91171_Auto_Edited.docx

WORD COUNT 1968
TIME SUBMITTED 16-FEB-2024 08:04AM
PAPER ID 106929504

Primary gastroduodenal tuberculosis presenting as gastric outlet obstruction: A Case Report and Literature Review

Abstract

BACKGROUND

Mycobacterium tuberculosis is the causative agent of tuberculosis (TB), a chronic granulomatous illness. This disease is prevalent in low-income countries, posing a significant global health challenge. Gastrointestinal tuberculosis (GI–TB) is one of the three forms. The disease can mimic other intra-abdominal conditions, leading to delayed diagnosis owing to the absence of specific symptoms. While gastric outlet obstruction (GOO) remains a frequent complication, its incidence has declined with the advent of proton pump inhibitors and *Helicobacter pylori* eradication therapy. Gastroduodenal TB can cause upper gastrointestinal hemorrhage, obstruction, and malignancy-like tumors.

CASE SUMMARY

A 23-year-old male presented with recurrent epigastric pain, distension, nausea, vomiting, and weight loss, prompting a referral to a gastroenterologist clinic. Endoscopic examination revealed distorted gastric mucosa and signs of chronic inflammation. However, treatment was interrupted, possibly owing to vomiting or comorbidities such as human immunodeficiency virus (HIV) infection or diabetes. Subsequent surgical intervention revealed a dilated stomach and diffuse thickening of the duodenal wall. Resection revealed gastric wall effacement with tuberculosis.

CONCLUSION

Primary gastric tuberculosis is rare, frequently leading to GOO. Given its rarity, suspicions should be promptly raised when encountering relevant symptoms, often requiring surgical intervention for diagnosis and treatment.

Keywords: Tuberculosis, Gastrointestinal tuberculosis, Gastric outlet obstruction, Gastroduodenal tuberculosis, Case report.

Core Tip: Primary gastric tuberculosis is rare, often causing obstruction of the gastric outlet. In cases of prevalence, suspicions should be raised and surgery is often required for diagnosis and treatment.

INTRODUCTION

Tuberculosis (TB) is a chronic granulomatous disease caused by aerobic bacteria *Mycobacterium tuberculosis* [1,3].

Its global incidence is high in low-income nations, posing a significant health concern. Abdominal TB ranks as the third most common extrapulmonary form [2].

Gastrointestinal tuberculosis (GI–TB) is one of the three forms of abdominal TB. The other types include visceral, peritoneal, and tuberculous lymphadenopathy [4].

Mycobacterium tuberculosis enters the gastrointestinal system via hematogenous spread, ingestion of contaminated sputum, or direct dissemination from infected adjacent lymph nodes and fallopian tubes [5].

Diagnosing abdominal TB is often hindered by the absence of distinct clinical signs and symptoms, leading to delayed identification because the disease can mimic other intra-abdominal pathologies [6].

The most common causes of gastric outlet obstruction (GOO) are peptic ulcer disease (PUD) and gastric cancer. However, the frequency of GOO owing to PUD has declined since the development of proton pump inhibitors and Helicobacter eradication therapy.

Gastric bezoars, pancreatitis-related fluid collection, caustic ingestion, massive gastric polyps, Crohn's disease, and complications following gastric surgery contribute to GOO [7].

Gastroduodenal TB presents with various manifestations, including upper gastrointestinal hemorrhage, obstruction, and gastric or periampullary tumors suggestive of malignancy [8].

Similar to our patient, most individuals with gastroduodenal TB exhibit signs of GOO, often attributed not only to intrinsic duodenal lesions but also to extrinsic compression by tuberculous lymph nodes [9].

Here, we present a case of gastroduodenal TB-associated GOO in a previously healthy 23-year-old patient who exhibited no signs of pulmonary TB.

CASE PRESENTATION

Chief complaints

A 23-year-old male patient came to the Department of Surgery with recurrent epigastric pain, vomiting, and weight loss that had persisted for a year despite the patient having no prior history of chronic illness.

History of present illness

The patient had a year-long history of recurrent epigastric pain, distension, low appetite, nausea, vomiting, and weight loss. He did not mention any fever, jaundice, or changes in bowel habits. Hemostasis and cough were absent, and there were no notable findings from the assessment of the other systems.

History of past illness

The patient had experienced similar symptoms during a prior stay in South Africa eight months earlier, during which an endoscopy revealed distortions and chronic inflammatory changes in the distal gastric mucosa without evidence of *H. pylori* infection. Subsequently, anti-tubercular medication was planned, but the patient ceased treatment after one week owing to intractable vomiting.

Personal and family history

There was no family history of similar conditions or TB,

Physical examination

The patient presented with normal vital signs, absence of pallor, and no cervical, axillary, or inguinal lymphadenopathy.

Clear chest examination findings were noted. Upon abdominal examination, the abdomen was soft and non-tender with mild distension, and no masses, organomegaly, or ascites were noted.

Laboratory examinations

CRP 200 mg/L

Stool H Pylori- Negative

No abnormalities were found in routine blood and urine analyses.

Imaging examinations

An abdominal contrast-enhanced CT scan revealed gastric distension and diffuse wall thickening in the first and second parts of the duodenum. A thoracic CT scan showed no evidence of prior TB sequelae [Figure 1]. Upper endoscopy was subsequently performed, revealing longitudinal hyperemic mucosal streaks in the gastric corpus and antrum, along with multiple glandular nodules and severely inflamed mucosa in the post-pyloric duodenum. Additionally, obstruction was observed at the junction of the first and second parts of the duodenum.

A gastric biopsy revealed chronic gastritis with severe inflammation, absence of metaplasia and dysplasia, and a positive result for *H. pylori*.

The patient was admitted for optimization of preoperative fitness in preparation for surgery.

FINAL DIAGNOSIS

Primary Gastro-duodenal Tuberculosis

TREATMENT

A decision was made to relieve the obstruction surgically. Intraoperatively, the following findings were noted: dilation of the stomach and diffuse wall thickening of the duodenum, along with multiple enlarged mesenteric lymph nodes (located at the lesser curvature, transverse colon mesentery, small bowel mesentery, paraduodenal area, and interaortocaval region). Subsequently, antrectomy and Roux-en-Y reconstruction with gastrojejunostomy were performed, along with lymph node dissection [Figure 2].

The pathology report from the resected distal part of the antrum and lymph nodes revealed findings indicative of gastric TB, characterized by effacement of the gastric wall architecture and numerous caseating granulomatous inflammation. Specifically, the distal margins displayed positivity for granulomatous inflammation. Additionally, 12 lymph nodes exhibited suppurative and non-suppurative granulomatous inflammation, further supporting the TB diagnosis. PAS stains yielded negative results for fungal hyphae. These findings were consistent with gastroduodenal TB [Figure 3]. Notably, cytology analysis for AFB staining and Gene Xpert testing were not pursued owing to the absence of peritoneal ascites detected during the operation.

OUTCOME AND FOLLOW-UP

The postoperative course was uneventful, and the patient was discharged from the hospital following successful tolerance of full enteral feeding. Plans were made for follow-up visits at the polyclinic and referral to a TB center to initiate anti-tubercular treatment. Currently, the patient is adhering to the anti-tubercular medication regimen well and has experienced no adverse effects.

DISCUSSION

GI–TB most frequently affects the ileocecal area, with the colon and jejunum following closely after. A total of 64% of GI–TB cases are caused by jejunal and ileocecal TB [10]. However, the esophagus, stomach, and duodenum are rarely affected. The duodenum and gastric are the primary sites of involvement for gastric TB.

Primary and isolated stomach TB is exceptionally rare, documented only in very few cases in the literature. A total of 0.4–2% of all GI–TB cases are associated with primary gastric TB, while 2–2.5% are associated with primary duodenal TB [11].

Several factors contribute to the low frequency of gastric TB, including the bactericidal properties of gastric acid, the presence of thick and intact gastric mucosa, and the absence of lymphoid structures in the gastric mucosa [10]. Treatment with H2 blockers increases the likelihood of involvement of the lesser curvature and pylorus in gastric TB, often presenting as ulcerating lesions [4]. Manifestations such as pyloric stenosis, miliary tubercles, and hypertrophic variations may also occur [10]. Gastric TB is frequently associated with TB lymphadenitis, as observed in cases where peripancreatic lymph nodes are involved, alongside visible ulcerative lesions in the prepyloric region.

The third part of the duodenum is commonly affected in cases of primary duodenal TB.

Both intrinsic and extrinsic factors can contribute to duodenal involvement [12]. Extrinsic involvement is most prevalent and is often related to lymphadenopathy in the duodenum's C-loop. Intrinsic variations may manifest as ulcerative, hypertrophic, or ulcer hypertrophic forms, potentially leading to fistula or stricture formation.

Various modalities contribute to GI–TB involvement, including ingestion of contaminated milk or food (primary TB), ingestion of contaminated sputum (secondary TB), hematogenous spread from a distant TB focus, or contiguous dissemination from infected neighboring foci via the lymphatic channels [13, 14]. Given the absence of evidence of extra-abdominal TB in our case, we believe the infection to be primary gastric TB involving the peripancreatic lymph node.

Gastroduodenal TB presents with nonspecific clinical characteristics commonly associated with weight loss, epigastric pain, and fever. In certain cases, GOO may be the presenting feature. Gastric TB may also mimic lymphoma or carcinoma, complicating the differential diagnosis. A study by Yannam et al. conducted at a single center in India reported that among 23 patients with histologically confirmed gastroduodenal TB,

vomiting (60.8%) and epigastric pain (56.5%) were the most prevalent presenting symptoms, with characteristics of GOO observed in 61% of cases (14 patients) [15]. While two patients had pyloric stenosis, the remaining twelve experienced obstruction owing to duodenal stricture. Additionally, out of the 23 patients studied, four had diabetes mellitus, and none were HIV-positive. Similar to this study, our patient presented with vomiting, epigastric pain, and early satiety, suggestive of GOO.

The lack of specific clinical symptoms and diagnostic indicators often leads to underdiagnosis of gastroduodenal TB.

Histopathological examination of gastroduodenal biopsy specimens obtained via endoscopy remains the gold standard for diagnosing gastroduodenal TB. However, owing to the submucosal nature of granulomatous lesions, they are often challenging to identify, even in biopsy samples. A review revealed that granulomas were detected in only seven out of 27 patients who underwent endoscopic biopsy for duodenal TB [16].

Similarly, Yannam et al. reported positive biopsies in their study in only 2 out of 20 patients [15].

Complications of gastroduodenal tuberculosis (GD-TB) may include GOO, hemorrhage, and perforation, which can significantly increase morbidity [7].

As outlined in a review by Yannam et al., in cases of TB-related GOO, truncal vagotomy and gastrojejunostomy (with or without feeding jejunostomy) were performed in twelve out of fourteen patients [15].

While abdominal TB can affect individuals of any age, there is a significant predominance among women, particularly those between the ages of 25 and 45 [17]. In patients with gastroduodenal TB presenting with obstruction or mass, the yield of endoscopic biopsy is typically low [18].

Dyspeptic symptoms suggestive of gastric lesions often lead to suspicion of peptic ulcer, while weight loss may prompt consideration of gastric cancer as the primary diagnosis.

Up to 20% of patients undergoing examinations may exhibit evidence of pulmonary TB on chest X-ray [19], and duodenal bulb deformity may be detected during upper GI endoscopy [20]. Because ulcerated lesions predominantly reside in the submucosa, conventional endoscopic biopsies often yield poor results and rarely uncover granulomas [21].

Although preoperative diagnosis of duodenal TB is exceedingly rare [12], Sharma et al. have demonstrated that an endoscopic ultrasound (EUS) is a valuable modality for characterizing lesions and obtaining samples for cytological confirmation [1]. In cases where histological diagnosis cannot be established by other means, intraoperative fine-needle aspiration cytology (FNAC) may be employed to obtain samples from the affected duodenal section [22].

Most lesions diagnosed with TB before surgery respond well to appropriate antitubercular treatment and may not require surgical intervention [23]. Antituberculosis medication therapy is the cornerstone of medical treatment for gastric TB. However, surgery becomes necessary for patients experiencing severe GOO owing to hypertrophic TB, with antituberculosis medication therapy following surgery [24].

In cases of GOO, gastrojejunostomy is preferred over pyloroplasty owing to the severe fibrosis around the pyloroduodenal junction, which may compromise the safety of pyloroplasty.

This study adheres to the SCARE 2016 criteria [25]

CONCLUSION

Primary gastric TB is rare and often poses a diagnostic challenge, particularly when it presents as GOO. In regions where TB is prevalent, heightened suspicion is warranted. Surgery is often essential for diagnosis and treatment.

91171_Auto_Edited.docx

ORIGINALITY REPORT

3%
SIMILARITY INDEX

PRIMARY SOURCES

1	mail.ijsurgery.com Internet	28 words — 1%
2	"Clinical Vignettes – Stomach", The American Journal of Gastroenterology, 10/2009 Crossref	15 words — 1%
3	www.oalib.com Internet	13 words — 1%
4	f6publishing.blob.core.windows.net Internet	12 words — 1%

EXCLUDE QUOTES	ON	EXCLUDE SOURCES	< 1%
EXCLUDE BIBLIOGRAPHY	ON	EXCLUDE MATCHES	< 10 WORDS