Invasive Gastrointestinal Mucormycosis Presenting as Pneumonia

Avinash Bhat Balekuduru
Athish Shetty
Gajendra Ramachandraiah
Satyaprakash Bonthala Subbaraj

Department of Gastroenterology, M.S.Ramaiah Hospitals, Bengaluru, India.

Corresponding Author: *Dr Avinash Bhat Balekuduru*
Email: avinashbalekuduru@gmail.com

Mucormycosis (zygomycosis) is a mould infection caused by a group of ubiquitous fungi (*Mucorales*). *Rhizopus oryzae* is the most common cause of mucormycosis[1]. The risk factors for the development of invasive mucormycosis are diabetes mellitus, particularly with ketoacidosis, corticosteroid use, neutropenic states in organ/stem cell transplantation or hematologic malignancies, malnourished states in renal failure, low birth infants, human immunodeficiency virus (HIV) infection and states of iron overload or treatment with desferoxamine. Nosocomial outbreaks may occur by transmission through contaminated bandages, tongue depressors and intravenous catheters[1].

We present a case of invasive intestinal mucormycosis presenting with pulmonary symptoms in an elderly diabetic woman.

Case Report

A 75-year-old female presented to us with complaints of shortness of breath for the last 10 days and cough with purulent expectoration for one week. She also had severe right lower quadrant abdominal pain for the last two days. Her medical history revealed that she had hypertension for the past 15 years, uncontrolled diabetes for the last 10 years on oral hypoglycaemic agents glimepiride and metformin and chronic obstructive airway disease (COAD) treated with bronchodilators and inhalation steroids. She had been evaluated at an outside hospital for her current complaints with contrast enhanced computed tomography (CT) abdomen which revealed long segment enhancing circumferential wall thickening in the distal ileal loops, cecum and ascending colon. She was referred to us for further management. On admission, she was hypotensive with a blood pressure of 80/50 mm of Hg, and pulse rate of 120 beats/min. Systemic examination was normal except for bilateral pitting pedal oedema and right basal coarse crepitations. Laboratory examination showed haemoglobin of 14gm/dL, total leucocyte count of 22800/mm^3 with neutrophils being 92%, and platelet count of 1.85 L/mm^3. Serum creatinine was 0.85 mg/dL. Serum electrolytes were normal. Liver function tests were normal except for hypoalbuminemia (serum albumin: 3gm/dl). She had normal coagulation parameters.

Her chest radiograph showed right lower lobe consolidation (**Figure 1**). Arterial blood gas analysis showed metabolic acidosis. She was intubated in view of tachypnea and hypoxemic respiratory failure. She was admitted in an intensive care unit and started on parenteral broad spectrum antibiotics, inotropes and

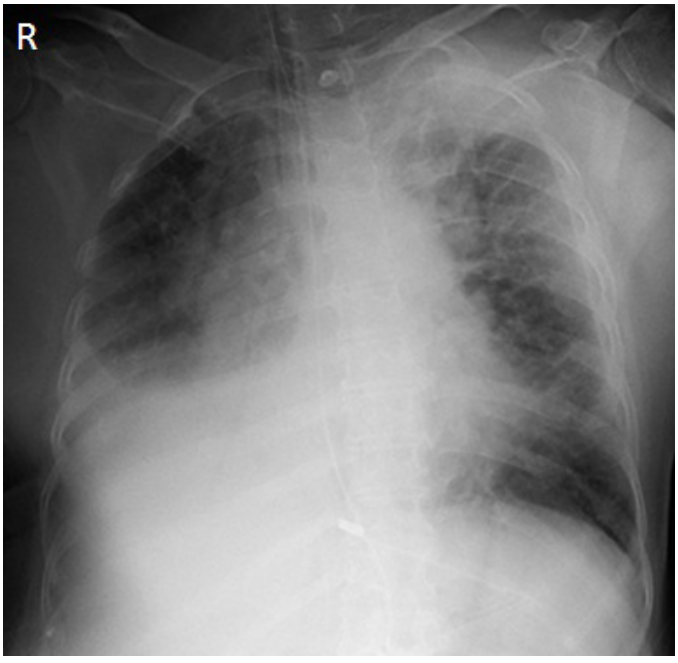

Figure 1: Chest radiograph showing right lower zone consolidation with increased bronchovascular markings.

fluid resuscitation. Bed side colonoscopy revealed a semi-circumferential slough-covered ulcerated friable lesion with luminal compromise at the hepatic flexure (**Figure 2**). The rest of the visualised colonic mucosa was normal. Multiple biopsies were taken from the abnormal mucosa to ascertain the aetiology. Histopathology showed focal areas of ulceration and acute suppurative inflammation. Several broad , sparsely septate , wide angle branching fungal elements were noted within the mucoid material and within the crypt lumen suggestive of mucormycosis (**Figure 3**). Microbiological culture and species subtyping was not done. She was started on intravenous amphotericin-B (5 mg/kg/day), but she succumbed to the illness after 5 days of treatment.

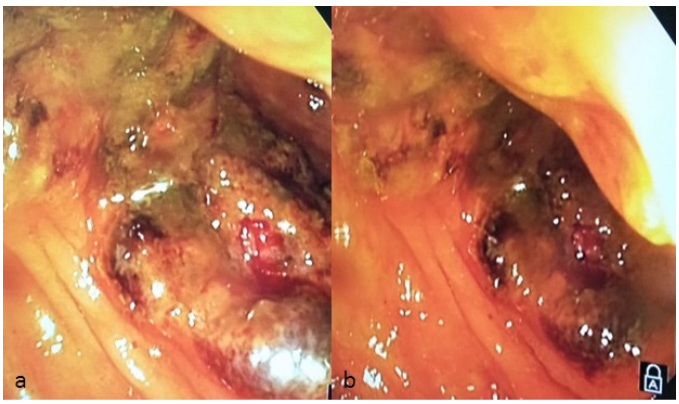

Figure 2: Colonoscopic pictures revealed semi circumferential slough covered ulcerated friable lesion with luminal compromise at hepatic flexure.

Discussion

Mucormycosis may be classified as rhino-cerebral, pulmonary, cutaneous, GI or disseminated disease. Rhino-cerebral mucormycosis is due to the inhalation of spores into the paranasal sinuses of susceptible individuals and is the most common clinical presentation. Gastrointestinal (GI) mucormycosis is rare and is acquired by the ingestion of pathogens in foods such as fermented milk, dried bread products, and fermented porridge[2]. The stomach is the most common site (58%), followed by the colon (32%). The ileum and the oesophagus are rare sites of involvement. Patients present with abdominal pain, hematemesis and necrotic ulcers that can lead to perforation peritonitis and shock with poor prognosis[2].

The diagnosis of GI mucormycosis relies upon the identification of organisms on endoscopic biopsy and histopathology. Tissue culture often yields no growth. Mucormycosis hyphae are broad (5 to 15 micron diameter), irregularly branched, and have rare septations in contrast to Aspergillus septate hyphae which are narrower (2 to 5 micron diameter) with regular branches. Serum tests (1,3-beta-D-glucan assay and the Aspergillus galactomannan assay) can detect aspergillosis but not mucormycosis due to the lack of these cell wall components[1]. The presence of more than 10 pulmonary nodules, pleural effusion, concomitant sinusitis, reverse halo signs on CT chest and prior voriconazole prophylaxis is more suggestive of mucormycosis than aspergillosis[3].

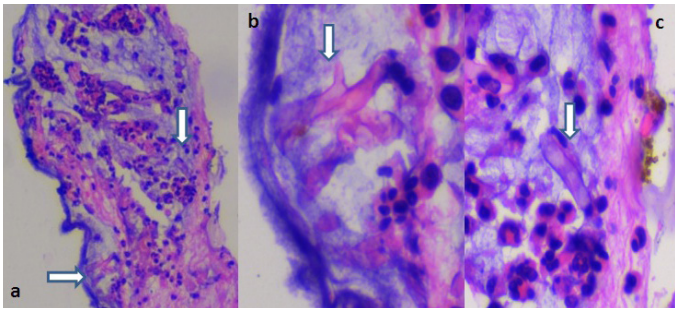

Figure 3: Histopathology: Hematoxylin and eosin (H&E) stain (a): focal area sofulceration (horizontal arrow), acute suppurative inflammation (inverted arrow) and several broad, sparsely septate (b): wide-angle branching (c): fungal elements of zygomycetes (inverted arrows).

Tissue necrosis due to angioinvasion and vascular thrombosis by hyphae prevents tissue penetration of antifungal agents and surgical debridement becomes essential in treatment[1].

Treatment of GI mucormycosis involves a combination of surgical debridement of involved tissues, antifungal therapy and elimination of predisposing factors for infection, such as hyperglycemia, metabolic acidosis, deferoxamine administration, immunosuppressive drugs, and neutropenia[1]. Intravenous amphotericin B is the drug of choice for initial therapy[1]. Posaconazole is used as step-down/salvage therapy for patients who have responded to /intolerant of amphotericin B[1].

The duration of antifungal treatment is guided by the resolution of clinical and radiological features which may take 6 to 8 weeks. The recommended therapeutic drug level of posaconazole is a trough concentration of 1 µg/mL or higher[4]. Delayed presentation with inability to remove the infected intestinal segment and use of parenteral antifungal therapy alone in an immunocompromised patient might have led to fatality in our case. Therefore, a high degree of clinical suspicion, early diagnosis and prompt surgical debridement with anti -fungal therapy might improve survival.

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Intra-Biliary Colorectal Metastasis in Lynch Syndrome: Fifteen Years After the Index Cancer

Anju Mullath[1]
Abraham Koshy[1]
Roy J Mukkada[1]
Antony Paul Chettupuzha[1]
Shelly C Paul[1]
Pradeep G Mathew[1]
Pushpa Mahadevan[2]
Murali Krishna[3]

[1]Department of Gastroenterology, [2]Department of Pathology, Lakeshore Hospital and Research Centre, Cochin, [3]Department of General Surgery, Military Hospital, Palampur, India.

Corresponding Author: *Dr Anju Mullath*
Email: anjuachuthamenon@gmail.com

Colorectal cancer is the third most common malignancy in the world. About 5 % of cases are attributed to hereditary causes with Lynch syndrome being among the commonest. Although the prevalence rate in the Indian subcontinent is comparatively low, the percentage of hereditary cases remains surprisingly the same as worldwide estimates. The lack of awareness and non-existent screening protocols often lead to preventable morbidity in these cases. Here we present a case of hereditary colorectal carcinoma with recurrent malignant lesions and a relatively rare intra-biliary metastasis from colorectal carcinoma.

Case Report

A 35-year-old male patient presented to our hospital with complaints of yellowish discolouration of the eyes and pruritis. He had undergone multiple medical and surgical interventions in the past.

At the age of 20 years, he was detected to have rectal carcinoma with liver metastasis. He underwent neoadjuvant chemotherapy with oxaliplatin and