

Case Report

Wilkie syndrome as a cause of upper intestinal obstruction and its minimally invasive surgical management: a case report and review of the literature

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ABSTRACT

Upper bowel obstruction is a diagnostic and therapeutic challenge due to the proximity of vascular structures and the anatomical constraints imposed by the thorax. Wilkie syndrome, a rare cause of upper GI obstruction, is associated with severe aortomesenteric compression and is usually diagnosed in children and adolescents. We present the case of an 18-year-old male patient with postprandial fullness, recurrent vomiting and epigastric pain. Contrast tomography showed severe gastric dilatation, a reduced aortomesenteric angle (11°) and an aortomesenteric distance of 3 mm, confirming the diagnosis. Endoscopy showed signs of gastric ischaemia with suspected perforation. Emergency diagnostic laparoscopy was performed and gastric perforation with mediastinal communication and duodenal ischaemia was identified. A partial gastrectomy with latero-lateral duodeno-jejunal anastomosis was performed. The postoperative course was favourable with tolerance to diet on day 3 and no complications at 3 months follow-up. Laparoscopic surgery represents a safe and effective alternative in the management of Wilkie's syndrome, reducing morbidity and optimising postoperative recovery.

Keywords: Wilkie syndrome, Upper GI obstruction, Aortomesenteric compression, Laparoscopic surgery, Minimally invasive, Gastrointestinal surgery, Surgical management, Partial gastrectomy, Duodenojejunal anastomosis

INTRODUCTION

Upper bowel obstruction is a diagnostic and therapeutic challenge, given the proximity of vital structures such as the aorta and the superior mesenteric artery (SMA). MSA syndrome, also known as Wilkie syndrome or Cast syndrome, is defined by compression of the third segment of the duodenum between the MSA and the aorta, attributable to a narrowing of the aortomesenteric angle. The first description of this phenomenon is attributed to Rokitansky, who mentioned it in his 1861 work.¹ Subsequently, between 1921 and 1927, Wilkie described a set of 75 cases and established the clinical and anatomical basis of this entity.²

Currently, the pathogenesis of the syndrome is mostly related to the loss of retroperitoneal adipose tissue, which acts as a natural buffer between the MSA and the aorta; this fat depletion, common in catabolic states, rapid weight loss, malnutrition and eating disorders, favours narrowing of the angle.³ Anatomical factors, such as a short ligament of Treitz or variations in the insertion of the MSA, may also predispose to the development of this compression.^{3,4}

Diagnosis of the syndrome is based on clinical suspicion and confirmed by imaging studies. Computed tomography (CT) with three-dimensional reconstruction allows accurate assessment of the aortomesenteric angle and distance, positioning it as the diagnostic modality of reference.⁴ Similarly, abdominal ultrasound offers a non-invasive, inexpensive but operator-dependent alternative. Early detection is crucial to avoid potentially serious complications such as ischaemia, perforation or necrosis of the duodenum.^{4,5}

Initial management of Wilkie's syndrome is conservative, focusing on gastric decompression and correction of nutritional disturbances. However, in cases where conservative treatment fails or complications arise, surgical intervention is required and laparoscopic duodenojejunostomy has become the technique of choice due to its high efficacy and low morbidity rate.⁵

The present case report describes the evolution of an 18-year-old patient who presented with symptoms of upper gastrointestinal obstruction. The integration of historical data with current diagnostic and therapeutic advances illustrates the complexity and comprehensive management of this rare entity.

CASE REPORT

The patient was an 18-year-old male, previously healthy, who presented to the emergency department with a 48-hour postprandial fullness and repeated vomiting (10 episodes) of gastric contents. He denied fever and had no neurological changes.

On physical examination, the abdomen was visibly distended, with pain on palpation in the epigastrium and

peristaltic rumbling in the mesogastrium. There was no evidence of peritoneal irritation. Table 1 shows the laboratory results on admission, noting the absence of leucocytosis and neutrophilia.

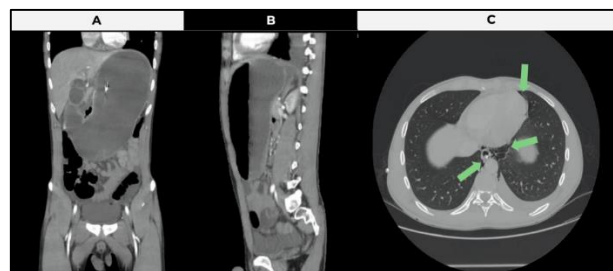


Figure 1: Contrast abdominopelvic tomography scan. Gastric distension and thin wall; dilatation of the duodenum in the coronal (A) and sagittal (B) planes. Periesophageal and pericardial gas is seen (C).

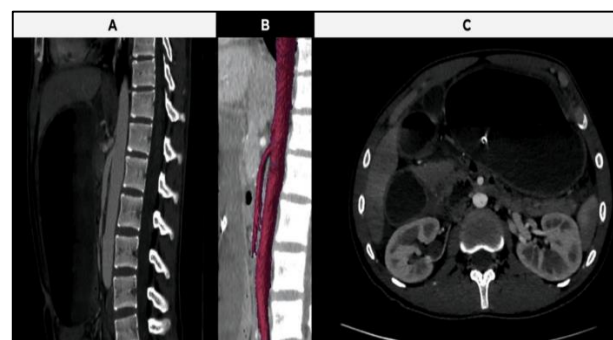


Figure 2: Measurement of aortomesenteric angle and distance. (A and B) Measurement of the aortomesenteric angle (C) Measurement of the aortomesenteric distance. Note the marked reduction which favours extrinsic compression of the third duodenal portion.

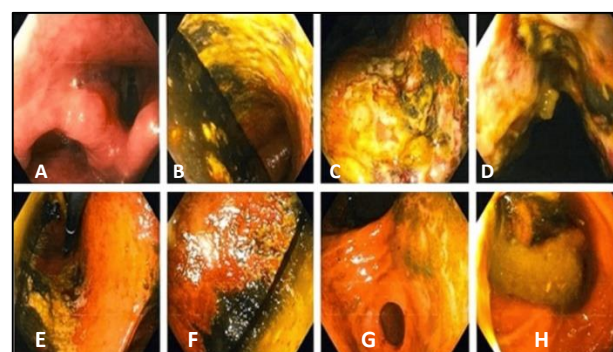


Figure 3 (A-H): Endoscopy.

Contrast-enhanced abdominopelvic computed tomography (CT) showed a markedly distended stomach with a hydro-aerial level, gas around the oesophagus and in the pericardial region, and a thin gastric wall. A transitional zone was observed in the third portion of the duodenum, which was dilated to a diameter of 40 mm. In addition, an aortomesenteric angle of 11° and a distance of

3 mm were measured, findings consistent with extrinsic compression (Figure 1 and Figure 2).

Upper gastrointestinal endoscopy showed necrosis of the gastric mucosa on the posterior aspect with ulceration; perforation was not ruled out due to the large amount of food debris that could not be aspirated. Extrinsic compression of the third duodenal portion was also evident (Figure 3). Gastric necrosis at the posterior aspect with ulceration; gastric perforation cannot be excluded due to the abundance of food debris which prevented complete aspiration. Extrinsic compression is seen in the third duodenal portion.

In view of these findings, an urgent diagnostic laparoscopy was indicated. During the operation, the presence of a perforation in the gastric fundus (posterior aspect) with communication to the mid-mediastinum was confirmed, as well as ischaemia proximal to the transition zone in the third duodenal portion. A partial gastrectomy was performed with an articulated Echelon Flex stapler (60 mm×4.1 mm) and a lateral-lateral duodeno-jejunal

anastomosis was created with a stapler (60 mm×2.6 mm). The closure line was reinforced with 3-0 monocryl suture, checked for leakage by pneumatic testing, and a Blake drain was placed adjacent to the anastomosis.

In the immediate post-operative period, the patient showed a satisfactory clinical evolution with no evidence of a systemic inflammatory response or febrile peaks. Clinical and laboratory controls were performed to assess his haemodynamic and metabolic stability, the main results of which are summarised in Table 2.

As can be seen, both body temperature and markers of systemic inflammation remained within normal parameters and there was no evidence of sepsis or infectious complications. On day three, the patient reported clinical improvement and was tolerating a soft diet, so discharge from hospital was considered appropriate. At the three-month review, the patient was found to be asymptomatic and fully reintegrated into his normal diet with no complications from the surgical procedure.

Table 1: Laboratory values on admission.

Parameter	Value	Reference range
White blood cells	8.0×10 ³ /mm ³	4.0–11.0×10 ³ /mm ³
Neutrophils (%)	60%	40–70%
Haemoglobin	14 g/dl	13.5–17.5 g/dl
Platelets	250×10 ³ /mm ³	150–400×10 ³ /mm ³
Sodium	138 mEq/l	135–145 mEq/l
Potassium	4.0 mEq/l	3.5–5.0 mEq/l
Chloride	102 mEq/l	98–107 mEq/l
Creatinine	0.8 mg/dl	0.7–1.2 mg/dl

Table 2: Postoperative clinical and laboratory course.

Parameter	Postop day 1	Postop day 3	Reference range
Temperature (°C)	36.7	36.6	36.0-37.5
Heart rate (bpm)	85	78	60-100
Blood pressure (mmhg)	110/70	115/75	-
White blood cells (X10 ³ /mm ³)	8.5	7.8	4.0-11.0
Neutrophils (%)	62%	58%	40-70%
C-reactive protein (mg/dl)	2	0.8	< 5
Lactate (mmol/l)	1.2	1	0.5-2.2

DISCUSSION

High intestinal obstruction affecting the gastric, duodenal, and jejunal outflow can arise from variations in the anatomical relationships between critical structures such as the aorta, the mesenteric vessels, and the gastrointestinal tract. In young, previously healthy patients presenting with symptoms of nausea, vomiting, anorexia, weight loss and, in some cases, malnutrition, it is essential to consider aortomesenteric syndrome as a differential diagnosis-

especially in contexts where it must be distinguished from neuropathic disorders (for example, megaduodenum) or eating disorders-in order to initiate timely management and prevent severe or even fatal complications.^{1,2} Superior mesenteric artery (SMA) syndrome, or Wilkie's syndrome, occurs due to extrinsic compression with symptomatic obstruction of the third portion of the duodenum, which becomes trapped between the aorta (posteriorly) and the SMA (anteriorly). This phenomenon was first described in 1861 by Carl Freiherr von

Rokitansky, based on cadaveric studies, and later, in the first half of the twentieth century, Wilkie presented a series of 75 cases that established the clinical and anatomical foundations of the condition.³ Over the past 150 years, despite ongoing controversies-even to the extent that its very existence has occasionally been questioned-multiple reports support the clinical relevance and impact of this syndrome.

Regarding its epidemiology, Wilkie's syndrome is more prevalent in women, with an approximate ratio of 3:2, and radiological studies report a general population incidence ranging from 0.013% to 0.78% in patients with functional dyspepsia, the incidence may be as high as 10.8%.⁴ The clinical presentation is highly variable: some patients may exhibit an acute, complete obstruction, while others display chronic, non-specific symptoms that can be mistaken for gastritis, peptic ulcer disease, irritable bowel syndrome or gastroparesis.⁵

The role of retroperitoneal adipose tissue is crucial in normal physiology, as it acts as a "cushion" that separates the SMA from the aorta, thereby allowing the duodenum to pass through unimpeded. Significant weight loss or catabolic states-observed in eating disorders, cancer, chronic infections and following surgical interventions (for example, in the management of scoliosis or bariatric surgery)-reduce this fatty cushion, narrowing the aortomesenteric angle and leading to duodenal compression.^{6,7}

Diagnosing this condition requires a high index of suspicion, as its symptoms can be vague and progressive. Patients may experience gastro-oesophageal reflux, early satiety, postprandial pain or fullness, nausea, bile-stained vomiting, distension and frequent belching. As the condition evolves, the anorexia induced by the fear of triggering symptoms perpetuates weight loss and, consequently, further reduces the fatty cushion, worsening the degree of compression.^{8,9} In addition, it is important to exclude other causes, such as chronic inflammatory processes, neoplastic disorders or eating disorders.

Traditionally, diagnosis was achieved through barium studies; however, owing to the risk of toxicity in the event of perforation, contrast-enhanced computed tomography (CT) has become increasingly relevant. In this modality, the characteristic findings include measurement of the aortomesenteric angle, the duodenal calibre and the distance between the aorta and the SMA.

Normal values range from 38 to 56 degrees for the angle and from 10 to 28 mm for the distance in patients with Wilkie's syndrome, these parameters can be reduced to values of 6-22 degrees and 2-8 mm, respectively. Diagnostic precision is optimised by using submillimetre cuts, three-dimensional reconstructions and, in some cases, angiography.^{9,10} Although magnetic resonance imaging offers high precision, its use is limited by longer study times and higher costs, and ultrasonography, despite

being a cost-effective alternative, has limitations in accurately measuring these parameters except when using colour Doppler.^{9,10}

Regarding therapeutic management, various strategies have been adopted. Initially, a conservative approach is taken, based on nutritional support, gastric decompression and postural modifications (for example, adopting a prone or left lateral decubitus position, which alleviates compression via gravitational effects). However, in cases where conservative treatment fails or the obstruction is severe, surgical intervention is required. Techniques employed include the release of the Treitz ligament (the Strong procedure), duodenal mobilisation, placement of feeding tubes, and anastomotic procedures such as duodenojejunostomy or gastrojejunostomy. Advances in minimally invasive surgery, both laparoscopic and robot-assisted, have reduced morbidity, shortened the postoperative period and facilitated a swift return to daily activities, thereby establishing this approach as the treatment of choice.⁸⁻¹¹

Wilkie's syndrome is a complex clinical entity that necessitates a multidisciplinary approach for both its diagnosis and management. Correct identification of predisposing factors, precise evaluation through imaging studies and timely therapeutic intervention are fundamental to preventing severe complications, such as profound malnutrition or perforation, and to improving patient outcomes.

CONCLUSION

Superior mesenteric artery syndrome, or Wilkie's syndrome, is a rare but important condition. It is caused by compression of the third part of the duodenum due to a narrowed aortomesenteric angle. This is typically caused by loss of retroperitoneal fat. It predominantly affects young, previously healthy individuals and presents with nonspecific gastrointestinal symptoms such as nausea, vomiting, anorexia, and weight loss. Early recognition is crucial to prevent serious complications like duodenal perforation or ischaemia. This relies on a high index of suspicion supported by advanced imaging-particularly contrast-enhanced CT with three-dimensional reconstruction-to confirm the diagnosis and differentiate it from other similar pathologies.

The therapeutic approach has evolved toward a multidisciplinary strategy, integrating nutritional optimisation, gastric decompression, and postural measures with minimally invasive surgical techniques such as duodenojejunostomy. This shift has led to reduced morbidity, faster recovery, and improved patient outcomes. The combination of timely diagnosis, high-resolution imaging, and individualised management highlights the importance of coordinated surgical and medical care in addressing this complex syndrome. It also underscores the ongoing need for research to refine its diagnostic and therapeutic standards.

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