CLINICAL VIGNETTE

Superior Mesenteric Artery (SMA) Syndrome in a Patient with Gunshot Wound to Head

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SMA syndrome is an uncommon but well recognized disorder that can be a clinical enigma. It is especially difficult to recognize in trauma victims.[1] SMA is caused by compression of the 3rd portion duodenum, between the aorta and the SMA. Trauma patients who requires prolong hospitalization may develop significant weight loss and SMA syndrome can be a potential issue that needs to be recognized and treated.

Case Presentation

A 24-year-old male was brought to the emergency room after a self-inflicted gunshot wound to the head. He placed a gun to his chin and shot himself. The exit wound was his mid forehead. Patient was alert and awake in the emergency room. CT scan head showed soft tissue swelling and extensive bullet fragments in the frontal brain soft tissue. Pt was taken to surgery and had a craniotomy with debridement of necrotic brain tissue. A left frontal lobectomy was performed and cranioplasty was done. ENT reconstructed his skull base. Shortly after surgery, a tracheostomy was done. Pt was post-operative day three when a gastroenterology consultation was called to evaluate for PEG (percutaneous endoscopic gastrostomy) placement for nutritional support.

His past medical history was only remarkable for childhood asthma. There was no history of anxiety, depression, or other psychiatric disorder.

Physical exam revealed a thin male with BP 106/49 P 90 R 15 T 99.4. Weight 120.5 pounds with BMI 17.8. HEENT: dressing from craniotomy, bilateral orbital ecchymosis, closed eyes, oral clear, neck supple. Heart: regular rate and rhythm. Lungs clear. Abdomen: soft, nontender, with no hepatosplenomegaly. Extremity: no edema. Neurologic exam shows patient extremely lethargic with difficulty answering simple questions.

After craniotomy, his hospital course was prolonged and stormy. He had persistent fevers treated with IV antibiotics. On post op day 4, a PEG was placed and he was started on tube feedings. Thirteen days after PEG placement, he had increased G tube residuals. Abdominal series showed non-specific bowel gas pattern and Gastrograffin SB series showed marked gastric and duodenal distention with collapse of distal loops of small bowel. The gastrograffin contrast flowed through the small bowel with normal transit time. CT abdominal/pelvis revealed aortomesenteric narrowing of at the level of 3rd portion duodenum and a narrow aortomesenteric angle. These findings were consistent with SMA syndrome.

His jaw was wired after reconstruction preventing converting PEG to percutaneous endoscopic jejunostomy (PEJ) by esophagogastroduodenoscopy (EGD). Interventional radiology converted his PEG to a gastrojejunostomy and he was able to tolerate tube feeding at 70 cc per hour via his gastrojejunostomy tube.

After one month acute hospitalized care, he was transferred to the acute rehabilitation unit (ARU). On admission to ARU, patient's weight was 87 pounds, a loss of 33.5 pounds since acute care admission with BMI 12.9. Mental status on admission to ARU showed significant improvement from care. He was alert and oriented and was able carry on brief conversations. He was switched to clear liquids and one week later, placed on soft diet. He was discharged after three weeks in the ARU. He was tolerating a soft diet up to 50% with discharge weight of 91 pounds with BMI 13.4.

Discussion

SMA syndrome is a rare disorder that needs to be clinically recognized and treated. Other names for SMA syndrome includes Cast syndrome (from developing the syndrome after being in a full body cast), Wilkie syndrome, and arteriomesenteric duodenal obstruction. Von Rokitansky first described SMA syndrome in 1842 as compression of duodenum between the aorta and SMA resulting in intermittent vomiting.[2]

The 3rd portion of the duodenum is sandwiched between the superior mesenteric artery and the aorta. A mesenteric fat pad prevents the SMA from collapsing onto the aorta. The fat pad elevates the SMA root off the aorta. The normal angle between the SMA and aorta (aortomesenteric angle) is 45º (range of 38 and 60º) and on cross-sectional imaging, the distance between the aorta and SMA (aortomesenteric distance) is 10 to 20 mm. With severe weight loss, there is decrease in the mesenteric fat pad and the aortomesenteric angle can shrink to 6-25º and the aortomesenteric distance reduced to 2-8 mm, both of these can result in SMA syndrome.[3] Ozkurt H, et al has shown a strong correlation between BMI and the aortomesenteric angle, and between BMI and the aortomesenteric distance.[4]

Those at risk for developing SMA syndrome commonly have severe illness with profound weight loss. These conditions

include malignancy, major gastrointestinal surgery, traumatic brain injury (as in our report), surgical treatment for obesity, anorexia nervosa, chronic malabsorption, and major trauma.[1] Corrective surgery for scoliosis in which the spine is straightened alters and narrows the aortomesenteric angle and increases the risk for SMA syndrome.[5] Other factors that can also contribute to SMA syndrome are those with congenital anomalies including acute aortomesenteric angle, high fixation of the ligament of Treitz, low origin of SMA, and lumbar lordosis.[6]

Diagnosing SMA syndrome is frequently delayed and requires a high index of suspicion. Often the diagnosis is made after an exhaustive process of exclusion. Females and young adults (18-35 years) are more likely to be susceptible to the condition.[7] Classic symptoms includes postprandial epigastric pain associated with nausea and vomiting, abdominal bloating, and weight loss. SMA syndrome may also present with progressive mild to moderate symptoms over years or decades.[1] Lying in a prone or at left lateral position may help with symptom relief.[8] These positions most likely temporarily reduces the pressure due to gravity of the SMA off the duodenum.

Plain abdominal films show marked gastric dilation. Small bowel series show various findings including dilation of 1st and 2nd portion of duodenum, abrupt vertical or oblique compression of the 3rd portion of duodenum, anti-peristaltic waves of barium proximal to obstruction, significant delay in gastroduodenal transit, and relief of obstruction with postural changes.[1,9] CT abdomen support the small bowel series findings but will also allow for measuring the aortomesenteric angle and aortomesenteric distance. EUS can also be helpful measuring the aortomesenteric distance and seeing an absence or decrease in mesenteric fat pad.[9]

SMA syndrome needs to be differentiated from other causes of megaduodenum including diabetes mellitus, systemic lupus erythematosus, scleroderma, amyloidosis, or chronic idiopathic intestinal pseudo-obstruction.[10] In addition, other causes of postprandial epigastric pain with nausea and vomiting can be excluded by EGD including GERD, peptic ulcer disease, gastric or duodenal malignancy. Also, a gastric emptying study may be needed to rule out gastroparesis.

Since weight loss is the most common cause of SMA syndrome, a trial of conservative management for nutritional support is the best initial approach. Nasogastric tube for decompression of dilated stomach and proximal duodenum should help improve patient's symptoms. Ideally, a jejunostomy feeding tube can be placed into or beyond the fourth portion of duodenum either endoscopically or radiologically. This would bypass the obstruction and give patients enteral feedings to build up their mesenteric fat pad. However, enteral feeding may not be possible and patients may need to receive total parenteral nutrition.

Patients that fail conservative management may need surgical intervention. Duodenojejunostomy for SMA syndrome bypasses the 3rd portion of the duodenum. The procedure was first described by Stavely in 1908[7] and is still regarded the surgery that has the most favorable outcome. The Strong procedure involves moving the duodenum by dividing the ligament of Treitz. This procedure maintains bowel integrity but there is a 25% failure rate due to short branches of the inferior pancreaticoduodenal artery not permitting the duodenum to fall inferiorly away from the aorta.[7] Gastrojejunostomy brings up a loop of jejunum to the stomach for a side to side anastomosis.

In summary, SMA syndrome is a rare condition that an astute clinician needs to recognize and treat. Our young patient had drastic weight loss which decreased the mesenteric fat pad. This caused narrowing of the aortomesenteric angle and shortening the aortomesenteric distance resulting in severe obstruction of the 3rd portion of duodenum. He was treated conservatively and fed through a jejunostomy tube. With weight gain in his mesenteric fat pad increased and his duodenal bowel obstructive symptoms improved allowing him to tolerate an oral diet.

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