Case Report ISSN 2639-9334

Gastroenterology, Hepatology & Digestive Disorders

"Wilkie's Syndrome; Diagnosis and Surgical Treatment a Case Report and Review"

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Received: 06 May 2026; **Accepted:** 08 Jun 2026; **Published:** 19 Jun 2026

Citation: Daniela Elizabeth Caro López, Alma Jocelyn-Rosas Zamora, Jaime Ponce De León Palomares. "Wilkie's Syndrome; Diagnosis and Surgical Treatment a Case Report and Review". Gastroint Hepatol Dig Dis. 2026; 9(2): 1-5.

ABSTRACT

Superior mesenteric artery syndrome (SMAS) is a rare cause of duodenal obstruction resulting from compression of the third portion of the duodenum between the aorta and the superior mesenteric artery (SMA), with nonspecific clinical manifestations that may be similar to functional dyspepsia or eating disorders, which often delays diagnosis. We present the case of a young fitness trainer who presented with early postprandial satiety, abdominal pain that improved in the genupectoral position, and progressively worsening intolerance to oral intake, associated with a weight loss of 9 kg in one year. Endoscopic evaluation and CT angiography revealed a reduced aortomesenteric angle and duodenal compression consistent with SMAS. She underwent laparoscopic duodenojejunostomy with a favorable postoperative course, adequate oral tolerance, and weight regain. This case highlights the importance of maintaining a high index of clinical suspicion in young patients with weight loss and nonspecific symptoms of obstruction; once the diagnosis is confirmed, the safest and most effective surgical option in treatment-refractory cases is minimally invasive duodenojejunostomy.

Keywords

Case report, Duodenal obstruction, Duodenojejunostomy, Superior mesenteric artery syndrome, Wilkie's syndrome.

Introduction

Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome or Cast's syndrome, is a rare cause of proximal intestinal obstruction resulting from compression of the third portion of the duodenum between the superior mesenteric artery (SMA) and the abdominal aorta [1]. It has an incidence of 0.013% to 0.03%, with a higher frequency in young women at a ratio of 3:2. The pathophysiology is due to a decrease in the aorto-mesenteric angle caused by loss of the mesenteric fat pad that separates the two structures, reducing the aorto-mesenteric space from 10–28 mm to 2–8 mm; under normal conditions, this angle typically measures 38° to 65° [2].

It can be congenital or acquired. The acquired causes involve significant weight loss, such as eating disorders, malabsorption disorders, post-bariatric surgery, and hypermetabolic conditions (burns, sepsis, cancer). They have also been observed in healthy individuals with a low body mass index, such as athletes or people with scant retroperitoneal adipose tissue. Among the pediatric population, a congenitally short or hypertrophic ligament of Treitz is the etiological factor [3].

Clinical manifestations are nonspecific symptoms of upper intestinal obstruction, such as postprandial fullness, early satiety, nausea, vomiting of alimentary or biliary contents, cramp-like abdominal pain localized in the epigastrium that intensifies in the supine position and is relieve in the lateral position (genupectoral position), and progressive weight loss [4]. The diagnosis is confirmed by CT angiography, which shows an aortomesenteric angle ≤25° and an aortomesenteric space distance ≤8 mm [5].

The initial management is conservative, consisting of gastrointestinal decompression, nutritional support, and correction of electrolyte imbalances; this noninvasive treatment option have a successful rate of nearly 70%, with recurrence occurring in up to 16% of cases. Refractory cases require surgical intervention, and the preferred technique is laparoscopic duodeno-jejunostomy due to its high success rate of >90% [2,3].

Case Presentation

A 29-year-old female patient, a physical trainer, without any history of degenerative diseases. Her surgical history includes a laparoscopic appendectomy at age 15. She reports occasional alcohol and tobacco consumption.

Her symptoms began one year ago and consisted of early postprandial fullness associated with abdominal pain localized in the epigastrium, of a cramp-like nature, which improved in the genupectoral position. She was treated conservatively with prokinetic agents, proton pump inhibitors, and diet modifications; however, the patient has shown gradual clinical deterioration, characterized by an intolerance to eating solid foods, and weight loss about 9 kg (BMI 18.29 kg/m²) during 9 months, and required 3 hospitalizations to treat electrolyte imbalances and signs of dehydration and malnutrition.

At the time of his last hospitalization, an endoscopy was performed, revealing gastric retention and a narrowed lumen in the third portion of the duodenum (Figure 1). Given the high suspicion of Wilkie syndrome, an abdominopelvic CT angiography was performed, showing a reduction in the aortomesenteric space from 7.06 mm to 5.74 mm, and an aortomesenteric angle of 28.25°, as well as gastric and proximal duodenal dilation, confirming the diagnosis of superior mesenteric artery syndrome (Figure 2).

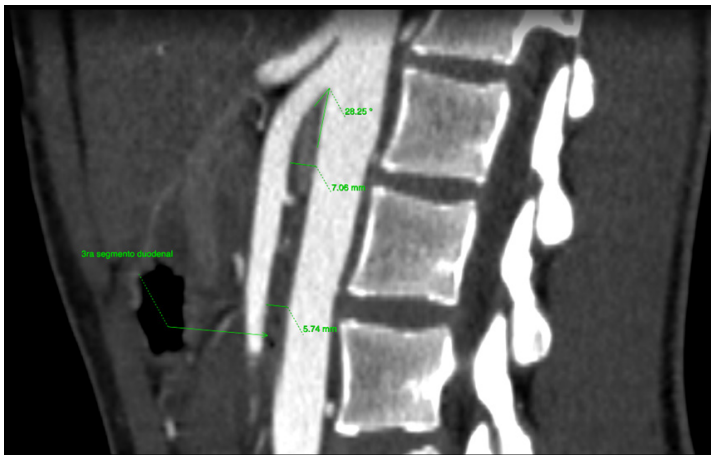

Figure 2: Angiotomography sagittal section at the level of the third portion of the duodenum, showing a reduction in the aortomesenteric space from 7.06 mm to 5.74 mm and an angle of 28.25°.

Based on these findings and the failure of conservative management, the decision was to proceed with a surgical intervention. A laparoscopic duodenojejunostomy was performed with balanced general anesthesia, and the patient was placed in the French position. Four trocars were placed: One 12-mm and one 5-mm trocar on the midclavicular line on the left flank, a third 5-mm trocar on the right flank on the midclavicular line, and a fourth trocar in the umbilical scar for the placement of the 30° endoscope. The surgeon positioned himself to the patient's left and the first assistant to the right (Figure 3).

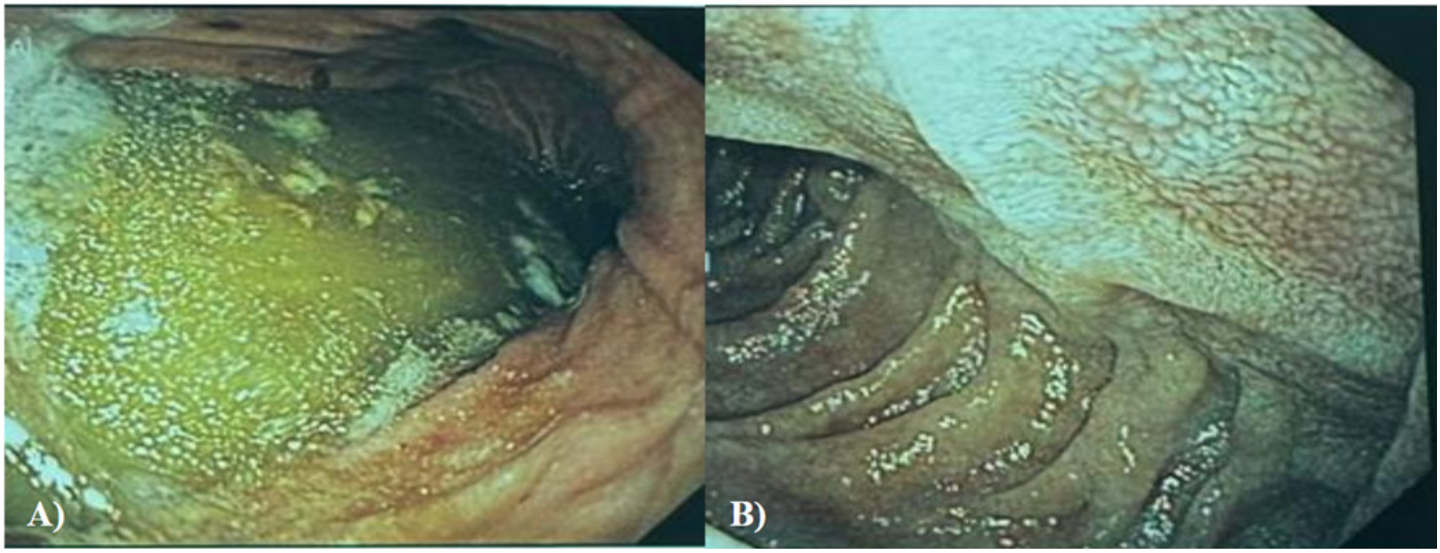

Figure 1: A) Endoscopy in the anteversion position showing a distended stomach containing food residues. B) Third portion of the duodenum with a narrowed lumen due to extrinsic compression.

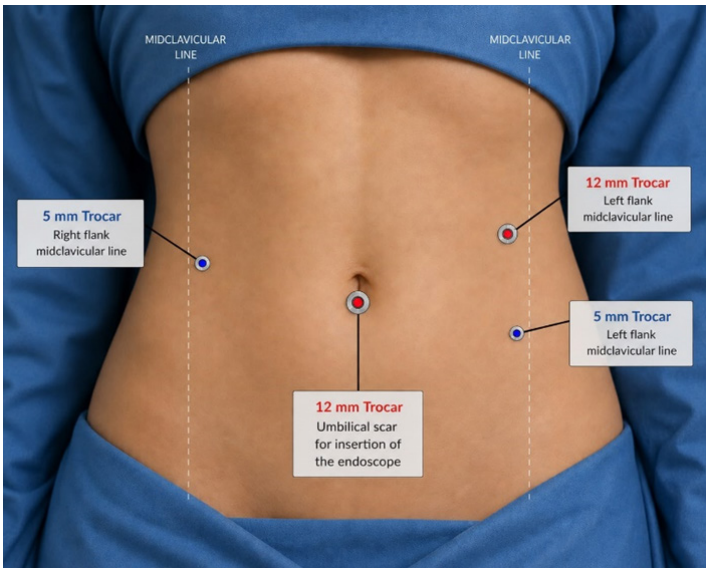

Figure 3: Placement of laparoscopic ports.

Laparoscopic examination revealed gastric dilatation, as well as dilatation of the proximal duodenum extending to the origin of the superior mesenteric artery. The retroperitoneum was opened until the ligament of Treitz was identified; the jejunal segment was counted 30 cm distal to the duodenojejunal junction, marked with a staple, and the selected jejunal segment was sutured to the duodenal segment with simple 2-0 prolene sutures, where a duodenostomy and jejunostomy were performed (Figure 4).

A mechanical isoperistaltic side-to-side enteric anastomosis was performed using a 60-mm endoGIA stapler with white staples (2.5 mm). The common enterostomy was closed in two layers: the first with Connell-Mayo sutures and the second with Lembert sutures, using 2-0 Monocryl (Figure 5). A 10-mm closed silicone drain was placed proximal to the anastomosis, and the ports were closed conventionally.

At 24 hours postoperatively, the patient started oral intake of clear liquids, which were well tolerated, and at 48 hours, an endoscopy was performed, revealing adequate patency in the third portion of the duodenum with an intact anastomosis (Figure 6). The drain was removed on the second day. The patient's recovery was satisfactory, with progressive oral tolerance, and she was discharged from the hospital on the fourth day.

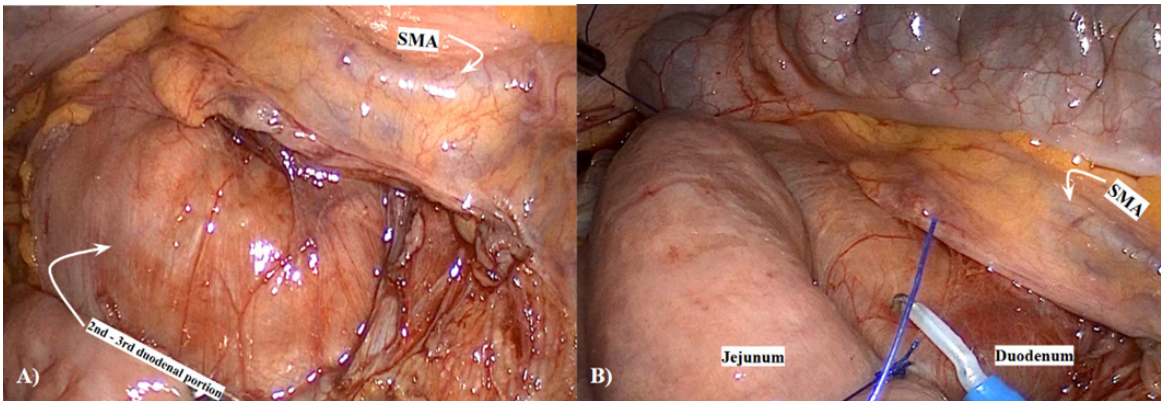

Figure 4: A) Retroperitoneal dissection with exposure of the superior mesenteric artery (SMA) and the second and third portions of the duodenum. B) Duodenostomy and jejunostomy 30 cm from the angle of Treitz.

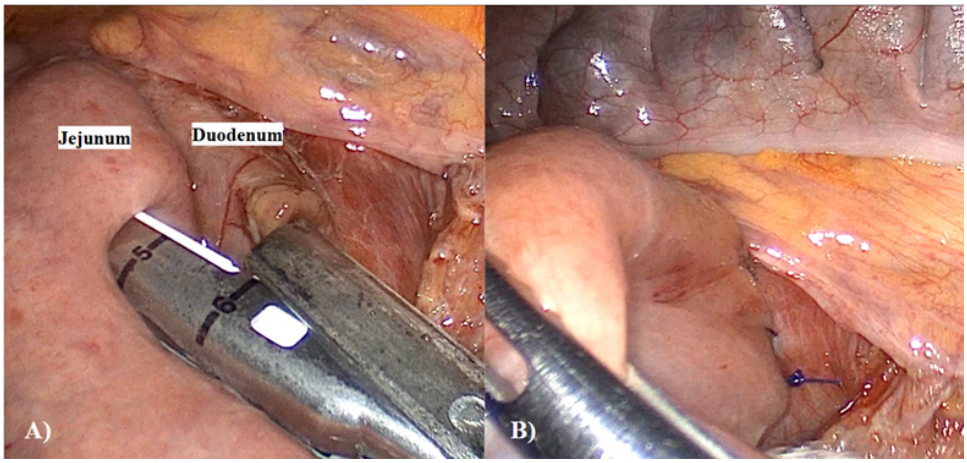

Figure 5: A) Mechanical isoperistaltic side-to-side enteric anastomosis. B) Reinforcement of enterotomies with Connell-Mayo and Lembert sutures

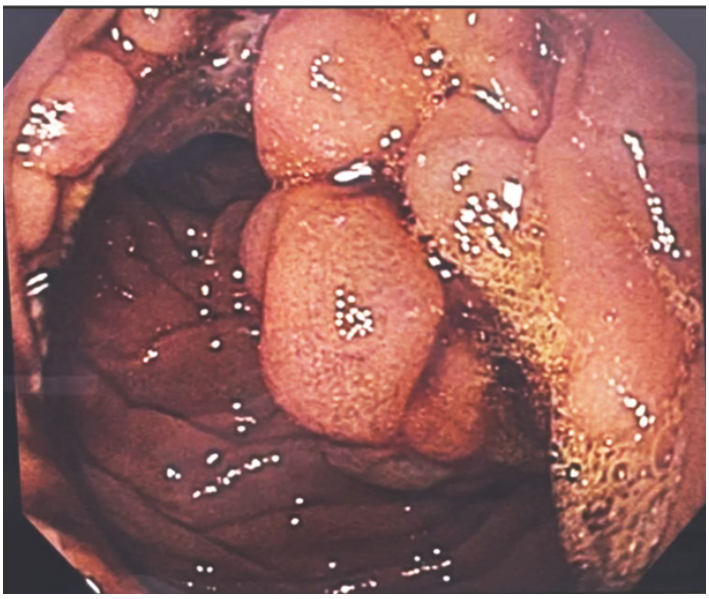

Figure 6: Endoscopy showing integrity of the enter-enter anastomosis with adequate passage through the 3rd duodenal portion.

Discussion

Superior mesenteric artery syndrome (SMAS) is a rare and underdiagnosed condition that is characterized by extrinsic compression of the third portion of the duodenum between the superior mesenteric artery and the abdominal aorta caused by a decrease in the aortomesenteric angle as a result of decreased retroperitoneal fat thickness. It has been reported in cases of intestinal malrotation, anorexia nervosa, severe burns, cancer, malabsorption syndromes, neurological disorders such as cerebral palsy, and patients who are post-operative for scoliosis [1,5]. The pathophysiology is due to rapid weight loss, even in the absence of comorbidities, but in patients with low body fat percentages, such as athletes or patients with high metabolic demands secondary to acute diseases, or bariatric patients [6].

The symptoms are vague, with chronic episodes of intestinal subocclusion that are often mistaken for functional dyspepsia or gastroparesis, delaying diagnosis. Clinical features include postprandial fullness, nausea, and vomiting of gastrobiliary contents along with progressive weight loss and abdominal pain that usually improves by adopting positions that reduce mesenteric tension, such as the left lateral decubitus position or genupectoral position [7,8].

The gold standard in diagnostic imaging studies is contrast-enhanced CT angiography, which allows measurement of the aortomesenteric angle (<25°) and the aortoduodenal distance (<8 mm). Additional imaging modalities, useful for supporting the diagnosis include a simple abdominal X-ray, which reveals the gas in the stomach and the initial portions of the duodenum. Barium fluoroscopy shows interruption of contrast passages in the third portion of the duodenum and antiperistalsis proximal to the obstruction site [9].

Endoscopic findings are gastric and proximal duodenal dilation with retention of digestive contents and secondary esophageal, gastric, or duodenal ulcers caused by alkaline reflux [10].

The initial treatment is a conservative approach based on a high-calorie diet, gastric decompression, changes in posture to the lateral decubitus position, hydroelectrolytic correction, and use of prokinetics, all with the aim of increasing visceral fat. Medical management has a success rate of 70% [11]. There have been reports of severe malnutrition requiring total parenteral nutrition or nasojejunal tube feeding. However, in patients with chronic disease or persistent symptoms after 6 weeks of conservative management, surgical intervention should be considered [12].

Surgical options include laparoscopic, open, or robotic gastrojejunostomy; gastroduodenostomy; duodenojejunostomy; the Strong procedure; anterior transposition of the third portion of the duodenum; duodenal descent; the Ladd procedure; and transabdominal duodenojejunostomy, which are some of the treatments described; however, laparoscopic duodenojejunostomy continues to be the surgical technique of choice, with success rates higher than 90% [2,13].

Conclusion

Superior mesenteric artery syndrome is an uncommon cause of upper intestinal obstruction, but it must be considered in thin young patients or those with a history of significant weight loss, who present with symptomatology of postprandial fullness, progressively worsening oral intolerance, and abdominal pain that gets better in the left lateral decubitus position. The diagnosis requires a high index of suspicion and angiotomography to measure the aortomesenteric space, which confirms its narrowing and the compression of the third portion of the duodenum, in the absence of any other cause. The surgical treatment with laparoscopic duodenojejunostomy is a safe and efficient alternative with excellent functional outcomes.

In this case, the patient had a background of strenuous exercise. The clinical presentation in her case was insidious, developing over several years and resulting in a delayed diagnosis, perhaps due to her low body fat percentage and intense physical activity, which led to mild malnutrition according to NRS criteria. Therefore, since there was no improvement with conservative management, a decision was made to perform minimally invasive surgery through a duodenojejunostomy this surgical treatment completely resolved the symptoms and allowed for a sustained weight gain, which yielded favorable results, complete recovery, and weight regain.

Early recognition and a multidisciplinary approach are essential to prevent serious nutritional complications and improve the patient's quality of life.

Acknowledgments

I extend my sincerest gratitude to Dr. Jaime Ponce De León Palomares for his valuable advice, expertise, and methodological

support throughout the clinical, surgical, and scientific management of this case. I would also like to thank Dr. José Sergio Verboonen Sotelo and Dr. Jeffry Romero Manzano for providing access to institutions for surgical practice and for contributing academic resources that significantly aided in the accomplishment of this work.

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