

stent in 9 cases.⁵ No complications apart from distal stent migration was reported.

We placed the guidewire in the antrum of our patient under endoscopic guidance. We did not face any technical difficulty in placing the stent. Histacrylglue was injected into the varices prior to the stent placement, but there was no pneumomediastinum. The stiff guidewire used for stent deployment is the only plausible explanation we have for the gastric perforation.

Since SEMS placement is usually done at the bedside in such cases, without radiological assistance, we recommend that the stiff guidewire enclosed in the package be avoided; less stiff guidewires (0.035") may reduce the likelihood of this complication.

AJINKYA SONAMBEKAR
DEVENDRA DESAI

¹Department of Gastroenterology, P. D. Hinduja Hospital and Medical Research Centre, Mumbai, India

*Corresponding Author: Dr Devendra Desai
Email: drddesai@hindujahospital.com*

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An Opportunistic Infection and a Lower Gastrointestinal Bleed: Rare But Real

Investigating a patient presenting with gastrointestinal bleed in a tropical country is challenging and we occasionally encounter rare causes. Invasive fungal infection due to mucormycosis is one such cause. Massive overt GI bleed as clinical presentation of mucormycosis is very rare. Less than 25% of GI mucormycosis get diagnosed with ante-mortem investigations. The mortality rate is high as these infections are usually seen in immunocompromised patients. This suggests the seriousness of condition and the low awareness. We discuss a case of lower GI bleed secondary to mucormycosis of the caecum.

Case Report

A 41 year old lady was hospitalised with history of lower abdominal pain and loose stools since 1 week and passing large quantity of blood clots mixed with stools since 2 days. No history of hematemesis. She was diagnosed to have rapidly progressive glomerulonephritis due to anti-GBM disease one month prior to this episode. She had received cortico steroids and pulse cyclophosphamide therapy followed by 5 sessions of plasmapheresis. She was on thrice weekly maintenance haemodialysis since then. She was hypotensive and had tachycardia. Pallor was present. Abdominal examination revealed mild hypogastric and periumbilical tenderness. Per rectal examination showed fresh blood. Lab investigations revealed haemoglobin of 4.7 gm/dl and elevated leucocyte count (19,800 cells/cu.mm). After hemodynamic stabilisation and blood transfusion, colonoscopy was done which showed a large 4x5 cms deep cecal ulcer with possible clot/blackish pigmentation over the base (**Figure 1**). Ileum appeared normal. Rest of the colonic mucosa was normal. Histopathological examination of biopsies from the cecal ulcer showed broad aseptate hyphal forms consistent with morphology of mucor (**Figure 2**). She was started on intravenous amphotericin and gradually improved. Her bleeding decreased over the next 2 days and stopped. She was discharged from the hospital but however she was lost for follow-up.

Discussion

Mucormycosis infection is most commonly seen in patients with underlying immunocompromised status like diabetes mellitus, HIV disease, corticosteroid use, underlying hematologic malignancy or post transplant status with immunosuppressant use. Other risk factors are deferoxamine use, military injuries (blast injuries) and natural disasters.¹

Mucormycosis can manifest in humans in 5 different forms: rhinocerebral, pulmonary, cutaneous, gastrointestinal and disseminated forms. Gastrointestinal

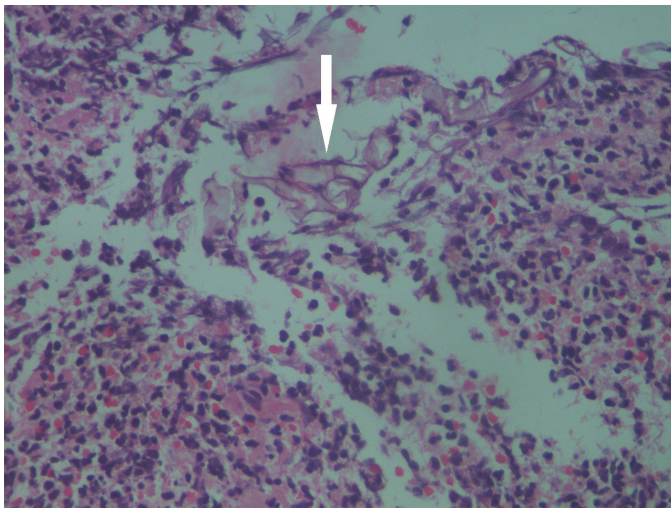


Figure 1: H and E staining biopsy specimen of cecal ulcer showing broad aseptate hyphal forms of mucor (arrow).

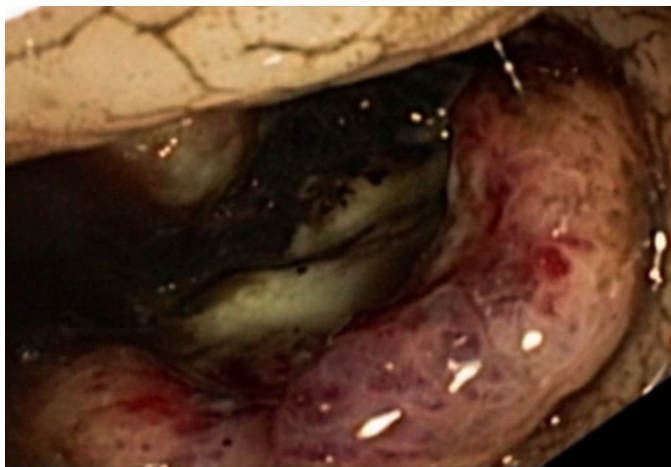


Figure 2: Colonoscopy image showing ulcer with areas of blackish pigmented base in the cecum.

involvement is rare and seen in 7% of cases.² Stomach is the most common organ involved followed by colon and ileum. Clinical presentation of GI mucormycosis is nonspecific with symptoms ranging from abdominal discomfort, diarrhoea, perforation and bleeding. GI involvement is more common in infants. Most common cause of mortality is bowel perforation. Presentation with massive lower GI bleed is rare.³ Mucormycosis invades the GI mucosa and submucosa and classically causes thrombosis of endarteries. This leads to necrosis and ulcers of the GI mucosa. The ulcers have been described as large deep well demarcated with exudative base and rarely eschar like pigmentation. Rarely proliferative lesions mimicking malignancy have been described.⁴

Diagnosis is usually by histopathology and there are no clinical pathognomonic features. Only 25-50% of the cases are diagnosed antemortem.⁵ Presence of aseptate hyphal forms of the fungus in a background of mononuclear infiltrate is seen in the histopathological specimens. Treatment depends on depth of involvement, clinical manifestation and general condition of patients. Extensive debridement of the area and liposomal amphotericin B are often required in severe cases. Survival was 3% for cases that were not treated, 46-61% for cases treated with amphotericin B deoxycholate, 57% for cases treated with surgery alone, and 70%-81% for cases treated with amphotericin B and surgery.⁵

We discuss this case to highlight the need to maintain a high index of suspicion for unusual opportunistic infections as cause of GI bleed in immunocompromised patients.

SATISH K NAYAK¹
 PIRAMANAYAGAM PARAMASIVAM¹
 PALLAV PARIKH¹
 MAYA MENON²
 SUBBA RAO B³

¹Department of Gastroenterology, ²Department of Pathology,
³Department of Nephrology, Apollo Hospitals, 21 Grems Lane, Grems Road, Chennai-6, India

Corresponding Author: Dr Satish K Nayak
 Email: bsatishnayak@gmail.com

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Celiac Disease in Patients with Liver Cirrhosis

Celiac Disease (CD) is a chronic immune-mediated multi-systemic disease that can affect any organ. Extra-intestinal manifestations are seen in up to 30% of patients with CD.¹ Modest elevation of serum aminotransferase levels is common in untreated CD, occurring in 15%-55% of patients.² Cirrhosis in patients with CD is described only in case reports.^{3,4} Cirrhosis is often associated with increased morbidity/mortality, decrease in health related quality of life and substantial health care costs.⁵ Without a liver transplantation survival is very poor; prevention of development of cirrhosis and its progression are considered standard of care. Therefore recognizing CD in the setting of cirrhosis is essential in order to institute a GFD to potentially prevent further morbidity and

mortality. Data is still lacking on correlation of CD with cirrhosis and the response to GFD in cirrhotics. Here we present six cases of CD with cirrhosis of liver where we have studied duration of disease, mode of presentation and effects of GFD on liver disease. Diagnosis of cirrhosis was based on clinical, biochemical, endoscopic and imaging findings. Patients with histological evidence of cirrhosis were also included. The diagnosis of alcoholic cirrhosis was made on the basis of history of any form of daily alcohol consumption of >80g/dl in men and >40g/dl in women for 10yrs. Diagnosis of CD was done as per European Society of Pediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) guidelines (score of 4 or more was considered diagnostic of CD). IgA anti endomysial antibodies (EMA) was done using ELISA kit named D-TEK from Belgium. Normal range is 0-25 units/ml. Value of 25 units/ml or more is considered positive.

Case 1

A 48 years old female with presented 8 years back with complain of painless progressive symmetrical distension of abdomen and painless swelling of both lower limbs since 1 month. She had no co morbidities and no addictions. After all laboratory investigations, upper gastrointestinal endoscopy (small oesophageal varices) and ultrasound of abdomen (USG abdomen) (moderate ascites, nodular liver surface and mild splenomegaly) and liver biopsy (moderate interface activity, chronic hepatitis with cirrhosis); she was diagnosed to have autoimmune hepatitis with decompensated cirrhosis of liver and was treated with tapering doses of steroids and azathioprine. One year later she presented with complains of small bowel type diarrhoea for 2 months. On investigations she had iron deficiency anaemia, USG abdomen showed moderate ascites and UGIscopy showed small oesophageal varices, duodenum was normal but biopsy was taken from second part of duodenum. Histopathology showed >40 intraepithelial lymphocytes/100 enterocytes with normal crypts and normal villi. Colonoscopy was normal. EMA was elevated more than 3 times the upper limit of normal. HLA typing showed HLA DQ2 positive. Patient was started on GFD. On one year of strict GFD patient's symptoms improved, ascites resolved diuretics were stopped and there was no worsening in liver functions.