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Brief Reports

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Superior Mesenteric Artery Syndrome Presenting with Pneumoperitoneum and Pneumomediastinum

A 19-year-old woman presented to the emergency department at our institution; she denied any pertinent medical or surgical history before this presentation. She endorsed a four- to six-month history of chronic daily nonbloody nonbilious emesis with a 30-pound weight loss. She had not sought medical attention for her complaints until she noted an acute onset of diffuse abdominal pain, resulting in her presentation to the emergency department. Physical examination revealed an emaciated-appearing, acutely ill female with parotid gland hypertrophy and dental erosions consistent with chronic vomiting. Her abdominal examination was diffusely tender but relatively nondistended.

Laboratory evaluation was significant for a serum blood urea nitrogen and creatinine of 50 and 2.45 mg/dL, respectively, and serum carbon dioxide was 22 mmol/L. Amylase and lipase were 181 and 299 U/L, respectively. White blood cell count was normal but exhibited a neutrophilic shift (89.6%). At this point, with the etiology of her symptoms still unclear, the patient underwent computed tomography of the chest, abdomen, and pelvis. The patient was unable to tolerate oral contrast as a result of persistent vomiting and inability to place a nasogastric tube and was unable to tolerate intravenous contrast as a result of acute renal insufficiency. Computed tomography revealed marked dilation of the stomach and proximal duodenum with small amounts of pneumoperitoneum (Fig. 1A–B). The examination of the chest showed a significant amount of pneumomediastinum (Fig. 1C). The presentation was confusing with pneumomediastinum and pneumoperitoneum as well as what appeared to be a chronic obstruction of the stomach with vomiting. The differential diagnosis included Boerhaave's syndrome, a perforated hollow viscus, a duodenal neoplasm, and malrotation with volvulus. Given the presence of pneumoperitoneum, the patient was taken for urgent abdominal exploration. A thorough abdominal exploration revealed no evidence of perforation. The stomach and duodenum were decompressed with the passage of a nasogastric tube. Intraoperative upper endoscopy revealed no evidence of esophageal perforation, and with manipulation of the intraluminal endoscope, it was possible to advance the endoscope beyond the third portion of the duodenum, revealing no evidence of duodenal web or other obstruction. At this point, because the patient clearly required further resuscitation and the diagnosis was not entirely clear, the abdomen was closed having ruled out acute life-threatening surgical conditions. We performed a thin barium swallow postoperatively, revealing complete obstruction at the midthird portion of the duodenum in a linear pattern pathognomonic for superior mesenteric artery syndrome (Fig. 1D). After suitable nasogastric decompression, we re-explored the patient to perform definitive repair with a hand-sewn side-to-side duodenojejunostomy with the distal limb brought through a defect in the transverse mesocolon. As a result of her profound malnutrition, we began total parenteral nutrition until the patient could tolerate enteral feedings. We were able to discharge the patient on hospital Day 9 on a regular diet and she is currently doing well.

Superior mesenteric artery (SMA) syndrome was first reported by Rokitansky in 1842.[1] It is an extremely uncommon condition, but there are several exacerbating factors that make such a presentation much more likely. These include conditions that result in chronic weight loss or immobility (malignancy, paraplegia, cerebral palsy, drug abuse), catabolism (burn injury, trauma), anorexia nervosa, malabsorption syndromes, and anatomic variations (high insertion of the ligament of

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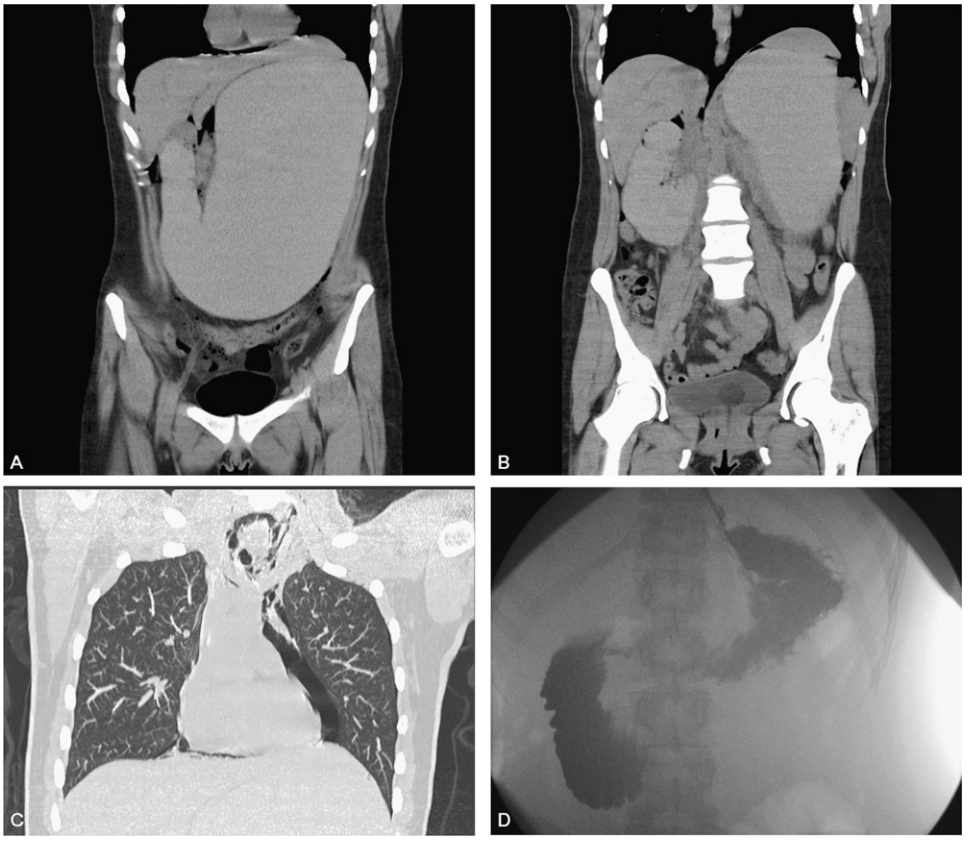


FIG. 1. Coronal reconstruction of abdominal computed tomography indicates a grossly dilated stomach (*A*) and proximal duodenum (*B*). Computed tomography of the chest revealed a significant amount of pneumomediastinum (*C*). After initial exploration to rule out perforated viscus, barium swallow revealed cessation of contrast flow at the third portion of the duodenum (*D*), pathognomonic for superior mesenteric artery syndrome.

Treitz, malrotation, adhesions, and low insertion of the SMA).[2] The final common pathway among many of these states is reduction of the angle between the aorta and the SMA that results in compression of the duodenum; in one case series of eight patients with SMA syndrome, the aortomesenteric angle was less than 20° in each patient[1]; in our case, the angle between the SMA and the abdominal aorta was approximately 13.5° (Fig. 2, arrow indicates SMA). Although pneumomediastinum has been previously reported with SMA syndrome, the reason why these would coexist remains unclear[3]; one possibility would be a microperforation of the esophagus resulting in pneumomediastinum with subsequent sealing of the perforation before onset of significant contamination that would otherwise result in mediastinitis.

Multiple techniques have been reported for the surgical management of SMA syndrome, including both traditional open and more advanced laparoscopic techniques such as minimally invasive Roux-en-Y duodenojejunal bypass.[4] In the case of our patient, her acute presentation and the presence of pneumoperitoneum/pneumomediastinum necessitated standard laparotomy, and thus performing an open duodenojejunostomy



FIG. 2. Sagittal reconstruction illustrates the angle between the superior mesenteric artery (indicated by arrow) and the adjacent abdominal aorta. The aortomesenteric angle in this patient was approximately 13.5°, which is similar to measurements based on computed tomographic studies that are reported in the literature.

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technique first performed by Stavely in 1908[2] seemed to be the most appropriate course. Alternatives such as the Strong's procedure (division of the ligament of Treitz) has a failure rate approaching 25 per cent. A review of 146 open cases using the Strong procedure to address SMA syndrome revealed that it was significantly inferior to open duodenojejunostomy.[2] Gastrojejunostomy is the wrong procedure for SMA syndrome because it does not address the obstruction of the duodenum, creates a blind loop, and may necessitate further procedures.[1]

Although SMA syndrome is uncommon, it is essential that surgeons be familiar with its presentation to facilitate early diagnosis and treatment before the onset of critical obstruction. Patients with early and appropriate treatment have excellent functional outcomes.

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