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# Superior Mesenteric Artery Syndrome: A Case Study

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**Abstract** | **Case Report**

Superior mesenteric artery (SMA) syndrome (known as Wilkie's syndrome) is a rare cause of upper gastrointestinal obstruction. It is an acquired disorder in which acute angulation of the SMA causes compression of the third part of the duodenum between the SMA and the aorta. This is commonly due to loss of fatty tissue as a result of a variety of debilitating conditions. We report a 25-year-old male who presented with intermittent abdominal pain and intractable vomiting following significant weight loss, he developed acute high intestinal obstruction, which was diagnosis by a CT scan. This case emphasizes the challenges in the diagnosis of SMA syndrome and the need for increased awareness of this entity. This will improve early recognition in order to reduce irrelevant tests and unnecessary treatments.

**Keywords:** Wilkie's syndrome, Superior mesenteric artery syndrome, Intestinal obstruction.



## INTRODUCTION

Superior mesenteric artery (SMA) syndrome (known as Wilkie's syndrome) is defined by extrinsic compression of the third duodenal segment between the superior mesenteric artery and the aorto-spinal plane. It was first described by Von Rokitansky in 1861. Long controversial, today this pathology is well recognized.

This is commonly due to loss of fatty tissue as a result of a variety of debilitating conditions.

## OBSERVATION

25-year-old patient, with a history of epigastralgia, admitted to the emergency room for a high occlusive syndrome with notion of vomiting and an important weight loss.

Biology: Hydro-electrolyte disorder with hypokalaemia.

Esogastroduodenal fibroscopy revealed a stasis stomach with diffuse duodenal dilation extending to the 3rd duodenum (D3).

Abdominal CT scan: Gastric and duodenal distension secondary to compression of the 3rd duodenum by an anatomical aortomesenteric syndrome with reduced aorto-mesenteric distance measured at 3.6mm and lower aorto-mesenteric angle calculated at 17 °, without sign of digestive distress (Fig 1 & 2).

The patient underwent a gastroenteroanastomosis after failure of medical treatment. The operative consequences were simple, and the evolution was marked by the disappearance of the symptoms and no recurrence was noted after a 9-month follow-up.

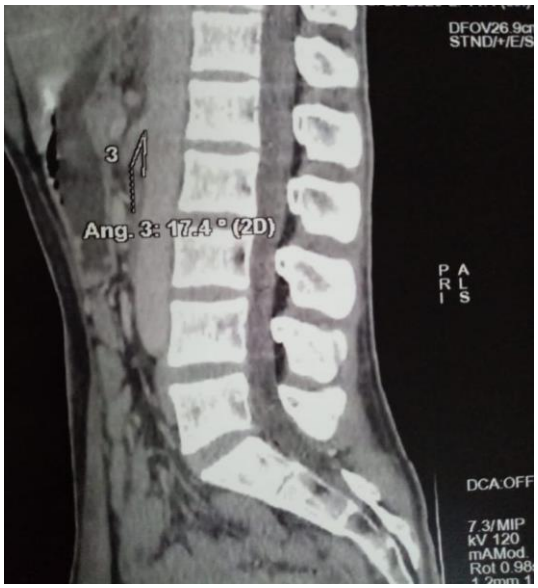


**Fig-1: Sagittal CT image showing the entrapment of the duodenum between the aorta and the SMA with an aorta-SMA angle of 17,4°**

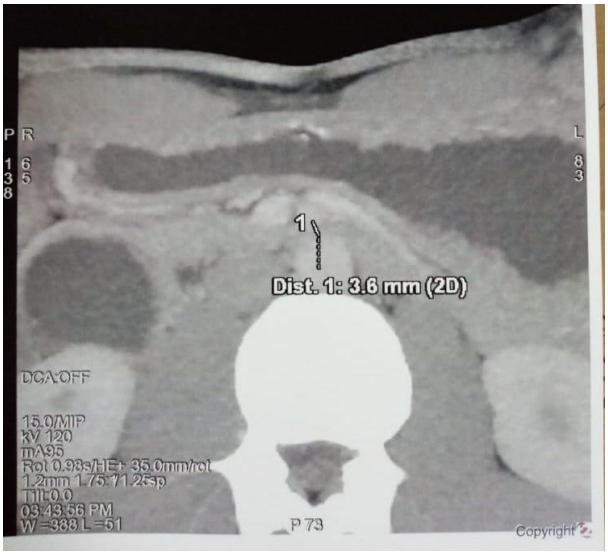


**Fig-2: CT scan showing the SMA and the aorta separated by a small distance of 3.6 mm and duodenal distension**

## DISCUSSION

SMA syndrome is a rare pathology with an incidence that ranges between 0.013 and 0.3% [1]. The defining feature of this entity is upper gastrointestinal obstruction caused by compression of the third part of the duodenum between the SMA anteriorly and the aorta posteriorly [2]. In humans, the aorta-SMA angle ranges from 38 to 65°, due to the erect posture, while in quadrupeds, it is nearly a right angle [3]. The main anatomic feature of SMA syndrome is a narrowing of the aorta-SMA angle to <25°, and as a result, the aortomesenteric distance decreases to <10 mm, from normally 10 to 28 mm [2].

Etiological factors can be either a congenital or an acquired anatomic abnormality or, more commonly, a debilitating condition causing severe weight loss. Congenital etiologies include abnormally low insertion of the SMA or high insertion of the angle of Treitz dislocating the duodenum to a cranial position. Acquired anatomic abnormalities can occur following corrective spinal surgery such as scoliosis surgery by a relative lengthening of the spine [4], spinal trauma, and after abdominal surgery such as total proctocolectomy and ileal J-pouch anal anastomosis due to tension and caudal pull of the small bowel mesentery [5].

Patients with SMA syndrome may present acutely, with chronic insidious symptomatology, or with an acute exacerbation of chronic symptoms. Acute presentation is usually characterized by signs and symptoms of duodenal obstruction which is the cas of our patient. Chronic cases like our patient may present with long-standing vague abdominal symptoms or recurrent episodes of abdominal pain, associated with vomiting. Other less common symptoms are esophageal reflux, early satiety with a sensation of fullness owing to increased gastroduodenal transit time, and gastric distension [6, 2, 3, 4, 7].

The diagnosis of the SMA syndrome is challenging and often delayed due to its insidious presentation. High clinical suspicion is warranted and diagnosis is based on clinical evidence supported by radiological findings. Barium radiography demonstrates dilatation of the flrst and second part of the duodenum with or without gastric dilatation, anti-peristaltic flow of barium proximal to the obstruction and a delay of 4–6 h in gastroduodenojejunal transit time, with relief of obstruction when the patient is placed in the prone, knee-chest or left lateral position [1, 6]. Contrast-enhanced CT or magnetic resonance angiography enable visualization of the vascular compression of the duodenum and precise measurement of the aortomesenteric angle and distance. Endoscopic examination may visualize a pulsatile extrinsic compression suggestive of this condition [8].

Traditionally treatment has consisted of conservative measures such as gastric decompression, parenteral nutrition and/or post-pyloric feeding when possible, followed by oral diet as tolerated [1]. Posturing maneuvers during meals and motility agents may be helpful in some patients. No time limit has yet been defined for the medical treatment. Surgery may be considered if conservative treatment fails [6]. Duodenojejunostomy is the operation of choice to relieve the obstruction, with a success rate up to 90% [6]. Another less invasive surgical option, known as Strong's procedure, involves lysis of the ligament of Treitz with mobilization of the duodenum with a gastroenteroanastomosis

## CONCLUSION

We feel that SMA syndrome is underdiagnosed. High clinical suspicion is of utmost importance, especially in patients with severe weight loss and symptoms of gastric distension. Surgical stress should as well be considered as a trigger factor of SMA syndrome. Interdisciplinary teamwork provides the most beneficial diagnostic and therapeutic result in this often underestimated disease. Lastly, we would like to point out the difficulty of achieving an accurate diagnosis of SMA syndrome in an atypical clinical setting similar to what happened in our case. Heightened awareness is advised for early recognition to avoid unnecessary suffering for the patient.

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