Retroperitoneal fungal abscess presenting as superior mesenteric artery syndrome

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Summary
We report a patient two years post-cholecystectomy for gallstone pancreatitis presenting with superior mesenteric artery syndrome due to a retroperitoneal fungal abscess.

Keywords: duodenum, superior mesenteric artery syndrome, fungal abscess

Vascular obstruction of the duodenum is a rare disorder due to compression of the third part of the duodenum anteriorly by the superior mesenteric artery against the aorta and the vertebral bodies posteriorly. Variously known as the superior mesenteric artery syndrome, chronic duodenal ileus occurs when occurring after surgical correction of scoliosis, Cast syndrome, this entity presents with chronic upper abdominal symptoms and sometimes as subacute small bowel obstruction. We report a case of a retroperitoneal fungal abscess causing a subacute obstruction of the duodenum.

Case report
A 35-year-old man presented with a two-year history of intermittent bilious vomiting. This had become progressively worse over the two months prior to admission. Prior to the onset of his presenting symptoms he had undergone an open cholecystectomy following an episode of gallstone pancreatitis. This was performed at another hospital and records were not available. His history and physical examination were suggestive of an upper small bowel obstruction with bile in the nasogastric aspirate which drained around two liters daily. Blood investigations including a white cell count and erythrocyte sedimentation rate were within normal range. Plain abdominal X-rays revealed a dilated stomach and first and second part of duodenum with a paucity of gas distally. An upper gastrointestinal endoscopy was performed and found to be normal up to the third part of duodenum. Barium meal examination showed a sharp vertical cut-off in the third portion of the duodenum with duodenal dilatation (figure), and a diagnosis of superior mesenteric artery syndrome was made.

At laparotomy an 8 × 8 cm retroperitoneal abscess was found to be compressing the duodenum against the superior mesenteric artery. The abscess was debrided and contained a non-foul-smelling cheesy material. Initial clinical assessment at surgery was of a tuberculous abscess. A gastrojejunostomy was performed and a drain placed in the abscess cavity. Recovery was uneventful.

Histopathological and microbiological examination of the abscess contents failed to confirm a diagnosis of tuberculosis. Postoperative physical examination and X-rays of the spine were normal. However, microscopic examination showed budding yeasts and pseudohyphae. Culture of the abscess contents grew Candida parapsilosis. The patient remains well eight months postoperatively.

Discussion
The third part of the duodenum crosses the vertebral column at the third lumbar vertebrae.

Figure Barium meal showing duodenal dilatation with characteristic vertical linear extrinsic pressure in the third part of the duodenum due to the superior mesenteric artery

Superior mesenteric artery syndrome: definition

Compression of the third, or transverse, portion of the duodenum against the aorta by the superior mesenteric artery, resulting in chronic, intermittent, or acute, complete or partial duodenal obstruction

Box 1
The superior mesenteric artery originates from the aorta at the level of the first lumbar vertebra. Any factor causing narrowing of the angle between the superior mesenteric artery and the aorta may cause entrapment of the third part of the duodenum. There are many known aetiologies for this condition (box 2). Rarer aetiologies include an abnormally high position of the ligament of Treitz with an upward displacement of the duodenum or an unusually low origin of the superior mesenteric artery. This syndrome should not be confused with a megaduodenum due to other conditions like diabetes, collagen vascular diseases, myotonic dystrophy, or chronic idiopathic intestinal pseudo-obstruction. Diagnosis depends on the barium meal findings of duodenal dilatation, retention of barium within the duodenum and characteristic vertical linear extrinsic pressure in the third part of the duodenum (figure).

How this patient developed a retroperitoneal fungal abscess is unclear. As his symptoms started six weeks following open cholecystectomy, one plausible explanation is that the organism was introduced during cholecystectomy, during which the duodenum was Kocherized. Another possibility is that following pancreatitis, peripancreatic necrosis was undetected at the time of cholecystectomy, and subsequently became infected by fungus. The interesting points are the duration of the symptoms (two years) and the lack of systemic manifestations, ie, pyrexia with normal blood indices. To our knowledge retroperitoneal fungal abscess has not been reported previously, either as a primary entity or as a complication following cholecystectomy.


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