Superior Mesenteric Artery Syndrome: Embracing the Possibility in Healthy Adult! A Case Report

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ABSTRACT:
Wilkie's syndrome, also known as superior mesenteric artery (SMA) syndrome, is a rare cause of duodenal outflow obstruction due to compression of the third part of the duodenum from a narrowing of the angle between the superior mesenteric artery and abdominal aorta. Despite its rarity, SMA syndrome should be considered particularly in patients with relevant symptoms and risk factors. We discuss the case of a healthy 34-year-old man with no comorbidities who presented with complaints of acute abdominal pain, distension, and vomiting after a heavy meal. Owing to compression of the third part of the duodenum between the superior mesenteric artery and aorta, the stomach and proximal duodenum were dilated on computed tomography, and conservative management was successful in this case.

INTRODUCTION:

Von Rokitansky first characterized the Superior Mesenteric Artery (SMA) syndrome in 1842 [1]; Wilkie later developed a deeper understanding of it under the names Wilkie's syndrome or Cast syndrome. SMA syndrome is an uncommon condition, with an incidence of approximately 0.0024-0.3%.It occurs when the superior mesenteric artery (SMA) compresses the third part of the duodenum as a result of the loss of angle between the SMA and aorta [2]. Weight loss is a sign of the condition, as well as one of its primary causes. Postprandial abdominal pain, early satiety, vomiting, weight loss, and malnutrition are prominent signs and symptoms of this condition [3]. Surgical intervention is necessary if conservative management is unsuccessful. Death rates exceed approximately 33% despite being rare [4]. Here, we present the case of a man with superior mesenteric artery syndrome who was an otherwise healthy individual with no comorbidities and no family history.

CASE PRESENTATION:

A 34-year-old male, presented to the emergency department with acute abdominal pain which gradually worsened after taking a heavy meal in the function on the same day, bilious vomiting and obstipation for three days duration. On admission, the patient's vital signs were normal. The patient was 158 cm tall, weighed 50 kg, and BMI of 20. He had no history of weight change or weight loss in the past year.

Abdominal palpation revealed generalized fullness and tenderness with absence of bowel sounds, but no guarding was detected. Per rectal and other examinations, including blood tests, the results were unremarkable.

Gastric decompression was performed with a nasogastric tube, and an aspirate of approximately 2 L was removed instantly, following which his distension and pain were reduced. Despite the patient's abdominal findings, his severe abdominal pain and fullness suggested the possibility of intestinal obstruction. To confirm this, a contrast-enhanced CT scan of the abdomen and pelvis was performed, which showed a grossly distended stomach with dilated 1st and 2nd part of the duodenum and compressed 3rd part of the duodenum between the aorta and super mesenteric artery with marked enhancement of the wall of the stomach and duodenum.The aortomesenteric angle was 18.1° (normal range: 25°–60°) and Aortomesenteric distance 4.2 mm (normal range: 10–28 mm) [5,6], (FIGURE 1-3) respectively. CT abdomen angiography confirmed the above-mentioned findings, and no other vascular abnormalities were observed.

Based on these radiological findings and clinical history, the patient was diagnosed with Superior mesenteric artery syndrome. Initially, conservative management, gastric decompression, intravenous fluid replacement, and electrolyte correction were performed, followed by nutritional assistance using enteral feeding, TPN, and gradual oral diet. Left lateral positioning during enteral feeding allowed for quick temporary resolution of the occlusive state in the early phase of treatment. The patient was successfully managed with conservative treatment and proper nutritional therapy.

FIGURE 1 : Sagittal CT image showing the entrapment of the duodenum between the aorta and the SMA with an aorta-SMA angle of 18.1°.

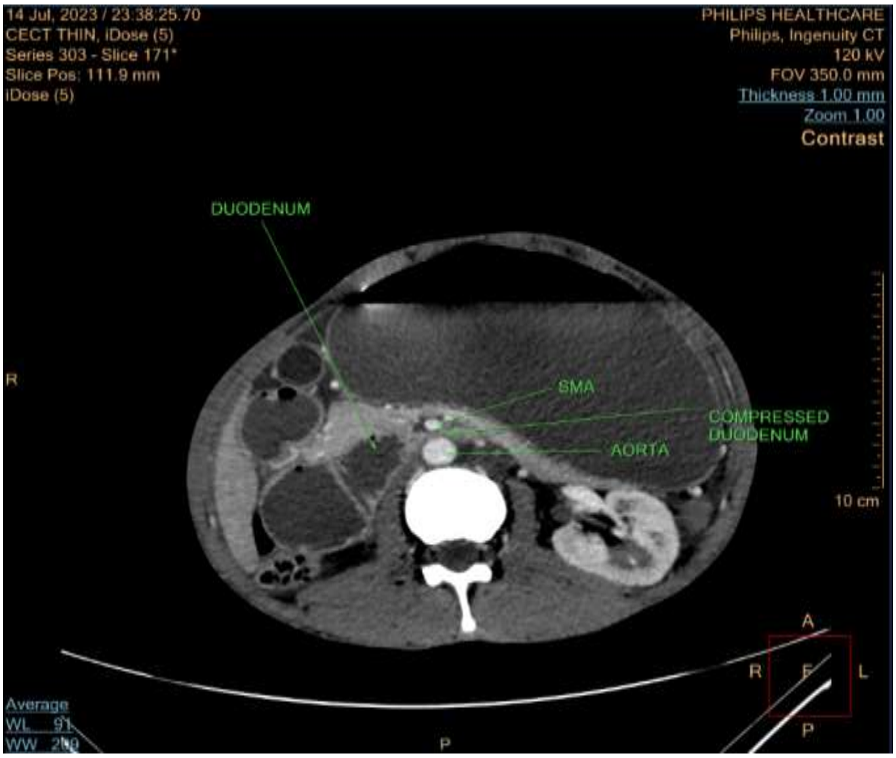

FIGURE 2 :This axial section of CT abdomen and pelvis shows compressed third part of duodenum between abdominal aorta and superior mesenteric artery.

FIGURE 3 :CT scan showing the SMA and the aorta separated by a small distance of 4.2 mm and severe gastric distension.

DISCUSSION:

The third part of the duodenum is situated between the aorta and superior mesenteric artery (SMA). It crosses the aorta anteriorly at the L3 vertebra and is supported by the Treitz ligament. Typically, the aortomesenteric angle and distance are 25–60° and 10–28 mm, respectively [5,6]. In SMA syndrome, the angle can be reduced to 7° and the distance to 2 mm, leading to compression of the duodenum [5,6].To assess the aortomesenteric vasculature, a contrast-enhanced computed tomography (CT) scan is used. Severe weight loss, which depletes the fatty cushion surrounding the SMA, is the primary cause of SMA syndrome. Etiological factors are divided into debilitating conditions that cause significant weight loss, and anatomical abnormalities (both congenital and acquired). Congenital anomalies include a high angle of Treitz insertion or an unusually low SMA insertion. Acquired anomalies can result from spinal trauma, corrective spinal surgery, or abdominal surgery due to strain in the small intestinal mesentery. Severe weight loss causes include eating disorders (as anorexia nervosa), burns, or other catabolic states, and wasting diseases (such as malabsorptive states or neoplasia) [7]. Common symptoms include anorexia, postprandial epigastric discomfort, bloating, eructation, oesophageal reflux, early satiety, acute metabolic alkalosis, hypokalemia, and chronic intractable bilious vomiting leading to severe dehydration [8]. The treatment for SMA syndrome can be surgical or conservative. Initial conservative management includes gastric decompression, intravenous fluid and electrolyte replacement, followed by nutritional support via enteral feeding, TPN, and gradual oral diet resumption. Surgery is considered if conservative treatment fails, including the Strong procedure, which divides the ligament of Trietz to mobilize the duodenum, but has a 25% failure rate [9]. Additional surgical options include gastrojejunostomy and duodenojejunostomy, with the latter preferred despite the benefits of gastrojejunostomy owing to its failure to eliminate the obstruction, potentially causing symptom recurrence [9,10].

CONCLUSION:

Despite its rarity as a cause of intestinal obstruction, SMA syndrome should be considered particularly in patients with relevant symptoms and risk factors. Delayed diagnosis can affect both mental health and acceptance of adverse outcomes. Computed tomography (CT) and angiography are essential for diagnosis. Early diagnosis allows for conservative management, whereas delayed diagnosis or unsuccessful medical treatment necessitate surgical intervention.

REFERENCE

1. Rokitansky C: Handbuch der pathologischen Anatomie. vol (ed): 3; 187.
2. Madhu B., Govardhan B., Krishna B: Cast syndrome. Oxf. Med. Case Rep. 20192019, 10.1093/omcr/omz025
3. Catarino Santos S., Loureiro A.R., Simão R., Pereira J., Pinheiro L.F., Casimiro C: Wilkie's syndrome: a case report of favourable minimally invasive surgery. J. Surg. Case Rep. 20182018. 10.1093/jscr/rjy027
4. Kothari TH, Machnicki S, Kurtz L: Superior mesenteric artery syndrome. Can J Gastroenterol. 2011, 25:599-600. 10.1155/2011/617201
5. Neri S, Signorelli SS, Mondati E., et al.: Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. J Intern Med. 2005, 257:346-51. 10.1111/j.1365-2796.2005.01456.x
6. Hines JR, Gore RM, Ballantyne GH: Superior mesenteric artery syndrome. Diagnostic criteria and therapeutic approaches. Am J Surg. 1984, 148:630-2. 10.1016/0002-9610(84)90339-8
7. Zaraket, V., & Deeb, L. (2015a: Accessed: June 5). Wilkie's syndrome or superior mesenteric artery syndrome: Fact or fantasy. Karger Publishers: https://doi.org/10.1159/000431307.

8. Mandarry M.T., Zhao L., Zhang C., Wei Z.Q: A comprehensive review of superior mesenteric artery syndrome. Eur. Surg. 2010, 42:229-236. 10.1007/s10353-010-0561-y
9. Sahni S, Shiralkar M, Mohamed S, et al.: Superior mesenteric artery syndrome: the dark side of weight loss. Cureus. 2017, 9:1-5. 10.7759/cureus.1859
10. Merrett ND, Wilson RB, Cosman P, Biankin AV: Superior mesenteric syndrome: diagnosis and treatment strategies. J Gastrointestinal Surg. 2009, 13:287-92. 10.1007/s11605-008-0695-4