

Superior mesenteric artery syndrome following scoliosis surgery: Its risk indicators and treatment strategy

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Abstract

AIM: To investigate the risk indicators, pattern of clinical presentation and treatment strategy of superior mesenteric artery syndrome (SMAS) after scoliosis surgery.

METHODS: From July 1997 to October 2003, 640 patients with adolescent scoliosis who had undergone surgical treatment were evaluated prospectively, and among them seven patients suffered from SMAS after operation. Each patient was assigned a percentile for weight and a percentile for height. Values of the 5th, 10th, 25th, 50th, 75th, and 95th percentiles were selected to divide the observations. The sagittal Cobb angle was used to quantify thoracic or thoracolumbar kyphosis. All the seven patients presented with nausea and intermittent vomiting about 5 d after operation. An upper gastrointestinal barium contrast study showed a straight-line cutoff at the third portion of the duodenum representing extrinsic compression by the superior mesenteric artery (SMA).

RESULTS: The value of height in the seven patients with SMAS was above the mean of sex- and age-matched normal population, and the height percentile ranged from 5% to 50%. On the contrary, the value of weight was below the mean of normal population with the weight percentile ranging from 5% to 25%. Among the seven patients, four had a thoracic hyperkyphosis ranging from 55° to 88° (average 72°), two had a thoracolumbar kyphosis of 25° and 32° respectively. The seven patients were treated with fasting, antiemetic medication, and intravenous fluids infusion. Reduction or suspense of traction was adopted in three patients with SMAS during halo-femoral traction after anterior release of scoliosis. All the patients recovered completely with no sequelae. No one required operative intervention with a laparotomy.

CONCLUSION: Height percentile < 50%, weight percentile < 25%, sagittal kyphosis, heavy and quick halo-femoral traction after spinal anterior release are the potential risk indicators for SMAS in patients undergoing correction surgery for adolescent scoliosis.

INTRODUCTION

Superior mesenteric artery syndrome (SMAS) results from compression of the third portion of duodenum as it crosses underneath the superior mesenteric artery (SMA), the second branch of the abdominal aorta (Figure 1). Several authors have reported series or individual cases of the condition after the trunk is immobilized with a cast, also known as cast syndrome^[1,2] or Wilkie's syndrome^[3-5]. It is rarely reported that SMAS occurs after scoliosis correction with newly-developed three-dimensional derotation technique. The extrinsic compression of duodenum by the SMA produces a characteristic contrast with the upper gastrointestinal tract. Clinically, the syndrome may progress to serious sequelae with a mortality rate of 33% or follow a benign course^[6]. Therefore, its early diagnosis and treatment should be emphasized. We report seven cases of SMAS following surgery for scoliosis and the risk indicators, pattern of clinical presentation and treatment strategy.

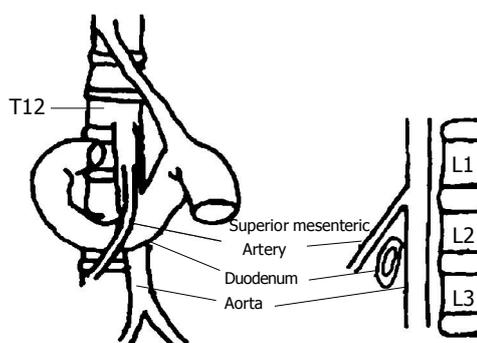


Figure 1 Anatomic relationship between SMA and duodenum.

MATERIALS AND METHODS

From July 1997 to October 2003, 640 patients (228 boys and

412 girls) with adolescent scoliosis who had undergone surgical treatment with three-dimensional correction technique were evaluated prospectively. Seven of them suffered from SMAS after operation, including three males and four females, aged from 13 to 16 years. Three of them were diagnosed as congenital scoliosis and four as idiopathic scoliosis with their coronary Cobb angle ranging between 45° and 116° (average, 82°). SMAS occurred in three patients during halo-femoral traction following anterior spinal release, in three after posterior correction with three-dimensional TSRH (Texas Scottish Rite Hospital, Sofamor-Danek, USA) derotation technique, and in one after anterior correction with CDH (CD-Horizon, Sofamor-Danek, USA) instrumentation.

Charts were reviewed for weight and height at admission. Each patient was assigned a percentile for weight and a percentile for height. The percentile was obtained by referring to the normal data reported by the National Center for Student Status and Health Statistics in 2001, and the tabulated data were sex- and age-specific. Values of the 5th, 10th, 25th, 50th, 75th, and 95th percentiles were selected to divide the observations. The sagittal Cobb angle across the involved segments was used to quantify thoracic or thoracolumbar kyphosis. Values were interpreted with respect to normative data for sagittal alignment in adolescents.

All the seven patients presented with nausea and intermittent vomiting about 5 d after operation. The vomit was brown green and turbid liquid. Between bouts of vomiting, the patients felt appetitive. On examination, the epigastrium was distended and soft with mild tenderness and resonant percussion, and bowel sounds were often active. An upper gastrointestinal barium contrast study showed a barium-filled dilatation of the proximal part of duodenum and a straight-line cutoff at the third part of the duodenum representing extrinsic compression by the SMA.

RESULTS

In the seven patients, the correction rate of coronary Cobb angle ranged from 57% to 74%, with an average of 66%. The height increased 4-15 cm after surgery.

All the seven patients with SMAS had a disproportionately thin habitus in relation to their height. The value of height was above the mean of sex- and age-matched normal population, and the height percentile ranged from 5% to 50%. On the contrary, the value of weight was below the mean of sex- and age-matched normal population with their weight percentile ranging from 5% to 25% (Table 1). The normal thoracic kyphosis was defined as 20°-50°, and the

normal thoracolumbar alignment was 0°^[6]. Among the seven patients, four had a thoracic hyperkyphosis ranging from 55° to 88° (average 72°), one had a thoracic alignment of 48° within the normal range of thoracic kyphosis but close to the upper normal value. Thoracolumbar kyphosis was identified in the other two patients with the sagittal Cobb angle of 25° and 32° respectively.

The seven patients were treated with fasting, antiemetic medication, and intravenous fluids infusion. A left lying position was mandatory, and reduction or suspense of traction was adopted in three patients who developed SMAS during halo-femur traction after anterior release of scoliosis. Symptoms were alleviated after 5-7 d in five patients. The other two patients had an obstinate and repetitive vomiting, conservative management was continued, and feeding was not started until days 12 and 18. At that time, their weight reduced 7 and 11 kg respectively. All the patients recovered completely with no sequelae. No one required operative intervention with a laparotomy.

DISCUSSION

The etiology of SMAS is related to the anatomy of the third part of duodenum, which passes between the aorta and its SMA branch. The SMA arises from the anterior aspect of the aorta at level of L1 and L2 vertebral body. It is encased in fat and lymphatic tissue at its origin and descends downwards at an acute angle into the mesentery. In normal individuals, the SMA to aortic angle is 20°-50°. The duodenum usually passes across the aorta at level of L3 vertebral body and is suspended in acute angle between the aorta and the SMA by Treitz ligament^[7]. This special anatomical relationship has been referred to as "a nutcracker"^[8]. Any factors disturbing the close relationship of this anatomy may lead to extrinsic compression of duodenum. SMAS may occur due to weight loss in patients suffering from malabsorption, anorexia nervosa, extensive burns, or multiple trauma, when fat is depleted at origin of the SMA surrounding duodenum^[9-12]. Other causes include extrinsic vascular obstruction secondarily due to bowel dilatation resulting from atrophy of bowel musculature and its replacement by collagen in connective tissue disorders.

Since 1990s, three-dimensional derotation technique has improved the corrective rate of scoliosis greatly^[8,13]. Although SMAS has decreased sharply due to the adoption of this technique, SMA compression of duodenum is still a potentially life-threatening complication of scoliosis surgery^[14]. The incidence of adverse sequelae from the syndrome

Table 1 Height, weight, and sagittal kyphosis data of patients with SMAS

Caseno.	Sex	Age (yr)	Height percentile	Weight percentile	Pre-operative sagittal kyphosis
1	M	16	25	5	85° (T)
2	F	15	50	5	48° (T)
3	M	16	25	10	88° (T)
4	F	14	25	25	55° (T)
5	M	15	10	5	60° (T)
6	F	13	5	25	32° (TL)
7	F	14	25	25	25° (TL)

M, male; F, female; T, thoracic; TL, thoracolumbar.

remains alarmingly high. Aspiration pneumonia, acute gastric rupture, and cardiovascular collapse have been documented as morbid or fatal complications of SMAS^[15-17]. Scoliosis surgery also relatively lengthens the spine, displacing the SMA origin cephalad at the expense of lateral mobility. Crowther *et al*^[15], showed that there can be an acute increase in vertebral column length with spinal instrumentation, resulting in traction on the SMA and narrowing of the arteriomesenteric angle, and postoperative weight loss results in loss of retroperitoneal fat that protects duodenum from compression^[18,19].

We emphasize the importance of identifying those patients at risk and maintaining a high index of suspicion. Paying attention to the various risk factors that may be present before surgery may be beneficial to the understanding and treatment of SMAS. Munns *et al*^[20], identified high-risk patients as “those with a thin, asthenic habitus”. Hutchinson and Bassett^[21] reported that five patients undergoing operative treatment of scoliosis have a “disproportionately thin habitus in relation to their height”. However, there are no clinically useful parameters to identify individuals susceptible to SMAS^[22]. In order to quantify the degree of asthenia, we calculated the absolute difference in these patients’ height percentile and weight percentile and compared it with the sex- and age-matched normal population. In our series, all the seven patients who developed SMAS had also a disproportionately thin habitus in relation to their height, the value of height was above the mean of normal population with the height percentile ranging from 5% to 50%, and the value of weight was below the mean of normal population with the weight percentile ranging from 5% to 25%. We propose that the height percentile <50% and the weight percentile <25% might be potential risk indicators for SMAS in patients undergoing correction surgery for adolescent scoliosis. Attention should also be given to sagittal kyphosis. Scoliosis associated with sagittal kyphosis is usually accompanied with severe trunk collapse. Following scoliosis correction or heavy skull-femur traction, the trunk elongation becomes more remarkable. This may lead to further narrowing of the arteriomesenteric angle. In this series, six patients had the sagittal abnormality including four with thoracic hyperkyphosis and two with thoracolumbar kyphosis. In addition, the high-risk patients include those who undergo heavy and quick halo-femoral traction after spinal anterior release (three patients in our group).

Symptoms of obstruction often become apparent 5-7 d after surgery. Clinicians should be on the alert for the persistent nature of vomiting. This is accompanied with upper abdominal distension, epigastric tenderness, and tympanic percussion. Bowel sounds are present on auscultation in contrast to the findings in postoperative ileus. Postoperative paralytic ileus caused by general anesthesia, analgesic, electrolytic imbalance, or greater splanchnic nerve injury during anterior spinal release usually occurs at an earlier period after operation and disappeared spontaneously in 3-5 d. Plain abdominal radiograph has no remarkable value in early diagnosis. An upper gastrointestinal barium contrast radiograph should be mandatory, the classic findings of which are specific for SMAS. Computed tomography scan and endoscopy do not contribute to the diagnosis of SMAS^[15].

Treatment of SMAS varies from nonoperative to operative procedures^[23-26]. Anderson *et al*^[27], reported that five of nine cases require exploratory laparotomy. Crowther *et al*^[15], proposed that most patients recover after conservative measures, but occasionally surgical intervention may be required. Van Brussel *et al*^[4], states that the first choice of treatment for SMAS is conservative therapy. In our series, through fasting, gastrointestinal decompression, changing posture, maintaining electrolytic balance or relieving halo-femoral traction, the seven patients recovered without operative intervention and internal fixation removal. We consider that conservative therapy is effective if early diagnosis, appropriate measures, and gastrointestinal physicians’ participation are obtained.

In conclusion, patients undergoing correction surgery for adolescent scoliosis have multiple potential risk indicators for SMAS. The main risk indicators include height percentile <50%, weight percentile <25%, sagittal kyphosis, heavy and quick halo-femoral traction after spinal anterior release.

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REFERENCES

- 1 **Griffiths GJ**, Whitehouse GH. Radiological features of vascular compression of the duodenum occurring as a complication of the treatment of scoliosis (the cast syndrome). *Clin Radiol* 1978; **29**: 77-83
- 2 **Sprague J**. Cast syndrome: the superior mesenteric artery syndrome. *Orthop Nurs* 1998; **17**: 12-15; quiz 16-17
- 3 **Raissi B**, Taylor BM, Taves DH. Recurrent superior mesenteric artery (Wilkie’s) syndrome: a case report. *Can J Surg* 1996; **39**: 410-416
- 4 **Van Brussel JP**, Dijkema WP, Adhin SK, Jonkers GJ. Wilkie’s syndrome, a rare cause of vomiting and weight loss: diagnosis and therapy. *Neth J Med* 1997; **51**: 179-181
- 5 **Lundell L**, Thulin A. Wilkie’s syndrome--a rarity? *Br J Surg* 1980; **67**: 604-606
- 6 **Boseker EH**, Moe JH, Winter RB, Koop SE. Determination of “normal” thoracic kyphosis: a roentgenographic study of 121 “normal” children. *J Pediatr Orthop* 2000; **20**: 796-798
- 7 **Meyers MA**. Treitz redux: the ligament of Treitz revisited. *Abdom Imaging* 1995; **20**: 421-424
- 8 **Vitale MG**, Higgs GB, Liebling MS, Roth N, Roye DP. Superior mesenteric artery syndrome after segmental instrumentation: a biomechanical analysis. *Am J Orthop (Belle Mead NJ)* 1999; **28**: 461-467
- 9 **Wilkinson R**, Huang CT. Superior mesenteric artery syndrome in traumatic paraplegia: a case report and literature review. *Arch Phys Med Rehabil* 2000; **81**: 991-994
- 10 **Milner EA**, Cioffi WG, McManus WF, Pruitt BA. Superior mesenteric artery syndrome in a burn patient. *Nutr Clin Pract* 1993; **8**: 264-266
- 11 **Laffont I**, Bensmail D, Rech C, Prigent G, Loubert G, Dizien O. Late superior mesenteric artery syndrome in paraplegia: case report and review. *Spinal Cord* 2002; **40**: 88-91
- 12 **Ahmed AR**, Taylor I. Superior mesenteric artery syndrome. *Postgrad Med J* 1997; **73**: 776-778
- 13 **Labelle H**, Dansereau J, Bellefleur C, Poitras B, Rivard CH, Stokes IA, de Guise J. Comparison between preoperative and postoperative three-dimensional reconstructions of idiopathic scoliosis with the Cotrel-Dubousset procedure. *Spine (Phila Pa 1976)* 1995; **20**: 2487-2492
- 14 **Shapiro G**, Green DW, Fatica NS, Boachie-Adjei O. Medical complications in scoliosis surgery. *Curr Opin Pediatr* 2001; **13**: 36-41

- 15 **Crowther MA**, Webb PJ, Eyre-Brook IA. Superior mesenteric artery syndrome following surgery for scoliosis. *Spine (Phila Pa 1976)* 2002; **27**: E528-E533
- 16 **Sapkas G**, O'Brien JP. Vascular compression of the duodenum (cast syndrome) associated with the treatment of spinal deformities. A report of six cases. *Arch Orthop Trauma Surg* 1981; **98**: 7-11
- 17 **Todd SR**, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. *Am Surg* 2000; **66**: 709-710
- 18 **Baltazar U**, Dunn J, Floresguerra C, Schmidt L, Browder W. Superior mesenteric artery syndrome: an uncommon cause of intestinal obstruction. *South Med J* 2000; **93**: 606-608
- 19 **Bapat VN**, Rastogi S, Moorthy K, Kulgod S, Supe An. Acute superior mesenteric artery syndrome due to rapid weight loss following massive small bowel resection. *Indian J Gastroenterol* 1996; **15**: 154
- 20 **Munns SW**, Morrissy RT, Golladay ES, McKenzie CN. Hyperalimentation for superior mesenteric-artery(cast) syndrome following correction of spinal deformity. *J Bone Joint Surg Am* 1984; **66**: 1175-1177
- 21 **Hutchinson DT**, Bassett GS. Superior mesenteric artery syndrome in pediatric orthopedic patients. *Clin Orthop Relat Res* 1990; **250**: 250-257
- 22 **Rosa-Jimenez F**, Rodriguez Gonzalez FJ, Puente Gutierrez JJ, Munoz Sanchez R, Adarraga Cansino MD, Zambrana Garcia JL. Duodenal compression caused by superior mesenteric artery: study of 10 patients. *Rev Esp Enferm Dig* 2003; **95**: 485-489 480-484
- 23 **Lippl F**, Hannig C, Weiss W, Allescher HD, Classen M, Kurjak M. Superior mesenteric artery syndrome: diagnosis and treatment from the gastroenterologist's view. *J Gastroenterol* 2002; **37**: 640-643
- 24 **Murthi GV**, Raine PA. Superior mesenteric artery syndrome in children. *Scott Med J* 2001; **46**: 153-154
- 25 **Kim IY**, Cho NC, Kim DS, Rhoe BS. Laparoscopic duodenojejunostomy for management of superior mesenteric artery syndrome: two cases report and a review of the literature. *Yonsei Med J* 2003; **44**: 526-529
- 26 **Bermas H**, Fenoglio ME. Laparoscopic management of superior mesenteric artery syndrome. *JLS* 2003; **7**: 151-153
- 27 **Anderson JR**, Earnshaw PM, Fraser GM. Extrinsic compression of the third part of the duodenum. *Clin Radiol* 1982; **33**: 75-81

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