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Case report

Ileocecal Tuberculosis (Surgical and Pathological Diagnosis) Mimicking Inflammatory Bowel Disease: A Case Report and Literature Review

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Abstract

Introduction: We report the case of a patient with suspected inflammatory bowel disease (IBD) who failed to respond to vedolizumab therapy and was subsequently diagnosed with *Clostridioides difficile* infection and gastrointestinal tuberculosis (GITB). **Case Presentation:** A 21-year-old woman with IBD receiving biological therapy and corticosteroids presented with vomiting, abdominal pain, and bloody diarrhea. *C. difficile* toxin testing was positive, and vancomycin therapy was initiated. Given the persistence of symptoms, colonoscopy was performed and revealed segmental ulcerative colitis. Biopsy specimens and cultures confirmed the presence of *Mycobacterium tuberculosis*, and a four-drug antituberculous regimen was subsequently initiated. **Conclusion:** GITB and Crohn's disease share overlapping clinical and histological features, making diagnosis particularly challenging. Considering GITB in cases of refractory IBD, as well as the possibility of its coexistence with *C. difficile* infection, is essential for appropriate management and for preventing complications associated with the inappropriate use of immunosuppressive agents and biological therapies.

Keywords

Tuberculosis; gastrointestinal tuberculosis; Crohn's disease; stenosis.

INTRODUCTION

In emerging countries such as Colombia, the use of biologic therapy for inflammatory bowel disease (IBD) is increasing, although tuberculosis remains more prevalent. In patients who do not improve with initial treatment, it is essential to rule out infectious causes, particularly those associated with biologic or immunosuppressive therapy.

CASE DESCRIPTION

A 21-year-old woman with no known medical history presented in another city with a one-month history of generalized abdominal pain predominantly located in the right iliac fossa. An upper gastrointestinal endoscopy was performed, showing moderate congestive antral gastropathy and moderate bulboduodenitis, while colonoscopy revea-

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led multiple discontinuous ulcers with irregular borders in the cecum, ascending colon, and transverse colon, predominantly involving the cecum. Biopsies were obtained, demonstrating severe chronic active inflammation with focal architectural distortion, ulcer fragments, and granulation tissue, without evidence of granulomas, cryptitis, or microorganisms. The findings were negative for dysplasia or neoplastic involvement.

Treatment was initiated for probable IBD consistent with Crohn's disease (CD) with prednisolone 40 mg and oral mesalazine 500 mg every 24 hours for one month. However, due to lack of improvement, she sought care at another institution. Given the lack of response to first-line therapy, her case was discussed in a multidisciplinary medical board, and initiation of treatment with an integrin receptor antagonist (vedolizumab 300 mg intravenously) using an induction regimen was considered pending infectious disease approval. Serologic testing showed negative hepatitis B and C virus antibodies, negative hepatitis B surface antigen, nonreactive human immunodeficiency virus (HIV) testing, and a negative tuberculin skin test. Additionally, chest radiography demonstrated interstitial opacities and thickening of the peribronchovascular interstitium, without other relevant findings (**Figure 1**).



Figure 1. Chest radiograph obtained prior to initiation of vedolizumab treatment. Author's file.

The patient was admitted to the emergency department at our institution two months later due to worsening abdominal pain associated with bloody watery stools. Upon evaluation, an abdominal computed tomography scan was requested (**Figure 2**), which showed infectious inflammatory changes involving the ascending colon and cecum, along with an increased number of lymph nodes at the mesenteric root. Given the suspicion of gastrointestinal infection in the context of CD, a toxin assay was requested and returned positive. Inpatient management with oral vancomycin was initiated; however, one week later, the patient developed fever spikes and a significant increase in pain predominantly located in the right iliac fossa. A repeat colonoscopy was performed, revealing multiple segmental ulcers with fibrin in the cecum, ascending colon, and transverse colon (**Figure 3**), with involvement of the ileocecal valve and sparing of the ileum. Cultures and biopsies were therefore obtained, respectively.

Laboratory testing demonstrated the presence of *Mycobacterium tuberculosis* on the acid-fast bacillus (AFB) test (**Figure 4**), with subsequent isolation in culture. The patient was therefore evaluated by the Infectious Diseases Department, and quadruple therapy with isoniazid, rifampin, ethambutol, and pyrazinamide was initiated.

Due to lack of improvement with antituberculous therapy and signs of intestinal obstruction, the patient underwent surgery, during which 70 cm of small intestine were resected starting from the ileocecal valve, followed by side-to-side mechanical anastomosis. Pathology findings (**Figure 5**) revealed chronic inflammation with necrotizing granulomas, ruling out CD and confirming gastrointestinal tuberculosis (GITB).

DISCUSSION

GITB occurs in 20%–25% of patients with concomitant pulmonary tuberculosis. It is the sixth most common form of extrapulmonary tuberculosis after lymphatic, genitourinary, bone and joint, miliary, and meningeal tuberculosis[1]. It may affect any segment of the gastrointestinal tract; however, most cases occur at the jejunal and ileocecal levels, accounting for 64% of cases, which is explained by the high concentration of lymphoid aggregates and possibly by prolonged mucosal contact with the bacilli[1].

Transmission may occur following primary tuberculosis through hematogenous or lymphatic spread, or through swallowed sputum mediated by macrophages. Epithelial cells are also involved in direct infection through microfold cells (M cells) and with the assistance of dendritic cells[2]. Previously, GITB was associated with *Mycobacterium bovis* rather than *Mycobacterium tuberculosis*, probably due to the consumption of contaminated dairy products. Following the introduction of milk pasteurization and improvements in detection methods, the number of gastrointestinal tuberculosis infections decreased dramatically, as this process is generally sufficient to eliminate mycobacteria[3].



Figure 2. Contrast-enhanced abdominal and pelvic computed tomography showing concentric mural thickening of the cecum and distal ascending colon segment (7.5 mm) with hyperenhancement, surgical absence of the cecal appendix, and an increased number of lymph nodes at the mesenteric root. Author's file.

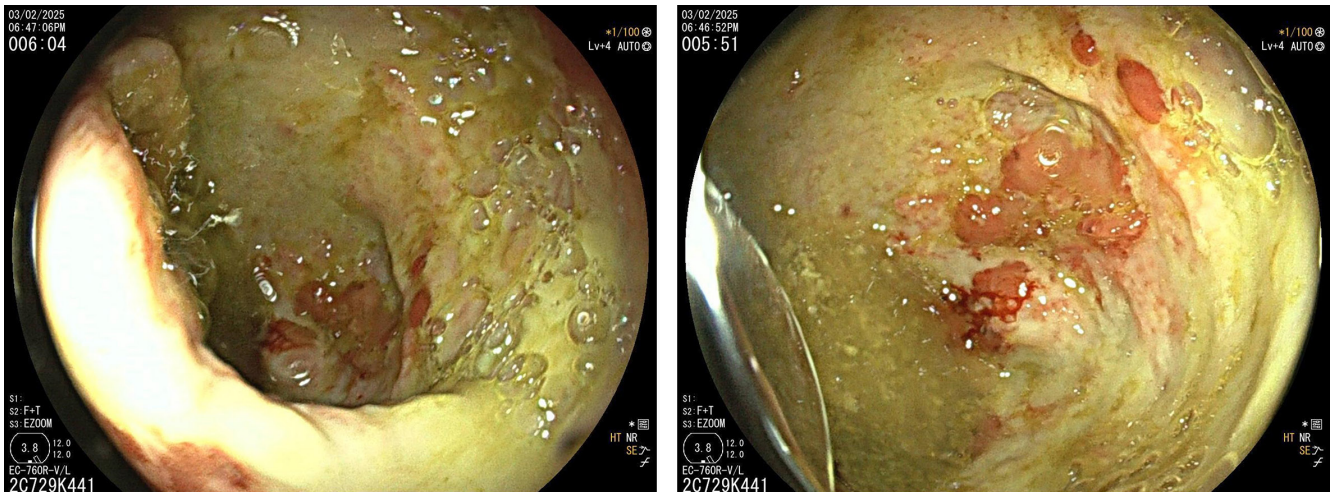


Figure 3. Segmental ulcerative colitis involving the cecum. Author's file.

In Colombia, case reports of gastrointestinal tuberculosis have been published in patients with HIV and in another patient with previous pulmonary tuberculosis[4,5].

The most common clinical manifestations of GITB are abdominal pain (30%–88%), fever (21%–73%), diarrhea (5%–47%), loss of appetite (30%–90%), weight loss (8%–80%), and rectal bleeding (5%–15%). Patients may also present with obstructive symptoms, pain in the right iliac fossa, or a palpable mass in the right iliac fossa[6].

Although it is considered a disease of developing countries, cases continue to be reported in developed countries due to migration and in immunocompromised individuals;

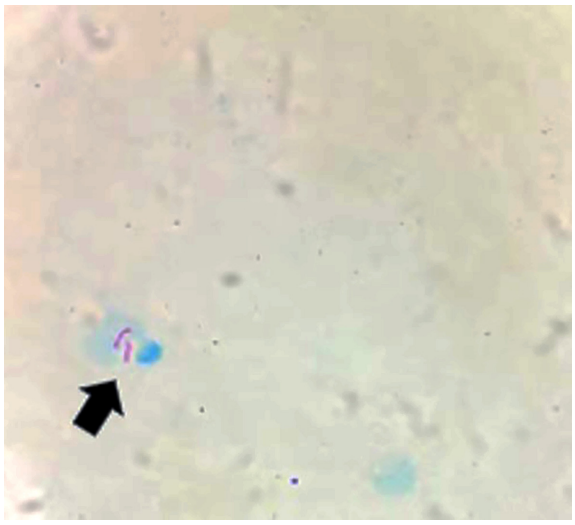


Figure 4. Isolation of *Mycobacterium tuberculosis* (AFB) in the cecum. Author's file.

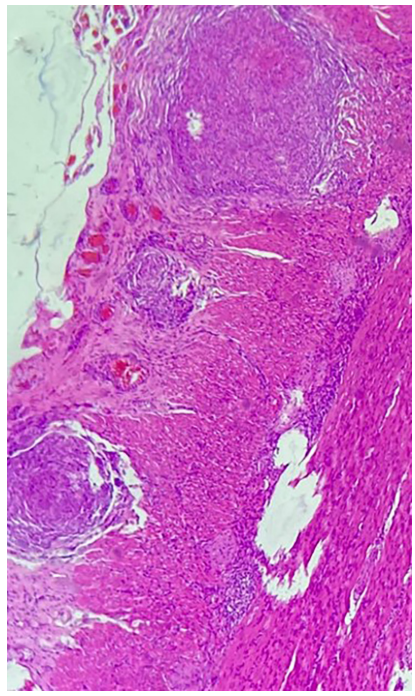


Figure 5. Ileal resection biopsy stained with hematoxylin and eosin showing necrotizing granulomatous chronic inflammation. Author's file.

for example, immunosuppression related to HIV and associated therapies, including anti-tumor necrosis factor (anti-TNF) agents and steroids[7]. GITB results in chronic intestinal inflammation that, like CD, is associated with granuloma formation[8]. Distinguishing gastrointestinal tuberculosis from CD is difficult because of the similarities in clinical manifestations and the low sensitivity of microbiological tools for the diagnosis of GITB. Seo H and colleagues showed that approximately 11% of patients with GITB were misdiagnosed with CD, whereas approximately 18% of patients with CD received a diagnosis of GITB[9]. These findings raise the following question: should greater caution be exercised when administering biologic therapy for suspected IBD in our setting?

In our case, the patient underwent chest radiography and tuberculin skin testing, taking into account the reported sensitivity and specificity of approximately 80% and 97%–98%, respectively[10]. However, several causes of false-negative results have been reported in a retrospective cohort, including HIV infection, chronic kidney disease, alcohol abuse, drug use, and age ≥65 years[11].

According to a meta-analysis that included 316 patients with gastrointestinal tuberculosis and 376 patients with CD, three histological features with high specificity for diagnosing GITB compared with CD were identified. These features included caseous necrosis, confluent granulomas, and ulcers lined by macrophages, with combined sensitivities of 21%, 38%, and 41%, and combined specificities of 100%, 99%, and 95%, respectively[12].

For the treatment of GITB, patients should receive a complete course of antituberculous therapy. Currently, a two-month treatment course is recommended for uncomplicated digestive tract tuberculosis. Longer treatment regimens should be avoided because they are associated with poor adherence and a higher risk of adverse effects from potentially toxic medications[13]. Surgical intervention is reserved for complications such as fibrosis, stenosis, and acute abdomen, or when diagnostic uncertainty persists[13].

For its part, *Clostridioides difficile* infection is the most common infectious cause of hospital-acquired diarrhea, generally preceded by the use of antibiotics such as fluoroquinolones, cephalosporins, broad-spectrum penicillins, and clindamycin[11,14]. Several studies estimate the incidence of *C. difficile* infection at between 2.8% and 3.73% in patients with ulcerative colitis (UC), and around 1% in CD, compared with rates below 0.5% in the general population. In addition, it is estimated that 8.2% of patients with UC and 1% of patients with CD are asymptomatic carriers of *C. difficile* without clinical manifestations of active infection[15]. This makes our clinical case even more noteworthy, given its rarity.

CONCLUSIONS

GITB and CD are chronic granulomatous diseases that primarily affect the terminal ileum. These conditions are

often confused in clinical practice because of the increasing emergence of IBD in developing countries. It is important to emphasize that the clinical, endoscopic, and imaging findings are not pathognomonic for either condition, creating a challenge for the treating physician. Considering the above, and given that this is an uncommon presentation reported in the literature, we describe this case with the aim of raising awareness within the medical community, since distinguishing GITB from CD is of immense importance because of the consequences that biologic therapy and immunosuppression may have in patients with an incorrect diagnosis.

Conflict of Interest

All authors declare that there are no conflicts of interest.

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None.

Use of Artificial Intelligence

No artificial intelligence tools were used in the development of this article.

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REFERENCES

1. Sharma MP, Bhatia V. Abdominal tuberculosis. Indian J Med Res. 2004;120(4):305-15.
2. Moule MG, Cirillo JD. *Mycobacterium tuberculosis* Dissemination Plays a Critical Role in Pathogenesis. Front Cell Infect Microbiol. 2020;10:65. https://doi.org/10.3389/fcimb.2020.00065
3. de la Rua-Domenech R. Human Mycobacterium bovis infection in the United Kingdom: Incidence, risks, control measures and review of the zoonotic aspects of bovine tuberculosis. Tuberculosis (Edinb). 2006;86(2):77-109. https://doi.org/10.1016/j.tube.2005.05.002
4. Gómez-Zuleta MA, Viveros-Carreño D, Cañón DP. Tuberculosis intestinal: reporte de caso y revisión de la literatura. Infectio. 2012;16(3):178-82. https://doi.org/10.1016/S0123-9392(12)70009-7
5. Martínez Montalvo CM, Botello A, Herrera Ortega LM, Leal Bernal SF, Guerrero Becerra ME. Tuberculosis ileocecal: A propósito de un caso. Rev Colomb Gastroenterol. 2021;36(Supl. 1):30-6. https://doi.org/10.22516/25007440.515
6. Jha DK, Pathiyil MM, Sharma V. Evidence-based approach to diagnosis and management of abdominal tuberculosis. Indian J Gastroenterol. 2023;42(1):17-31. https://doi.org/10.1007/s12664-023-01343-x
7. Kedia S, Das P, Madhusudhan KS, Dattagupta S, Sharma R, Sahni P, et al. Differentiating Crohn's disease from intestinal tuberculosis. World J Gastroenterol. 2019;25(4):418-432. https://doi.org/10.3748/wjg.v25.i4.418
8. Zhao Y, Xu M, Chen L, Liu Z, Sun X. Levels of TB-IGRA may help to differentiate between intestinal tuberculosis and Crohn's disease in patients with positive results. Ther Adv Gastroenterol. 2020;13:1756284820922003. https://doi.org/10.1177/1756284820922003
9. Seo H, Lee S, So H, Kim D, Kim SO, Soh JS, et al. Temporal trends in the misdiagnosis rates between Crohn's disease and intestinal tuberculosis. World J Gastroenterol. 2017;23(34):6306-14. https://doi.org/10.3748/wjg.v23.i34.6306
10. Mangione CM, Barry MJ, Nicholson WK, Cabana M, Chelmow D, Coker TR, et al. Screening for latent tuberculosis infection in adults: US Preventive Services Task Force recommendation statement. JAMA. 2023;329(17):1487-94. https://doi.org/10.1001/jama.2023.4899
11. Almeida Santos J, Duarte R, Nunes C. Tuberculin skin test and predictive host factors for false-negative results in patients with pulmonary and extrapulmonary tuberculosis. Clin Respir J. 2020;14(6):541-8. https://doi.org/10.1111/crj.13166
12. Du J, Ma YY, Xiang H, Li YM. Confluent granulomas and ulcers lined by epithelioid histiocytes: new ideal method for differentiation of intestinal tuberculosis and Crohn's disease? A meta-analysis. PLoS One. 2014;9(10):e103303. https://doi.org/10.1371/journal.pone.0103303
13. Tahiri M, Goh KL, Abbas Z, Epstein D, Min-Hu C, Mulder CJJ, et al. Digestive Tract Tuberculosis Guideline. J Clin Gastroenterol. 2023;57(7):643-50. https://doi.org/10.1097/MCG.0000000000001819
14. Brazier JS. The epidemiology and typing of Clostridium difficile. J Antimicrob Chemother. 1998;41(Suppl C):47-57. https://doi.org/10.1093/jac/41.suppl_3.47
15. Ramos-Martínez A, Ortiz-Balbuena J, Curto-García I, Asensio-Vegas Á, Martínez-Ruiz R, Múñez-Rubio E, et al. Risk factors for Clostridium difficile diarrhea in patients with inflammatory bowel disease. Rev Esp Enferm Dig. 2015;107(1):4-8.