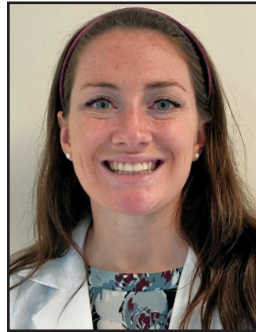


Neha D. Shah, MPH, RD, CNSC, CHES, Series Editor  
Elizabeth Wall, MS, RDN-AP, CNSC, Series Editor

## Superior Mesenteric Artery Syndrome: A Nutrition-Oriented Review



David Hakimian



Bailey Torkelson

**Superior mesenteric artery syndrome, a rare condition, is the result of duodenal compression between the aorta and the superior mesenteric artery. This compression is caused by decreased mesenteric fat tissue or abnormal anatomy leading to a narrowed aortomesenteric angle and ultimately duodenal compression. Patients can present with abdominal pain, lack of appetite, nausea, vomiting, and unintentional weight loss. The syndrome is a diagnosis of exclusion and as such, is often overlooked. An upper gastrointestinal series is typically used for diagnosis, however, computed tomography angiography can solidify the diagnosis. Generally, treatment requires weight gain with surgery only if conservative measures fail. This review will describe the syndrome, diagnostic criteria, and treatment options including medical nutrition therapy.**

### INTRODUCTION

**S**uperior mesenteric artery (SMA) syndrome is a rare condition also known as duodenal ileus, aortomesenteric artery compression, Cast syndrome, Wilkie's disease, and duodenal arterial mesenteric compression. The syndrome was originally described by Rotikansky in 1842 when he completed autopsies of thin young women with history of abdominal pain and emesis.<sup>1</sup>

Wilkie published his comprehensive case series and detailed the pathophysiology and diagnostic findings of the syndrome in 1921.<sup>2</sup> SMA syndrome is characterized by compression of the third segment of the duodenum due to reduced space between the SMA and aorta.<sup>3</sup>

SMA syndrome occurs either from a congenital anomaly in children or a significant, unintentional weight loss in adults, predominantly affecting young women, with the majority of patients between the ages of 10-39 years.<sup>4,5</sup> The incidence of SMA syndrome is reported as 0.013%-0.3% in the general population, 0.3% in hospitalized patients,

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David Hakimian, MD<sup>1</sup> Bailey Torkelson<sup>2</sup>  
<sup>1</sup>University of Chicago Medicine, Department of Internal Medicine, Section of Gastroenterology, Hepatology and Nutrition, Chicago, IL <sup>2</sup>Lurie Children's Hospital of Chicago, Chicago, IL

1.1% in burn patients, and 4.9% in patients with unexplained abdominal pain.<sup>6</sup> The diagnosis can be somewhat challenging and may take many years to diagnose in patients with nonspecific symptoms that do not correlate with duodenal compression nor resolve with empiric treatments.<sup>4</sup> Most often SMA syndrome is treated conservatively with weight gain in order to expand the mesenteric fat mass to relieve the obstruction. The purpose of this review is to describe the syndrome's pathophysiology, etiology, presentation, diagnostic criteria, and treatment with a special focus on nutrition therapy.

### Pathophysiology

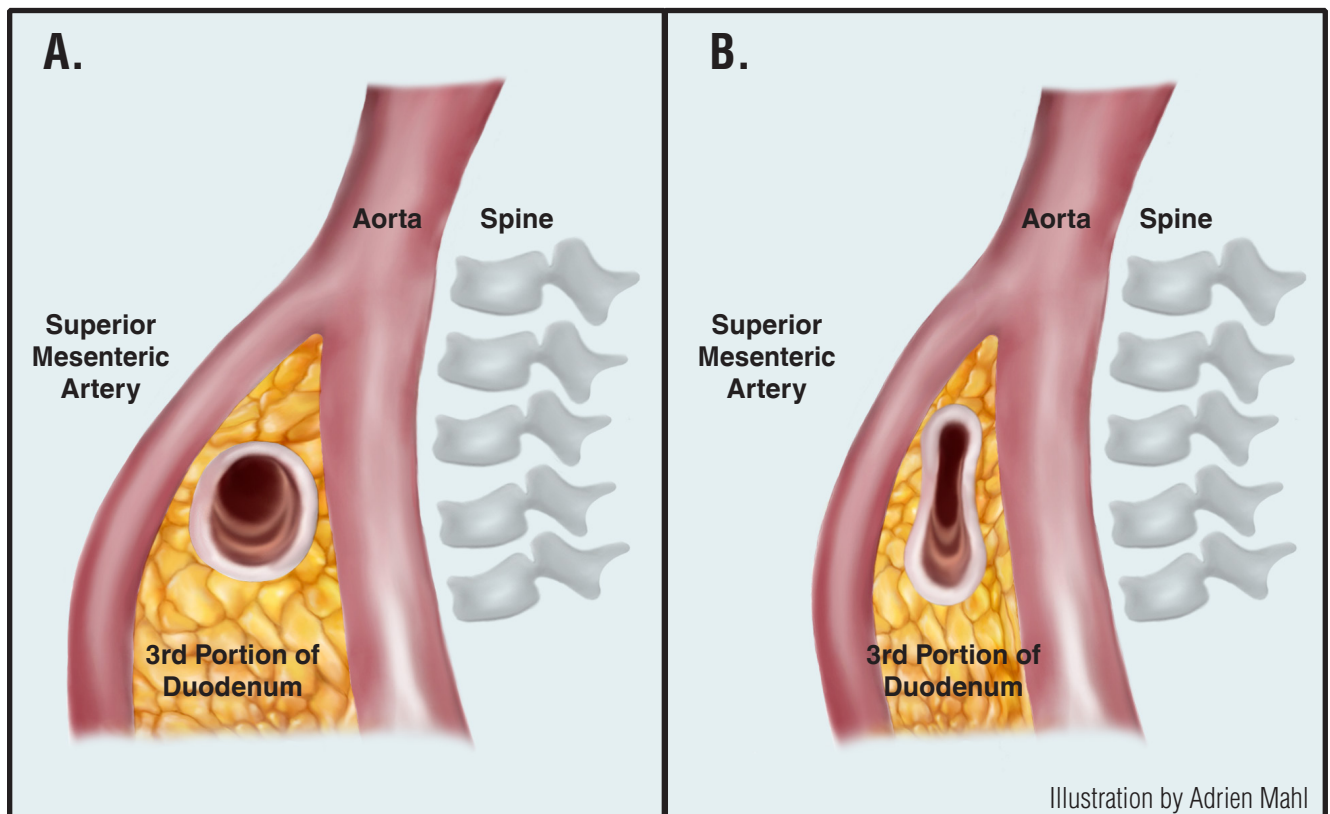
Normally, the duodenum crosses the abdomen anterior to the aorta at the level of the third lumbar vertebral body, suspended by the ligament of Treitz, and passes between the aorta and the SMA.<sup>7</sup> The SMA arises from the anterior abdominal aorta, behind the body of the pancreas at the level of the first lumbar vertebral body, adjacent to the origin of

the celiac trunk. The SMA runs inferiorly, forming a small arch to the right with its convexity to the left, crossing over the third part of the duodenum (see Figure 1). Together, the SMA and the aorta form an acute aortomesenteric angle (AMA) that the third portion of the duodenum passes through. The AMA is normally between 25°–60°, is related to the retroperitoneal fat tissue which holds the SMA off the spine, and is correlated with the patient's body mass index (BMI).<sup>2,8</sup> The aortomesenteric distance (AMD), defined as the length between the aorta and SMA, is typically 10–28 millimeters.<sup>8</sup> Irrespective of the inciting disorder, reduction of the AMA to < 25° and the AMD to < 8–10 millimeters raises the risk of duodenal pinching and small bowel obstruction (SBO).<sup>9–11</sup>

### Etiology

SMA syndrome in adults is most often a consequence of significant weight loss related to an underlying disorder. The various disorders

**Figure 1. Normal Anatomy and SMA Syndrome Anatomy**



**A.** Normal anatomy. **B.** SMA Syndrome. Clinical presentation is due to narrowed AMA defined as less than 25 degrees, and reduced AMD which is less than 8–10 mm.

predisposing patients for weight loss and SMA syndrome are broadly stratified into two categories: loss of mesenteric fat tissue and abnormal anatomy (Table 1). In healthy adolescents, SMA syndrome is reported after inadequate weight gain relative to height growth, causing duodenal compression without weight loss but decreased BMI.<sup>12</sup> Abnormal anatomy is generally seen as congenital anomalies in children and post-surgical alterations in adults.

**Symptoms**

Symptoms of SMA syndrome are often attributed to limited flow of chyme through the duodenum. It may present acutely or progressively over time. The severity of symptoms ranges from mild postprandial discomfort to bilious emesis and weight loss depending on the degree of the compression. Acute presentations often occur in post-surgical cases due to overextension of the SMA.<sup>13</sup> Progressive cases are more likely seen when patients have epigastric pain, nausea, and/or weight loss.<sup>14</sup> Patients with chronic symptoms of SMA syndrome may anticipate postprandial discomfort and develop aversions to food, perpetuating further weight and mesenteric fat tissue losses.

**Establishing the Diagnosis**

The diagnosis of SMA syndrome is often a diagnosis of exclusion since the symptoms can be

nonspecific and mimic other gastrointestinal (GI) and non-GI disorders. Clinical symptoms alongside imaging studies are used to diagnose the disorder. Common physical examination findings are listed in Table 2.<sup>15</sup> Laboratory values are usually normal, with the exception of patients with severe vomiting and dehydration who present with significant electrolyte abnormalities such as metabolic alkalosis or hypokalemia. Delay in diagnosis results in continuation of duodenal compression, discomfort, weight loss, malnutrition, electrolyte abnormalities, gastric dilation and perforation, peptic ulcer disease, pancreatitis, and even death.<sup>6</sup>

**Diagnostic Testing**

The vague and nonspecific symptoms of SMA syndrome often lead to inconclusive diagnostic testing. The radiologic tests most sensitive for SMA syndrome are upper gastrointestinal series (UGI) and computed tomography angiography (CTA) as depicted in Figures 2-4.<sup>6</sup> Table 3 describes the usual findings from both UGI and CTA testing.

An upper GI series can demonstrate prolonged retention of barium proximal to the third portion of duodenum, dilation of the duodenum and stomach, and backward flow of contrast from reverse peristalsis (known as “to and fro” peristalsis). Postural changes during an upper GI study can demonstrate changes in vascular compression of the duodenum; obstruction is typically greatest

**Table 1. Predisposing Disorders to SMA Syndrome**

Loss of Mesenteric Fat Tissue	Abnormal Anatomy
Anorexia nervosa	Congenital <ul style="list-style-type: none"> <li>• Low origin of the superior mesenteric artery</li> <li>• Intestinal malrotation</li> <li>• High insertion of the ligament of Treitz</li> </ul>
Cancer	Aortic aneurysm repair
Cardiac cachexia	Spinal instrumentation, scoliosis surgery
Burn injury	Nissen fundoplication
Drug abuse	Neoplastic growth in the mesenteric root
Paraplegia	Bariatric surgery
Malabsorption	
Trauma	
Human immunodeficiency virus (HIV)	
Cerebral palsy	

in the supine position and improved in the prone and left lateral decubitus position.<sup>16</sup> An UGI series allows for real-time evaluation by the radiologist to administer proper test maneuvers and evaluate the flow of contrast through the duodenum for an accurate diagnosis of SMA syndrome (Figure 3).

CTA using a three-dimensional technique provides a precise method for measurement of the aortomesenteric angle and distance. CTA may demonstrate narrowed AMA, decreased AMD, and dilated duodenum and stomach to secure the diagnosis of SMA syndrome.<sup>17,18</sup> An advantage of CTA is that it can shed light on SMA etiologies and

preexisting anatomical conditions (Figures 3,4).<sup>19</sup>

Upper endoscopy can be useful for differentiating SMA syndrome from other etiologies.<sup>15</sup> It does not serve as a diagnostic tool but should trigger a workup to confirm the diagnosis. Endoscopic ultrasound (EUS) has been used to diagnose SMA syndrome. The ultrasound probe allows for identification of the anatomical cause of the obstruction, and in some cases may be used to perform a minimally invasive bypass of the obstruction.

### Treatment and Management

The fundamental treatment of SMA syndrome aims to provide symptom relief, treat and manage the underlying disorder, weight restoration, and/or pursue surgery if weight gain is not successful. Surgical procedures should only be utilized when conservative measures fail or for anatomical reconstruction. There are no protocols or guidelines regarding the duration of conservative management nor optimal timing of surgery after failure; symptomatic improvements are observed within a few days or may take as long as a few months.<sup>6</sup> Nevertheless, whether managed conservatively or

**Table 2. General Findings in SMA Syndrome**

High-pitched bowel sounds
Nausea
Vomiting
Abdominal pain
Abdominal bloating
Early satiety
Weight loss

**Table 3. Diagnostic Imaging Performed for SMA Syndrome**

Imaging Modality	Findings	Advantages	Disadvantages
<b>UGI</b>	<ul style="list-style-type: none"> <li>Blockage or retention of barium proximal to the third portion of duodenum</li> <li>Dilation of the duodenum and stomach</li> <li>Back flow of contrast (“to and fro” peristalsis)</li> </ul>	<ul style="list-style-type: none"> <li>Commonly used for diagnosis</li> <li>Postural changes can support the diagnosis</li> <li>Real-time evaluation of obstruction or delay in contrast flow</li> <li>Shows characteristic anti-peristaltic pattern</li> </ul>	<ul style="list-style-type: none"> <li>Radiation exposure</li> <li>Requires ingestion of oral contrast</li> <li>Other anatomical structures are less visible</li> <li>Requires a trained radiologist</li> <li>Prolonged examination</li> </ul>
<b>CTA</b>	<ul style="list-style-type: none"> <li>Obstruction of duodenum if oral contrast used</li> <li>Gastroduodenal artery dilation with an abrupt narrowing in bowel caliber at the SMA</li> <li>Narrow AMA</li> <li>Short AMD</li> </ul>	<ul style="list-style-type: none"> <li>High accuracy, can show degree of compression and bowel wall thickness</li> <li>Can measure the AMA and AMD</li> <li>Creates three-dimensional reconstruction of anatomy for better accuracy</li> <li>Oral contrast can show the obstruction</li> <li>More sensitive and specific when used with intravascular contrast</li> </ul>	<ul style="list-style-type: none"> <li>Radiation exposure</li> <li>Requires ingestion of oral contrast</li> <li>Risk for contrast nephropathy</li> <li>No real-time assessment of obstruction</li> </ul>

surgically, a multidisciplinary team approach is beneficial including gastroenterologists, dietitians, radiologists, and psychiatrists is cardinal to ensure the patient’s well-being and quality of care.

### Nutrition Therapy

Nutrition assessment of patients with SMA syndrome includes: diet recall, weight history, anthropometric evaluation, biochemical data, and physical examination to assess for fat mass loss, muscle mass loss, fluid status, and signs of micronutrient deficiencies. First, the best route of feeding must be determined (Table 4). Many patients with SMA are not only at risk of refeeding syndrome, but also Wernicke’s encephalopathy if emesis has been an ongoing issue. Once past the refeeding stage, energy needs to support weight restoration should be determined. Medical nutrition therapy (MNT) requires a calorie surplus to promote anabolism and fat mass expansion in the epigastric area to alleviate obstructive symptoms. Depending on the patient’s weight history and anthropometric data, full recovery of lost weight is not always necessary, as small gains may be sufficient for symptomatic relief.<sup>20</sup>

The gold standard for measuring energy expenditure in the clinical setting is indirect calorimetry, which is particularly useful for underweight or malnourished patients as predictive equations are less accurate for patients



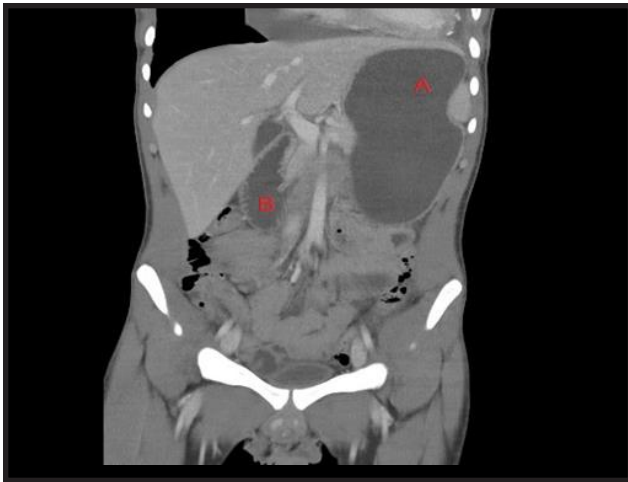
**Figure 2. Upper GI series with oral contrast of SMA syndrome. The stomach and second part appear to be dilated with retention of barium proximal to the duodenal third part. Jejunal loops distal to obstruction appears collapsed without contrast.**

with abnormal body composition.<sup>21</sup> If indirect calorimetry is available to measure resting energy expenditure (REE), this value is then multiplied by an activity factor. When indirect calorimetry is unavailable, using 30 kilocalories/kilogram or a predictive equation may best approximate REE, which is bolstered by multiplying by activity and stress factors or adding a fixed amount of additional kilocalories.<sup>21,22</sup>

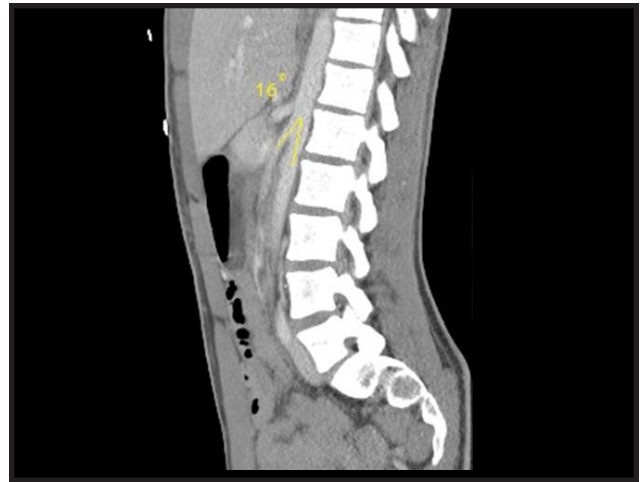
Case reports of MNT for SMA syndrome are heterogeneous; therefore, clear MNT guidelines

**Table 4. SMA Syndrome Nutrition Treatment Strategies**

Nutrition Intervention	Strategies for safe restorative feeding
<b>Oral diet</b>	<ul style="list-style-type: none"> <li>• First line of therapy</li> <li>• Focus on nutrient-dense liquids</li> <li>• Encourage low volume, sipping throughout the day</li> <li>• Post-prandial postural positioning (prone or left lateral)</li> </ul>
<b>Enteral nutrition</b>	<ul style="list-style-type: none"> <li>• Utilize when oral intake fails to improve symptoms</li> <li>• Nasoenteric access below the level of compression</li> <li>• Polymeric, isotonic enteral formula</li> <li>• Gradual introduction and advancement when concern for refeeding syndrome</li> <li>• Useful as adjunct to oral or parenteral support</li> </ul>
<b>Parenteral nutrition</b>	<ul style="list-style-type: none"> <li>• Utilize <u>only</u> when oral and enteral support fail</li> <li>• Can be used to supplement insufficient oral/enteral intake</li> <li>• Wean as soon as weight restoration is sufficient to allow for oral/EN</li> </ul>
<b>Supplements</b>	<ul style="list-style-type: none"> <li>• Complete daily vitamin/mineral supplement</li> <li>• Assessment for micronutrient deficiencies and specific repletion as indicated</li> <li>• Monitoring and repletion of potassium, phosphorus, and magnesium as necessary</li> <li>• Severely malnourished/refeeding risk – start thiamin 100 mg daily (oral or intravenous)</li> </ul>



**Figure 3. Coronal imaging of abdominal CT. Dilated stomach and duodenum proximal to the obstruction are both seen in SMA syndrome.**



**Figure 4. Sagittal abdominal CT imaging showing the narrowing of the aortomesenteric angle calculated here as 16°.**

have not been established. For example, management with a dense (4 kcal/mL), low-volume formula was effective in an 83-year-old male suffering from post-operative SMA when given in small doses orally.<sup>20</sup> In a 16-year-old female with anorexia nervosa, providing half of the needed calories through a nasojejun tube to supplement oral intake was beneficial for weight gain.<sup>23</sup> Some patients may be unable to tolerate adequate oral intake despite their efforts,<sup>24</sup> others may be able to tolerate enteral nutrition (EN) with proper tube placement that takes gastrointestinal

anatomy and function into account,<sup>25</sup> and others are unable to tolerate parenteral nutrition (PN) due to fluid overload or hepatotoxicity.<sup>26</sup> Successful weight restoration is possible via oral, enteral, or parenteral routes, but often requires a combination of modalities. Clinicians must use judgement to apply interventions based upon the etiology of the compression and weigh the risks and benefits of treatment plans for each individual patient.

Patients with SMA syndrome may best tolerate small frequent meals. Liquids will be easiest to pass through the compressed area; high-calorie,

**Table 5. Nutrition Interventions to Prevent SMA Syndrome for Select Clinical Etiologies**

Condition	Interventions
<b>Severe catabolic injury/illness (e.g. burn, trauma)</b>	<ul style="list-style-type: none"> <li>• Begin EN within 24-48 hours if hemodynamically stable, PN as soon as possible if unstable and severely malnourished</li> <li>• High-protein formulas or modular</li> <li>• Volume-based feeding protocols</li> <li>• Enhanced Recovery After Surgery programs to optimize perioperative nutrition delivery</li> </ul>
<b>Cancer</b>	<ul style="list-style-type: none"> <li>• Early nutrition interventions at the first sign of weight loss</li> <li>• Small frequent meals (6 times/day)</li> <li>• High-calorie high-protein foods, oral nutrition supplements</li> <li>• Soft and moist foods, blenderized meals</li> <li>• If nauseous, avoid foods with strong smell or flavor</li> </ul>
<b>Eating disorders</b>	<ul style="list-style-type: none"> <li>• Psychiatric treatment with pharmacotherapy and psychotherapy</li> </ul>
<b>Bariatric surgery</b>	<ul style="list-style-type: none"> <li>• Small frequent meals, prioritize protein sources</li> <li>• Daily multivitamin specialized for increased micronutrient needs</li> <li>• Avoid concentrated sweets</li> <li>• Space liquids 30 minutes apart from solid food intake</li> </ul>

high-protein liquids should be encouraged to optimize oral intake.<sup>20</sup> Positional maneuvers can provide symptomatic relief by removing tension from the mesentery and increasing the AMD. Lying prone or on the left side postprandially and using prokinetics or antiemetics may improve tolerance to oral intake.<sup>27</sup>

If oral feeding fails, the enteral route should be pursued next. Endoscopic tube placement beyond the duodenal compression is useful for both diagnostic and therapeutic purposes.<sup>26</sup> For a short-term trial, a temporary nasojejunal tube may be placed. If EN is tolerated and the anticipated need exceeds one month, then more permanent enteral access such as a gastrostomy tube with a jejunal extender or a direct jejunostomy tube may be required. If EN is poorly tolerated or fails to improve symptoms, then PN should be utilized. Parenteral support can be used in the short term until there is enough weight gain to allow for tolerance to oral intake, after which it is best to combine PN with oral intake or EN to provide adequate calories, expedite weight restoration, and minimize complications.<sup>26</sup>

Many patients with SMA syndrome presenting with intolerance of oral intake and weight loss meet malnutrition criteria. When initiating EN or PN support in a malnourished patient, it is prudent to take precautions against and monitor symptoms of refeeding syndrome. This is accomplished by “starting low and going slow” with general guidelines to initiate nutrition with 50-150 grams carbohydrates, or 10-20 kilocalories/kilogram, and advance by 33% of goal every 1-2 days. While advancing nutrition support, potassium, phosphorus, and magnesium levels should be monitored every 12 hours for repletion as needed. Given the high risk for refeeding syndrome in those with SMA syndrome, it is recommended to supplement with 100 mg thiamin supplementation for 5-7 days in addition to a therapeutic vitamin with mineral supplement until full nutrition support is achieved.<sup>28</sup>

Nutritional restoration is frequently met with physical and psychological challenges that impact resolution of SMA syndrome. Table 5 lists clinical conditions associated with SMA syndrome and suggested nutrition interventions to prevent or reduce the likelihood of mesenteric fat tissue loss.

Collaboration of care with a registered dietitian will help patients achieve their nutrition therapy goals, with timely adjustments to the nutrition care plan for optimal recovery from catabolic illnesses and reduced sequela of malnutrition.

## CONCLUSION

SMA syndrome is associated with a significant, unintentional weight loss in a wide range of predisposing clinical settings. The syndrome is characterized by compression of the third portion of the duodenum resulting in unexplained postprandial abdominal pain, anorexia, nausea, vomiting, or weight loss. When suspecting SMA syndrome, clinicians should begin with an upper GI series for evaluation and assessment of an obstruction. CTA with oral contrast can solidify the diagnosis and offer information about the underlying etiology of obstruction. Initial treatment is conservative and focuses on weight gain. Surgery may be required if medical management fails or there are predisposing factors such as abnormal anatomy. Employing a multidisciplinary team is imperative for successful treatment of SMA syndrome. ■

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Answers to this month's crossword puzzle:

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