



Case Report

Isolated enteric aspergillosis in a non severely immunocompromised patient. Case report and literature review



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ABSTRACT

Invasive aspergillosis is a life-threatening, opportunistic fungal infection that mainly affects immunocompromised patients, particularly those receiving immunosuppressive therapy or high-dose corticosteroids. The most common portal of entry in aspergillosis is the lung, so the isolated occurrence of intestinal aspergillosis in the absence of pulmonary disease is an unusual pattern.

Case report: We report a case of a lung cancer patient who did not receive chemotherapy, presenting to the emergency department with fever and chills. The patient also developed abdominal pain and bloody diarrhoea during his hospitalisation. Colonoscopy revealed diffuse epithelial and subepithelial oedema of the terminal ileum, linear ulcers, denudation of the mucosa, and necrosis. Histopathological examination of terminal ileum diffuse biopsies showed haemorrhagic infiltrations, while microscopy revealed Y-shaped, branching septate hyphae, diagnostic for *Aspergillus* fungi. Additionally, the culture of biopsy samples showed the growth of *Aspergillus fumigatus*. The patient was negative for *Aspergillus* antibodies in his blood, and bronchoscopy and sputum samples were negative as well.

Conclusion: This is a rare case of isolated aspergillosis of the small intestine, as there is no pulmonary or other systemic participation, as it also happens in a patient not being in a severely immunocompromised state.

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Introduction

Fungal infections are frequent in patients treated with chemotherapy in oncologic and haematologic departments [1]. Disseminated aspergillosis is a rapidly progressive and highly lethal infection in patients with altered host defenses resulting mainly from immunosuppressive therapy. The disease most frequently involves the respiratory tract, and the signs and symptoms include fever, cough, dyspnoea, and hemoptysis. Secondary dissemination to the brain, gut, liver, kidney, thyroid, bone, eye, skin, and heart has been reported. The number of cases of invasive fungal infections is increasing, with a 14-fold increase in the number of cases observed between 1978 and 1992 in an autopsy survey, which is probably related to the increasing use of intensive cytotoxic chemotherapy [2]. According to one report, the gastrointestinal tract was the site of 41% of deep fungal infections in

immunocompromised patients, with 22% affecting the lower part of the gastrointestinal tract [3]. Although *Aspergillus* species can invade the gastrointestinal tract, the vast majority of patients with bowel involvement in the setting of disseminated disease have been reported to invariably present with coexisting, clinically predominant lesions in other organs, which almost always include the lungs. In a limited number of cases, the intestine is the only site of infectious localisation. In this situation, *Aspergillus* usually makes its way through the intestinal mucosa eventually disrupted by chemotherapy's toxicity with consequent deterioration of gastrointestinal immunity. This solitary clinical presentation can have a high death rate [4,5]. Prescott et al. reported that 12 of 14 patients with lower gastrointestinal fungal infection also had pulmonary and upper gastrointestinal infection [3]. Similarly, Schwesinger et al. reported that 25 of 27 patients with aspergillosis had pulmonary involvement [6]. Disseminated aspergillosis can cause bowel infarction or diffuse peritonitis due to small bowel perforation [7–10]. Moreover, the majority of these patients succumbed within a short time [11].

Histopathology continues to be a rapid and cost-effective means of providing a presumptive or definitive diagnosis of an invasive fungal infection. Proven invasive fungal infection requires fungus

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Fig. 1. Small bowel follow-through showed dilatation, thickening, and oedema of the intestinal wall mainly at the ileum region (arrow).

detection by histological analysis or direct microscopy of a specimen of tissue taken from a site of disease, in which hyphae or melanised yeast-like forms are seen accompanied by evidence of associated tissue damage. Culture in combination with evidence of tissue invasion on histopathology provides the most certain evidence of invasive aspergillosis. However, both microscopic examination and culture are insensitive methods. Additionally,

performing a biopsy is not feasible in some patients due to the risk of bleeding or other complications. In those cases, clinicians will have to rely on alternative testing [12]. *Aspergillus* spp. are usually described as thin (3–12 μ m), septate, acute-angle (45°), or dichotomous branching hyphae. Histopathological examination can usually distinguish *Aspergillus* spp. and the other fungi; however, several filamentous fungi have similar appearances [13]. A study of 122 specimens showed concordance in 83% of cases with septated, acute-angle-branching hyphae in histology and the presence of *Aspergillus* spp. in culture [14].

Galactomannan is a major constituent of *Aspergillus* cell walls that is released during growth of hyphae. A double sandwich enzyme-linked immunosorbent assay (ELISA) that detects the galactomannan antigen is used on serum and bronchoalveolar lavage (BAL) fluid as an adjunctive test for the diagnosis of aspergillosis [15]. Finally, a systemic review and meta-analysis suggested that the sensitivity and specificity of polymerase chain reaction (PCR) to detect invasive aspergillosis was 88% and 75%, respectively [16].

Case report

A 76-year-old male, recently diagnosed with unresectable lung cancer and scheduled for chemotherapy, was admitted to the emergency department with fever and chills. The patient did not report any other specific symptoms. His medical history included chronic pulmonary obstructive disease, in the context of cigarette smoking (40 pack-years), and arterial hypertension. The complete blood count showed leukocytosis and biochemical tests showed elevation of C-reactive protein. The chest X-ray was normal. Stool testing was negative for *Clostridium difficile* enterotoxin and urine

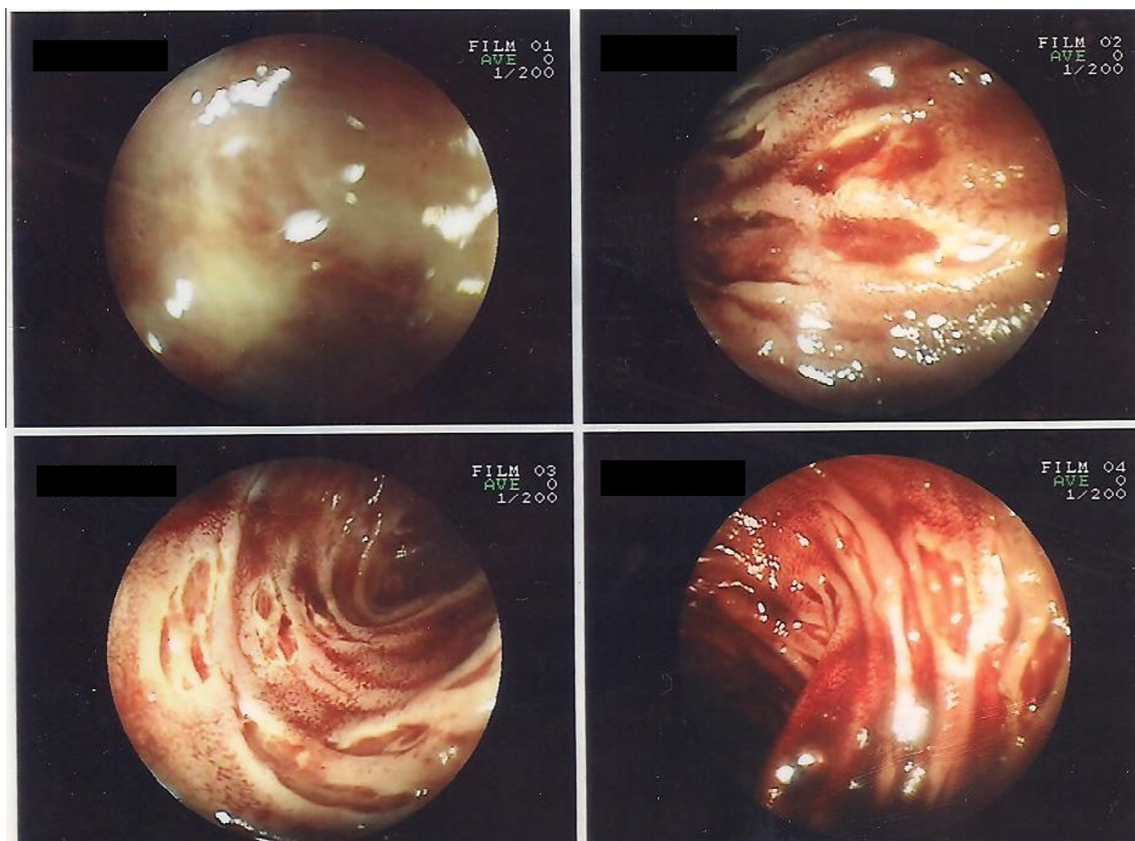


Fig. 2. For a depth of at least 40 cm into the ileum, the lumen was found full of fresh bleeding, with diffuse epithelial and subepithelial oedema, continuous serpiginous and linear ulcers, denudation of the mucosa, and necrosis.

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