International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Gastrointestinal Disseminated Histoplasmosis Masquerading as Sigmoid Colon Cancer in an Immunocompromised Patient

Nathan Jatczak[1], Joey Marcuccilli, OMS IV, John Hiscock, OMS IV, Evan Nuels, MD

[1]OMS IV
Corresponding Author Email: *njatczak4[at]gmail.com*

[2]OMS IV

[3]OMS IV

[4]MD

Abstract: ***Disseminated histoplasmosis is a rare fungal infection that may mimic colorectal malignancy, especially in immunocompromised individuals. We present the case of a 74-year-old male with multiple myeloma who developed near-obstructive sigmoid colon lesions initially suspected to be malignant. Histopathology confirmed Histoplasma capsulatum infection. Despite amphotericin B followed by itraconazole, the patient's condition progressed to complete obstruction, necessitating colectomy. This report underscores the diagnostic challenge of gastrointestinal histoplasmosis, the limitations of antifungal therapy in advanced lesions, and the importance of considering surgical intervention when medical management fails.***

Keywords: Histoplasmosis, Gastrointestinal Infection, Sigmoid Colon, Immunocompromised Patient, Colectomy

1. Introduction

Histoplasmosis, caused by *Histoplasma capsulatum*, is an endemic fungal infection most commonly affecting the lungs. Immunocompromised individuals, particularly those with hematologic malignancies such as multiple myeloma, are at higher risk for disseminated disease [1]. Although gastrointestinal (GI) involvement occurs in approximately 70–90% of disseminated histoplasmosis cases, it often remains subclinical and is rarely the initial presentation [1,2]. When symptomatic, GI histoplasmosis can manifest with nonspecific complaints such as diarrhea, abdominal pain, and weight loss, making it difficult to distinguish from inflammatory bowel disease or colorectal malignancy [3]. These overlapping features contribute to delays in diagnosis and treatment, increasing the risk of complications. Here, we present a case of disseminated histoplasmosis with predominant colonic involvement, initially mistaken for malignancy, ultimately leading to surgical resection due to failed medical therapy. The purpose of this case report is to highlight the diagnostic and therapeutic complexities of gastrointestinal histoplasmosis in immunocompromised patients, emphasizing the importance of early recognition, multidisciplinary management, and timely surgical consideration when medical therapy fails.

2. Methodology

This case report was prepared by reviewing the patient's clinical records, imaging studies, operative findings, and histopathology results. Relevant peer-reviewed literature on gastrointestinal histoplasmosis was reviewed to provide context for the discussion. All patient identifiers were removed to ensure confidentiality.

3. Results & Discussion

Case Presentation

A 74-year-old male with a history of multiple myeloma, previously treated with stem cell transplant and ongoing immunosuppressive therapy, presented with several weeks of progressive diarrhea, fatigue, and unintentional weight loss. Initial evaluation at an outside hospital suggested colitis, and he was started on broad-spectrum antibiotics. However, his symptoms persisted, prompting a general surgery consultation.

Colonoscopy revealed a circumferential, near-obstructing mass approximately 9 cm in length located in the sigmoid colon (Figure 1a, 1b). In the rectum, multiple ulcers were noted and biopsied (Figure 1c, 1d). Biopsies demonstrated *Histoplasma capsulatum* on histopathology, confirming a diagnosis of gastrointestinal disseminated histoplasmosis.

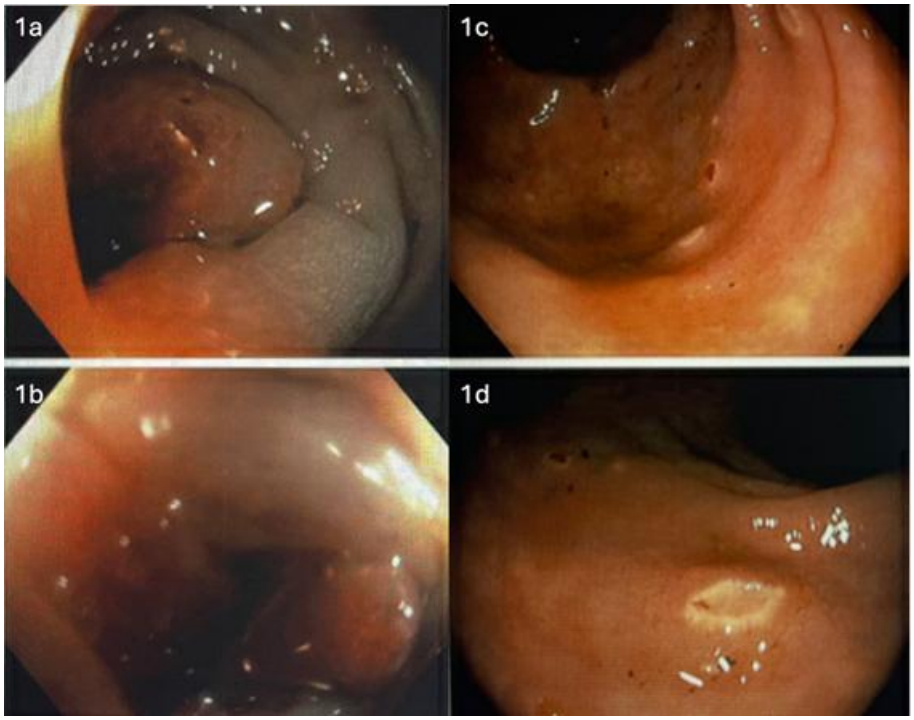

Figure 1: Colonoscopic images. (a, b) Near-obstructing circumferential sigmoid colon mass located 19 cm from the anal verge. (c, d) Multiple rectal ulcers that were biopsied. All specimens tested positive for disseminated histoplasmosis.

Following the biopsy results, a contrast-enhanced CT scan of the abdomen and pelvis (Figure 2) was performed, which demonstrated severe colonic wall thickening of the sigmoid colon, raising concern for potential malignancy versus severe infectious colitis.

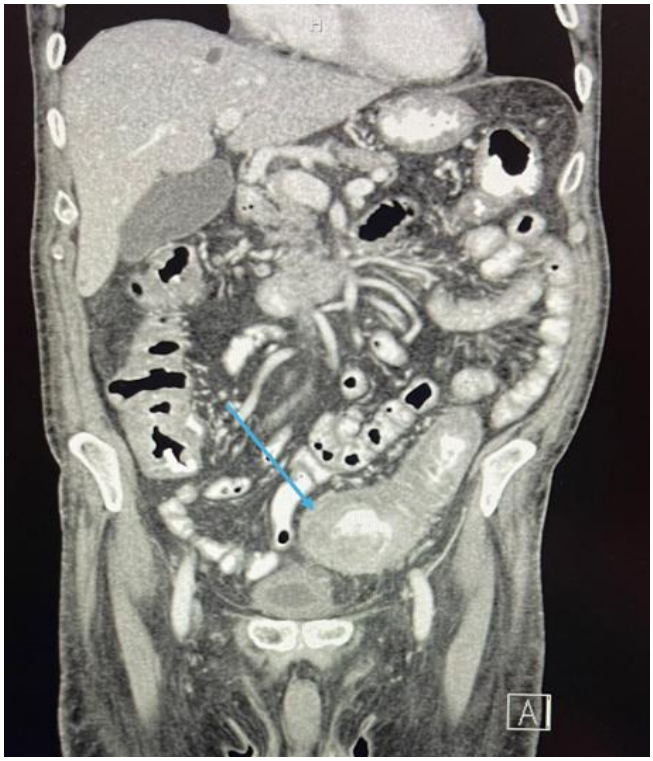

Figure 2: CT scan of the abdomen and pelvis prior to antifungal treatment.

Treatment and Outcome

Given the extent of the disease, intravenous amphotericin B was initiated. However, therapy was complicated by hypokalemia, necessitating temporary discontinuation. Once electrolyte disturbances were corrected, antifungal therapy was resumed and completed over a 15-day course, followed by a transition to oral itraconazole.

Despite appropriate antifungal therapy, the patient's symptoms improved only minimally. A follow-up CT scan performed two weeks after discharge (Figure 3) revealed persistent sigmoid wall thickening with an 8 cm segment of abnormality now in direct contact with the bladder, suggestive of a stricture and ongoing inflammation.

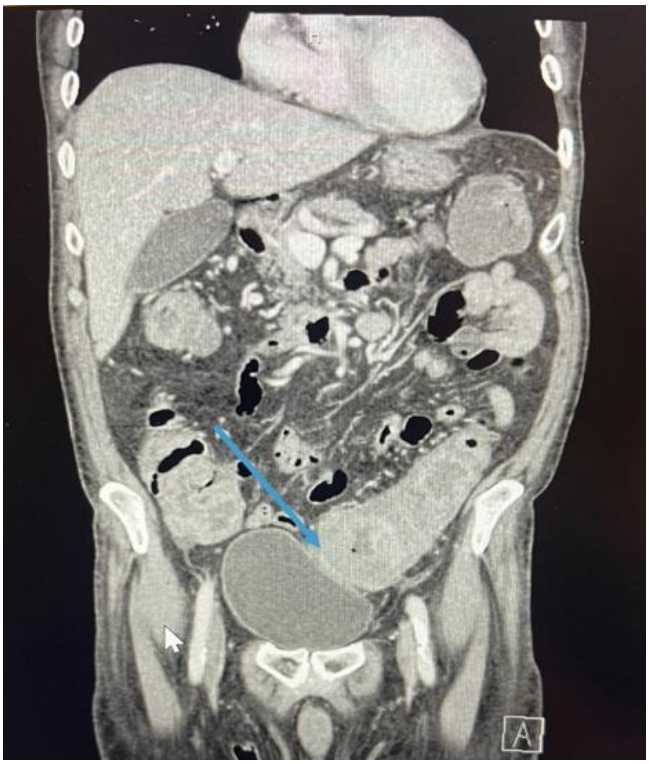

Figure 3: Follow-up CT scan two weeks post-discharge.

Seventeen days after discharge, the patient returned to the emergency department with clinical signs of large bowel obstruction, including severe abdominal distension, obstipation, and vomiting. Surgical intervention was deemed necessary due to failure of medical management and evidence of complete obstruction.

Sigmoid colectomy was performed, and intraoperative findings confirmed a firm, fibrotic stricture with marked mural thickening and adherence to surrounding tissues (Figure 4). Pathology showed transmural inflammation with granulomas and fungal organisms consistent with *Histoplasma capsulatum*, with no evidence of malignancy.

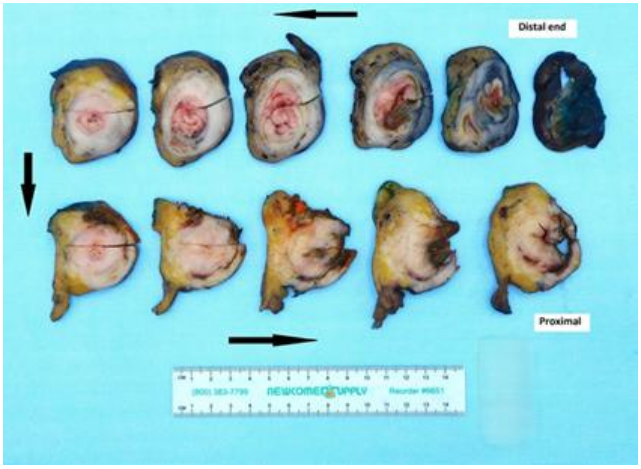

Figure 4: Gross pathology of the resected sigmoid colon demonstrating complete luminal obstruction with evidence of fungal infection.

4. Discussion

This case adds to the limited but growing body of literature on gastrointestinal histoplasmosis, illustrating that even with appropriate antifungal therapy, fibrotic stricture formation can lead to medical treatment failure and necessitate surgery. Gastrointestinal histoplasmosis is a frequently overlooked manifestation of disseminated *Histoplasma capsulatum* infection, particularly in immunocompromised individuals. The nonspecific nature of presenting symptoms such as diarrhea, weakness, and weight loss can closely mimic colorectal cancer or inflammatory bowel disease, leading to delayed or missed diagnoses [1,3,4].

In this case, initial treatment with antibiotics for presumed colitis delayed the diagnosis. It was only upon colonoscopy and biopsy that histoplasmosis was identified. Early recognition of fungal etiology is essential, as antifungal therapy can prevent progression and obviate the need for surgery [5]. Unfortunately, in this patient, amphotericin B therapy was interrupted due to hypokalemia—a well-known complication that underscores the importance of electrolyte monitoring during treatment [6,7].

Despite appropriate treatment resumption and transition to itraconazole, the lesion failed to regress. Follow-up imaging revealed continued thickening of the colonic wall and eventual development of a complete colonic obstruction, necessitating surgical intervention.

One potential reason for failure of medical therapy is the development of fibrotic strictures during the healing phase of infectious colitis. Chronic inflammation from fungal infections may lead to transmural fibrosis and luminal narrowing, which do not respond to antifungal medications alone [3,8]. Case reports and histopathologic studies have shown that persistent fibrotic changes can require surgical management despite microbiologic cure.

Furthermore, antifungal therapy may paradoxically enhance immune response leading to increased inflammation and scarring, a phenomenon somewhat analogous to immune reconstitution inflammatory syndrome (IRIS) seen in other infections [9]. These possibilities support the need for close follow-up and imaging to assess treatment response and to consider surgery if obstruction or fistula formation occurs.

Additionally, this case adds to the growing literature that suggests GI histoplasmosis should be included in the differential for patients with obstructive colorectal lesions, especially in those with immunocompromising conditions such as hematologic malignancies or organ transplant recipients [1,2,4]. A high index of suspicion is essential in endemic areas, and definitive diagnosis requires histopathological confirmation with fungal staining or culture. Cross-sectional imaging can support the diagnosis but is often nonspecific.

The utility of serum and urine antigen detection tests has been demonstrated in disseminated histoplasmosis and may aid in diagnosis when tissue is unavailable [5,6]. However, in localized gastrointestinal cases without systemic spread, antigen tests may yield false-negative results. Therefore, clinicians should rely heavily on endoscopic evaluation and biopsy in cases of suspected GI involvement.

Surgical intervention, while not always necessary, becomes critical in cases of complete obstruction, perforation, or failure of medical therapy. In our case, surgery provided symptom relief and allowed for definitive pathologic diagnosis, confirming the presence of invasive fungal disease without malignancy. The postoperative course in such patients should include continued antifungal therapy to prevent recurrence and ensure close outpatient follow-up.

5. Conclusion

This case demonstrates that gastrointestinal histoplasmosis, though rare, can closely mimic colorectal cancer in immunocompromised patients. Early colonoscopic biopsy is crucial for diagnosis. While amphotericin B remains first-line therapy, clinicians must anticipate possible stricture formation leading to obstruction. Prompt surgical evaluation in such scenarios, coupled with continued antifungal therapy postoperatively, offers the best chance for recovery.

References

[1] Assi M, Martin S, Wheat LJ, Hage CA. Histoplasmosis in the immunocompromised host. *J Fungi (Basel)*. 2019;5(3):71. doi:10.3390/jof5030071

[2] Kauffman CA. Histoplasmosis: a clinical and laboratory update. *Clin Chest Med*. 2009;30(2):217-225. doi:10.1016/j.ccm.2009.02.004
[3] Goodwin RA Jr, Shapiro JL, Thurman GH, Thurman SS, Des Prez RM. Disseminated histoplasmosis: clinical and pathologic correlations. *Medicine (Baltimore)*. 1980;59(1):1-33. doi:10.1097/00005792-198001000-00001
[4] Riddell J 4th, Wheat LJ. Gastrointestinal histoplasmosis. *Curr Gastroenterol Rep*. 2003;5(4):292-296. doi:10.1007/s11894-003-0051-4
[5] Wheat LJ, Freifeld AG, Kleiman MB, et al. Clinical practice guidelines for the management of patients with histoplasmosis: 2015 update. *Clin Infect Dis*. 2016;63(7):665-675. doi:10.1093/cid/ciw444
[6] Azar MM, Loyd JL, Relich RF, Wheat LJ, Hage CA. Current concepts in the epidemiology, diagnosis, and management of histoplasmosis syndromes. *Semin Respir Crit Care Med*. 2020;41(1):13-30. doi:10.1055/s-0039-3399503
[7] Pappas PG, Kauffman CA, Andes DR, et al. Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2016;62(4):e1-e50. doi:10.1093/cid/civ933
[8] Kauffman CA, Malani AN. Fungal infections in older adults. *Clin Geriatr Med*. 2007;23(3):561-576. doi:10.1016/j.cger.2007.03.007
[9] Shelburne SA, Visnegarwala F, Darcourt J, et al. Incidence and risk factors for immune reconstitution inflammatory syndrome during highly active antiretroviral therapy. *AIDS*. 2005;19(4):399-406. doi:10.1097/01.aids.0000161763.38251.64