An Unusual Cause of Duodenal Obstruction

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**Introduction:** Superior mesenteric artery (SMA) compression of the duodenum is well described, but duodenal obstruction caused by extrinsic compression from an abdominal aortic aneurysm (AAA) occurs rarely.

**Report:** This case report describes a patient who presented with nausea and bilious vomiting and was found to have SMA syndrome due to an 8.6 cm suprarenal AAA. He declined an open aneurysm repair, but elected to have an enteric bypass procedure for symptomatic relief.

**Discussion:** SMA syndrome is caused by compression of the third portion of the duodenum between the aorta and the SMA, resulting in complete or partial duodenal obstruction. Only 22 cases of SMA syndrome caused by an AAA have been reported in the literature; usually there is an inflammatory process involved with the aneurysm or there is an infrarenal aortic neck. Definitive treatment consists of open aneurysm repair or decompress the aneurysm sac; other options are enteric bypass, duodenal transposition or duodenal mobilization and caudal displacement (Strong’s procedure).

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**CASE REPORT**

A 76-year-old man presented to a hospital after a two-week history of nausea and bilious vomiting. He reported constipation, urinating less than usual and 6.8 kg weight loss over two months. He is known to have a 7 cm suprarenal abdominal aortic aneurysm (AAA) but has declined repair.

On physical exam, he was 1.8 m tall and weighed 78.4 kg. He had a distended abdomen with a pulsatile, non-tender mass in the midline. He underwent abdominal computerized axial tomographic scanning, which revealed an 8.6 cm (anterior—posterior) × 7.2 cm (transverse) atherosclerotic suprarenal abdominal aortic aneurysm (AAA) that extended from the level of the SMA to both common iliac arteries. He had a distended stomach and complete duodenal obstruction from extrinsic compression of the AAA on the third portion of the duodenum (Fig. 1). The aortomesenteric angle was 19°.

Laboratory testing was significant for blood urea nitrogen 21 mg/dL, creatinine 2.0 mg/dL, white blood cell count 7.8 K/µL and hemoglobin 12.9 g/dL. The diagnosis of SMA syndrome was established. A nasogastric tube was placed and he was hydrated for pre-renal acute renal failure. He again declined repair of the AAA. At laparotomy, the preoperative diagnosis was confirmed and there was no evidence of an intraluminal cause of the obstruction, which was also confirmed by upper endoscopy. He underwent gastrojejunostomy for relief of symptoms.

**DISCUSSION**

SMA syndrome, also known as Wilkie’s syndrome, Cast syndrome and chronic duodenal ileus, is characterized by compression of the third portion of the duodenum between the aorta and the SMA, resulting in complete or partial duodenal obstruction.1,2 The SMA trunk usually forms an acute angle with the aorta (normal range 38–56°) and courses cephalad to the third portion of the duodenum. Any factor which narrows the aorto-mesenteric angle or decreases the aorto-mesenteric distance (normal is 10–20 mm) can lead to entrapment of the duodenum.3,4

SMA syndrome is most common in thin patients aged 10–30 years.4,5 Rapid linear growth without compensatory weight gain, such as in adolescents and rapid severe weight loss in catabolic states can precipitate narrowing of the aortomesenteric angle. In rare cases, it is caused by an abnormally high and fixed ligament of Treitz or an unusually low SMA origin. SMA syndrome is rarely associated with abdominal aortic aneurysms; when it occurs, there is usually an inflammatory or infectious aortic process or an infrarenal aortic neck.4,5

Presentation includes epigastric pain, nausea, bilious vomiting, postprandial discomfort and early satiety. Diagnosis of SMA syndrome is by imaging (CT or upper GI). CT criteria for SMA syndrome includes an aortomesenteric angle of less than 22° and an aortomesenteric distance of less than 8–10 mm. CT scan can also identify other problems that would require intervention, such as pneumatosis, portal venous gas or other causes of duodenal obstruction.

Treatment consists of supportive care initially with nasogastric tube decompression, proper positioning of the patient after eating and adequate nutrition, which can be achieved using a nasojejunal tube passed distal to the obstruction. Surgical intervention is required in order to correct the inciting problem or when conservative measures fail. Options include a duodenojejunostomy, gastrojejunostomy, division...
of the ligament of Treitz with complete mobilization and caudal displacement of the duodenum (Strong’s procedure), or transposition of the third part of the duodenum anterior to the superior mesenteric vessels.1,3 In this case, open repair of the AAA, which decompresses the aneurysm sac, would have been definitive management for duodenal decompression. Since the patient did not wish to undergo this operation with its substantial associated risks, the next best option was a decompressive enteric bypass procedure.

In summary, this is a rare case of SMA syndrome from compression of the third part of the duodenum between a normally located SMA and an abnormally prominent, anteriorly displaced suprarenal AAA.

CONFLICT OF INTEREST/FUNDING
None.

REFERENCES