## References

1. Watson JG, Bird AG. Laboratory investigations and normal value. In: Handbook of Immunological Investigations in Children. Wright 1990 : 214-51.

2. Stephan JL, Viekava JL, Le Deist F, Blanche S, Donadieu J, De Saint-Basille G, Duran DY, Griscell C, Fischer A. Severe combined immunodeficiency ; A retrospective single centre study of clinical representation and outcome in 117 patients. J Paed 1993;123 : 564-72.

3. Le Deist F, Fischer A. Primary T cell Immunology. In: Clinical Immunology, Principles and Practice ed. Rich RR, Fleisher TA, Schwartz BD, Shearr WT, Strober W. Mosby, 1996 : 637-61.


# Oesophageal Tuberculosis – An Unusual Site of Tuberculous Infection

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## Summary

A female patient presenting with post-prandial epigastric pain and weight loss was diagnosed to have oesophageal tuberculosis by endoscopic biopsy. She responded well to standard anti-tuberculosis treatment.


*Key Words:* Tuberculosis, Oesophagus

## Introduction

Tuberculosis is a very ancient disease which is certain to have been present before the beginning of recorded history. In the past decade, there has been a resurgence of tuberculosis in many parts of the world attributed to a number of factors, including the association with HIV infection/AIDS. Tuberculosis remains a major health problem in Malaysia despite effective modern chemotherapy: it is the leading cause of death from any single infectious disease and over 10,000 new cases are treated annually by government hospitals. Tuberculosis is capable of mimicking many disease processes and may involve virtually any anatomical structure. We report a case of oesophageal tuberculosis; to the best of our knowledge, this is the first such case reported in a Malaysian patient.

## Case Report

NAB, an 18-year-old Malay girl, was admitted to the surgical ward in January 1995 with a 3-week history of post-prandial epigastric pain and weight loss of 4 kg. There were no other symptoms and no past medical history of note. She worked in a factory and was a non-smoker. Her father had suffered from pulmonary tuberculosis in 1980 and had completed treatment. On examination, she weighed 46.5 kg and had 2 BCG scars. There were no positive findings apart from mild epigastric tenderness on palpation of the abdomen. She was treated symptomatically for gastritis and discharged the following day. Outpatient upper gastrointestinal endoscopy in February 1995 revealed an area of irregularity about 25 cm from the incisor teeth on the right posterior wall of the

oesophagus, 2 cm in length and occupying about one-quarter of the circumference of the oesophageal lumen. No other abnormality was seen. Biopsy of the lesion then was inconclusive, showing inflamed granulation tissue heavily infiltrated with plasma cells, lymphocytes and polymorphs. Barium swallow done in late February 1995 showed a constant area of irregularity and ulceration in the oesophagus at the level of the fourth and fifth thoracic vertebrae (Fig. 1). A repeat upper gastrointestinal endoscopic examination done in early April 1995 showed the same lesion and biopsy revealed inflamed tissue with areas of caseating epitheloid granuloma containing multinucleated giant cells, one of which was of Langhan type. Photomicrographs of part of the oesophageal biopsy, with epitheloid granuloma formation, are shown in Figure 2.

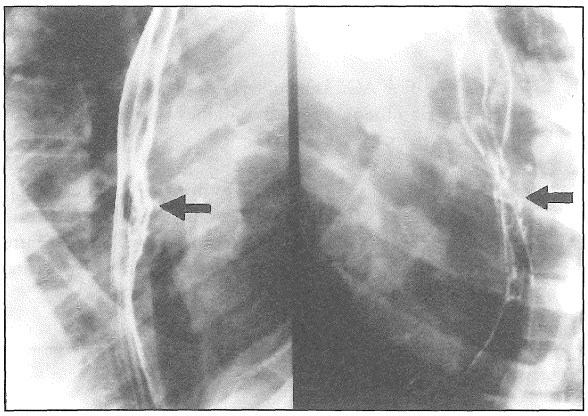

**Fig. 1: Barium swallow showing an area of ulceration (arrowed) at the level of T4/T5**

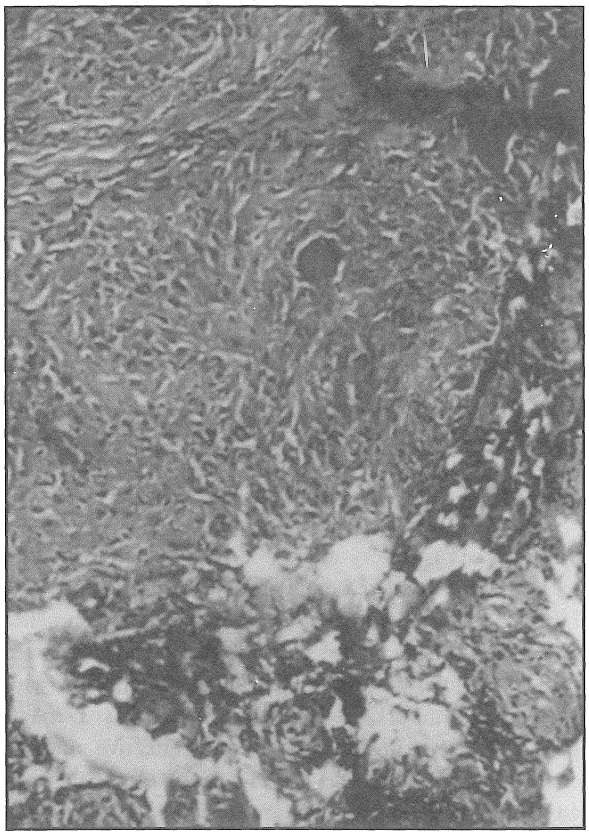

**Fig. 2: Oesophageal biopsy showing epitheloid granuloma, with Langhans giant cell**

In May 1995, she was referred to the Chest Clinic for further management. Her chest X-ray was normal with no features suggestive of pulmonary tuberculosis or mediastinal lymphadenopathy. The Mantoux test (10 T.U.) reading was 21 mm and erythrocyte sedimentation rate was 86 mm/hr. Three gastric lavage specimens were negative for acid fast bacilli on Ziehl-Neelsen staining and culture on Lowenstein-Jensen medium. She was commenced on daily supervised streptomycin, isoniazid, rifampicin and pyrazinamide and was asymptomatic on follow-up 8 weeks later. Upper gastrointestinal endoscopy done the next day revealed no obvious oesophageal lesion and treatment was changed to biweekly streptomycin, isoniazid and rifampicin. On review in November 1995 she had gained 11.5 kg over her pre-treatment weight and anti-tuberculosis drugs were stopped. Since then, she has remained well and asymptomatic on follow-up.

## Discussion

Tuberculosis of the oesophagus is remarkably rare, even where the disease is endemic. There have been sporadic case reports but until 1987 only 20 cases had been reported in the Western literature[1]. One of the largest recent series consists of 11 cases seen over a 18-year period in South Africa[2]. In most cases, the symptoms are similar to those of oesophageal carcinoma, a much more common cause of oesophageal lesion. The majority of patients present with dysphagia, although a smaller proportion may have haemetemesis, hoarseness of voice or cough. Seven out of the 11 cases from the South African series had chest X-ray abnormalities either suggestive of pulmonary

tuberculosis or mediastinal, paratracheal or thoracic outlet mass.

Oesophageal tuberculosis may be caused by swallowing infected sputum, direct spread from lung, mediastinal nodes or spine, or by retrograde lymphatic spread[1]. The rarity of oesophageal tuberculosis can be partly explained by protective mechanisms such as the stratified squamous epithelial lining, the tubular structure and short transit time through the oesophagus, which prevent prolonged contact of swallowed infectious material with the mucosa. With regard to spread of tuberculosis from mediastinal lymph nodes to the oesophagus, it has been postulated that caseous nodes causing oesophageal compression may erode into the lumen causing oesophageal ulceration. By the same mechanism, tuberculous mediastinal nodes can erode into the bronchus and tracheo-oesophageal fistula is also a known complication of tuberculous mediastinal lymphadenopathy[3].

The diagnosis of oesophageal tuberculosis depends on demonstration of caseous granuloma or acid fast bacilli in the lesion. Some authors have resorted to culturing the biopsy to establish the diagnosis[2]. In cases where there is strong clinical suspicion but no bacteriological or histological confirmation, a trial of anti-tuberculosis therapy may be useful. The treatment of oesophageal tuberculosis is primarily medical and a full course of combination anti-tuberculosis drug therapy should be given.

Although there was no bacteriological confirmation of tuberculosis in our case, the diagnosis was supported by the history of contact with tuberculosis, positive tuberculin test, histological evidence of caseating granuloma on oesophageal biopsy and rapid response to anti-tuberculosis treatment. Her chest X-ray and barium swallow did not indicate the presence of pulmonary tuberculosis or mediastinal lymphadenopathy but a small focus may have been missed. A C.T. scan of the chest would have been helpful but was not done in this patient. This case illustrates well the multi-disciplinary approach often necessary in the management of extrapulmonary tuberculosis since this patient benefited from discussion and collaboration between the Surgeon, Radiologist, Pathologist and Physician.

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## References

1. Gupta SP, Arora A, Bhargava DK. An unusual presentation of oesophageal tuberculosis. Tubercle Lung Dis 1992;73 : 174-6.

2. Mokoena T, Shama DM, Ngakane H, Bryer JV. Oesophageal tuberculosis: a review of eleven cases. Postgrad Med J 1992; 68 : 110-5.

3. Lee JH, Shin DH, Kang KW, Park SS, Lee DH. The medical treatment of tracheo-oesophageal fistula. Tubercle Lung Dis 1992;73 : 177-9.