TUBERCULOSIS OF THE OESOPHAGUS

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Tuberculosis of the oesophagus is a rare entity with variable presentations[1,2]. Both primary and secondary involvement of the oesophagus have been reported[3]. The diagnosis is almost always made on histopathology and response to anti-tuberculosis therapy is good. We present a case of primary tuberculosis of the oesophagus which presented with dysphagia.

CASE REPORT

A 65 years old female was referred to this Centre with 6 months history of progressive dysphagia for liquid and solids both and heartburn. She also complained of fever with chills (100.103°F) since 6 weeks especially at night. She denied any history of weight loss, nausea, vomiting or G.I. bleeding. She was addicted to pan with tobacco (betel leaf with lime, catechu and areca nut topped with tobacco leaves) and was taking 30-40 pans daily since 40 years. She gave a history of being treated with antibiotics and H2 receptor antagonists for fever and heartburn but with no relief. Physical examination revealed an elderly lady who weighed 46 kg, was normotensive and had no lymphadenopathy or visceromegaly. Barium meal showed a small hiatus hernia with gastro-oesophageal reflux and tertiary waves in the oesophagus; a small diverticulum was noted in the 2nd part of the duodenum. In view of persistent dysphagia she underwent an upper G.I. endoscopy with Olympus 2T10 scope, without any sedation. An ulcerating growth with curled up edges was seen at 25 cms from the incisors. The growth was bypassed with the scope and rest of the oesophagus. Stomach and duodenum were normal. Multiple biopsies were taken from the edge of ulcerated growth. Histopathology of the lesion showed edge of an ulcer with few suspected malignant cells and a repeat biopsy was therefore suggested. Repeat examination and biopsy a week later on H&E showed oesophageal mucosa covered with hyperplastic epithelium. The underlying flbrovascular connective tissue showed mixed inflammatory infiltrate including oesinophils. There were scattered epitheloid cells and few giant cells of the langerhans and foreign body type (Figure).

FIGURE. H&E stain of oesophagus showing a polypoid lesion covered on two sides by stratified squamous epithelium. A granuloma with a multinucleated and langerhans type of giant cell is seen at its base.

Neutrophils and extravasated red blood cells were seen on surface epitheium. Acid fast bacilli were seen with Ziehi-Neelsen stain and a diagnosis of chronic grannulomatous inflammation consistent with tuberculosis was thus made. Haematology showed a haemoglobin of 11.4 g/dl, total white cells count was 7,300/mm with 82% neutrophils, 12% lymphocytes, 4% eosinophils and 2% monocytes. ESR was 105 mm in first hour. Liver function tests were within normal limits with total proteins of 7.7 G/dl (4.6 albumin). Random sugar was 115 mg, urea 22 and creatinine 0.9 mg, x-ray chest was normal. Her fever persisted, especially in the evening and at night, alongwith weakness. She also developed oedema on both ankles and diarrhoea (6-7 BM/day). She was started on tablet rimactazid 450 mg and myambutol 800 mg once a day after meal. She became afebrile after 3 weeks of therapy and her dysphagia also started to subside. Three months later she was swallowing normally and was afebrile. Myambutol was stopped but rimactazid was continued, Five months later she developed breathlessness which was more on exertion and also had bilateral ankle oedema. A mild diuretic (moduritic) was given on alternative days. Chest examination did not reveal any abnormality. At the end of one year anti-TB treatment was stopped. At this time she had no dysphagia, no oedema (still on diuretics) and weighed 52 kg. A repeat endoscopy showed a non9ulcerated raised area at 25 cms which was biopsied. Rest of the examination was essentially normal. Histopathology revealed a stratified squamous epithelium with no grannuloma. Last follow-up, 9 months after the cessation of therapy, has shown no recurrence of symptoms.

DISCUSSION

Tuberculosis of the oesophagus is a rare entity especially the primary involvement of the oesophagus. This is due to the protective mechanism of this organ which includes constant peristalsis, gravitational pull, coating of bacilli by the saliva and mucus and the protective role of the stratified squamous epithelium. All these factors prevent stasis and mucosal invasion in the oesophagus[4-6]. Moreover the encapsulated mycobacterium only becomes active whenever and wherever it finds a feasible environmen[7]. A preformed stricture, ulcer, growth or fungal oesophagitis often act as aground for the innoculation of the ingested mycobacterium8, resulting in primary tuberculosis of the oesophagus. Secondary involvement of the oesophagus though more common, is often a local spread from a neighbouring organ like lung, lymph node or larynx[3]. Any part of the oesophagus can be affected but middle 1/3 is more involved. Types include ulcerative, hyperplastic, granular and neoplastic[1,2]. Ulcerative lesion is more common, which is usually single but can be multiple. The presentation depends upon the type of the lesion and its length of involvement. An asymptomatic presentation has also been reported[9]. Though endoscopy or barium studies are helpful in defining the lesion but biopsy is mandatory. Culture of AFB from the tissue sample and presence of AFB on Ziel-Neelsen staining give variable results[4]. Histopathology of the tissue is therelore the only diagnostic tool on which diagnosis and treatment can be evaluated. Suspected malignant cells and the edge of the ulcer seen on histology in our case alongwith her presentation of dysphagia and a growth on endoscopy were all going in favour of a carcinoma and would have subjected her to unnecessary surgery. In developing countries where tuberculosis is common, any suspected lesion anywhere in the GIT should always be biopsied and inconclusive reports or small tissue sample should always be supplemented by a repeat biopsy to confirm the diagnosis.

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