

Letters to the Editor

Duodenal tuberculosis – a continuing diagnostic challenge

Sir,
Duodenal tuberculosis is a difficult diagnosis to make for several reasons and instigation of appropriate treatment is often delayed. In comparison with the relative ease of diagnosis of pyloric tuberculosis described in the January 1990 issue of your *Journal*,¹ the following case illustrates some of the difficulties in diagnosis of duodenal tuberculosis.

A 45 year old Ethiopian man was referred for further management of a presumed peptic ulcer. He had presented 3 years earlier with epigastric pain and an upper gastrointestinal endoscopy had revealed mucosal ulceration in the pylorus and first part of duodenum. He was treated with cimetidine with some relief of his symptoms but recurrence of his symptoms prompted his physician to double the dose of cimetidine. He re-presented one year later with vomiting and weight loss and was referred to this hospital.

Systems review and physical examination were unremarkable. Investigations revealed an iron deficient anaemia, with a haemoglobin of 9.0 g/dl and a white cell count of 3000/mm. A chest radiograph was normal. A barium meal showed the duodenal cap and second part of the duodenum failing to expand. The mucosal folds were coarse and there was a constant niche of barium in the duodenal cap. A diagnosis of duodenal ulceration and duodenitis was made from these appearances. A repeat upper gastrointestinal endoscopy showed a normal oesophagus and stomach. The pylorus was irregular, and it was impossible to enter the duodenum. Maximum doses of cimetidine were continued. Two months later, he was readmitted with vomiting and a diagnosis of pyloric obstruction was made. At laparotomy, a tightly closed pylorus was found, with a posterior penetrating ulcer through the first part of the duodenum. It was impossible to separate the duodenal cap from a surrounding inflammatory mass. A Polya gastrectomy was performed, and the resected specimen sent for histology. Microscopy of the duodenal ulcer showed a granulomatous infiltration extending through the muscularis mucosa into the submucosa and to the surrounding lymph nodes. There were caseating granulomas with Langerhans giant cells, but the Ziehl-Nielsen stains on the specimen were negative. A diagnosis of tuberculosis was made at this point. Sputum and early morning urine samples remained negative for acid-fast bacilli, and a Mantoux test also was negative. Antituberculous chemotherapy was commenced, his condition improved, and he was discharged home.

The difficulty in the diagnosis of duodenal tuberculosis arises for several reasons and the true diagnosis is often delayed for several years in areas where tuberculosis is not common, or where clinical suspicion is low. As in our case, the disease most commonly presents with an insidious onset of upper abdominal discomfort, nausea and weight loss. Physical signs are few. Duodenal obstruction occurs late in the disease process. Rarely a mass may be felt, and even less commonly the first sign may be frank bleeding or perforation.²

Basic laboratory blood tests are non-specific. Although the Mantoux test may be positive, this has little relevance in endemic areas and, as the chest radiograph is frequently normal, the diagnosis of duodenal tuberculosis is not even considered. In the majority of cases of duodenal tuberculosis, the chest X-ray has been negative for active or old pulmonary tuberculosis, even in retrospect. Barium studies may show the site of the lesion, the presence of obstruction and involvement of other parts of the gastrointestinal tract by tuberculosis. The radiological features of duodenal tuberculosis include bulbar and post-bulbar ulceration, mucosal fold thickening, spasm and narrowing. Strictures, fistulae and sharp cut off areas of obstruction are also a feature.³

Upper gastrointestinal endoscopy is also non-specific with duodenal tuberculosis being misdiagnosed as duodenitis, peptic ulceration or even primary duodenal neoplasia. There may be stricture formation, stenosis and difficulty with cannulation of the duodenum. Fistula formation may give a clue to the diagnosis.

Identification of the bacillus has been negative in many reported series and many patients have been treated blind. Adequate response to chemotherapy has been considered to be good supportive diagnostic evidence.

Histology obtained by endoscopy and biopsy may also give rise to diagnostic difficulties. Often only non-specific inflammatory changes are present and, as the characteristic granulomas are in the submucosa, superficial mucosal biopsies will not yield a diagnostic result.⁴ Obviously direct visualization of the granulomas is impossible and biopsy may therefore be blind. The only way to reliably diagnose duodenal tuberculosis is to identify caseating granulomas with acid-fast bacilli,² and almost invariably, open operation with frozen section is required for positive diagnosis.

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References

1. Gupta, B., Mathew, S. & Bhalla, S. Pyloric obstruction due to gastric tuberculosis – an endoscopic diagnosis. *Postgrad Med J* 1990, **66**: 63–65.
2. Gleason, T., Prinz, R.A. Kirsch, E.P. *et al.* Tuberculosis of the duodenum. *Am J Gastroenterol* 1979, **72**: 36–40.
3. Gupta, S.K., Jain, A.K., Gupta, J.P., Agrawal, A.K. & Berry, K. Duodenal tuberculosis. *Clin Radiol* 1988, **39**: 159–161.
4. Thompson, J.N., Keshavarzian, A. & Rees, H. Duodenal tuberculosis. *J R Coll Surg Edin* 1984, **29**: 292–295.