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SUPERIOR MESENTERIC ARTERY SYNDROME MASQUERADING AS GASTRIC OUTLET OBSTRUCTION: A CASE REPORT HIGHLIGHTING THE IMPORTANCE OF CONSIDERING RARE VASCULAR CAUSES OF CHRONIC VOMITING

Dr. Guna Sahithi Lebaka[1]*, Dr. Nagarajan Duraisamy[2], Dr. Divyan Devasir[3], Dr. Giridhar Aravind[4], Dr. Sudarshan[5]

[1]Postgraduate, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India
[2]Professor and Head of Department, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India
[3]Associate Professor, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India
[4]Assistant Professor, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India
[5]Professor and HOD, Department of General Surgery (Research and Minimal Access Surgery)

ABSTRACT

Background: Superior mesenteric artery (SMA) syndrome is an uncommon cause of upper gastrointestinal obstruction resulting from compression of the third part of the duodenum between the SMA and the aorta, owing to a narrowed aortomesenteric angle and distance. It is frequently misdiagnosed because its presentation mimics more common conditions such as peptic ulcer disease and gastric malignancy.

Case Presentation: We report a 55-year-old male farmer who presented with a four-month history of postprandial upper abdominal pain, self-induced vomiting of partially digested food 10–15 minutes after meals, and unintentional weight loss of 20 kg over one month. Examination revealed a cachectic patient (BMI 16.1 kg/m²) with a scaphoid, non-distended abdomen and a positive succussion splash. Contrast studies and contrast-enhanced computed tomography (CECT) of the abdomen demonstrated marked dilatation of the duodenum up to the duodenojejunal flexure with an abrupt transition, a reduced aortomesenteric distance of 7 mm, and a narrowed aortomesenteric angle of 20 degrees, confirming SMA syndrome.

Conclusion: SMA syndrome should be considered in the differential diagnosis of chronic postprandial vomiting associated with significant weight loss, particularly when initial investigations such as ultrasonography and endoscopy are inconclusive. A high index of suspicion combined with targeted cross-sectional imaging is essential for timely diagnosis and management, avoiding unnecessary interventions directed at more common differentials.

KEYWORDS: Superior mesenteric artery syndrome, Wilkie's syndrome, duodenal obstruction, aortomesenteric angle, weight loss, gastric outlet obstruction

INTRODUCTION

Superior mesenteric artery (SMA) syndrome, also known as Wilkie's syndrome or cast syndrome, is a rare cause of proximal intestinal obstruction. It occurs when the third part of the duodenum becomes compressed within the narrow space between the SMA anteriorly and the aorta and vertebral column posteriorly. Normally, the aortomesenteric angle measures between 38° and 65°, and the aortomesenteric distance ranges from 10–28 mm; the mesenteric fat pad cushions the duodenum within this space. When this angle narrows to less than 25° and the distance falls below 8 mm, the duodenum is compressed, producing partial or complete obstruction. The condition classically develops in settings that reduce the retroperitoneal and mesenteric fat pad, most commonly rapid or significant weight loss from any cause — malignancy, malabsorption, eating disorders, prolonged immobilization, burns, or severe systemic illness. Anatomical predispositions such as a high or fixed duodenojejunal flexure, a low origin of the SMA, or an abnormally short ligament of Treitz have also been implicated. Because of its non-specific presentation, SMA syndrome is frequently misdiagnosed initially as peptic ulcer disease, gastroparesis, or malignancy, leading to delays in diagnosis. We describe a case in which the classic clinical triad and radiological findings led to a confirmed diagnosis after several more common differentials were reasonably excluded.

Case Presentation

A 55-year-old male, employed as a farmer and residing in Tiruvannamalai, presented with a four-month history of postprandial upper abdominal pain associated with discomfort, aggravated by food intake and relieved by vomiting. The vomiting was self-induced, occurring approximately 10–15 minutes after meals, and consisted of partially

digested food particles that were bile-stained but non-blood-stained and non-feculent. Over the preceding month, he had experienced an unintentional weight loss of approximately 20 kg. He also reported reduced urine output. There was no history of fever, hematemesis, jaundice, loose stools, blood in stools, breathlessness, hemoptysis, or abdominal bloating. He was not a known case of type 2 diabetes mellitus, systemic hypertension, bronchial asthma, seizure disorder, or tuberculosis, and had no significant surgical history. He reported a two-year history of alcohol intake and denied smoking. His personal history revealed a mixed diet with irregular bowel and bladder habits and adequate sleep.

General Examination: The patient was conscious, oriented, and afebrile, but poorly built, poorly nourished, and dehydrated, with no pallor, icterus, cyanosis, clubbing, or pedal edema. Vital signs were: blood pressure 100/60 mmHg, heart rate 64/min, respiratory rate 18/min, SpO_2 98% on room air, and BMI 16.1 kg/m².

Abdominal Examination:

●Inspection: Scaphoid abdomen with visible upper abdominal fullness; no scars, sinuses, dilated veins, visible gastric peristalsis, or hernial swellings; umbilicus inverted.

●Palpation: Soft, non-rigid abdomen with mild epigastric tenderness; no guarding, rebound tenderness, palpable mass, or hepatosplenomegaly.

●Percussion: Tympanic note over the upper abdomen with normal liver dullness; no shifting dullness or fluid thrill.

●Auscultation: Bowel sounds present; succussion splash was elicited and was positive.

Systemic examination of the cardiovascular, respiratory, and central nervous systems was unremarkable. Per-rectal examination revealed skin tags at the 1 o'clock and 5 o'clock positions with normal sphincter tone, no fissure or fistula, and stool-stained gloves.

Investigations

Table 1. Laboratory Investigations

Investigation	Value	Investigation	Value
Hemoglobin	14.3 g/dL	Total protein	8.0 g/dL
PCV	40.4%	GGT	521 U/L
Platelets	3.50 lakh/µL	Urea	46 mg/dL
TLC	7,400 /µL	Creatinine	0.8 mg/dL
Neutrophils	70.6%	Uric acid	6.1 mg/dL
Lymphocytes	20.7%	Sodium	128 mEq/L
HIV / HBsAg / HCV	Non-reactive	Potassium	2.8 mEq/L
Total bilirubin	0.83 mg/dL	Chloride	102 mEq/L
Direct bilirubin	0.31 mg/dL	Bicarbonate	12 mEq/L
AST	36 U/L	PT / INR	12.1 s / 1.01
ALT	52 U/L	APTT	34.6 s
ALP	85 U/L	Serum calcium	8.5 mg/dL

Notably, the patient had hyponatremia, hypokalemia, and low bicarbonate, consistent with prolonged vomiting and poor oral intake, and a markedly elevated GGT.

Radiological Findings

●Ultrasonography of the abdomen: No significant abnormality detected.

●Barium enema/meal: Marked dilatation of the proximal duodenum and stomach, with abrupt, smooth extrinsic narrowing of the third part of the duodenum and delayed transit of contrast into the jejunum.

●Upper GI endoscopy: Raised suspicion of carcinoma stomach (linitis plastica), prompting further evaluation.

●CECT abdomen: Gross dilatation of the D1–D4 segments of the duodenum (maximum diameter 8 mm) with an abrupt transition at the duodenojejunal flexure and collapse of distal small bowel loops, consistent with duodenal/small bowel obstruction. The aortomesenteric distance was reduced to 7 mm, and the aortomesenteric angle was narrowed to 20 degrees, with direct compression of the third part of the duodenum — findings diagnostic of superior mesenteric artery syndrome.

Differential Diagnosis

Given the presentation of postprandial vomiting, epigastric pain, and weight loss, the following differentials were considered before imaging confirmed the diagnosis:

●Peptic ulcer disease with gastric outlet obstruction

●Gastric carcinoma (linitis plastica)

●Chronic pancreatitis

●Duodenal tumors

●Annular pancreas

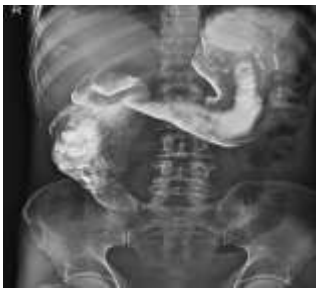
●Intestinal malrotation

●Intestinal pseudo-obstruction

●Tuberculous stricture of the duodenum

Diagnosis

Based on the classical clinical triad of postprandial vomiting, epigastric pain relieved by vomiting, and significant weight loss, together with barium studies showing duodenal dilatation with abrupt extrinsic narrowing at the third part of the duodenum, and CECT confirming a reduced aortomesenteric angle (20°) and distance (7 mm) with duodenal compression, a diagnosis of superior mesenteric artery (SMA) syndrome was established.



Intraoperative Findings

At operation, grossly and diffusely dilated, thin-walled, edematous stomach and loops of proximal small bowel were confirmed proximal to an abrupt transition point, corresponding to the site of extrinsic vascular compression identified on preoperative imaging. The affected bowel showed prominent, engorged serosal vessels consistent with chronic proximal distension.

Figure 1. Intraoperative photograph showing grossly dilated, stomach and edematous loops of proximal small bowel with engorged, prominent serosal vessels, confirming marked bowel distension proximal to the site of obstruction.

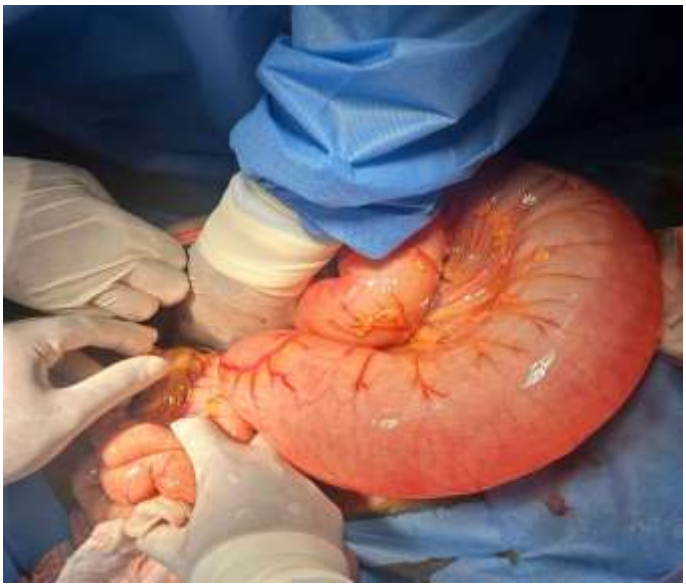


Figure 2. Intraoperative photograph demonstrating markedly dilated, thin-walled proximal small bowel loops proximal to the site of extrinsic compression.

DISCUSSION

SMA syndrome is an uncommon but important cause of chronic upper gastrointestinal obstruction that is easily overlooked because its symptoms overlap substantially with far more common conditions. In this patient, the initial clinical picture — postprandial pain, self-induced vomiting, and profound weight loss — raised concern for peptic ulcer disease with gastric outlet obstruction and, following an equivocal endoscopic impression of linitis plastica, gastric malignancy. Ultrasonography was unremarkable, which is typical, since SMA syndrome is a functional/mechanical vascular compression rather than a parenchymal or mucosal disease and is therefore not reliably detected on ultrasound. The diagnostic breakthrough in this case came from contrast radiology and CECT, which remain the investigations of choice. Barium studies classically show dilatation of the stomach and proximal duodenum with an abrupt vertical or oblique cut-off at the third part of the duodenum and to-and-fro peristalsis proximal to the obstruction. CT angiography or CECT with vascular measurements is considered the gold standard, as it allows direct visualization and measurement of the aortomesenteric angle and distance, and can exclude alternative mechanical causes such as tumors, annular pancreas, or malrotation. In this patient, an aortomesenteric angle of 20° (normal 38°–65°) and distance of 7 mm (normal 10–28 mm) were diagnostic. A notable feature of this case is the vicious cycle underlying SMA syndrome: weight loss reduces the mesenteric fat pad, narrowing the aortomesenteric angle and worsening duodenal compression, which in turn causes vomiting and reduced oral intake, perpetuating further weight loss. This self-reinforcing mechanism explains why patients can deteriorate rapidly once the syndrome is established, and why nutritional rehabilitation forms the cornerstone of initial management. The patient's metabolic profile — hyponatremia, hypokalemia, and low serum bicarbonate — reflected the physiological consequences of prolonged vomiting and poor intake, and required correction alongside definitive management of the underlying obstruction. Management of SMA syndrome is generally guided by symptom duration and severity. Conservative management — nutritional optimization via nasojejunal or parenteral feeding, correction of electrolyte imbalances, and positional maneuvers (left lateral decubitus or knee-chest positioning after meals) to increase the aortomesenteric angle — is typically attempted first, particularly in acute or recently diagnosed cases, and can be successful once adequate weight and mesenteric fat are restored. In patients with chronic, refractory symptoms or significant weight loss unresponsive to conservative measures, surgical intervention is warranted, with duodenojejunostomy considered the procedure of choice; other options include gastrojejunostomy and the Strong procedure (division of the ligament of Treitz), the latter now largely of historical interest.

This case underscores that SMA syndrome should remain an active differential whenever postprandial vomiting is accompanied by disproportionate weight loss, especially when first-line investigations such as ultrasonography are unremarkable and endoscopic findings are equivocal. Prompt recognition prevents unnecessary invasive work-up directed at malignancy and allows timely, targeted management of a potentially reversible condition.

CONCLUSION

Superior mesenteric artery syndrome, though rare, should be actively considered in patients presenting with chronic postprandial vomiting, epigastric pain, and marked weight loss, particularly when initial imaging is inconclusive. A high index of clinical suspicion supported by contrast studies and CECT with vascular measurements allows accurate

diagnosis and helps direct patients toward appropriate conservative or surgical management, thereby breaking the self-perpetuating cycle of weight loss and duodenal compression that characterizes this condition.

Conflict of Interest
The authors declare no conflict of interest.

Source of Funding
None.

Informed Consent
Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images/investigations.

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