Case Report

A case of superior mesenteric artery syndrome ✡

Shiavax J Rao, MDa,*, Pallavi Lakra, MBBSa, Kalyan Paudel, MDb, Christopher J Haas, MD, PhDc,d

a Department of Medicine, MedStar Union Memorial Hospital, MedStar Health Internal Medicine Residency Program, Baltimore, MD, USA
b Department of Radiology, MedStar Harbor Hospital, Baltimore, MD, USA
c Department of Medicine, MedStar Health, Baltimore, MD, USA
d Department of Medicine, Georgetown University Medical Center, Washington, DC, USA

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ABSTRACT

Superior mesenteric artery (SMA) syndrome, also known as Cast syndrome, Wilkie’s syndrome, or duodenal ileus, is a rare condition involving compression of the duodenum between the aorta and the SMA, primarily attributed to loss of the intervening mesenteric fat pad [1]. From a radiological perspective, this results in shortening of the aortomesenteric distance and narrowing of the aortomesenteric angle [2]. The reported prevalence of SMA syndrome is approximately 0.1%-0.3%, with common causes including extreme weight loss, debilitating illness, malignancy, malabsorption syndromes, trauma, neurologic injury, eating disorders, and substance abuse. Here, we present a case of SMA syndrome in a 24-year-old woman presenting with nausea, vomiting, and abdominal pain who improved with conservative management.

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Background

Superior mesenteric artery (SMA) syndrome, also known as Cast syndrome, Wilkie’s syndrome, or duodenal ileus, is a rare condition involving compression of the duodenum between the aorta and the SMA, primarily attributed to loss of the intervening mesenteric fat pad [1]. From a radiological perspective, this results in shortening of the aortomesenteric distance and narrowing of the aortomesenteric angle [2]. The reported prevalence of SMA syndrome is approximately 0.1%-0.3%, with common causes including extreme weight loss, debilitating illness, malignancy, malabsorption syndromes, trauma, neurologic injury, eating disorders, and substance abuse [3]. Clinical symptoms of SMA syndrome vary in intensity based on the severity of obstruction, and include postprandial epigastric abdominal pain, nausea, emesis, and weight loss. We describe an interesting case of SMA syndrome.

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* Corresponding author.
E-mail address: shiavax.j.rao@medstar.net (S.J. Rao).
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in a 24-year-old woman presenting with nausea, vomiting and abdominal pain.

Case presentation

A 24-year-old woman presented to the emergency room in the context of nausea, vomiting, and abdominal pain of 2 days duration. She had recently given birth and was discharged following an uncomplicated delivery and post-partum course 2 weeks prior to presentation. She endorsed ongoing substance use of opiates and cannabinoids. She denied fevers, chills, lightheadedness, dizziness, palpitations, melena, or hematochezia. Medical history was remarkable for seizure disorder managed with oral levetiracetam, which she was unable to take for several days owing to her current symptoms. She had a witnessed seizure after her initial presentation to the hospital for which she received intravenous levetiracetam. At the time of presentation, she was hypertensive (181/108 mm Hg) with otherwise preserved vital signs, and BMI was 21.08 kg/m². On examination, she was thin, ill-appearing, with decreased skin turgor, and tenderness in the epigastic and suprapubic regions. Bowel sounds were present on auscultation and there was no rebound tenderness or guarding. Initial laboratory diagnostics revealed leukocytosis (11.5 k/ul; reference range: 4.0–10.8 k/ul) and hypokalemia (3.1 mmol/L; reference range: 3.4–4.5 mmol/L). Urinalysis was remarkable for proteinuria, hematuria, pyuria with bacteria, and trace leukocyte esterase. Molecular testing for COVID-19 was negative. A contrast enhanced computed tomography (CT) scan of the abdomen and pelvis revealed diffuse wall thickening of the gastric pylorus, narrowing of the duodenum at the junction with the SMA and aorta, dilation of the first part of the duodenum, decreased aortomesenteric distance of 6 mm (Fig. 1) and decreased aortomesenteric angle of 19.9 degrees (Fig. 2). She was admitted for further management of her symptoms, attributed to SMA syndrome, urinary tract infection, dehydration, electrolyte abnormalities, and the noted seizure. She was managed conservatively for her abdominal symptomatology; upon resolution of her nausea and abdominal discomfort, she was initiated on a clear liquid diet and oral medications with dietary advancement. Electrolyte abnormalities were corrected with intravenous and oral electrolyte replacement therapy. She was eventually transitioned back to oral levetiracetam at her usual home dose. She was discharged from the hospital with planned outpatient follow-up with gastroenterology.

Discussion

SMA syndrome is a rare clinical entity, and risk factors include rapid weight loss, malignancy, malabsorption, cachexia, trauma, rating disorders, burns, and mechanical/structural variations (congenitally short or hypertrophic ligament of Treitz, duodenal malrotation, Ladd’s bands, and lumbar hyperlordosis), with a noted female predisposition [4,5]. Our patient had several of these risk factors including female sex, pregnancy associated lordosis and post-partum weight loss. Her symptoms were classic - nausea, vomiting, and abdominal pain. CT scan showed an aortomesenteric distance of 6 mm (normal: 10–34 mm) and aortomesenteric angle of 19.9 degrees (normal: 28–65 degrees) along with a narrowed duodenal junction; the latter is due to the loss of mesenteric fat pad resulting in a decrease in the angle and reduced distance between the aorta and SMA leading to duodenal compression [2,6].

Diagnosis may be challenging as the clinical presentation can mimic several conditions; however, when paired with strong suspicion in the appropriate clinical setting, symptom analysis along with radiographic imaging may aid in the diagnosis. A variety of imaging modalities may be utilized including plain abdominal radiographic films (with demonstration of gastroduodenal distention), upper gastrointestinal barium study (demonstrating anti-peristaltic flow, also known as a ‘to and from’ pattern of peristalsis), and CT scan (demonstrating a decreased angle and distance between the aorta and SMA) [2]. An aortomesenteric angle of less than 22-28 degrees on CT is considered the most sensitive finding, particularly when coupled with an aortomesenteric distance of less than 8 mm [6,7]. Other studies such as magnetic resonance imaging and ultrasound may also be used and may provide additional information and a correlation to confirm diagnosis [2].

Initially, treatment is conservative, focusing on fluid resuscitation, electrolyte repletion, and bowel rest with eventual dietary progression with nutritional support if required [2,4,8]. Some patients, especially those with cachexia require nutrition support with total parenteral nutrition to help increase the mesenteric fat pad which in turn increases the aortomesenteric distance and angle [5]. Our patient was managed conservatively and eventually discharged. With appropriate treatment, and resolution of symptoms, CT findings may improve and revert to normal [9]. If conservative therapy fails, surgical interventions such as laparoscopic duodenojejunostomy.
should be considered. This procedure carries a 90% success rate and is the procedure of choice due to its immediate relief with minimal postoperative complications. In certain cases, gastrojejunostomy may also be performed. Although the latter helps decompress and provide a bypass, it does not re-

solve the duodenal obstruction, and may lead to alternative gastrointestinal syndromes as a result of a residual blind loop or peptic ulceration secondary to biliary reflux [4,8].

While SMA syndrome is a rare condition, it does have high rates of morbidity related to small bowel obstruction. Prompt recognition, diagnosis, and management is important to prevent imminent minacious consequences.

**Patient consent**

Consent for publication has been obtained and the individual who is being reported on is aware of the possible consequences of that reporting.

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