CASE REPORT

Postmortem Diagnosis of Fatal Gastric Mucormycosis in a Young Male

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Abstract

Background: Gastric mucormycosis is a rare and highly aggressive fungal infection, typically seen in immunocompromised individuals. Its nonspecific clinical presentation often leads to delayed diagnosis and poor outcomes. Pediatric cases, particularly without clear risk factors, are exceedingly uncommon.

Case Presentation: We report the postmortem diagnosis of gastric mucormycosis in a 13-year-old male who presented with undifferentiated fever and altered consciousness. The patient died before a definitive diagnosis could be made. Autopsy revealed significant gastric mucosal necrosis and ulceration. Histopathological examination with PAS and GMS stains demonstrated broad, non-septate, angioinvasive fungal hyphae with intravascular thrombosis—hallmarks of mucormycosis. No fungal involvement was found in other organs, suggesting primary gastric infection.

Discussion: This case highlights the diagnostic challenge of gastric mucormycosis, particularly in a young, seemingly immunocompetent host. While commonly associated with diabetes, malignancy, or immunosuppression, our patient lacked overt predisposing factors, though borderline low blood counts suggested a possible undiagnosed immunocompromised state. The aggressive course and fatal outcome underscore the importance of maintaining a high index of suspicion in atypical presentations.

Conclusion: Gastric mucormycosis, though rare, should be considered in unexplained systemic deterioration, even in young patients without clear risk factors. Early diagnosis through histopathology remains critical for timely, life-saving intervention.

Keywords: Angioinvasion, Gastric mucormycosis, Invasive fungal infection, Postmortem diganosis.

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Introduction

Mucormycosis is a rare, opportunistic, and highly aggressive fungal infection caused by moulds of the order Mucorales.[1–4] While it is most commonly reported in immunocompromised patients, there has been an increasing number of cases seen in seemingly immunocompetent hosts.[2] Rhinocerebral and pulmonary forms are the most frequent presentations.[1,3–5] Gastrointestinal (GI) involvement is considered uncommon, reported in approximately 5% to 15% of cases, though the incidence in adults has been reported to be on the rise.[1,2,4,5] Within the GI tract, the stomach is the most commonly affected organ, followed by the colon and ileum.[1,2,4–6]

Gastric mucormycosis is a particularly challenging diagnosis due to its often non-specific clinical presentation, which can delay identification and treatment, leading to poor outcomes.[1,2,5] The angioinvasive nature of the fungus, causing thrombosis and subsequent tissue necrosis, is a hallmark pathological feature that contributes significantly to morbidity and mortality, often resulting in severe complications such as massive bleeding and perforation.[1,4,5,7,8] Mortality rates for GI mucormycosis are reported to be very high, approximately 85% in some series.[1,4,5]

Diagnosis typically relies on histopathological evidence of fungal invasion in tissue biopsies obtained via endoscopy or surgery.[1,2,5,6] Antemortem diagnosis is infrequent, with some reports indicating that only about 25% of cases are diagnosed before death.[5] We report a rare case of fatal gastric mucormycosis diagnosed postmortem in a 13-year-old male patient, highlighting the aggressive nature of this infection and the difficulties in clinical diagnosis, particularly in individuals without readily apparent risk factors and in younger age groups.

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Case Presentation

A 13-year-old male patient, of average build and nourishment, was admitted to the hospital with a clinical picture suggestive of undifferentiated fever, unconsciousness, and suspected brainstem injury. The specific details of his hospital course leading up to his death are not available, but he succumbed

to his illness due to reasons that remained unknown prior to the postmortem examination.

The patient's age is notable, as while gastric mucormycosis has been reported in premature neonates and malnourished children, primary gastric involvement in older children or adults is often associated with specific underlying risk factors.[1,2,4,8] The clinical presentation of undifferentiated fever and neurological signs did not immediately point towards a primary gastric pathology, further complicating antemortem diagnosis.[1–3,6]

Postmortem Examination Findings

An autopsy was performed on the patient to ascertain the cause of death:

External Examination: External examination revealed the presence of rigor mortis in all limbs. Postmortem lividity was fixed in dependent parts of the body. There were no external signs of icterus, cyanosis, lymphadenopathy, or pedal oedema.

Internal Findings: Upon internal examination, the brain weighed 1.4 kg and showed signs of cerebral oedema. Macroscopically, there was no evidence suggestive of meningitis or encephalitis. Other major organs, including the heart, spleen, and bone marrow, appeared histologically unremarkable. There was no evidence of haematological malignancy or systemic bacterial infection. Blood counts obtained prior to death were reported to be on the lower side, which, in retrospect, suggested the possibility of a fungal sepsis.

Stomach Findings: The stomach was a key organ with significant pathological findings. Grossly, the gastric mucosa exhibited necrotic and ulcerative lesions (Figures 1 & 2). Such lesions are frequently observed in gastric mucormycosis and are a result of the fungal invasion.[1,2,4]

Histopathological examination was crucial for diagnosis. Sections taken from the stomach were stained using Periodic acid-Schiff (PAS) and Gomori's methenamine silver (GMS) stains. These special stains are known to highlight fungal elements.[1,4,7] Microscopic analysis of these stained sections revealed the presence of fungal hyphae. The hyphae were described as broad, non-septate, and ribbon-like.[1–4,5,7] Importantly, the histopathology demonstrated angioinvasion and fibrin thrombi within the gastric vessels. Angioinvasion, the invasion of blood vessel walls by fungal hyphae, is a critical pathological feature of mucormycosis that leads to thrombosis, tissue infarction, and necrosis.[1,4,5,7,8] The presence of thrombi within the vessels is a direct consequence of this process.[2,4,5] The combination of these histological features – broad, aseptate hyphae and angioinvasion – is characteristic of Mucorales infection.[1,2,4,5,7]

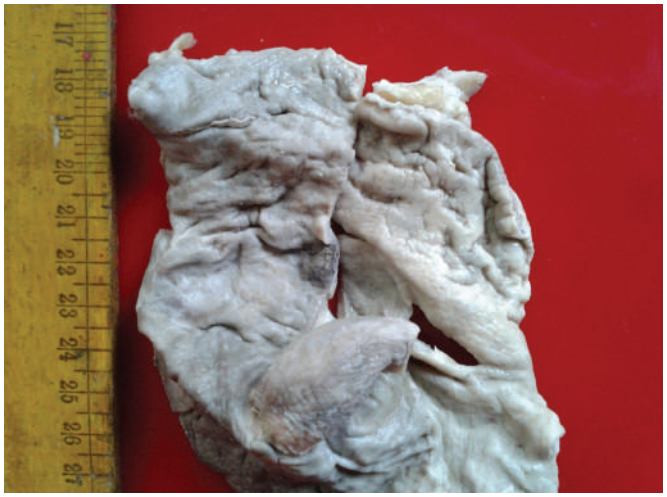

Figure 1: Gross: Cut opened stomach showing a ulcer.

Figure 2: Gross: Cut opened stomach showing an ulcer measuring 1.5 x 0.8 cm.

Examination of other organs, including the lungs and brain, did not reveal any fungal invasion, suggesting that the infection was primarily located in the stomach.[1,4,5] Based on the distinctive histopathological findings in the gastric tissue, particularly the presence of broad, non-septate, angioinvasive hyphae of Mucorales and associated vascular thrombosis, the final diagnosis of fatal gastric mucormycosis was established postmortem.

Discussion

This case underscores the insidious nature and high mortality associated with gastric mucormycosis, particularly when the diagnosis is delayed. The presentation with undifferentiated fever and neurological symptoms, without overt GI complaints or clear predisposing risk factors, contributed to the diagnostic challenge in this young patient.

Gastric mucormycosis typically arises in individuals with certain risk factors that compromise their immune defences.[1,2,6] The most common risk factors documented in the literature include:

- **Uncontrolled Diabetes Mellitus**, especially with ketoacidosis.[1,3–5,8]

- **Immunosuppression** due to conditions like neutropenia, haematological malignancies, solid organ or stem cell transplantation, and corticosteroid therapy.[1–6]
- **Chronic Renal Failure** and patients undergoing dialysis.[2,4]
- **Alcoholism**, including alcoholic liver disease or cirrhosis.[1,2,4]
- **Malnutrition**. [1,2,4]
- **Iron Overload** and deferoxamine therapy.[1,4,8]
- **Major Trauma**, including penetrating abdominal injury and burns.[6,8]
- **HIV/AIDS**.[4,8]

- **Hemophagocytic Lymphohistiocytosis (HLH)** is also reported as a risk factor.[30]

While primary gastric mucormycosis has been reported in neonates and malnourished children,[1,2,4] it is less common in the early adolescent age group (13-years-old) without clear, pre-existing severe immunocompromise or other typical risk factors. The patient was described as averagely built and nourished, and the autopsy found no haematological malignancy or evidence of systemic bacterial infection. However, his blood counts being on the lower side could hint at an underlying, undiagnosed immunocompromised state that was not clinically apparent or specifically investigated before his death. The absence of obvious risk factors is noted as unusual in some case reports of gastric mucormycosis.[3]

The clinical presentation of gastric mucormycosis is notoriously non-specific.[1,2,5] Common symptoms include abdominal pain, nausea, vomiting, and fever.[1] More severe presentations involve gastrointestinal bleeding (haematemesis, melaena, haematochezia) and potentially life-threatening perforation leading to peritonitis.[1,4,5] Gastritis-like symptoms refractory to standard treatment can also be an indicator.[3] This patient presented with fever and altered consciousness, symptoms that are not directly suggestive of primary gastric pathology, further highlighting the diagnostic difficulty.

Endoscopic findings often include gastric ulcers that may appear large, with necrotic bases, sharply demarcated edges, and signs of haemorrhage or oedema.[1,2,6] The gross findings of necrotic and ulcerative lesions in the stomach during the autopsy are consistent with these descriptions.

The definitive diagnosis of mucormycosis relies on the microscopic identification of characteristic fungal hyphae in affected tissue. Histopathology of gastric biopsies or resected tissue is the gold standard.[1,2] As seen in this case, the key features are the presence of broad (typically 6-25 µm), thin-walled, aseptate or sparsely septate hyphae (Figure 3), which often exhibit irregular or obtuse-angled branching (although branching angle was not specifically noted in this patient's autopsy description). The angioinvasive nature, leading to thrombosis and tissue necrosis, is pathognomonic [1,4,5,7,8] (Figures 4 & 5). The detailed autopsy findings in this patient, specifically the broad, non-septate, ribbon-like hyphae with angioinvasion and fibrin thrombi demonstrated by PAS

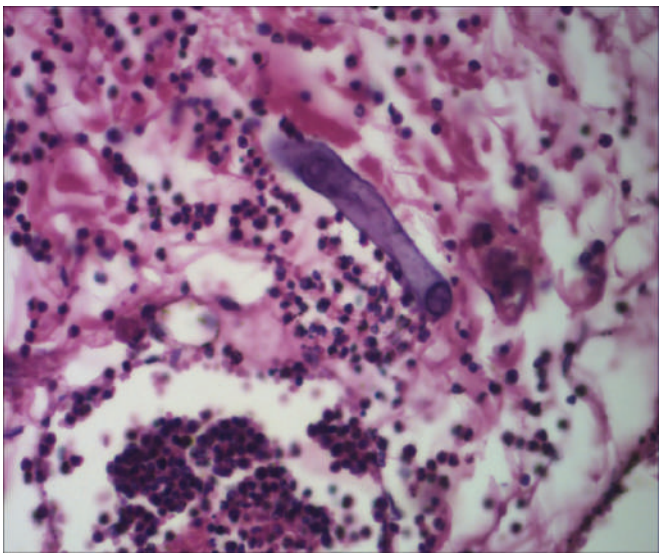

Figure 3: Microscopy: Showing fungal filaments broad septae with mixed inflammation.

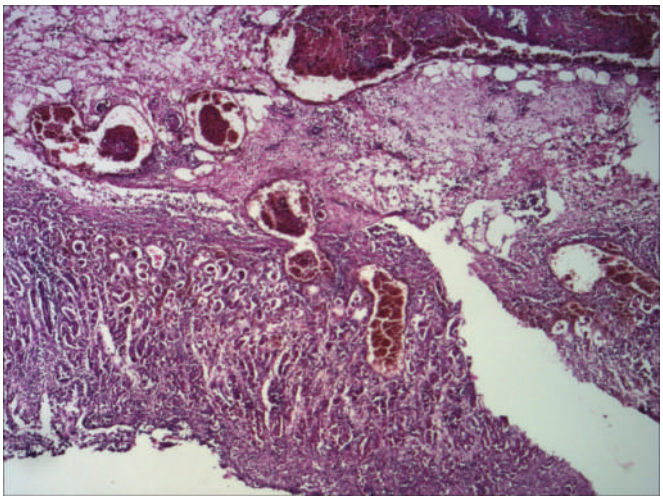

Figure 4: Microscopy shows Angioinvasion by fungal filaments with inflammation.

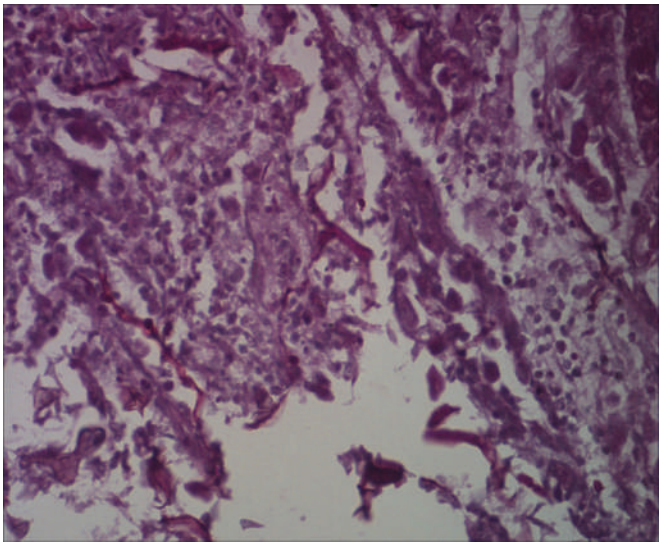

Figure 5: Microscopy shows broad septae in vessels s/o Angioinvasion by fungus.

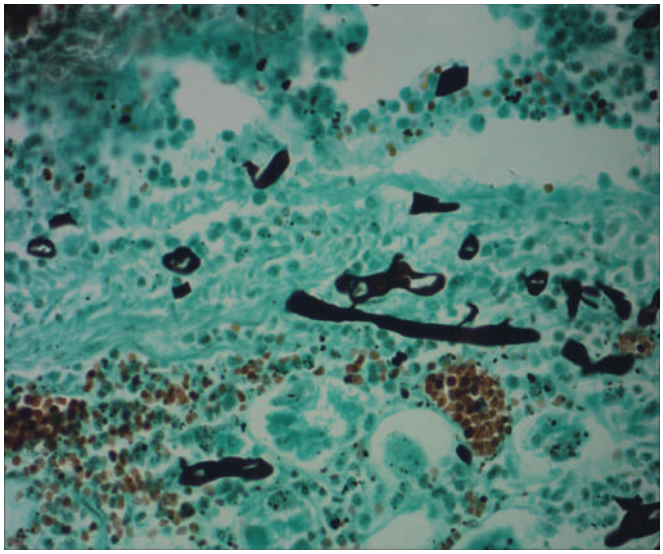

Figure 6: Microscopy shows broad septae special stain used GMS s/o Mucormycosis.

and GMS stains, precisely match the histological criteria for diagnosing mucormycosis (Figure 6). The lack of fungal invasion in other organs examined suggests primary gastric involvement, likely acquired via ingestion.[1,4,5]

The rapid progression and high fatality rate of mucormycosis necessitate prompt diagnosis and aggressive treatment, which typically involves a multidisciplinary approach combining surgical debridement of infected tissue with systemic antifungal therapy, most commonly with liposomal amphotericin B.[1,3–5,7] Surgical resection of necrotic areas is crucial because the angioinvasion causes poor penetration of antifungal agents into the affected tissue.[1,7,8] Delayed initiation of appropriate therapy is strongly associated with poor outcomes.[1,4–6] Unfortunately, in this case, the diagnosis was made after the patient's death, precluding timely intervention.

This case highlights the importance of maintaining a high index of suspicion for invasive fungal infections, including mucormycosis, even in seemingly atypical presentations or in patients without overt, classical risk factors.[3,4] While rare, swift diagnosis via histopathology is paramount to enabling potentially life-saving medical and surgical treatment. This case serves as a stark reminder that fatal outcomes can occur rapidly if this aggressive infection is not identified early.

Conclusion

We report a fatal case of gastric mucormycosis in a 13-year-old male, diagnosed postmortem. The patient presented with non-specific systemic symptoms and died before a clinical diagnosis of fungal infection could be established. Autopsy revealed severe necrotic and ulcerative lesions in the stomach, with characteristic histopathological findings of broad, non-septate, angioinvasive fungal hyphae consistent with Mucorales, along with vascular thrombosis. This case demonstrates that gastric mucormycosis can occur in younger patients and may present without the typical clinical features or obvious risk factors, leading to diagnostic delays and devastating outcomes. Histopathological examination remains the cornerstone for diagnosis. This report underscores the need for heightened awareness of this rare but deadly infection, particularly when facing unexplained clinical deterioration, and reinforces the critical role of postmortem examination in identifying unexpected causes of death.

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