

Case Report

Chronic abdominal pain as the sole symptom of colitis tuberculosis: A case report with endoscopic insights and histopathological findings

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Abstract

Tuberculous colitis remains a challenging clinical diagnosis because its presentation is often nonspecific and may be mistaken for inflammatory bowel disease (IBD) or colon cancer. A diagnosis may take time to be established, particularly when typical systemic symptoms of tuberculosis are not present. Herein, a case of tuberculous colitis presenting solely with long-standing abdominal pain is reported. The value of combining multiple diagnostic modalities, including endoscopy, biopsy, and GeneXpert testing, in establishing an accurate diagnosis is highlighted. A 25-year-old man was referred to the gastroenterology clinic with chronic abdominal pain that had persisted for 1 year, without fever, diarrhea, or weight loss. On physical examination, no remarkable findings were noted except for mild periumbilical tenderness. Routine laboratory investigations were found to be unremarkable. On colonoscopy, multiple ulcers and polypoid lesions with confluent granulomas in the cecum were identified. On histopathological examination, mucosal erosion, mild crypt distortion, and infiltration of polymorphonuclear cells were observed, favoring neutrophilic cryptitis. *Mycobacterium tuberculosis* was detected at a very low level by GeneXpert, whereas Ziehl-Neelsen staining was found to be negative. Based on the overall clinical, endoscopic, histopathological, and molecular findings, a diagnosis of tuberculous colitis was established. Chronic abdominal pain, although rare and nonspecific, may be observed as the sole presenting symptom of tuberculous colitis. Diagnostic yield may be increased through a multimodal approach incorporating endoscopy, histopathology, and molecular techniques such as GeneXpert when routine investigations are inconclusive. Tuberculous colitis should be considered an important differential diagnosis in patients with nonspecific gastrointestinal symptoms, particularly in tuberculosis-endemic areas.

Keywords: Tuberculous colitis, chronic abdominal pain, GeneXpert, endoscopy, granulomatous inflammation

Introduction

Tuberculosis remains one of the most lethal infectious diseases worldwide, with a significant global disease burden [1]. The World Health Organization (WHO) estimates that almost 10 million new cases of tuberculosis are reported every year, with 1.3 million deaths attributed to the



disease [2]. The majority of tuberculosis cases occur in the lungs, while extrapulmonary tuberculosis occurs in approximately 11–16% of all cases of tuberculosis [3]. Extrapulmonary tuberculosis may involve additional sites or organs, including the lymph nodes, bones, genitourinary tract, central nervous system, and gastrointestinal (GI) tract [4]. Of these manifestations, GI tuberculosis is likely the most difficult to diagnose, largely due to vague, nonspecific symptoms that can mimic other GI diseases such as inflammatory bowel disease (IBD) and colonic cancer [5]. In this case, a diagnosis of GI tuberculosis is mostly made in a belated manner, and the patients may suffer from serious complications such as stricture, perforation, or intestinal obstruction [6]. Moreover, the difficulty of diagnosis is enhanced by the fact that tuberculous colitis may not present typical systemic manifestations of tuberculosis infection, such as fever, night sweats, or loss of weight [7]. Thus, a full comprehension of the clinical presentation and variations of this ailment is especially critical in countries like Indonesia, which has been reported to be among the three top countries burdened by tuberculosis in the world [8, 9].

Tuberculous colitis is a term used to define chronic granulomatous infection of the colon by *Mycobacterium tuberculosis* [10]. It can be caused by hematogenous spread from an initial pulmonary focus, oral ingestion of infected sputum, or direct extension from adjacent infected structures [11]. The most common site of involvement is in the ileocecal region due to rich lymphatics, slow intestinal transit time, and high concentration of dense lymphoid tissue [12]. Tuberculous colitis has been reported to present with chronic diarrhea, fever, weight loss, or abdominal pain [13]. However, in some cases, chronic abdominal pain without systemic manifestations may be observed as the sole presentation, which may lead to delayed diagnosis or inappropriate management because it can mimic IBD or colonic neoplasm [14]. On endoscopy, ulcerative, polypoid, or nodular lesions may be identified, although these findings are nonspecific [15]. Therefore, histopathological examination, together with molecular analyses such as GeneXpert, is required to establish a more definitive diagnosis [16].

This case report was primarily intended to describe the clinical features and diagnostic measures used in a patient with tuberculous colitis who presented solely with chronic abdominal pain and no constitutional symptoms. It also highlighted tuberculous colitis as an important differential diagnosis in patients with nonspecific gastrointestinal symptoms. In addition, the need for a multimodal diagnostic approach incorporating endoscopy, histopathology, and GeneXpert was illustrated in establishing the diagnosis. Finally, the importance of clinician awareness in high-burden settings was emphasized to facilitate earlier diagnosis and treatment, reduce diagnostic delay, and lessen the overall burden of disease in affected patients.

Case

A 25-year-old man with no prior medical history presented to the emergency department of Saiful Anwar Hospital, Malang, Indonesia, with chronic abdominal pain persisting for one year prior to admission. The pain was localized to the periumbilical region and was not associated with meals. The patient denied diarrhea, constipation, nausea, vomiting, fever, night sweats, or significant weight loss, although the patient reported a mild decrease in appetite. There was a history of household contact with tuberculosis. Physical examination revealed mild periumbilical tenderness without palpable masses or organomegaly.

Routine laboratory tests were unremarkable. A colonoscopy was performed in order to exclude any possible intraluminal pathology. Colonoscopy findings in the ascending colon revealed multiple ulcerations with polypoid lesions and confluent granulomas covered with pus surrounding the cecum (**Figure 1A**). Within the cecal segment, an irregular polypoid mass surrounded by edematous and erythematous mucosa was observed (**Figure 1B**). The cecum also had small polypoid nodules with erythematous mucosa and focal exudates (**Figure 1C**). This was suggestive of a chronic granulomatous inflammatory process, and an infectious cause was suspected, along with GI tuberculosis.

Histopathological examination of biopsy specimens revealed partial mucosal erosion and mild crypt architectural distortion at 10× magnification (**Figure 2A**). At higher magnification (40×), polymorphonuclear cell infiltrations were observed within the epithelium and crypts, consistent with neutrophilic cryptitis (**Figure 2B**). The lamina propria demonstrated increased

infiltration of neutrophils and lymphoplasmacytic cells (**Figure 2C**). No well-formed epithelioid granulomas, Langhans giant cells, or caseating necrosis were identified. These findings were consistent with chronic active colitis, and differentiation from Crohn's disease remained necessary. Ziehl–Neelsen staining for acid-fast bacilli was negative; however, GeneXpert testing was positive for *M. tuberculosis* at a “very low” level. Based on the combined clinical, endoscopic, histopathological, and molecular findings, a diagnosis of intestinal tuberculosis was considered most likely.



Figure 1. Endoscopic findings in the colon. (A) The ascending colon shows multiple ulcerations and polypoid lesions with pus-covered confluent granulomas near the cecum. (B) The cecal segment demonstrates an irregular polypoid mass with adjacent mucosal edema and erythema. (C) The cecum displays smooth polypoid nodules surrounded by erythematous mucosa and focal exudate.

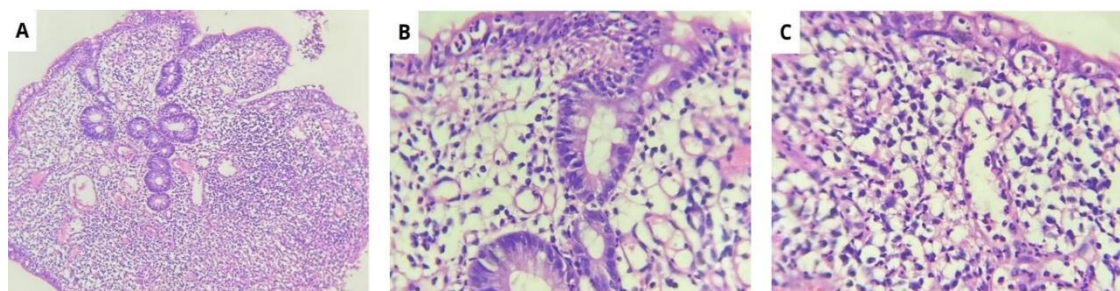


Figure 2. Histopathological findings of the colonic mucosa (Hematoxylin and Eosin staining). (A) At 10× magnification, the intestinal mucosa appears partially eroded with mildly distorted crypt architecture forming branching structures. (B) At 40× magnification, infiltration of polymorphonuclear cells is observed within the epithelium and crypts, consistent with neutrophilic cryptitis. (C) At 40× magnification, the lamina propria shows an increased number of neutrophilic and lymphoplasmacytic inflammatory cells.

The patient was initiated on standard anti-tuberculosis therapy consisting of a 2-month intensive phase with isoniazid, rifampicin, pyrazinamide, and ethambutol (2HRZE), followed by a continuation phase of isoniazid and rifampicin (4HR). The patient reported clinical improvement in abdominal pain and appetite during a one-year follow-up.

Discussion

In this case report, a patient with tuberculous colitis was reported to have presented atypically with chronic abdominal pain and no constitutional symptoms, making the diagnosis challenging. In this patient, an atypical presentation with nonspecific clinical findings was observed. Chronic abdominal pain was presented as the main symptom, while ulcerative and polypoid lesions of the colon with confluent granulomas were identified on endoscopy, suggesting an underlying granulomatous inflammatory process. These findings are consistent with early reports of tuberculous colitis appearing clinically and endoscopically like Crohn's disease, often in the terminal colon, delaying clinical diagnosis [17]. Historically, similar study of colitis has shown that the primary differential criteria for the two diseases is through histopathology and finding *M. tuberculosis* using molecular testing or tissue cultures [18]. One of the interesting differences

in the present case was the lack of typical caseous necrosis in the pathology, likely due to low bacterial burden (paucibacillary) [19]. Therefore, our findings support that the combined endoscopic and molecular findings form a solid basis for the diagnosis of tuberculous colitis, even without histopathology that may be considered classic.

Differentiating intestinal tuberculosis from Crohn's disease remains challenging due to overlapping clinical, endoscopic, and histopathological features. In this case, several findings favored intestinal tuberculosis. The involvement of the ileocecal region with ulcerative and polypoid lesions is more typical of intestinal tuberculosis, whereas Crohn's disease more commonly presents with longitudinal ulcers and a cobblestone appearance. Histopathologically, although classic caseating granulomas were not observed, the presence of granulomatous inflammation combined with a positive GeneXpert result supported tuberculosis. In contrast, Crohn's disease typically demonstrates small, poorly formed non-caseating granulomas without microbiological confirmation. Additionally, the patient's epidemiological background and history of tuberculosis exposure further supported the diagnosis.

Diagnostic examination in this case presented a discordance between the Ziehl–Neelsen stain and GeneXpert molecular test. Ziehl–Neelsen stain did not reveal acid-fast bacilli; however, GeneXpert detected *M. tuberculosis* DNA with a "MTB very low" positive report. This correlates with the evidence indicating that GeneXpert is more sensitive in the detection of extrapulmonary tuberculosis compared to conventional methods [20]. GeneXpert also be able to detect bacterial DNA fragments even at low concentrations, whereas Ziehl–Neelsen staining requires a sufficient bacillary load to allow microscopic detection [21]. Pathophysiologically, this is due to the DNA amplification capability of the GeneXpert system with a detectable ability in paucibacillary conditions [22]. Consequently, these findings suggest that GeneXpert could provide higher sensitivity in paucibacillary extrapulmonary tuberculosis; however, results should be interpreted cautiously [23]. Nevertheless, GeneXpert has certain limitations, as it may detect DNA from non-viable bacilli, potentially leading to false-positive results. Therefore, results should be interpreted in conjunction with clinical, endoscopic, and histopathological findings. Mycobacterial culture, although considered the gold standard, was not performed due to limited resources and longer turnaround time.

Tuberculous colitis arises as a result of *M. tuberculosis* infection of the GI tract, primarily the ileocecal region, being the major predilection area [5]. Infection may occur by hematogenous dissemination from a primary pulmonary focus or by ingestion of sputum containing tubercle bacilli [24]. Upon reaching the intestinal mucosa, the bacilli settle and cause activation of cell-mediated immunity involving T lymphocytes, macrophages, and dendritic cells [25]. Activation of cells results in the production of cytokines such as interferon- γ and tumor necrosis factor-alpha (TNF- α), which participate in the formation of epithelioid granulomas as a host reaction to immunoprotection against the bacilli [26,27]. Granulomas serve to contain the infection, but chronic inflammation that is repeated can lead to tissue damage and, in some cases, central caseous necrosis of the granuloma [28]. Non-caseating granulomas are more commonly observed in paucibacillary disease as a result of a relatively well-balanced host-pathogen immune response [29]. This theory explains tuberculous colitis as an expression of generalized *M. tuberculosis* infection, perpetuated by a multifactorial interaction of bacterial virulence, site of colonization, and host immune response.

There are some clinical implications in this case report. Firstly, it highlights the importance of adding tuberculous colitis to the differential diagnosis in young patients with chronic abdominal pain and ulcerative-polypoid lesions of the colon, particularly in high-prevalence regions for tuberculosis. Second, a combination of endoscopic diagnosis and molecular testing, such as GeneXpert, may provide enhanced diagnostic sensitivity compared to the conventional methods. Third, in addition to accelerating diagnosis, the utilization of GeneXpert also enhances early rifampicin resistance detection. In addition, the findings are supportive of a multi-modal approach—clinical, endoscopic, histopathological, and molecular assessment—to arrive at a diagnosis of tuberculous colitis.

A number of limitations are also present in this case report. It is a single-case report, so the generalizability of findings is still limited. Diagnosis of tuberculosis was primarily based on GeneXpert alone and not necessarily with bacterial culture as the gold standard confirmation.

The lab resources and facilities available in endemic regions may impact the completeness of the testing. The absence of classic caseating granulomas represents a limitation; however, this may occur in paucibacillary disease and does not exclude intestinal tuberculosis. These findings still remain clinically relevant, highlighting the effectiveness of GeneXpert as a sensitive diagnostic tool for tuberculous colitis in cases with few symptoms and non-classic histopathology.

Conclusion

Tuberculous colitis may present with chronic abdominal pain as its sole manifestation, despite the symptom being subtle and nonspecific. Because its clinical presentation may overlap with that of other gastrointestinal diseases, the diagnosis should be established through a combination of endoscopic findings, histopathological examination, and molecular testing. Tuberculous colitis should therefore be considered as an important differential diagnosis in patients with relevant risk factors or those from tuberculosis-endemic areas, particularly when chronic granulomatous inflammation is suspected. Early recognition and appropriate management may be achieved through a multimodal diagnostic approach, thereby reducing delays in treatment and preventing complications.

Ethics approval

Written informed consent was obtained from the patient for the publication of this case report. The patient agreed that the clinical details and supporting materials may be published.

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None.

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

This study utilized artificial intelligence (AI) tool, Quillbot, for language refinement, including improving grammar, sentence structure, and readability of the manuscript. We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results. The final decisions and interpretations presented in this article were made solely by the authors.

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