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Case Report

Duodenal candidiasis: a rare cause of giant duodenal perforation in immunocompetent patient

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ABSTRACT

Candida is a commensal found even in the gut of healthy individuals and varying rate of incidence have been reported in autopsy studies. Pathological role of Candida is rarely seen in stomach and first part of duodenum as low pH and commensal bacteria inhibits its growth. Any imbalance in these factors or immunocompromised status can lead to fungal overgrowth. Most of the cases of duodenal perforation are seen as the complications of *H. pylori* infection, due to the intake of non-steroidal anti-inflammatory drugs (NSAIDS) or traumatic/iatrogenic. Authors are reporting first case of a 35year old male who presented with duodenal perforation peritonitis with perforation edge biopsy revealing the presence of fungal hyphae. Fungal microorganisms as a cause of duodenal perforation per se, is very rare.

Keywords: Candida, Duodenal perforation, Proton pump inhibitors

INTRODUCTION

Perforation peritonitis is a common disease presenting to surgery emergency out of which a major group is of gastro-duodenal perforation. Most of the cases of duodenal perforation are seen as the complications of *H. pylori* infection, due to the intake of non-steroidal anti-inflammatory drugs (NSAIDS) are traumatic/iatrogenic. Candida is a commensal found even in the gut of healthy individuals as reported in autopsy studies.[1,2] Gastroduodenal candidiasis can be seen as secondary infection of ulcer or primary mucosal disease especially in immunocompromised person. Multiple case reports of gastric perforation due to fungal invasion proven on histopathology are documented in literature but no case of Fungal microorganisms, documented on histopathology, as a cause of duodenal perforation has been reported in literature.[3,4] Authors are therefore reporting a case of a 35year old immunocompetent male who presented with duodenal perforation peritonitis with perforation edge biopsy revealing the presence of fungal hyphae.

CASE REPORT

A 35years old male presented to surgery emergency with complaint of pain epigastric region for 3 days which became generalised to whole abdomen for 2 days. It was associated with non-passage of flatus/faeces. Patient had no previous history of peptic ulcer disease/dyspeptic symptoms or use of Proton Pump Inhibitors (PPI) or NSAIDs. There was no history of diabetes or any immune deficiency. On examination patient had tachycardia with a stable blood pressure. Abdomen was distended with generalized guarding, tenderness and rebound tenderness. Haemogram showed total leucocyte count of 16000 with no other changes. Renal function test, *S. amylase* and *S. lipase* were normal. X-ray Chest

PA Erect with both domes of diaphragm showed free gas suggestive of intestinal perforation. After adequate resuscitation, patient was taken up for exploratory laparotomy. A giant perforated duodenal ulcer (3x3cm) with necrotic margins was noted in first part of duodenum. Margins of perforation was freshened and sent for biopsy. Primary repair of perforation with retrograde duodenostomy and feeding jejunostomy with gastric decompression via Ryle's tube was done. Post operatively patient did well. There was no fever, TLC normalized gradually, and patient moved his bowels. On day 7 patient was started orally and day 10 duodenostomy and feeding jejunostomy were removed. Histopathology report was suggestive of ulcer bed formed by mixed inflammatory infiltrate (Figure 1), with entrapped pseudo-hyphae of yeast and candida (Figure 2). In view of histopathological finding of candidiasis, patient was given a course of fluconazole 150mg once daily for 14 days. 3 months post operatively, upper GI endoscopy was done which did not show any ulceration.

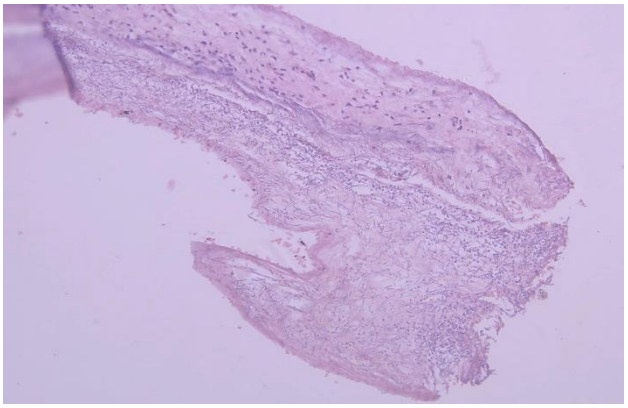

Figure 1: Ulcer bed formed of inflammatory granulation tissue with surface necrosis (Hematoxylin and Eosin x200).

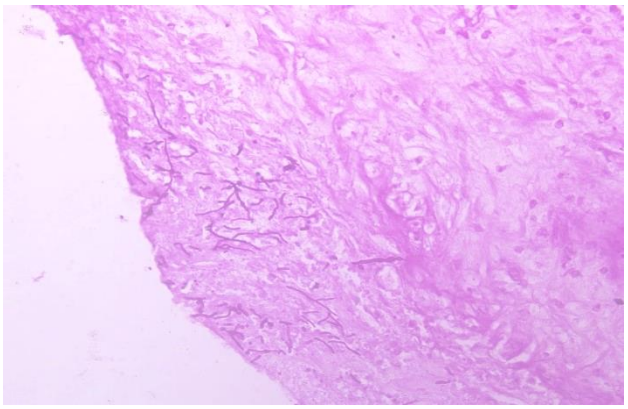

Figure 2: Yeast and pseudohyphal forms of candida highlighted by periodic Schiff stain x 200.

DISCUSSION

Candida is a commensal found even in the gut of healthy individuals and varying rate of incidence have been reported in autopsy studies. In a study by Ears et al., gut mycosis was observed in 109 (4.35%) of the 2517 cases.[1] Tsukamoto et al, from Japan reported that gut mycosis was present in 196 (5.9%) of the 3,339 cases. In these reports, the most commonly affected organ was oesophagus, followed by stomach, small intestine and large intestine.[2]

Pathological role of Candida is rarely seen in stomach and first part of duodenum as low pH and commensal bacteria inhibits its growth. Any imbalance in these factors or immunocompromised status can lead to fungal overgrowth. Therefore, candidial growth has been seen in conditions such as administration of steroids, excessive antibiotics, immunosuppressive drugs or anticancer drugs, HIV status, malignancy, uncontrolled diabetes, old age or systemic illness. The role of acid suppression in Gastric/Duodenal Candidiasis is also controversial. Some studies suggested a predisposition to Candida overgrowth with H2-receptor antagonist therapy.[5] However, a recent prospective study, did not report higher Candida culture rates in patients receiving H2-blockers or proton pump inhibitor treatment compared to no treatment with acid suppressants.[6]

Candidiasis of gastroduodenal region can present as secondary infection of peptic ulcers, or invasive disease of mucosa or systemic dissemination due to vascular invasion. The clinical diagnosis of candidiasis is very difficult due to varied presentation which can range from features of dyspepsia with ulcer like pain, weight loss, anorexia as seen in mucosal disease to fever with sepsis like presentation as seen in vascular invasion.

On endoscopy the candidiasis lesion can be of three types[7]

- Thrush type presents as a white or green-white membrane which can be easily removed to expose underlying inflamed mucosa.
- Nodular type presents as nodular projections with a diameter of a few millimetres, overlaid by highly inflamed mucosa, and mainly located in the antrum.
- Ulcerated type had no particular endoscopic features that can differentiate them from other peptic ulcer forms.

Therefore, to differentiate secondary growth of candidiasis in ulcer from invasive diseases, culture of brushings from ulcer surface is not a definite proof of candidiasis unless proven by histopathological examination. Role of candidiasis in ulcer healing is also debatable. Some studies reported that fungal infected ulcers are larger in size as compared to non-infected ulcers. These studies also reported delayed healing of fungal infected ulcers when treated without anti-fungal therapy and therefore support use of antifungal therapy.[8]

Whereas some studies show no effect of candidial growth on ulcer healing and support use of only PPI.[9-11] Special

mention is needed for study by Jung et al because the criterion for the diagnosis of candidiasis was the finding of yeast and hyphae in the tissue or ulcer on histological sections of biopsies. Surface fungi were not considered infections and demonstration of Candida in smears and cultures was not considered reliable evidence for candidiasis and therefore excluded secondary fungal growth cases from study.

On extensive research of literature author can find multiple case reports of gastric perforation due to fungal invasion documented on histopathological examination in immunocompromised individuals and few cases in immunocompetent individuals.[3,4] Author also found case reports/case series of fungal peritonitis post intestinal perforation where majority of cases of fungal peritonitis belongs to gastro duodenal perforation (70%).[12] But author could not find any case reported where fungal invasion as cause of duodenal perforation was documented as in our case and also patient had no predisposing factors for candidiasis.

CONCLUSION

Most of the cases of duodenal perforation are result of complicated *H. pylori* infection or the intake of NSAIDs but in immunocompromised patient fungal infection should be kept as one of possible cause of perforation. Role of PPI as causative factor and role of anti-fungal treatment is not clear with contradicting evidence.

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