Case Report

DOI: 10.7860/JCDR/2023/65582.18623

Gastric Candidiasis Leading to Gastric Perforation: A Case Report

Surgery Section

MAREEDU SRI HARI RAO[1], HIMANISH SEN[2], MAREEDU RADHIKA RANI[3], MAREEDU SUHAS[4]

ABSTRACT

Gastric candidiasis is commonly seen in immunocompromised patients with malignant conditions and patients who have undergone gastrectomy. It is also observed in patients who frequently use antacids. The most common presentation of gastric candidiasis is single or multiple ulcerations, with perforation being an infrequent occurrence. The present case report describes a 38-year-old male with a known history of peptic ulcer disease, who presented with abdominal pain and shortness of breath for the past two days. The patient had been using antacids and Proton Pump Inhibitors (PPIs) for two years. Chest and abdomen radiographs revealed an air shadow under the right diaphragm. Emergency surgery was performed, revealing two stomach perforations. The patient underwent primary repair and a Modified Graham's patch repair. Histological examination of the perforation margin showed invasive candidal colonisation. The patient experienced a series of postoperative complications during their hospital stay, all of which were successfully managed. The patient was discharged on the 40th postoperative day. Therefore, it was concluded in this case that the chronic use of antacids and PPIs may have created an alkaline environment, facilitating candidal colonisation of the stomach, ultimately leading to ulceration and perforation.

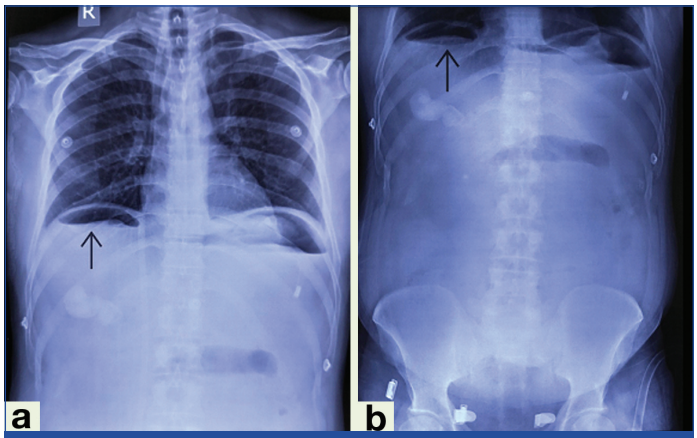
Keywords: Alkanised environment, Gastrointestinal candidiasis, Peptic ulcer perforation

CASE REPORT

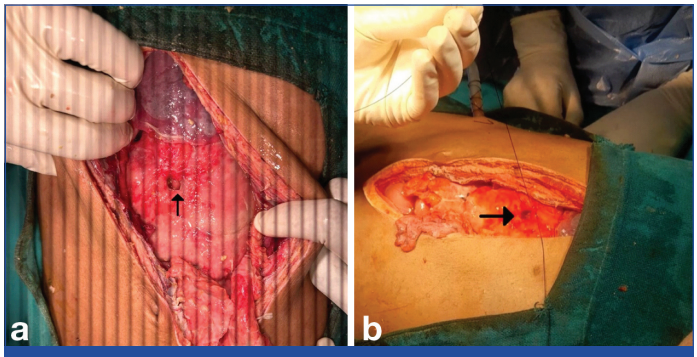
A 38-year-old man presented to the emergency room with a complaint of abdominal pain for the last two days. The pain started around the umbilicus and then spread to the entire abdomen. It was sudden in onset and progressed in severity from mild to severe. He also experienced shortness of breath. The pain worsened with movement and respiration and was accompanied by abdominal distension and rigidity. He had constipation and obstipation for the last two days but no history of vomiting. The patient had a previous diagnosis of peptic ulcer disease and had been using PPIs and antacids chronically. He had experienced two previous episodes of abdominal pain, one three months ago and another two years ago, both of which subsided with treatment. He had no other co-morbidities but was a chronic smoker and alcoholic. There was no history of fever, jaundice, haematemesis, melena, or any previous surgeries.

On examination upon admission, his pulse rate was 128/min (feeble), blood pressure was 80/50 mmHg, and oxygen saturation was 90% in room air. Pallor was present. Diffuse tenderness with guarding and rigidity was noted upon abdominal examination, and bowel sounds were absent. Chest X-ray and erect abdominal X-ray revealed gas under the right dome of the diaphragm [Table/Fig-1a, b]. Abdominal ultrasound showed evidence of moderate free fluid in the abdomen and pelvis. Laboratory results showed a haemoglobin level of 9.5 g/dL, a White Blood Cell (WBC) count of 6.2×103/mm^3 with relative neutrophilia (80.6%), an elevated lactate level (3.6 mmol/L), a prolonged prothrombin time (21 seconds) with an elevated International Normalised Ratio (INR) (1.9), elevated blood urea (51.36 mg/dL) and serum creatinine (1.41 mg/dL), and an elevated total bilirubin (1.88 mg/dL). Viral screening for Human Immunodeficiency Virus (HIV), Hepatitis B, and Hepatitis C were negative.

The patient was resuscitated with Intravenous (i.v.) fluids, and nasogastric tube decompression was performed. An indwelling urinary catheter was inserted, and the patient was planned for an emergency exploratory laparotomy. A midline laparotomy was carried out, revealing 500 mL of purulent fluid in the peritoneal cavity, along with pus flakes and food particles throughout the abdomen, which were suctioned out. The greater omentum was found to be partially adhered to the greater curvature of the stomach. Two stomach perforations were identified. One perforation, measuring 1×1 cm, was located on the anterior wall of the stomach body with indurated margins [Table/Fig-2a]. Another perforation, measuring 4×2 cm, was found in the prepyloric region, and both were closed [Table/Fig-2b]. An edge biopsy of the perforated ulcer was taken before repair and sent for histopathological examination. Peritoneal fluid was also sent for culture and sensitivity. A Weitzel's



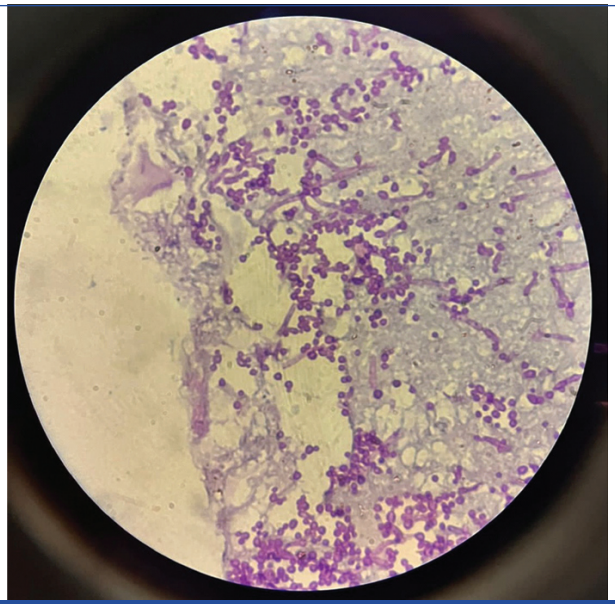
[Table/Fig-1]: a) Chest X-ray posteroanterior view; b) X-ray erect abdomen. Arrows showing gas under the right dome of the diaphragm.



[Table/Fig-2]: Intraoperative images: a) Arrow showing the perforation over the anterior aspect of the body of stomach; b) Primary repair of the perforation.

feeding jejunostomy was placed. The patient had a successful postoperative recovery with improved blood pressure, pulse rate, and O_2 saturation levels, and was transferred to the surgical Intensive Care Unit (ICU) on postoperative day 1.

The histopathology report {Haemotoxylin and Eosin (H&E)} revealed fibromuscular and necrotic tissue in the perforated wall segment, showing spores and septate hyphal structures, indicating "Perforation site candidiasis" [Table/Fig-3]. The culture report showed no bacterial growth.



[Table/Fig-3]: Histopathological slide showing invasive candidal septal hyphae; Haematoxylin and Eosin stain; Magnification 100x (Oil emersion).

During the patient's stay in the ICU, they experienced multiple postoperative complications, including acute kidney injury, leakage from the site of primary repair, pulmonary oedema, pleural effusion, pneumonia, surgical site infection, wound gaping, erythematous rashes caused by levofloxacin, and electrolyte imbalances. However, all of these complications were successfully managed. The patient was treated with Fluconazole 200 mg tablets orally twice daily for 14 days to address the fungal infection. They were discharged on postoperative day 40 and followed-up on a monthly basis for three months post-discharge, during which they had no new complaints or complications.

DISCUSSION

Candida sp is a commensal of the human Gastrointestinal Tract (GIT), and its presence is generally benign. However, recent studies have shown that high-level Candida colonisation is associated with several GIT diseases [1]. Species such as Candida albicans, C. tropicalis, and C. parapsilosis can be found as natural, asymptomatic microbes in the human GIT. The published estimate of Candida albicans carriage in healthy individuals ranges from 30% to 60% [2].

The yeast form of Candida sp is benign, while the fungal form with septae and hyphae is invasive and causes severe diseases. Candida sp exists in its yeast form in an acidic environment, and in its fungal form in an alkaline medium [3]. Therefore, an increase in pH anywhere in the GIT predisposes it to the risk of Candida colonisation and invasion. Prolonged use of PPIs and antacids may have led to Candida colonisation, resulting in ulcers and eventually perforation. A disturbance in the composition of the gut microbiome is also responsible for fungal overgrowth in the GIT. The bacteria-fungi ratio may be altered due to unjustified and excessive use of antibiotics, which suppress the bacterial population and allow fungal colonisation [4]. Thus, several conditions, such as immunocompromised patients, injudicious use of antibacterial agents, and prolonged use of antacids in immunocompetent individuals, can increase gastric habitation by Candida sp.

Candidial infections in the GIT are rare, and the true incidence of GI candidiasis is not known. Documented cases usually involve some abnormality. The oesophagus is the most common site, followed by the stomach and small intestines. Gastric candidial lesions have mostly been found in patients with peptic ulcer disease, malignant neoplasms, or post gastric resection patients [5]. Gastric candidiasis typically presents in two forms: diffuse mucosal (rare) and focal invasion of benign gastric ulcers [5]. The most commonly observed lesions in gastric candidiasis are single or multiple ulcers containing Candida, infiltrating deep into the ulcer beds. Less common presentations include chronic gastric ulcer, gastric perforation, and malignant gastric ulcer with concomitant Candida infection [3]. The most common presentation in gastric candidiasis is single or multiple ulcerations, while gastric perforation is an infrequent presentation.

Lesions with gastric localisation have been predominantly found in patients with peptic ulcer disease, malignant neoplasms, or those who have undergone gastric resection surgeries. Candidial gastric ulcer is more common than previously considered, occurring not only in patients with predisposing conditions but also in apparently healthy individuals [6-10].

Gastric candidiasis is generally managed medically with Tab Fluconazole 400 mg for 14 days. In cases of critically ill patients with Fluconazole-resistant species, Caspofungin 50 mg is recommended. Initially, Amphotericin-B is not used for the treatment of this condition [11]. In cases presenting with a visceral perforation of an ulcer, surgical intervention is mandatory.

However, the present case had a history of antacid and PPI use, which is suspected to be the predisposing factor, leading to candidial colonisation in an immunocompetent individual. Gastric candidiasis can cause gastric ulcers (with Candida in ulcer bed), which may progress to perforation and present as a surgical emergency.

A similar case reported by Kavyashre M et al., had a patient with comparable predisposing factors, clinical presentation, and a similar management plan as in the present case [12]. The renal function was severely deranged in the former, while it was less severely affected in the latter. However, both had a common site of perforation at the prepyloric area, with the present case having an additional perforation at the anterior aspect of the stomach body. Moreover, compared to the uneventful hospital stay of the former patient, the presently reported patient had multiple postoperative complications, all of which were successfully managed, resulting in a longer duration of hospital stay.

Since there is a lack of adequately documented cases of gastric candidial perforation, and only a few cases have been reported so far, it is expected that further studies will be conducted in the future to explore this complication and measures to prevent it.

CONCLUSION(S)

The present case report presents a rare case of candidal infection leading to perforation. The fact that the patient was a known case of peptic ulcer disease and was on chronic use of PPIs and antacids indicates that a decrease in the acidic pH of the stomach might have led to this complication. Therefore, it can be concluded that excessive and unjustified use of antacids and PPIs may lead to candidal colonisation of an alkaline stomach mucosa, resulting in candidal ulcers and their perforations. Considering the adverse effects of candidal colonisation, including rare but potentially fatal complications like perforation, a prolonged use of PPIs and antacids should be discouraged for the treatment of peptic ulcer disease. Instead, other preventive measures should be considered, such as periodic follow-up of the patient with Upper Gastrointestinal (UGI) endoscopy to detect and treat the disease early.

REFERENCES

[1] Kumamoto CA. Inflammation and gastrointestinal Candida colonization. Curr Opin Microbiol. 2011;14(4):386-91. Doi: 10.1016/j.mib.2011.07.015. Epub 2011 Jul 28. PMID: 21802979; PMCID: PMC3163673.

[2] Heather E, Hallen-Adams, Suhr MJ. Fungi in the healthy human gastrointestinal tract. Virulence. 2017;8(3):352-58. Doi: 10.1080/21505594.2016.1247140.
[3] Chen H, Zhou X, Ren B, Cheng L. The regulation of hyphae growth in Candida albicans.Virulence. 2020;11(1):337-48. Doi: 10.1080/21505594.2020.1748930. PMID: 32274962; PMCID: PMC7161696.
[4] Ukekwe FI, Nwajiobi C, Agbo MO, Ebede SO, Eni AO. Candidiasis, a rare cause of gastric perforation: A case report and review of literature. Ann Med Health Sci Res. 2015;5(4):314-16. Doi: 10.4103/2141-9248.160187. PMID: 26229723; PMCID: PMC4512127.
[5] Edwards JE Jr., in Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (Eighth Edition), 2015.
[6] Katzenstein AL, Maksem J. Candidal infection of gastric ulcers. Histology, incidence, and clinical significance. Am J ClinPathol. 1979;71(2):137-41. Doi: 10.1093/ajcp/71.2.137. PMID: 425930.
[7] Neeman A, Avidor I, Kadish U. Candidal infection of benign gastric ulcers in aged patients. Am J Gastroenterol. 1981;75(3):211-13. PMID: 7234843.
[8] Vilotte J, Toutoungi M, Coquillard A. Ulcères gastriques colonisés par Candida. Caractéristiques cliniques et biologiques [Candida infection of gastric ulcers. 6 cases (author's transl)]. Nouv Presse Med. 1981;10(18):1471-74. French. PMID: 7255120.
[9] Scott BB, Jenkins D. Gastro-oesophageal candidiasis. Gut. 1982;23(2):137-39. Doi: 10.1136/gut.23.2.137. PMID: 7068036; PMCID: PMC1419554.
[10] Minoli G, Terruzzi V, Ferrara A, Casiraghi A, Rocca F, Rainer H, et al. A prospective study of relationships between benign gastric ulcer, Candida, and medical treatment. Am J Gastroenterol. 1984;79(2):95-97. PMID: 6364799.
[11] Solomkin JS, Mazuski JE, Bradley JS, Rodvold KA, Goldstein EJ, Baron EJ, et al. Diagnosis and management of complicated intra-abdominal infection in adults and children: Guidelines by the surgical infection society and the infectious diseases Society of America. Clin Infect Dis. 2010;50(2):133-64. https://doi.org/10.1086/649554.
[12] Kavyashree M, Pal B, Dutta S, Badhe BA, Ramakrishnaiah VPN. Gastric candidiasis leading to perforation: An unusual presentation. Cureus. 2021;13(9):e17878. Doi: 10.7759/cureus.17878. PMID: 34660077; PMCID: PMC8502732.

PARTICULARS OF CONTRIBUTORS:
1. Professor, Department of General Surgery, Osmania Medical College, Hyderabad, Telangana, India.
2. Junior Resident, Department of General Surgery, Osmania Medical College, Hyderabad, Telangana, India.
3. Associate Professor, Department of Radiotherapy, MNJ Cancer Hospital, Hyderabad, Telangana, India.
4. Medical Student, All American Institute of Medical Sciences, Black River, St. Elizabeth, Jamaica.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Himanish Sen,
c/o Dr. M Sri Hari Rao, Flat C-123, Krishna Apartments, 8-3-324, Yellareddy Guda, Ameerpet, Hyderabad-500073, Telangana, India.
E-mail: senhimanish@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: May 31, 2023
- Manual Googling: Aug 05, 2023
- iThenticate Software: Aug 21, 2023 (09%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? No
- For any images presented appropriate consent has been obtained from the subjects. No

Date of Submission: **May 30, 2023**
Date of Peer Review: **Jul 26, 2023**
Date of Acceptance: **Aug 23, 2023**
Date of Publishing: **Oct 01, 2023**