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EDITORIAL

Foregut tuberculosis: Too close but miles apart

Yumna Shahid, Muhammad Aarish Anis, Shahab Abid

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Abstract

The worldwide burden of tuberculosis (TB) has increased and it can involve virtually any organ of the body. Intestinal TB accounts for about 2% of the cases of TB worldwide. The ileocecal region is the most commonly affected site, and the foregut is rarely involved. The reported incidence is approximately 0.5%. Esophageal TB presents with dysphagia, weight loss, and hematemesis in rare cases. Gastroduodenal TB usually manifests with symptoms such as nausea, vomiting, weight loss, and sometimes with gastric outlet obstruction. Gastroscopy may reveal shallow ulcers in stomach and duodenal deformity when underlying TB is suspected, therefore histopathology plays pivotal role. On computed tomography, duodenal TB typically manifests as duodenal strictures predominantly, accompanied by extrinsic compression, and occasionally as intraluminal mass. But their diagnosis can easily be missed if proper biopsies are not taken and samples are not sent for GeneXpert testing, TB polymerase chain reaction investigation and histopathological analysis. Despite being in close proximity to the lungs, the esophagus and stomach are rare sites of TB. The reasons could be low gastric pH and acidity which does not let mycobacterium grow. But there are various case reports of TB involving the foregut. We have summarized the rare cases of foregut TB in different sections and highlighted the importance of esophagogastroduodenoscopy, histopathology and advanced techniques like endoscopic ultrasound in establishing the diagnosis.

Key Words: Foregut; Tuberculosis; Gastroduodenal; Esophagogastroduodenoscopy; Mycobacterium; Caseating granulomas

Core Tip: Gastroduodenal tuberculosis (TB) often mimics peptic ulcer disease and malignancies, presenting with symptoms such as gastric outlet obstruction and hematemesis, which necessitate urgent gastroscopic intervention. Differentiating TB from other conditions based solely on endoscopic examination is challenging. Therefore, histopathological analysis and molecular tests like GeneXpert are crucial for an accurate diagnosis. We aim to delve into the intricate details of these unexpected findings, highlighting the diagnostic challenges they present and their potential impact on patient outcomes.

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INTRODUCTION

Tuberculosis (TB) is a perilous malady capable of afflicting virtually any organ system. The worldwide burden of TB approaches nearly 12 million cases. The 2013 report of the World Health Organization estimated the global annual incidence of TB was at 8.6 million, with 1.3 million individuals succumbing to the disease in the year 2012[1]. TB can occur in any part of gastrointestinal (GI) tract. Intestinal TB (ITB) accounts for about 2% of the cases of TB worldwide[2]. TB can affect the abdomen *via* various mechanisms. Firstly, mycobacterium enters the intestine *via* ingestion of infected milk or sputum, infecting the mucosal layer of the GI tract and forming epithelioid granulomas in the lymphoid tissue of the submucosa. Within a few weeks, caseous necrosis of these tubercles causes ulceration of the overlying mucosa and spreads to deeper layers, adjacent lymph nodes, and the peritoneum. Bacilli can also enter the portal circulation, affecting solid organs such as the liver, pancreas, and spleen but this is very rare. Secondly, spread can also occur *via* the blood if a focus of infection elsewhere in the body reaches abdominal solid organs, kidneys, lymph nodes, or the peritoneum. Thirdly, direct spread to the peritoneum can occur from adjacent infected sites and through lymphatic channels from infected nodes[1]. The ileocecal region is the most commonly affected site, followed by the small bowel in cases of ITB. The predominant involvement of the ileocecal area is attributed to the abundance of lymphoid tissue, fecal stasis, increased absorption, and the easy access of mycobacterium to the mucosal lining[3]. Most common symptoms manifesting in ITB are abdominal pain, fever, weight loss, anorexia, nausea and vomiting, and diarrhea[4]. The foregut is rarely involved. According to an autopsy series, the reported incidence was approximately 0.5%. Most common symptoms of gastroduodenal TB are abdominal pain and vomiting. Patients usually present with gastric outlet obstruction or GI hemorrhage. Gastroscopy may reveal shallow ulcers in stomach and duodenal deformity when there is underlying suspected TB, therefore histopathology plays pivotal role[5]. On computed tomography, duodenal TB typically manifests as duodenal strictures predominantly accompanied by extrinsic compression, and occasionally as intraluminal mass[6]. Endoscopic biopsies seldom reveal granulomas, primarily owing to the submucosal localization of these lesions and the inadequacy of routine endoscopic procedures to encompass the submucosal layer[7]. Sharma *et al*[8] reported that endoscopic ultrasonography (EUS) was an exemplary modality for both characterizing the lesion and acquiring a sample for cytological confirmation of the diagnosis. Recent data support the use of newer modalities that help in the diagnosis of TB. Traditional investigations such as culture, acid-fast bacilli (commonly referred to as AFB) staining, and histopathology have low sensitivities, necessitating the use of other diagnostic methods. Modern tests based on molecular techniques include GeneXpert, polymerase chain reaction (PCR), interferon-gamma release assays (commonly referred to as IGRAs), multiplex-PCR and immunological markers. These are anticipated to aid in diagnosing ITB. CD73 is an important immunological marker that can help differentiate between Crohn's granulomas and TB granulomas. CD73 is found more in TB granulomas[9]. This editorial comments on the article about gastroduodenal TB and highlights the diagnostic difficulties faced by clinicians[10].

ESOPHAGEAL TB

Even in countries with the highest burden of TB, the esophagus is a rare site to be affected by mycobacterium. The incidence of esophageal TB is around 0.2%-1% in the GI tract[1,11]. The esophagus has dynamic mechanisms to protect its mucosa, including constant peristaltic movement, mucus, saliva, enzymes, stratified squamous epithelium, and other mucosal factors that may contribute to the low incidence of TB. Hence, primary esophageal TB is extremely rare, and secondary esophageal TB is more common[12]. Multiple theories have been postulated to predict the mechanisms by which the esophagus may become involved. Savage *et al*[13] discussed some mechanisms of spread, first mentioned by Rubinstein in 1958, including direct contact with sputum, *via* the pharyngeal or laryngeal route or extension *via* lymph nodes.

The clinical presentation may vary amongst patients, but the most frequent symptom of esophageal TB is dysphagia in 90% of cases[14], which is possibly related to factors such as mass compression by enlarged lymph nodes, mediastinal fibrosis or stricture, esophageal ulcer, or decreased peristaltic activity[12]. Other common symptoms include hematemesis, retrosternal pain, and cough during swallowing[15]. Coughing during meals indicates the possibility of an esophageal-tracheal fistula, present in 13%-50% of cases, which can cause aspiration of gastric contents into airway[16]. The presence of hematemesis also raises suspicion of a fistula[17]. Clinicians may confuse esophageal TB with esophageal

cancer or Crohn's disease because they have similar disease presentations, but their treatment options are entirely different[18].

Esophagogastroduodenoscopy (EGD) is the cornerstone in managing dysphagia. Diagnosing esophageal TB typically involves histopathology and TB PCR, although detecting caseating granulomas in endoscopic samples has limited sensitivity (25.0%-60.8%)[19]. Owing to the submucosal location of caseating granulomas, it is recommended that multiple biopsies be taken from the ulcer margins during EGD[18]. EUS is another useful modality that can accurately determine the specific layer of esophageal wall from which the lesion arises. In cases of submucosal pathologies, EUS-guided fine needle aspiration (FNA) is preferred over regular endoscopic biopsy because of its higher sensitivity[19]. For esophageal TB, the mainstay of treatment is anti-TB therapy, including all complicated cases[20,21]. Esophageal TB is initially treated for 2 months with isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by 4 months of isoniazid, rifampicin, and ethambutol[22]. Table 1 summarizes case reports of esophageal TB[11-13,15,17,18,22-32].

Table 1 Esophageal tuberculosis

Ref.	Age, sex	Presentation	Diagnosis and treatment
Diallo *et al* [11]	58-year-old man	Dysphagia for 3 months with non-quantified weight loss, anorexia and fever	Esophageal biopsy showed caseous granuloma. Treated with ATT
Mao *et al*[12]	27-year-old woman	1-month history of progressive dysphagia, accompanied by post-sternal pain, belching, acid regurgitation, heartburn, and nausea	Esophageal endoscopy with biopsy performed showed interstitial granulation tissue. TB PCR was positive. Treated with ATT
Savage *et al* [13]	46-year-old woman	6-week history of dysphagia and retrosternal chest pain unrelieved by antacids	Barium swallow showed a 10 cm plaque-like mid-esophageal lesion. Biopsies taken during esophagoscopy were inconclusive. Repeat biopsy 1 month later demonstrated caseating granulomata in the submucosa. Treated with ATT
Savage *et al* [13]	26-year-old man	6-week history of dysphagia for solids and retrosternal pain	Barium meal: Extrinsic compression in the mid-esophagus. Chest X ray: Sub carinal lymphadenopathy; Mantoux test: Strongly positive at 1:10000; Endoscopic biopsies were non-specific. A diagnosis of esophageal TB was made on radiological appearances and strongly positive Mantoux test. Treated with ATT
Han *et al*[15]	56-year-old woman	2-week history of mild dysphagia	Esophageal biopsies showed numerous epithelioid caseating granulomas. The PPD skin test, T-SPOT. TB assay and polymerase chain reaction testing for MTB were positive. Treated with ATT
Jain *et al*[17]	15-year-old man	Bouts of hematemesis and melena for 2 days	EGD followed with biopsy revealed caseating epithelioid granulomas with lymphocytic infiltrate. PCR positive for TB. Treated with ATT
Khan *et al* [18]	25-year-old man	1-month progressive dysphagia for both solids and liquids associated with epigastric pain	Endoscopy followed with biopsy showed granulation tissue. Cultures of biopsied tissue were positive for MTB PCR was positive. Treated with ATT
Zahra *et al* [22]	27-year-old man	Burning chest pain for 3 months; Intermittent fever, weight loss, loss of appetite for few weeks	EGD followed by biopsy revealed granulomatous caseating lesion. Nucleic acid amplification test was positive for TB. Treated with ATT
Abid *et al* [23]	45-year-old woman	Progressive dysphagia and weight loss (8 kg) for 6 months	Endoscopic biopsy of esophageal ulcers revealed granulomatous changes. AFB staining and MTB complex PCR was negative. Treated with ATT
Abid *et al* [23]	80-year-old woman	Massive hematemesis without any history of dysphagia, odynophagia, regurgitation	Endoscopy showed multiple ulcers in proximal esophagus, biopsy of which received granulomatous lesions. AFB stain for MTB was negative, but a positive PCR. Patient died next day because of massive hematemesis
Abid *et al* [23]	85-year-old woman	Dysphagia and cough. 5 kg weight loss in 2 months	Endoscopic biopsy of a hyperemic patch in esophagus revealed granulomatous changes. AFB stain and PCR for MTB was negative. Patient died 3 days after hospitalization due to aspiration pneumonia
Abid *et al* [23]	29-year-old man	Odynophagia and retrosternal burning	Endoscopic biopsy of esophageal ulcer revealed positive MTB PCR, although cultures showed no growth. Treated with ATT
Mohan *et al* [24]	30-year-old woman	Fever, malaise and decreased appetite for 3 weeks. She had dysphagia and cough during swallowing for 4 days	Chest X ray: Miliary TB. Upper GI endoscopy showed ulcer, biopsy of which revealed chronic inflammation with granuloma. Treated with ATT
Olson *et al* [25]	35-year-old man	2 months of progressive dysphagia and odynophagia	AFB culture positive for MTB from surgically removed mediastinal lymph node. Treated with ATT
Fujiwara *et al*[26]	82-year-old man	Progressive dysphagia	Esophagoscopy with biopsy showed epithelioid granulation. CT chest showed enlarged sub carinal lymph nodes. Responded to ATT
Hu *et al*[27]	75-year-old man	Progressive dysphagia for 1 month	EUS followed by histopathology showed tuberculoid granuloma. PPD skin test and TB spot both were positive
Mahmoudi *et al*[28]	55-year-old	Progressive dysphagia for 3 months, weight loss, loss of appetite, intermittent	EGD followed by histopathological examination revealed epithelioid cell granulomas without caseous necrosis. Sputum examination for acid-fast bacilli

	woman	fever	was positive. Treated with ATT
Salad *et al* [29]	23-year-old man	Cough on eating and halitosis, mild weight loss	EGD followed with biopsy showed inflammatory lesions with epithelioid granulomas. Treated with ATT
Khanna *et al* [30]	60-year-old man	Progressive dysphagia for 4 months with loss of weight and appetite	Endoscopy followed by biopsy showed epithelioid granulomas with caseating necrosis. TB PCR was positive. Treated with ATT
Danna *et al* [31]	69-year-old man	Persistent fever and dysphagia for 4 weeks	EGD followed showed deep esophageal ulcerations followed with biopsy with special stains positive for AFB. Treated with ATT
Baleguli *et al* [32]	24-year-old woman	1 month of dysphagia and odynophagia associated with low-grade fevers, dry cough, night sweats, anorexia, sore throat, and pound weight loss	EGD followed with esophageal biopsies showed active ulcerative and granulomatous esophagitis with mycobacterial organisms. AFB culture grew MTB and MTB PCR was positive. Treated with ATT

AFB: Acid-fast bacilli; ATT: Anti-tuberculosis therapy; EGD: Esophagogastroduodenoscopy; EUS: Endoscopic ultrasonography; GI: Gastrointestinal; MTB: *Mycobacterium tuberculosis*; PPD: Purified protein derivative; TB: Tuberculosis; PCR: Polymerase chain reaction.

GASTRIC TB

TB involvement of the stomach has been reported to occur in 0.5%-3% of all cases of GI TB[33,34]. The incidence of gastroduodenal TB is 0.5%[9], and isolated gastric TB is even rarer, comprising 0.1%-2% of all cases of TB. Gastric TB has been reported to be more common in men than in women, with a ratio of 2.8:1[7]. Gastric TB may occur as a primary or secondary infection, usually secondary to pulmonary infection or as a part of multifocal GI TB or miliary TB[35]. Primary or isolated gastric TB is a rare occurrence, and the main reason for it may be the consumption of unpasteurized milk infected with bovine TB or a severely immunocompromised condition[36,37]. The rarity of gastric TB can be attributed to several factors, the lack of lymphoid tissue in the gastric mucosa, the high acidity and bactericidal properties of gastric acid, the rapid transit time of the stomach because of its continuous motor activity, which swiftly moves organisms through the stomach, and the intact gastric mucosa that contributes to the local immunity of the gastric wall[34,38,39]. The symptoms of gastric TB differ depending on the site involved[40]. Patients may present with abdominal pain, vomiting, fever, weight loss, lethargy, upper GI bleed, and gastric outlet obstruction[1].

The diagnosis of gastric TB is challenging, and endoscopy often plays a vital role. Gastroscopy usually reveals submucosal pathology or ulceration in mucosa. Lesions found by EUS are described as hypoechoic lesions in the lamina propria, making it difficult to distinguish from gastric stromal tumors. Percutaneous ultrasound-guided FNA, Tru-Cut biopsy, and EUS-guided FNA can aid in the diagnosis of TB because the lesions are often located in the submucosa; however, the diagnostic accuracy of histopathology in diagnosing gastric TB is low[41]. PCR of gastric aspirates is also useful in the diagnosis of gastric TB, but the specificity is only approximately 85%[42]. Table 2 summarizes case reports of gastric TB[40,41,43,44-54].

DUODENAL TB

Duodenal TB comprises 2%-2.5% of cases of GI TB, which most commonly affects the third part of duodenum[1]. Gupta *et al*[55] described the characteristics of duodenal TB, which include inflammatory changes in duodenal mucosa, narrowing of duodenal lumen and granulomas seen on histopathology. Most of the published case reports describe duodenal TB as presenting with weight loss, persistent vomiting, and gastric outlet obstruction[56]. Gastric outlet obstruction can occur *via* two mechanisms, intrinsic (due to mucosal involvement), or extrinsic (due to external compression of enlarged lymph nodes)[57]. Duodenal TB frequently faces challenges in accurate diagnosis, often leading to its misidentification as Crohn's disease. The presence of granulomas in a duodenal stricture, particularly in the absence of caseating lesions and when accompanied by a sinus tract, complicates the differentiation between duodenal TB and Crohn's disease. This complexity may result in delays in administering appropriate anti-TB treatment and the unfortunate initiation of corticosteroid therapy. The key to a definitive diagnosis lies in identifying mycobacteria in gastroduodenal lavage material, biopsy specimens, or direct detection by acid-fast staining or culture. Therefore, a cautious approach to steroid administration is warranted when Crohn's disease is suspected. Comprehensive investigations should be conducted to exclude the possibility of ITB[58]. As per the guidelines, a conclusive diagnosis of GI TB is attainable when any of the following four criteria are met, detection of AFB, a positive result in TB PCR testing, the presence of caseating granulomas, or a positive TB culture obtained from a biopsy specimen. Nevertheless, establishing a diagnosis of duodenal TB using regular biopsy material is infrequent due to the predominant localization of TB granulomas in the submucosa[59]. Therefore deeper EUS-guided biopsies may have a higher diagnostic yield[60]. Duodenal TB case reports are summarized in Table 3 [56-58,60,61-69].

CONCLUSION

Despite being in close proximity to the lungs, the esophagus and stomach are rare sites of TB. The reasons could be low

Table 2 Gastric tuberculosis

Ref.	Age, sex	Presentation	Diagnosis and treatment
Eray *et al* [40]	42-year-old woman	Loss of appetite, post-prandial epigastric pain, weight loss	Post- operation (on prediagnosis of gastric cancer) biopsy of tissue showed caseating granulomatous inflammation. TB PCR was positive. Treated with ATT
Zhu *et al* [41]	25-year-old woman	Upper abdominal discomfort and distension	EUS along with biopsy revealed granulomatous inflammation. PPD skin test was positive. Treated with ATT for 1 year
Zhu *et al* [41]	52-year-old woman	Upper abdominal pain and swelling for 6 months, 3 kg weight loss over 2 months, low-grade fever and night sweats for 1 week	EUS followed with biopsy showed granulomatous inflammation with caseous necrosis. Treated with ATT for 13 months
Zhu *et al* [41]	16-year-old man	Upper abdominal pain and fatigue for 3 months along with 4 kg weight loss	Biopsy post ESD (done with suspected diagnosis of interstitial tumor) showed caseous granuloma. PPD skin test was positive. Treated with ATT for 6 months
Seetlani *et al*[43]	55-year-old man	Persistent episodes of nonbilious vomiting and epigastric pain for 3 months, associated with weight loss	Endoscopic biopsies showed epithelioid cell granulomas. Multiple solid enhancing lymph nodes were seen on CT Abdomen. Treated with ATT for 6 months
Lv *et al*[44]	60-year-old woman	Endoscopy from another hospital showed smooth protruding lesion from gastric cardia	ESD performed (due to suspicion of gastric stromal tumor) followed with biopsy showed caseous necrosis. TB PCR was positive. Treated with ATT
Yan *et al* [45]	53-year-old woman	Upper abdominal pain and discomfort for 4 years	Histological analysis post laparoscopic surgical resection (due to suspected gastric tumor) showed granulomatous inflammation. GeneXpert MTB was positive
Lim *et al* [46]	38-year-old woman	Epigastric discomfort for 1 month along with palpable abdominal mass	Endoscopic biopsy showed necrotic granuloma with an abscess. AFB stain and PCR were positive for MTB. Due to resistance kanamycin, moxifloxacin, prothionamide and cycloserin and pyrazinamide were given for 1 year
Ma *et al*[47]	26-year-old woman	Constant gastric pain for 1 month accompanied with acid reflux	Gastroscopic biopsy showed caseous granuloma. MTB PCR was positive. Treated with ATT for 18 months
Talukdar *et al*[48]	30-year-old woman	Epigastric pain for 1 year along with progressive loss of appetite and 15 kg weight loss	Thickened gastric wall on CT scan. Endoscopic biopsy showed epithelioid granuloma with caseation. Ziehl–Nielsen staining of the biopsy specimen revealed AFB
Manoria *et al*[49]	39-year-old woman	Epigastric pain for 1 month accompanied with 3 kg weight loss	Endoscopic biopsy revealed multiple granulomas with epithelioid cells and multinucleate cells. Mantoux test was positive. Treated with ATT
Sharma *et al*[50]	21-year-old woman	2-day history of acute abdominal pain with bouts of hematemesis. CT showed gastric perforation	Biopsy post emergency distal gastrectomy revealed tuberculosis granulation tissue and acid- fast bacilli in the ulcer
Zhang *et al* [51]	40-year-old woman	Painful enlarged cervical lymph nodes for 2 weeks	Gastroscopic biopsy showed chronic granulomatous inflammation. AFB was positive. Treated with ATT for 6 months
Liu *et al*[52]	68-year-old man	Epigastric pain for a few months	Subtotal gastrectomy (suspected tumor) followed by histopathological examination revealed necrotizing granulomatous inflammation. MTB PCR was positive
Liu *et al*[53]	52-year-old woman	Upper abdominal pain for 6 months	Endoscopic biopsy showed caseous granuloma. Acid-fast stain and MTB PCR were positive
Khan *et al* [54]	29-year-old man	Epigastric pain for 1 month with 7 kg weight loss	Endoscopic biopsy specimen revealed caseating granulomas with AFB. Positive cultures for MTB

AFB: Acid-fast bacilli; ATT: Anti-tuberculosis therapy; CT: Computed tomography; EGD: esophagogastroduodenoscopy; ESD: Endoscopic submucosal dissection; MTB: *Mycobacterium tuberculosis* PCR: Polymerase chain reaction; PPD: Purified protein derivative.

gastric pH and acidity which do not let mycobacterium grow. However, the case reports mentioned earlier are examples of uncommon instances where typical symptoms such as vomiting, weight loss, or abdominal pain occur. If these symptoms are overlooked or not thoroughly investigated, they can easily go unnoticed. Foregut TB frequently eludes a correct diagnosis by clinicians owing to superficial biopsies and the omission of tissue analysis for *Mycobacterium tuberculosis* PCR and GeneXpert testing. When TB is strongly suspected, conducting a thorough diagnostic examination is essential to minimize morbidity. TB is a manageable condition, hence swift diagnosis and appropriate treatment can help alleviate complications. EUS-FNA helps to obtain deeper core tissue samples, which have a positive yield in histopathology compared with superficial biopsies obtained with forceps. Therefore employing advanced endoscopic techniques such as EUS may prove advantageous with a higher diagnostic yield. Clinicians should always suspect ITB, especially in

Table 3 Duodenal tuberculosis

Ref.	Age, sex	Presentation	Diagnosis and treatment
Mani and Rananavare[56]	18-year-old man	Persistent vomiting due to duodenal stricture as shown in endoscopy	Underwent laparotomy and resection. Post-surgical specimen revealed tuberculosis granulomas
De *et al*[57]	38-year-old woman	Recurrent vomiting	CT showed mural thickening of duodenum, deeper endoscopic biopsies revealed non-necrotizing granulomata. Treated with ATT
Naouri *et al*[58]	69-year-old man	Duodenal stenosis	Misdiagnosed as Crohn's disease
Sato *et al*[60]	35-year-old man	Abdominal fullness, vomiting, CT showed the duodenal wall thickening, luminal narrowing, multiple enlarged abdominal lymph node	EUS-FNA biopsy of duodenum showed caseating granulomas with multinucleated giant cells, and AFB were positive by Ziehl–Nielsen staining. Underwent laparoscopic gastrojejunostomy, and ATT after surgery
Antón Rodríguez *et al*[61]	60-year-old woman	Weight loss, bloating, duodenal thickening on CT scan	Endoscopic biopsy showed granulomatous inflammation and positive PCR for MTB. Treated with ATT
Chang *et al*[62]	52-year-old man	Early satiety, post meal abdominal pain, weight loss	Duodenal tissue culture showed growth of MTB. Treated with ATT
Pratap *et al*[63]	43-year-old woman	Nausea, vomiting, fever, weight loss	Duodenal ulcer on endoscopy with biopsy showing active duodenitis and positive AFB stain, lymph node biopsy also confirmed TB. Treated with ATT
Dahiya *et al*[64]	23-year-old man	Post-prandial fullness, weight loss	Duodenal narrowing on endoscopy. Duodenal segment resection with duodenojejunostomy, biopsy showed granulomatous inflammation suggestive of TB
Zhang *et al*[65]	71-year-old man	Upper abdominal pain	Space occupying lesion on CT scan, endoscopic biopsy showed granulomas. Treated with ATT
Moirangthem *et al*[66]	17-year-old man	Gastric outlet obstruction	Underwent laparotomy which revealed duodenal mass. Biopsy showed caseating tuberculosis
Berney *et al*[67]	22-year-old man	Duodenal ulcer perforation	Underwent laparotomy. Margins of ulcer were resected. Biopsy showed giant-cell-granulomatous inflammation. Lymph node biopsy showed caseating necrosis
Souhaib *et al*[68]	33-year-old woman	Perforated duodenal ulcer which was initially diagnosed as acute cholecystitis	Underwent laparotomy. Peri-duodenal lymph node and the gallbladder lymph node showed caseating necrosis
Sharma *et al*[69]	32-year-old man	Recurrent vomiting, weight loss, thickened duodenum seen on CT scan and endoscopy	Histopathology revealed features of chronic inflammation, giant cells, and granulomas. Ziehl–Nielsen stain of tissue specimens was positive for AFB. Treated with ATT for 8 months

AFB: Acid-fast bacilli; ATT: Anti-tuberculosis therapy; CT: Computed tomography; EUS: Endoscopic ultrasound; FNA: Fine needle aspirate; MTB: *Mycobacterium tuberculosis*; PCR: Polymerase chain reaction.

low middle income countries where prevalence of TB is higher. This editorial intends to inform about newer modalities to consider that enhance diagnostic accuracy and improve patient care. In situations where there is a high suspicion of TB indicated by a mass-forming lesion, irregular mucosa, weight loss, vomiting, and biopsies have are for malignancy, specimens should be tested using GeneXpert and TB culture. Molecular studies, such as IGRA, may also provide valuable diagnostic information.

FOOTNOTES

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