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Superior mesenteric artery syndrome: a clinically oriented review of diagnosis, pitfalls, and management

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Abstract

Superior mesenteric artery (SMA) syndrome is an uncommon cause of proximal small-bowel obstruction caused by compression of the third portion of the duodenum between the SMA and the abdominal aorta. Although a reduced aortomesenteric angle and distance are useful diagnostic clues, they are not synonymous with disease, because similar measurements may be seen in thin or asymptomatic individuals. This narrative review was prepared for a gastroenterology readership to integrate clinical presentation, functional obstruction, cross-sectional imaging, endoscopy, nutritional management and surgical decision-making. A targeted literature search was updated through May 2026, prioritizing primary imaging studies, clinically oriented reviews, prospective or larger observational studies, and recent systematic reviews relevant to malnutrition and postoperative states. The available evidence supports a clinicoradiologic diagnosis in which symptoms, a compatible predisposing context and objective evidence of obstruction are more important than isolated vascular measurements. Computed tomography (CT) with multiplanar reconstruction is the most practical first-line modality for defining anatomy and excluding alternative causes, whereas an upper gastrointestinal contrast series can provide functional confirmation when CT findings are borderline or discordant. Endoscopy is valuable mainly to exclude intrinsic disease and assess mucosal consequences of stasis. Management should begin with gastric decompression, correction of fluid and electrolyte abnormalities, and carefully monitored nutritional rehabilitation, with explicit attention to refeeding syndrome. Surgery, most often laparoscopic duodenojejunostomy, is reserved for persistent, severe or chronic obstruction after appropriate conservative therapy, or when nutritional restoration is not feasible.

Keywords Superior mesenteric artery syndrome, duodenal obstruction, computed tomography, malnutrition, duodenojejunostomy

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Introduction

Superior mesenteric artery (SMA) syndrome, historically referred to as Wilkie's syndrome, is an uncommon cause of proximal small-bowel obstruction in which the third portion of the duodenum is compressed between the SMA anteriorly and the abdominal aorta or vertebral column posteriorly [1,2]. The disorder occupies a difficult space between anatomy and function: many patients have a demonstrably narrowed aortomesenteric angle or distance, yet not every patient with such measurements has clinically meaningful obstruction [3-6].

This distinction is clinically important. Delayed diagnosis may expose patients to progressive vomiting, dehydration,

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electrolyte disturbance, severe malnutrition, and rarely gastric or duodenal perforation. Conversely, overdiagnosis may lead to unnecessary anxiety, inappropriate attribution of symptoms to a radiologic measurement, prolonged tube feeding, or surgery in patients whose symptoms are better explained by gastroparesis, disorders of gut-brain interaction, eating-disorder-related symptoms without mechanical obstruction, chronic intestinal pseudo-obstruction, intrinsic duodenal disease, pancreatic or retroperitoneal pathology, or another vascular compression syndrome [7,8].

Previous reviews have catalogued the causes, diagnostic tests and operations used in SMA syndrome, and the principles of clinicoradiologic correlation and multidisciplinary care are already established. The practical contribution of this review is therefore not a claim that these individual concepts are new. Rather, it provides an integrated diagnostic pathway that explicitly ranks clinical context and objective obstruction above isolated morphometric thresholds, links discordant CT findings to functional testing, and redirects evaluation toward relevant mimics when obstruction is not demonstrated. The review also connects this diagnostic framework to risk-stratified nutritional rehabilitation and explicit reassessment points for escalation to surgery.

Literature review methodology

A targeted narrative search was performed in PubMed/MEDLINE and Google Scholar, supplemented by citation tracking from key reviews and original articles. The final search was updated on 22 May 2026. Search terms included "superior mesenteric artery syndrome", "Wilkie syndrome", "aortomesenteric angle", "aortomesenteric distance", "duodenal obstruction", "vascular compression syndrome", "nasojejunal feeding", "duodenojejunostomy", and "refeeding syndrome". Priority was given to primary imaging studies, normal-population studies, prospective or larger observational studies, systematic reviews and clinically relevant reviews. Case reports were used selectively when they illustrated uncommon mechanisms, complications or management dilemmas not represented in larger series. Because the evidence base is heterogeneous and predominantly observational, no formal meta-analysis or GRADE assessment was undertaken. For management statements, Table 1 provides pragmatic certainty labels (moderate, low or very low) based on study design, consistency, directness and reliance on consensus guidance; these labels are intended to make the evidentiary basis transparent and should not be interpreted as formal society recommendations.

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Anatomy and pathophysiology

In normal anatomy, the SMA arises from the anterior aorta and descends anteriorly to the left renal vein and the third portion of the duodenum (Fig. 1). Retroperitoneal and mesenteric fat maintain the aortomesenteric space and prevent compression of the duodenum. Reported normal values vary by technique and population, but commonly cited ranges are approximately 28-65 degrees for the aortomesenteric angle and 10-34 mm for the aortomesenteric distance [4-6] (Supplementary Table 1).

The key pathophysiologic event in acquired SMA syndrome is loss of the fat pad between the aorta and SMA, most often after rapid weight loss or severe catabolic illness. As the angle and distance decrease, the third portion of the duodenum may be compressed, producing partial or complete proximal obstruction. A self-perpetuating cycle may then develop: postprandial discomfort and vomiting reduce oral intake, progressive weight loss further narrows the aortomesenteric space, and obstruction worsens (Supplementary Fig. 1) [1,3,4].

Reduced angle or distance is best understood as an anatomical risk factor rather than the disease itself. Ozkurt *et al* studied 524 routine abdominal CT examinations in a normal population and showed that aortomesenteric measurements vary with body mass index, indicating that low values may occur in thin individuals without necessarily implying obstruction [6]. In the imaging study by Unal *et al*, an aortomesenteric distance cutoff of 8 mm had high discriminatory value in their selected cohort, whereas an angle cutoff of 22 degrees had low sensitivity despite high specificity; importantly, the authors also emphasized the correlation of gastric or duodenal dilatation with symptoms [5]. These findings support a diagnostic model in which anatomy must be linked to clinical and functional consequences.

Etiology and epidemiology

The true population prevalence of SMA syndrome is unknown. The often-quoted prevalence of approximately 0.1-0.3% is derived from selected clinical or radiologic populations, rather than population-based screening, and should not be interpreted as a general-population prevalence estimate [1,3]. SMA syndrome is most often described in adolescents and young adults, with a slight female predominance in many series, but it can occur at any age when the anatomical and nutritional context is appropriate [1,3,9,10].

Acquired SMA syndrome is more common than congenital disease. Predisposing settings include eating disorders, severe malnutrition, malignancy-related cachexia, chronic infection or inflammatory disease, burns, trauma, prolonged immobilization, spinal cord injury and rapid postoperative weight loss (Supplementary Table 2) [11]. Postoperative

Table 1 Management strategy, reassessment targets, and evidence certainty

Clinical situation	Preferred initial approach	Reassessment target/timeframe	Escalation trigger	Evidence certainty
Acute or recent onset with reversible weight loss	Nasogastric decompression if markedly distended; correct fluid/electrolytes; oral or post-pyloric feeding as tolerated	Reduced vomiting/pain, prescribed calories delivered, and an upward weight trend. A monitored trial of approximately 4-6 weeks may be used when stable and improving; direct duration evidence is mainly pediatric [36]	Persistent vomiting, inability to establish nutrition, worsening obstruction, or no objective nutritional progress	Low - observational series and expert synthesis [1,3,9,12,36]
Severe malnutrition or high refeeding risk	Dietitian-led feeding; baseline and serial phosphate, potassium, magnesium, glucose and fluid assessment; risk-based thiamine; gradual caloric advancement	Stable electrolytes and fluid status while calories advance toward full needs over approximately 4-7 days, or per local protocol	Electrolyte deterioration, fluid overload, organ dysfunction, or inability to deliver nutrition safely	Low - NICE/ASPEN consensus guidance, indirect to SMA syndrome [30,31]
Eating disorder or persistent food avoidance	Integrated gastroenterology, nutrition, and mental health care; confirm objective obstruction before attributing symptoms to SMA syndrome	Participation in nutritional rehabilitation, symptom improvement, and sustained weight restoration	Confirmed obstruction with inability to restore nutrition despite coordinated treatment	Very low - case series and expert opinion [19,20]
Post-bariatric or complex postoperative anatomy	Early CT review, exclusion of anastomotic/internal hernia causes, surgical consultation, and post-pyloric nutritional support	Resolution of obstruction and successful nutritional delivery after the postoperative cause is defined	Refractory obstruction or anatomy that prevents safe feeding	Low - systematic review of case reports/series and observational evidence [13]
Chronic or recurrent objective obstruction	Short monitored conservative trial when safe, with early multidisciplinary surgical planning	Predefined symptom, intake, weight, biochemical, and obstruction endpoints. Avoid prolonging an ineffective trial beyond about 3 months	Recurrent admissions, progressive malnutrition, persistent objective obstruction, or complications	Low - prospective/ retrospective series and systematic review; no randomized comparison [9,12,19]

Certainty labels are pragmatic rather than formal GRADE ratings: moderate=consistent direct evidence from systematic review or prospective comparative evidence; low=observational evidence or indirect consensus guidance; very low=case reports, small case series, or expert opinion
NICE, National Institute for Health and Care Excellence; ASPEN, American Society for Parenteral and Enteral Nutrition; SMA, superior mesenteric artery; CT computed tomography

SMA syndrome has been described after scoliosis correction, bariatric surgery, esophagectomy, and other operations that alter weight, posture or mesenteric tension [1,12-15]. In older or frail adults, cancer cachexia, sarcopenia and chronic illness may be more relevant than the classic adolescent phenotype, and should be actively considered because they influence both treatment tolerance and prognosis.

Congenital or anatomic factors include high insertion of the ligament of Treitz, low origin of the SMA, intestinal malrotation, exaggerated lumbar lordosis, and other variants that increase the susceptibility of the duodenum to compression, even without marked recent weight loss [3,4]. Such factors may explain some cases with recurrent symptoms or incomplete response to weight restoration.

Clinical presentation

Symptoms are usually nonspecific and overlap with many upper gastrointestinal disorders. Typical features include postprandial epigastric pain, early satiety, nausea, bilious or non-bilious vomiting, abdominal distension, food fear and weight loss. Symptoms may worsen in the supine position and improve in the prone, left lateral decubitus or knee-chest position. However, similar positional improvement may occur in gastroparesis or other disorders of gastric emptying; positional relief is therefore supportive rather than diagnostic, and should not substitute for objective evidence of duodenal obstruction [1,3,16-18].

Acute presentations may involve severe gastric and proximal duodenal distension, dehydration, hypokalemic metabolic alkalosis and aspiration risk. Chronic presentations are more subtle and may mimic functional dyspepsia, gastroparesis, cyclic vomiting syndrome or an eating disorder. In patients with anorexia nervosa or avoidant restrictive food intake disorder, a particularly difficult bidirectional relationship may occur: malnutrition predisposes to SMA syndrome, while obstructive symptoms reinforce food avoidance and delay weight restoration [14,19,20].

The clinical question should therefore be framed as follows: does the patient have symptoms compatible with proximal obstruction, a plausible predisposing context, and objective evidence that the duodenum is functionally obstructed at

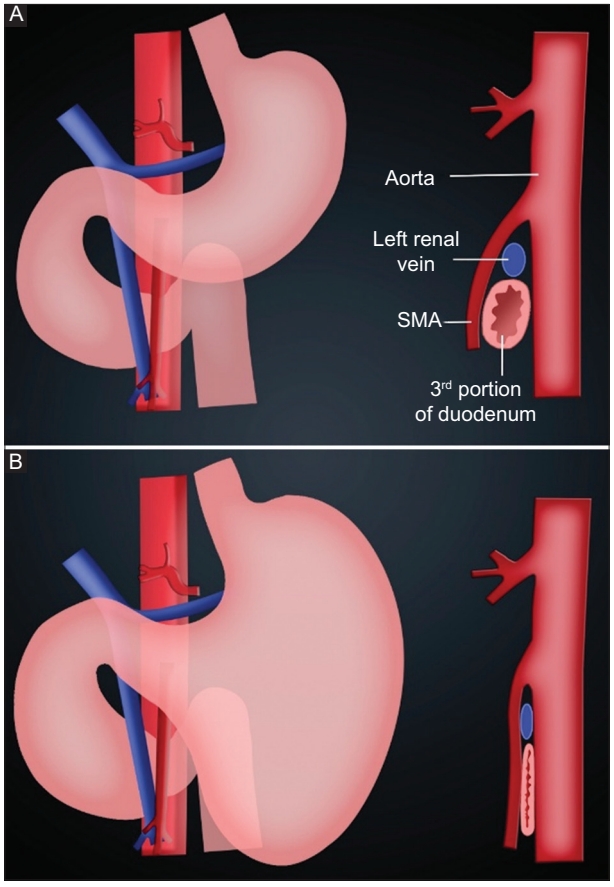


Figure 1 An illustration comparing normal anatomy with SMA syndrome. Panel A shows the normal relationship between the duodenum, aorta, SMA, and left renal vein. Panel B shows gastric and proximal duodenal distension caused by compression of the third portion of the duodenum between the SMA and aorta
SMA, superior mesenteric artery

the aortomesenteric crossing? If 1 component is absent, the diagnosis should remain provisional until alternative explanations are addressed.

Diagnosis: integrating anatomy, symptoms, and functional obstruction

No universally accepted diagnostic gold standard exists. In practice, diagnosis rests on convergence among 3 domains: (1) a compatible clinical syndrome; (2) reduced aortomesenteric angle or distance with compression of the third portion of the duodenum; and (3) objective evidence of upstream obstruction or delayed transit. The relative importance of these domains is unequal. Clinical context and functional obstruction are more diagnostically decisive than angle or distance alone, because isolated vascular narrowing can be incidental in thin individuals [5,6].

A practical diagnostic threshold is not a single number but a pattern. Measurements such as an aortomesenteric angle less than approximately 22-25 degrees and a distance less than approximately 8-10 mm are useful when they coexist with gastric or proximal duodenal distension, abrupt caliber change at the third portion of the duodenum, and delayed contrast passage (Supplementary Fig. 2) [5,7,8]. Borderline measurements without obstruction should be reported cautiously as predisposing anatomy rather than diagnostic SMA syndrome.

Computed tomography (CT)

Multidetector CT with multiplanar reconstruction is the most practical first-line imaging test in most adults with suspected SMA syndrome (Supplementary Table 3) [21]. CT allows measurement of the aortomesenteric angle and distance, demonstration of the duodenal transition point, assessment of gastric and proximal duodenal dilatation, and exclusion of more common causes of obstruction, such as intrinsic duodenal tumors, pancreatic disease, annular pancreas, inflammatory masses, retroperitoneal lymphadenopathy, adhesional obstruction or malrotation (Fig. 2) [5,7,8]. Ancillary findings, such as distension of the stomach and proximal duodenum, left renal vein compression and the abrupt vertical course of the SMA on sagittal reconstructions, further support the diagnosis [22,23].

The measurement technique should be standardized. The angle is usually measured on sagittal or sagittal-oblique reformations at the SMA origin, while the aortomesenteric distance is measured at the level where the duodenum crosses between the aorta and SMA. Reported measurements can vary with slice selection, patient position, respiratory phase, hydration, gastric distension and observer technique. Radiology reports should therefore avoid wording that equates measurement abnormality with disease. A more clinically useful report explicitly states whether there is upstream gastric or proximal duodenal dilatation, abrupt narrowing at the third portion of the duodenum, and whether an alternative cause is present.

Upper gastrointestinal contrast series

An upper gastrointestinal contrast series remains useful when CT findings and symptoms are discordant. Its advantage is functional: it can show delayed gastric or duodenal emptying, dilatation of the first and second portions of the duodenum, an abrupt vertical or oblique cutoff at the third portion, to-and-fro contrast movement, antiperistaltic waves proximal to the obstruction, and improvement with positional change [1,3,16].

A normal contrast study does not absolutely exclude intermittent or position-dependent SMA syndrome, but it lowers the probability of clinically significant fixed obstruction when CT also lacks upstream dilatation. Conversely, a contrast study showing reproducible stasis and delayed passage can support the diagnosis even when CT measurements are borderline, provided alternative causes of impaired transit have been considered.

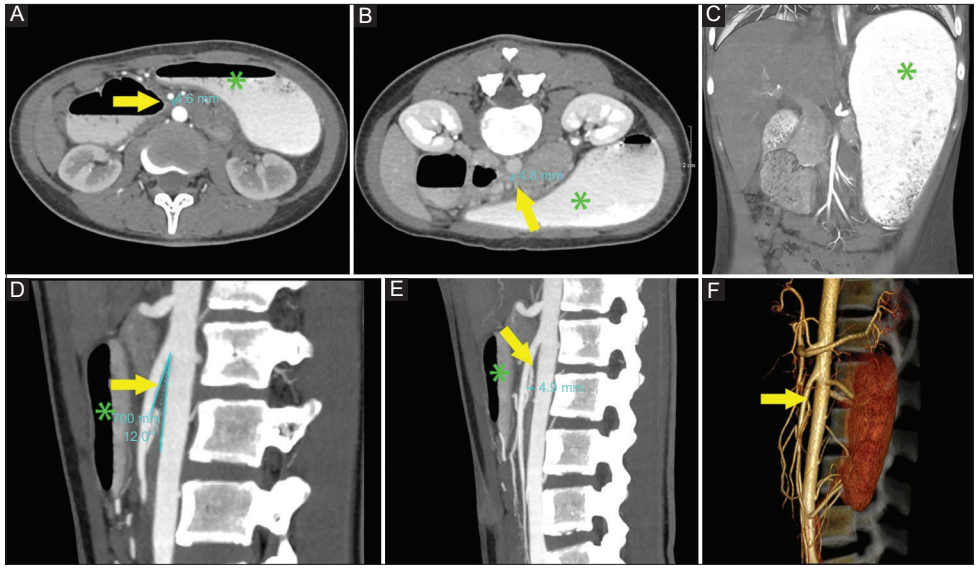


Figure 2 Typical computed tomography (CT) findings of superior mesenteric artery syndrome. (A and B) Supine and prone axial CT images obtained after oral contrast administration and arterial-phase intravenous contrast enhancement demonstrate focal compression of the third portion of the duodenum at the aortomesenteric crossing (yellow arrows), with upstream dilatation of the stomach and proximal duodenum (green asterisks). (C) Coronal reformatted image demonstrates marked upstream gastroduodenal dilatation (green asterisk). (D and E) Sagittal reformatted images demonstrate narrowing of the aortomesenteric angle and distance (cyan measurements), compression of the third portion of the duodenum (yellow arrow), and associated gastroduodenal dilatation (green asterisks). (F) Three-dimensional vascular reconstruction demonstrates the narrowed aortomesenteric relationship (yellow arrow)

Endoscopy and endoscopic ultrasound

Upper endoscopy should not be considered a definitive diagnostic test for SMA syndrome, because the extrinsic compression occurs in the third portion of the duodenum and may be intermittent or difficult to traverse. Its major roles are to exclude intrinsic mucosal or luminal disease, assess consequences of stasis, such as esophagitis, gastritis, retained gastric fluid or duodenitis, and identify features that should prompt cross-sectional or functional evaluation [16,24].

Endoscopic findings described in children and adults include a dilated stomach or duodenum, retained bile-stained fluid, pulsatile vertical or oblique narrowing in the third portion of the duodenum, and failure of the narrowed segment to open adequately with insufflation [16,24]. However, the literature does not provide reliable percentages for how often endoscopy is normal despite true SMA syndrome. A normal endoscopy that reaches the third portion of the duodenum reduces the likelihood of intrinsic obstruction, but does not rule out extrinsic dynamic compression, especially if symptoms, CT or contrast fluoroscopy remain compelling.

Endoscopic ultrasound has been reported as an adjunctive technique for measuring the aortomesenteric angle and distance, including in patients with normal body mass index [25]. Its role remains limited and non-standardized. It should be considered or used only as an adjunct in selected cases, not as a routine diagnostic requirement.

Ultrasound, magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA)

Ultrasound can measure the aortomesenteric angle and distance without radiation, and may be useful in children, adolescents, pregnant patients or repeated follow up, especially where experienced operators are available [26]. It may also permit dynamic assessment during positional maneuvers. Its limitations are substantial: bowel gas, gastric distension, body habitus, pain and operator dependence may impair visualization. Ultrasound is therefore best regarded as a screening or adjunctive tool rather than a standalone test.

MRI and MRA can demonstrate the vascular–duodenal relationship without ionizing radiation and are useful in selected patients who require radiation avoidance, or when CT is contraindicated. However, standardized MRI protocols for SMA syndrome are not established, acquisition is longer, and spatial or temporal resolution may be less practical in acutely obstructed patients. Although cine or dynamic MRI is increasingly used in other gastrointestinal contexts, its diagnostic role in SMA syndrome remains investigational rather than established.

Differential diagnosis and diagnostic pitfalls

The most common diagnostic error is overinterpreting a narrowed aortomesenteric angle or distance without proving obstruction. In a thin patient with chronic nausea, early satiety

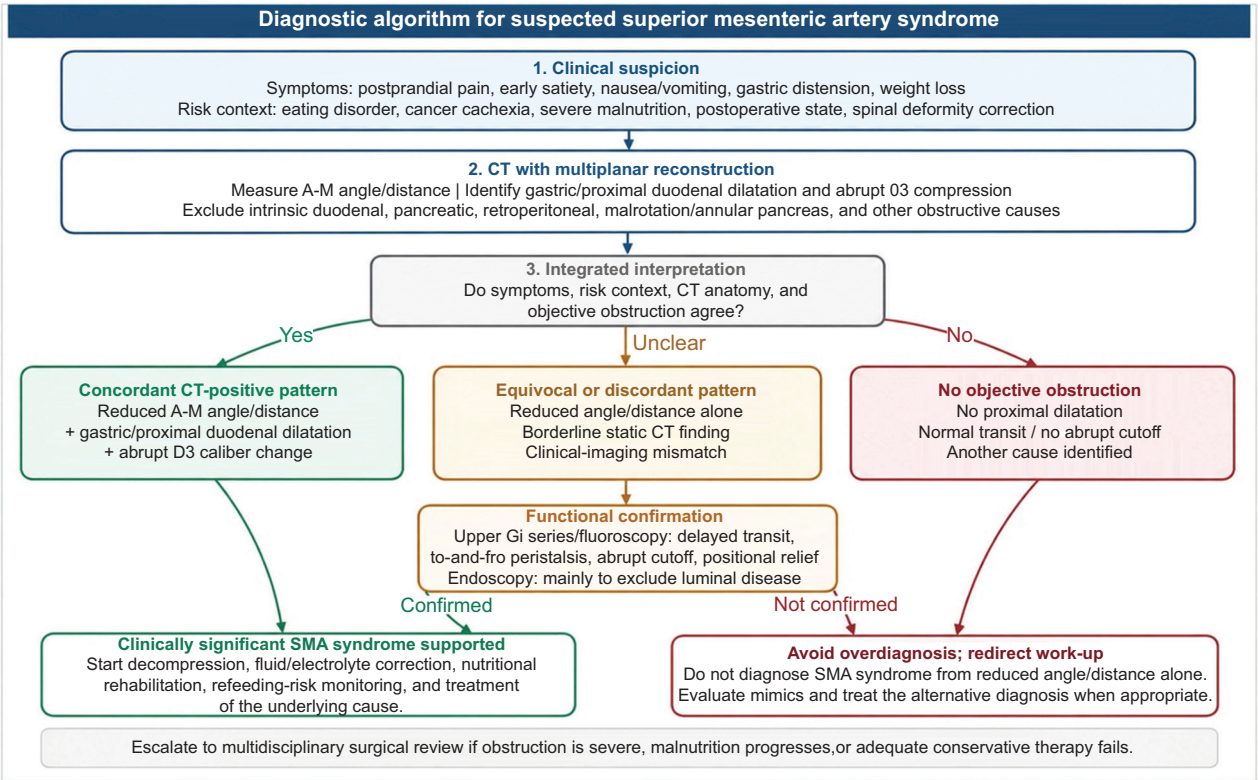
and a low angle on CT, but no gastric or proximal duodenal dilatation, the finding may represent body habitus rather than SMA syndrome [5,6]. The opposite error is underdiagnosis in patients with severe malnutrition or postoperative anatomy, where symptoms may be misattributed to functional disease, eating disorder behavior or expected postoperative intolerance. An integrated diagnostic pathway that combines symptoms, predisposing context, CT anatomy, functional obstruction, endoscopy and differential diagnosis helps reduce both errors (Fig. 3).

Important mimics include gastroparesis, functional dyspepsia, cyclic vomiting syndrome, chronic intestinal pseudo-obstruction, eating-disorder-related food avoidance without mechanical obstruction, intrinsic duodenal stenosis or tumor, peptic stricture, Crohn disease, pancreatitis, pancreatic neoplasm, retroperitoneal mass or lymphadenopathy, annular pancreas, malrotation with Ladd bands, adhesions and postoperative anatomic complications (Table 2) [3,7,8,23,27]. Coexisting vascular compression phenomena, particularly nutcracker (left renal vein) compression, may accompany a narrowed aortomesenteric angle and should be recognized rather than mistaken for the cause of duodenal obstruction [7,8,28]. In these situations, endoscopy, CT, MR enterography, gastric emptying studies or surgical consultation may be more appropriate than simply repeating angle measurements.

SMA syndrome also belongs to a broader family of abdominal vascular compression syndromes. Nutcracker phenomenon may coexist, because the left renal vein also passes between the SMA and aorta. However, nutcracker phenomenon is not equivalent to nutcracker syndrome: hematuria, flank pain, pelvic congestion symptoms or venous collateralization are needed before clinical significance is assigned [7,8,28]. Median arcuate ligament syndrome and May–Thurner syndrome can also coexist in thin patients, but each requires its own clinical-radiologic correlation.

Management principles

Management is individualized because no randomized trials or universally endorsed SMA-syndrome-specific society guideline defines the optimal pathway. Most authors recommend initial conservative therapy when the patient is clinically stable, particularly in acute or recent-onset disease with a reversible nutritional trigger [1,3,9,12]. This recommendation is supported mainly by prospective and retrospective observational series, case series and expert synthesis, rather than high-certainty comparative evidence. Table 1 therefore pairs each practical recommendation with a transparent evidence-certainty label.



Abbreviations: A-M. aortomesentenc; CT. computed tomography; D3. third portion of the duodenum; SMA. superior mesenteric artery.

Figure 3 Clinically oriented diagnostic pathway emphasizing the integration of symptoms, predisposing context, CT anatomy, functional obstruction, endoscopy, and differential diagnosis
CT, computed tomography; GI, gastrointestinal; SMA, superior mesenteric artery

Table 2 Common mimics and false-positive scenarios

Scenario	How it mimics SMA syndrome	How to reduce error
Low BMI with incidental narrow angle [5,6]	Reduced aortomesenteric measurements without obstruction	Require symptoms plus gastric/proximal duodenal dilatation or delayed transit before diagnosing SMA syndrome
Gastroparesis or severe functional dyspepsia [3,7,8,23,27]	Early satiety, nausea, vomiting, gastric retention	Consider gastric emptying evaluation, recognize that positional improvement can also occur, and confirm absence of a fixed third-duodenal transition point
Eating-disorder-related symptoms without mechanical obstruction [14,19,20]	Food fear, weight loss, nausea, abdominal discomfort	Use CT/UGI evidence of obstruction and psychiatric-nutritional assessment
Chronic intestinal pseudo-obstruction [3,7,8,23]	Recurrent distension and vomiting without mechanical cause	Look for diffuse bowel dysmotility rather than focal third-duodenal transition
Intrinsic or extrinsic duodenal disease [7,8,23,27]	Duodenal narrowing or proximal obstruction	Use endoscopy, CT, MR enterography, or biopsy when tumor, Crohn disease, peptic stricture, annular pancreas, or pancreatitis is suspected
Malrotation or Ladd bands [7,8,23,27]	Obstructive symptoms and abnormal duodenal course	Assess duodenojejunal junction position and mesenteric vessel orientation; surgical consultation when suspected

SMA, superior mesenteric artery; BMI, body mass index; CT, computed tomography; UGI, upper gastrointestinal imaging; MR, magnetic resonance

Initial treatment has 4 goals: relieve distension, correct fluid and electrolyte abnormalities, restore nutrition safely, and treat the underlying driver of weight loss (Table 1). Gastric decompression with a nasogastric tube is appropriate in patients with severe vomiting or marked gastric distension. Fluid resuscitation and correction of potassium, chloride, magnesium, phosphate and acid-base abnormalities should begin promptly. Positional therapy and small frequent meals may help mild cases, but are rarely sufficient when objective obstruction and severe malnutrition are present.

Nutritional support should bypass the obstructed segment whenever feasible. Nasojejunal feeding distal to the compression is generally preferred over nasogastric feeding, because it avoids worsening gastric distension and improves the likelihood of adequate calorie delivery (Supplementary Fig. 3). If nasojejunal access is unsuccessful, or prolonged support is expected, endoscopic, radiologic or surgical jejunal access may be considered [29,30]. Parenteral nutrition is reserved for patients in whom enteral feeding is not feasible, not tolerated or insufficient [9,12,19]. Prokinetic agents such as metoclopramide may have a limited adjunctive role when coexisting delayed gastric emptying persists after decompression, but they do not relieve fixed third-duodenal compression and should not be used when complete or high-grade mechanical obstruction is suspected [3].

Refeeding syndrome and multidisciplinary care

Many patients with SMA syndrome are malnourished, and nutritional rehabilitation can precipitate refeeding syndrome. Risk should be assessed before calories or intravenous dextrose are started. The National Institute for Health and Care Excellence (NICE) considers a patient at high risk when at least 1 major criterion is present (body mass index <16 kg/m^2, unintentional weight loss >15% within 3-6 months, little or no nutritional intake for >10 days, or low potassium, phosphate or magnesium before feeding), or when at least 2 of the following coexist: body mass index <18.5 kg/m^2, unintentional weight loss >10% within 3-6 months, little or no intake for >5 days, or a history of alcohol misuse or treatment with insulin, chemotherapy, antacids or diuretics. The American Society for Parenteral and Enteral Nutrition (ASPEN) additionally stratifies risk by the severity and number of abnormalities [30,31]. Baseline phosphate, magnesium, potassium, glucose, renal function and fluid status should be documented, with frequent reassessment during the first days of feeding.

Caloric advancement should follow a defined local protocol rather than immediate delivery of the full target. Commonly cited approaches include approximately 10-20 kcal/kg during the first 24 hours, with advancement by about a third of the goal every 1-2 days (ASPEN), or a maximum of 10 kcal/kg/day in high-risk patients and 5 kcal/kg/day in extreme-risk patients with progression toward full needs over 4-7 days (NICE) [30,31]. Thiamine is indicated before feeding or dextrose in patients at risk of deficiency or refeeding; ASPEN recommends 100 mg before feeding and then daily for 5-7 days or longer in severe starvation, whereas NICE recommends 200-300 mg/day during the initial 10 days. Routine thiamine supplementation is not necessary in a well-nourished, low-risk patient. Feeding should be slowed, and electrolyte deficits corrected, if phosphate, potassium or magnesium fall substantially, or organ dysfunction develops.

A multidisciplinary approach is particularly important in patients with eating disorders, cancer cachexia, frailty, sarcopenia, chronic systemic illness or postoperative states. Gastroenterologists, surgeons, radiologists, dietitians, mental health professionals and the primary treating team should agree on whether symptoms are truly obstructive, whether weight restoration is achievable, and which prespecified endpoints define response or failure. A recent systematic review reported a mean weight gain of approximately 5.6 kg (12.1% of body weight) over about 20 days among conservatively treated patients who

improved, but the underlying studies were heterogeneous and at high risk of bias; these values should be treated as contextual benchmarks rather than universal individual targets [19].

When to consider surgery

Surgery should be considered when severe or persistent obstructive symptoms continue despite a monitored conservative trial, when recurrent admissions or progressive malnutrition occur, when post-obstructive complications are present, or when safe nutritional restoration cannot be delivered (Table 3). Response should be reassessed against explicit endpoints: reduction in vomiting and postprandial pain, achievement of prescribed caloric intake, a sustained upward weight trend, correction of dehydration and electrolyte abnormalities, and improvement or persistence of objective obstruction. Lack of improvement in 1 isolated measurement should not by itself trigger surgery.

The duration of conservative therapy cannot be rigidly standardized. A monitored trial of approximately 4-6 weeks is commonly used in a clinically stable patient with recent-onset disease and a reversible trigger, although direct duration evidence is mainly pediatric; 1 pediatric cohort recommended at least 6 weeks before considering surgery [36]. In chronic or recurrent disease, Ganss *et al* used stringent operative criteria that included symptoms at least weekly for >6 months, compatible imaging, failure of at least 3 months of medical therapy, or severe complications; the same series argued against extending an ineffective trial beyond approximately 3 months. Merrett *et al* likewise emphasized that prolonged conservative treatment in chronically symptomatic adults often has low success and can delay definitive care [9,12]. These timeframes are decision aids rather than mandatory waiting periods: earlier escalation is appropriate for complete obstruction, perforation, aspiration risk, repeated inability to establish enteral access, progressive organ dysfunction or worsening malnutrition.

Laparoscopic duodenojejunostomy is generally regarded as the preferred definitive operation when surgery is required, because it bypasses the compressed segment while preserving gastric emptying physiology more effectively than gastrojejunostomy (Supplementary Fig. 4). Symptom improvement is frequently reported, but estimates vary, because patient selection, symptom duration, coexisting motility disorders and follow up differ substantially [9,32-34]. Cienfuegos *et al* reported durable but not universal relief after minimally invasive duodenojejunostomy, reinforcing the need to exclude coexisting gastric motility or functional disorders before surgery [32].

Strong's procedure, or division of the ligament of Treitz with duodenal mobilization, avoids an anastomosis but may fail if mobilization is incomplete, or vascular branches limit descent of the duodenum. Gastrojejunostomy decompresses the stomach, but does not directly relieve duodenal compression, and may lead to persistent symptoms, blind-loop problems, bile reflux or marginal ulceration. SMA transposition and other vascular operations are rarely used, and should be limited to highly selected cases in expert centers [9,12,32-35]. In patients with anorexia nervosa or other eating disorders, psychiatric assessment is not merely supportive: it is central to determining whether postoperative nutrition and long-term recovery are achievable.

Prognosis

Prognosis depends on symptom duration, reversibility of the precipitating condition, degree of malnutrition, presence of coexisting motility or psychiatric disorders, and timeliness of management. Patients with acute weight loss and early recognition often improve with decompression and nutritional

Table 3 Procedural and surgical options and expected roles

Procedure	Mechanism	Advantages	Limitations/current role and evidence
Laparoscopic duodenojejunostomy	Bypasses the compressed third portion of the duodenum	Most widely accepted definitive procedure; high reported symptom improvement in selected series	Requires surgical expertise; symptoms may persist if motility disorder or functional pain coexists. Evidence certainty: low (observational surgical series) [9,32-34]
Strong's procedure	Division of ligament of Treitz and duodenal mobilization	No bowel anastomosis; preserves continuity	Higher risk of incomplete relief; less commonly preferred as definitive therapy. Evidence certainty: very low (small series/case reports) [9,12,32,35]
Gastrojejunostomy	Bypasses gastric outlet into jejunum	Technically simpler in selected high-risk or hostile anatomy cases	Does not relieve duodenal compression; risk of bile reflux, blind loop, marginal ulcer or persistent symptoms. Evidence certainty: very low [9,12,32-35]
SMA transposition/ vascular procedures	Moves SMA to relieve compression	Anatomically direct in theory	Rare and technically demanding; reserve for highly selected expert-center cases. Evidence certainty: very low [12,35]
Feeding access procedures	Nasojejunal, gastrojejunal or jejunal access distal to obstruction	Bridge to weight restoration or optimization before surgery	A bridge to weight restoration or preoperative optimization, not definitive treatment when fixed obstruction persists. Evidence certainty: low/very low [29,30]

SMA, superior mesenteric artery

rehabilitation. Patients with long-standing symptoms, severe malnutrition, postoperative altered anatomy or coexisting disorders of gut motility are more likely to require surgery, and may have persistent symptoms even after technically successful bypass [9,13,19,32].

Complications of untreated or undertreated disease include dehydration, electrolyte abnormalities, aspiration, severe malnutrition, gastric pneumatosis, and rarely gastric or duodenal perforation. Complications of treatment include line-associated complications from parenteral nutrition, tube-related intolerance or displacement, refeeding syndrome and surgical morbidity. These competing risks justify early multidisciplinary review, rather than prolonged sequential referrals.

Knowledge gaps and future directions

The most important gap is the absence of standardized diagnostic criteria that integrate anatomy, functional obstruction and clinical severity. Future studies should compare symptomatic patients, thin asymptomatic controls and disease mimics using standardized CT measurement protocols, contrast transit assessment, nutritional indices and patient-reported outcomes. A second gap concerns management endpoints: the literature rarely defines how much weight gain is sufficient, how long conservative therapy should continue, or which patients should move earlier to surgery.

Further work is also needed in special populations, including adolescents with eating disorders, adults with cancer cachexia or sarcopenia, older frail patients, and patients who have undergone bariatric or spinal surgery. Dynamic imaging, including positional ultrasound and potential cine MRI, may be useful, but its incremental value over CT plus contrast fluoroscopy requires validation. Finally, outcome studies should distinguish persistent mechanical obstruction from coexisting gastroparesis or disorders of gut–brain interaction, because these conditions may explain poor surgical response.

Concluding remarks

SMA syndrome is best understood as a clinicoradiologic syndrome of proximal duodenal obstruction, not as an isolated measurement abnormality. Reduced aortomesenteric angle and distance are important clues, but diagnosis requires compatible symptoms, a plausible predisposing context and objective evidence of functional obstruction. CT is the most practical first-line modality, because it defines anatomy and excludes alternative disease, while upper gastrointestinal contrast study, endoscopy, ultrasound, MRI and MRA have complementary roles in selected circumstances.

Management should begin with decompression, correction of fluid and electrolyte abnormalities, and carefully monitored nutritional rehabilitation, with explicit prevention and monitoring for refeeding syndrome. Surgery, particularly laparoscopic duodenojejunostomy, should be considered for persistent, severe or chronic obstruction after appropriate conservative therapy, or when nutritional restoration is not feasible. The most defensible approach is multidisciplinary, individualized and cautious with regard to both underdiagnosis and overdiagnosis.

Acknowledgments

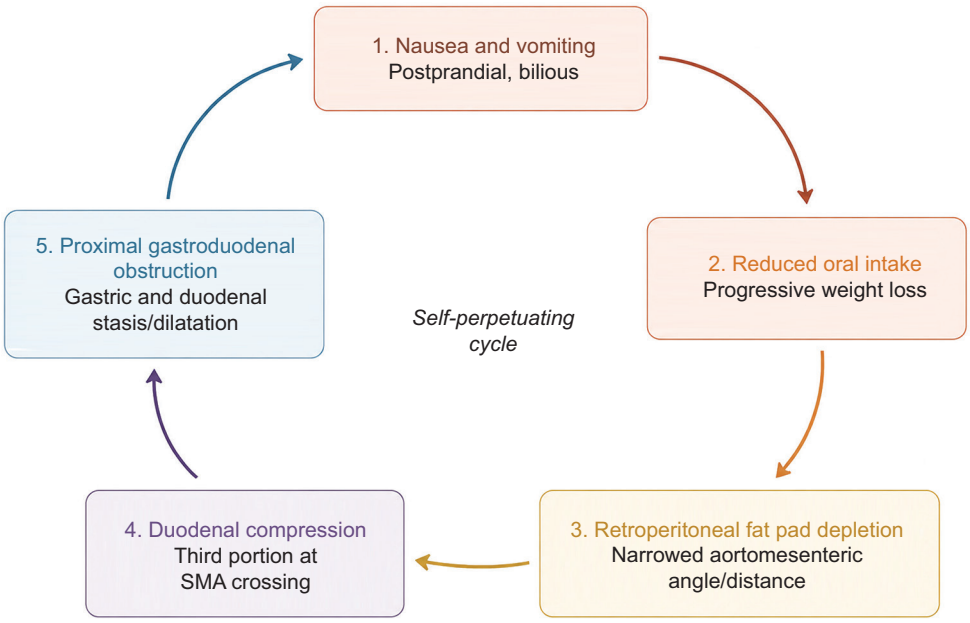
Artificial intelligence-assisted tools were used for language refinement, grammar checking, editorial formatting support and preparation of schematic illustrations. The authors reviewed and revised all text and figures, verified the references, and take full responsibility for the accuracy, integrity, and conclusions of the manuscript

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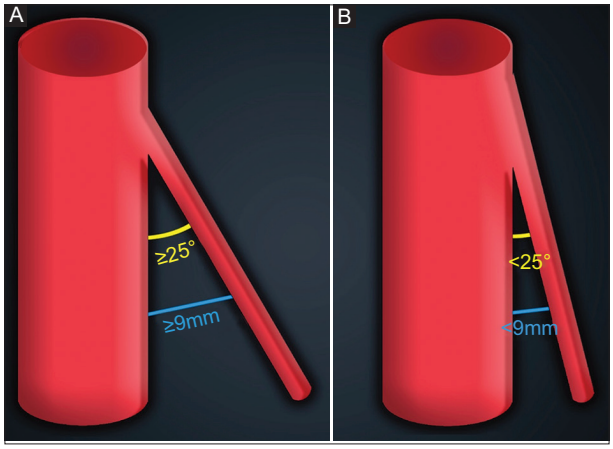
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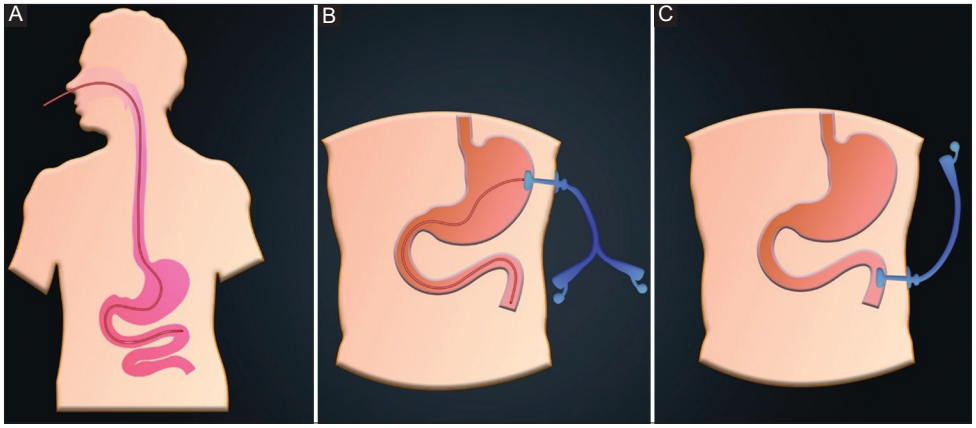
Supplementary material



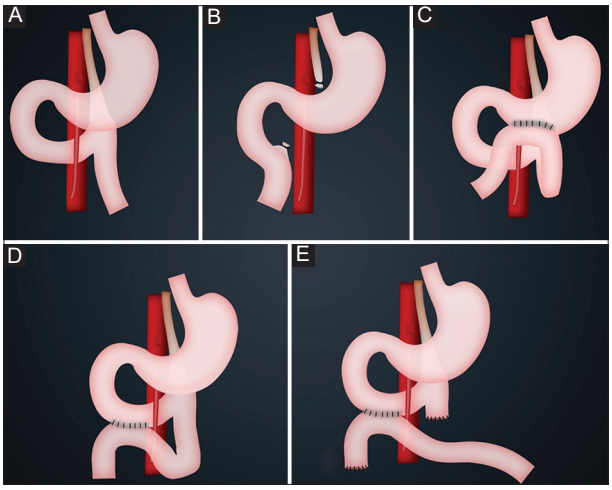
Supplementary Figure 1 Self-perpetuating pathophysiological cycle in SMA syndrome. Nausea and vomiting reduce oral intake and promote weight loss, leading to loss of the retroperitoneal fat pad and narrowing of the aortomesenteric angle. This compresses the third portion of the duodenum at the SMA crossing, producing proximal obstruction with gastric and duodenal stasis, which in turn aggravates nausea and vomiting\
SMA, superior mesenteric artery



Supplementary Figure 2 (A and B) Schematic representation of commonly cited imaging thresholds. Reduced aortomesenteric angle and distance are supportive findings but should be interpreted only in conjunction with symptoms and objective evidence of obstruction



Supplementary Figure 3 (A-C) Jejunal feeding approaches for nutritional support. Options include nasojejunal feeding when the tube can be advanced beyond the narrowed duodenum, gastrojejunal access, and direct jejunal access in selected cases



Supplementary Figure 4 (A-E) Surgical approaches for SMA syndrome. The diagram illustrates normal anatomy, duodenal derotation/Strong's procedure, gastrojejunostomy, and duodenojejunostomy options
SMA, superior mesenteric artery

Supplementary Table 1 Evidence and limitations of anatomical criteria in SMA syndrome

Topic [ref.]	Key data	Clinical interpretation	Main limitation
Normal-population CT measurements [6]	Aortomesenteric angle and distance varied across body mass index categories in 524 routine CT examinations	Low body mass index may be associated with smaller measurements even without proven obstruction	Not designed to diagnose symptomatic SMA syndrome
Symptom-enriched imaging cohort [5]	An 8-mm distance cutoff had high discriminatory value in the selected cohort; a 22-degree angle cutoff had low sensitivity but high specificity	Distance may be more informative than angle, but both require clinical and obstructive correlation	Small number of confirmed cases and selected population
Vascular compression imaging reviews [7,8]	CT depicts the aortomesenteric relationship, duodenal compression, and coexisting vascular compression phenomena	CT is practical for anatomy and differential diagnosis; the transition point and upstream effects should be reported	Imaging findings may be incidental unless symptoms and obstruction are present
Clinical series and reviews [9,12,13,19]	Evidence consists mainly of prospective or retrospective series, systematic reviews of observational reports, and case reports, rather than randomized trials	Diagnostic and treatment decisions should be individualized and multidisciplinary	Heterogeneous diagnostic criteria, treatments, and follow up limit certainty

SMA, superior mesenteric artery; CT, computed tomography

Supplementary Table 2 Predisposing causes and clinical contexts

Category	Examples	Clinical clue	Management implication
Rapid weight loss/ malnutrition	Eating disorders, severe dietary restriction, malabsorption, chronic vomiting, severe systemic illness	Low BMI or recent weight loss with postprandial vomiting and food fear	Nutritional rehabilitation is central; assess refeeding risk and psychological comorbidity
Catabolic disease and frailty	Malignancy cachexia, chronic infection, burns, trauma, sarcopenia, prolonged immobilization	Severe malnutrition or muscle wasting, often in medically complex patients	Treat underlying disease; conservative therapy may be difficult and requires close monitoring
Postoperative states	Scoliosis correction, bariatric surgery, esophagectomy, major abdominal surgery	Symptoms after rapid weight loss or altered anatomy/posture	Early surgical input may be needed if anatomy limits nutritional recovery
Congenital/ anatomic predisposition	High ligament of Treitz, low SMA origin, malrotation, exaggerated lordosis	Recurrent or early-onset symptoms, sometimes without major weight loss	Consider anatomic correction or bypass when conservative therapy fails
Coexisting vascular compression	Nutcracker phenomenon/syndrome, median arcuate ligament syndrome	Flank pain, hematuria, pelvic congestion, postprandial pain patterns beyond duodenal obstruction	Do not treat incidental vascular compression without syndrome-specific symptoms

BMI, body mass index; SMA, superior mesenteric artery

Supplementary Table 3 Diagnostic modalities and practical roles

Modality	Useful findings	Best use	Cautions
CT with multiplanar reconstruction	Reduced angle/distance, duodenal transition at SMA crossing, gastric/proximal duodenal dilatation, exclusion of mimics	First-line test in most adults and acute presentations	Measurements alone are not diagnostic; standardize technique
Upper GI contrast series	Delayed transit, abrupt cutoff, to-and-fro movement, positional improvement	Functional confirmation when CT is borderline or discordant	Less anatomic detail; may be normal in intermittent disease
Endoscopy	Retained gastric fluid, duodenal dilatation, pulsatile narrowing; exclusion of intrinsic lesions	Rule out mucosal/luminal disease and assess stasis complications	Normal endoscopy does not exclude extrinsic dynamic compression
Ultrasound	Angle/distance measurement; possible positional assessment	Screening or follow up in young/ pregnant/radiation-sensitive patients	Operator dependent; limited by gas, pain, and gastric distension
MRI/MRA	Vascular-duodenal anatomy without radiation	Selected patients with contraindication to CT or need for radiation avoidance	No standardized dynamic MRI pathway for SMA syndrome

CT, computed tomography; SMA, superior mesenteric artery; GI, gastrointestinal; MRI, magnetic resonance imaging

CT, computed tomography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; SMA, superior mesenteric artery; UGI, upper gastrointestinal