotericin therapy (5mg/kg) and planned for surgical debridement. However, patient developed refractory shock and had sudden cardiac arrest and succumbed to the illness.

DISCUSSION

Fungal infections causing UGIB is rare. Mucormycosis is a highly invasive and progressive fungal disease with significant mortality and morbidity³. Conditions associated with greater risk of acquiring infection include uncontrolled diabetes and diabetic ketoacidosis, immuno-compromised states (Acquired immuno-deficiency syndrome, malnutrition), immuno-suppression (steroid use, post-transplant states and neutropenia). Mucormycosis is categorised into six clinical syndromes with rhino-orbital cerebral disease being the commonest form (39%). Pulmonary (24%), Cutaneous (19%), Cerebral (9%), Gastrointestinal (7%), Disseminated (3%) and Renal (2%) comprise the other systems that are involved^[3]. The mortality rate depends on underlying patient condition, type of fungus and body site involved. There have been very few reports of GI infection with mucor. Most frequently affected part of the GIT is stomach (58%) and then the colon (32%), ileum, duodenum and jejunum. The pathology varies from peptic ulcer colonisation to angio-invasion and dissemination. Clinical manifestations are protean ranging from Fever, Nausea, Non-specific Abdominal Pain and Vomiting to hematemesis, Melena, Hematochezia, or Gastrointestinal Perforation and Even Death^{1,3}. High mortality is due to GI perforation and massive bleeding. Definitive diagnosis involves targeted surgical or



Fig 1 — Endoscopic images showing ulceroproliferative friable growth along the greater curvature of the stomach

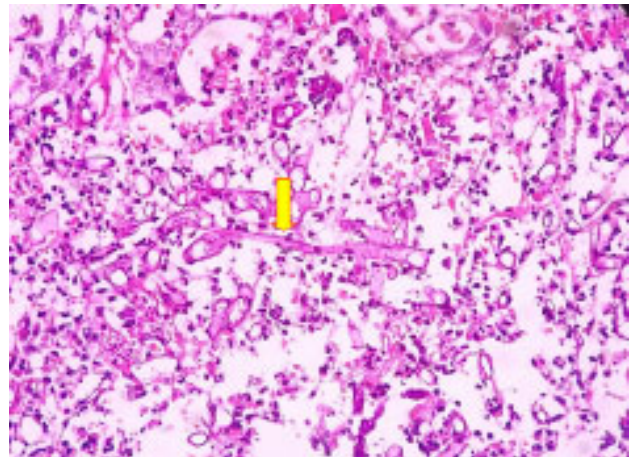


Fig 2 — Hematoxylin and Eosin stain of fundic growth biopsy revealing fungal growth by broad branching filamentous structures resembling Mucormycosis

endoscopic biopsy and histopathological identification of the organism showing characteristic ribbon shaped thick walled, aseptate hyphae⁴. CT can be used to aid diagnosis. Successful management includes aggressive Blood Sugar control, normal acid base status, antifungal therapy with Amphotericin B or Posaconazole and surgical debridement of all necrotic tissue^{4,5}. Surgical debridement is an independent factor determining survival. Prolonged treatment upto 3-6 weeks is often required to achieve cure and should be individualised to every patient. Nonsiderophore iron chelators, hyperbaric oxygen, cytokine therapy are other treatment options.

With the rise in COVID-19 cases and the associated use of steroids, treating clinicians must consider Mucor as a precipitant for diabetic ketoacidosis in uncontrolled diabetes. Frequent occurrence of this infection warrants high degree of suspicion, early targeted biopsy, risk factor screening and timely commencement of treatment with polyene antifungals while waiting biopsy confirmation.

Conflict of Interest : None

Financial Support : None

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