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Disseminated Intra-Abdominal Aspergilloma With Abdominal Wall Invasion in a Patient With Acute Myeloid Leukemia: A Case Report

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Clinical Practice Points

- Invasive aspergillosis represents a major cause of morbidity and mortality in immunocompromised patients. Involvement of the gastrointestinal (GI) tract by *Aspergillus* infection is mostly reported as part of a disseminated infection from a primary pulmonary site and only rarely as an isolated organ infection.
- The case we report here describes an aggressive *Aspergillus* infection that arose from the jejunum and invaded the abdominal wall in a patient with acute myeloid leukemia (AML) after induction chemotherapy. There was no evidence of pulmonary or disseminated *Aspergillus* infection.

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Introduction

Acute myeloid leukemia (AML) is considered the most common form of acute leukemia in adults, with an expected significant increase in incidence with the aging population.[1-3] Treatment of AML is based on an initial induction phase to generate remission followed by a subsequent consolidation phase to prevent relapse.[3] Standard of care for induction in patients < 60 years of age with good performance status consists of a combination of cytarabine and an anthracycline, which is known as the "7 + 3 regimen."[3] In most cases, treatment can be complicated by various infections, which are exacerbated by an altered immune status from the leukemia in the setting of toxic side effects of chemotherapy. Among these infections, fungal infections carry high mortality. Simultaneously, because of the extensive development of new chemotherapeutic agents and the increase in bone marrow transplantation, the incidence of invasive aspergillosis in treated patients with hematologic malignancies has increased considerably.[3]

Aspergillus is an inhaled ubiquitous fungus known to be present mainly in the respiratory pathways, which can cause severe invasive pulmonary infections in immunocompromised hosts, with possible dissemination to other organs through hematologic spread. However, primary extrapulmonary cases involving the gastrointestinal (GI) tract, heart, kidney, central nervous system, and liver have been reported as the initial presentation of the disease.[4] GI tract invasive aspergillosis commonly presents with high-grade fever, abdominal pain or distention, nausea, vomiting, diarrhea, and GI bleeding. Despite the absence of a single specific radiologic sign diagnostic of GI aspergillosis, the first-line imaging modality that should be performed when in doubt is contrast-enhanced computed tomography (CT), with findings ranging from segmental mural thickening of the small bowel to inflammation of the mesenteric fat to bowel distention, obstruction, or perforation.[4,5]

However, the definitive diagnosis is made by the histopathologic findings, which reveal the presence of *Aspergillus* hyphae and mycelial growth, with surrounding evidence of inflammatory cells with mucosal alterations or exclusive tissue invasion with microvascular involvement, or both. These findings can be obtained by pathologic examination of an endoscopic biopsy specimen or surgically resected bowel specimen.[6] The optimal treatment of aspergilloma is a controversial subject and relies on antifungal or surgical treatment, or both.[7,8]

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In this report, we describe a very rare case of GI aspergillosis infection arising from the jejunum and invading both the bowel wall and the abdominal wall.

Case Report

We report the case of a 66-year-old man who was a remote heavy smoker and was known to have a psychotic disorder (treated with trihexyphenidyl, chlorpromazine, and perphenazine). He was transferred to our care for further diagnostic workup and management of pancytopenia. His initial studies on presentation revealed anemia, neutropenia, and thrombocytopenia (hemoglobin, 11.0 g/dL; white blood cell count, 1.03×10^3 cells/μL; platelet count, 122×10^3/μL). A bone marrow aspirate and biopsy specimen revealed AML subtype M1 according to the French American British classification, with > 40% blast cells. His cytogenetic analysis revealed a normal karyotype, and molecular analysis revealed no *FLT3* or *NPM1* mutations. The patient was then given 7 + 3 induction chemotherapy with cytarabine (200 mg/m^2) for 7 days (continuous infusion) and idarubicin (12 mg/m^2) intravenously for 3 days.

Even before starting chemotherapy, the patient complained of continuous intractable nausea with bilious vomiting that was refractory to multiple antiemetic agents. His symptoms worsened after receiving chemotherapy. Emergent CT of the abdomen showed no obstruction but revealed extensive ileitis and colitis. On day 14 after induction, a bone marrow aspirate showed acellular bone marrow with no evidence of blastosis. On day 15, he was prescribed granulocyte-colony stimulating factor (G-CSF). He also experienced neutropenic fever (absolute neutrophil count < 50 Cells/mm^3 with fever up to 39.3°C). He was managed with multiple broad-spectrum antibacterial and antifungal agents (fluconazole, which was then switched to voriconazole) with no significant improvement; his nausea and vomiting persisted. Because severe hypotension that was unresponsive to fluid resuscitation developed, the patient was transferred to the intensive care unit, where he remained in a pancytopenic state. A bone marrow aspirate on day 30 continued to be hypocellular, with no evidence of residual leukemia. Five days later, the absolute neutrophil count began to increase; concomitantly, a hard palpable abdominal mass was noticed in the left upper quadrant on physical examination. CT of the abdomen then revealed a hypoattenuating area peripherally circumscribed by a thin enhancing rim measuring 6 × 5 cm that was in continuity with an area of the jejunal loop distention, with total effacement of the bowel wall and dense infiltration of the surrounding fat, favoring aspergilloma over a leukemic infiltrate (Figure 1). Pathologic results from an ultrasonographically guided core biopsy specimen showed necrotic tissue enclosing septate branching hyphae consistent with *Aspergillus* abscess. Liposomal amphotericin B therapy was then initiated at a dose of 5 mg/kg/d.

The patient's white blood cell count recovered by day 45, and he underwent laparoscopic exploration, which revealed a large mass involving multiple jejunal loops and the surrounding part of the transverse colon. Extension to the abdominal wall was also obvious over a large surface area. Dissection and excision of this mass necessitated the removal of 33 cm of the jejunal loop and 7 cm of the transverse colon. Resection of the abdominal wall at the site of

Figure 1 **(A) Computed Tomographic Scan of the Abdomen Showing the Jejunal Mass Invading the Bowel Wall Outward Into the Abdominal Wall. (B) Computed Tomographic Scan of the Abdomen, Showing Segmental Small Bowel Wall Thickening, Characteristic of *Aspergillus* Infection of the Small Bowel. (C) Computed Tomographically Guided Biopsy of the Abdominal Mass, Leading to the Diagnosis of Jejunal Aspergilloma With Abdominal Wall Invasion**