Case Reports and Reviews

Case Report

Gastric Perforation Caused by Candida Albicana : A Rare Clinical Case

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Abstract

Gastric perforation due to Candida albicans is an extremely rare occurrence, typically associated with immunocompromised individuals. We report the case of a 23-year-old previously healthy male who presented with symptoms of acute abdomen. Radiological imaging confirmed visceral perforation, and emergency laparotomy revealed a 3 cm gastric perforation at the antrum. Histopathological analysis of the biopsy confirmed Candida albicans as the causative agent. The patient underwent successful surgical repair with a modified Graham's patch and antifungal therapy. This case highlights the importance of considering fungal etiology even in immunocompetent individuals presenting with gastric perforation.

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Keywords

Candida albicans, gastric perforation, fungal infection, case report, immunocompetent

Introduction

Candidiasis is a fungal infection caused by Candida species, most commonly Candida albicans. While Candida is a normal commensal organism of the gastrointestinal tract, infections are rare due to the acidic gastric environment and competitive gut flora. Gastric candidiasis is typically seen in immunocompromised individuals and is classified into three forms: thrush, nodular, and ulcerative. Gastric perforation secondary to candidiasis is exceedingly rare and has seldom been reported in healthy individuals [1-3].

The common causes of gastric perforation include peptic ulcer disease (PUD), nonsteroidal anti-inflammatory drug (NSAID) use, gastric neoplasms, and trauma. Fungal causes are rare and generally associated with predisposing conditions such as immunosuppression, prolonged antibiotic use, and chronic acid suppression therapy [4,5].

Case Presentation

A 23-year-old male soldier presented to the emergency department with a two-day history of severe generalized abdominal pain, vomiting, and fever. Initially, the pain was localized to the epigastric region and was burning in nature, non-radiating, and unrelieved by food. It progressed to severe, diffuse pain accompanied by persistent vomiting of greenish content, high-grade fever, and abdominal distension. He reported no bowel movement since the onset of symptoms.

The patient had no prior history of diabetes, hypertension, surgery, or blood transfusions. His medical history included gastroesophageal reflux disease (GERD), for which he had been taking proton pump inhibitors (PPIs) and antibiotics for two months. There was no history of alcohol, smoking, or recreational drug use. He had recently returned from Egypt, where he had been consuming canned food. Symptoms developed shortly after resuming military training.

On examination, he appeared acutely ill and dehydrated. Vital signs were: temperature 38.9°C, blood pressure 100/60 mmHg, pulse 78 bpm, respiratory rate 28 breaths/min. The abdomen was rigid with rebound tenderness. Chest auscultation revealed normal breath sounds. Initial management included nasogastric tube insertion, intravenous fluids, and routine laboratory investigations. Chest and abdominal radiographs revealed air under the diaphragm and air-fluid levels, indicating hollow viscus perforation.

Emergency laparotomy via a midline incision revealed approximately 2000 mL of straw-colored peritoneal fluid and a 3 cm perforation at the gastric antrum. A biopsy was taken from the perforation edge, and primary closure was performed using an omental (modified Graham's) patch.

Postoperative Management

The patient was managed with:

- Cefazolin 1g BID for 7 days
- Metronidazole 500mg TID for 5 days
- Omeprazole 40mg BID IV for 7 days
- Paracetamol infusion TID
- Adequate daily IV fluids

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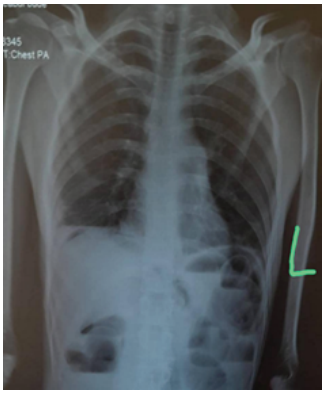

Figure 1. *Chest x ray 1*

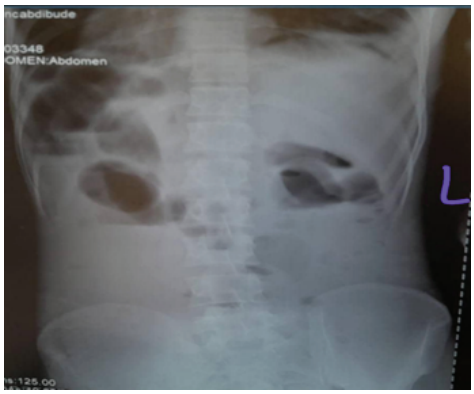

Figure 2. *Abdominal x ray 1*

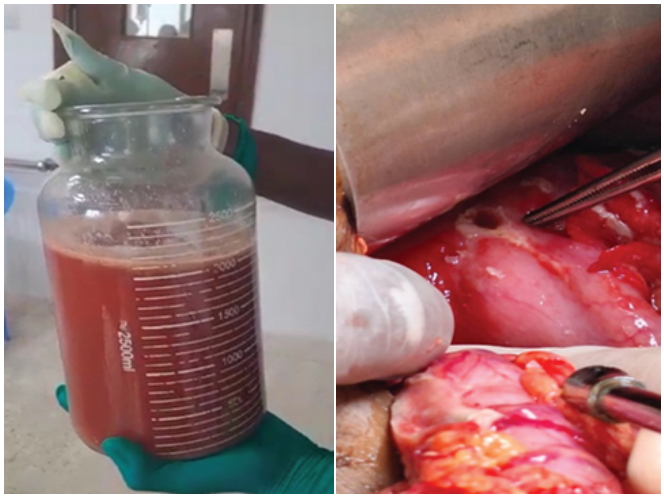

Figure 3. *Image to exemplify the abnormalities of Wünderlich Syndrome*

On postoperative day 2, the nasogastric and Foley catheters were removed. By day 3, the patient was started on oral fluids, and by day 5, he resumed a soft diet. He was discharged on postoperative day 7 in stable condition on PPI therapy alone. On postoperative day 10, histopathology confirmed Candida albicans as the cause of the perforation. Consequently, Fluconazole 200 mg daily for 14 days was initiated.

Discussion

While Candida species are commonly found in the gastrointestinal tract, their progression to invasive disease is typically seen in patients with compromised immune systems. Risk factors include long-term antibiotic use, acid suppression therapy, diabetes, malignancy, or organ transplantation [4,6]. In this case, however, the patient was young, immunocompetent, and otherwise healthy.

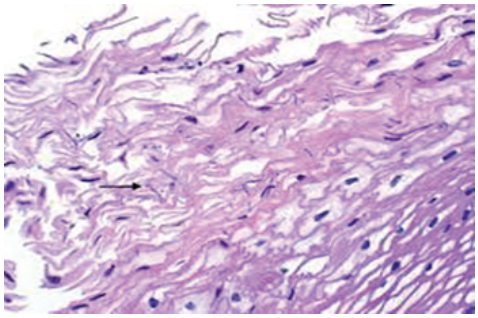

Figure 4. *Histological of Candida albiCana*

The patient's prolonged use of antibiotics and PPIs likely altered the gastric microbiome and pH, facilitating fungal overgrowth. Ingestion of canned food may have further contributed to fungal colonisation. Though Candida-associated gastric ulcers have been reported, progression to perforation is exceedingly rare in immunocompetent individuals [2,3].

Studies indicate that fungal gastrointestinal infections account for 7% of primary GI infections but are associated with an 85% mortality rate. The stomach is the most commonly affected site, followed by the colon and small intestine [4,5]. Early diagnosis and treatment are critical to improving outcomes [1,3].

Conclusion

Gastric perforation due to Candida albicans is rare, especially in healthy individuals. However, prolonged antibiotic and antacid use may predispose even immunocompetent individuals to fungal overgrowth and serious complications. This case underscores the importance of considering fungal pathogens in differential diagnoses of gastric ulcers and perforations, particularly when conventional etiologies are absent.

Ethical Considerations

Informed consent was obtained from the patient for publication of this case report.

Acknowledgments

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