Superior Mesenteric Artery Syndrome Secondary to Tuberculosis Induced Cachexia

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Abstract
Superior mesenteric artery (SMA) syndrome is a rare cause of obstruction of 3rd part of duodenum between abdominal aorta and the overlying superior mesenteric artery caused by decrease in the angle between the two vessels as a result of rapid loss of retroperitoneal fat. It is seen in conditions causing severe weight loss and catabolic states. We report a case of pulmonary tuberculosis leading to superior mesenteric artery syndrome.

Introduction
Superior mesenteric artery syndrome was first described by Carl Freiherr von Rokitansky in 1861. It is one of the rarest gastroenterological disorders known to medical science. The incidence was found to be 0.013-0.3% in a series of upper Gl barium studies. SMA syndrome is also known as Wilkie’s syndrome, cast syndrome, mesenteric root syndrome, chronic duodenal ileus and intermittent aortomesenteric occlusion.

Case Report
A 59-year old farmer had presented with complaints of colicky upper abdominal pain and vomiting since 4 days and oliguria since one day. He had generalised weakness and weight loss since one month. He had received antitubercular treatment for pulmonary tuberculosis one year before. Presently he did not complain of fever or cough. On examination he had pulse of 100/min, BP of 100/70 mm Hg, respiratory rate 32/min, pallor and clubbing. He was cachexic and dehydrated. There was no lymph node enlargement or edema. Per abdominal examination revealed a lump in left hypochondrium and epigastrum with visible peristalsis, tenderness in epigastrum and decreased bowel sounds. Respiratory system examination showed decreased intensity of breath sounds in right infrascapular region and right apical crepitations. The clinical impression was abdominal tuberculosis causing intestinal obstruction with prerenal acute renal failure. Differential diagnosis of intraabdominal malignancy was also kept in mind.

His investigations were as follows: Hb-10.9 gm/dL, CBC and platelet count normal, BUN-43 mg/dL, Creatinine-4.0 mg/dL, Na+-144 mEq/L, K+-3.1 mEq/L, liver function tests and blood sugar were normal, ABG showed mild hypoxia. ESR was 115; HIV, HBsAg, anti HcV were negative. Chest radiograph showed right apical opacity, right mild pleural effusion and decreased breath sounds. Respiratory system examination showed decreased intensity of breath sounds in right infrascapular region and right apical crepitations. The clinical impression was abdominal tuberculosis causing intestinal obstruction with prerenal acute renal failure. Differential diagnosis of intraabdominal malignancy was also kept in mind.

abdominal aorta (AA) which was 20⁰ (the normal being 38⁰-56⁰) causing obstruction of 3rd part of duodenum (Figs. 1 and 2). HRCT chest was showing peribronchovascular nodules and tree in bud appearance suggestive of infective etiology, tuberculosis. As patient was unable to produce sputum his gastric aspirate was sent for Ziehl-Nelson staining which was positive for acid fast bacilli. The pleural fluid assay showed high protein content and lymphocytic predominant cytology with high level of ADA suggesting tuberculosis.

The patient was managed conservatively with no oral intake and continuous nasogastric aspiration. He was hydrated and hypokalemia was corrected. The creatinine value reduced to normal after 4 days. He was started on total parenteral nutrition. Since there was no obstruction to orally administered liquid contrast on CT scan the patient was subsequently started on small liquid feeds and antitubercular treatment. The surgeons planned to insert a feeding jejunostomy followed by duodenojejunostomy after improvement of patient’s general condition. However before these procedures could be done the patient suddenly developed acute respiratory failure; he did not respond to the treatment in ICU and succumbed to death. The respiratory failure was probably because of the pulmonary tuberculosis.

Discussion
SMA Syndrome is characterized by compression of third part of duodenum between AA and overlying SMA. Normally the angle between AA and SMA is 38°-56°. Due to rapid loss of retroperitoneal fat the angle may reduce to 6°-25° when it can give rise to SMA syndrome. Depletion of mesenteric fat pad is caused by severe and rapid weight loss due to, anorexia nervosa, malabsorption syndrome, chronic congestive heart failure, diabetes mellitus, catabolic states like cancer, burns and surgery. Spinal deformity, spine surgery, use of body cast for treatment of scoliosis or vertebral fractures may also decrease the AA-SMA angle leading to SMA syndrome. Anatomical anomalies may cause congenital SMA syndrome.

The symptoms include nausea, early satiety, bilious vomiting of partially undigested food, postprandial abdominal pain, abdominal distension. The malnutrition due to persistent vomiting increases wasting which in turn increases the duodenal obstruction thus leading to a vicious cycle. The symptoms are slightly relieved in left lateral or knee-chest position. The investigations include Barium meal, hypotonic duodenography. The computerised tomography with angiography or magnetic resonance angiography can demonstrate reduced AA-SMA angle and shortened AA-SMA distance (2-8 mm, normal being...
10-20 mm) (Fig. 2). It can also be used to assess retroperitoneal fat content and to rule out any other cause of obstruction like lymph nodes or tumour.

The treatment comprises of removal of precipitating factor, nasogastric decompression, maintenance of nutrition by entera feeding using nasojejunal tube, slow and gradual introduction of small, frequent, soft meals, left lateral or prone position after meals. Surgical treatment is indicated when conservative measures fail and it consist of open or laparoscopic duodenojejunostomy.6

In literature search we have found case reports of SMA syndrome caused by anorexia nervosa, cardiac cachexia, diabetes mellitus, spinal surgery. However infectious cause like tuberculosis was not found. So probably this is the first such case report to the best of our knowledge. Duodenal tuberculosis is a rare entity and can mimic SMA syndrome.7 The duodenal obstruction in such situations is caused by retroperitoneal lymph node enlargement or mucosal ulceration, edema and cicatrisation. Both were absent in our case.

Fig. 1: Abdominal CT angiography showing that the angle between abdominal aorta and superior mesenteric artery has been reduced to 20°; the normal being 38°-56°.

Fig. 2: The abdominal CT scan: the arrows point towards abdominal aorta and superior mesenteric artery showing decreased aortomesenteric distance. There is absence of ascites or lymph node enlargement.

References