

Superior Mesenteric Artery Syndrome as an Atypical Aetiology of Upper Intestinal Obstruction in a Young Adult

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ABSTRACT:

Superior mesenteric artery (SMA) syndrome is a rare cause of proximal bowel obstruction in young adults. Weight loss is one of the most significant aetiology of this condition. In young adults presenting with post-prandial abdominal pain, abdominal distension, anorexia and voluminous vomiting, the diagnosis of superior mesenteric artery syndrome should be considered. These symptoms are due to compression of the third part of the duodenum against the aorta by the overlying superior mesenteric artery. Computed tomography (CT) angiography of the abdomen is currently favoured in the literature to establish the diagnosis. We report a case of SMA syndrome in a 20-year-old Malay gentleman with a history of significant weight loss and kratom abuse for a year. A CECT scan of the abdomen was performed which established the diagnosis. This patient responded to conservative treatment of nutritional supplementation.

Keywords:

Superior mesenteric artery syndrome, acute abdomen, vascular compression syndrome

INTRODUCTION:

Superior mesenteric artery (SMA) syndrome was described in 1861 by Rokitansky. Later, in 1927, Wilkie described in more detail the clinical, anatomy and pathophysiological features and named it chronic duodenal ileus. SMA syndrome is also known as Cast syndrome or Wilkie syndrome. Typically, there is retroperitoneal fat surrounding the third part of the duodenum (D3), which acts as a cushion between the SMA anteriorly and the aorta posteriorly. SMA syndrome is caused by the loss of this fat, causing

a decrease in the aorto-mesenteric distance and angle, which leads to obstruction of the D3 part of the duodenum due to compression between the SMA and the aorta. This could result in various scenarios, including acute, intermittent and chronic, and partial or total duodenal obstruction. SMA syndrome is a rare cause of proximal duodenal obstruction.

CASE PRESENTATION:

A 20-year-old Malay gentleman with no known medical illness presented to the Accident and

Emergency Department with recurrent central abdominal pain associated with multiple episodes of vomiting. The symptoms progressively worsened over two weeks. The pain was localised to the epigastric region without any radiation to other areas. There were no relieving or aggravating factors. It was associated with increasing distension of the upper abdomen over one week. Prior to this, he had poor appetite, early satiety, intolerance to solid food and recurrent post-prandial vomiting for the past two months. This patient has also noticed a significant weight loss of approximately 10kg since he regularly consumed kratom a year ago. On examination, he had a thin build with a distended upper abdomen. The abdomen was mildly tender to palpation, but there was no guarding. Blood investigation parameters were unremarkable.

The supine abdominal radiograph showed a grossly dilated gastric cavity extending from the left upper quadrant to the central abdomen (Figure 1). No small or large bowel dilatation was seen. No pneumoperitoneum features were seen in the erect chest and supine abdominal radiographs. Ultrasound of the abdomen showed similar features (Figure 2). No free fluid was seen. The rest of the solid intraabdominal organs were unremarkable. In order to identify the cause of the obstruction, a contrast-enhanced computed tomography (CECT) scan of the abdomen was done which showed a severely dilated stomach and proximal duodenum up the level of the D3 part of the duodenum. A transition point with abrupt tapering was noted at the duodenum traversing between the superior mesenteric artery and abdominal aorta (Figure 3). The aortomesenteric angle was reduced, measuring 15 degrees (Figure 4). The aortomesenteric distance was also reduced, measuring 4mm (Figure 5). The location of the superior mesenteric vein (SMV) relative to the superior mesenteric artery (SMA) was normal in this patient, with the SMV lying to the right of the SMA, which pointed against a diagnosis of malrotation. No whirlpool sign was seen to suggest volvulus. No pneumoperitoneum or pneumatosis intestinalis was seen. The rest of the small and

large bowels were not dilated and were unremarkable. Ground glass opacities were seen at the left lower lobe, most likely secondary to aspiration pneumonia due to recurrent vomiting.

A diagnosis of SMA syndrome was made. This patient was treated conservatively. A nasogastric tube was inserted to decompress the gastric cavity. He was referred to a dietician who started him on enteral nutrition composed of titrated Ensure® Gold milk (Abbott Laboratories, IL, USA) through nasogastric tube feeding, targeting a daily calorie intake of 1700 kcal per day. He had significant clinical improvement and subsequently started on a high-calorie liquid and soft diet. He was also referred to social welfare and drug rehabilitation after discharge. On his last follow-up, he remained asymptomatic.

DISCUSSION:

SMA syndrome is rare, its prevalence in the chronic hospital setting is approximately 0.0965% and 0.0011 to 0.0052% in an acute hospital setting[1]. 75% of the cases occur in individuals 10 to 39 years old, with a slight female preponderance of approximately 64% [2]. Symptoms include post-prandial epigastric pain, fullness in the upper abdomen, nausea, vomiting and anorexia[2]. The pain, as in our patient, is most likely caused by stretching of the gastric cavity and might be relieved by a prone or left lateral decubitus position. The SMA originates at the level of L1-L2 and courses anterior and inferiorly, forming an angle with the aorta named the aortomesenteric angle (AMA)[3]. The D3 part of the duodenum, which is usually surrounded by retroperitoneal fat, passes between the aorta and proximal SMA. The fat layer acts as a cushion between the SMA and the aorta [2]. Loss of this fat causes the SMA to compress onto the D3 part of the duodenum against the aorta, as seen in our patient [4]. Repeated vomiting with weight loss will result in a self-propagating cycle leading to further progressive loss of the retroperitoneal fat, causing reduced cushioning of the D3 segment of duodenum and smaller aortomesenteric distance (AMD) and AMA [3].

SMA syndrome is known to be caused by rapid and significant weight loss as a result of conditions such as acquired immunodeficiency syndrome, malabsorption, cancer, burns, major surgery, eating disorders, drug abuse and bariatric surgery [2,3]. Our patient's history of regular kratom abuse could have predisposed him to this condition. Kratom contains mitragynine and 7-hydroxymitragynine, which act on opioid receptors [5]. Long-term use of kratom is known to cause poor appetite, gastrointestinal discomfort and weight loss [5]. In the literature, there are reported cases of SMA syndrome caused by heroin abuse, which similarly binds to opioid receptors [6]. SMA syndrome could also be found among post-corrective scoliosis surgery patients due to increased tension on the mesentery after lengthening the spine [3]. Another less common cause includes the application of external abdominal pressure by a body or hip spica cast [3].

Radiological examination is the primary investigative tool used to diagnose these patients, with CECT gaining the most traction. The CECT provides an assessment of the aortic vascular anatomy, D3 duodenal compression, and demonstrates dilated proximal bowels while excluding other obstruction causes. Hence, it is the diagnostic imaging modality of choice for SMA syndrome [3]. The scan is recommended to be done in the late arterial phase to allow simultaneous demonstration of the vessel anatomy and the obstructed bowels optimally. Positive oral contrast agents may be excluded in patients with severe obstruction, considering the risk of aspiration pneumonia [2,3]. The AMA is the angle between the SMA and the aorta. It is measured in a sagittal plane where the third part of the duodenum passes underneath SMA. The standard AMA is reported to be 28 to 65 degrees. The AMD is the distance between the SMA and the aorta. It is measured in an axial plane at the level of D3 duodenum as it travels between the SMA and the aorta. The typical AMD measurement is 10 to 28mm [2,3]. The two key imaging features in diagnosing SMA syndrome include an AMA which is smaller than 22 degrees and an AMD of

lesser than 8mm. An AMA of smaller than 22 degrees has a sensitivity of 42.8% and a specificity of 100%, while an AMD of lesser than 8mm has a sensitivity of 100% and a specificity of 100% for diagnosing SMA syndrome [2]. Other ancillary imaging findings include a dilated gastric cavity and proximal duodenum up to the level of D3 segment of the duodenum, followed by abrupt tapering where the horizontal part of the duodenum passes between the SMA and aorta [2]. All of these findings were demonstrated in our patient.

The initial management of SMA syndrome is usually conservative, which includes decompressing the stomach and duodenum using a nasogastric tube followed by providing enteral feeding [7]. This is followed by nutritional support with a small quantity of high-calorie liquid meals aimed at restoring the retroperitoneal fat and helping in weight gain in order to build up the fatty cushion surrounding the duodenum and increase the AMA and AMD [8]. Surgery is indicated when conservative management is unsuccessful or in the presence of severe symptoms [8]. The surgery of choice would be duodenojejunostomy which could be performed either open or laparoscopically [8]. Our patient showed an excellent response to conservative therapy.

CONCLUSION:

SMA syndrome is a rare cause of proximal bowel obstruction but should be kept in mind in young adults with a history of weight loss and persistent post-prandial vomiting. A CECT scan of the abdomen provides the diagnosis as well as excludes other causes. Chronic conditions such as malabsorption, eating disorders and substance abuse should be considered as contributory factors in patients with SMA syndrome. Once the patient has been stabilised, the long-term management should be focused on correcting the aetiology of SMA syndrome. Conservative management by providing high-calorie nutritional support can be attempted to solve the symptoms prior to surgical management.

CONFLICTS OF INTEREST:

The authors have no potential conflicts of interest to report regarding this presentation.

PATIENTS' CONSENT FOR THE USE OF IMAGES AND CONTENT FOR PUBLICATION:

Consent had been acquired from the patient to publish images and content.

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FIGURE LEGENDS:

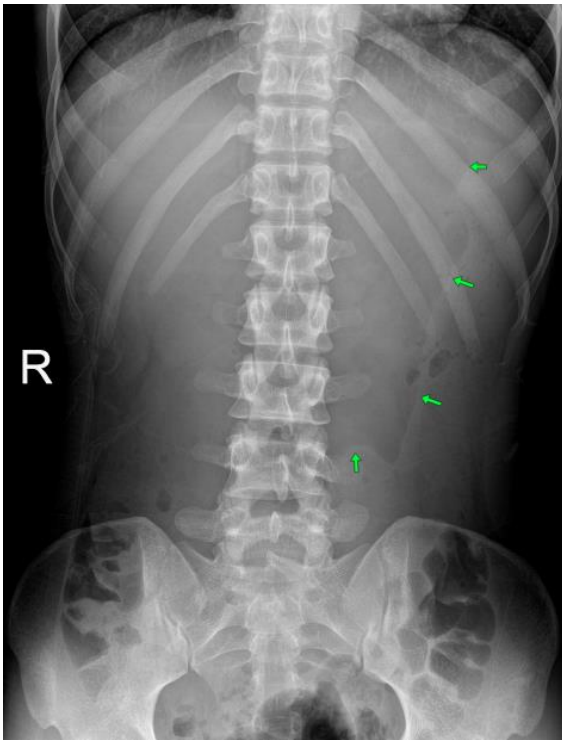


Figure 1: Supine abdominal radiograph showing gross dilatation of the gastric cavity (green arrows) extending to the central abdomen. No evidence of small and large bowel dilatation. No features of pneumoperitoneum.

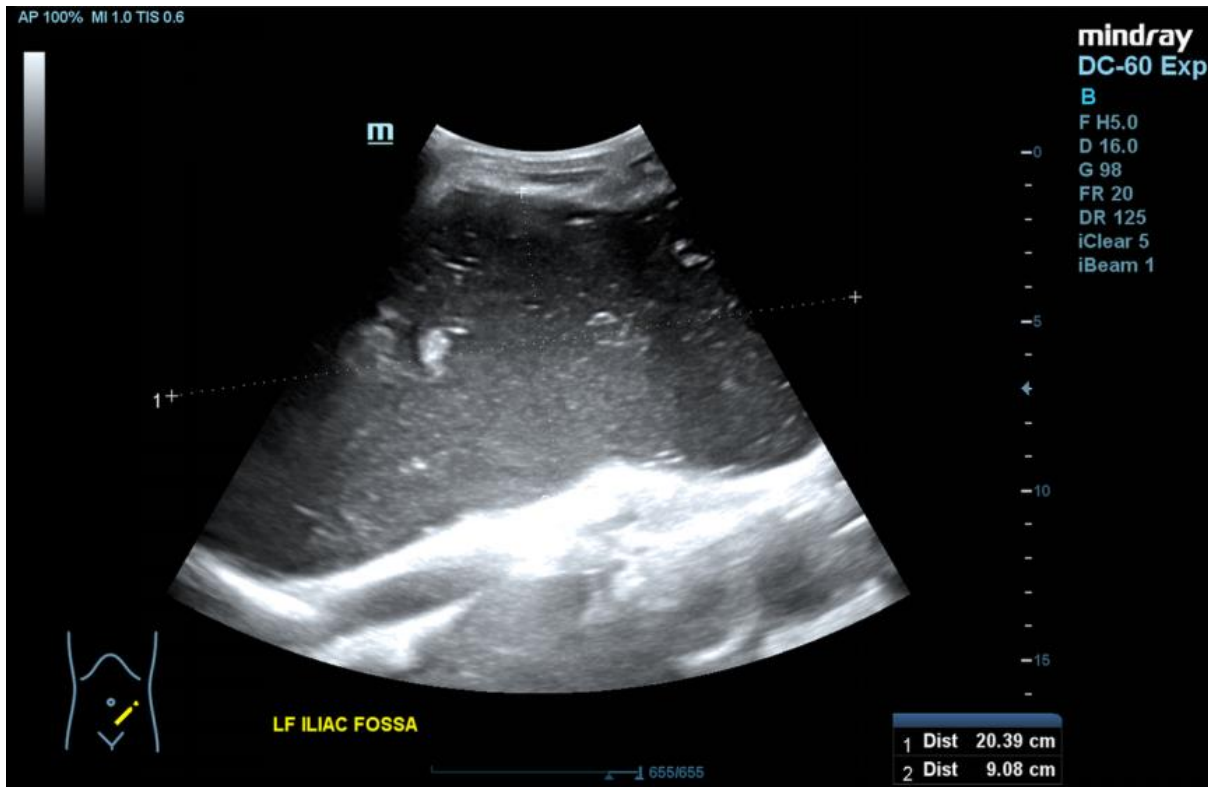


Figure 2: Abdomen ultrasound image showing dilated gastric cavity, which is fluid-filled, extending to just above the left iliac fossa.

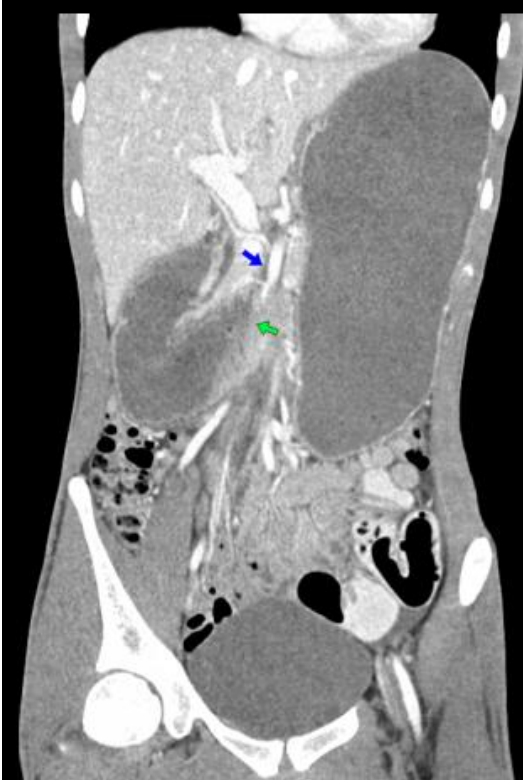


Figure 3: Coronal CT images showing grossly dilated gastric cavity and proximal duodenum. The transition point (green arrow) is at the origin of the SMA (blue arrow) from the aorta.

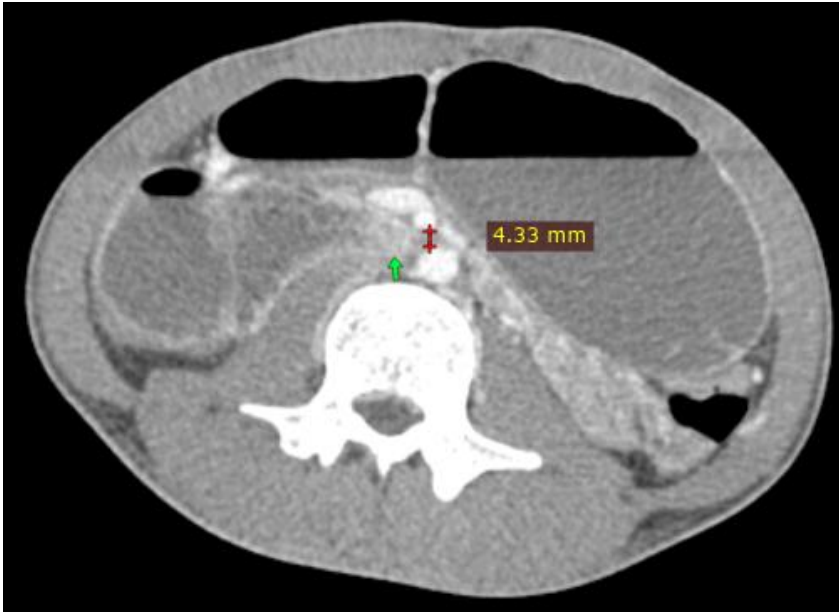


Figure 4: Axial CT image showing abrupt tapering of the third part of the duodenum (green arrow) as it passes between the SMA and the aorta. The aorto-mesenteric distance is reduced, measuring 4mm.



Figure 5: Sagittal CT image showing a narrowed aorto-mesenteric angle of 15 degrees.