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# SUPERIOR MESENTERIC ARTERY SYNDROME: A CASE REPORT FROM A MEDICAL COLLEGE IN EASTERN INDIA

Surgery

**Sramana Palit** Intern, Department of Surgery, ESIC-PGIMSR, Joka, Kolkata, West Bengal, India

**Subhadip Sarkar*** Associate Professor, Department of Surgery, ESIC-PGIMSR, Joka, Kolkata, West Bengal, India *Corresponding Author

## ABSTRACT

Superior mesenteric artery (SMA) syndrome, also known as Wilkie's syndrome, is an uncommon condition which is caused by compression of the third part of duodenum between aorta and superior mesenteric artery. It occurs due to narrowing of the aorto-mesenteric angle (less than 15 degree). There are many causative factors like rapid weight loss, external compression by spica casts, anatomical anomalies & post-spinal surgeries. We report a case of SMA syndrome in a 21 yrs old male, presented with history of intermittent upper abdominal pain, recurrent post-prandial bilious vomiting and significant loss of weight for the last 6 months. He underwent series of invasive and non-invasive investigations to come to a proper diagnosis. Initially conservative management was tried but failed. After that, he underwent duodenojejunostomy and finally got cured. Our case emphasizes the challenges faced in diagnosing of SMA syndrome and the need of increased awareness among health care personnel regarding this clinical entity.

## KEYWORDS

SMA syndrome, Wilkie's syndrome

## INTRODUCTION

Superior mesenteric artery syndrome (SMA) syndrome is a rare clinical entity of proximal small bowel obstruction. It is also known as Wilkie's syndrome. The prevalence rate is reported as 0.1-0.3% with just around 400 cases reported in the literature [1, 2]. It is mostly seen in adolescents and young adults within age group 10-39 yrs but it can occur in any age. The female: male ratio is found to be 3:2. However, no ethnic predisposition has been described in literatures [3, 4, 5]. It is also described as cast syndrome, arterio-mesenteric duodenal obstruction & chronic duodenal ileus.

The third part of duodenum is positioned within an angle of approximately 45 degrees formed by the root of superior mesenteric artery and aorta. When this angle is narrowed down to less than 25 degrees, the superior mesenteric artery impinges on the duodenum, thereby leading to duodenal obstruction. Patients mostly presents with the symptoms of intermittent upper abdominal pain, recurrent post-prandial bilious vomiting and significant loss of weight.

A delay in the diagnosis can potentially lead to many complications like dyselectrolytemia, malnutrition, gastric perforation and peritonitis. The aim of conservative management is to achieve weight gain, either orally or parenterally, with a view to restituting the mesenteric pad of fat and subsequent increase in the aortomesenteric angle [6].

If the non-surgical approach becomes unsuccessful, surgical management is the only option and duodenojejunostomy being the treatment of choice [7].

However, advancement in imaging studies such as computed tomography (CT) and magnetic resonance imaging (MRI) have been extremely helpful to visualise the angle between the aorta and the SMA and thus the diagnostic rate has also been improved [8].

### Case Report

We present here a case of 21 yrs old male who presented to the emergency with a history of upper abdominal pain for the last 5 months. The pain was diffuse, constant, non-radiating and squeezing in type, unrelated to any food intake, partially relieved by medications. The pain was associated with non-projectile, non- bilious vomiting, containing food particles.

The frequency of the vomiting was around 4-5 episodes/ day. There was also an associated history of significant loss of appetite and loss of weight. There was no history suggestive of fever, loose motion or jaundice. No medical comorbidities were present.

On examination, he was cachectic with a BMI of 14.3 kg/m2. His vital parameters were stable. Pallor was present .On palpation, diffuse abdominal tenderness with upper abdominal fullness were noted. Other systemic examinations were within normal limits.

### Investigations:-

Hb- 8.3gm%
Total protein- 5.1 g/dl
Albumin - 2.8 g/dl
RFT- WNL
Sodium:- 137
Potassium - 3.2
Chest x- ray (PA) and ECG (all leads) were normal. Ultrasound of the whole abdomen didn't conclude any significant findings.

In view of persistent vomiting, we did an upper GI endoscopy which showed erosive antral gastritis with narrowing at D1-D2 junction **(Figure 1).**

We did CECT (WHOLE ABDOMEN), which revealed a hugely distended stomach and duodenum up to D2. Significant narrowing also noted at D3, which appears to be compressed between SMA and aorta. **(Figure 2 & 3).**

Once the diagnosis is confirmed, we have started conservative management with IV fluid, nasogastric tube placement, total parenteral nutrition. However, no clinical improvement was noted even after conservative management. Then we decided to proceed with side to side duodenojejunostomy.

Post-operative recovery was uneventful. On day 3, intestinal peristalsis returned fully and we started allowing him orally. On day 8, we discharged the patient. Follow up was done after 1 month, 3 months and 6 months interval. He was absolutely symptom free during follow up visits. Moreover he gained 5 kg weight after 6 months from surgery.

## DISCUSSION

SMA syndrome was first described by the Austrian professor, Carl Freiherr von Rokitansky in 1861, as an autopsy finding [8]. Later, Wilkie provided more detailed clinical and pathophysiological descriptions in a series of 64 patients and suggested treatment approaches [9].

However, during initial days, there were many controversies regarding the actual existence of the syndrome because, all the symptoms were nonspecific and there were many other differential diagnoses.

- Anorexia nervosa
- Chronic idiopathic intestinal pseudo obstruction
- Collagen vascular diseases
- Diabetes mellitus
- Mega duodenum
- Peptic ulcer disease

Duodenal compression is usually due to the loss of mesenteric fat pad

between the aorta and the SMA, resulting in narrowing of the angle between those two. The fat pad cushion helps to hold the SMA off the spine and protect it from duodenal compression.

The normal aortomesenteric angle is 38 to 65 degrees. However, the angle less than 25 degrees causes compression to the third part of duodenum. Decrease in the aortomesenteric angle can be either congenital or acquired. SMA syndrome is mostly associated with significant weight loss, including hypermetabolic conditions like trauma and burns, dietary conditions like anorexia nervosa, malabsorbtive diseases, and cachexia causing conditions like AIDS, advanced malignancy.

There are few other risk factors also like surgical correction of scoliosis, congenitally short ligament of treitz, duodenal malrotation, Ladd's band, abdominal aortic aneurysm, lumbar hyperlordosis and mesenteric root neoplasm.

Patients with SMA syndrome may present with either acute Symptoms or with acute exacerbation of chronic symptoms. Acute presentation is usually characterized by signs and symptoms of duodenal obstruction.

However, the chronic cases may present with recurrent episodes of abdominal pain with vomiting, cachexia, sense of fullness after meal with early satiety due to increased gastroduodenal transit time and gastric distension [10, 11, 12].

Diagnosis is mainly done on the basis of clinical findings along with imaging studies. There are various imaging studies which can be used, for example, barium meal x-ray study of stomach & duodenum, CECT (W/A), Magnetic resonance angiography (MRA).

Upper GI endoscopy is very much useful to detect the complications of the disease including gastric stasis, reflux biliary gastritis and duodenal ulcers and also to rule out any intraluminal cause of duodenal obstruction. CECT (W/A) allows accurate measurement of aortomesenteric angle which helps in confirmation of SMA syndrome and this has replaced MRA as the gold standard diagnostic modality.

In a study which reviewed 8 cases of SMA syndrome, a reported aortomesenteric angle cut off of 22 degrees revealed a 42.8% sensitivity and 100% specificity and a distance of 8 mm was both 100% sensitive and specific [13].

Laboratory investigations like serum electrolytes, serum protein and albumin level are usually supportive in case of cachexia. Though it is rare, it is very much important to consider SMA syndrome as diagnosis because delay in diagnosis may result in significant morbidity and mortality from malnutrition, dehydration, dyselectrolytemia, GI haemorrhage and gastric perforation [14, 15, 16]

Traditionally, the treatment includes conservative measures such as gastric decompression via nasogastric tube, parenteral nutrition/ nasojejunal feeding (wherever possible), followed by oral feeding as tolerated [6]. Motility agents may be helpful in some patients. Surgery is indicated if conservative management fails [7].

Duod enojejunostomy is the surgical treatment of choice, with a success rate of approximately 90% [7]. Another surgical option, known as String's procedure, involves detachment of the ligament of treitz with mobilisation of the duodenum. However, this surgery has a failure rate of around 25% [11].

Gastrojejunostomy, which was the preferred surgery of choice in initial days, has been abandoned because of increased post-operative complications like blind loop syndrome and recurrent vomiting due to non-decompression of dilated duodenum.

## CONCLUSION

From our case study, we can conclude that SMA syndrome is under diagnosed. We have to show high clinical suspicion, especially in patients with severe cachexia and symptoms of gastroduodenal obstruction.

Computed tomography angiography is considered as the gold standard diagnostic modality. In case of unsuccessful conservative management, Duodenojejunostomy is the surgical treatment of choice.

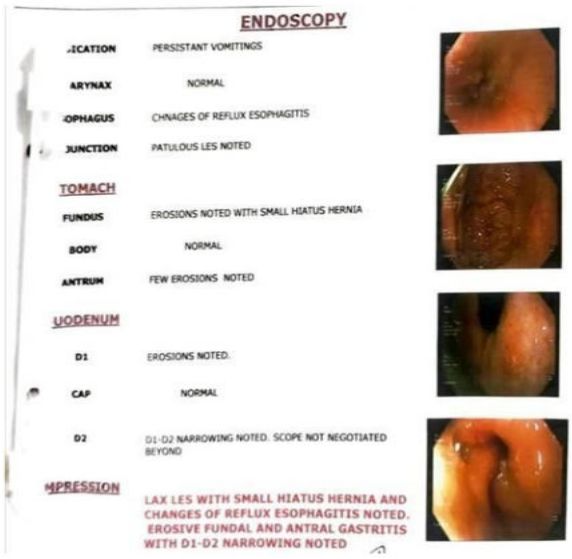


**Figure 1:- Upper GI endoscopy report showing narrowing at D1-D2.**

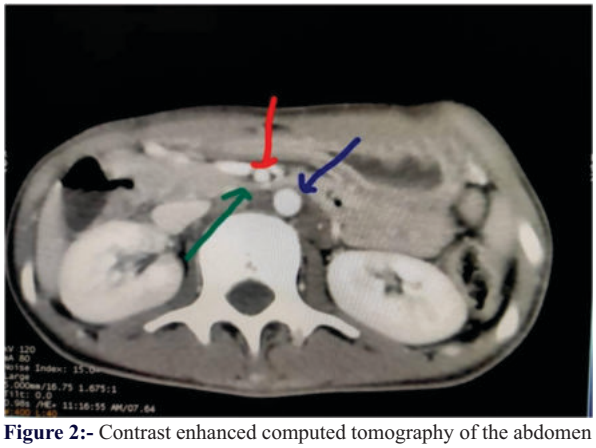

**Figure 2:-** Contrast enhanced computed tomography of the abdomen in venous phase showing compression of third part of the duodenum between superior mesentery artery and aorta. Blue arrow- aorta, red arrow – superior mesenteric artery, Green arrow – 3rd part of duodenum

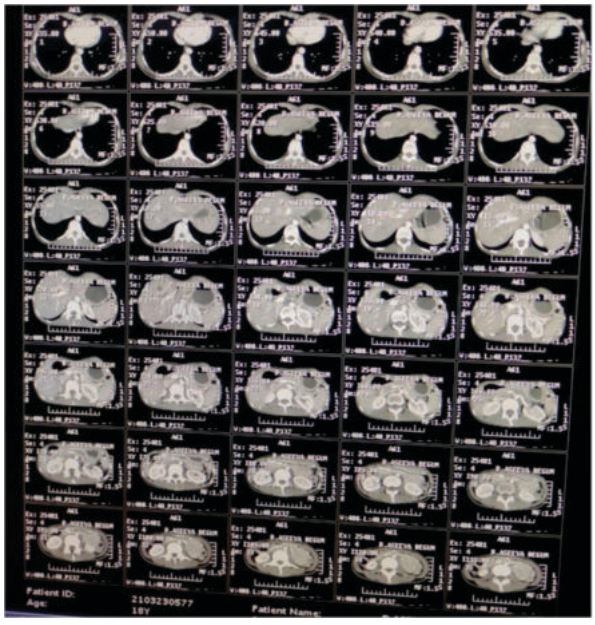

**Figure 3:-** Contrast enhanced computed tomography of the abdomen revealed compression of third part of the duodenum between superior mesentery artery and aorta.

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