

Wilkie's Syndrome as an Uncommon Cause of Duodenal Obstruction: Case Report

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ABSTRACT

Wilkie's syndrome, also known as superior mesenteric artery syndrome, is a rare clinical entity characterized by compression of the third portion of the duodenum between the abdominal aorta and the superior mesenteric artery, leading to partial or complete duodenal obstruction. Due to its low prevalence and the nonspecific nature of its clinical presentation, diagnosis is often delayed, which can result in nutritional deterioration and the development of complications. We present the case of a young female patient with postprandial abdominal pain, nausea, recurrent vomiting, and significant weight loss, in whom imaging studies confirmed a decreased aortomesenteric angle and duodenal compression consistent with Wilkie's syndrome. Conservative management was initially attempted, but clinical improvement was not achieved, so surgical treatment via open duodenoduodenostomy was performed. Postoperative evolution was favorable, with resolution of symptoms, adequate oral intake tolerance, and progressive improvement of nutritional status. This case report highlights the importance of considering Wilkie's syndrome in the differential diagnosis of upper intestinal obstruction, as well as the relevance of timely diagnosis and appropriate therapeutic intervention to improve patient outcomes.

KEYWORDS: Wilkie's syndrome, duodenal obstruction, superior mesenteric artery, duodenoduodenostomy, extrinsic duodenal compression, weight loss.

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INTRODUCTION

Wilkie's syndrome, also called superior mesenteric artery syndrome, is a rare gastrointestinal disorder characterized by extrinsic compression of the third portion of the duodenum between the superior mesenteric artery and the abdominal aorta. This compression occurs due to a reduction in the aortomesenteric angle and distance, resulting in partial or complete duodenal obstruction. Although its incidence is low, it represents a relevant cause of upper intestinal obstruction and should be considered in the differential diagnosis of patients with persistent gastrointestinal symptoms.

Pathophysiologically, Wilkie's syndrome is mainly associated with the loss of mesenteric fat, which normally acts as a cushion between vascular structures and the duodenum. This condition is often observed in patients with significant and rapid weight loss, malnutrition, eating disorders, chronic diseases, hypercatabolic states, prior surgeries, or prolonged immobilization. Certain anatomical or postural conditions can also contribute to the development of this syndrome.

The clinical presentation of Wilkie's syndrome is variable and often nonspecific, which makes early recognition

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difficult. The most frequent symptoms include postprandial abdominal pain, early satiety, nausea, bilious vomiting, and progressive weight loss, creating a vicious cycle in which intestinal obstruction exacerbates malnutrition, which in turn

perpetuates duodenal compression. Due to these nonspecific symptoms, diagnosis is often delayed, and the patient may initially be evaluated by various specialties.

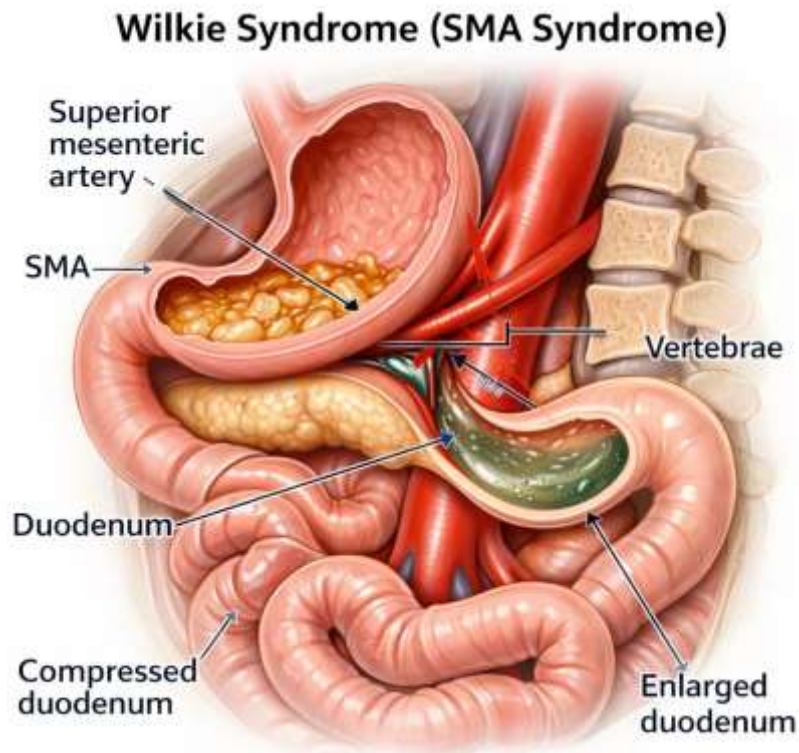


Figure 1: illustrative image of Wilkie's syndrome.

Diagnosis relies primarily on imaging studies, with contrast-enhanced abdominal computed tomography being the method of choice. This study allows visualization of gastric and proximal duodenal dilation, as well as reduction of the aortomesenteric angle and distance. Other diagnostic tools, such as upper digestive endoscopy or contrast studies, can help rule out differential diagnoses and support clinical suspicion.

Management of Wilkie's syndrome depends on the severity of the clinical picture and response to initial treatment. Conservative management is recommended as a first-line approach, focusing on gastric decompression, correction of fluid and electrolyte imbalances, and nutritional rehabilitation. However, in refractory cases or those with significant clinical compromise, surgical treatment is an effective option, with duodenoduodenostomy being the most commonly employed procedure.

The following case report aims to emphasize the importance of timely recognition of Wilkie's syndrome as a rare cause of duodenal obstruction, describing its clinical presentation, diagnostic approach, and clinical course, thus contributing to awareness and clinical suspicion of this uncommon entity.

CASE REPORT

A 24-year-old female patient, with no relevant personal or surgical history, presented to the emergency department with

a six-month history of recurrent abdominal pain, primarily located in the epigastrium and mesogastrium, progressively worsening, and exacerbated after meals. The pain was accompanied by persistent nausea, frequent postprandial vomiting of food and bilious content, early satiety, and involuntary weight loss estimated at 12 kg over the previous four months.

The patient reported that symptoms worsened in the supine position and partially improved in the left lateral decubitus or knee-chest position. She denied fever, diarrhea, gastrointestinal bleeding, chronic medication use, or history of eating disorders. Physical examination revealed a conscious, oriented patient with signs of malnutrition, hemodynamically stable, and a body mass index of 17 kg/m². The abdomen was flat, soft, and depressible, with deep epigastric tenderness, without palpable masses or signs of peritoneal irritation. Bowel sounds were present and increased.

Initial laboratory studies revealed normal hemoglobin, leukocytes, and biochemical parameters, with no relevant electrolyte abnormalities. Upper digestive endoscopy showed dilation of the stomach and first and second portions of the duodenum with retained contents, without evidence of intraluminal obstruction or visible extrinsic compression.

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Due to persistent symptoms, contrast-enhanced abdominal CT was performed, showing marked dilation of the stomach and proximal duodenum, with narrowing of the third portion of the duodenum caused by compression between the superior mesenteric artery and the abdominal aorta. A significantly decreased aortomesenteric angle was observed, compatible with Wilkie's syndrome. Based on clinical and radiological findings, a definitive diagnosis was established. Conservative management was initiated with nasogastric decompression, bowel rest, correction of fluid and electrolyte

imbalances, and progressive nutritional support. Despite these measures, the patient showed minimal clinical improvement, with persistent vomiting and oral intolerance, prompting surgical consultation.

An open surgical intervention was performed with a side-to-side duodenoduodenostomy. The procedure was uneventful. Postoperatively, the patient remained under observation, gradually resuming oral intake from day four, with good tolerance. Abdominal pain and vomiting resolved completely.

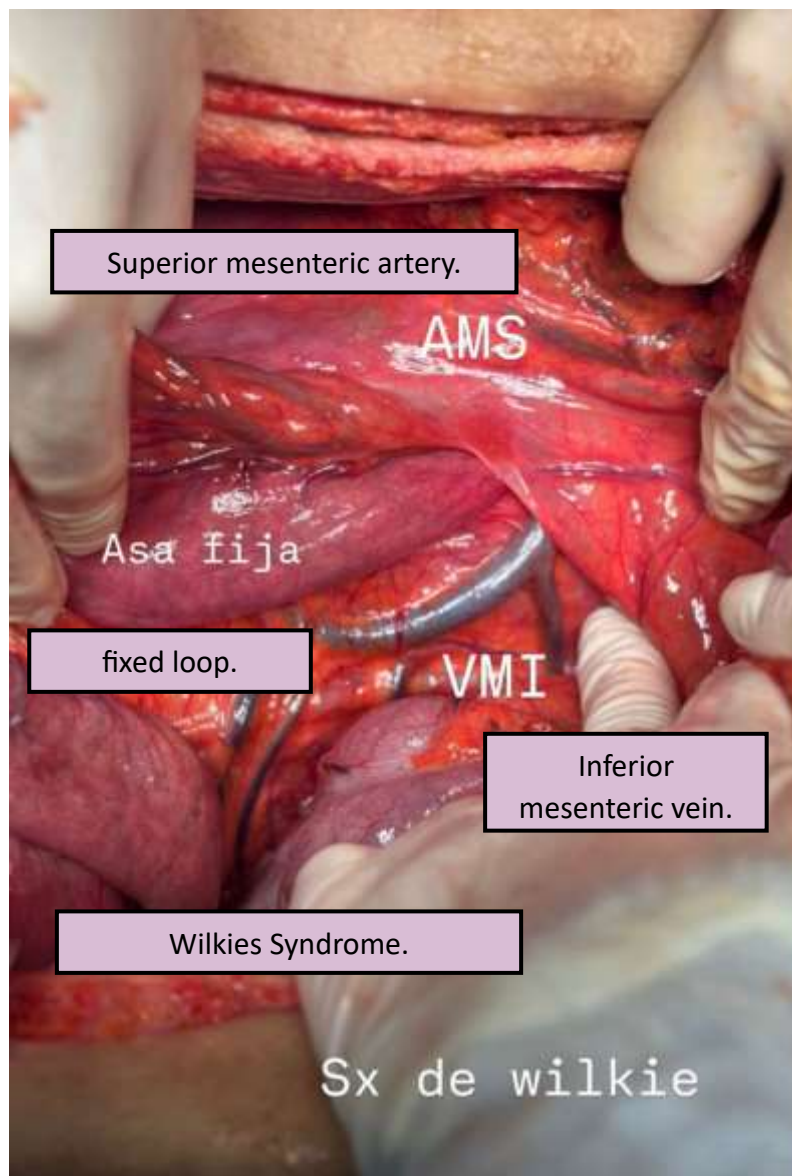


Figure 2: intraoperative finding.

Subsequent evolution was favorable, with progressive weight gain and improvement of nutritional status. The patient was discharged in good general condition and continues outpatient follow-up, with no recurrence of symptoms to date.

DISCUSSION

Wilkie's syndrome, also known as superior mesenteric artery syndrome, is a rare and underdiagnosed clinical entity characterized by compression of the third portion of the

duodenum between the abdominal aorta and the superior mesenteric artery. Its low prevalence and nonspecific presentation contribute to diagnostic delays, which can result in progressive malnutrition and increased morbidity and mortality. It is more commonly described in young adults, particularly women, and is often associated with significant weight loss, malnutrition, and reduction of retroperitoneal fat. Several predisposing factors have been identified in the literature, summarized in Table 1. These include rapid weight

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loss, nutritional disorders, chronic illnesses, prior surgeries, and hypercatabolic states, all contributing to a reduced aortomesenteric angle and promoting duodenal compression.

Table 1. Risk factors associated with Wilkie’s syndrome

Category	Associated factors
Nutritional	Rapid weight loss, malnutrition, anorexia nervosa
Medical	Chronic diseases, hypercatabolic states
Surgical	Bariatric surgery, scoliosis correction
Traumatic	Severe trauma, burns
Postural	Prolonged immobilization

Pathophysiologically, the main mechanism involves a decrease in the angle and distance between the superior mesenteric artery and the abdominal aorta, causing extrinsic duodenal obstruction. Normal and pathological values of

these anatomical parameters are described in Table 2, highlighting a vicious cycle in which duodenal obstruction leads to oral intolerance and weight loss, perpetuating the clinical picture.

Table 2. Normal and pathological anatomical parameters

Parameter	Normal	Wilkie’s Syndrome
Aortomesenteric angle	25°–60°	< 22°
Aortomesenteric distance	10–28 mm	< 8 mm
Duodenal passage	Normal	Partial or absent

The clinical presentation of Wilkie’s syndrome is variable and frequently nonspecific, necessitating consideration of

multiple differential diagnoses, particularly other causes of upper intestinal obstruction (Table 3).

Table 3. Differential diagnosis of Wilkie’s syndrome

Pathology	Differential features
Pyloric stenosis	Intraluminal obstruction
Chronic pancreatitis	Radiating pain, enzymatic alterations
Duodenal neoplasms	Mass visible on imaging
Paralytic ileus	No mechanical obstruction
Functional disorders	Normal imaging

Diagnosis relies primarily on imaging studies, with contrast-enhanced CT being the method of choice due to its ability to evaluate vascular and duodenal anatomy. Other

complementary studies and their utility are summarized in Table 4.

Table 4. Diagnostic methods in Wilkie’s syndrome

Method	Utility	Limitations
CT scan	Confirms diagnosis	Radiation exposure
Upper endoscopy	Rules out intraluminal cause	Cannot measure angle
Barium study	Evaluates functional obstruction	Lower specificity
MRI	Radiation-free alternative	Less availability

Management depends on clinical severity and response to initial treatment. Conservative management is first-line in selected cases; however, persistent symptoms or progressive

nutritional deterioration require surgical intervention. Therapeutic options and indications are summarized in Table 5.

Table 5. Therapeutic options based on clinical course

Type of management	Indications	Expected outcome
Conservative	Mild cases	Clinical improvement

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Nutritional support	Severe malnutrition	Weight recovery
Open surgery	Failed conservative management	Definitive resolution
Duodenoduodenostomy	Procedure of choice	High success rate

Several surgical techniques have been described (Table 6). Duodenoduodenostomy, performed either open or laparoscopically, remains the most accepted procedure due to

favorable long-term outcomes. In the present case, the open approach allowed definitive resolution of the clinical picture with significant nutritional improvement.

Table 6. Described surgical techniques

Technique	Advantages	Disadvantages
Duodenoduodenostomy	High efficacy	Invasive procedure
Gastrojejunostomy	Technical alternative	Does not correct pathophysiology
Division of the ligament of Treitz	Less invasive	Variable outcomes

Wilkie's syndrome should be considered in young patients with upper obstructive symptoms and significant weight loss. Timely recognition and appropriate therapeutic intervention prevent complications and improve prognosis, as demonstrated in this case report.

CONCLUSION

Wilkie's syndrome represents a rare but clinically significant entity within the differential diagnosis of upper intestinal obstruction, especially in young patients with significant weight loss and persistent gastrointestinal symptoms. Its nonspecific clinical presentation and low prevalence often result in delayed diagnosis, favoring nutritional deterioration and increased risk of complications.

This case emphasizes the importance of maintaining a high index of suspicion in patients with postprandial abdominal pain, nausea, recurrent vomiting, and oral intolerance, particularly when associated with malnutrition or progressive weight loss. Accurate interpretation of imaging studies, especially contrast-enhanced abdominal CT, is crucial for confirming the diagnosis by evaluating the aortomesenteric angle and distance and identifying characteristic duodenal compression.

Although conservative management is first-line in selected cases, focusing on nutritional recovery and gastric decompression, it is not always sufficient. As observed in the present case, lack of response to medical treatment requires timely surgical intervention. Open duodenoduodenostomy remains an effective and safe therapeutic option, with high success rates and definitive symptom resolution, especially in patients with significant clinical compromise or prolonged disease course.

This case also highlights the importance of a multidisciplinary approach involving gastroenterology, general surgery, nutrition, and radiology to optimize diagnosis, treatment, and follow-up. Comprehensive management ensures resolution of duodenal obstruction, nutritional recovery, and improved long-term quality of life. In conclusion, although rare, Wilkie's syndrome must be promptly recognized to avoid diagnostic and therapeutic

delays. Case reports such as this contribute to increasing awareness, promoting early identification, and establishing appropriate therapeutic strategies, ultimately leading to better clinical outcomes for affected patients.

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