An Overlooked Cause of Upper Gastrointestinal Obstruction in Children

Al Munther Al Oraimi[1], Yusriya Al Rawahi[1,2*], Yarab Al Balushi[3], Aftab Anwar[2], Sumaiya Al Hadhrami[4] and Naeema Al Shibili[5]

[1]*Department of Child Health, College of medicine and health sciences, Sultan Qaboos University, Muscat, Oman*

[2]*Pediatric Gastroenterology, Hepatology and Nutrition Unit, Department of Child Health, Sultan Qaboos University, University Medical City, Muscat, Oman.*

[3]*Department of Radiology and Nuclear Medicine, Sultan Qaboos University Hospital, University Medical City, Muscat, Oman.*

[4]*Development unit, Department of Child Health, Sultan Qaboos University, University Medical City, Muscat, Oman.*

[5]*General Pediatic unit, Department of Child Health, Sultan Qaboos University, University Medical City, Muscat, Oman.*

Received: 31 August 2025

Accepted: 22 October 2025

*Corresponding author: yusria@squ.edu.om

DOI 10.5001/omj.2025.95

A 10-year-old girl presented to the Emergency Department (ED) with severe epigastric pain, nausea, vomiting and 15% weight loss. The vomiting was non-bloody, non-bilious and associated with poor oral intake for two months. There was no associated loose stools, fever or headache. Her past medical history was significant for vegan diet for two years. However, there was no history suggestive of body image issues. Clinical examination revealed a thin girl with reduced muscle bulk. Her weight was 27 Kg at 5th centile, her height was 137 cm at 25th centile and her BMI was 14 at the 5th centile. Her vital signs showed blood pressure of 104/75 mmHg, heart rate of 89bpm and temperature of 36.7 C. There was no orthostatic tachycardia or hypotension. Her abdominal examination revealed a scaphoid abdomen which was soft with some epigastric tenderness. Her cardiopulmonary examinations were unremarkable.

Investigations showed haemoglobin of 10.4 g/dL (Reference range: 11.5 g/dl-15.5 g/dl). with MCH of 29 pg (24 -33), MCV of 85.5 fL (73- 95 fl), RDW of 14.5% (1.5- 16.5%). She has normal white cell count with normal differntials). She has normal inflammatory markers, normal liver enzymes and normal renal function test., Her thyroid function test, Vitamin D and Lactate levels were normal. Her bone profile showed Alkaline phosphate (ALP): 80 U/L (129-417 U/L), with normal albumin, calcium and phosphorus levels.

For evaluation of abdominal pain and vomiting, she had an upper gastrointestinal contrast study [Figure 1], and an abdomen computed tomography [Figure 2].

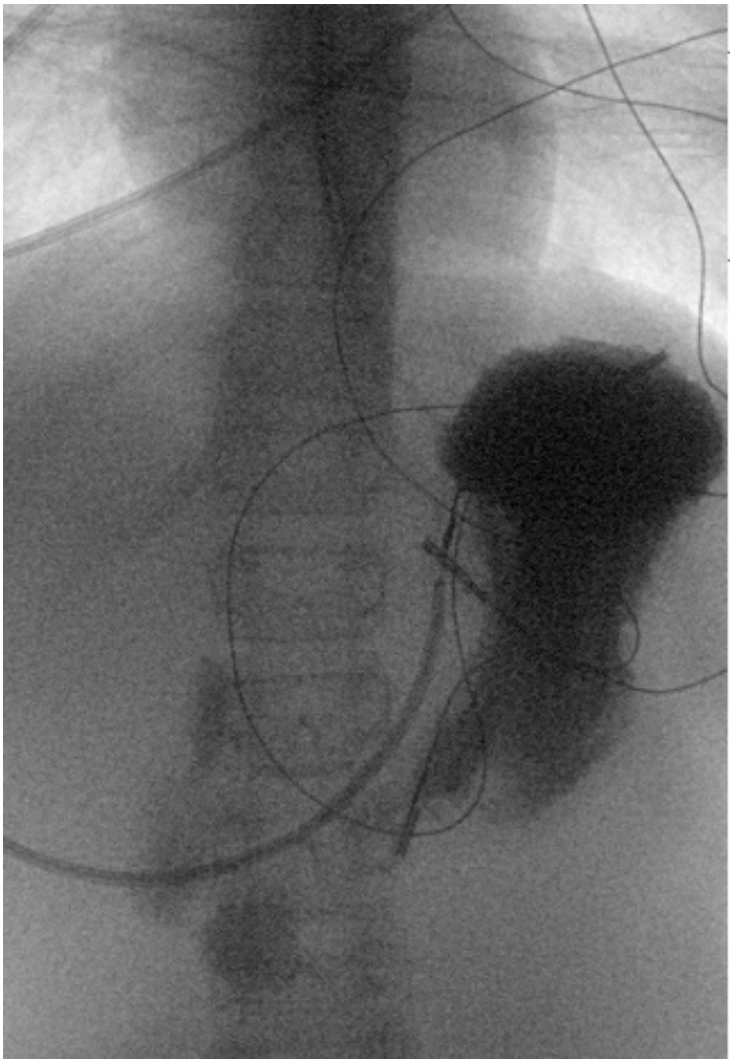

Figure 1: Un upper GI single contrast study. Prolonged contrast hold-up was noted at the mid duodenum. The stomach was partially decompressed by nasogastric tube.

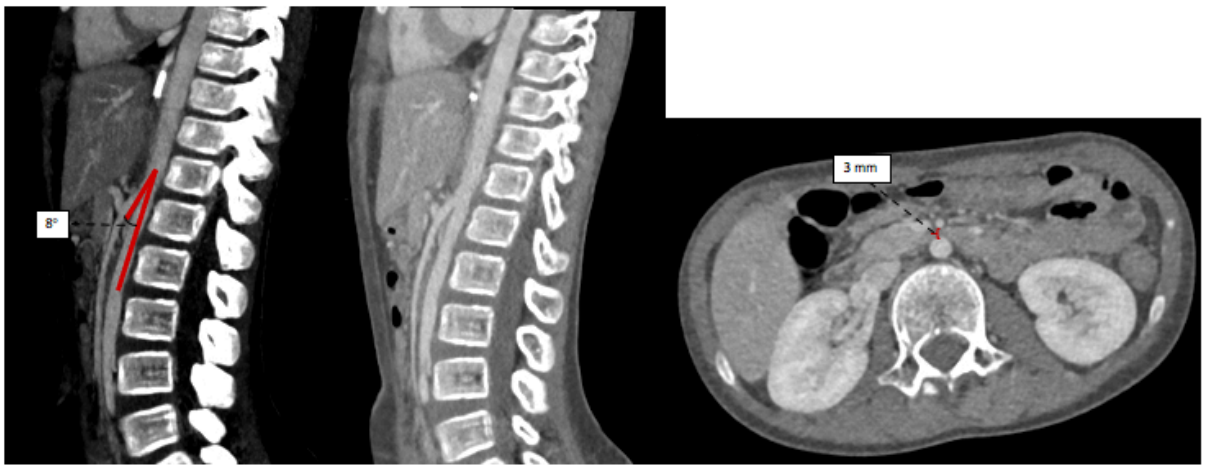

Figure 2: Contrast-enhanced CT of the Abdomen and Pelvis. Reduced aortomesenteric angle measures 8° and reduced aortomesenteric distance of 3 mm. Third part of the duodenum is compressed. Measurements along with fluoroscopic findings are suggestive of SMA syndrome.

Questions

1. What is the most likely diagnosis?
2. What is the management of this condition?

Answers

1. Superior mesenteric artery syndrome
2. Gastric decompression with nasogastric tube, fluid and electrolyte correction, and nutritional rehabilitation, enterally or parentally to increase weight.

Discussion

The upper gastrointestinal study showed contrast hold-up at the level of the third part of the duodenum [Figure 1]. Abdominal CT showed reduced aortomesenteric angle of 8 degrees and reduced distance of 3 mm [Figure 2], in keeping with a diagnosis of Superior Mesenteric Artery Syndrome (SMAS). SMAS, also known as Wilkie's syndrome, is a rare condition that typically presents as mid- to upper gastrointestinal obstruction caused by an acquired vascular compression of the duodenum between the aorta and the superior mesenteric artery.[1,2] Although it most commonly affects young females in their twenties, it can occur at any age. Recent literature has also reported an increasing number of cases among elderly patients.[3] SMAS in children is extremely rare, and its exact prevalence remains unknown as no pediatric-specific epidemiological studies are available. The prevalence is thought to be similar to that reported in the general population (0.01–0.3%).[3] However, the incidence rises to 2–3% among children undergoing spinal surgery.[4]

SMAS is usually an acquired condition caused by conditions associated with significant weight loss or catabolic states, such as anorexia nervosa, severe burns, malabsorption syndromes, malignancy, and recent upper abdominal gastrointestinal surgeries. Mesenteric fat helps preserve this angle; however, with significant weight loss, the fat cushion diminishes leading to sharper angle, resulting in the SMA compressing the 3rd part of the duodenum. This leads to further weight loss and, thereby perpetuating a vicious cycle.[5]

Additionally, patients undergoing scoliosis surgery may develop SMAS due to increased tension on the mesentery following spinal correction.[3] However, congenitally short or hypertrophic ligament of Treitz, low origin of the superior mesenteric artery, an inherently narrow angle between the SMA and aorta, reduced aortomesenteric distance, and intestinal malrotation may contribute to the development of SMAS in children.[3]

Patients with SMAS typically present with postprandial abdominal pain, persistent emesis, prolonged absence of stooling, and weight loss. They are often misdiagnosed with constipation. SMAS should be considered as a differential diagnosis, especially with the patients with acute weight loss.[2,6] The diagnosis of SMAS requires a high index of clinical suspicion. In addition to the clinical picture, several imaging modalities can aid in diagnosis. Abdominal sonography may reveal a dilated duodenum with or without gastric dilatation. CT scan is considered the gold standard as it allows for measurement of the aortomesenteric angle and visualization of duodenal compression. The aortomesenteric angle is considered normal if it is between 28 and 65 degrees. The aortomesenteric distance is considered normal if it is between 10 to 34 mm.[7] The upper gastrointestinal barium study may demonstrate contrast pooling in the stomach with delayed transit into the duodenum. Endoscopy can be used to detect any gastric complications.[3, 7]

Management of SMAS is typically conservative and focuses on bowel decompression, correction of the fluid and electrolyte imbalance, and nutritional support such as total parenteral nutrition. Once the patient is stabilized, small, frequent, high-caloric oral feeds are introduced to promote gain weight.[1] Adjustment in feeding position, such as knees-to-chest or right lateral decubitus position, may also help alleviate the symptoms by facilitating duodenal passage of food.[3] If symptoms persist, placement of a nasojejunal tube beyond the obstruction point may allow for continuous enteral feeding. When conservative management fails, particularly in the presence of profound proximal dilatation and stasis, surgical intervention becomes the second line. Based on the clinical scenarios, surgical procedures such as duodenojejunostomy, gastrojejunostomy, or resection of the ligament of Treitz, may be considered to manage the obstruction.[1] In our patient, after confirming the diagnosis, she was initially managed with a nasogastric tube for gastric decompression. Enteral nutrition was

later initiated via a nasojejunal tube (NJT), resulting in a 7 kg weight gain during admission. Subsequently, she was started on oral feed and discharge home on stable condition.

In summary, although SMAS is a rare cause of duodenal obstruction in children. It is typically characterized by compression of the third part of the duodenum and a reduced aortomesenteric angle and distance. Clinicians should maintain a high index of suspicion for it, especially in the context of recent weight loss or surgery, such as spinal correction procedures. Management includes gastric decompression, fluid and electrolyte correction, and nutritional support. Early recognition and appropriate management are vital to improving outcomes and avoiding complications.

References

1. Biank V, Werlin S. Superior mesenteric artery syndrome in children: a 20-year experience. J Pediatr Gastroenterol Nutr. 2006 May;42(5):522-5. doi: 10.1097/01.mpg.0000221888.36501.f2. PMID: 16707974.
2. Waheed KB, Shah WJ, Jamal A, Mohammed HR, Altaf B, Amjad M, Bassam MA, Almutawa DH, Arulanantham ZJ. Superior mesenteric artery syndrome: An often overlooked cause of abdominal pain! Saudi Med J. 2021 Oct;42(10):1145-1148. doi: 10.15537/smj.2021.42.10.20210509. PMID: 34611011; PMCID: PMC9129241.
3. Oka, A., Awoniyi, M., Hasegawa, N., Yoshida, Y., Tobita, H., Ishimura, N., & Ishihara, S. (2023). Superior mesenteric artery syndrome: Diagnosis and management. World Journal of Clinical Cases, 11(15), 3369–3663. https://doi.org/10.12998/wjcc.v11.i15.3369
4. Tsirikos, A. I., & Jeans, L. A. (2005). Superior mesenteric artery syndrome in children and adolescents with spine deformities undergoing corrective surgery. *Journal of spinal disorders & techniques*, *18*(3), 263–271.
5. Bozzola, E.; Irrera, M.; Cirillo, F.; Zanna, V.; Petrelli, I.; Diamanti, A.; Scire, Y.; Park, J.;Marchesi, A.; Marchili, M.R.; et al. Superior Mesenteric Artery Syndrome in Anorexia Nervosa: A Case Report and a Systematic Revision of the Literature. Nutrients 2024, 16, 541. https://doi.org/ 10.3390/nu16040541
6. England J, Li N. Superior mesenteric artery syndrome: A review of the literature. JACEP Open.2021;2:e12454. https://doi.org/10.1002/emp2.12454
7. Kim JY, Shin MS, Lee S. Endoscopic features for early decision to evaluate superior mesenteric artery syndrome in children. BMC Pediatr. 2021 Sep 8;21(1):392. doi: 10.1186/s12887-021-02848-0. PMID: 34496824; PMCID: PMC8424886.
8. Lamba R, Tanner D, Sekhon S, McGahan J, Corwin M, Lall C. Multidetector CT of Vascular Compression Syndromes in the Abdomen and Pelvis. Radiographics. 2014;34(1):93-115. doi: 10.1148/rg.341125010. Erratum in: Radiographics. 2015 May-Jun;35(3):973. doi: 10.1148/rg.2015154007.