

Superior Mesenteric Artery Syndrome (Wilkie syndrome): A Diagnostic Challenge in High Intestinal Obstruction: Case Report

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ABSTRACT

Wilkie's syndrome is a rare condition characterized by compression of the third portion of the duodenum between the superior mesenteric artery and the aorta. We present the case of a 50-year-old male patient who presented with a six-month history of postprandial epigastric pain, early satiety, nausea, and vomiting, which worsened two weeks prior to admission. A contrast-enhanced abdominal CT scan revealed dilation of the stomach and duodenum, as well as narrowing of the aortomesenteric angle and space. This article highlights the importance of considering this condition in cases of high intestinal obstruction, particularly in patients with risk factors. It also emphasizes the importance of imaging studies and CT criteria for diagnosis.

KEYWORDS: Wilkie's syndrome, superior mesenteric artery, Cast syndrome

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INTRODUCTION

Wilkie's syndrome, also known as superior mesenteric artery syndrome (SMAS), or Cast syndrome, is a rare condition characterized by compression of the third portion of the duodenum between the superior mesenteric artery and the abdominal aorta (1, 2, 3).

Its low incidence and nonspecific symptoms often lead to diagnostic delays, frequently resulting in confusion with functional gastrointestinal disorders or other more prevalent causes of upper intestinal obstruction (2).

From a pathophysiological perspective, the main mechanism is a decrease in the aortomesenteric angle ($<22^\circ$) and the distance between the aorta and the superior mesenteric artery ($<8-10$ mm), generally secondary to the loss of retroperitoneal fat. This condition is associated with significant weight loss, hypercatabolic states, eating disorders, and major surgical procedures, and a possible familial predisposition has even been described (3,4).

Additionally, a dynamic component is recognized in its pathophysiology, in which factors such as mesenteric tension, anatomical variations of the ligament of Treitz, and postural changes influence the degree of duodenal obstruction. This phenomenon explains the clinical variability and symptomatic improvement with postural maneuvers, as well as the establishment of a vicious cycle in which the

obstruction promotes progressive weight loss, perpetuating vascular compression (5).

Clinically, patients typically present with postprandial epigastric pain, nausea, bilious vomiting, early satiety, and weight loss, with possible improvement in positions such as the left lateral decubitus or knee-chest position (6).

The diagnosis is established through clinical and imaging correlation, with contrast-enhanced computed tomography being the study of choice. Initial management is conservative, focused on nutritional recovery; however, in refractory cases, laparoscopic duodenojejunostomy is the surgical treatment of choice (1-3, 7-9).

CASE DESCRIPTION

A 50-year-old male patient denies chronic degenerative diseases, has ceased chronic alcoholism two years ago, has a history of smoking, and suffered a spinal cord injury two years and seven months ago, undergoing spinal surgery for the same injury, which currently results in paraplegia.

His symptoms began six months ago with postprandial epigastric pain, early satiety, nausea and vomiting on several occasions, as well as weight loss, requiring him to restrict his diet to liquids and soft foods. Two weeks prior to admission, the patient experienced increased vomiting with gastrobiliary characteristics and began to develop abdominal distension.

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Upon admission to this hospital, the patient was conscious and oriented, with dehydrated mucous membranes.

He reported abdominal pain and an inability to defecate for the past week.

Physical examination revealed a distended abdomen, absent peristalsis, generalized tenderness to palpation, and tympanic membranes on percussion without signs of peritoneal irritation.

Laboratory tests were within normal limits, and a clinical diagnosis of intestinal obstruction was made. Non-surgical management was initiated. Subsequently, a contrast-enhanced abdominal CT scan was requested, which revealed a decrease in the angle and distance between the aorta and mesentery, and gastric and duodenal dilation. (Figure 1, Figure 2).

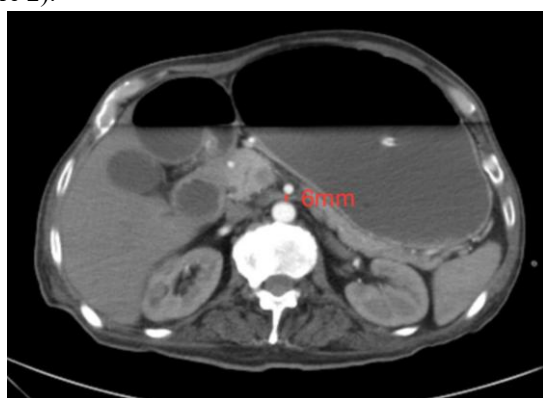


FIGURE 1. Abdomino-pelvic axial CT scan with intravenous contrast: decrease in the distance between the aorta and mesentery of 6 mm.



FIGURE 2. Abdomino-pelvic sagittal section computed tomography: decrease in the aortomesenteric angle of 20°.

A diagnosis of Wilkie's syndrome was made, and conservative management was chosen, consisting of fluid

resuscitation and total parenteral nutrition, as well as placement of a nasogastric tube for enteral feeding upon hospital discharge. Follow-up was scheduled in the outpatient clinic to monitor the patient's progress and determine if surgery was necessary should conservative treatment fail.

DISCUSSION

Wilkie's syndrome, also known as superior mesenteric artery syndrome (SMAS), is a rare condition characterized by compression of the third portion of the duodenum between the superior mesenteric artery (SMA) anteriorly and the aorta with the spine posteriorly. (1, 2, 3)

Its low incidence and nonspecific clinical presentation frequently lead to diagnostic delays and confusion with functional gastrointestinal disorders or more prevalent conditions such as gastroparesis or partial mechanical obstruction of other etiologies. (2)

From a pathophysiological perspective, the reduction of the aortomesenteric angle ($<22^\circ$) and the distance between the aorta and the superior mesenteric artery ($<8-10$ mm) has been consistently described as the main underlying mechanism.

This phenomenon is typically associated with the loss of retroperitoneal fat, as occurs in patients with significant weight loss, hypercatabolic states, eating disorders, or following major surgical procedures. A possible familial predisposition has even been reported. (3, 4)

However, beyond this static mechanism, evidence suggests that it is a dynamic process involving additional factors such as mesenteric tension, variations in the insertion of the ligament of Treitz, and postural changes, all of which can modulate the degree of obstruction. This dynamic nature partially explains the observed clinical variability and the symptomatic improvement with specific postural maneuvers. A pathophysiological vicious cycle has also been described in which duodenal obstruction leads to hyporexia and progressive weight loss, which in turn exacerbates the narrowing of the aortomesenteric angle and perpetuates vascular compression. (5)

Typically, patients present with postprandial epigastric pain, nausea, vomiting (frequently of bile), early satiety, and progressive weight loss. A distinctive, though not universal, clinical finding is the improvement of symptoms with postural changes such as the left lateral decubitus or knee-chest position, reflecting the dynamic component of this condition. This clinical constellation, in the context of predisposing factors such as rapid weight loss or hypercatabolic states, should raise diagnostic suspicion, particularly in the absence of more common obstructive causes. (6)

The diagnosis is based on the correlation of clinical symptoms with radiographic findings. (4)

Diagnostic modalities include: Contrast-enhanced computed tomography (CT): currently the diagnostic standard, it demonstrates the reduced aortomesenteric angle and distance,

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as well as the extent of duodenal obstruction. Contrast-enhanced upper gastrointestinal series: shows the exact site of luminal compression and duodenal obstruction. Ultrasound and magnetic resonance imaging (MRI): non-ionizing radiation alternatives, especially useful in the pediatric population. (5, 6)

Initial management is conservative and includes fluid resuscitation, total parenteral nutrition (TPN), placement of a nasoenteric tube beyond the obstruction for enteral feeding, small meals, and positional feeding. Hyperalimentation aims to increase retroperitoneal and mesenteric fat to widen the aortomesenteric angle. (7, 8)

When conservative treatment fails, surgical intervention is indicated. Laparoscopic duodenojejunostomy is currently the procedure of choice. (1-3)

Other surgical options include gastrojejunostomy (although it does not relieve proximal duodenal obstruction), the Strong procedure (division of the ligament of Treitz, with a 25% failure rate), and, in exceptional cases, transposition of the superior mesenteric artery to the infrarenal aorta. (9)

CONCLUSION

Wilkie's syndrome should be considered in patients with signs of high intestinal obstruction and predisposing factors, especially weight loss or debilitating conditions. The diagnostic key lies in the clinical correlation with characteristic CT findings. Timely diagnosis prevents treatment delays and improves prognosis.

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