Primary duodenal tuberculosis masquerading as chronic liver disease : an unusual presentation

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To the Editor,

Primary involvement of the duodenum by tuberculosis (TB) occurs infrequently. It poses diagnostic challenges owing to rarity, nonspecific clinical features and equivocal imaging results (1-3). As per literature, the usual presentation is in the lines of gastrointestinal obstruction, upper gastrointestinal bleeding or acid-peptic disorders (2-4). The diagnosis is sometimes established even intra-operatively during exploratory laparotomy (3). The authors describe an unusual case of a 43-years old woman with primary duodenal TB, with features mimicking chronic liver disease (CLD). She complained of diffuse pain abdomen and abdominal distension followed by bilateral leg swelling for about a month. Treated as a case of CLD elsewhere, but without any improvement, she was referred to our centre. There was no history of recurrent vomiting, diarrhoea, fever, weight loss, cough, dyspnoea, rashes or arthralgia. Past history of TB, diabetes and hypertension was absent. The general examination documented: temperature 37.2^{0}C, blood pressure 90/60 mmHg, pulse 96 beats/ minute, respiratory rate 20 breaths/ minute, oxygen saturation 100% at room air, moderate pallor and bilateral pitting pedal edema. The abdomen was soft on palpation with mild diffuse tenderness, and fluid thrill was present. Respiratory system examination detected dullness on percussion in bilateral infrascapular areas with decreased air entry. The remainder of the clinical examination was unremarkable.

Blood investigation revealed anaemia (hemoglobin 8.6 g/dL), hypoproteinemia (4.7 g/dL), and hypoalbuminaemia (1.8 g/dL). Renal function tests were normal, as were electrocardiography and echocardiography. Chest-radiograph showed bilateral blunting of costophrenic angles. Abdominal ultrasound revealed mesenteric lymphadenopathy and ascites. Contrast enhanced computed tomography (CECT) abdomen showed diffuse wall thickening involving second part of duodenum without significant luminal narrowing, intraabdominal discrete lymph nodes, ascites and peritoneal thickening. A tubercular etiology was suspected. On diagnostic paracentesis, a slightly turbid and straw-coloured ascitic fluid, with total cell count of 200 cells/mm^3 (92% lymphocytes, 8% polymorphs) and protein concentration of 0.8 g/dl was observed. The serum-ascites albumin gradient (SAAG) was < 1.1 gm/dL. The ascitic fluid adenosine deaminase (ADA) activity was 26 IU/L. Upper gastrointestinal endoscopy showed a normal esophagus with hypertrophy and nodularity in the second part of duodenum (Fig. 1A). Duodenal biopsy (endoscopic) showed caseating granuloma on histopathology, and stained positive for acid-fast bacilli by Ziehl-Neelsen method (Fig. 2A-2D). The diagnosis of duodenal TB was established.

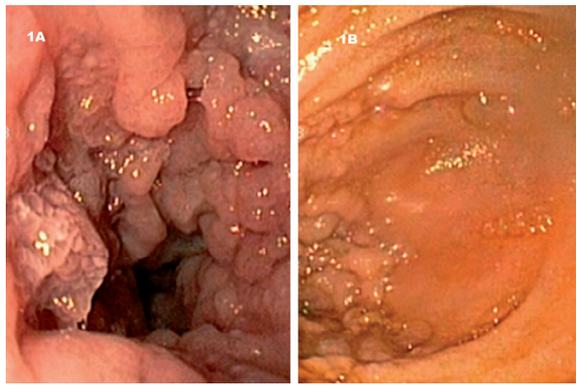

Figure 1. — Upper GI endoscopy showing hypertrophy and nodularity in second part of duodenum (1A) ; Repeat upper GI endoscopy after one month of anti-tubercular therapy showing decreasing hypertrophy of duodenum (1B).

Anti-tubercular drugs were started: rifampicin (450 mg), isoniazid (300 mg), ethambutol (800 mg) and pyrazinamide (1500 mg) under directly observed therapy, short course (DOTS) (category 1) for 2 months followed by a continuation phase of rifampicin and

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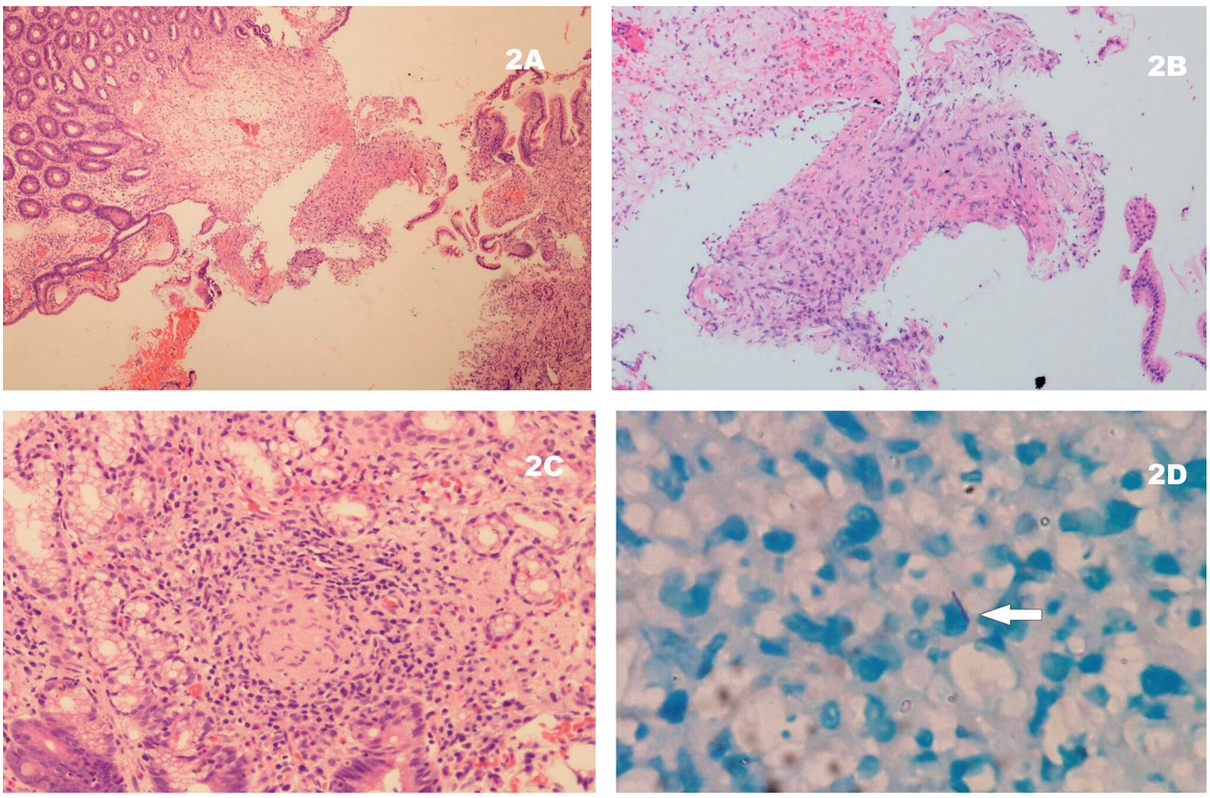

Figure 2. — Duodenal tissue with epithelioid granuloma in lamina propria (4x magnification) (A); Epithelioid granuloma (10x magnification) (B); Epithelioid granuloma with peripheral cuffing of lymphocytes with activity in glands (40x magnification) (C); Acid fast bacillus on Ziehl-Neelsen stain (D).

isoniazid for 4 months. Tablet prednisolone (40 mg/day) was added with the view of preventing stricture formation. On follow-up after 2 months, the patient was found to have improved greatly. Laboratory tests noted remarkable improvements in total serum protein (6.1 g/dL), serum albumin (3.5 g/dL) and hemoglobin (13.2 g/dL). Pedal edema had disappeared, and abdominal sonography revealed an absence of ascites. On repeat upper gastrointestinal endoscopy, earlier hypertrophy of the duodenal mucosa was found to have subsided considerably (Fig. 1B). Further, a repeat biopsy showed normal study.

Presentation of duodenal TB mimicking CLD is intriguing. It is plausible that duodenal hypertrophy causes decreased absorption area, resulting in malabsorption. The consequent hypoproteinemia may lead to ascites (transudative), pedal edema and also fluid collection in the pleural cavity – the sequelae with which our patient presented. These were subsequently corrected on anti-tubercular therapy. Thus, a high index of suspicion is necessary to diagnose unusual forms of duodenal TB. Contrary to popular perception, although the role of endoscopic biopsy in detecting tubercular involvement of the duodenum is often underrated (1, 2), it can be of value in facilitating the diagnosis (4,5).

Conflict of interests

* These are joint first authors who contributed equally. There are no conflicts of interests, financial or otherwise to disclose.

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