Oesophageal tuberculosis mimicking a tumour during treatment for nodal tuberculosis

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Abstract

A patient with cervical lymph node tuberculosis developed a tubercular ulcer in the oesophagus eight weeks after starting treatment. This was probably due to a drug related hypersensitivity reaction in an adjacent mediastinal lymph node and subsided with continued treatment.

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Despite the frequent involvement of other mediastinal structures,¹ oesophageal tuberculosis is rare. We describe a patient with tuberculous ulceration of the oesophagus mimicking a mass lesion which appeared eight weeks after institution of antituberculous treatment.

Case report

A 26 year old housewife presented with general ill health and fatigue for 2-3 months. She

had no documented fever, anorexia, or loss of weight, but had been aware of a gland in the neck for the preceding year. In 1985 she had pleurisy and had been treated with rifampicin, isoniazid, and ethambutol. She took the treatment irregularly for only five months. On examination she weighed 45 kg and was 134 cm tall. Two right posterior cervical lymph nodes were palpable and one was 1.5 cm in diameter, soft, fluctuant, and non-tender. There were no other palpable lymph nodes. The abdomen, cardiovascular, and respiratory systems were normal. Investigations revealed a haemoglobin level of 11.2 g/dl with a normal total and differential cell count. Erythrocyte sedimentation rate was 20 mm in the first hour (Wintrobe). Blood film showed predominantly normocytic normochromic erythrocytes with a few macrocytes. Serum biochemistry tests gave normal results. The Mantoux test with 10 tuberculin units was 18 × 18 mm. The chest radiograph was normal. Fine needle aspiration cytology of the cervical lymph nodes yielded white particulate material composed of multiple epithelioid cell granulomas, giant cells, and reactive lymphoid cells. Concentration smear and culture of the material did not yield acid fast bacilli. She was started on rifampicin 450 mg, isoniazid 300 mg, and pyrazinamide 1200 mg daily.

Eight weeks later she felt better. The cervical lymphadenopathy had subsided but she complained of transient moderate dysphagia which occurred only during the main meal and was not related to the type of food. Oesophagoscopy revealed an ulcer, 1.2 cm in diameter, with rolled up margins. It felt hard when probed with biopsy forceps. The ulcer crater was situated on a smooth bulge, about 2.5-3 cm in diameter, in the mid oesophagus.

Figure 1 A, B Barium oesophagograms showing the mass lesion and an ulcer crater in two views. There is no dilatation of the oesophagus above the lesion.

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Barium swallow revealed an ulcerated mass lesion in the oesophagus (fig 1A, B) and a biopsy of the lesion showed extensive mixed inflammatory exudate, necrosis, and vascular proliferation. She received amoxycillin 500 mg eight hourly for five days. Later, when *Candida* species were seen on PAS stain, she was given ketoconazole 400 mg daily for 10 days.

Repeat oesophagoscopy after one week revealed the same findings except that the ulcer crater looked less angry. Needle aspiration of the smooth bulge, away from the ulcer crater, yielded whitish particulate material which showed only benign squamous epithelial cells and erythrocytes. There were no inflammatory cells, *Candida* hyphae, or acid fast bacilli in the smears. Biopsy from the ulcer bed revealed epithelioid granulomas with Langhans giant cells (fig 2), but acid fast bacilli were not found. Antituberculous treatment was continued.

Four weeks later endoscopic examination revealed a soft bulge at 25 cm with a residual dimple at the site of the ulcer which had healed completely. Fluoroscopy during a barium swallow revealed persistence of a soft tissue shadow causing extrinsic compression of the oesophagus. Repeat endoscopy and barium swallow nine months after institution of antituberculous treatment showed that the mass lesion and mucosal abnormality had disappeared. Antituberculous treatment was continued for a total duration of 18 months, with pyrazinamide for the first three months only.

Discussion

Tuberculosis of the oesophagus is rare.² In this patient symptoms attributable to the oesophageal disorder occurred almost eight weeks after the institution of antituberculous treatment. It is therefore difficult to hypothesise that tuberculosis continued to spread during this period. The mediastinum can

only be incompletely evaluated by readily available methods and the chest radiographs were normal; nevertheless it is most likely that pre-existing nodal tuberculosis in the mediastinum³ caused these glands to enlarge on the antituberculous drugs. This has been well documented in the literature during treatment of superficial tuberculous lymphadenitis⁴ and there is no reason why tuberculous mediastinal lymph nodes should not also enlarge.

A chest radiograph and computed tomography of the thorax timed to show the mediastinal condition could have helped to elucidate the sequence of events.⁵ However, chest radiographs have been reported to be normal in other patients with dysphagia caused by oesophageal tuberculosis.²

Hypersensitivity to tuberculo-protein has been thought to be the likely explanation for the increase in size of pre-existing nodes or the appearance of fresh nodes during therapy,⁴ and may also be related to the bacterial load. Fluctuation of enlarged lymph nodes and discharging sinuses during treatment have also been documented.⁴ Oesophageal tuberculosis, however, usually results from direct extension of caseating subcarinal lymph nodes.⁶ The stratified squamous epithelium and the short transit time through it render the normal oesophagus resistant to invasion by swallowed tubercle bacilli.^{6,7} Two striking observations in this patient, however, need to be highlighted. Firstly, the severity of the reaction to tuberculo-protein which led to ulceration of the mucosal surface of the otherwise relatively spared oesophagus⁴ eight weeks after starting treatment and, secondly, the progression of the reaction in the mediastinal nodes in the face of resolution of the cervical lymphadenopathy.⁸

Oesophageal tubercular ulceration caused by direct inoculation of swallowed acid fast bacilli does not appear to be likely in this patient. The other mechanism that could explain the ulceration is obstruction by the nodal mass leading to a local hold up of swallowed drugs with resultant mucosal damage followed by secondary infection with *Candida*.

Figure 2 Photomicrograph of oesophageal biopsy (haematoxylin and eosin) showing epithelioid cell granulomas, Langhans giant cells, and lymphocytic infiltrate typical of tuberculosis.

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