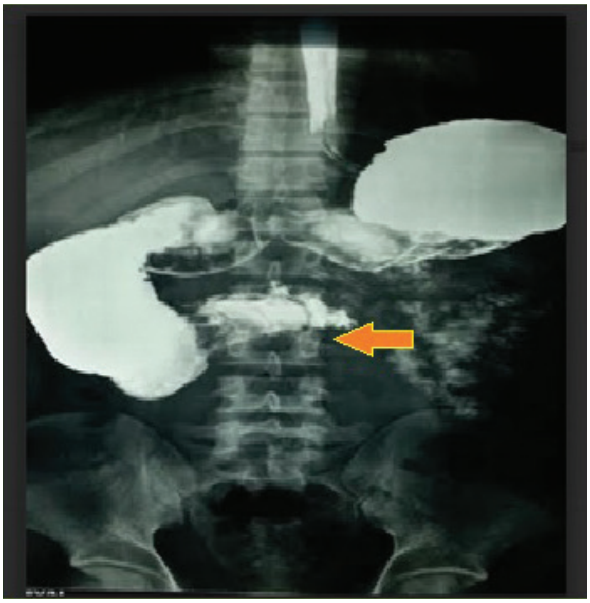

Figure 4: Barium meal series showing a halo sign suggestive of IDD.

duodenum leads to formation of a sac like structure which is the IDD. The median age of presentation is 3rd to 4th decade. Most common presentation is pain abdomen followed by partial obstruction ,pancreatitis and upper UGI bleed.[4] The usual site of IDD is proximal to ampulla in 52%, at the ampullary level in 30% and distal to ampulla in 18%. The close differentials are choledochocele and duplication cysts which can be differentiated by imaging studies where the choledochocele will be continuous with bile duct and duplication cyst will not take up barium/ contrast as it is not connected with duodenal lumen. Endoscopic management by snare technique and needle knife resection has been proposed as this is not a true diverticulum and has duodenal mucosa covered on both sides, but always a careful delineation of ampullary, bile duct and pancreatic duct anatomy is very important before endoscopic management as this can result in permanent bile duct injuries.[5] Our case presented with partial duodenal obstruction and hence we offered a surgical resection of the diverticulum after which he became asymptomatic. As it resembled a kangaroo's pouch we named it kangarooed duodenum.

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Saccharomyces cerevisiae fungemia in inflammatory bowel disease

Saccharomyces cerevisiae is commonly used in the food industry as probiotic for prevention and treatment of diarrheal diseases. It is present as commensal in human

gut and has been found in the respiratory tract in 7% of patients of COPD, occasionally in the vaginal flora, throat, stool and urine. Whether it colonizes the digestive tract persistently or transiently is still unknown.[1] Iinitially it was considered non pathogenic, there have been reports of invasive infections, seen more frequently in immunocompromised patients.[1,2] Predisposing factors for acquisition are intravenous catheter use and previous antibiotic therapy.[1]

Case Report

Here we present a case of *Saccharomyces cerevisiae* fungemia in a young male. To our knowledge this is the first report from India. Patient presented with bloody diarrhea and vomiting for 5 days, fever for 3 days and altered sensorium for 1 day. He was a known case of Inflammatory bowel disease (IBD). Pulse rate was 137/ minute, blood pressure 100 /60 mm Hg, oxygen saturation 98%. Abdomen was distended with absent bowel sounds. Pupillary response was normal. Glasgow Coma Scale was 15/15. Blood investigations revealed haemoglobin 7.0 gm/dl, total leucocyte count 24,000 cells/µl with 92% neutrophils, blood urea 119.0 mg/dl, creatinine-2.4 mg/dl, sodium-123 mmol/l, potassium-2.4 mmol/l. Procalcitonin was 17.2 nmg/ml. An immediate decision of exploratory laporotomy was taken. On exploration, the colon was found dilated with internal haemorrhage due to multiple perforations. The operative diagnosis was IBD necrotizing enterocolitis. A total proctocolectomy with jejunal re-anastomosis was performed and a drain was put in situ. Antibiotics started were Metronidazole 500mg and Meropenem 1 gm both thrice daily i.v. Blood cultures were sterile initially. Histopathological study reported ulcerated mucosa with multiple foci of haemorrhage, mucosal ischemia and crypt abscesses. On Day 20, massive haemorrhagic discharge was noticed in the drain. Internal haemorrhage was suspected and reexploration laporotomy was done whereby a perforation closure was performed. A stoma was created in the lower abdomen. The patient continued to be critical. On day 22 he developed high grade fever. Twin sets of blood culture grew yeast cells. Identification of the isolate as *Saccharomyces cerevisiae* was done on VITEK 2 (Biomerieux, USA). Molecular

Figure 1: Saccharomyces cerevisiae on Sabourad dextrose agar.

identification was confirmed at Vallabhbhai Patel Chest Institute, Delhi by sequencing of ITS and D1/D2 region of ribosomal DNA. DNA extraction and amplification were performed as described.[3] Briefly, the extracted DNA was subjected to amplification with the established primers ITS1 (5=-TCCGTAGGTGAACCTGCGG-3=) and ITS4 (5=-TCCTCCGCTTATTGATATGC-3=) for ITS region amplification and NL1 (5=-GCATATCAATAAGCGGAGGAAAAG-3=) and NL4 (5=-GGTCCGTGTTTCAAGACGG-3=) for LSU region amplification. The amplicons of both regions were purified (Wizard SV Gel and PCR Clean-up System; Promega) and sequenced. The sequencing reactions were carried out by using a cycle sequencing kit (Big-Dye Terminator v3.1 cycle sequencing kit RR100; Applied Biosystems, Foster City, CA) with ITS1 and ITS4 as sequencing primers for ITS region amplicons and NL1 and NL4 as sequencing primers for D1/D2 region amplicons. The strands were sequenced with an ABI 3130XL genetic analyzer (Applied Biosystems, Foster City, CA). Both ITS and D1/D2 domain consensus sequences were then subjected to Basic Local Alignment Search Tool (BLAST) searches at GenBank and revealed 99% homology with *S. cerevisiae* GenBank accession no. HG532105. E-strips

(Biomerieux, USA) recorded the isolate susceptible to Caspofungin, Voriconazole, Flucytosine and resistant to Amphotericin (MIC > 32 microgram/ml). He was started on Caspofungin 70 mg load and 50 mg daily for 21 days.

Discussion

Saccharomyces cerevisiae is ubiquitous and used as probiotic. Fungemia due to S. cerevisae has been documented in immunocompromised, burn patients and where it has been used as probiotic. Munoz P et al (2005) described 60 cases of *S. cerevisiae* fungemia; commonest factor was its use as probiotic. 60% were nosocomial and 70 % had enteral feed.[4] In IBD, patient shows abnormal responses to food antigens which may be secondary to inflammation and damage to integrity of the intestinal wall, causing increased exposure of the immune system to antigenic contents of the bowel lumen including *S. cerevisiae* antigens.[5] Interestingly, Saccharomyces cerevisiae has been used as supportive therapy in Crohn's disease (CD) and have shown decrease in its relapse.[6] *S. cerevisiae* colonizes the duodenal mucosa. IBD is associated with increased permeability and disruption of gut mucosa. Probably our patient suffered fungemia due to break in endogenous gut mucosal barrier. He had risk factors for nosocomial acquisition but Differential time to positivity was not significant nor was he on probiotics. The intestine had multiple abscesses and perforations. Surgeries must have disrupted the local defense mechanisms and yeast must have translocated from the intestinal mucosa into blood stream. Literature reveals that most cases were treated with Amphotericin B.[7] However antifungal of choice is not established. Low susceptibility to azole group has been documented. Caspofungin has not been reported to be used but unpublished data suggest good efficacy.[1] Here, Caspofungin cleared the fungus as demonstrated with repeat blood cultures and was shifted out of the ICU after 45 days. Subsequently he was discharged from the hospital. We conclude that *S. cerevisiae* fungemia though uncommon, is an emerging infectious disease. This case gives an insight into the other collaterals that need attention in managing such cases of IBD.

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