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Case Report

Bowel infarct with mucormycosis of the intestine

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ABSTRACT

Gastrointestinal mucormycosis is a relatively uncommon opportunistic fungal infection. The classical presentation is that of a rhino-cerebral infection. It is a life threatening, angio-invasive condition typically affecting immunocompromised individuals. Only a handful cases of intestinal mucormycosis have been reported to date. Here, we discuss our experience regarding an adult male patient who succumbed to bowel infarction and perforation with septic shock due to intestinal mucormycosis.

Keywords: Fungal, Gastrointestinal, Mucormycosis, Rhizopus

INTRODUCTION

Fungi of the subphylum *Mucoromycotina*, order *Mucorales* cause mucormycosis-a rare, often fatal, angioinvasive infection, primarily of immune-compromised hosts.[1] Rhino cerebral and pulmonary involvements of this infection are the most common followed by gastrointestinal infections. All parts of the intestine are vulnerable to infection, with stomach, ileum, and colon being most commonly involved.[2] The incidence of gastro-intestinal mucormycosis appears to be on the rise, highlighted by an increase in the number of cases indexed on PubMed-50 publications between 2000 and 2011 compared with eight between 1959 and 1989. Authors present a case of a male patient with bowel infarction and perforation due to intestinal mucormycosis.

CASE REPORT

A 42-year-old gentleman presented with complaints of sudden onset of multiple episodes of non-bilious vomiting and loose stools for two days followed by non-colicky, mild, generalized abdominal pain . He was non-diabetic. He was previously diagnosed with nasopharyngeal carcinoma for which he had received chemoradiation in 2005. At the time of admission, he was hypotensive (blood pressure 70/50 mm of Hg) with tachycardia and was dehydrated. Systemic examination was grossly normal. He was admitted with the provisional diagnosis of acute gastroenteritis with hypovolemic shock. His initial blood panel revealed leucocytosis (23000/cu mm) and raised serum creatinine (2.3 mg/dl) with deranged electrolytes. Other blood works were normal with negative viral markers for HIV, HCV and HBsAg. X-ray abdomen and chest were grossly normal. Ultrasonogram of the abdomen revealed mild ascites. His urine output started to drop associated with further derangement of the kidney function with metabolic acidosis. He underwent haemodialysis multiple times over the next few days, but the renal function did not improve. He developed constipation and obstipation associated with progressive abdominal distension. The non-contrast CT scan revealed extensive pneumoperitoneum with moderate free fluid in perihepatic, peri splenic, bilateral paracolic gutters and pelvis, with dilated duodenum, proximal jejunum and proximal ileum with bowel wall edema and extraluminal air, and fat stranding in surrounding mesentery suggestive of ileal perforation. He was taken up for an emergency

exploratory laparotomy. The intraoperative findings revealed around 3 litres of feculent, bilious and purulent fluid in the peritoneal cavity, necrotic small bowel with thickened and thrombosed mesenteric veins and multiple large perforations in distal jejunum and distal ileum.

Around 6 feet of small bowel was resected and sent for histopathology, and a distal jejunostomy was fashioned. The patient continued to require mechanical ventilation postoperatively and was on inotropic support. He developed metabolic acidosis with oliguria. His inotropic requirement increased gradually, and he developed a cardiac arrest on the 3rd post op day for which CPR was initiated as per protocol but the patient succumbed to the illness. The histopathological examination showed transmural haemorrhagic necrosis of bowel wall with infiltration by branching, broad, aseptate fungal hyphae. Vessels showed thrombosis and fungal invasion suggestive of invasive intestinal mucormycosis.

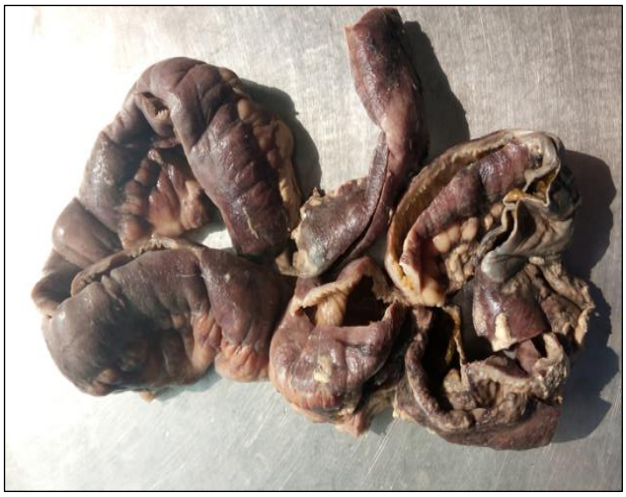

Figure 1: Resected gross specimen with multiple perforations in small bowel.

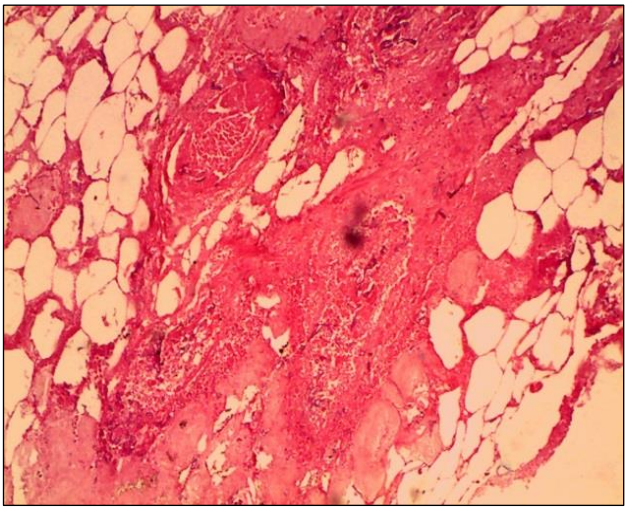

Figure 2: 100X magnification showing fungal colonies infiltrating wall of intestine.

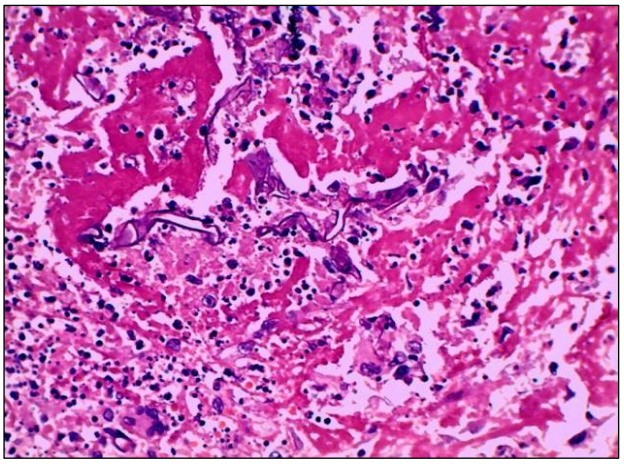

Figure 3: 400X magnification showing fungal colonies infiltrating wall of intestine.

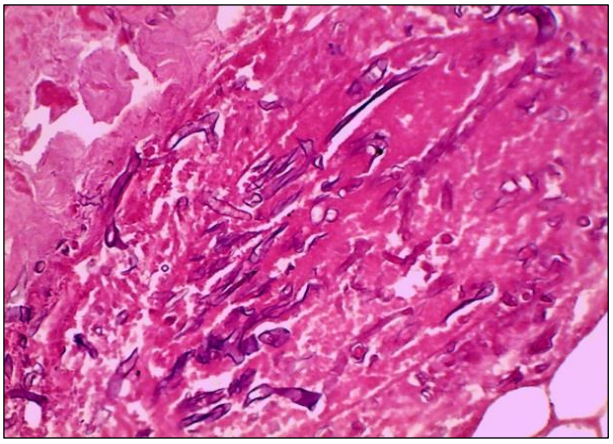

Figure 4: 400X magnification shows broad, aseptate, fungal hyphae and spores invading vessel wall.

DISCUSSION

Mucormycosis is an infection caused by fungal agents in the order *Mucorales*. Infection is commonly due to *Rhizopus* (47%), *Rhizomucor*, *Absidia*, and *Mucor* species, in the family *Mucoraceae*. The invasion of blood vessels by hyphae leads to arterial thrombosis, tissue infarction, and necrosis, whereas venous invasion causes hemorrhages.[3] Paltauf et al reported the first case of mucormycosis in 1885.[4] All parts of the alimentary tract are vulnerable to gastrointestinal mucormycosis infection, with stomach (the most common site), ileum and colon being commonly affected. The route of infection is believed to be secondary to ingestion of the fungi, which then may colonize the gastrointestinal tract.[5]

The diagnosis of gastrointestinal mucormycosis is often delayed because of the non-specific presentation like in our case; abdominal pain, distention and vomiting are the most common presenting symptoms. Infection may present with an abdominal mass (appendiceal, cecal or ileal) mistakenly thought to be an intra-abdominal abscess.[7] The pathological hallmark of mucormycosis is

infarction of host tissue resulting from angioinvasion by hyphae. The fungus invades the bowel wall and blood vessels, leading to bowel ischemia, necrosis, perforation, peritonitis or massive hemorrhage.[6] Diagnosis of this condition can be elusive and is most often revealed by histopathology only.[8] The successful management of mucormycosis requires early diagnosis, reversal of predisposing risk factors, Surgical debridement, and prompt antifungal therapy. There are no recommendations specific to gastrointestinal infection.[8] Thrombosis and necrosis result in poor tissue penetration and delivery of antifungal therapy to the infected site. Debridement of necrotic tissue appears to be critical for complete eradication.[9] Given the mortality attributable to gastrointestinal mucormycosis, aggressive surgical intervention is paramount. In 2014, Cornely et al, and the European Society of Clinical Microbiology and Infectious Diseases, and the European Confederation of Medical Mycology strongly recommended that liposomal or lipid complex Amphotericin-B be initiated as first-line therapy in addition to surgical debridement and the treatment of any underlying disease processes. Posaconazole is strongly recommended as a treatment in refractory cases.[10] These recommendations do not specifically address invasive mucormycosis of the gastro-intestinal tract, which was reported in our case. There are many open studies and areas that have yet to be addressed in these papers that provide an avenue for future recommendations, guidelines, and specific protocols in such cases.

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REFERENCES

1. Kwon-Chung K. Taxonomy of fungi causing mucormycosis and entomophthoramycosis (zygomycosis) and nomenclature of the disease: mole- cular and mycologic perspectives. Clin Infect Dis. 2012;54(1):8-15.
2. Azhar A, Calubiran OV, Kilaru RM, Lincoln JA, Moshenyat I, Basti K, et al. Patient with Abdominal Pain-Concealing Gastrointestinal Mucormycosis. Gastroenterol Hepatol (NY). 2009;5(9):657-61.
3. Kontoyiannis DP, Lewis RE. Invasive zygomycosis: update on pathogenesis, clinical manifestations, and management. Infect Dis Clin North Am. 2006;20(3):581-607.
4. Paltauf A. Mycosis mucorina. Virchows Archiv. 1885;102:543-64.
5. Prasad BNS, Shenoy A, Nataraj KS. Primary Gastrointestinal mucormycosis in an immunocompetent person. J Postgrad Medicine. 2008;54(3);211-3.
6. Roussy JF, Allard C, Germain GS, Pepin J. Gastrointestinal mucormycosis following a streptococcus pyogenes toxic shock syndrome in a previously healthy patient: A case report. Case Rep Infect Dis. 2012;2012:476719.
7. Petrikkos G, Skiada A, Lortholary O, Roilides E, Walsh T, Kontoyiannias D. Epidemiology and clinical manifestations of mucormycosis. Clin Infect Dis. 2012;54(1):23-34.
8. Spellberg B, Walsh J, Kontoyiannis D, Edwards J, Ibrahim A. Recent advances in the management of mucormycosis: from bench to bedside. Clin Infect Dis. 2009;48(12):1743-51.
9. Roden M, Zaoutis T, Buchanan W, Knudsen T, Sarkisova T, Schaufele R, et al. Epidemiology and outcome of zygomycosis: a review of 929 reported cases. Clin Infect Dis. 2005;41(5):634-53.
10. Cornely O, Arikan-Akdagli S, Dannaoui E, Groll AH, Lagrou K, Chakrabarti A, et al. ESCMID and ECMM joint clinical guidelines for the diagnosis and management of mucormycosis 2013. Clin Microbiol Infect. 2014;20:5-26.

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