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# Acute-onset of superior mesenteric artery syndrome following surgical correction of scoliosis: Case report and review of literature



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#### ABSTRACT

Superior mesenteric artery (SMA) syndrome is a rare condition caused by compression of the third portion of duodenum by the angle between the superior mesenteric artery against the aorta. A rare presentation of SMA syndrome is following scoliosis repair and spinal fusion with a low incidence and most of these patients present with symptoms within one to two weeks or even more after the surgical repair. A high suspicion index after surgical correction of scoliosis with well-known risk factors (low BMI, low percentile of weight for height, and a high degree of change in the Cobb's angles) can anticipate the postoperative diagnosis. Management has been described for postsurgical scoliosis repair with a late onset presentation of SMA syndrome with nutritional support with good success rates, but there is no data for best treatment management for acute onset especially when the surgical correction of the spine causes complete duodenal obstruction and a surgical intervention might be warranted. Here in, we present a 14 year-old boy with an acute 24-h postoperative SMA syndrome following surgical correction of scoliosis.

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#### 1. Introduction

Superior mesenteric artery (SMA) syndrome is a rare condition caused by compression of the third portion of duodenum by the angle between the superior mesenteric artery against the aorta, described by Von Rokitansky in 1861 and the first case series report on 1927 by Wilkie [1]. Its overall incidence is unknown [2,3]. Symptoms of SMA syndrome may include nausea, bilious emesis, abdominal pain, early satiety, anorexia, failure to gain weight, indigestion, esophageal reflux, sense of fullness, persistent weight loss, and low BMI [3,4]. SMA syndrome can be developed after scoliosis repair and spinal fusion causing narrowing the aortomesenteric angle with an incidence of 2.5% [5]; most of these patients present with symptoms within one or two weeks and even more after the surgical repair [6,7]; here in, we present to our knowledge

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the first case report of acute onset (24-h) postoperative SMA syndrome following surgical correction of scoliosis in a 14 year old male patient.

#### 2. Case report

Fourteen (14) year-old male patient with a BMI of 13.4 kg/m<sup>2</sup> [weight 40 kg (2 percentile), height 1.73 m (85 percentile)] was scheduled for thoracolumbar T3-L2 spinal fusion procedure for scoliosis (Cobb's angles: thoracic 70° and lumbar 44°). Past medical and surgical history included umbilical hernia repair and circumcision at 6 years old, with no other medical history. The patient underwent a successful spinal correction of scoliosis (Fig. 1), with a final thoracic angle of 22° and lumbar 10°, with a total increase in height of 8 cm. 24-hours postoperatively the patient developed nausea, bilious vomiting and abdominal pain. A nasogastric tube was placed obtaining bilious output.

An upper gastrointestinal (UGI) series with barium was done showing third portion duodenal obstruction with slight dilatation of the second portion of the duodenum (Fig. 2). An abdominal CT

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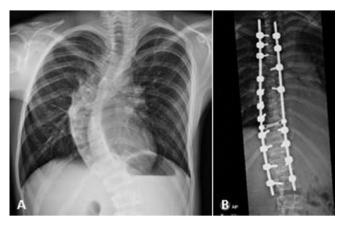


Fig. 1. A) Chest X-ray: Scoliosis with right convexity. B) Postoperative Chest X ray.

angiography confirmed the presence of a narrow aortomesenteric angle of 14° with a 3.8 mm diameter (Fig. 3). A SMA syndrome was diagnosed. The placement of a feeding tube assisted with an upper endoscopy was attempted and was unsuccessful since there was a complete obstruction of duodenum. The patient was then scheduled for surgery and a duodenum-jejunum bypass was done to relieve the obstruction. The patient was discharged one-week after surgery tolerating full oral feeds.

#### 3. Discussion

Superior artery mesenteric syndrome is a rare condition, its overall true incidence is unknown and an incidence of 0.0024–0.034% has been described for the general population in some series, being young adults and women the most commonly affected [1,3,8,9].

Symptoms of obstruction are caused by compression of the third portion of duodenum by the angle between de abdominal aorta and the origin of the SMA, which normally can vary between 20° and



Fig. 2. UGI series with barium: Third portion duodenal obstruction and dilatation of the second duodenal portion.

70°; most of the population in general being between 30° and 56°, and an aortomesenteric distance of 10 or 20 mm [1,10]. An angle between 6 and 16° and an aortomesenteric angle distance 2–8 mm confirms its diagnosis [11]. Our patient had an angle of 14° and an aortomesenteric diameter of 3.8 mm after the surgical correction based on the CT angiography.

Conditions that can cause the SMA syndrome has been associated most commonly to catabolic states and malnutrition such as rapid weight loss, AIDS, cancer, cerebral palsy, high degree burns, anorexia nervosa, drug abuse and patients with very low body mass index or as on our case after scoliosis repair and spinal fusion [5,8,12–14].

The surgical correction of scoliosis causes realignment and longitudinal traction of the aorta increasing the height of the patient and subsequently causing narrowing of the angle between the aorta and the superior mesenteric artery, causing obstruction of the third portion of the duodenum [10], especially in patients with a BMI <18 or a weight percentile for height of 5% that suggests low or no periphery duodenal visceral fat which is a well-known risk factor [14,15]. Our patient met the previous risk factors with a BMI of 13.4 kg/m<sup>2</sup> and a weight percentile for height of 2% which predicted the postoperative SMA syndrome. The correctional change in curvature angles in the sagittal and coronal planes also plays a role in SMA syndrome risk. A greater Cobb's angle correction is accompanied by a greater increase in patient height [5]; our patient had preoperatively a thoracic 70° and lumbar 44° and postoperatively thoracic angle of 22° and lumbar 10° with an increase of 8 cm in height.

Usually the SMA syndrome following spinal fusion for scoliosis presents one week postoperatively, with an average of between day 5 or 6 [10,11,16]. Progressive weight loss recurrent vomiting, abdominal distension, marked dehydration, bilious vomiting are some common features described and usually presented as a late onset [6,7]. Although we predicted a postoperative SMA syndrome on our patient [10], we did not expect such an acute onset. This can be explained by the fact of the complete duodenal obstruction caused by the 8 cm sudden increase in height after the spinal fusion and narrowing of the aortomesenteric angle, causing a 24-h postoperatively bilious vomiting.

Although there is no gold standard for diagnosis of SMA syndrome, some series suggest that both UGI series and CT angiography will be necessary for diagnosis [1,11,17]. On our patient an UGI showed obstruction of the third portion of duodenum, and an abdominal CT angiography confirmed an aortomesenteric 14° angle and a 3.8 mm distance.

Management of the overall SMA syndrome with nutritional support has a success rate of 71%–85% [1,18], with a recurrence rate of 15% [1], and treatment should be at least for a period of 6 weeks [19], including patients who develop SMA syndrome with late onset symptoms following spinal fusion with some series having good outcomes [10]; placement of an enteral tube beyond the ligament of Treitz or parenteral nutrition, postural changes such as postprandial left lateral decubitus, prone or knee-chest position and nasogastric decompression have all been described as a good option to increase the aortomesenteric fat and the aortomesenteric angle allowing the relief of the duodenal obstruction [1,11,16]. Nevertheless, all of these reports were made for patients who developed symptoms one week after the procedure, none of these describes management after an acute onset with complete duodenal obstruction that was confirmed with endoscopy and for that reason we were unable to offer this option to our patient, in fact the nutritional management is not always successful and surgical intervention might be needed [3].

We decided to perform a duodenum-jejunum anastomosis since it remains as the surgery of choice; it has a 90% success rate and

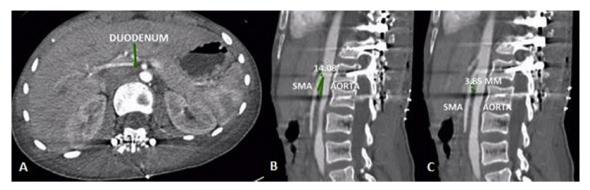


Fig. 3. Abdominal CT angiography: A) Duodenal impingement. B) Aortomesenteric angle of 14°. C) Aortomesenteric diameter of 3.8 mm.

allows the functional preservation of the pylorus [2]. Some other surgical options have been described including the ligament of Treitz division or Strong Procedure, and gastro-jejunum bypass. Strong procedure has the advantage of keeping the intestinal integrity and it is feasible [11], but has a 25% failure rate [20]. Gastro-jejunum anastomosis provides appropriate gastric decompression but fails in liberating completely the duodenal obstruction, leading to the persistence of symptoms, needing a duodenum-jejunum bypass in some cases [2]. Finally, some authors support the resection of the abnormal duodenal segment instead of performing a bypass of the third portion of the duodenum, arguing that SMA syndrome is a variation of a motility disorder more than a real mechanical obstruction [21] with unknown success and failure rates. Laparoscopic or open approaches are well described and both have the similar success rates as the open approach [20].

### 4. Conclusion

SMA syndrome is a well-described complication after surgical correction of scoliosis especially in patients with high risk factors such as low BMI and low percentile of weight for height; most of these symptoms have a late onset within a week or later. Post-operative 24-h bilious vomiting should increase the suspicion of complete duodenal obstruction caused by the sudden increase in patient height and the narrowing of the aortomesenteric angle. Although conservative treatment has been described for the late onset postoperative SMA syndrome with good rates of success, there is no data on patients with acute onset presentation with an almost complete duodenal obstruction and a surgical intervention might be warranted. More data is needed to predict which patient will develop acute versus late onset SMA symptoms after surgical correction of scoliosis.

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