

Massive Lower Gastrointestinal Bleeding Secondary to Colonic Mucormycosis

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SUMMARY

Lower gastrointestinal bleeding is usually due to haemorrhoids, diverticular disease, or colorectal cancer. Infective causes of gastrointestinal bleeding are rare. A 70-year-old lady was admitted with septic shock secondary to community acquired pneumonia. She later developed massive lower gastrointestinal bleeding secondary to colonic mucormycosis. Her condition deteriorated rapidly and she died of septicemia. Mucormycosis of the colon is extremely rare and is still associated with a high mortality.

KEY WORDS:

haematochezia, Bleeding per rectum, Opportunistic infection, Colonic bleeding

INTRODUCTION

Gastrointestinal (GI) bleeding is an important cause for acute hospital admissions and is still associated with significant morbidity and mortality. Compared to upper GI bleeding, lower GI bleeding is generally less common and is often due to haemorrhoids, diverticular disease, angiodysplasia and colorectal neoplasm^{1,2}. However, it is important to be aware of the less common causes of bleeding. We report a case massive lower GI bleeding secondary to colonic mucormycosis.

CASE REPORT

A 70-year-old lady with a background history of hypertension, ischaemic heart disease and previous gastric ulcers was admitted with septic shock. There was history of fever for one day. At presentation, she was hypoxic, hypotensive and disorientated. Respiratory examination showed diffuse crepitations. The rest of the system examinations were unremarkable. She was immediately intubated, stabilised and transferred to the intensive care unit.

Investigations showed leukocytosis ($13.9 \times 10^9/L$, [4-11]) with 80% neutrophils, anaemia (8.8 gm/dL, [12-15]), mild thrombocytopenia ($140 \times 10^9/L$, [150-440]), elevated C-reactive protein (14.2 mg/dL, [0-0.5]), mild renal impairment (serum urea 9.5 mmol/L, [2.5-6.7]) and creatinine of 120 $\mu\text{mol/L}$ [50-98]). Clotting and liver profiles were normal apart from a mildly depressed serum albumin. Chest radiography showed extensive consolidation of both lung fields consistent with severe pneumonia or adult respiratory distress syndrome. She was started on meropenem 1 gm t.i.d and

ionotropic support to maintain her blood pressure.

A computed tomography showed extensive ground glass appearance, thickened interlobular septae and honeycombing consistent with pulmonary fibrosis with superimposed pneumonia. There were no masses or lymphadenopathies seen. Autoimmune profiles that included rheumatoid factor, antinuclear antibody and lupus anticoagulant were all positive. Cultures from blood, sputum, urine and stool all yielded no growth. A rheumatologist consult strongly suggested autoimmune disease with underlying pulmonary fibrosis that was complicated by pneumonia. Intravenous hydrocortisone, 100mg t.i.d was initiated.

Three weeks into admission and after eight days of steroid therapy, she developed a massive lower GI bleed with a four grams drop of haemoglobin. An urgent colonoscopy showed a stenosing nodular lesion near the hepatic flexure with friable mucosa suspected to be colorectal cancer. Mucosa beyond the nodularity appeared inflamed and covered with necrotic yellow slough. Multiple biopsies were taken. The rest of the colon was normal. Despite continued aggressive therapy, her condition deteriorated and she died three days later. Post mortem examination was not done for religious reason. Surprisingly, the histology did not show any evidence of malignancy but showed multiple non-septate hyphae invading into the mucosa and submucosa (Fig. 1). The findings were consistent with mucormycosis and the cause of death was septicemia and probable disseminated mucormycosis.

DISCUSSION

Mucormycosis is an opportunistic mouldy ubiquitous fungus that is found in soil and decaying organic matter. The organism is mainly acquired either through ingestion or inhalation. Clinical infections in human are extremely rare and typically occur in those with immune-compromised state such as haematological malignancies, post-transplantations, severe acidosis with poorly controlled diabetes mellitus or severe malnutrition are particularly at risk^{3,4}. It has also been reported in patients with iron overload disorders receiving chelation therapy, post-cytotoxic therapy and even after biologic therapy with infliximab for Crohn's disease. Of concern is that the incidence of mucormycosis is reported to be increasing, paralleling the increase in the number of patients with haematologic malignancies or bone marrow transplants.

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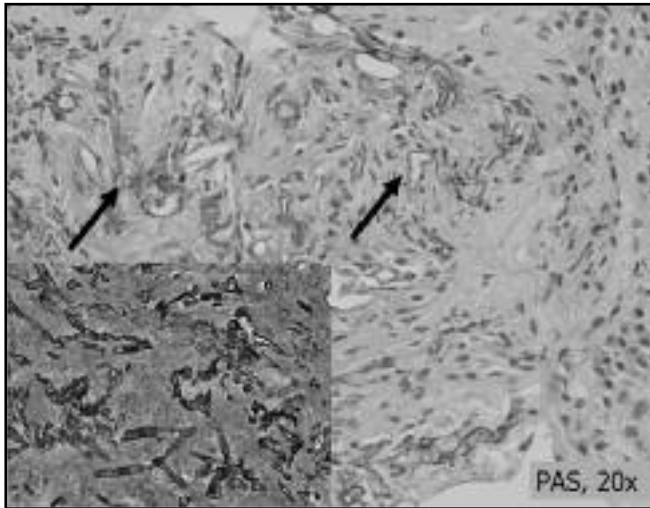


Fig. 1: Histology showing non-septate hyphae invading into the mucosa and submucosa (Periodic acid Schiff, x20) and insert showing non-septate hyphae variable contour branching at right angle (Methenamine Silver or Grocotts stain).

GI mucormycosis is rare and only account for seven percent of all mucormycosis⁵. In a review of 87 cases of GI mucormycosis, the most commonly affected site was the stomach (57.5%) followed by colon (32.2%), small bowel (ileum, 7%, duodenum 2.3% and jejunum 1.1%) and oesophagus (7%)⁵. Oesophageal involvement was often associated with involvement the other part of GI tract.

The hallmark of mucormycosis is vascular invasion leading to endarteritis, thrombosis resulting in tissue ischaemia and infarction. Therefore, ischaemic colitis like presentations is a common manifestation of colonic mucormycosis. However,

the clinical presentations of colonic mucormycosis can be non-specific, ranging from non-specific abdominal pain resembling constipation to diarrhoea and perforation. Apart from our case, there has only been one other reported case manifesting with massive lower GI bleed that was caused by a mixed infections of mucormycosis with cytomegalovirus in a renal transplant patient. This patient survived with appropriate and adequate treatment. Similar to what have been widely reported, the actual diagnosis was only made after histological examination.

Invasive GI mucormycosis carries a high mortality rate of over 90%³. Most are still diagnosed late or at post mortem. Fortunately, the overall prognosis of mucormycosis (inclusive of other organs affected) is presently improving with aggressive tissue debridement and prolonged anti-fungal therapy.

In conclusion, we report a case of massive lower GI bleeding secondary to mucormycosis of the colon in a patient without underlying reported risk factors. The only risk factor was a short course on intravenous steroid used to treat a newly diagnosed autoimmune disease with underlying pulmonary fibrosis and possibly old age.

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