# SUPERIOR MESENTERIC ARTERY SYNDROME

RESEARCH AWARENESS AND SUPPORT

## What is SMAS?

SMAS or Superior Mesenteric Artery Syndrome is the obstruction of the 3rd portion of the duodenum due to compression by the Superior Mesenteric Artery (SMA ) towards the Abdominal Aorta ( AA ) reducing the angle between these two structures to 6 – 22° ( normal 28 – 65° ) hence decreasing the aortomesenteric distance to 2 – 8 mm ( normal 10 – 34mm ). The obstruction can be intermittent, partial or complete. The etiology is not well understood but it can be due to congenital predisposition or acquired or both. The location of the Ligament of Treitz can affect angulation of the SMA and AA. Loss of mesenteric fat from weight loss will decrease the angle between the SMA and AA. Elongated mesentery can cause a high aortomesenteric angular torque hence the “pendulum effect” can be a factor. During adolescence a rapid growth spurt in height without concomitant increase in abdominal girth can be a factor. Rapid weight loss from any cause can also be a factor. The most commonly mentioned incidence ranges from 0.013% to 0.3% worldwide which translate to 41,000 to 96,000 U.S. Citizens. From a personal observation, it appears that the incidence is higher than is written in the literature.

The symptoms include postprandial epigastric pain, nausea, bloating, early satiety and “food fear”. Intermittent bilious vomiting can occur. The result of chronic duodenal obstruction is enlargement if the duodenum above the blockage and enlarged stomach ( gastromegaly ) with delayed emptying of the stomach or gastroparesis ( paralysis of the stomach ). Constipation can also occur due to delay or inability to empty the stomach or with opioid use. There also reverse peristalsis ( to and fro motion ) of the proximal duodenum. Weight loss follows which aggravates the problem due to loss of the mesenteric fat decreasing the distance between the SMA and AA. Symptoms can be relieved by knee chest position or right lateral decubitus ( right side down ) to allow gravitational emptying of the stomach.

Physical examinations include an asthenic built, frail with muscle wasting and a scaphoid abdomen. There is a bounding pulse ( “second heart “ ) and palpable thrill and a bruit over the SMA.

The most definitive diagnostic study is a CT Angiogram ( CTA ) because it can measure the angle between the SMA and AA ( normal is 28 to 65* ) and the distance between the SMA and AA ( normal is 10 to 34mm ). An MRI can also be used with the same result. A doppler ultrasound can determine blood flow variations but is not diagnostic. Contrast study ( UGIS )

which was employed in the past can show the obstruction at the 3rd portion of the duodenum with a filling defect by the SMA. One can also observe a reverse peristalsis ( to and fro motion ) of the contrast at the proximal duodenum. Other studies needed include gastric emptying study to rule out gastroparesis. CCK-HIDA ( Cholecystokinin – Hepatobiliary iminodiacetic acid scan ) is done to rule out gallbladder dysfunction ( biliary dyskinesia ). MALS ( Median Arcuate Ligament Syndrome ) also known as " Coeliac Steal Syndrome" ) must be ruled out because it could be associated with SMAS or could be causing the pain. If there is left flank pain, pelvic pain and hematuria, Nutcracker Syndrome must be ruled out by CTA and Doppler ultrasound. Management is based upon etiology. Primary is probably a congenital predisposition with decreased angle between the SMA and AA with decreased distance between the 2 structures causing the obstruction. Secondary is rapid weight loss from other causes hence loss of mesenteric fat pad which in turn narrows the angle and the distance between the SMA and AA. Trial of conservative management of frequent small feedings, use of supplemental predigested formulas followed by the knee chest posture or right lateral decubitus posture when symptoms arise. Naso-jejunal feedings can also be used. If no response after 6 weeks surgical therapy is recommended. Secondary is when there is a rapid weight loss from any cause with loss of mesenteric fat can be treated with frequent small feedings, naso-jejunal feedings with or without Total Parenteral Nutrition ( TPN ). Treatment of the underlying illness causing the illness is paramount. These patients should do well with conservative management hence surgical intervention should be reserved for rare cases. The need for team approach to therapy is needed. Gastroenterologist, nutritionist, pain therapist, psychologist or psychiatrist and a social worker should be part of the team. A surgeon dedicated to the care of SMAS patients is needed when surgical therapy becomes necessary. Team approach to follow up following any treatment is a must.

Review of the literature yielded the following :

- 5
  Gastro – jejunostomy : This procedure can relieve some symptoms temporarily but does not relieve the SMA compression, pain persist , blind loop syndrome can occur as well as bile gastritis and potential for anastomotic ulcers.

- . Duodenal transposition anterior to the SMA does not relieve the SMA compression hence persistence of pain,

- SMA transposition below the renal vein or below the duodenum is a complex vascular procedure only done in Germany.

- Duodeno-jejunostomy : most popular procedure and relieve most of the symptoms in 80 to 90% according to the literature. The SMA compression persist hence pain may persist or worse in some.

- Duodenal Derotation with our without doudeno-jejunostomy at the site of the ligament of Treitz. This procedure will completely relieve the compression of the SMA and relief of all symptoms.

What is the appropriate name of this disease? Wilkie's Syndrome , Cast syndrome, Aortomesenteric Artery Compression, Arteriomesenteric Artery Duodenal Compression, Duodenal Vascular Compression, Superior Mesenteric Artery Syndrome, Superior Mesenteric Artery Compression Disorders or Superior Mesenteric Artery Compression Syndrome (SMACS). The SMACS best describe the disease.

Domingo T. Alvear, M.D.,FACS,FICS