

Case Report

Duodenal tuberculosis mimicking superior mesenteric artery syndrome

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ABSTRACT

Tuberculosis of stomach and duodenum is quite uncommon. Its preoperative diagnosis still remains a challenging problem for surgeons. Herein, we report the case of isolated abdominal tuberculosis in a 28 years old male presenting with features of gastric outlet obstruction due to stricture in duodenum. Oesophagogastroscope revealed doubtful extrinsic compression of first part of duodenum with mild gastritis. CECT abdomen showed pronounced dilatation of stomach, 1st and 2nd part of duodenum with narrowing and compression of 3rd part of duodenum between superior mesenteric artery and aorta; suggesting superior mesenteric artery syndrome (SMA). He was taken up for exploration which revealed peritoneal, small and large bowel mesentery and mesocolon seedlings. Mesenteric lymphadenopathy was seen and duodenum showed narrowing between 1st and 2nd part of duodenum with proximal dilated and distal narrow duodenum without any compression by SMA. Duodeno-duodenostomy was done. Histopathological examination of biopsied mesenteric lymph node was consistent with the features of tuberculosis. Patient was started on anti-tubercular therapy. His symptoms were relieved and he gained weight rapidly.

Keywords: Gastrointestinal, Superior mesenteric artery, Tuberculosis

INTRODUCTION

Abdominal tuberculosis is the sixth most common site of extra pulmonary tuberculosis.¹ TB may affect any portion of the GI tract however, in 85% of cases ileocecal region is affected whereas gastroduodenal region is involve only 0.3-2.3% of TB cases of abdominal TB.² When gastroduodenal TB is seen without any other lesion of TB in body it is known as primary gastroduodenal TB, which is very rare and only few cases had been reported in the literature.^{3,4}

Duodenal obstruction due to TB is very rare and needs high index of suspicions for diagnosis; and usually it is suspected on intraoperative findings.⁵ Strictures of duodenum are very rare and could be due to tuberculosis. Superior mesenteric artery compression has been suggested as a cause of obstruction to the third part of duodenum.

CASE REPORT

Twenty eight years old male presented to the emergency department with complain of intermittent colicky pain all over the abdomen of four months duration with aggravation after food intake. This forced him to reduce his food intake which was mainly liquid. During past 10 days he had 4-5 daily episodes of non-bilious, non-projectile vomiting. Patient had history of constipation since last 2 months and loss of appetite and significant loss of weight (13kgs) during last 4 months. There was no history of fever, cough, evening rise of temperature, hematemesis or malena. Patient was diagnosed to have peptic ulcer disease, 8 years back for which he had taken conservative treatment for one month and was relieved. Patient had no history of tuberculosis (TB) in the past and no contact history of TB.

Examination revealed a grossly malnourished patient with BMI of 16.6 kg/m². His vital signs were within normal limits. Examination of abdomen revealed visible

peristalsis from left to right in epigastric region but no visible mass or dilated veins. There was no hepatosplenomegaly or palpable lump. No free fluid was detected in peritoneal cavity.

Laboratory evaluation was essentially normal except for Serum electrolytes which showed hyponatraemia (134 mmol/lit.), hypokalaemia (3.4 mmol/lit) and elevated bicarbonate levels (31 mmol/lit.). He tested negative for HIV. Montoux's test was negative. X-Ray chest did not show active or healed tuberculosis. X-Ray erect abdomen showed dilated gas filled stomach. Ultrasonography (abdomen and pelvis) was suggestive of dilated stomach and duodenum which were extending up to right Iliac fossa with air fluid levels but normal peristalsis.

Oesophagogastrosocopy revealed doubtful extrinsic compression of first part of duodenum with mild gastritis. Upper GI scope could not be negotiated beyond first part of duodenum and washing were negative for acid fast bacilli.

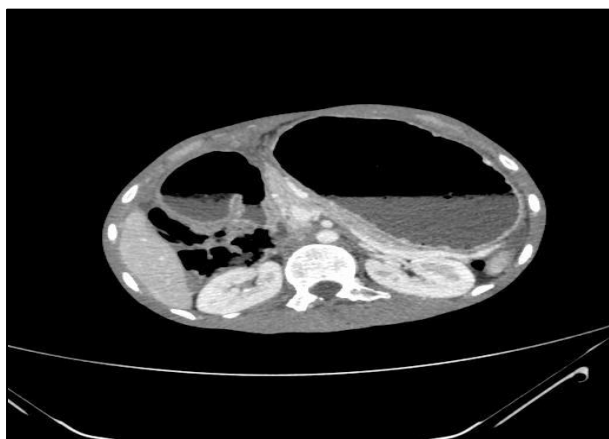


Figure 1: Axial contrast enhanced CT image of dilated stomach.



Figure 2: Sagittal reformatted image narrowed aorto mesenteric angle and distance

CT scan (abdomen and pelvis) with contrast revealed pronounced dilatation of stomach (Figure 1), 1st and 2nd part of duodenum with narrowing and compression of 3rd part of duodenum between SMA and aorta (Figure 2). The aorto-mesenteric angle (AO) and AO distance were 10 degree and 6mm respectively. Diagnosis of superior mesenteric artery (SMA) syndrome was suggested.

Exploratory laparotomy was undertaken with diagnosis of SMA syndrome. On exploration, parietal peritoneum was found studded with tubercles and contents of peritoneal cavity appeared as cocoondue to filmsy adhesions (Figure 3). Area of stomach, duodenum, upper jejunum and right half of transverse colon was defined after careful adhesiolysis. Multiple tubercles were seen in mesentery, mesocolon, over small bowel, transverse colon and peritoneum. Mesenteric lymphadenopathy was noted.

No compression by SMA was detected and we were surprised to find duodenal narrowing between 1st and 2nd part of duodenum with proximal dilated and distal narrow duodenum (Figure 4). Duodeno-duodenostomy was done. HPE of an excised mesenteric lymph node showed fibroadipose tissue infiltrated by confluent granulomas which comprised of epithelioid histiocytes, Langerhan's type of giant cells and lymphocytes; although acid-fast bacilli could not be demonstrated.

Post-operative recovery was uneventful. He could tolerate oral feeds from third post-operative day, graduating from liquids to normal diet during next 3 days. He was started on anti-tubercular therapy (DOTS category I) consisting of isoniazid 5mg/kg, rifampicin 10mg/kg, pyrazinamide 25mg/kg and ethambutol 15mg/kg 3 days per week. It is proposed to follow regimen of 2 months of intensive phase and 4 months of continuation phase. Patient has responded well after surgery and was completely relieved of the symptoms and has gained 12 kgs within a short period of 2 months. Repeat CECT performed showed normal aorto-mesenteric angle and distance (Figure 5 and 6).



Figure 3: Tubercles seen over mesentery, mesocolon and transverse colon.

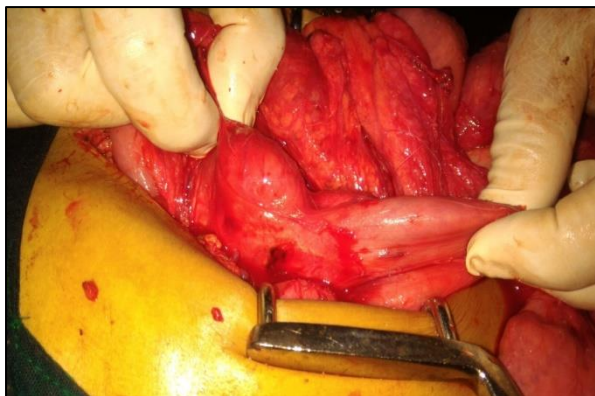


Figure 4: Narrowing of second part of duodenum.



Figure 5: Axial CT image of normal aorto-mesenteric distance at 3 month follow up.



Figure 6: Post-op sagittal reformatted image of normal AM angle at 3 month follow up.

The duodenum is an uncommon site for tuberculosis even in endemic areas like India. Third part is the most commonly affected site in the duodenum.⁶ Duodenal lesion may be intrinsic (ulcerative, hypertrophic or ulcero-hypertrophic) or extrinsic i.e. compression of duodenum by enlarged periduodenal lymph nodes from the outside or both.⁷ Increasing incidence has been reported in HIV infected individuals. Clinical manifestations of duodenal TB are non-specific and can

mimic that of other GI diseases. They may present with dyspeptic symptoms and features of peptic ulcer complications like bleed or perforation, gastric outlet obstruction, or duodenal obstruction. Pain (56.5%) and vomiting (60.8%) are common symptoms of duodenal TB which may be associated with fever, weight loss and palpable epigastric mass (33%). This patient presented with duodenal obstruction and did not have any evidence of pulmonary TB. Duodenal TB though rare should be considered in the differential diagnosis of GOO of recent origin in endemic countries like India.⁸ Surgery was required either for diagnosis or treatment in all but one of the 40 cases (97%) in two large series from India.^{9,10}

SMA syndrome, also known as Wilkie's syndrome is a rare pathology; first described by Rokitsky et al and has an incidence of 0.013-0.3%. The defining feature of this entity is upper gastrointestinal obstruction caused by compression of the third part of the duodenum between the SMA anteriorly and the aorta posteriorly.¹¹ The normal aorto-mesenteric angle has been previously noted as 38-65 degrees.¹² The normal distance from the aorta to the SMA at the duodenal crossing is 13-34 mm.¹² Diagnostic criteria for SMA syndrome include a decrease in the aorto-mesenteric angle to 6-16 degrees and a decrease in the aorta-SMA distance to 5-11 mm. In our case AM angle was 10 degree and A-SMA distance was 6 mm.

Etiological factors for SMA syndrome can be either a congenital or an acquired anatomic abnormality or, more commonly, a debilitating condition causing severe weight loss. Aortomesenteric angle's width is related to the body mass index.¹³ Anatomic causes include exaggerated lumbar lordosis, abnormally high fixation of the duodenojejunal flexure of the ligament of Treitz, an unusually low origin of the SMA, or a decrease in retroperitoneal fat in the aorto-mesenteric angle. Severe weight loss leading to a depletion of the fatty cushion around the SMA is a major cause of SMA syndrome. Lack of retroperitoneal and periduodenal fat pads can lead to a more acute angle resulting in duodenal "clamping." SMA syndrome is thus triggered by any condition compromising the normal fat cushions and the mesenteric angle. In our case CT scan findings of AM angle of 10 degree and A-SMA distance of 6 mm could be result of sudden weight loss (13kgs), thus mimicking SMA syndrome. Vomitus was non-bilious whereas it should be bilious in SMA syndrome. On exploration, site of narrowing was above the crossing of SMA in the second part of duodenum.

CONCLUSION

Although diagnosis of SMA syndrome based on CT findings was correct but the etiology of GOO in our case was stricture of duodenum due to tuberculosis, which caused loss of retroperitoneal fat and features of SMA syndrome. Duodenal tuberculosis though rare should be considered in differential diagnosis of GOO.

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