CASE REPORT

An unusual case of gastric outlet obstruction caused by tuberculosis: challenges in diagnosis and treatment

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SUMMARY

Gastroduodenal tuberculosis (GDTB) is rare in the West. Its presentation can be non-specific and often mimics other more common conditions such as peptic ulcer disease, malignancy and Crohn's disease. Our case describes a 33-year-old Indian immigrant who presented with a 3-year history of dyspepsia and underwent balloon dilation for gastric outlet obstruction (GOO). While biopsies from the duodenum revealed only non-caseating granuloma, a high index of suspicion was maintained and colonoscopy, performed despite the absence of lower gastrointestinal symptoms, revealed a single discrete nodular and ulcerated area in the proximal transverse colon; this eventually grew *Mycobacterium tuberculosis*. Our patient avoided undergoing major surgery and was successfully treated with balloon dilation and antitubercular medication. We highlight the importance of having a concerted, proactive approach to diagnosis. We discuss the therapeutic challenges involving this rare condition and explain the rationale for high-dose antisecretory therapy.

BACKGROUND

Gastroduodenal tuberculosis (GDTB) constitutes only 2% of gastrointestinal tuberculosis (GITB) even in parts of the world where tuberculosis (TB) is endemic.¹ Its presentation can be non-specific. The diagnosis is further complicated by frequently negative cultures and microscopy for acid-fast bacilli. It is for these reasons that gastric outlet obstruction (GOO), secondary to TB, has been traditionally managed by surgery for both diagnosis and treatment.

GOO as the primary manifestation of TB is rare. There are only isolated case reports of TB presenting with GOO from the west and the diagnosis in all was made postoperatively.²⁻⁴ To our knowledge, this is the first report from the western literature where a patient has been treated with balloon dilation and chemotherapy resulting in a complete resolution of the disease.

CASE PRESENTATION

A 33-year-old male Indian IT professional, who had moved to the UK in 2003, was referred to the gastroenterology clinic in August 2009 with a 3-year history of dyspepsia. Initially, the dyspepsia was intermittent, but it had progressed to become continuous and was unresponsive to lansoprazole, 30 mg daily. He presented with early satiety and occasional vomiting. He had lost 4 kg of weight.

He had no lower gastrointestinal symptoms. Apart from lansoprazole, he was on no other medication. In particular, there was no history of the usage of prescribed or over the counter aspirin or non-steroidal anti-inflammatory drugs. He was a non-smoker and had no significant medical history.

Physical examination was unremarkable; in particular, there was no succussion splash.

INVESTIGATIONS AND MANAGEMENT

Blood tests showed haemoglobin 15.7 g/dl, erythrocyte sedimentation rate 4 mm/h, white cell count $5.1 \times 10^9/l$ and lymphocytes $0.6 \times 10^9/l$. Urea and electrolytes, liver function tests and thyroid function tests were within normal limits.

Lansoprazole was discontinued and an upper gastrointestinal (GI) endoscopy (OGD, oesophago-gastroduodenoscopy) was performed 4 weeks later.

OGD showed a nodular, inflamed and distorted pylorus and the bulb of the duodenum (figure 1A,B). There was a complete GOO and it was not possible to pass a 9 mm endoscope into the second part of the duodenum (D2). Dilation was not attempted as the procedure was carried out without sedation with pharyngeal lignocaine spray.

In view of the concerns about the compromised bioavailability of antisecretory medication (AST), he was treated aggressively with a combination of high dose of proton pump inhibitor, lansoprazole 60 mg twice daily and ranitidine 600 mg syrup at bedtime.

The rapid urease test was negative. Biopsies from the pyloroduodenal stricture showed a large epithelioid granuloma and a slight increase in plasma cells and eosinophils in the lamina propria. *Helicobacter pylori* (HP) were not seen. The Ziehl-Neelsen stain was negative. The faecal HP antigen test was negative. A fasting serum gastrin level was normal. A CT scan of the abdomen was unremarkable.

OGD was repeated a week later and the stricture was dilated with a balloon using the through the scope (TTS) technique to 15 mm; it was then possible to get a 9 mm gastroscope past the stricture into the second part of the duodenum. Beyond the grossly abnormal pyloroduodenal area, in the second part of the duodenum, there was a single separate discrete ulcerated nodular lesion (figure 2).

The mucosa beyond this area was normal. Biopsies taken from this lesion were reported as showing focal ulceration and the presence of an epithelioid non-caseating granuloma. Histology

To cite: Padmanabhan H, Rothnie A, Singh P. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008277

Figure 1 (A, B) Oesophagogastroduodenoscopy showing nodular, inflamed and distorted pylorus and the bulb of the duodenum.

from the stricture showed non-specific inflammation. The Ziehl-Neelsen stain was again negative.

At this point, two possible diagnoses were considered: Crohn's disease and TB. However, the possibility of idiopathic peptic ulcer disease could not be ruled out. A high-dose AST was continued.

A follow-up OGD, 4 weeks later, showed an improvement in that now it was possible to pass a 9 mm endoscope with a gentle push through the pyloroduodenal stenosis. The first part of the duodenum (D1) and the D1/D2 junction were still

Figure 2 Oesophagogastroduodenoscopy showing a single separate ulcerated nodular lesion in the second part of the duodenum.

grossly nodular. Further biopsies showed the same changes as before.

A number of investigations were carried out to look for evidence of TB. Mantoux test was positive at 20 mm. Chest radiograph was normal. A urine test for acid-alcohol-fast bacilli (AAFB) was negative. Histology and culture from the gastroduodenal area were negative for AAFB.

Despite the absence of lower gastrointestinal symptoms, an ileocolonoscopy was performed to search for evidence of Crohn's disease elsewhere in the GI tract. The ileocolonoscopy showed a totally normal terminal ileum up to an approximate insertion depth of 20 cm. However, there was a single discrete nodular and ulcerated area with luminal narrowing in the proximal transverse colon (figure 3). The rest of the colon was completely normal.

Biopsies were taken from this lesion for histology and culture for TB. The histology showed a large non-caseating granuloma with pale staining histiocytes (figure 4). The Ziel Neelsen stain was negative.

The differential diagnosis was now between the Crohn's disease and gastroduodenal plus colonic tuberculosis. The differentiation between the two conditions was crucial because the treatment would be so different. Even though Crohn's disease was a very likely diagnosis, there was a reluctance to treat him with steroids in case the actual diagnosis was TB. After a detailed discussion with him, we were about to embark on the empirical antitubercular treatment (ATT) when fortuitously, 6 weeks after colonoscopy, the growth of *Mycobacterium* was reported from the colonic biopsies.

TREATMENT AND FOLLOW-UP

OGD was repeated to ensure the patency of the gastric outlet so that we could be confident that the ATT would get past into the small bowel to be available for absorption. OGD showed a patent outlet and a grossly nodular and narrowed pyloroduodenum. He was started on quadruple therapy for TB (rifampicin, isoniazid and pyrazinamide (rifater) and ethambutol) along with AST. He was monitored closely with regular follow-up and OGDs to ensure drug compliance and bioavailability which was a major concern.

A follow-up colonoscopy after 3 months of ATT showed complete healing with scarring and pseudopolyp formation of the lesion in the transverse colon (figure 5). Similarly, a follow-up OGD (figure 6) demonstrated a patent gastric outlet and complete healing of the inflammation with heavy scarring.

After 4 months of quadruple therapy, the ATT was changed to dual therapy, rifampicin and isoniazid (rifinah), for the remaining 8 months. Meanwhile, over a period of time under close endoscopic surveillance, the level of acid suppression was progressively reduced such that 12 months after the start of ATT all drugs were withdrawn. At the cessation of therapy, and subsequently on clinic follow-up 6 months later, he was completely symptom free.

DISCUSSION

GITB is rare even in the regions of the world where TB is endemic.⁵⁻⁷ However, it is possible that the prevalence of this condition is underestimated. In 1985, a case series from an urban hospital in Britain, serving a large immigrant population, showed an incidence of GITB to be very similar to newly diagnosed Crohn's disease (5 cases per 100 000/annum).⁸ Ramesh *et al*⁹ found that 91% of patients with GITB in the Northwest region of England were of South Asian origin. A retrospective epidemiological study of abdominal tuberculosis in the city of

Figure 3 (A–D) Colonoscopy showing a single discrete nodular and ulcerated area with luminal narrowing in the proximal transverse colon.

Leicester from 1972 to 1989 reported that the majority of cases of GITB arose among immigrants rather than their offspring.¹⁰

The most common location for GITB is the ileocaecal region.¹ Gastroduodenum is rarely involved, accounting for 2% of all cases of GITB.^{1–11} Postulated mechanisms for the rarity of TB in this location are the high acidity, paucity of lymphoid tissue and the relatively rapid emptying time of the stomach.^{12–13}

Figure 4 Histology from the colonic lesion showing a large granuloma.

Rao *et al*,¹⁴ in a paper based on a retrospective case series published in 2003, reported only 23 cases of GDTB over a 15-year period from a large tertiary centre in India. Fourteen of the 23 patients presented with features of GOO. In only 2 of the 23 patients, the diagnosis was suspected preoperatively based on the presence of caseating granulomas in endoscopic biopsies. They make no comment about the positive culture in any of their patients. Only 14% of their patients had evidence of pulmonary TB. All but one patient, who had presented with a gastrointestinal bleed, underwent surgery for diagnosis and treatment.

In a similar retrospective case series, Gupta *et al*⁷ described their experience of 30 cases of duodenal TB over a period of 20 years, of which 73% had symptoms of GOO. In all the patients, the diagnosis of TB was only made postoperatively. Caseating granulomas were found in lymph node biopsies in 18 of the 30 cases; in the remaining, the diagnosis was based on a suspicion of coexistent ileocaecal and/or pulmonary TB together with a good clinical response to ATT.

The two case series by Gupta *et al*⁷ and Rao *et al*¹⁴ illustrate a number of key issues relating to GDTB in general and GOO related to GDTB in particular. First, this is an uncommon condition. Second, obtaining microbiological diagnosis is very difficult. Third, the diagnosis is usually made postoperatively and

Figure 5 Follow-up colonoscopy showing complete healing of the colonic lesion with scarring and pseudopolyp formation.

Figure 6 Follow-up oesophagogastroduodenoscopy showing a patent gastric outlet.

even then it is often based on soft criteria such as the presence of caseating granulomas rather than positive identification of *Mycobacterium tuberculosis*. Finally, the majority of patients do not have the supporting evidence of TB such as an abnormal chest radiograph.

The problem of making a diagnosis is compounded by the fact that GDTB can closely mimic other more common conditions such as peptic ulcer disease,^{5 15} malignancy² and Crohn's disease.¹⁶

Most case series describing GDTB have reported major difficulties in obtaining an objective-positive diagnosis of TB based on Ziel-Neelsen stain or a positive culture, even postoperatively. Identification of acid-fast bacilli from endoscopic biopsy samples is possible in only 4–6% of cases due to the paucibacillary nature of GDTB.¹⁷ The difficulty in obtaining definitive evidence may be because the granulomas are located in the sub-mucosa and endoscopic biopsies are limited to the mucosa.¹⁸

In contrast, however, a recent paper from India reported a remarkably high positive diagnosis based on the histological demonstration of granulomas as well as a positive culture.¹⁹

The two remarkable features of this series of 13 cases were the positive objective evidence of TB and a successful outcome with a combination of endoscopic dilation and ATT without requiring surgery. Puri *et al*¹⁹ used an unusual technique to obtain large and deep tissue samples using endoscopic mucosal resection which may have been the reason for their remarkable success in obtaining tissue evidence of TB. As far as we are aware, no other group has reported this technique for diagnostic purposes in this setting.

In our case, the main differential diagnoses were Crohn's disease and TB. Differentiating Crohn's from GITB remains a major challenge to clinicians as they have marked overlap in clinical, radiological, endoscopic, surgical and histological features.²⁰ Repeated biopsies had failed to demonstrate the histological or microbiological evidence of TB. Serology tests, such as antineutrophil cytoplasmic antibody and anti-*Saccharomyces cerevisiae* antibody, are not useful in differentiating GITB from Crohn's disease.^{20 21} PCR for *M tuberculosis* (TB PCR) was not carried out in our patient. TB PCR is highly specific for GITB, but has poor sensitivity.²² It was felt that TB had to be given serious consideration as a possibility as the patient was an immigrant from an endemic area. The other two unlikely possibilities were malignancy and idiopathic peptic ulcer disease. There are a number of examples of GITB misdiagnosed and treated as Crohn's disease reported in the literature.^{23–25}

We performed an ileocolonoscopy, even in the absence of lower GI symptoms, in the hope of finding an evidence of Crohn's disease elsewhere in the GI tract. The normal ileoscopy and discrete nature of the lesion in the colon supported the likelihood of TB. This investigation was crucial to the success of our case as it was only from the colonic biopsies that the *M tuberculosis* was cultured. Even though he had no risk factors, the HIV test was carried out which was negative. It is reported that the relative annual risk of developing active TB when coinfected with HIV is 20–30 times that of a seronegative.²⁶

GOO presents a twofold challenge in drug delivery and bio-availability. First, it physically compromises the delivery of all drugs for absorption from the small bowel. Second, GOO has an additional impact on the bioavailability of proton pump inhibitors (PPI). PPIs are acid-labile prodrugs designed to be absorbed from the small bowel in their lipid-soluble form. Once the lipid-soluble prodrug enters the secretory canaliculi of the parietal cells and comes into contact with acidic pH, it gets converted into its water-soluble active sulfenamide product which then irreversibly binds to the proton pump. In the presence of GOO, the prodrug is subjected to a prolonged contact with an acidic environment prematurely converting into the active sulfenamide product, rendering it ineffective.²⁷ The reasons for starting powerful AST were that first we had no clear evidence supporting either TB or Crohn's disease and idiopathic peptic ulcer disease could not be ruled out. Second, it is well recognised that the diseases of the upper GI tract other than peptic ulcer disease often respond extremely well to acid suppression. We had to adopt a concerted proactive approach to ensure patency of the gastric outlet tract combining endoscopic therapy and powerful AST.

Cases of GOO secondary to GDTB from the west have all been managed surgically and the diagnosis reached postoperatively.^{2–4}

To our knowledge, this is the first report from the western world where a patient has been successfully treated with a combination of balloon dilation and chemotherapy resulting in a complete resolution of the disease. The diagnosis was only possible because of a high level of suspicion and even though the presenting feature was GOO, TB was isolated from the colonoscopy, which was performed despite the fact the patient had no lower GI symptoms.

Learning points

- ▶ Primary gastroduodenal tuberculosis is rare and frequently presents with non-specific symptoms mimicking other much more common conditions. Preoperative diagnosis is often hampered by negative cultures and biopsies.
- ▶ Gastric outlet obstruction secondary to tuberculosis makes the case more challenging as drug delivery is compromised.
- ▶ Traditionally, surgical intervention has been the primary mode for diagnosis and treatment. Case reports from India have shown that balloon dilation of pyloroduodenal strictures appears to be a safe alternative to surgery and we have replicated this in a report from the west.
- ▶ Ensuring adequate drug availability of both antitubercular treatment and antisecretory medication is paramount when managing cases such as this endoscopically. Close monitoring for compliance and patency of gastric outlet through symptom assessment and surveillance oesophagogastroduodenoscopies is an essential part of the treatment.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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