Rare disease

Oesophageal tuberculosis

Latha Bonthala,¹ Eleanor Wood²¹Barts and The London SMD, London, UK;²Gastroenterology Department, Homerton University Hospital, London, UK**Correspondence to** Miss Latha Bonthala, ha08342@qmul.ac.uk**Summary**

The case discussed is that of a previously healthy 48-year-old female who presented with a week long history of epigastric pain and continuing weight loss. A series of investigations and supporting literature alluded to a diagnosis of oesophageal tuberculosis (TB), and antituberculous medication was commenced accordingly. An accompanying discussion considers the incidence, differential diagnoses, pathogenesis, clinical features, investigations and aspects of management of oesophageal TB.

BACKGROUND

Tuberculosis (TB) is re-emerging in less affluent areas of the UK and Europe.¹ TB, most commonly caused by *Mycobacterium tuberculosis*, can target pulmonary and extrapulmonary sites. The gastrointestinal (GI) system is the sixth most common extrapulmonary site to be affected at a 3–5% incidence rate of all TB cases.² Extremely rarely, the oesophagus is a site of TB infection and inflammation.

CASE PRESENTATION

The case is that of a previously healthy 48-year-old Turkish female who has been a resident in the UK for the last 10 years. She presented to her general practitioner with a 2 week history of epigastric pain on swallowing solid foods, dysphagia and 2 kg weight loss. She did not present with odynophagia, vomiting or abdominal distension. No distinct respiratory symptoms of cough, sputum production or haemoptysis were reported. She was not taking any medication at the time of presentation. Significant family history is that her father was previously diagnosed with pulmonary TB. She was urgently referred to the gastroenterology department at Homerton University Hospital; images from an oesophagogastrroduodenoscopy (upper GI endoscopy) showed a small mid length oesophageal ulcer (in close proximity to the tracheal carina) with rolled, raised edges (figure 1). Numerous biopsies taken from the ulcer edge showed profound inflammation with non-necrotising epithelioid granulomas. A subsequent CT scan outlined a subcarinal lymph node mass measuring 5.4×5.2×2 cm (T3, N1, M0) inseparable from the oesophagus (figures 2 and 3). She was referred to the TB clinic and demonstrated a positive tuberculin skin test. This, the positive family history for TB and supporting literature with empirical evidence² helped formulate a diagnosis of oesophageal TB. The diagnosis was made and she was commenced on antituberculous chemotherapy.

INVESTIGATIONS

Except for the investigation results detailed above a normal posteroanterior chest x-ray was reported. A positive tuberculin skin test (induration of 16 mm 72 h post

injection) confirmed the diagnosis in question. Routine blood parameters demonstrated a mixed anaemia, but were otherwise unremarkable. The patient's HIV status was negative.

TREATMENT

Following a referral to respiratory physicians, the patient was commenced on a strict regime of antituberculous therapy, as well as ferrous sulphate 200 mg daily, hydroxocobalamin initially 1 mg monthly for 3 months and pyridoxine 25 mg daily. With regard to antituberculous chemotherapy, initial phase therapy prescribed was a weight – adjusted dose of the combination preparation rifater (rifampicin 120 mg; isoniazid 50 mg; pyrazinamide 300 mg) at five tablets daily, and ethambutol 1 g daily, for 2

Figure 1 An image taken from an upper gastrointestinal endoscopy showing a small mid length oesophageal ulcer (in close proximity to the tracheal carina) with rolled, raised edges.

Figure 2 A coronal CT image demonstrating an enlarged subcarinal mediastinal lymph node measuring $5.4 \times 5.2 \times 2$ cm (arrowed).

Figure 3 An axial CT image demonstrating an enlarged subcarinal mediastinal lymph node measuring $5.4 \times 5.2 \times 2$ cm (arrowed).

months. This was followed by continuation phase antituberculous chemotherapy, 4 months in duration, of rifinah 300 (rifampicin 300 mg, isoniazid 150 mg) twice daily at a weight-adjusted dose.

OUTCOME AND FOLLOW-UP

As antituberculous chemotherapy was commenced, the patient showed signs of clinical improvement. A follow-up upper GI endoscopy undertaken towards the end of

continuation phase therapy (approximately 6 months after initial symptom presentation) showed a mild gastritis but complete healing of the oesophageal ulcer. This finding of improvement did not warrant the need for a follow-up CT. A follow-up chest x-ray was unremarkable.

DISCUSSION

Oesophageal TB is extremely rare, as it accounts for only 0.2–1% of GI TB cases reported, where GI TB accounts

for 3–5% of all TB cases.³ Due to the paucity of cases reported, there are poorly defined clinical, endoscopic and radiological diagnostic features. Oesophageal viral for example, cytomegalovirus, and fungal infections, syphilis, Crohn's disease and most significantly, oesophageal carcinoma are considered in the differential diagnoses, the latter condition justifying urgent referrals to gastroenterology departments.⁴

Primary TB is typically pulmonary in origin; it is uncommon to have a primary oesophageal infection with *M tuberculosis*, therefore the oesophageal infection can occur most commonly as a result of post primary TB (reactivated lung infection) or extension from infected hilar or tracheobronchial mediastinal lymph nodes. These nodes can soften and become inseparably attached to the oesophagus.⁵ As the CT chest of this patient showed a well-defined enlarged mediastinal lymph node and the x-ray chest was clear it is most likely that extension from an infected subcarinal lymph node mass was the causative pathological event. The most commonly reported presenting feature in oesophageal TB as a result of oesophageal stricturing is dysphagia. Other associated clinical features include odynophagia, retrosternal or epigastric pain, coughing with swallowing solid foods and haematemesis. In addition, weight loss and other systemic symptoms can be reported.⁶

Investigation modalities used in the diagnosis of oesophageal TB include haematological investigations such as inflammatory markers (erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)), haematinics and HIV screening, as approximately upto 15% of new annual TB cases globally are HIV positive.⁷ Interferon γ (IFN γ) cytokine assays such as QuantiFERON-TB Gold or the established Mantoux test are needed. Imaging of the lesion is achieved by CT and/or upper GI endoscopy during which a biopsy is done. If a biopsy is not possible, or not contributory to formulating a diagnosis, then an endoscopic ultrasound – guided fine needle aspirate is required.^{3–8} Lesions in GI TB may be ulcerative, hypertrophic or ulcerohypertrophic. The ulcerative form is seen commonly in oesophageal TB; typically it is a shallow solitary ulcer with a grey, purulent, cavitating base and irregular, rolled edges. Multiple biopsies are taken from the edge of ulcers for histopathological analysis. The 'typical' ulcer of oesophageal TB is similar to that of oesophageal carcinoma.^{4–9} The most cited treatment protocol, which is standard antituberculous bacteriostatic chemotherapy, was initiated in our patient. Follow-up with upper GI endoscopy approximately 6 months after the initial symptom presentation is advised. In our patient, this showed regression of the solitary oesophageal ulcer, which coincided with symptom cessation. The role of surgery must not be disregarded, as it can be indicated for management of complications such as perforation and oesophageo-tracheal fistula formation. Endoscopic stenting or bronchoscopic stenting may be preferred to surgery in some cases.¹⁰

In conclusion, the prevalence of GI, in particular oesophageal TB is low. However, its prevalence in developed nations may increase due to the concomitant increase

of HIV, which can be complicated by tuberculous infection. Its presence must also be considered in the immunocompromised and homeless. Due to the scarcity of cases, clinical, endoscopic and radiological diagnostic criteria are poorly defined and thus it is the onus of clinicians to be aware of the salient features of this condition and formulate appropriate differential diagnoses. As oesophageal TB is responsive to antituberculous chemotherapy, this should be initiated as soon as this infection is suspected.

Learning points

- ▶ TB is re-emerging in less affluent areas of the UK. The GI system is the sixth most common extrapulmonary site to be affected.
- ▶ Oesophageal TB is extremely rare, with an incidence of 0.2–1% of GI TB cases.
- ▶ Clinical features of oesophageal TB include dysphagia, odynophagia, retrosternal/epigastric chest pain and haematemesis.
- ▶ Important differential diagnoses to consider are oesophageal viral infections such as cytomegalovirus, Crohn's disease and oesophageal carcinoma.
- ▶ A definite diagnosis is achieved by haematological tests (eg, ESR, CRP, HIV testing), TB specific tests (QuantiFERON-TB Gold or Mantoux) and biopsy with imaging (upper GI endoscopy, CT or endoscopic ultrasound – guided fine needle aspirate).
- ▶ Management typically involves a standard antituberculous chemotherapy regime and follow-up with upper GI endoscopy, recommended at 6 months.

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Competing interests None.

Patient consent Obtained.

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