Case Report

DOI:

Wilkie's Syndrome Revisited: Lessons from Two Case Reports

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Abstract:

Background: Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome, is an uncommon cause of proximal intestinal obstruction caused by compression of the third portion of the duodenum between the superior mesenteric artery and the aorta, most often following significant weight loss.

Case Presentation: Authors describe two young patients with superior mesenteric artery syndrome (SMAS) precipitated by weight loss. The first was a 23-year-old woman who presented with four months of postprandial epigastric pain, early satiety, bilious vomiting, and an unintentional weight loss of 8 kg, with her body mass index (BMI) decreasing from 21 to 18 kg/m². Upper gastrointestinal endoscopy suggested extrinsic duodenal compression without intrinsic pathology.

The second case involved a 22-year-old man with a one-year history of left lower abdominal pain, recurrent vomiting, and increased bowel frequency following intentional weight loss. Endoscopy revealed only erosive gastritis. Laboratory investigations were unremarkable in both cases.

Computed tomography of the abdomen revealed a reduced aortomesenteric angle in both patients, confirming the diagnosis of SMAS. Both were managed conservatively with nutritional rehabilitation and postural therapy.

Conclusion: These cases highlight weight loss as a key precipitating factor for SMAS and underscore the importance of considering this diagnosis in young patients with unexplained gastrointestinal symptoms. Early imaging facilitates timely conservative management and favorable outcomes.

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Introduction:

Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome, is an uncommon but potentially debilitating cause of proximal intestinal obstruction resulting from compression of the third part of the duodenum between the superior mesenteric artery and the abdominal aorta.

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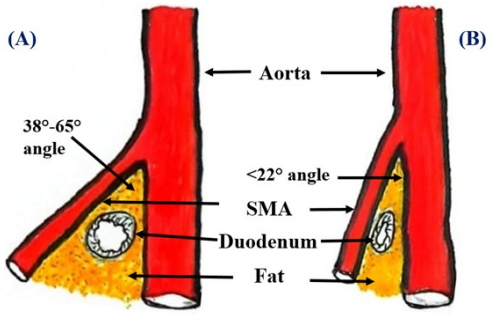
The condition is typically associated with a reduction in the aortomesenteric angle (normally 28°-65°) and distance (normally 10-28mm) **[Figure 1]**, most often precipitated by rapid or significant weight loss leading to depletion of the mesenteric fat pad.[1]Although rare, SMAS should be considered in young, otherwise healthy individuals who present with postprandial abdominal pain, early satiety, nausea, and bilious vomiting, particularly in the context of recent weight reduction.

The diagnosis can be challenging due to its nonspecific clinical presentation and overlap with more common gastrointestinal disorders.[2]Cross-sectional imaging, especially contrast-enhanced computed tomography, plays a crucial role in demonstrating a narrowed aortomesenteric angle and confirming duodenal compression.[3] Early recognition is important, as most patients

respond well to conservative management aimed at nutritional rehabilitation and restoration of retroperitoneal fat, thereby relieving vascular compression and preventing chronic morbidity.[1,4] In this article, the authors report two cases with similar clinical presentations, imaging findings, and outcomes. This report, along with previously published case reports on SMAS[5,6,7], highlights the elusive nature of the condition, which may contribute to delayed diagnosis and result in serious long-term complications. In the index cases, the underlying etiology was benign weight loss; however, in other cases, more serious causes of weight loss, such as malignancy, chronic illness, sepsis, substance abuse, and eating disorders, may be responsible.[8] Therefore, early recognition and prompt management of this condition are essential.

Figure-1: Normal (A) and narrow (B) aortomesenteric angle and distance



Case Summary

Case 1

A 23-year-old female with no significant past medical history presented with a 4-month history of progressive upper gastrointestinal symptoms. She complained of persistent postprandial upper abdominal fullness associated with epigastric pain and early satiety, which significantly restricted her oral intake. The symptoms were accompanied by frequent bilious vomiting occurring several times daily, typically shortly after meals. She reported unintentional weight loss of approximately 8kg during this period, with a decline in body mass index (BMI) from 21 to 18 kg/m². There was no history of prior abdominal or spinal surgery, trauma, eating disorders, medication use, or chronic systemic illness. Family history was unremarkable.

On physical examination, the patient appeared lean and mildly dehydrated but was hemodynamically stable. There was no pallor, jaundice, lymphadenopathy, or peripheral edema. Abdominal examination revealed mild epigastric tenderness without guarding, rebound tenderness, palpable masses, or organomegaly. Bowel sounds were normal. Cardiovascular, respiratory, and neurological examinations were within normal limits.

Laboratory investigations, including complete blood count, serum electrolytes, liver and thyroid function tests, serum amylase, and inflammatory markers, were within normal ranges, excluding infectious, metabolic, hepatobiliary, endocrine, and pancreatic causes. Abdominal ultrasonography demonstrated normal liver, gallbladder, biliary tree, pancreas, spleen, kidneys, uterus, adnexa, and urinary bladder, with no evidence of biliary dilatation or free intraperitoneal fluid. Upper gastrointestinal (GI) endoscopy revealed normal esophageal and gastric mucosa without ulceration, erosion, or mass lesions. Bile was noted within the stomach, suggestive of duodenogastric reflux. Although no intrinsic obstructive lesion was identified, a subtle external impression at the level of the antrum raised suspicion of extrinsic compression. The duodenal bulb and second portion were unremarkable **(Figure 2)**. Further evaluation with CT angiography and three-dimensional vascular reconstruction revealed a markedly reduced aortomesenteric angle of 17° and a decreased aortomesenteric distance of 4.5 mm. There was evident compression of the third portion of the duodenum between the superior mesenteric artery anteriorly and the abdominal aorta posteriorly, with mild proximal duodenal dilatation **(Figure 3)**. These findings were diagnostic of superior mesenteric artery syndrome or Wilkie's syndrome.

The patient was managed conservatively with nutritional rehabilitation aimed at weight gain, including small, frequent high-calorie meals and postural modifications (prone, left lateral decubitus, and knee-to-chest positions after meals) to alleviate duodenal compression. Surgical intervention, such as duodenojejunostomy, was reserved for failure of conservative therapy. At follow-up, the

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patient demonstrated gradual symptomatic improvement with conservative management.

Figure-2: Endoscopy upper gastrointestinal tract: bile in stomach and subtle extrinsic compression at antrum

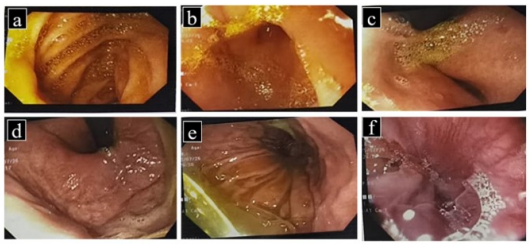
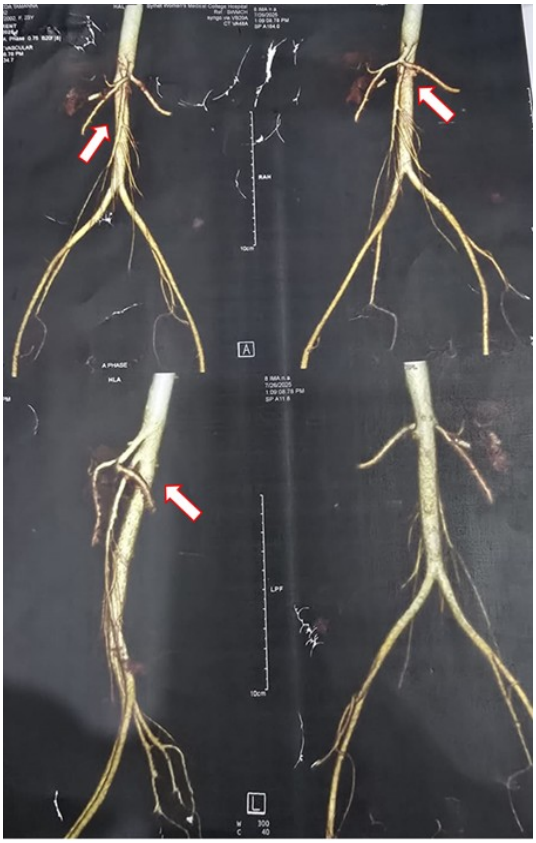


Figure-3: CT angiogram of abdominal aorta: compression of the third part of duodenum in the narrow aortomesenteric angle (arrows)



Case 2

A 22-year-old man presented with a one-year history of left lower abdominal pain and postprandial vomiting, typically occurring after meals. His bowel movements were infrequent (every 2–3 days) and were occasionally preceded by cramping pain. He also reported associated weight loss.

On examination, his body mass index (BMI) was 18.5 kg/m² (height 171 cm, weight 54 kg). His vital signs were stable (blood pressure 110/70 mmHg, pulse 80/min). Mild anemia was noted; however, there was no jaundice, edema, organomegaly, or other significant systemic findings.

Upper gastrointestinal endoscopy revealed mucosal erythema suggestive of erosive antral gastritis **(Figure 4)**. A contrast-enhanced computed tomography (CT) scan of the abdomen demonstrated normal liver, gallbladder, biliary tree, pancreas, spleen, kidneys, adrenal glands, urinary bladder, prostate, and major vessels. No mass lesion, lymphadenopathy, ascites, or abnormal enhancement was identified. However, vascular measurements revealed a markedly reduced aortomesenteric angle (approximately 14°) and a reduced aortomesenteric distance (approximately 6 mm), both significantly below normal values **(Figure 5)**. These radiological findings were suggestive of superior mesenteric artery syndrome (SMAS), likely contributing to his chronic postprandial vomiting and weight loss. On follow-up, he also showed improvement with conservative management.

Figure-4: Endoscopy upper gastrointestinal tract: antral erosive gastritis (arrow)

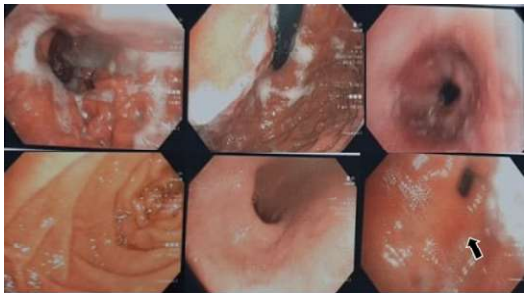
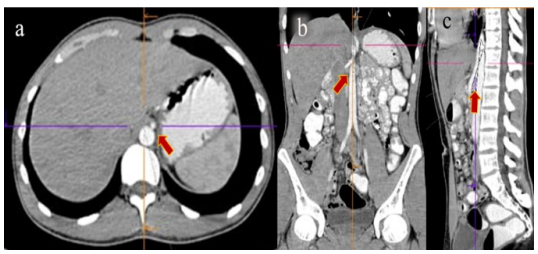


Figure-5: CT scan of abdomen (a-horizontal view, b-coronal view, c- sagittal view): reduced aortomesenteric angle and distance (arrows)



Discussion:

Superior mesenteric artery syndrome (SMAS) is typically associated with a reduction in the aortomesenteric angle and distance, most commonly precipitated by significant or rapid weight loss leading to depletion of the mesenteric fat pad.[9]

The two cases presented here highlight the classic clinical and radiological spectrum of SMAS in young individuals with weight loss. Both patients were young (22-year-old male and 23-year-old female) and had progressive postprandial symptoms accompanied by reduction in BMI to borderline underweight levels (18–18.5 kg/m²). In both cases, weight loss likely contributed to narrowing of the aortomesenteric angle, predisposing to duodenal compression. SMAS can affect individuals of any age; however, in older patients, it is more commonly associated with atherosclerosis.[10] While it may arise from benign factors, including short-term fasting during Ramadan, it can also indicate a serious underlying pathology, such as a hidden malignancy.[3,11]

Clinically, SMAS often presents with nonspecific upper gastrointestinal symptoms including postprandial fullness, epigastric pain, early satiety, nausea, and bilious vomiting. The female patient exhibited typical features of high intestinal obstruction, including frequent bilious vomiting and early satiety, whereas the male patient had chronic postprandial vomiting with associated abdominal pain and constipation. These variations illustrate the heterogeneity of presentation and the potential for delayed diagnosis due to symptom overlap with functional dyspepsia, gastritis, or peptic ulcer disease.[12]

Endoscopic findings in both cases were either normal or nonspecific. In case 1, bile in the stomach and subtle extrinsic compression raised suspicion of a mechanical etiology which was later on confirmed by imaging. In case 2, erosive gastritis was noted, which may have represented either a coincidental finding or a consequence of chronic bile reflux and vomiting. Importantly, endoscopy may fail to visualize the site of compression directly, underscoring the need for cross-sectional imaging when clinical suspicion persists.[4]

CT angiography with vascular reconstruction was diagnostic in both cases, demonstrating markedly reduced aortomesenteric angles (14° and 17°) and decreased aortomesenteric distances (6 mm and 4.5 mm, respectively), along with compression of the third part of the duodenum and proximal dilatation. These findings fall well below established normal ranges and are considered highly suggestive of SMAS when correlated with compatible clinical features.[3]

SMAS has been reported more frequently in females, particularly among young, lean individuals. This observed female predominance may be related to the associated risk factors, such as lower body fat and rapid weight loss, which can reduce the fat pad surrounding the third part of the duodenum and increase the risk of vascular compression. However, whether female sex itself confers an intrinsic biological susceptibility to SMAS remains uncertain.[13]

Management of SMAS is initially conservative, focusing on nutritional rehabilitation to restore the mesenteric fat pad and increase the aortomesenteric angle. Both patients were managed with dietary modification, frequent high-calorie meals, and postural measures to reduce duodenal compression. During follow-up, both patients showed symptomatic improvement, with better tolerance of oral intake and gradual weight gain, indicating a favorable response to conservative management. Surgical intervention, such as duodenojejunostomy, is reserved for refractory cases or those with persistent obstruction despite adequate nutritional therapy.[14]

The index cases emphasize several important points. First, SMAS should be considered in young, lean patients presenting with chronic postprandial vomiting and weight loss. Second, normal laboratory tests and nonspecific endoscopic findings do not exclude the diagnosis. Third, CT angiography with measurement of the aortomesenteric angle and distance plays a crucial role in establishing the diagnosis. Early recognition is essential to prevent progressive malnutrition and unnecessary investigations.[15] Together, these two cases reinforce the importance of maintaining a high index of suspicion for

SMAS in patients with unexplained upper gastrointestinal obstructive symptoms, particularly in the context of recent or significant weight loss.

Conclusion:

Superior mesenteric artery syndrome should be kept in mind when evaluating patients, particularly the young ones, who present with unexplained weight loss, abdominal discomfort, and recurrent vomiting. Prompt recognition, supported by thorough clinical assessment and appropriate imaging, is essential for timely diagnosis. In many cases, non-surgical approaches, especially nutritional rehabilitation, can successfully alleviate symptoms and lead to favorable outcomes.

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