

# Infrarenal transposition of the superior mesenteric artery: A new approach in the surgical therapy for Wilkie syndrome

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The superior mesenteric artery syndrome, also known as Wilkie syndrome or as arteriomesenteric obstruction of the duodenum, is a rare condition of upper intestinal obstruction in which the third part of the duodenum is compressed by the overlying, narrow-angled superior mesenteric artery against the posterior structures. It is characterized by early satiety, recurrent vomiting, abdominal distention, weight loss, and postprandial distress. When nonsurgical management is not possible or the problem is refractory, surgical intervention is necessary. Usually a laterolateral duodenojejunostomy or Roux-en-Y reconstruction for reconstruction of the intestinal passage is performed. We report the first successful transposition, to our knowledge, of the superior mesenteric artery into the infrarenal aorta in the therapy of Wilkie syndrome. (*J Vasc Surg* 2008;47:201-4.)

Wilkie syndrome, also known as arteriomesenteric obstruction of the duodenum, is a rare condition of upper intestinal obstruction in which the third part of the duodenum is compressed by the overlying, narrow-angled superior mesenteric artery (SMA) against the posterior structures. It is characterized by early satiety, recurrent vomiting, abdominal distention, weight loss, and postprandial distress. When nonsurgical management is not possible or the problem is refractory, surgical intervention is necessary.

## CASE REPORT

A 37-year-old woman presented with the sensation of fullness, early satiety, epigastric pain, postprandial distress after a few bites, and tympanitis. There was weight loss of 8 kg reported due to postprandial epigastric pain. She said, that “. . . she would eat just because of sense of duty . . .” There was no indication in her medical history of gastrointestinal disorders or evidence of any abdominal surgery. To this date she had led an active life style.

Physical examination revealed an underweight woman, with a body-mass index of 16 kg/m<sup>2</sup> (calculated from a weight of 46 kg and height of 168 cm). Palpation and percussion of the abdomen did not set off any unpleasant sensation or pain during examination, or palpable mass. Auscultation of heart, lungs, and abdomen was normal. Pulse was well palpable in all extremities. The mental status was adequate. Results of routine laboratory analysis of her blood were normal; in particular, there were no signs of anemia.

Gastroscopy revealed gastritis with no indication of a *Helicobacter pylori* infection. The result of the colonoscopy was normal. Owing to the prolonged history of abdominal distress, without any

pathologic findings, the patient had been referred to a psychologist, who recommended antidepressive medication.

Eventually, an abdominal computed tomography scan was performed that revealed scant retroperitoneal fatty tissue, massive dilatation of the stomach and the proximal duodenum, and an abrupt cutoff on the third portion of the duodenum (Fig 1). The SMA-aortic angle was decreased to less than 14° (normal range, 30° to 45°)<sup>1,2</sup> (Fig 2). These findings suggested Wilkie syndrome. An upper gastrointestinal series demonstrated duodenal narrowing due to extrinsic compression of the third portion of the duodenum and marked delay in the passage of contrast medium of the stomach through the duodenal loop (Fig 3).

Because the initial conservative treatment failed, the patient was referred to our department for surgical treatment. An upper median laparotomy was performed under general anesthesia. The colon transversum and the small bowel were placed next to each other. After dissection of the retroperitoneum, the juxtarenal aorta was exposed by the senior author (W. S.). The obstruction of the duodenum due to a narrow angle between the aorta and the SMA was verified.

An intravenous bolus of 2500 IU heparin was administered, and the SMA was ligated with a suture right above the aorta and transversely dissected. The aorta was clamped tangentially at the infrarenal level with a Satinsky clamp, and a midline aortotomy was performed. Without shortening the SMA, an end-to-side anastomosis between the SMA and the aorta was performed with 5-0 polydioxanone suture in a single-stitch technique.

After declamping, the anastomosis was patent, with a good pulse distally. The duodenal obstruction was resolved by the transposition, and the laparotomy was closed. The patient's postoperative course was uneventful. The postoperative upper gastrointestinal series (Fig 4) and the reconstruction of the aorta and its branches in volume-rendering technique (Fig 5) showed normal morphology with no signs of duodenal obstruction.

The patient recovered well and was discharged home in good condition on postoperative day 10. During the 9-month follow-up, the patient remained in good physical condition. She reported

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**Fig 1.** Preoperative axial arterial phase computed tomography scan after contrast administration shows an obstruction of the horizontal part of the duodenum.



**Fig 2.** Sagittal reconstruction of an arterial phase preoperative computed tomography scan after contrast administration shows a narrow angle between the aorta and the superior mesenteric artery of  $14^\circ$ .

complete alleviation of the abdominal pain and gained weight rapidly after surgery.

## DISCUSSION

SMA syndrome is a rare entity in which the third part of the duodenum is compressed by the overlying SMA. It was first described in 1842 by C. von Rokitansky<sup>3</sup> and later on in 1927 by D. P. D. Wilkie<sup>4</sup> as “chronic duodenal ileus.” It



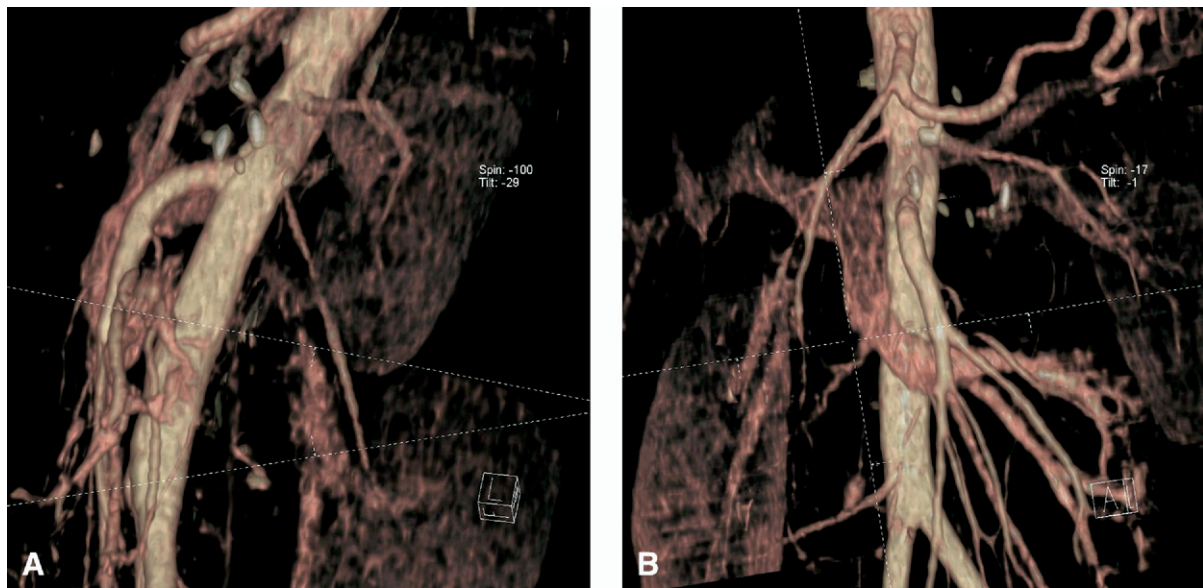
**Fig 3.** Preoperative barium follow-through study shows obstruction on the level of the superior mesenteric artery.



**Fig 4.** Postoperative barium follow-through study shows normal morphology.

is recognized by early satiety, recurrent vomiting, nausea, abdominal distention, weight loss, and postprandial distress. The anatomic features of this entity are a narrow angle between the aorta and the SMA, together with the high fixation of the duodenum by the ligament of Treitz or an anomalous SMA crossing directly over the aorta at its intersection with the duodenum, or both.

The literature consists of several case reports and single-center reports that vary widely in their description of the usual severity of the syndrome, the cause, and the treatment required. The SMA syndrome can occur after rapid weight loss from anorexia nervosa,<sup>5,6</sup> trauma,<sup>7,8</sup> brain injury,<sup>9</sup> orthopedic surgery for correction of spinal deformities,<sup>2,8</sup>



**Fig 5. A and B,** Postoperative volume-rendering technique reconstructions of a computed tomography scan show a distal transposition of the superior mesenteric artery (SMA) with a normal physiologic angle between the aorta and the SMA.

**Table.** Clinical data of four series of patients with Wilkie syndrome

First author, year	Patients, no.	Sex	Age, y, median (range)	Treatment	Outcome
Lee, <sup>14</sup> 1978	17	7 M/10 F	—	5 med 8 DJ 4 LLT 2 GJ*	8/17 no follow up 5/17 no S/S 4/17 persistent S/S
Weber, <sup>15</sup> 1979	14	7 M/10 F	30 (15-54)	No med 13 DJ 1 LLT	1/14 no follow-up 11/14 no S/S 2/14 persistent S/S
Gustafsson, <sup>16</sup> 1984	11	2 M/9 F	36 (17-67)	No med 10 DJ 1 GJ	11/11 no S/S 3/11 redo-operation due to adhesions or stenosed anastomosis
Ylinen, <sup>17</sup> 1989	18	1 M/17 F	28.5 (19-54)	No med 18 DJ	2/8 no follow-up 14/18 no S/S 2/18 persistent S/S

S/S, Signs and symptoms; med, medical; GJ, gastrojejunostomy; DJ, duodenojejunostomy; LLT, lysis of the ligament of Treitz.

\*Multiple procedures possible.

immobilization with a body cast (cast syndrome),<sup>10</sup> gastro-intestinal surgery, compression by the median arcuate ligament of the diaphragm, extrinsic compression by an abdominal aortic aneurysm,<sup>11,12</sup> cardiovascular surgery (correction after aortic coarctation), and even familial cases have been reported.<sup>13</sup>

Because Wilkie syndrome is very rare, it is usually diagnosed after ruling out other more common causes and thus can take some time. The initial treatment is usually conservative, and surgical intervention should be sought if it fails. In addition to simple release of the ligament of Treitz, several types of reconstruction such as laterolateral duodenojejunostomy or Roux-en-Y reconstruction are performed—recently also by using laparoscopy—for the reconstruction of the intestinal passage.

A review of the literature for Wilkie syndrome by using PubMed showed that duodenojejunostomy was considered to be the best procedure for severe cases (Table).<sup>1,14-17</sup> Such results were not achieved after gastrojejunostomy and lysis of the ligament of Treitz. Gastrojejunostomy provided adequate decompression of the stomach but was inadequate for releasing duodenal obstruction. Downward displacement of the duodenum was a difficulty that occurred in some patients after division of the ligament of Treitz, and the symptoms of obstruction persisted necessitating later duodenojejunostomy.

None of the published surgical concepts solved the anatomic problem itself, but performed a small-bowel bypass-procedure with accompanying disruption of the physiologic intestinal continuity. Unfortunately, opera-

tions have their own complications such as suture insufficiency or suture stenosis. Patients are also more likely to encounter dumping syndrome.

## CONCLUSION

To our knowledge, this case report is first the description of a new surgical approach in the therapy of Wilkie syndrome. It consists of removal of the compressing SMA, thus avoids contact and compression of the duodenum. The transposition of the SMA in the infrarenal aorta is already an established and standardized vascular surgical procedure and a valuable treatment option in patients with chronic visceral ischemia. The SMA syndrome can be successfully treated by transposition of SMA without disrupting the continuity of the small bowel.

## REFERENCES

1. Dietz UA, Debus ES, Heuko-Valiati L, Valiati W, Friesen A, Fuchs KH, et al. Das aortomesenteriale Kompressionssyndrom. *Chirurg* 2000;71:1345-51.
2. Zhu Z, Qiu Y. Superior mesenteric artery syndrome following scoliosis surgery: its risk indicators and treatment strategy. *World J Gastroenterol* 2005;11:3307-10.
3. Von Rokitansky C. *Lehrbuch der pathologischen Anatomie*. 1st edition. Vienna: Braumüller & Seidel; 1842.
4. Wilkie DPD. Chronic duodenal ileus. *Am J Med Sci* 1927;173:643-9.
5. Elbadawy MH. Chronic superior mesenteric artery syndrome in anorexia nervosa. *Br J Psychiatry* 1992;160:552-4.
6. Sours JA, Vorhaus LJ. Superior mesenteric artery syndrome in anorexia nervosa: a case report. *Am J Psychiatry* 1981;138:519.
7. Lescher TJ, Sirinek KR, Pruitt BA Jr. Superior mesenteric artery syndrome in thermally injured patients. *J Trauma* 1979;19:567-71.
8. Loeb T, Loubert G, Morsly R, Gabillet JM, Pasteyer J. Superior mesenteric artery syndrome. *Ann Fr Anesth Reanim* 1999;18:1000-4.
9. Pedoto MJ, O'Dell MW, Thrun M, Hollifield D. Superior mesenteric artery syndrome in traumatic brain injury: two cases. *Arch Phys Med Rehabil* 1995;76:871-5.
10. Hall LW. The cast syndrome incognito. *Am J Surg* 1974;127:371-6.
11. Sostek M, Fine SN, Harris TL. Duodenal obstruction by abdominal aortic aneurysm. *Am J Med* 1993;94:220-1.
12. Bhama JK, Ogren J, Guinn G, Fisher WE. Unique cause of duodenal obstruction by an abdominal aortic aneurysm. *J Vasc Surg* 2001;34:1130-2.
13. Iwaoka Y, Yamada M, Takehira Y, Hanajima K, Nakamura T, Murohisa G, et al. Superior mesenteric artery syndrome in identical twin brothers. *Intern Med* 2001;40:713-5.
14. Lee CS, Mangla JC. Superior mesenteric artery compression syndrome. *Am J Gastroenterol* 1978;70:141-50.
15. Weber H, Gumrich H, Klotz E. Arteriommesenteric duodenal compression. *Chirurg* 1979;50:503-7.
16. Gustafsson L, Falk A, Lukes PJ, Gamklou R. Diagnosis and treatment of superior mesenteric artery syndrome. *Br J Surg* 1984;71:499-501.
17. Ylinen P, Kinnunen J, Hockerstedt K. Superior mesenteric artery syndrome. A follow-up study of 16 operated patients. *J Clin Gastroenterol*. 1989;11:386-91.

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