

## Case Report

# Superior Mesenteric Artery Syndrome: Need for an Awareness

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## I N F O

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## A B S T R A C T

Superior Mesenteric Artery (SMA) syndrome is an upper gastrointestinal disorder caused by the compression of the third part of the duodenum between Aorta and Superior mesenteric artery. SMA syndrome is a rare pathology with an incidence that ranges between 0.013 and 0.3%. It's a rare cause of duodenal obstruction which may be complete or partial but is a life-threatening disorder as it poses a diagnostic dilemma. A patient was admitted in the emergency with complaints of subacute intestinal obstruction along with peritonitis. He underwent exploratory laparotomy along with surgical resection of gangrenous segment. Histopathological examination showed gangrenous bowel, however, no specific cause could be identified. CT findings were reviewed for a possible compression/ obstruction aetiology. It revealed features suggestive of superior mesenteric artery syndrome.

**Keywords:** Gastrointestinal Disorder, Superior Mesenteric Artery Syndrome, Superior Mesenteric Artery

**Introduction**

Third part or transverse part of the duodenum is in fixed compartment bounded anteriorly by the root of mesentery and Superior mesenteric artery and posteriorly by the Aorta and lumbar spine. This third part of the duodenum very rarely gets compressed between aorta and superior mesenteric artery.<sup>1</sup> It manifests as chronic upper abdominal symptoms such as epigastric pain, nausea, eructation, voluminous vomiting, post prandial discomfort, early satiety and sometimes subacute small bowel obstruction and is called superior mesenteric artery syndrome. It's a rare cause of duodenal obstruction which may be complete or partial but is a life-threatening disorder and is difficult to diagnose and is often missed.<sup>1</sup> Loss of the fat pad between the superior mesenteric artery and aorta, reduces the angle between the two vessels to less than 20 degree causing compression of the duodenum.<sup>3</sup>

**Case Report**

A 20 years young male got admitted to surgery emergency with complaints of abdominal pain, epigastric fullness, nausea and repeated vomiting for 8 days. He had not been passing stool and flatus since 4 days.

His vitals were normal. His routine blood and urine investigations were within normal limit. Ultrasound showed markedly distended bowel loops. He had no other medical and surgical finding.

Per abdominal examination revealed epigastric fullness. There was marked tenderness over all quadrants of the abdomen along with guarding, rigidity and rebound tenderness. Bowel sounds were absent. A clinical diagnosis of subacute intestinal obstruction along with perforation peritonitis was made. A CT scan showed duodenal distension. After several bouts of failed conservative treatment patient and his family gave consent for surgery.

A specimen consisting of part of duodenum, small intestine, along with caecum, appendix and part of ascending colon, altogether measuring 250 cm, was received in the department of pathology. Grossly most of the intestine showed gangrenous changes, with blackish discoloration and foul smell. The proximal part just before the gangrene showed dilation. A narrow-compressed area at the junction of gangrene and dilated part (in the duodenum) was seen in the anterior part measuring 0.5 cm in width, however the wall was not thickened, ruling out any stricture. The resected ends appeared partially viable. The intestinal wall was thinned out and the mucosa sloughed off throughout the gangrenous area. Lumen showed blood clots. The mesentery, on cut, showed congestion. 15 lymph nodes were identified varying from 0.4 cm to 1 cm in diameter.

On microscopic examination, the gangrenous areas showed complete necrosis along with severe acute inflammation. The compressed area showed acute on chronic inflammation only. The resected ends showed acute on chronic inflammation but were viable. Sections from mesentery showed marked congestion and inflammation. All the lymph nodes showed reactive hyperplasia.

A diagnosis of gangrene intestine and ileocecal region, was entertained but pathologically no definite cause of the compression could be found. It was when we went back to the patient's clinical records, and checked for any preoperative radiological cause of compression. We found a close proximity of superior mesenteric vessels and aorta. The angle between the two vessels were measured and found to be less than 20 degree with evidence of compression of the transverse duodenum. Thus, a final diagnosis of superior mesenteric artery syndrome was made.



**Figure 1.** Barium follow through showing compression of small bowel

## Discussion

The normal distance between superior mesenteric artery and Aorta is 8-12 mm, when the distance between these vessels reduces there is loss of obliquity that result in compression of the third part of duodenum as it runs between these two vessels.<sup>3</sup> The diagnosis is confirmed by the loss of the angle below 20 degree between the Superior mesenteric artery and Aorta (normally 38-65 degree).<sup>2</sup> In many previous reports many other reasons have been suggested such as a low congenital low origin of Superior mesenteric artery, high insertion of duodenum at the ligament of Treitz, compression of duodenum caused by peritoneal adhesion and after duodenal malformation. Out of all theory the compression by superior mesenteric artery is not only rare but controversial, as many authors have expressed their doubts and attributed its clinical features to other underlying causes like inflammation, neoplasia and electrolyte imbalance none of these were found in our case.<sup>4</sup> Conventional radiology/ ultrasound cannot identify the compression. With the advent of newer radiological techniques, that imaging studies provided more conclusive evidence of superior mesenteric artery syndrome.<sup>5</sup>

## Conclusion

The Superior mesenteric artery syndrome can only be diagnosed based on clinical symptoms and obstructive evidence by Barium meal follow through X-ray studies and CT scan. Conservative treatment was failed so surgery was performed which is the only accepted way of managing superior mesenteric artery syndrome. Patient responded very well to surgery.

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