



## Case report

## Colonic mucormycosis in an immunocompetent patient with endocarditis



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## ABSTRACT

Mucormycosis is a fatal opportunistic fungal infection. Rarely it can occur in immunocompetent patients. Here, we present a case of colonic mucormycosis in immunocompetent patients.

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## Introduction

Mucormycosis is a fatal opportunistic infection that typically occurs in immunocompromised patients [1]. Most commonly, it manifests as a rhinocerebral infection [1]. Primary gastrointestinal mucormycosis is rarely reported in literature [3]. Immunosuppression is the main risk factor associated with mucormycosis infections, however there are other factors that have been shown to increase the incidence of primary GI mucormycosis mainly in patients predisposed to acute or chronic gastric ulcers secondary to smoking or alcohol use [2]. It is more often reported with transplant recipients who are on corticosteroids and immunosuppressant drugs [2]. In the absence of immunosuppression, breach in the gastric mucosa has been thought to be an important predisposing factor for this condition [2]. Primary gastrointestinal Mucormycosis is an uncommon disease associated with high mortality rate [3]. The most common site involved is stomach [3]. The patient with gastrointestinal mucormycosis has mortality rate of 85 % [3]. Mucormycosis is the second most common mold after aspergillus for invasive infections [3].

## Case report

A 72-year-old male with a history of Type 2 Diabetes mellitus well controlled, Benign Prostatic Hypertrophy, Gout, and splenectomy in 2011 due to trauma, originally presented to an outside

Emergency department with complaints of having shaking/rigors and few days of what he described as “a cold”. At that facility, he had a temperature of 102.8 F and was hypotensive and hypoxic. His blood pressure improved with fluid resuscitation, however he was emergently intubated due to worsening respiratory failure. Propofol was started for sedation and the subsequent drop in pressure resulted in the need for pressors, so norepinephrine was started. Additional workup included a CT Angiography of Chest which showed no Pulmonary Embolism. He was then transferred to our facility.

Upon arrival, he was found to have acute kidney injury with Creatinine of 1.79 mg/dl and an elevated lactic acid at 4.5 mmol/l. Norepinephrine was continued, but vasopressin and stress dose Corticosteroids were needed to help the patient maintain an adequate MAP. Vancomycin, cefepime, and metronidazole were started. Given his hemodynamic instability without a clear cause, a bedside Transthoracic echocardiogram was performed to assess tamponade, but it was essentially normal. His hemodynamics improved and his pressors were able to be titrated off over the next 72 h. His creatinine continued to worsen during this time. Blood cultures (two bottles) collected at the outside facility returned positive for *Streptococcus infantarius* (formerly *Bovis*). Broad spectrum antibacterials were continued. His creatinine did not improve and the patient had to be transitioned to continuous renal replacement therapy. Ultimately, he required hemodialysis. His blood cultures cleared, but the patient had continued leukocytosis. A TEE was performed which showed a 5 mm aortic valve vegetation. The patient was transitioned to ceftriaxone for endocarditis. He continued to be febrile with temperatures of 101.0 F and his rectal tube began to have melanotic stools, followed

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by bright red bloody output, which prompted further investigation with a CT of his abdomen and pelvis. Imaging revealed circumferential colonic wall thickening consistent with infectious, inflammatory, or ischemic colitis. Due to this finding, metronidazole was added to ceftriaxone for additional anaerobic coverage. A subsequent drop in hemoglobin resulted in the patient being transfused 2 units Packed Red Blood Cells.

Gastroenterology was consulted who performed bedside EGD and flexible sigmoidoscopy. The patient was still sedated with propofol at this time and remained on the ventilator. Flex sigmoidoscopy showed localized severe inflammation characterized by erosions, erythema, and granularity in the sigmoid colon. Biopsies were taken for histology. Endoscopy showed grade C reflux esophagitis with some non-bleeding gastric ulcers. Surgery was then consulted due to the biopsy findings of the sigmoidoscopy and a total colectomy was performed. Overall, the procedure was well tolerated. Over the next several days, the patient was weaned off the ventilator and transitioned to nasal cannula. The surgical pathology and sigmoid biopsies returned positive for fulminant colitis with full thickness necrosis secondary to mucormycosis. Fungal culture grew *Lichtheimia (Absidia)*. CT of abdomen, pelvis, chest, and sinuses was performed to investigate for any other possible manifestations of mucormycosis. All further imaging was essentially unremarkable. Patient was started on IV liposomal amphotericin B 500 mg every 24 h. After one week of therapy, repeat upper endoscopy and colonoscopy was repeated to search for persistent mucormycosis in the ileum or rectal stump in addition to ruling out malignancy. These biopsies were negative for infection as well as malignancy. Overall treatment plan was discussed at length, as the patient does not have known immunodeficiency and it was hypothesized that the endocarditis led to the ischemic colitis which harbored a reservoir for mucormycosis to grow. Regardless, the final plan was to treat with 14 days liposomal amphotericin B, followed by isavuconazole for several months with frequent Infectious Disease follow up appointments. He had infusion set up for ceftriaxone to finish the antibacterial therapy for endocarditis.

Patient was discharged to a long term acute care facility for rehabilitation (LTACH). However during a follow up with infectious disease, the patient complained of abdominal pain. CT of the abdomen and pelvis was done which showed small abscess formation. Patient was admitted to the hospital and two drains were placed by interventional radiology(IR). The culture from

abscess that was sent out once again grew *Lichtheimia (Absidia)*. Patient had been on isavuconazole at discharge which was changed to posaconazole at the follow up. After this finding, the patient was put back on amphotericin B. General surgery was consulted who recommended that the patient be transferred to a tertiary care center for surgical intervention. No surgical Intervention was done at the tertiary care center. The patient was discharged to a long term acute care facility (LTACH) and he continues to do well.

## Discussion

Colonic mucormycosis accounts for 9% cases of mucormycosis and it carries a high mortality [4]. The first case of mucormycosis was reported by Paultauf in 1885 [3,5]. Standard treatment of mucormycosis involves treating with intravenous liposomal amphotericin B initially, followed by a course of posaconazole/ isavuconazole. There have not been standardized treatment guidelines for GI mucormycosis, however it is common to start with amphotericin B. The role of combination therapy has not been elucidated [5]. An added confounder in this case is that the patient was not immune-suppressed unlike the typical mucormycosis patient. This posed additional questions when developing a treatment plan for the patient, as most research has been performed on immunocompromised individuals.

## Declaration of Competing Interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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