

## Case Report

# A rare case of gastric perforation caused by *Candida* infection

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## ABSTRACT

Fungi are usually a rare cause of gastric perforation (about 0.65%), with most cases of gastric perforation occurring as complications of peptic ulcer disease (PUD), nonsteroidal anti-inflammatory drugs (NSAIDs) and gastric neoplasms. Here, we report the case of a 60-year-old male who presented to our hospital with severe epigastric pain, multiple episodes of vomiting and no history of PUD, NSAIDs use or gastric neoplasm. Exploratory laparotomy revealed a pre-pyloric gastric perforation which was repaired with modified graham's patch repair. Histopathological examination of the gastric perforation edge biopsy revealed an intense *Candida* colonization invading and destroying the gastric wall. Intra-operative fluid sends for culture and sensitivity also revealed growth of candida species. He was subsequently treated with fluconazole antifungal and discharged home after an uneventful postoperative period.

**Keywords:** Candidiasis, Fluconazole, Gastric perforation, Peritonitis, Surgical pathology

## INTRODUCTION

Most cases of gastric perforation occur as complications of PUD, NSAIDs and gastric neoplasms.<sup>1</sup> Though *Candida* species are regarded as normal commensals of the gastrointestinal tract, infections of the gastrointestinal tract very rarely occur and have been reported as very rare causes of gastric perforation, seen mostly in immunocompromised and debilitated patients as well as in healthy persons who indulge in habitual use of strong antacids. Studies shows that nearly 12% of all cases of peritonitis are fungal peritonitis, and *Candida* is the most frequent agent identified.<sup>2</sup> Here, we report the case of a 60-year-old male with a pre-pyloric gastric perforation with candidiasis.

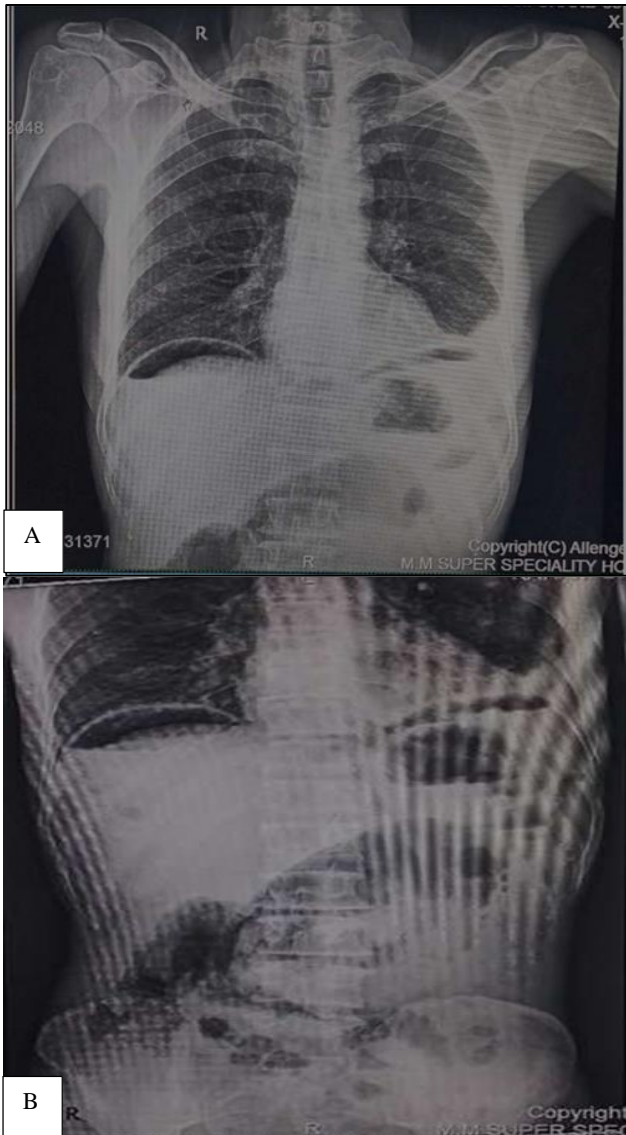
## CASE REPORT

A 60-year-old man, presented to our hospital in the emergency with a history of abdominal pain for the past 5

days. The pain was localized in the epigastric region; was severe in intensity, deep-seated, progressive, non-radiating and was not relieved by intake of food. It was associated with multiple episodes of vomiting. The patient also complained of inability to pass stool and flatus for the past 3 days. His condition worsened 2 days before presenting to us with generalized abdominal pain, abdominal distension and abdominal wall rigidity. There was no associated fever, weight loss, alcohol binge or trauma. No history of previous medication, PUD or abdominal surgery.

The patient was not a known diabetic, hypertensive, epileptic, asthmatic. Patient was a chronic smoker and had a history of cigarette smoking for more than 20 years. On clinical examination, the patient was conscious, tachypnoeic, had tachycardia and had a blood pressure of 110/70 mmHg. The abdomen was distended, tense and with minimal movement on respiration, marked generalized abdominal tenderness and rigidity. On

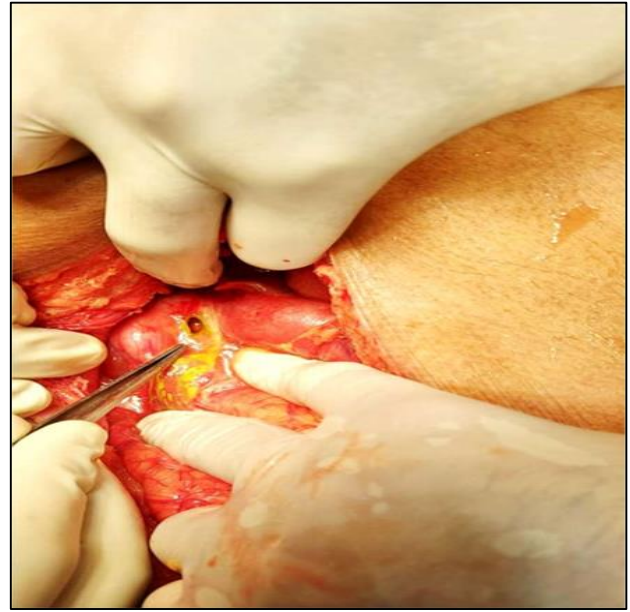
Auscultation there were sluggish bowel sound present. The examination of the liver, spleen, kidneys, and rectum were unremarkable. Upon investigation the preoperative laboratory investigations were hemoglobin-14.3 gm/dl, total leucocyte count-9.66×1000/cumm, urea-210 mg/dl, creatinine-3.9 mg/dl, serum electrolyte concentration - sodium (Na+) 130 mmol/L, potassium (K+) 5.8 mmol/L, chloride (Cl-) 101 mmol/L, and viral markers were negative. X-ray chest showed free air under the right dome of the diaphragm (Figures 1 A and B).



**Figure 1 (A and B): X-ray chest and abdomen showed free air under the right dome of the diaphragm.**

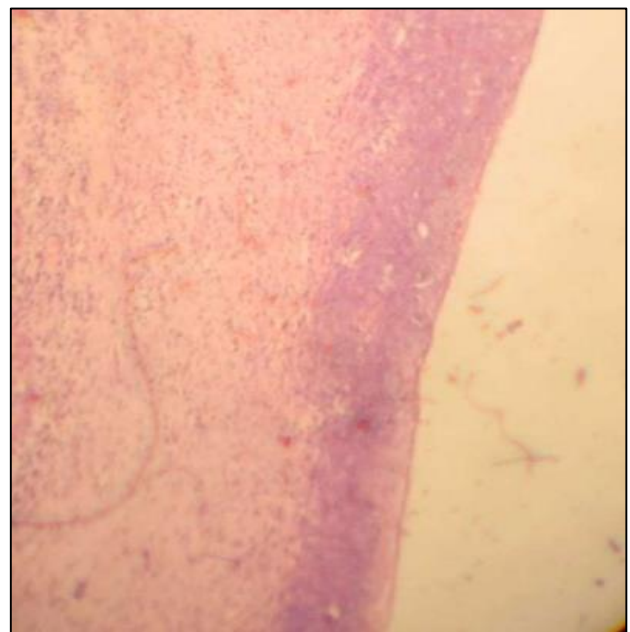
Emergency exploratory laparotomy was performed and a 1 cm by 1 cm, prepyloric perforation covered with fibrinous exudate was seen with vegetative material within the peritoneum (Figure 2). About 100 ml of bilious Fluid was drained and peritoneal lavage was done. An edge biopsy of the ulcerated perforation was taken and modified graham's patch repair was done. The specimen was sent for histopathology.

Fluid sample was sent for culture and sensitivity.

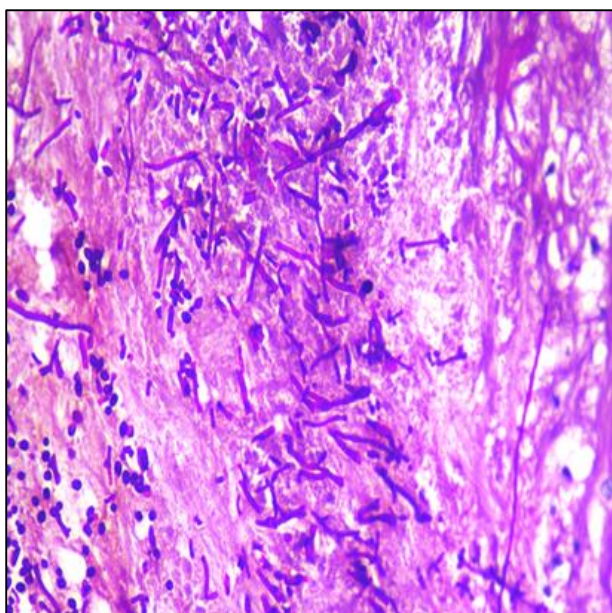


**Figure 2: 1×1 cm, prepyloric perforation covered with fibrinous exudate.**

The microscopic examination of the specimen showed extensive ulceration of the wall with ulcer bed lined by necro-inflammatory admixed with fungal spores and few pseudo-hyphae morphologically resembling *Candida* species (Figures 3 and 4). No evidence of atrophy, metaplasia, *Helicobacter pylori* like organisms or atypical cellular proliferations were seen. A histopathological diagnosis of pre-pyloric perforation from specimen, candidal colonization was made.



**Figure 3: Light microscope from perforation edge biopsy revealed fungal hyphae.**



**Figure 4: PAS stain showing entangled fungal hyphae.**

Fluid culture and sensitivity showed growth of *Candida* species (*Candida Albicans*) sensitivity to antifungal (fluconazole, voriconazole, caspofungin, amphotericin b, flucytosine).

Patient was subsequently treated with injection fluconazole and discharged after 6 days of an uneventful postoperative period. On discharge oral fluconazole was advised, alongside proton pump inhibitor therapy for 1 week. At one week follow-up, the patient was doing fine with no complaints. Further 15 days, 1 month and 3 month follow ups were done.

## DISCUSSION

Gastro-intestinal tract perforation and peritonitis is one of the common surgical emergencies in India. Infections are the most common cause of perforation in India which include *H. pylori* infection, tuberculosis, and typhoid fever in the adult population while foreign body, ischemia, diverticula, Crohn's disease and malignancy are common non-infectious causes of perforation.<sup>3</sup>

Primary fungal infection of the gastro-intestinal tract is uncommon and accounts for only 7% of cases but is associated with 85% mortality.<sup>4</sup> The most common site of fungal gastro-intestinal tract infection is the stomach, followed by the colon and ileum, however, gastric perforation is a rare clinical presentation in such cases.<sup>5</sup>

*Candida albicans* and other *Candida* species are ubiquitous fungi which are the normal commensal flora of the gastrointestinal tract in healthy humans.<sup>6,7</sup> *Candida* species cause infection mostly in immunocompromised and debilitating patients like diabetes, HIV/AIDS, patients on long-term steroid therapy and patients on chemotherapeutic drugs or radiation therapy but rarely in

healthy individuals with high-risk factors like prolonged or habitual use of strong antacids use.<sup>8,9</sup>

The gastrointestinal fungal colonization is normally controlled by the beneficial bacterial flora and low pH of GIT.<sup>10</sup> Improper use of antibacterial agents causing an imbalance in the bacterial flora, prolonged use of antacids resulting in an increase in gastric pH, hyperglycemia facilitating fungal growth, commonly candida infection and invasion leading to multiple ulcers ultimately leading gastric perforation.<sup>11</sup> But diagnosis of this condition is however, difficult due to the prevalence of colonization with nonspecific symptoms and variability of presentation.<sup>12</sup>

A conclusive and confirmatory diagnosis of gastric candidiasis can be made based only on histopathological findings of fungal hyphae with microbiologically confirmed *Candida* species.<sup>13</sup> Histological tissue staining using either the PAS or Gomori's methenamine silver stains to demonstrate Candidal elements like septate hyphae with characteristic dichotomous branching (at an angle of approximately 45°) and yeast cells.<sup>14</sup>

For *Candida albicans*, fluconazole with a dose of 400 mg per day is an appropriate choice, but in fluconazole-resistant *Candida* species and critically ill patients, echinocandin-like caspofungin with a dose of 50 mg per day is recommended. A minimum of two weeks of antifungal agents is recommended.<sup>15</sup>

The current guidelines do not recommend empirical antifungal use for all gastric perforation patients except patients of recurrent perforations, recent abdominal surgery, anastomotic leaks, and critically ill patients requiring ICU care and ventilator patients. Sometimes *Candida* colonization scores like "*Candida* score" may be used to identify who should receive antifungal prophylaxis.<sup>16</sup>

Surgery is mandatory when a patient presents with a visceral perforation following fungal ulcer.<sup>17</sup> Identification of visceral perforation and surgically draining is necessary for the treatment of peritonitis. Postoperatively, Early removing all possible sources like intravascular and urinary catheters, intraabdominal drains and prosthetic materials have to be removed because fungi can form biofilms over it.<sup>18</sup>

Similarly, our patient presented with perforation peritonitis, for which he underwent surgery and had *Candida albicans* in both tissue biopsy and peritoneal fluid. We started fluconazole as per sensitivity and a full 14 days course was completed.

## CONCLUSION

Though rare, surgeons should include candidiasis as a differential diagnosis in a patient with gastrointestinal perforation even in healthy patients. Early detection is



necessary for *Candida* infections as it is associated with significant morbidity and mortality.

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