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Isolated Ileocolonic Histoplasmosis in an HIV-Negative Patient

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Histoplasmosis infection typically occurs in HIV-positive patients manifesting as either pulmonary lesions or severe disseminated pattern implicating multiple organs. While gastrointestinal (GI) involvement is identified in over 70% of autopsy cases in disseminated disease, clinical symptoms from GI manifestation is uncommon, reported by only 3–12% of patients [1]. More infrequent is isolated ileocolonic histoplasmosis, described only in case vignettes [2,3]. However, this atypical presentation may suggest clinical under-diagnosis rather than disease rarity, as symptoms are nonspecific and histoplasmosis may seldom be considered in differential diagnosis, especially outside areas of prevalence and in HIV-negative patients. Nevertheless, accurate diagnosis and timely treatment are necessary to avoid life-threatening dissemination. We present a case of isolated gastrointestinal histoplasmosis in a patient with autoimmune diseases undergoing chronic immunosuppression.

A 58-year-old Hispanic male with systemic sclerosis and interstitial lung disease was referred for colonoscopy after a positive fecal occult blood test. He traveled to Mexico one year prior; otherwise travel history was unremarkable. A review of systems uncovered seven months of right-lower-quadrant pain relieved with defecation, and non-bloody diarrhea alleviated by loperamide. In addition to blood pressure medications and omeprazole, he had been taking prednisone 5mg daily and mycophenolate mofetil 1000mg twice daily for six years. Colonoscopy revealed villous blunting of the terminal ileum, clean-based ulcers in the terminal ileum (Figure A) and splenic flexure (Figure B), and mild rectal erythema, consistent with patchy colitis.

Given the history of underlying autoimmune disease and chronic immunosuppression the differential diagnosis for these colonoscopic findings includes both inflammatory and

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infectious causes. For example, Crohn's disease can present with discontinuous mucosal ulcerations and has been associated with both systemic sclerosis and interstitial lung disease. Infectious causes for these findings could include cytomegalovirus (CMV), intestinal tuberculosis, or amebiasis. In this patient, treatment was deferred pending pathologic confirmation of the diagnosis. Biopsies were significant for widespread inflammation extending from the terminal ileum to the rectum and the presence of granulomas (Figure C), which prompted further staining for fungi and acid-fast bacteria. Grocott-Gomori methenamine silver (GMS) stain revealed histoplasma organisms throughout the terminal ileum (Figure D) and the colon, with the highest densities at the terminal ileal and splenic flexure ulcers (Figure E). The patient was contacted urgently and urinary histoplasma antigen was obtained but was negative. Histoplasmosis complement fixation was performed and was also negative. Itraconazole was promptly initiated for treatment. However, the patient continued to experience abdominal pain, diarrhea and fatigue, and subsequently presented to our hospital. Following the recommendation of infectious diseases, the patient received four days of intravenous amphotericin, and was transitioned to oral itraconazole to complete a twelve-week course.

Signs and symptoms of GI histoplasmosis include diarrhea, abdominal pain, dysphagia, nausea/vomiting, and bowel obstruction or hemorrhage. Ulcers are the most common lesions seen on endoscopy and are frequently located in the terminal ileum [1]. Typically, these ulcers have raised border with a necrotic base and may bleed. Other findings include mucosal hyperemia, villous blunting, and obstructive masses. In the presence of these clinical manifestations, GI histoplasmosis must be considered even when patients are not from endemic areas of the Midwest and South-central United States [4], and especially if they are immunocompromised. Close communication between the gastroenterologist and pathologist to facilitate diagnosis is critical.

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Figure.