



ISOLATED GASTRIC MUCORMYCOSIS: AN INTERESTING CASE

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ABSTRACT

Mucormycosis (previously referred to as zygomycosis) is a rare but fatal infection caused by fungal species of the order Mucorales, family Mucoraceae. It is usually seen in immunocompromised individuals, such as those suffering from diabetes, recipients of organ transplants or patients on chemotherapy. Depending on the site affected, the disease can be pulmonary, cutaneous, rhinocerebral, gastrointestinal or disseminated. We present the case of a 40-year old male who was a known chronic alcoholic and was admitted with haematemesis from oesophageal varices for which endoscopic banding was done. However, his condition continued to deteriorate. He subsequently succumbed and a gastric ulcer due to mucormycosis was found among other observations.

KEYWORDS : Mucomycosis, Gastric ulcer, Chronic alcoholic**INTRODUCTION**

Rhizopus, Lichtheimia and Mucor are the most common species recovered from specimens taken from patients (Lass-Flörl, 2009; Prabhu & Patel, 2004; Ribes, Vanover-Sams, & Baker, 2000). Organisms belonging to the order Mucorales are ubiquitous-found in soil, faecal and decaying vegetable matter. 8.3%-13% of fungal infections found at autopsy in immunocompromised patients are found to be mucormycoses (Chamilos et al., 2006; Hotchi, Okada, & Nasu, 1980).

The incidence of gastrointestinal mucormycosis has been increasing since the past few decades- the most common site being the stomach, followed by the colon and ileum.

CASE REPORT

A 40-year-old man, alcoholic for 5-6 years, was admitted in a private hospital following a complaint of coffee ground coloured vomitus, mild abdominal pain and malaena. On admission, his haemoglobin was 6.0 gm/dl; total count 13,800/ μ l; platelet 1.65 lakh/ μ l; random blood sugar 240.8 mg/dl, urea 60.8 mg/dl, blood urea nitrogen 28.41 mg/dl, creatinine 1.0 mg/dl, total bilirubin: 0.9mg/dL, direct bilirubin: 0.3mg/dL SGOT 73.3 U/l, total protein 5.3 g/dl, albumin 3.0 g/dl, stool occult blood positive. Upper GI scopy showed oesophageal varices with clot in fundus. Variceal banding was done. In view of continued episodes of haematemesis, he was referred to our hospital after 5 days of stay in a local hospital. On admission, general condition was poor, BP 60 mmHg systolic. He succumbed within 40 minutes of stay at our hospital. A complete autopsy was performed to ascertain the final cause of death.

At autopsy, external examination showed no icterus or abdominal distention. On in-situ examination, there was no free fluid in the abdominal cavity. Systemic examination revealed oesophagus with blood clots in the lumen and 5 oval lesions of variceal banding (Fig 1). Stomach: serosa was unremarkable, no perforation seen, wall was thickened; altered blood and a 100gm blood clot noted with a 5x4cm single, irregular ulcer at the greater curvature in the body, floor was haemorrhagic (Fig 2). Liver was shrunken in size with vague nodularity seen on capsular and cut-surface (Fig 3). Lung showed smooth, shiny pleura; frothy fluid oozed out on pressure (suggestive of pulmonary oedema) (Fig 4).

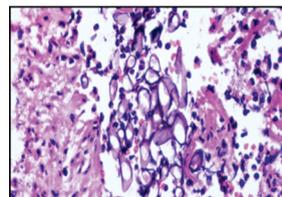
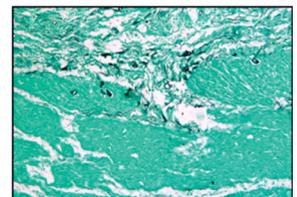
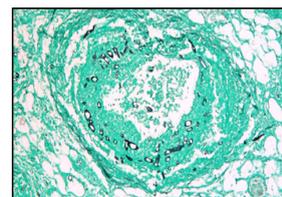
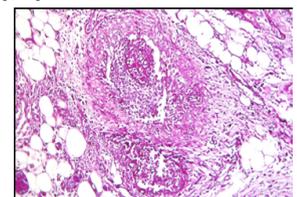
Histological findings:

Sections from the stomach showed an ulcer with extensive transmural neutrophilic inflammation. Also seen were areas of necrosis and transmural as well as vascular infiltration by broad, non-septate, non-branching fungal hyphae (Figs 5,6,7,8).

Sections from oesophagus also showed ulceration. Sections from

liver showed evidence of cirrhosis. No fungal hyphae were seen in these or in sections taken from other organs.

The final cause of death was: Hypovolaemic shock due to upper gastrointestinal bleed in a case of oesophageal varices and gastric ulcer (mucormycosis) (Natural).

**Fig 1: Oesophagus showing oval banded ulcers****Fig 2: Thickened stomach wall with irregular ulcer****Fig 3: Liver with vaguely nodular areas****Fig 4: Lung with smooth, shiny pleura; no focal lesion****Fig 5: Ulcer: fungal hyphae with surrounding inflammation (40x)****Fig 6: Gomori methenamine silver (GMS) stain showing hyphae infiltrating muscularis propria****Fig 7: Fungal hyphae showing angioinvasion (GMS)****Fig 8: Periodic acid Schiff (PAS) stain: fungal hyphae showing angioinvasion**

DISCUSSION:

Infection by *Mucorales* spp. occurs from sporangiospores that are either inhaled or directly inoculated in the disrupted mucosal surface, skin or intestine (Spellberg, 2012). These are followed by angioinvasion, formation of thrombus, tissue infiltration and necrosis (LEHRER et al., 1980).

Of all the cases of mucormycosis, gastrointestinal infections account for 7% (Vera, Hubscher, McMaster, & Buckels, 2002). These cases are seen in patients with haematological malignancies, recipients of organ transplant, diabetics, patients on long-term corticosteroids (Bullett, Andreu, Elias, Roig, & Cervantes, 1993).

Chronic alcoholism has not been reported to be a risk factor. However, gastric mucormycosis in alcoholics has been reported in 3 cases, besides ours, (Ho, Wu, Chen, & Wang, 2007; Park et al., 2002; Shahapure, Patankar, & Bhatkhande, n.d.). All these patients presented with abdominal pain. One had fever and vomiting. Another case had complaints of malaena and haematemesis, similar to our case. Ulcerated lesions with necrotic bases and raised edges were seen on endoscopy. In our case, endoscopy of the stomach did not identify the ulcer probably due to the presence of bleeding varices.

If reported on endoscopy, the diagnosis of mucormycosis could have been done by biopsy and special stains that show broad, aseptate fungal hyphae. In such a case antifungal treatment could have been added to the treatment. However, as the patient was unstable due to the continuous bleeding, this was not possible.

Although the association is not common, awareness of this potential pathogen may help clinicians in better managing such patients.

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