

Unusual Mechanism for Superior Mesenteric Artery Syndrome after Scoliosis Surgery

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ABSTRACT

Superior Mesenteric Artery (SMA) syndrome is an uncommon condition caused by mechanical obstruction of the distal third of the duodenum between the superior mesenteric artery and the abdominal aorta. SMA syndrome is associated with both operative and non-operative corrections of scoliosis, as well as anorexia nervosa, severe weight loss, tumors, burns, and other traumas.^[1-4] We report an unusual case of SMA syndrome following corrective surgery for scoliosis in which post-operative gastric distension caused duodenal compression that subsequently resolved with gastric decompression, as opposed to the conventional, reverse series of events in which SMA syndrome causes the gastric dilatation.

KEYWORDS: SMA syndrome, scoliosis, spine surgery complications, nasogastric decompression, ileus

CASE REPORT

An otherwise healthy 15-year-old girl presented with adolescent idiopathic scoliosis and a significant right thoracic curve necessitating surgical intervention (**Figure 1**). Her past medical history was noncontributory and there was no family history of idiopathic scoliosis or abdominal pathology. The patient was underweight with a BMI was 16.

The patient underwent a posterior spinal fusion from T2-L3. Pedicle screws were placed at every level on the left and selected levels on the right (**Figure 2**). A Stryker FluoroNav spin confirmed appropriate positioning of all screws. The scoliosis was corrected and reduced using a dual-rod construct. The patient tolerated the procedure well without any intraoperative complications.

The patient's hospital stay was uneventful; she tolerated a regular diet, had normal bowel function and was ambulating without assistance. She was discharged home

Figure 1. Pre-operative AP and Lateral Radiographs of the Spine shows a right thoracic curvature with a Cobb angle of 68 degrees. Pelvis is Risser Stage 4.



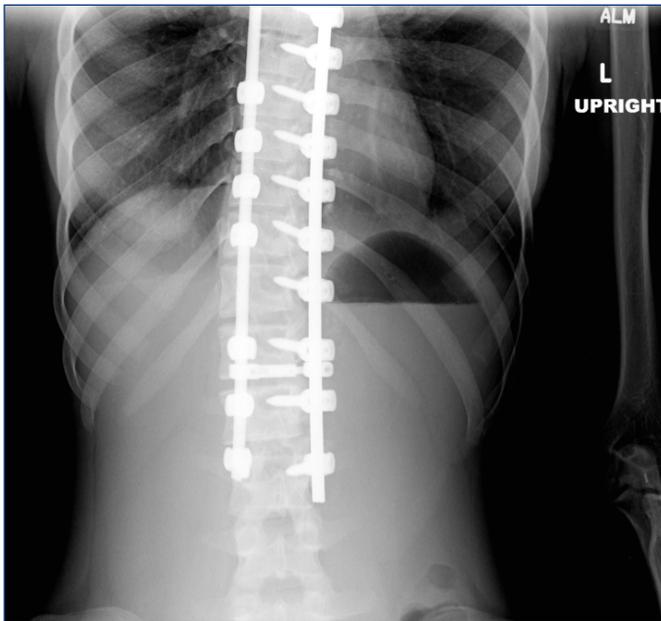
Figure 2. Post-operative AP and Lateral Radiographs of the Spine.

The patient underwent surgical correction of her scoliosis with posterior spinal fusion from T2-L3.



with her family on the fifth post-operative day. However, while at home on the eighth post-operative day she developed abdominal pain, had multiple bouts of emesis and was unable to tolerate food. She returned to the Emergency Department for evaluation. An AP upright abdominal radiograph revealed marked distention of the stomach with an air-fluid level consistent with an obstructive process (Figure

Figure 3. AP Upright Abdominal Radiograph reveals marked distention of the stomach with an air-fluid level. There is otherwise a lack of bowel gas except a small amount of gas and dilatation seen in the descending and rectosigmoid colon.



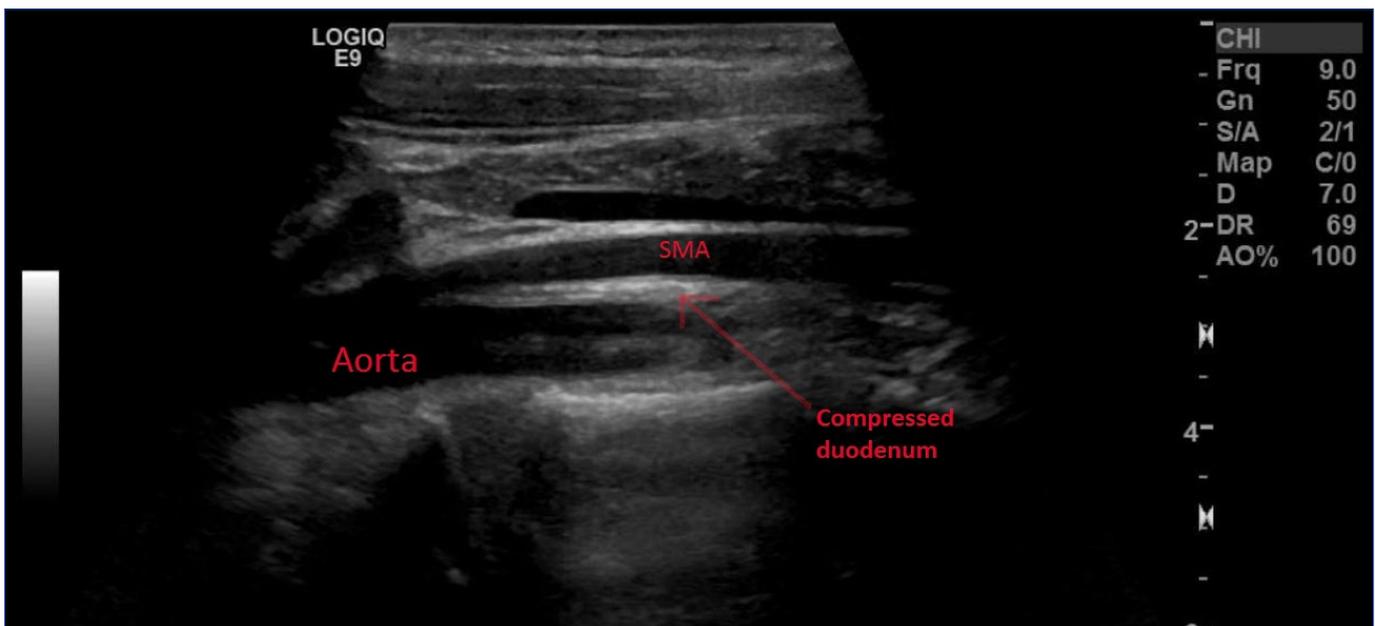
3). An abdominal ultrasound revealed a markedly dilated stomach with the gastric fundus extending down to the level of the aortic bifurcation. On the sagittal images, the superior mesenteric artery course was nearly parallel to the course of the abdominal aorta, such that there was very little space between the two vessels for the transverse duodenum to remain patent (Figure 4).

A nasogastric (NG) tube was placed and the stomach was decompressed overnight. A repeat ultrasound was conducted three days after the NG-tube was placed and revealed normal midgut rotation (Figure 5). Under fluoroscopic monitoring, a naso-duodenal tube was placed into the descending duodenum (attempts to reach the duodenal-jejunal flexure were unsuccessful). Fluoroscopic images confirmed that the gastric decompression resolved the SMA syndrome, and contrast flowed fairly readily from the duodenum to the proximal jejunum. (Figure 6) The patient received continuous decompression of stomach with the NG tube and slow, continuous feeding via the nasoduodenal feeding tube for five days at the hospital and for five days at home after she was discharged.

A week after discharge, the patient was evaluated for displacement of the nasoduodenal tube. Normal swallowing was confirmed with a barium study: the barium emptied promptly into the normal duodenum, proximal small bowel and into the jejunum without evidence of obstruction. Both the NG tube and the nasoduodenal tube were removed and she was transitioned to a regular oral diet.

The patient is now more than a year out from her initial operation. She is doing well in follow-up at one year; she has had no hardware complications or recurrence of her gastrointestinal symptoms.

Figure 4. Sagittal view of an abdominal ultrasound taken prior to nasogastric tube placement. An almost parallel aorta and SMA are seen with a very compressed duodenum passing in between the two vascular structures



DISCUSSION

SMA syndrome is a complication of corrective surgery for spinal deformities with reported incidence of over 4%. [5, 6] SMA syndrome often presents with early satiety after eating, intermittent nausea, bilious vomiting, and gastric dilatation. The onset of symptoms may begin immediately post-operatively, or up to several weeks following the surgery. [1, 6, 8] If untreated, repeated emesis as a result of SMA

syndrome may lead to dehydration, electrolyte imbalances, gastric perforation, circulatory collapse due to decreased intraluminal pressures and even death. [1, 3]

Diagnosis relies on a focused clinical history and a combination of imaging techniques to visualize the gastric and proximal duodenal dilatation, as well as an aortomesenteric angle $<20^\circ$. [1, 6, 8] In this case, the patient's suspected diagnosis was confirmed using upper GI barium contrast after ultrasound, though some studies suggest that the definitive imaging should be upper GI barium and concurrent angiography to visualize the aortomesenteric angle. [6]

The etiology of SMA syndrome in the setting of spinal deformity correction surgery is related to trunk height lengthening with instrumentation, resulting in traction of the SMA and narrowing of the aortomesenteric angle. [9,11] Recent studies have identified certain preoperative risk factors of patients likely to develop SMA syndrome including BMI <20 , laterally displaced lumbar curves, sagittal kyphosis, and a large correctional change in the angle of curvature. [5, 6, 9, 10, 11]

However, in the case reported in this article, despite the patient's recent history of corrective surgery for adolescent idiopathic scoliosis and low BMI, her SMA syndrome appears to have been secondary to acute gastric dilatation causing aortomesenteric impingement. Unlike other cases of SMA syndrome, her gastric dilatation was the cause, not merely the consequence, of her aortomesenteric duodenal obstruction. After gastric decompression, the patient's SMA syndrome resolved and the contrast was seen to readily flow through her distal duodenum and into her jejunum.

Acute gastric dilatation is a known consequence of duodenal occlusion and SMA syndrome, but there have been no reports of SMA syndrome resulting from acute gastric dilatation.

Figure 5. Sagittal view of an abdominal ultrasound after nasogastric tube placement.
 (a) Improved angle between the SMA and aorta are seen here.
 (b) a decompressed duodenum is seen between the two vascular structures

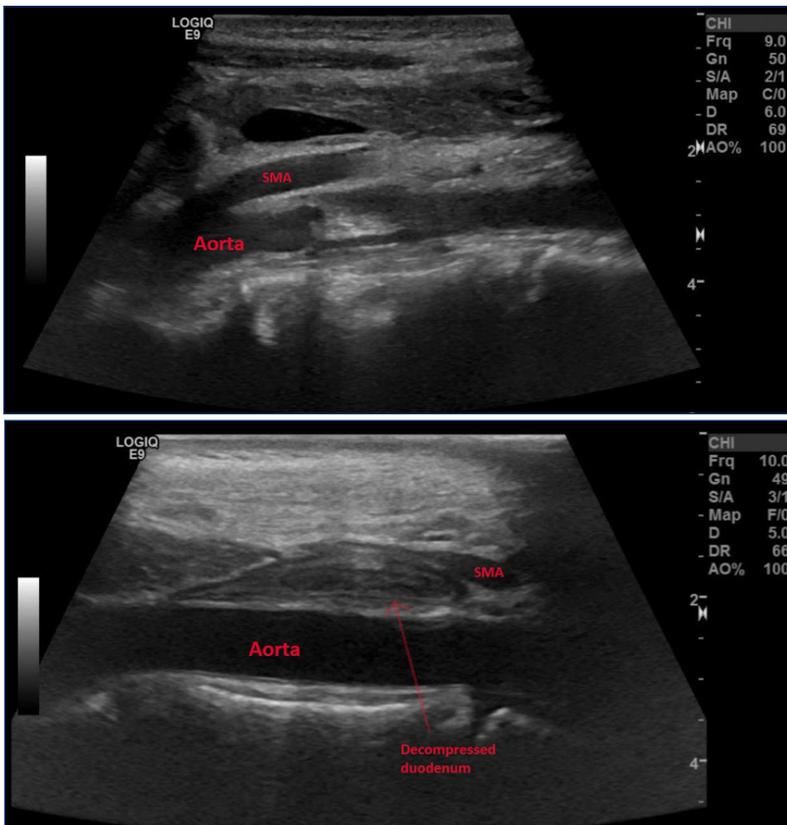
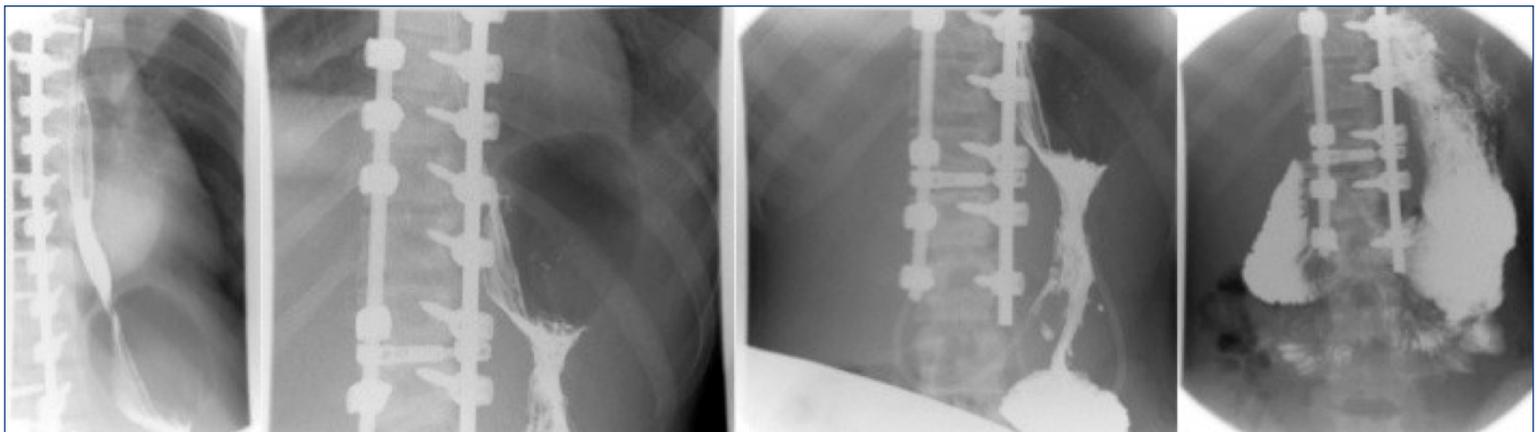


Figure 6. Upper GI Series with barium swallow showed the contrast emptied promptly from the stomach into the normal duodenum without any evidence of obstruction.



SMA syndrome induced by acute gastric dilatation is most commonly seen in patients with eating disorders, where episodes of binge-eating lead to acute stomach distension and compression of duodenum between the SMA and the aorta. [2, 12] This in turn, prevents emptying of the stomach and can cause the dreaded consequences of SMA syndrome.

This is the first described case of SMA syndrome resulting from acute gastric distension following corrective spine surgery for scoliosis. It is important to recognize that patients without identified risk factors for SMA syndrome (<25% weight percentile for height, <20 BMI, postoperative weight loss) may be at risk for developing this potentially fatal complication. Moreover, while the causes of SMA syndrome are mechanical and well understood, SMA syndrome secondary to acute gastric dilatation may be caused by decreased gastric motility (similar to postoperative ileus) or simply by eating too much too soon after surgery, which can be corrected by GI rest and placement of a decompressive NG tube. This finding also has implications for diet recommendations upon discharge.

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