

Gastric mucormycosis as a rare cause of gastric mucosal sloughing and gastrointestinal bleeding: A case report

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Case Report

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Abstract

Background

Gastric mucormycosis is a rare but fulminant invasive fungal infection associated with high mortality, particularly in immunocompromised individuals. Mucormycosis can induce gastric mucosal sloughing, ulcer formation, and subsequent gastric hemorrhage.

Case presentation

A 75-year-old male was admitted to hospital with pulmonary infection and sepsis. He had a history of psoriasis treated with secukinumab and apremilast. During hospitalization, he developed refractory upper gastrointestinal bleeding. Gastroscopy identified a large 4 cm × 5 cm mucosal detachment extending from the lower gastric body near the gastric antrum, spanning from the anterior wall to the posterior wall. The ulcer base showed probable muscularis propria exposure, diffuse oozing hemorrhage, and visible thrombi. Histopathological examination of endoscopic biopsy specimens confirmed gastric mucormycosis, revealing fungal elements consistent with Mucorales. Despite urgent treatment with liposomal amphotericin B and a subtotal gastrectomy, the patient suffered recurrent inflammation and persistent anemia, ultimately dying of progressive multiple organ failure one month postoperatively.

Conclusion

Gastric mucormycosis should be suspected in immunocompromised patients on long-term biologic agents who present with sepsis and unexplained gastrointestinal bleeding. Gastroscopic manifestations may include gastric mucosal sloughing and large, irregular, deep ulcerations. Early endoscopy, prompt antifungal therapy, and surgical debridement are critical for disease control.

Background

Mucormycosis is a life-threatening invasive fungal infection caused by ubiquitous fungi of the order Mucorales, which primarily affects immunocompromised human hosts[1]. Characterized by aggressive angioinvasion, tissue necrosis, and rapid disease progression, this infection confers an extremely high mortality rate despite standardized antifungal and surgical interventions. Gastrointestinal mucormycosis is the least common clinical subtype, accounting for only a small proportion of all reported cases[2]. Its lack of specific clinical manifestations in the early stage frequently causes missed diagnosis and delayed treatment. Here, we report a fatal case of invasive gastric mucormycosis in an elderly psoriasis patient receiving long-term combination biologic and immunomodulatory therapy, with the aim of characterizing the clinical, endoscopic, and histopathological features of this rare infection.

Case presentation

A 74-year-old male presented with a fever, generalized myalgia, and abdominal distension lasting for 3 days. His peak temperature reached 39.0°C. He denied obvious abdominal pain, diarrhea, sore throat, chest tightness, or dyspnea. He received empiric anti-infective and antipyretic symptomatic treatment at an outside hospital; however, his symptoms failed to improve. He was then admitted to the emergency department of our hospital for further evaluation. The patient had a more than 30-year history of psoriasis. His treatment regimen involved continuous secukinumab therapy over the past 3 years, with intermittent oral apremilast. Upon admission, the patient's vital signs were as follows: (1) Body temperature: 38.5°C; (2) Heart rate: 94 bpm; (3) Respiratory rate: 20 breaths/minute; and (4) Blood pressure: 92/54 mmHg. On auscultation, coarse breath sounds were heard bilaterally in both lungs.

Initial laboratory evaluations indicated leukocytosis, thrombocytopenia, inflammation, and multi-organ injury: white blood cell count (WBC): $11.16 \times 10^9/\text{L}$ (reference range: $3.5\text{--}9.5 \times 10^9/\text{L}$); neutrophil count: $10.30 \times 10^9/\text{L}$ (reference range: $1.8\text{--}6.3 \times 10^9/\text{L}$); lymphocyte count: $0.40 \times 10^9/\text{L}$ (reference range: $1.1\text{--}3.2 \times 10^9/\text{L}$); platelet count: $39 \times 10^9/\text{L}$ (reference range: $125\text{--}350 \times 10^9/\text{L}$); C-reactive protein (CRP): 201.10 mg/L (reference range: 0–5 mg/L); myoglobin: >1200.00 ng/mL (reference range: 10–92 ng/mL); high-sensitivity cardiac troponin-I: 0.580 ng/mL (reference range: 0–0.056 ng/mL); serum creatinine: 273.5 $\mu\text{mol}/\text{L}$ (reference range: 57–97 $\mu\text{mol}/\text{L}$); blood urea: 24.56 mmol/L (reference range: 3.1–8.0 mmol/L); serum albumin: 28.83 g/L (reference range: 40–55 g/L); total bilirubin: 29.40 $\mu\text{mol}/\text{L}$ (reference range: 0–26 $\mu\text{mol}/\text{L}$); and aspartate aminotransferase: 83.42 U/L (reference range: 15–40 U/L). Chest computed tomography revealed extensive patchy, high-density opacities in both lungs, suggestive of pulmonary infection.

Integrating the clinical history, imaging, and laboratory findings led to an initial diagnosis of sepsis secondary to a pulmonary infection, complicated by multiple-organ injury. Sputum and blood cultures were collected. Empiric broad-spectrum antimicrobial therapy with meropenem was initiated, alongside acid suppression, nutritional support, and other supportive measures. Blood cultures yielded methicillin-resistant *Staphylococcus aureus* (MRSA). Vancomycin was added for anti-MRSA therapy. Following treatment, the patient's inflammatory markers, including WBC and CRP, trended downward.

However, the patient developed melena accompanied by a progressive hemoglobin decline. Dark-red bloody fluid was drained via gastrointestinal decompression, raising high suspicion of upper gastrointestinal bleeding. Emergency gastroscopy identified a large 4 cm $\times$ 5 cm mucosal detachment extending from the lower greater curvature of the gastric body near the gastric antrum, spanning from the anterior wall to the posterior wall. The ulcer base showed probable muscularis propria exposure, diffuse oozing hemorrhage, and visible thrombi (Fig. 1). Four biopsy specimens were obtained from the surface and marginal mucosa of the ulcer. Histopathological examination demonstrated chronic active inflammation with ulceration and necrotic tissue. Fungal elements morphologically consistent with Mucorales were detected on the ulcer surface (Fig. 2). The final diagnoses included: (1) gastric

mucormycosis; (2) pulmonary infection; (3) sepsis; (4) multiple-organ injury (heart, liver, kidney); and (5) gastrointestinal bleeding.

After diagnosis of gastric mucormycosis, targeted antifungal therapy with liposomal amphotericin B (5 mg/kg/day) was initiated for mucormycosis. On the second day, subtotal gastrectomy was performed to achieve source control of the necrotic fungal nidus. Postoperatively, recurrent hemoglobin reduction and fluctuating inflammatory markers persisted. Approximately one month after surgery, the patient experienced an abrupt clinical deterioration. Despite aggressive resuscitation and intensive care, there was no clinical improvement, and the patient eventually died.

Discussion

This case report describes a fatal case of invasive gastric mucormycosis in an elderly patient with psoriasis receiving long-term biologic and immunomodulatory therapy.

Gastrointestinal bleeding in this patient resulted from local gastric mucosal necrosis, sloughing, and ulcer formation secondary to gastric mucormycosis. Mucormycosis is a rare, aggressive, invasive fungal infection caused by fungi of the order Mucorales. Mucorales organisms are ubiquitous in soil, air, and decaying organic matter. Human infection mainly occurs via spore inhalation, ingestion of contaminated food, or cutaneous traumatic inoculation. Based on anatomical sites of involvement, mucormycosis is conventionally classified into rhinocerebral, pulmonary, gastrointestinal, cutaneous, and disseminated forms[1]. Gastrointestinal mucormycosis accounts for 5%–13% of all mucormycosis cases[2, 3]. Owing to non-specific clinical manifestations and signs, gastrointestinal mucormycosis represents one of the most challenging forms to diagnose, with an overall mortality rate up to 40%, which may exceed 90% in disseminated cases[4, 5]. Gastric mucormycosis predominantly affects severely immunocompromised individuals, including patients with uncontrolled diabetic ketoacidosis, hematological malignancies, recipients of solid-organ or hematopoietic stem cell transplantation on long-term potent immunosuppressive agents, patients receiving long-term glucocorticoid therapy, and patients with disrupted cutaneous barriers secondary to trauma or burns[6].

The detrimental effects of Mucorales are multifactorial and synergistic, with angioinvasiveness serving as the core pathological hallmark. The broad, sparsely septate or aseptate hyphae of Mucorales can directly penetrate vascular endothelium and proliferate extensively within vessel lumina, triggering thrombosis and subsequent ischemic infarction and coagulative necrosis of involved tissues. This process generates a hypoxic, acidic microenvironment favorable for fungal proliferation and substantially impedes the delivery of antifungal agents to infectious foci via blood flow, thereby markedly attenuating pharmacological efficacy. Furthermore, ischemic necrosis rapidly disrupts native anatomical tissue barriers, facilitating rapid dissemination of infection along tissue planes, nerve tracts, or vascular bundles[7].

Following fungal invasion of the gastric mucosa, initial presentations may mimic non-specific gastritis or gastric ulcer, including epigastric pain, abdominal distension, nausea, vomiting, and upper

gastrointestinal bleeding. As the disease progresses, hyphae may infiltrate into the muscularis propria and even the serosa, leading to gastric perforation, full-thickness gastric wall necrosis, or life-threatening massive hemorrhage secondary to erosion of large blood vessels. Endoscopically, lesions often present as large, irregular, deep ulcers covered by gray-green necrotic exudates, with black necrotic or leather-like bases and a firm consistency during biopsy sampling[8–10]. Histopathological examination of endoscopic biopsy specimens remains the gold standard for diagnosis, requiring the identification of characteristic broad, aseptate, or pauciseptate hyphae with right-angle branching within tissue sections[11]. Gomori methenamine silver (GMS) and periodic acid-Schiff staining facilitate the visualization of these fungal structures[12].

Intravenous liposomal amphotericin B (5–10 mg/kg/day) is the globally accepted first-line therapeutic agent. Isavuconazole and posaconazole are important salvage or sequential therapy alternatives. The convenience of oral administration renders them suitable for long-term suppressive maintenance therapy [4, 12]. For gastric mucormycosis, timely surgical intervention constitutes a critical therapeutic modality. Partial or total gastrectomy may be indicated depending on disease extent. The surgical goal is complete resection of all devitalized, necrotic, and fungus-infiltrated tissues to minimize the overall fungal burden. Comparative studies have demonstrated significantly better survival among patients receiving combined surgical and antifungal therapy compared with those receiving monotherapy alone[2, 6].

Conclusion

Gastric mucormycosis, though rare, has a high mortality rate. Non-specific clinical manifestations pose substantial diagnostic and therapeutic challenges. Successful management relies on high clinical vigilance to achieve early diagnosis. For immunocompromised patients presenting with abdominal pain, fever, and gastrointestinal bleeding, prompt gastroscopy should be performed with heightened suspicion for gastric mucormycosis. Gastroscopic manifestations may include gastric mucosal sloughing and large, irregular, deep ulcerations. Treatment requires close multidisciplinary collaboration among infectious disease specialists, surgeons, intensivists, and clinical pharmacists to implement a comprehensive strategy comprising underlying disease control, surgical debridement or resection, and potent antifungal therapy.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Following review by the Medical Ethics Committee of the Affiliated Hospital of Qingdao University Hospital, the study was determined to meet the criteria for an ethics exemption and thus did not require formal ethical approval. The patient's relevant information has been de-identified to protect privacy.

Consent to participate

was not applicable for this single-case report.

Consent for publication

Written informed consent was obtained from the relatives of patient for publication of this case report and any accompanying images. A copy of the consent form is available for review by the Editor of this journal.

Competing interests

The authors declare that they have no conflicts of interest.

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Author Contribution

LB.L. and B.F. wrote the paper; P.Q. and B.C. designed the study and supervised the writing of the paper as co-corresponding authors; LB.L., KJ.X. and K.Z. collected clinical data and provided case information on patients; all authors have read and approved the final manuscript.

References

1. Kontoyiannis DP, Walsh TJ, Mucormycosis. *N Engl J Med*. 2026;394(7):684–98. 10.1056/NEJMra2412565.
2. Kaur H, Ghosh A, Rudramurthy SM, Chakrabarti A. Gastrointestinal mucormycosis in apparently immunocompetent hosts-A review. *Mycoses*. 2018;61(12):898–908. 10.1111/myc.12798.
3. Roden MM, Zaoutis TE, Buchanan WL, Knudsen TA, Sarkisova TA, Schaufele RL, et al. Epidemiology and outcome of zygomycosis: a review of 929 reported cases. *Clin Infect Dis*. 2005;41(5):634–53. 10.1086/432579.
4. Didehdar M, Chegini Z, Moradabadi A, Anoushirvani AA, Tabaeian SP, Yousefimashouf M, et al. Gastrointestinal mucormycosis: A periodic systematic review of case reports from 2015 to 2021. *Microb Pathog*. 2022;163:105388. 10.1016/j.micpath.2022.105388.
5. Liang M, Xu J, Luo Y, Qu J. Epidemiology, pathogenesis, clinical characteristics, and treatment of mucormycosis: a review. *Ann Med*. 2024;56(1):2396570. 10.1080/07853890.2024.2396570.
6. Dioverti MV, Cawcutt KA, Abidi M, Sohail MR, Walker RC, Osmon DR. Gastrointestinal mucormycosis in immunocompromised hosts. *Mycoses*. 2015;58(12):714–8. 10.1111/myc.12419.

7. Frater JL, Hall GS, Procop GW. Histologic features of zygomycosis: emphasis on perineural invasion and fungal morphology. *Arch Pathol Lab Med.* 2001;125(3):375–8. 10.5858/2001-125-0375-HFOZ.
8. Chang CF, Huang TY, Lin TY. Unusual giant gastric cardia ulcer. *Intern Emerg Med.* 2018;13(5):795–6. 10.1007/s11739-018-1814-1.
9. Chhaya V, Gupta S, Arnaout A. Mucormycosis causing giant gastric ulcers. *Endoscopy.* 2011;43 Suppl 2 UCTN:E289-90. 10.1055/s-0030-1256425
10. Metussin D, Telisinghe PU, Chong PL, Chong VH. Gastrointestinal: Gastric mucormycosis. *J Gastroenterol Hepatol.* 2017;32(9):1537. 10.1111/jgh.13661.
11. Guarner J, Brandt ME. Histopathologic diagnosis of fungal infections in the 21st century. *Clin Microbiol Rev.* 2011;24(2):247–80. 10.1128/CMR.00053-10.
12. Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, et al. Global guideline for the diagnosis and management of mucormycosis: an initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. *Lancet Infect Dis.* 2019;19(12):e405–21. 10.1016/S1473-3099(19)30312-3.

Figures

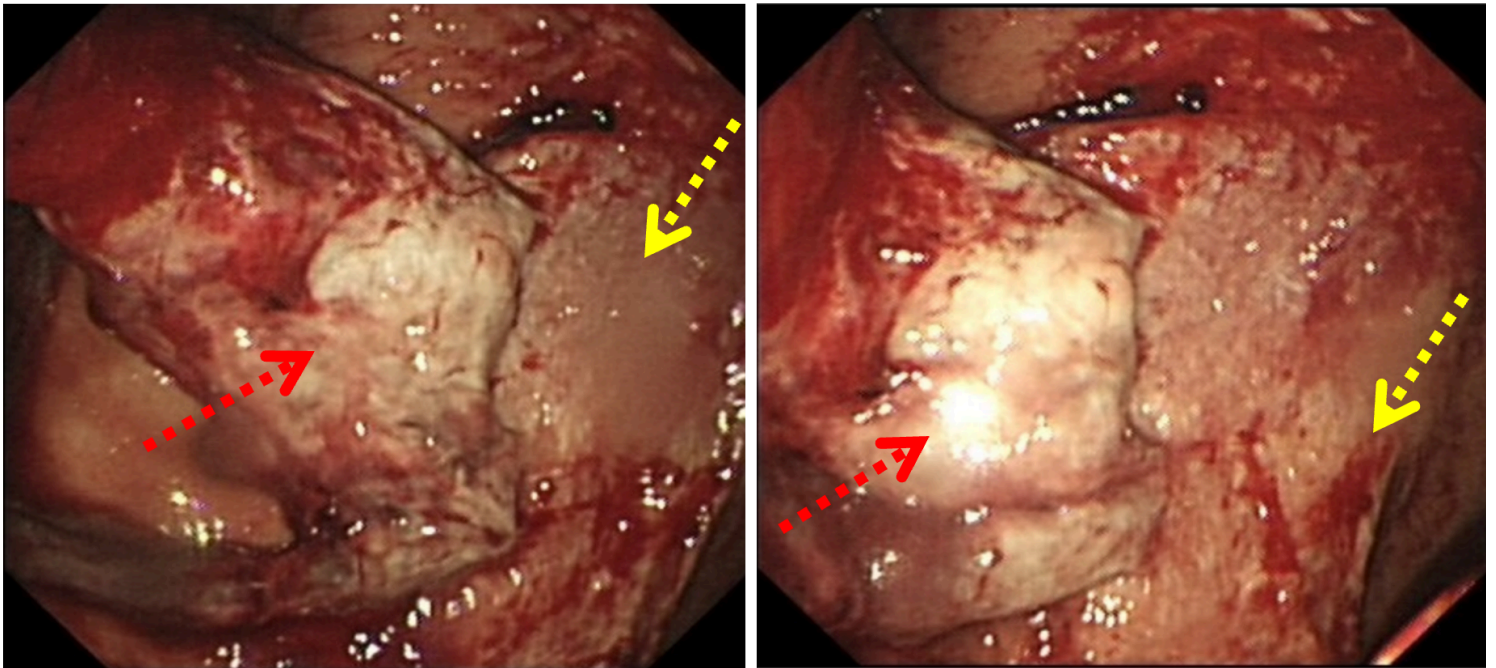


Figure 1

Gastrosocopy identified a 4 cm × 5 cm mucosal detachment (red arrow) extending from the lower greater curvature of the gastric body near the gastric antrum, spanning from the anterior wall to the posterior wall. The ulcer base showed probable muscularis propria exposure, diffuse oozing hemorrhage, and visible thrombi (yellow arrow).

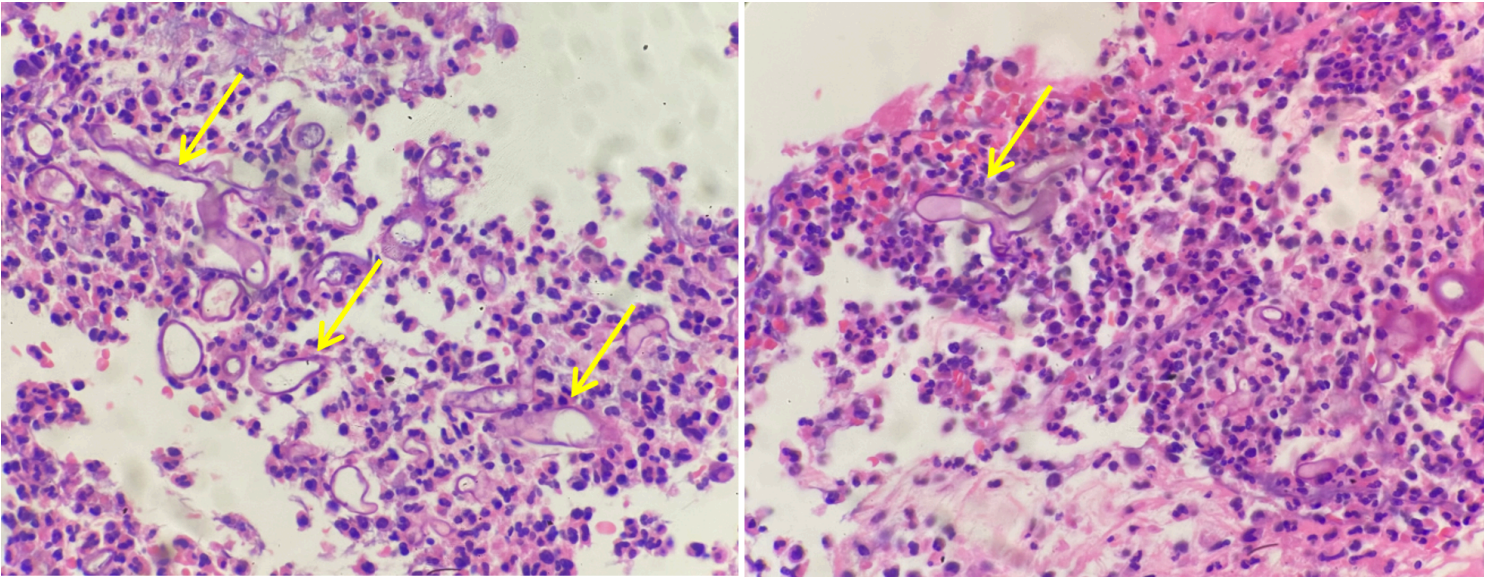


Figure 2

Histopathology from gastric biopsy with hematoxylin and eosin revealed broad, aseptate or pauciseptate fungal hyphae with right-angle branching (yellow arrow), consistent with gastric mucormycosis.