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Superior Mesenteric Artery Syndrome Mimicking Functional Vomiting in an Adolescent: A Case Report

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KEYWORDS

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ABSTRACT:

Superior mesenteric artery (SMA) syndrome, also known as Wilkie or Cast syndrome, is a rare cause of proximal small bowel obstruction resulting from compression of the third part of the duodenum between the SMA and aorta due to a narrowed aorto-mesenteric angle. It typically presents with postprandial abdominal pain, nausea, and vomiting, with diagnosis primarily confirmed through radiological imaging. A 14-year-old male presented with persistent vomiting for one week, along with a history of similar episodes over the preceding six years. Physical examination revealed mild dehydration, a scaphoid, soft, and non-tender abdomen. Imaging demonstrated a reduced SMA angle, decreased aorto-mesenteric distance, and duodenal compression, consistent with SMA syndrome. The patient was managed conservatively with aprepitant, leading to symptom resolution and an uncomplicated discharge. SMA syndrome remains a rare and often under-recognised condition; delayed diagnosis can lead to significant morbidity. Prompt recognition and appropriate management, whether conservative or surgical, generally result in favourable outcomes. Clinicians should maintain a high index of suspicion in patients with chronic, unexplained upper gastrointestinal symptoms to avoid diagnostic delay.

Background

Superior mesenteric artery (SMA) syndrome is a rare condition caused by vascular compression of the third part of the duodenum between the aorta and the SMA. The clinical features of duodenal obstruction include postprandial epigastric pain, nausea, vomiting, anorexia, and weight loss, and the condition is also termed Wilkie or cast syndrome. It was first described by the Austrian professor Carl von Rokitansky in 1842.(1) The SMA originates behind the neck of the pancreas at the level of the first lumbar vertebra and leaves the aorta at an acute angle. The aorto-mesenteric angle contains the left renal vein, uncinate process of the pancreas, and lymphatics, embedded in retroperitoneal fat. Loss of this fat is considered a key factor in reducing the angle. Three duodenal obstruction mechanisms have been described: (i) a very acute aorto-mesenteric angle; (ii) a high-positioned transverse duodenum due to a short ligament of Treitz; (iii) an anomalous route of the mesenteric artery or its branches displaced downwards and anterior to the spine.(2)

Symptoms may include intermittent or postprandial abdominal pain, vomiting, and anorexia. Examination may reveal abdominal distension, poorly localised pain, and altered bowel sounds.(3) Imaging modalities include upper GI series, CT, CT angiography, MR angiography, conventional angiography, ultrasonography, and endoscopy.(4) Conservative management aims at weight gain to restore retroperitoneal fat and increase the aorto-mesenteric angle.(4) If unsuccessful, surgery such as duodenojejunostomy is preferred.(5)

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Case Report

A 14-year-old male presented to the outpatient department with persistent vomiting for one week, unresponsive to ondansetron. The vomiting was spontaneous, contained food particles, was non-projectile, occasionally bile-stained, and never blood-stained. His mother reported similar episodes during certain months each year for the past six years, with complete symptom-free intervals in between. These episodes were previously managed symptomatically as functional or psychological vomiting, without thorough evaluation.

There was no history of fever, headache, altered sensorium, dizziness, photophobia, blurring of vision, syncope, seizures, abdominal trauma, jaundice, or tuberculosis contact. During symptomatic periods, his appetite decreased, and weight gain was poor.

On examination, the patient was mildly dehydrated, vitals stable, and BMI 13.35 (<3rd percentile, WHO). Abdominal exam was unremarkable. Cardiovascular, respiratory, and neurological findings were normal. Labs showed normal renal/liver function, haemoglobin 15.3 g/dL, mild leukocytosis with neutrophilia, lymphocytopenia, mildly elevated lipase (303 IU/L), normal amylase, electrolytes, and C-reactive protein.

Ultrasound abdomen showed anatomical variations without obstruction. Based on Rome IV criteria, cyclic vomiting syndrome was provisionally diagnosed, and ondansetron continued. On day two, he developed bilious vomiting. Oral intake was stopped, and a nasogastric tube inserted for decompression.

EEG ruled out Panayiotopoulos syndrome but showed epileptiform activity in the temporal region; anti-epileptics were started on neurology advice. Upper GI endoscopy revealed pangastritis and bulbar duodenitis without ulceration; biopsy was positive for *Helicobacter pylori* (Figure 1). Triple therapy was withheld as no ulceration was seen.

On literature review, SMA syndrome was considered. Repeat abdominal ultrasound, focusing on SMA parameters, showed reduced aorto-mesenteric angle (13.3°) and distance (3.3 mm) without proximal loop dilatation. Contrast-enhanced CT and CT angiography confirmed an acute angle (~20°), aorto-mesenteric distance 3 mm, mild compression of the third part of the duodenum, mild second-part dilatation, collapse of the fourth part, para-aortic lymph node (13×7 mm), compression of the left renal vein with pre-stenotic dilatation (compression ratio: 3), and reduced mesenteric fat (Figure 2).

Barium meal follow-through showed normal gastric emptying and contrast passage without hold-up (Figure 3).

The patient was started on parenteral nutrition for four weeks to promote weight gain. Multiple anti-emetics were ineffective until two doses of aprepitant stopped vomiting. Oral feeds with high-calorie supplements were gradually reintroduced; he gained 1.2 kg before discharge. Anti-epileptics were discontinued after two months. At six-month follow-up, he remained symptom-free with improved appetite and weight gain.

Discussion

SMA syndrome, first described by Carl von Rokitansky in 1842, refers to obstruction of the third part of the duodenum between the aorta and the superior mesenteric artery. The reported prevalence is 0.013%–0.3%.(6) In humans, the erect posture produces an aorto-SMA angle of 38–56°. The key anatomical feature is narrowing of this angle to <25°, reducing the aorto-mesenteric distance to <10 mm compared with the normal 10–28 mm.(4,7) Compression of the left renal vein with the duodenum is termed “Nutcracker syndrome”.(8) SMA syndrome occurs most often in females and young adults (18–35 years), often linked to eating disorders.(9) Our patient, a 14-year-old male, falls outside this typical demographic, making the presentation unusual.

Predisposing factors fall into three groups: i) severe weight loss in catabolic states, ii) external or intra-abdominal compression, and iii) mesenteric tension.(4) These include chronic wasting diseases (e.g., cancer, cerebral palsy, paraplegia, juvenile rheumatoid arthritis, cardiac cachexia, drug abuse), trauma (burn injury, brain injury, multiple injuries), dietary disorders (anorexia nervosa, malabsorption), post-operative states (bariatric surgery, proctocolectomy, Nissen fundoplication, aortic aneurysm repair, spinal instrumentation, scoliosis surgery, body casting), anatomical/congenital anomalies (high insertion of the ligament of Treitz, intestinal malrotation, peritoneal adhesions, low SMA origin,

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increased lumbar lordosis), and local pathology (mesenteric root tumours, dissecting aortic aneurysm).

Patients usually present with chronic abdominal symptoms and intermittent exacerbations. The most common is post-prandial abdominal pain (59–81%) with vomiting, nausea, anorexia, and weight loss.(4) In our case, the six-year history of seasonal, intermittent vomiting—with symptom-free intervals—was atypical, as SMA syndrome usually causes persistent or progressively worsening symptoms. This prolonged intermittent course in a paediatric patient is rarely documented. The condition was repeatedly labelled as functional/psychological vomiting, illustrating its ability to mimic functional gastrointestinal disorders and delay diagnosis. Complications include electrolyte imbalance, massive gastric dilatation, pneumatosis, and portal venous gas.(9) Severity correlates with the reduction in aorto-mesenteric distance.(3)

Diagnosis relies on barium meal follow-through, ultrasound with colour Doppler, CT, CT angiography, MR angiography, and endoscopy. CT achieves near 100% sensitivity and specificity when: (i) there is abrupt obstruction in the third duodenum with active peristalsis, (ii) an aorto-mesenteric angle <25°(10) and/or distance ≤8 mm,(11) and (iii) duodenal fixation by the ligament of Treitz or vascular variants.(11) Our patient's barium study showed normal contrast transit, which is unusual in SMA syndrome, yet targeted ultrasonography and CT angiography—prompted by persistent symptoms—confirmed severe angle narrowing and coexisting left renal vein compression, consistent with Nutcracker phenomenon.

Acute presentations generally respond to conservative management, though duration is undefined.(12) Treatment includes fluid/electrolyte correction, nutritional rehabilitation to restore retroperitoneal fat and widen the angle, gastric decompression, and positional therapy. In chronic cases like ours, surgery is often required; however, this patient achieved complete resolution with nutritional support and short-term aprepitant, gaining 1.2 kg during admission and remaining asymptomatic for six months. Surgical intervention is reserved for failed conservative management, progressive disease, or complications, with duodenojejunostomy having up to 90% success.(5)

This case highlights that even long-standing SMA syndrome in a paediatric male, with intermittent symptoms and initially normal barium findings, can resolve with targeted conservative therapy when the diagnosis is finally established.

Conclusion

Superior mesenteric artery syndrome is rare and often under-recognised. High clinical suspicion, targeted imaging, and complete evaluation are crucial. Early diagnosis allows effective conservative management, preventing unnecessary surgery and reducing prolonged morbidity, even in chronic, atypical paediatric presentations.

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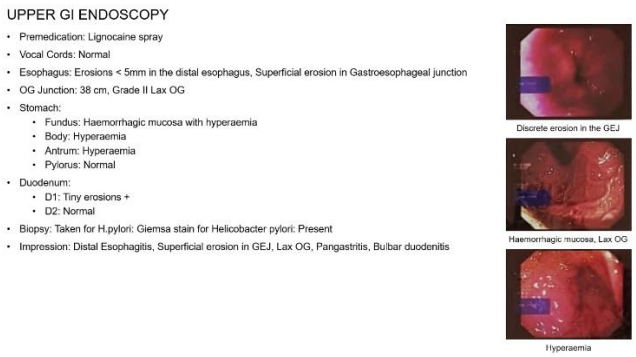

Figure 1: Upper gastrointestinal endoscopy showing discrete erosion in the gastroesophageal junction, haemorrhagic mucosa with lax gastro-esophageal junction, and gastric hyperaemia

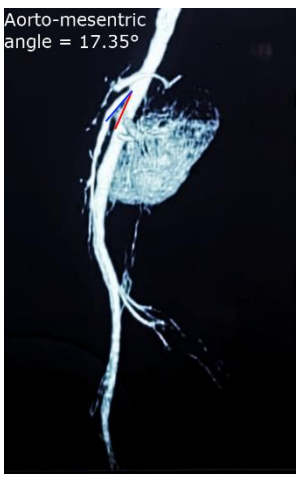

Figure 2: Contrast enhanced computed tomography with CT angiogram showing reduced aorto-mesenteric angle* = 17.35º with compression of the left renal vein (nutcracker syndrome)
**The sagittal CT angiogram image depicts the aorto-mesenteric angle measurement, indicative of narrowing. In the annotated image, the aorta is represented by a red line and the superior mesenteric artery by a blue line to illustrate the angle measurement.*

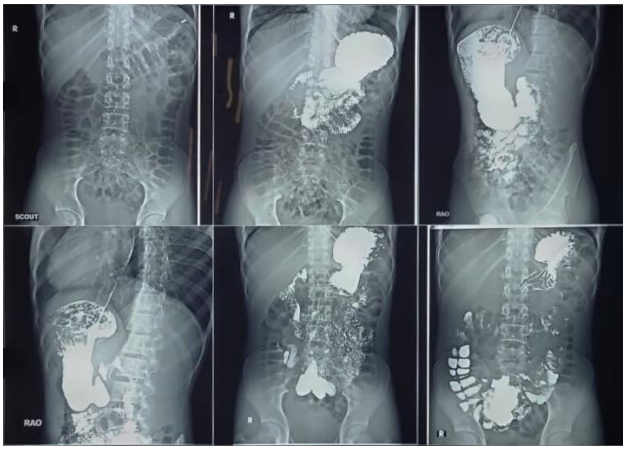

Figure 3: Barium meal follow-through series demonstrating normal passage of contrast through the stomach, duodenum, and small bowel without evidence of hold-up