CLINICAL VIGNETTE

Superior Mesenteric Artery Syndrome in a Patient with Underlying Functional Dyspepsia

Adrienne Lenhart, MD and Sonya Dasharathy, MD

Case Presentation

A 21-year-old female with anxiety, depression, borderline personality disorder, and anorexia nervosa in remission, presented to gastroenterology with six months of epigastric pain, bloating, early satiety, weight loss, and nausea. She denied significant heartburn, acid reflux, vomiting, changes in bowel habits, or GI bleeding and there was no change in abdominal pain with defecation. Her epigastric pain and bloating worsened with increased stress and varied with diet. She reported increased stress prior to symptom onset and developed anticipatory anxiety regarding food intake and dietary restrictions. Trigger foods included dairy, gluten, and fatty or greasy foods. She reported 12-pound weight loss in the preceding 6 months with symptoms several times a week, which severely limited socialization and activities of daily life.

Initial evaluation included BMI of 17.2 and unremarkable physical exam. Labs included normal complete blood count, metabolic panel, TSH and inflammatory markers. Celiac antibodies and Helicobacter pylori stool antigen were also negative. CT abdomen/pelvis without contrast and esophagogastroduodenoscopy (EGD) were also unremarkable. She was initially diagnosed with functional dyspepsia with overlapping epigastric pain and postprandial distress syndrome subtypes, and was started on nightly amitriptyline 25 mg. She worked on diaphragmatic breathing exercises and regular visits with her therapist. However, symptoms progressed over the next few months with worsening nausea, vomiting, and epigastric pain. She reported inability to eat due to pain and lost an additional 10 pounds since initial presentation with BMI of 15.23.

She was admitted to a local community hospital and a CT angiogram of the abdomen/pelvis was performed. This showed patency of the celiac axis, superior mesenteric artery (SMA), and inferior mesenteric artery (IMA). However, the aortomesenteric angle measured 17 degrees, consistent with SMA syndrome. Upper GI series (UGIS) with small bowel follow through (SBFT) did not show obstruction but did show slow movement of contrast through the third portion of the duodenum. Gastroenterology and surgery consultations discussed total parenteral nutrition (TPN) vs gastrojejunostomy placement for enteral feedings. Surgery would be considered if symptoms persisted after weight gain following conservative measures. She elected to proceed with TPN with a goal 15–20-pound weight gain.

Over the next few months, she was followed by GI, Psychiatry, the GI Integrative Clinic, and Nutrition and enrolled in an Intensive Outpatient Program for eating disorders. She started nightly mirtazapine 7.5 mg which was titrated to 30 mg qhs. She reached her goal weight and reported significant improvement in her GI symptoms. TPN was discontinued and she remained on mirtazapine.

Discussion

SMA syndrome is a rare cause of intestinal obstruction, with an incidence ranging from 0.013 to 0.3%, and is more common in women.[1] SMA syndrome is characterized by compression of the third portion of the duodenum from narrowing of the space between the superior mesenteric artery and the aorta. This is typically caused by loss of the mesenteric fat pad between the aorta and the SMA. With the mesenteric fat pad in appropriate position, the angle between the SMA and the aorta is between 38-65 degrees, with a normal aortomesenteric distance of 10-28 mm. However, in SMA syndrome, the aortomesenteric angle can be narrowed to as low as 6 degrees with the aortomesenteric distance decreasing to 2-8 mm.[2,3] This decreased distance can lead to subsequent duodenal compression.

There are several risk factors for SMA syndrome. The most common is weight loss leading to subsequent loss of the mesenteric fat pad. This usually occurs with severe illness, malignancy, malabsorption syndromes, psychological disorders, or eating disorders like anorexia nervosa.[4] There have also been case reports of SMA syndrome developing after spinal cord injuries or corrective surgery for scoliosis.[5]

Patients typically present with symptoms consistent with a proximal small bowel obstruction. Duodenal obstruction can be either partial or complete based on the degree of narrowing of the aortomesenteric angle. Patients present with varying degrees of postprandial abdominal pain, early satiety, nausea, vomiting, acid reflux, and weight loss depending on the severity of the obstruction.[6] Given the rarity of this condition and the nonspecific presenting symptoms, patients often undergo extensive evaluation and empiric treatments prior to diagnosis. One case series reported duration of symptoms ranging from 8-28 months prior to diagnosis.[7] Patient positioning can affect the degree of obstruction in SMA syndrome. Duodenal obstruction

is often maximal in the supine position, whereas prone or left lateral decubitus positioning can increase the aortomesenteric distance and help relieve symptoms.[1]

Different imaging techniques have been used in the diagnosis of SMA syndrome. UGIS with SBFT can show gastric or gastroduodenal distention, delayed gastroduodenal emptying, or potentially a band like defect across the third portion of the duodenum due to compression by the SMA.[8] However, CT with intravascular contrast or CT angiography allow for evaluation of the aortomesenteric vasculature as well as gastroduodenal caliber and are often the preferred diagnostic imaging modalities. CT angiography can provide the most precise evaluation of the aortomesenteric angle (6-22 degrees in SMA syndrome) and aortomesenteric distance (2-8 mm in SMA syndrome).[9] There are limited data regarding the utility of EGD in diagnosis of SMA syndrome. Case reports have largely reported normal EGD findings or changes consistent with reflux esophagitis. However, in severe cases of SMA syndrome, luminal narrowing or band-like, pulsating compressions of the third portion of the duodenum may be visualized.[10]

Initial management of SMA syndrome is conservative and includes IV fluid hydration, bowel rest, nutritional support and potential gastrointestinal decompression. The goal of conservative management is weight gain to increase the mesenteric fat pad, increasing the aortomesenteric angle and relieving duodenal compression. If conservative management is not successful, surgery can be considered. Small, oral feedings are preferred, but nutritional support is often required. Options for enteral feedings include a nasojejunal feeding tube or gastrojejunostomy tube placement. Feeding tubes should be placed distal to the area of duodenal compression. If enteral feedings are not an option, TPN can be utilized. TPN nutritional composition is personalized to meet the individual patient needs.

Currently there are no clear guidelines on the optimal modality or duration of nutritional support, nor how much weight needs to be gained prior to stopping nutritional support. One retrospective case series of 26 patients with SMA syndrome reported patients received enteral nutrition for an average of 10.1 months.[11] BMI, body weight, fat mass, and skeletal mass all increased with enteral nutrition, with significant improvement in GI symptoms reported at mean follow up of 24 months. While no definitive guidelines exist, different case series suggest a trial of at least 6-12 weeks of medical/nutritional therapy before considering surgery.[12]

In patients who have either failed medical therapy, have severe clinical presentations of SMA syndrome, or have developed complications from enteral feeding or TPN may be considered for surgical management. Surgical options include a gastrojejunostomy or duodenojejunostomy with or without division of the 4th portion of the duodenum.[13,14] A recent retrospective study of eighty patients with SMA syndrome compared the success rates of medical vs surgical management. Fifty-seven patients were managed medically with a 71.3% success rate and 15.8% recurrence rate at 5-month follow up. Fourteen patients were managed surgically with a 92.9% success rate at 12 months.[15]

Clinical symptoms of SMA syndrome, including postprandial epigastric pain and early satiety, overlap with symptoms of functional dyspepsia. These groups of patients may also have overlapping psychologic conditions or restricted eating, which can make the diagnosis challenging. One recent study evaluated the overlap between functional dyspepsia and SMA syndrome and reported 11% of functional dyspepsia patients met diagnostic criteria for SMA syndrome.[16] They concluded that determining SMA syndrome in pre-diagnosed functional dyspepsia patients requires a high level of suspicion. Diagnosis and evaluation should be considered selectively in patients with low BMI that are not improving with conventional therapy for functional dyspepsia.[16] Mirtazapine could potentially be helpful in patients with overlapping functional dyspepsia and SMA syndrome with appetite stimulation and weight gain.

This clinical vignette presents a unique patient with overlapping functional dyspepsia and SMA syndrome. She was able to achieve a successful outcome with conservative management, nutritional supplementation, and use of mirtazapine.

REFERENCES

1. **Welsch T, Büchler MW, Kienle P**. Recalling superior mesenteric artery syndrome. *Dig Surg*. 2007;24(3):149-56. doi: 10.1159/000102097. Epub 2007 Apr 27. PMID: 17476104.
2. **Derrick JR, Fadhli HA**. Surgical anatomy of the superior mesenteric artery. *Am Surg*. 1965 Aug;31:545-7. PMID: 14322848.
3. **Sapkas G, O'Brien JP**. Vascular compression of the duodenum (cast syndrome) associated with the treatment of spinal deformities. A report of six cases. *Arch Orthop Trauma Surg* (1978). 1981;98(1):7-11. doi: 10.1007/BF00389703. PMID: 7235883.
4. **Agarwal T, Rockall TA, Wright AR, Gould SW**. Superior mesenteric artery syndrome in a patient with HIV. *J R Soc Med*. 2003 Jul;96(7):350-1. doi: 10.1258/jrsm.96.7.350. PMID: 12835451; PMCID: PMC539542.
5. **Chung WH, Anuar AA, Lee KJ, Hasan MS, Chiu CK, Chan CYW, Kwan MK**. Superior mesenteric artery syndrome: A rare complication of scoliosis corrective surgery. *J Orthop Surg (Hong Kong)*. 2020 Sep-Dec;28(3):2309499020945014. doi: 10.1177/2309499020945014. PMID: 32909907.
6. **Li X, Tian M, Yang H, Liu Y, Chen J, Tian H**. Superior mesenteric artery syndrome after colectomy: A case report and literature review. *Medicine (Baltimore)*. 2022 Sep 2;101(35):e30427. doi: 10.1097/MD.0000000000030427. PMID: 36107595; PMCID: PMC9439752.
7. **Merrett ND, Wilson RB, Cosman P, Biankin AV**. Superior mesenteric artery syndrome: diagnosis and treatment strategies. *J Gastrointest Surg*. 2009

Feb;13(2):287-92. doi: 10.1007/s11605-008-0695-4. Epub 2008 Sep 23. PMID: 18810558.

8. **Warncke ES, Gursahaney DL, Mascolo M, Dee E**. Superior mesenteric artery syndrome: a radiographic review. *Abdom Radiol (NY)*. 2019 Sep;44(9):3188-3194. doi: 10.1007/s00261-019-02066-4. PMID: 31127323.
9. **Sankaran L, Ramachandran R, Bala Raghu Raji V, *et al***. The role of multidetector CT angiography in characterizing vascular compression syndromes of the abdomen. *Egypt J Radiol Nucl Med*. 2019;50:55. https://doi.org/10.1186/s43055-019-0063-2.
10. **Park JH, Lee SH, Park DH, Park JY, Park SH, Chung IK, Kim SJ**. Superior mesenteric artery syndrome (with video). *Gastrointest Endosc*. 2008 Feb;67(2):348-50. doi: 10.1016/j.gie.2007.08.009. Epub 2007 Dec 26. PMID: 18155203.
11. **Wan S, Zhang L, Yang J, Gao X, Wang X**. Superior Mesenteric Artery Syndrome Improved by Enteral Nutritional Therapy: A Retrospective Case-Series Study in a Single Institution. *Ann Nutr Metab*. 2020;76(1):37-43. doi: 10.1159/000506620. Epub 2020 Mar 13. PMID: 32172254.
12. **Shin MS, Kim JY**. Optimal duration of medical treatment in superior mesenteric artery syndrome in children. *J Korean Med Sci*. 2013 Aug;28(8):1220-5. doi: 10.3346/jkms.2013.28.8.1220. Epub 2013 Jul 31. PMID: 23960451; PMCID: PMC3744712.
13. **Richardson WS, Surowiec WJ**. Laparoscopic repair of superior mesenteric artery syndrome. *Am J Surg*. 2001 Apr;181(4):377-8. doi: 10.1016/s0002-9610(01)00571-2. PMID: 11438278.
14. **Massoud WZ**. Laparoscopic management of superior mesenteric artery syndrome. *Int Surg*. 1995 Oct-Dec;80(4):322-7. PMID: 8740677.
15. **Lee TH, Lee JS, Jo Y, Park KS, Cheon JH, Kim YS, Jang JY, Kang YW**. Superior mesenteric artery syndrome: where do we stand today? *J Gastrointest Surg*. 2012 Dec;16(12):2203-11. doi: 10.1007/s11605-012-2049-5. Epub 2012 Oct 18. PMID: 23076975.
16. **Kawanishi K, Shojima K, Nishimoto M, Abe H, Kakimoto T, Yasuda Y, Hara T, Kato J**. Superior Mesenteric Artery Syndrome May Be Overlooked in Women with Functional Dyspepsia. *Intern Med*. 2017 Oct 1;56(19):2549-2554. doi: 10.2169/internalmedicine.8647-16. Epub 2017 Sep 6. PMID: 28883239; PMCID: PMC5658518.