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CASE REPORT

Tuberculous Pyloric Stenosis; Successful Medical Therapy May Obviate Surgery

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Abstract
Gastroduodenal involvement occurs in only 0.3 to 2.3% of patients with tuberculosis (TB). Clinically, it may resemble peptic ulcer disease or malignancy. We present a 36 year old man with gastric outlet obstruction proven to be pyloric stricture due to primary pyloric tuberculosis. Diagnosis was established endoscopically and patient was successfully managed by standard anti-tuberculous therapy. Although rare, tuberculous involvement of the stomach should be considered in the differential diagnosis of gastric outlet obstruction especially in regions where tuberculosis is endemic. This case also emphasizes that medical management with anti-TB medications can be tried solely as the first line therapy for strictured pyloric stenosis. However, surgical intervention is required more often than none in cases where gastrointestinal obstruction is the main clinical problem.

Key words: Tuberculosis, Pyloric Stenosis, Gastric Tuberculosis

Introduction

Gastroduodenal tuberculosis (GD-TB) occurs in 0.3 to 2.3% only of patients with TB. Clinically it may resemble peptic ulcer disease or malignancy (1-5). Gastric outlet obstruction (GOO) is the most common complication of GD-TB. In one report, for example, GOO was the presenting feature in 61% of 23 patients with biopsy-proven GD-TB (6). The obstruction may be due to infiltration of the gastric antral or duodenal wall, or to extrinsic compression by adenopathy or a phlegmon (2,7). GD-TB is rare and as it exhibits no specific symptoms or signs and has no characteristic endoscopic or radiographic features (8,9), diagnosis requires a high index of suspicion. We report a case of endoscopically diagnosed and medically managed tuberculous pyloric stenosis.

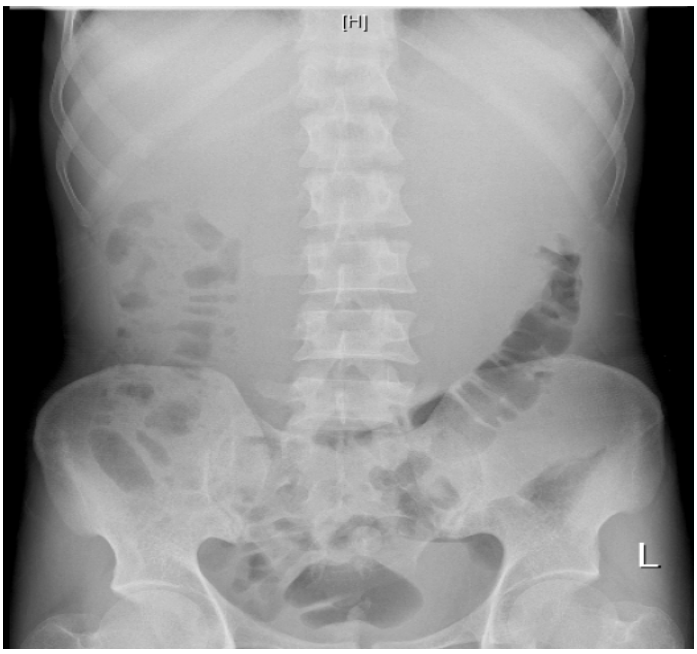

Figure 1. Abdomen KUB shows an almost gasless mid abdomen and lumbar region with displacement of the large bowel loops inferiorly.

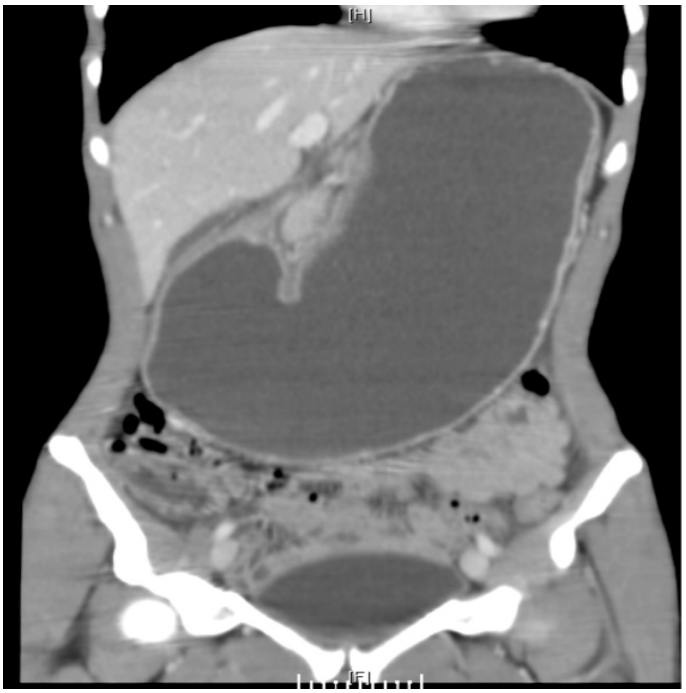

Figure 2. Markedly distended fluid-filled stomach with collapse of the duodenum and the small bowel loops. Findings are consistent with gastric outlet obstruction.

Case report

A thirty six year old man presented to the emergency room with intermittent epigastric pain of 8 month duration. This had been treated previously as gastritis. He had received H2 blockers and proton pump inhibitors (PPI) with some relief initially. Over the previous two weeks, pain had worsened; becoming persistent and associated with vomiting, constipation and sporadic abdominal colic despite treatment with Omeprazole 40 mg orally once daily. The patient admitted losing weight (about 2.5 kg) over the previous 2 months. He denied any chills, fever, night sweats, cough, hematemesis, melena or diarrhea. He had no past history of tuberculosis or any other significant illness. He had had appendectomy 25 years previously. On physical examination, the patient was conscious and oriented. His vital signs were normal with blood pressure of 103/74 mmHg, pulse rate of 64 beats per minute, temperature at 36.5°C, respiratory rate of 21 breaths per minute, and oxygen saturation was 100% while breathing ambient air. His weight was 40 kg and body mass index 15.4 kg/m^2. Examination of his head and neck, cardiovascular

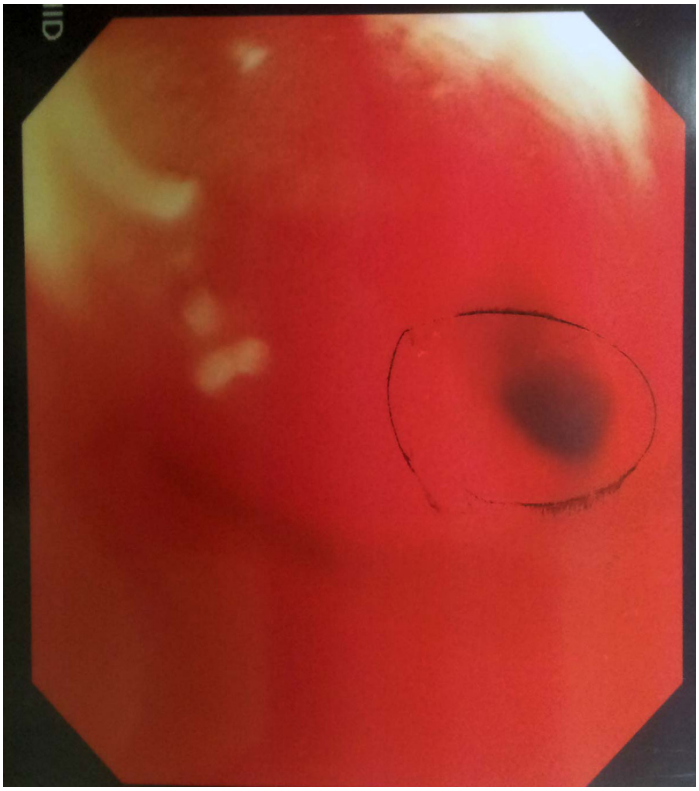

Figure 3. Endoscopic view of the pyloric stricture before medical treatment.

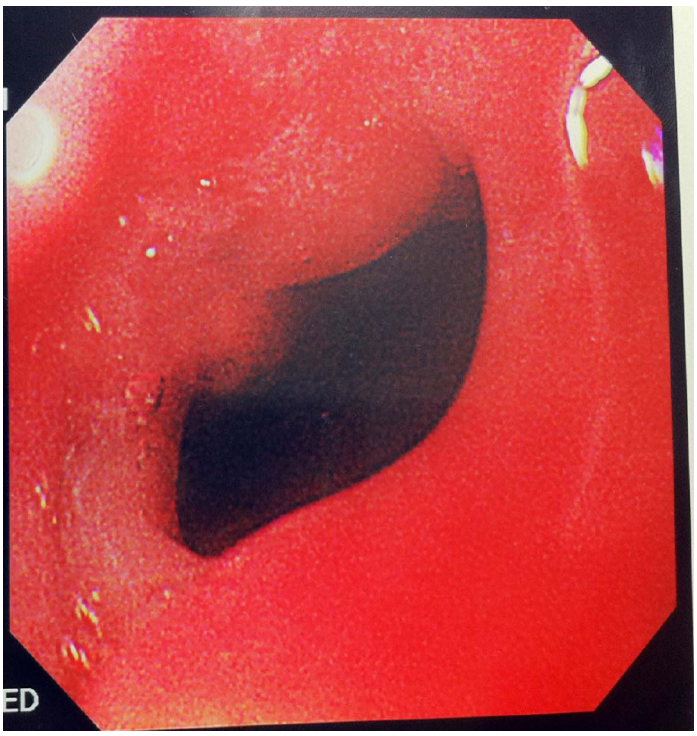

Figure 4. Endoscopic view of the pyloric stricture after medical treatment.

system and chest was unremarkable. Abdominal examination revealed visible epigastric peristalsis, a scar of previous appendectomy, mild epigastric tenderness but no rigidity or rebound tenderness. No masses or organ enlargement were detected. Basic investigations revealed a white blood cell count of 5.8 $x10^9$/L, a hemoglobin level of 118 g/L, a platelet count of 397 $x10^9$/L, normal serum amylase and normal renal and liver function tests. Chest radiograph was normal; abdominal plain KUB film showed that the mid abdominal and lumbar regions were almost gasless with displacement of the large bowel loops inferiorly (Figure 1). The patient was admitted to hospital with the initial impression of gastric outlet obstruction for further management. He was put on *nil per os* (NPO); with intravenous fluid drip was set up and an nasogastric (NG) tube was inserted for drainage. CT abdomen became available on the next day. This revealed a markedly distended fluid-filled stomach with collapse of the duodenum and the small bowel loops. These findings were consistent with gastric outlet obstruction. No obvious masses were demonstrable in the stomach (Figure 2). The patient was kept NPO and started on total parentral nutrition (TPN). Endoscopy revealed a small hiatus hernia, severe pan-gastritis and severe pyloric stricture. An adult scope could not be passed through however a pediatric scope passed (with some manipulation). Balloon dilatation up to 8 mm was done and biopsies were taken (Figure 3). The first part of the duodenum (D1) was thick-walled. The patient was started on parenteral PPI infusion, TPN was stopped and clear liquids initiated. Endoscopy was repeated 3 days later which revealed moderate gastritis with pyloric stricture that was dilated to 10 mm with no complications. The patient tolerated oral intake and was sent home on PPI with a follow up endoscopy appointment one week later. The endoscopic biopsy showed severe granulomatus gastritis with multinucleated giant cells but definite caseation could not be identified. Acid fast bacilli cultures were suggested. Endoscopy was repeated as planned and this showed normal esophagus but the pyloric stricture was tighter than before. Even the pediatric scope could not be passed on this occasion. TB culture was taken as recommended and the patient was referred for surgical evaluation. After multi-disciplinary discussion, it was agreed to start him on anti-tuberculous medications pending culture results. The patient was prescribed on daily Izonizide 300 mg, Pyrzinamide 1000 mg, Rifamicn 600 mg and Ethambutol 800 mg. Follow up endoscopy was scheduled after 2 months provided the patient continued to improve. This showed normal esophagus, the pyloric stricture was still present (but less severe than before), and the duodenal cap was deformed and the adult scope managed to pass through with some manipulation. The culture was negative for acid fast bacilli, but in view of improvement of his condition, it was recommended to continue the empiric anti-TB drugs for a period of one year. The patient was followed up in clinic jointly by the gastroenterologist and infectious disease specialist. His weight and physical condition improved. Endoscopy was repeated twice (After completing the treatment and 6 months later). In both occasions endoscopy revealed normal esophagus, stomach, and deformed duodenal cap with normal second and third parts of the duodenum (D2 and D3). The pyloric stenosis seen previously has now markedly improved (Figure 4).

Discussion

There is a resurgence of tuberculosis in western countries, mainly found either in immigrants from countries where TB is endemic or in immunosuppressed patients with leukemia or AIDS (10). The most common site for gut involvement of TB is the ileocecal region (2). Tuberculosis of the stomach

is rare and is usually secondary to pulmonary tuberculosis (11). Primary and isolated gastric tuberculosis without evidence of lesions elsewhere is fairly uncommon (12). Possible reasons for sparing of the stomach by TB infection include high acidity, a paucity of lymphoid tissue and rapid transit of food through the stomach (8,13-15). Hence, long-term therapy with H2 blockers increases the incidence of GD-TB (16). Due to its rarity, lack of specific diagnostic features and absence of co-existing pulmonary disease, GD-TB is not usually thought of as a cause of gastric outlet obstruction. Therefore, this may lead to unnecessary surgical interventions in cases that could be dealt with conservatively (2). In our case, no caseation was observed, in agreement with the previous reports suggesting that TB is not always present in mucosal biopsy (17). Surgery is usually reserved for patients who have developed complications, including perforation, confined perforation with abscess or fistula, massive bleeding, complete obstruction, or obstruction not responding to medical management (18-20). Our case is an example of tuberculous pyloric stenosis that was diagnosed endoscopically, did not respond to balloon dilatation but was successfully treated with anti-TB medications. Thus confirming that surgery is not the only approach (21-22).

Conclusion

Tuberculous involvement of the stomach, albeit rare, should be considered in the differential diagnosis of gastric lesions especially in geographical regions where tuberculosis is endemic. This case emphasizes that perseverance with medical management in strictured pyloric stenosis can be tried as a first line therapy before resorting to surgery.

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