Section: Medical Gastroenterology

Case Report

ISOLATED PYLORIC STENOSIS DUE TO GASTRIC TUBERCULOSIS IN AN ADULT FEMALE

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ABSTRACT

Tuberculosis remains a significant global health concern with diverse clinical manifestations. Gastric tuberculosis is an exceedingly rare form of extrapulmonary tuberculosis, often presenting diagnostic challenges due to nonspecific symptoms and overlap with other gastric pathologies. We report a case of a 37-year-old female presenting with progressive postprandial vomiting and significant weight loss over three years. Diagnostic evaluations revealed isolated pyloric stenosis caused by granulomatous inflammation with Langhans giant cells on biopsy. Despite negative acid-fast bacilli staining and molecular tests, a clinical diagnosis of gastric tuberculosis was made based on histopathological findings. The patient was initiated on anti-tuberculosis therapy, resulting in remarkable symptomatic improvement and substantial weight gain. This case underscores the importance of considering gastric tuberculosis in the differential diagnosis of gastric outlet obstruction, even in the absence of pulmonary involvement or positive laboratory tests. **Categories:** Gastroenterology, Infectious Disease and Surgery.

INTRODUCTION

Tuberculosis (TB) continues to be one of the leading infectious diseases worldwide, with an estimated 10 million new cases and 1.4 million deaths reported in 2019.[1] While pulmonary tuberculosis is the most common manifestation, accounting for approximately 85% of cases, extrapulmonary tuberculosis constitutes about 15% and can involve virtually any organ system.[2] Gastrointestinal tuberculosis is the sixth most common form of extrapulmonary TB, representing 3% of all TB cases.[3]

Within the gastrointestinal tract, the ileocecal region is most frequently affected due to the abundance of lymphoid tissue and prolonged transit time, which facilitates the localization of Mycobacterium tuberculosis.[4] Gastric tuberculosis is exceedingly rare, accounting for less than 2% of all gastrointestinal TB cases.[5] The stomach's hostile acidic environment, paucity of lymphoid tissue, and rapid transit time are thought to protect against mycobacterial infection.

Gastric tuberculosis often presents with nonspecific symptoms such as epigastric pain, vomiting, weight loss, and upper gastrointestinal bleeding.[5] These symptoms can mimic more common gastric pathologies like peptic ulcer disease, gastritis, or gastric malignancies, leading to diagnostic delays.[6-8] Risk factors for gastric TB include immunosuppression (e.g., HIV infection), malnutrition, chronic debilitating diseases, and previous or concurrent pulmonary TB.[6]

Diagnosing gastric TB is challenging due to its rarity and nonspecific presentation. It requires a high index of suspicion, especially in endemic areas. Endoscopic biopsies with histopathological examination remain the cornerstone of diagnosis, often supported by imaging studies and microbiological tests.[7] Histologically, the presence of granulomatous inflammation with Langhans giant cells is suggestive but not definitive for TB.[8] Microbiological confirmation through acid-fast bacilli (AFB) staining, culture, or molecular methods enhances diagnostic accuracy but may not always be positive due to the paucibacillary nature of the disease.[9]

We present a rare case of isolated pyloric stenosis due to gastric tuberculosis in an immunocompetent adult female without pulmonary involvement. This case highlights the importance of considering gastric TB in the differential diagnosis of gastric outlet obstruction and discusses the diagnostic challenges and management strategies.

CASE PRESENTATION

A 37-year-old female presented to our outpatient department in April 2024 with a chief complaint of vomiting after meals for the past three years. The vomiting was insidious in onset and had progressively worsened over the preceding six months. It occurred 2-3 hours postprandially, was

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more pronounced after solid meals than liquids, and was non-bilious and non-projectile. She denied associated nausea, dysphagia, hematemesis, abdominal pain, or epigastric burning sensation. There was no history of loss of appetite; however, she reported significant unintentional weight loss of 28 kilograms over three years. She denied any abdominal distension, bowel habit changes, fever, night sweats, or fatigue.

Her past medical history was unremarkable. There was no history of tuberculosis exposure, contact with individuals known to have TB, or symptoms suggestive of sarcoidosis or other granulomatous diseases. She was not on any immunosuppressive therapy and tested negative for HIV. She had been prescribed ondansetron by her primary care physician, which provided minimal relief.

On physical examination, she appeared lean built and undernourished. Vital signs were within normal limits. Abdominal examination revealed mild epigastric fullness without tenderness, palpable masses, or organomegaly. Bowel sounds were normal. Other systemic examinations were unremarkable.

Laboratory investigations showed mild anemia with a hemoglobin level of 9.8 g/dL and normal leukocyte and platelet counts. Renal and liver function tests, serum electrolytes, and blood glucose levels were within normal limits.

Table 1: Laboratory parameters

Parameters	Values	Reference values
Haemoglobin	9.8 g/dL	13.0-17.5 g/dl
Haematocrit	28.1 %	38.3 -48.6%
TLC	4.99 10^3/uL	4,000-11,000^3/uL
Platelet count	238 10^3/uL	150- 450^3/uL
Sodium	135 mmo/L	135-140 mmo/L
Potassium	3.7 mmo/L	3.5- 5.2 mmol/L
Urea	20.0 mg/dL	8–24 mg/dL
Creatinine	0.95 mg/dL	0.7–1.2 mg/dL
Total bilirubin	0.65 mg/dL	0.3- 1.2 mg/dL
Direct bilirubin	0.25 mg/dL	<0.3 mg/dL
AST	19.0 U/L	8-35 U/L
ALT	12.0 U/L	7-35 U/L
ALP	86.0 U/L	44-120 U/L
Total protein	6.0 g/dL	6.0- 8.3 g/dL
Albumin	3.3 g/dL	3.4- 5.4 g/dL

An upper gastrointestinal (UGI) endoscopy revealed a 2-centimeter stricture at the pyloric canal causing significant luminal narrowing [Figure 01]. The mucosa appeared edematous with mild erythema but no ulceration or mass lesions. A rapid urease test for Helicobacter pylori was negative. In the same session, balloon dilatation using a controlled radial expansion (CRE) balloon was performed up to 6 millimetres to alleviate the obstruction.

Contrast-enhanced computed tomography (CECT) of the abdomen demonstrated circumferential thickening of the pyloric region with luminal narrowing and a few sub-centimetric lymph nodes in the perigastric region [Figure 02]. There was no evidence of lesions in the lungs, liver, spleen, or other abdominal organs. A barium meal study showed a narrowed pyloric canal measuring 1.8 centimetres in length with a luminal diameter of 3.8 millimetres, consistent with pyloric stenosis.

Multiple biopsies were obtained from the pyloric region during endoscopy. Histopathological examination revealed granulomatous inflammation composed of epithelioid cells, lymphocytes, plasma cells, and Langhans-type giant cells [Figure 03]. No caseating necrosis was observed. Ziehl-Neelsen staining for AFB was negative. The findings suggested a granulomatous process, raising the possibility of tuberculosis or sarcoidosis.

Given the histological findings, a detailed history and further investigations were undertaken to differentiate between possible causes. The patient had no respiratory symptoms, skin lesions, ocular involvement, or arthralgias suggestive of sarcoidosis. A chest radiograph was normal, showing no pulmonary infiltrates or mediastinal lymphadenopathy. High Resolution Computed Tomography (HRCT) thorax was also normal. The Mantoux test (tuberculin skin test) was negative, and interferon-gamma release assay (IGRA) could not be performed due to financial constraints.

Two months later, the patient returned with worsening symptoms of gastric outlet obstruction, including increased frequency of vomiting and inability to tolerate oral intake. A repeat UGI endoscopy showed persistent pyloric stenosis with no significant improvement despite prior dilatation. Additional biopsies were taken, and balloon dilatation was performed up to 7 millimeters. Histopathology again demonstrated granulomatous inflammation with Langhans giant cells. AFB staining, cartridge-based nucleic acid amplification test (CBNAAT), and mycobacteria growth indicator tube (MGIT) cultures were all negative.

Considering the clinical presentation, histopathological evidence, high prevalence of tuberculosis in the region, and exclusion of other granulomatous diseases, a presumptive diagnosis of gastric tuberculosis causing pyloric stenosis was made. The patient was initiated on anti-tuberculosis therapy (ATT) comprising rifampicin (600 mg daily),

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isoniazid (300 mg daily), pyrazinamide (1500 mg daily), and ethambutol (1200 mg daily) for the intensive phase of two months, followed by rifampicin and isoniazid for the continuation phase.
Over the next four months, the patient showed remarkable clinical improvement. Her vomiting resolved completely, and she was able to tolerate a regular diet. She gained 21 kilograms, returning to her baseline weight. No further dilatation sessions were required Follow-up endoscopy were not done as the patient was not willing.

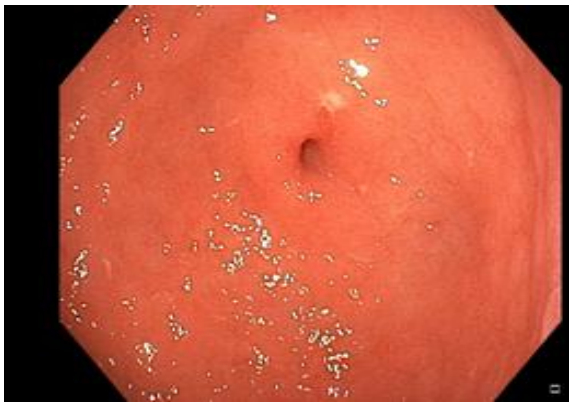

Figure 1: Pyloric stenosis before dilatation

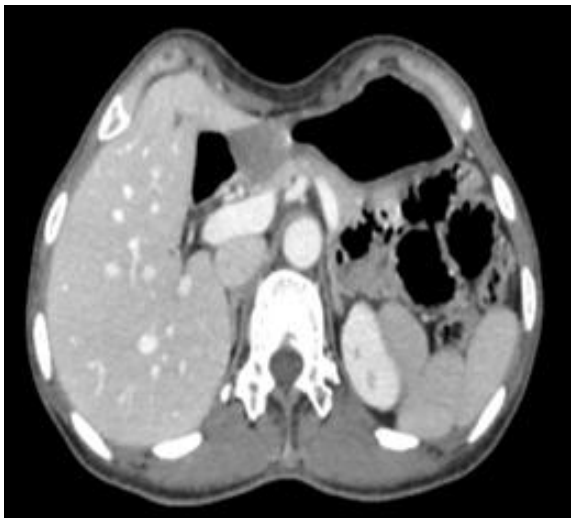

Figure 2: Image showing pyloric stenosis

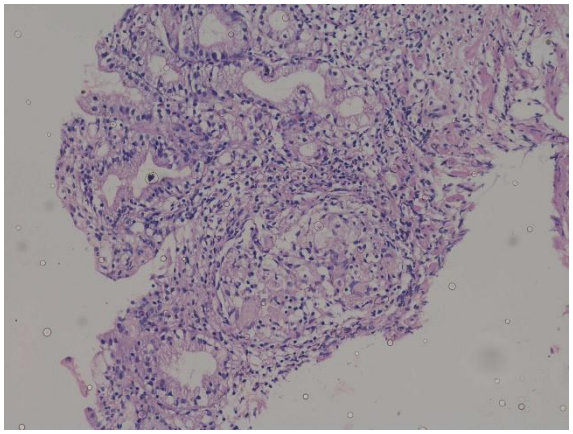

Figure 3: Histopathology showing caseating granuloma

DISCUSSION

Gastric tuberculosis is an uncommon entity, even in regions where tuberculosis is endemic. The rarity of gastric involvement is attributed to several factors: the acidic gastric environment is hostile to Mycobacterium tuberculosis, there is a relative paucity of lymphoid tissue in the stomach compared to other parts of the gastrointestinal tract, and the rapid transit time through the stomach reduces the contact time between the mucosa and the pathogen.
When gastric TB does occur, it often involves the distal stomach and pyloric region due to relative stasis and increased lymphoid tissue in these areas.[10]
The clinical presentation is nonspecific and can mimic other gastric diseases. Common symptoms include epigastric pain, vomiting, weight loss, anorexia, and occasionally upper gastrointestinal bleeding.[5] Our patient's primary symptoms of postprandial vomiting and significant weight loss over an extended period pointed towards a mechanical obstruction at the gastric outlet.
Diagnosing gastric tuberculosis is challenging. Endoscopic findings are variable and can include ulcers, nodules, mass lesions, or strictures.[11] In our case, endoscopy revealed a pyloric stricture without ulceration or masses. Imaging studies such as CECT and barium studies can provide supportive evidence but are not diagnostic.[6]
Histopathological examination of biopsy specimens remains the gold standard for diagnosis. The presence of granulomatous inflammation with Langhans giant cells is suggestive of TB but not definitive, as other conditions like sarcoidosis, Crohn's disease, and fungal infections can present similarly.[5,8] The absence of caseating necrosis does not exclude TB, especially in early or localized disease.[12]
Microbiological confirmation through AFB staining, culture, or molecular methods increases diagnostic accuracy but may not always yield positive results due to the paucibacillary nature of gastric TB.[9] In our case, repeated AFB staining and molecular tests were negative. Negative results do not rule out TB, particularly in endemic areas, and a trial of ATT may be justified based on clinical and histopathological findings.[13]
Differential diagnoses such as sarcoidosis were considered but deemed less likely due to the absence of systemic features, normal chest imaging including X-ray and CT.[14] Crohn's disease typically involves the terminal ileum and colon, with transmural inflammation and skip lesions, which were not observed in this patient.[15] Fungal infections were unlikely due to the patient's immunocompetent status and lack of exposure history.
The decision to initiate ATT was supported by the patient's clinical presentation, histopathological findings, exclusion of other granulomatous diseases, and the high prevalence of TB in the region. The significant clinical improvement and weight gain

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following ATT further supported the diagnosis of gastric tuberculosis.

Early initiation of ATT is crucial in managing gastric TB to prevent complications such as obstruction, perforation, bleeding, fistula formation, or progression to malignancy. Surgical intervention is reserved for cases with complications or when there is no response to medical therapy.[16]

This case highlights the importance of considering gastric tuberculosis in the differential diagnosis of gastric outlet obstruction, especially in endemic areas. Clinicians should maintain a high index of suspicion, even when microbiological tests are inconclusive, and rely on a combination of clinical judgment, histopathological findings, and epidemiological context.

CONCLUSION

Isolated pyloric stenosis resulting from gastric tuberculosis is exceptionally rare and can pose a formidable diagnostic challenge. In this case, the patient's predominant symptoms of gastric outlet obstruction—coupled with ambiguous endoscopic and radiological findings—demonstrated that classic features of gastrointestinal TB may not always manifest. The gradual onset and nonspecific clinical signs easily mimic more common etiologies, such as peptic ulcer disease, malignancies, or other granulomatous conditions. As a result, the risk of delayed or incorrect diagnosis remains high, emphasizing the necessity for heightened clinical vigilance, especially in regions where TB is endemic or when a patient's immune status is compromised.

This case also highlights how diligent histopathological and microbiological assessments can definitively establish the diagnosis. While suggestive radiologic and endoscopic changes can guide the suspicion, obtaining targeted biopsies is pivotal for confirming gastric tuberculosis. A low threshold for biopsy and histological workup is key, particularly when dealing with unexplained pyloric obstruction or refractory ulcerative lesions. Once diagnosed, timely initiation of anti-tuberculosis therapy is paramount to control disease progression and promote reversal of stenosis. In certain advanced cases, intervention to relieve the obstruction—such as endoscopic balloon dilation—may be required to manage symptoms until adequate treatment response is achieved.

Moreover, this patient's course illuminates the importance of multidisciplinary collaboration among gastroenterologists, radiologists, pathologists, and infectious disease specialists for both prompt diagnosis and integrated management. Identifying predictors of therapy failure or delayed response—such as persistent gastric outlet obstruction, weight loss, or ongoing systemic symptoms—can help clinicians decide if more aggressive or tailored therapeutic strategies are warranted. Lastly, recognizing the potential for isolated gastric TB to occur outside the typical pulmonary or disseminated patterns broadens our understanding of tuberculosis' protean presentations. Sharing such experiences in the literature not only enriches our collective knowledge but also underscores the crucial role of early suspicion and comprehensive evaluation in ensuring favorable outcomes for this rare but significant clinical entity.

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