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Tuberculous Duodenal Stenosis: A Rare Case Report and Review of the Literature

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Abstract

Extrapulmonary tuberculosis accounts for a growing proportion of all tuberculosis cases, due to the increasing incidence of HIV. Tuberculosis of the gastro-duodenal region is rare. It may manifest as a syndrome of upper digestive stenosis, evolving against a background of progressive deterioration in general condition. Diagnosis is based on biopsy specimens taken during meticulous upper digestive endoscopy. Treatment is usually medical, but surgery may be necessary in some cases. We report a case of duodenal stenosis of tuberculous origin in a 15-year-old female patient, which posed diagnostic and therapeutic challenges.

Keywords: Tuberculosis - duodenal stenosis - diagnosis - treatment.

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INTRODUCTION

Tuberculosis is an infectious and contagious disease caused by Mycobacterium tuberculosis. It remains a public health problem in our populations [1]. It mainly affects the lungs, but can spread to other organs. Extrapulmonary forms of the disease (EPT) account for a growing proportion of all tuberculosis cases, due to the increasing incidence of HIV [2]. The polymorphism of clinical manifestations often makes diagnosis difficult [2]. Abdominal tuberculosis accounts for approximately 12.8% of extrapulmonary tuberculosis cases [3]. It can manifest in different ways, through visceral, peritoneal, lymph node, or luminal involvement [4]. Tuberculosis of the gastro-duodenal region is rare and accounts for between 0.5 and 2.5% of all gastrointestinal tuberculosis [5]. Diagnosis can be difficult, as it can resemble the clinical manifestations of a gastroduodenal ulcer or a malignant condition. Endoscopic and histological findings usually reveal nonspecific inflammatory changes [6]. Treatment is most often medical, but surgery may be necessary in some

cases. We report a case of upper digestive tract stenosis due to duodenal tuberculosis.

OBSERVATION

Miss KAAD, aged 15, with a history of appendectomy four years ago, no history of tuberculosis infection. She was referred to general and digestive surgery for abdominal pain localized in the epigastrium, which occurred suddenly, radiated to the back, and was exacerbated by eating. The pain was associated with delayed and bilious vomiting that had been progressing for four months in the context of a gradual deterioration in her general condition (35% loss of body weight). On admission, the examination revealed epigastric pain, epigastric distension, and splashing on an empty stomach, along with signs of moderate malnutrition and dehydration. The patient weighed 39 kg and was 1.59 m tall (BMI: 15.4). Her retroviral serology was negative. Upper gastrointestinal endoscopy revealed gastric stasis and stenosis of the first part of the duodenum (Figure 1). Histology of the endoscopic biopsy specimen revealed

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granulomatous duodenitis. Abdominal CT scan was consistent with gastric stasis and thickening of the wall of the first part of the duodenum (Figures 2 and 3). Biological tests revealed moderate hypochromic microcytic anemia at 8.3 g/dl, hyponatremia at 125 at 96 mmol/l, hypochloremia mmol/l, and hypoproteinemia at 36.6 g/l; renal, glycemic, lipasemic, and hepatic function tests were normal. We performed a transfusion and rebalancing of fluids, electrolytes, and proteins. Screening for pulmonary tuberculosis was negative. After a multidisciplinary consultation involving pulmonologists, gastroenterologists, pathologists, and visceral surgeons, we scheduled a laparotomy, which revealed a stenotic rearrangement in the first part of the duodenum, whitish granulations (Figure 4), ascites, and multiple lymphadenopathies.

Surgical treatment included a trans- and submesocolic gastrojejunal anastomosis (Figure 5), coupled with lymph node biopsies. Histological analysis of the biopsies showed caseous follicular granulomatous adenitis (Figure 6). The postoperative course was uneventful, allowing for gradual feeding on day 6 and discharge on day 8 postoperatively. She was referred to pulmonology, where antituberculosis treatment was instituted for a period of 6 months. She underwent regular follow-up, and after 5 months, she had gained 15 kg; with a satisfactory follow-up endoscopy showing persistent stenosis and a normal gastro-anastomotic orifice and jejunal loop (Figures 7 and 8).

ANNEX



Figure 1: FOGD: Stasis stomach, stenosis of the first part of the duodenum



Gauche



Figures 2 and 3: Abdominal computed tomography: Stasis stomach with thickening of the wall of the first part of the duodenum.

Gauche

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Figure 4: Intraoperative image: Whitish granulations



Figure 5: Intraoperative image: Transmesenteric gastrojejunal anastomosis.



Figure 6: Histological image of the surgical specimen: caseous follicular granulomatous adenitis.



Figures 7 and 8: Follow-up FOGD showing persistent stenosis and a normal gastro-anastomotic opening and jejunal loop

DISCUSSION

Abdominal tuberculosis accounts for approximately 12.8% of extrapulmonary tuberculosis cases [3]. Tuberculosis of the gastro-duodenal region is rare, accounting for between 0.5 and 2.5% of all gastrointestinal tuberculosis cases, and most often affects ileocecal region [5,7]. Several routes of the contamination are assumed: via the bloodstream secondary to bacteremia, but mainly via the lymphatic system; duodenal involvement is thought to occur via lymphatic retrograde spread from subacute supramesocolic adenitis [7]. The initial lesion affects the submucosa, where tuberculous follicles develop and undergo caseation. The evacuation of caseum into the intestinal lumen, through the necrotic mucosa, leads to ulceration [7]. The hypertrophic form is due to the histiolymphoplasmacytic granulomatous inflammatory process. It may be a mixed ulcerative-hypertrophic form with a predominance of one of the components. Lymph node involvement in the surrounding area is constant [7]; its presence is a fundamental element of the diagnosis. Duodenal tuberculosis can affect all four portions of the duodenum: D3-D4 in 73.3% [7], D2: 17% and D1: 8%, as in our case [7]. The clinical manifestations of duodenal tuberculosis are non-specific and may mimic those of other gastrointestinal diseases. They may present as dyspeptic symptoms [8], hemorrhage, perforation [9], or gastroduodenal stenosis [10,11]. Pain and vomiting are the main signs of duodenal tuberculosis and may be associated with fever, weight loss, and an epigastric mass [12]. Obstructive jaundice, internal gastrointestinal fistulas, or fistulas with the kidneys and aorta have been reported [13-16]. Active pulmonary tuberculosis can be observed in 10% to 50% of patients [17]. In our patient, abdominal pain, vomiting, and deterioration of general condition were the signs observed. She showed no signs of pulmonary tuberculosis. Diagnosis can be difficult, as the clinical manifestations may resemble those of a peptic ulcer, a malignant condition, or Crohn's disease, imaging is nonspecific, and endoscopic and histological findings

usually reveal non-specific inflammatory changes [6,17]. In our case, the patient presented with upper digestive stenosis syndrome. Abdominal computed tomography showed thickening of the wall of the first part of the duodenum and gastric stasis. Upper digestive endoscopy and pathological examination of the biopsy specimen revealed granulomatous duodenitis, suggesting duodenal tuberculosis or Crohn's disease [18]. Gastrointestinal tuberculosis generally responds to medical treatment; early diagnosis can avoid the need for surgery [19]. Antituberculosis treatment lasts six months according to the protocol: two months (rifampicin, isoniazid, ethambutol, pyrazinamide) and four months (rifampicin, isoniazid) [20]. Endoscopic balloon dilation of the stenosis is successful and is a good treatment option in combination with anti-tuberculosis drugs. Laparotomy is indicated to diagnose and treat complications of the disease [7]. Stenosis can be treated by resection or bypass [7]. Surgical treatment must be combined with antituberculosis treatment. In our case, gastroenterostomy combined with anti-tuberculosis treatment for a period of 6 months was instituted, with satisfactory medium-term results.

CONCLUSION

Primary duodenal tuberculosis is rare. It poses a diagnostic problem and can present as a syndrome of upper digestive tract stenosis. Endoscopic biopsy histology can raise suspicion of the condition. The prognosis is favorable with a combination of surgical and medical treatment.

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