Primary oesophageal tuberculosis: A rare entity

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ABSTRACT

Tuberculous infection of the oesophagus is rare and primary oesophageal tuberculosis is seen even more infrequently. We report a case of oesophageal tuberculosis in a 32-year-old female patient who presented to us with odynophagia and weight loss. Endoscopy showed a solitary ulcerative oesophageal lesion. Further investigation resulted in a diagnosis of oesophageal tuberculosis with no manifestations of tuberculosis elsewhere. She responded well to antitubercular treatment. This case was classified as primary oesophageal tuberculosis.

Key words: tuberculosis; oesophagus; dysphagia; granuloma

INTRODUCTION

Mycobacterial involvement of the oesophagus is very rare, constituting only about 0.3% of cases of gastrointestinal tuberculosis.[1] Oesophageal tuberculosis is generally secondary to mycobacterial disease elsewhere and only a few cases of possible primary oesophageal tuberculosis have ever been described.[2-6]

CASE REPORT

A 32 year-old female patient presented to us with a 4-week history of odynophagia, anorexia and 6 kg weight loss. There was no history of fever, night sweats, vomiting, haematemesis, cough, or sputum production. No history of caustic ingestion, smoking, alcohol or illicit drug abuse was present. She denied prior history of tuberculosis or exposure to it. Physical examination was unremarkable. Blood tests revealed normochromic normocytic anaemia (haemoglobin 10.4 g/dl) and an elevated erythrocyte sedimentation rate of 64 mm/hour. The white cell count was 10,600/mm^3 with a normal differential count and the platelet count was 345,000/mm^3. Liver and renal function tests were normal. Chest x-ray was unremarkable. Upper endoscopy revealed an ulcerative lesion 3-4 cm in size at the lower oesophagus, 36 cm from the incisors (Figure1). Pathological examination showed multiple epithelioid non-caseating granulomas without any evidence of malignancy (Figure 2). No acid-fast organisms were observed on staining and culture and special staining for fungi were negative. Polymerase chain reaction (PCR) assay of the specimen was positive for tubercle bacilli. A purified protein derivative skin test was positive after 48 hours. CT scan of the chest showed thickened oesophagus with ulceration in its distal third. There was no pulmonary lesion, or mediastinal/hilar lymphadenopathy. Serologic testing for human immunodeficiency virus (HIV) was negative. Colonoscopy with ileoscopy, barium meal follow through and CT scan abdomen were normal. The patient was given isoniazid, rifampicin, pyrazinamide and ethambutol for two months and isoniazid, rifampicin for another seven months. The dysphagia disappeared and she gained weight (8 kg). Follow up endoscopy disclosed complete disappearance of the oesophageal lesion.

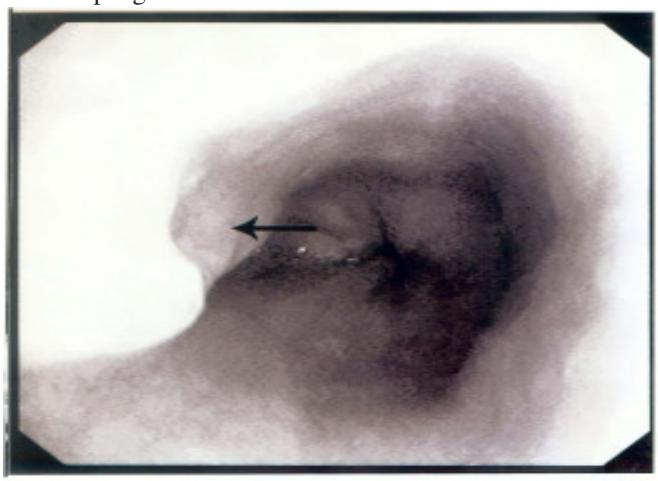

Fig 1: Upper endoscopy showing an ulcerative lesion (arrow) in the oesophagus at 36 cm.

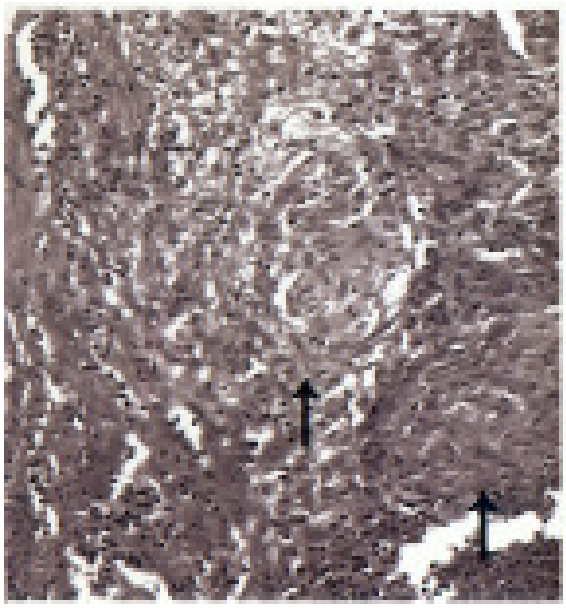

Fig 2: Photomicrograph of oesophageal biopsy specimen showing non ceaseating granuloma composed of epitheloid cells (arrows) (H&E × 40)

DISCUSSION

Oesophageal tuberculosis is very rare and primary oesophageal tuberculosis is seemingly even more exceptional.[2-7] Oesophageal tuberculosis is considered primary when there is no other detectable tubercular site and secondary when the oesophagus is involved by spread from adjacent structures.[3] The factors which protect the oesophagus from tubercular infection include the presence of a stratified squamous epithelial

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lining, tubular structure, coating of the mucosa with saliva and mucus, and rapid peristaltic transit of swallowed substances that prevents stasis and mucosal invasion by organisms.[8] The most common mechanism for secondary involvement of the oesophagus is reactivation in mediastinal lymph nodes and erosion into the oesophagus. It can also occur via local extension from pharyngeal or laryngeal disease, broncho-oesophageal fistulae, infected aortic aneurysms or infected bone, retrograde flow of lymphatic drainage from infected paratracheal, subcranial or peribronchial lymph nodes, and haematogenous spread.[8,9] The first case of primary oesophageal tuberculosis was reported by Torek in 1931.[10] Since then only a few cases have been reported.[2-6] The most common symptom is dysphagia occurring in over 90% of cases.[6,7,11,12] Other common symptoms include odynophagia and retrosternal pain. Constitutional symptoms like fever, weight loss, and anorexia may also occur. Cough on swallowing suggests the development of a tracheo-oesophageal or broncho-oesophageal fistula.[12] Oesophageal tubercular ulcers have a tendency to bleed because they are deep and massive bleeding has been reported because of deep penetration of the ulcer into the aorta with resultant oesophagoaortic fistula.[13] Tuberculous lesions can occur in any segment of the oesophagus but are most common in the middle-third of the oesophagus because of its proximity to the mediastinal and hilar lymph nodes.[4,6,7,11,12] The most common form of presentation is the ulcerative form. The ulcers are typically multiple, large, and deep with sharp irregular margins.[14] Strictures and fistulae are also common.[12,14] A hypertrophic growth mimicking oesophageal cancer can also occur.[15,16] Occasionally, oesophageal tuberculosis manifests as hyperaemic patches and nodules in the distal oesophagus that are difficult to differentiate from peptic oesophagitis.[4] Diagnosis requires endoscopy along with biopsy and Ziehl-Neelsen staining and culture of the biopsy specimen.[8] Endoscopic biopsies are useful but reveal the classical granuloma in only 50% of cases, whereas acid fast bacilli are demonstrated in less than 25%.[12,17] Recently, cytology and PCR have also proven useful in cases where the initial biopsies showed non-specific changes.[18,19] A CT scan of the thorax and/or endoscopic ultrasonography is mandatory for documentation of the secondary nature of the disease.[12] Oesophageal carcinoma, fungal and viral infections, ingestion of caustic material, syphilis and Crohn's disease should be considered in the differential diagnosis. A diagnostic dilemma may result in an unneccessary oesophagectomy.[16] The outcome is generally good and antituberculous chemotherapy alone is largely successful; surgery is reserved for complications including non-healing tracheo-oesophageal or broncho-oesophageal fistulae, stricture, or bleeding from an aorto-oesophageal fistula.[7,11,12] A 6-to-9 month course of antituberculous chemotherapy is sufficient with a regimen consisting of four first-line drugs, namely isoniazid, rifampicin, ethambutol, and pyrazinamide for the initial two months, and then continuing with isoniazid and rifampicin for another four to seven months.[7,12,20] In our patient, the histopathology, positive PCR, positive tuberculin skin test, inability to demonstrate tuberculosis elsewhere and complete response to anti-tuberculous treatment affirmed a diagnosis of primary tuberculosis of the oesophagus.

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