GASTRIC TUBERCULOSIS

(A Case Report)

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Introduction

Gastric tuberculosis (GTB) accounts for only 1-2 per cent of tuberculosis of the gastro-intestinal tract [1]. Most of these cases are secondary to pulmonary tuberculosis. A case of primary gastric tuberculosis who presented as prolonged pyrexia is described.

Case Report

A 58 years old male patient presented with two months history of intermittent fever, epigastric pain and vomiting. There was no history of cough, altered bowel habits or gastro intestinal bleeding. Clinical examination revealed an emaciated individual weighing 42 kg (ideal weight 68 kg) with pallor, pedal and sacral edema. There was epigastric tenderness but no organomegaly. Examination of other systems was unremarkable. Erythrocyte sedimentation rate was 64 mm fall first hour with haemoglobin of 8.8 gm% (normochromic normocyte). Biochemical parameters, chest X-ray and ultrasound of the abdomen were normal. Blood culture sterile, widal test negative and ELISA for HIV was negative. Mantoux test was 15x12 mm. Upper gastrointestinal endoscopy showed three well defined rounded submucosal lesions measuring 1 to 3 cm in diameter in the corpus of the stomach along the lesser curve. Endoscopic biopsy from these lesions revealed non-caseating granulomas in the lamina propria with increase in lymphomononuclear cells. Crush smears of the endoscopic biopsies were teeming with acid fast bacilli. CT Scan of the abdomen done with oral and intravenous contrast showed intramural polypoid lesions in the lesser curvature of the stomach (Fig 1). Anti-tuberculosis treatment was started and at three months follow-up the individual was asymptomatic and had gained 12 kg.

Discussion

The ileo-cecal region is the most common site of involvement of gastrointestinal tuberculosis. GTB is a rare clinical entity. Good reported an incidence of 0.03% in routine autopsies and 0.3% in the autopsies of patients with pulmonary tuberculosis [2].

The relative sparing of the stomach has been explained by the paucity of lymphoid tissue, low gastric

Fig. 1 : CT scan of the abdomen with oral contrast in the stomach showing intra-mural polypoid filling defects (arrows) along the lesser curvature

pH and the rapid emptying of gastric contents [3]. GTB usually develops secondary to pulmonary tuberculosis. In a review of literature, Broders found evidence of pulmonary tuberculosis in 34 out of 49 cases of GTB [4]. Besides direct mucosal invasion of swallowed bacilli, the other routes of infection include retrograde spread from celiac lymphnodes, haematogenous spread, direct extension or super - infection of a pre-existent gastric ulcer. There are very few reports of primary gastric tuberculosis i.e., without evidence of tuberculosis elsewhere [5].

The gastric antrum and the pre-pyloric area are the most common sites of involvement. Ulcerative lesions are by far the most common (80%) followed by hyper-

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trophic which present with linitus plastica. Other pathological types include ulcerative-hypertrophic, erosive gastritis and nodular. The clinical manifestations of GTB are varied. The patient may present with dyspeptic symptoms, gastric outlet obstruction, prolonged pyrexia or upper GI bleeding in various combinations. The diagnosis of GTB can be confirmed by demonstrating caseating granulomas and/or acid fast bacilli in the mucosa and sub-mucosa of the stomach. Conventional endoscopic biopsies may not provide adequate tissue for diagnosis, especially in the hypertrophic and nodular varieties of GTB. Endoscopic fine needle aspiration biopsy (FNAB) may be a better option as it can reach deeper tissues and is an effective modality in the diagnosis of abdominal tuberculosis.

GI tuberculosis responds well to chemotherapy. Although six months duration of therapy has been shown to be effective for GI tuberculosis, the duration of therapy is best individualised and guided by the response to treatment [7]. Surgery is usually required if

gastric outlet obstruction is present.

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