Superior mesenteric artery syndrome as a postoperative complication following splenectomy: A case report and review

A 24-year-old male was brought to emergency department with blunt trauma abdomen. Ultrasound of the abdomen showed gross hemoperitoneum and splenic injury. Emergency laparotomy was done in view of hemodynamic instability and splenectomy was performed. Postoperatively patient had persistent vomiting. Upper gastrointestinal endoscopy revealed external compression of the third part of duodenum—diagnostic of superior mesenteric artery syndrome. Duodenojejunostomy was performed due to failure of conservative management. Postoperative period was uneventful and is on regular follow up.

Key words: Superior mesenteric artery syndrome, splenectomy

INTRODUCTION
Superior mesenteric artery (SMA) syndrome is a rare cause of upper gastrointestinal tract obstruction caused by compression of the third part of the duodenum by SMA. Here we report a case who presented with SMA syndrome as a postoperative complication following splenectomy.

CASE REPORT
A 24-year-old male presented to emergency department with blunt trauma to abdomen following an accident. On admission the patient was in hemodynamic shock. Ultrasound of the abdomen showed gross hemoperitoneum with splenic injury. In view of hemodynamic instability, the patient was taken up for emergency laparotomy and splenectomy was performed, no other solid organ or bowel injuries were identified. Post-operatively the patient had prolonged ileus and oral feeds were started on sixth post-operative day. The patient developed persistent vomiting and on examination had dilated stomach.

Upper gastrointestinal endoscopy revealed external compression at level of third part of duodenum. Contrast-enhanced computer tomography showed dilated stomach and duodenum with non-passage of oral contrast beyond duodenum. There was acute angulation of superior mesenteric artery (SMA) from aorta causing external compression of duodenum [Figure 1 and 2]. The patient was diagnosed to have acute SMA syndrome.

Initially the patient was started on total parenteral nutrition and managed conservatively with continuous nasogastric tube aspiration. Post-operative day 14, upper gastrointestinal contrast x-ray showed persistent obstruction at duodenum and contrast failed to pass even on left lateral and prone position of the patient. The patient was re-operated and duodenojejunostomy performed. Post-operative period was uneventful and the patient started tolerating oral feeding from fourth post-operative day. The patient was discharged on post-operative day 25 and is on regular follow up.

DISCUSSION
SMA syndrome first described in 1861 by Von Rokitansky[1] is a rare condition. It usually affects young females from 10 to 39 years of age.[2]
The pathophysiology of this disorder relates to the angle between the SMA and the abdominal aorta at the level of the first lumbar vertebra. It is caused by trapping of the third part of the duodenum as it crosses between the SMA anteriorly and the aorta and vertebral column posteriorly. The normal angle between the SMA and the abdominal aorta is approximately 45° (range 38-56°) in standing position. Any factor that sharply narrows this angle to between 6° and 16° results in a vascular, extrinsic compression of the duodenum as it passes between the SMA and the aorta.[3]

Although the exact etiology is not known, certain predisposing conditions are well recognized.

- Diseases associated with rapid severe weight loss with a decrease in the retroperitoneal and mesenteric fat such as anorexia nervosa,[4] malabsorption syndrome, increased catabolic states as in cancer and burns
- Neurological disorders causing dystonic denervation of the abdominal wall and spinal muscles such as acute trauma to the spine,[5] patients who had spinal surgery, the use of a body cast in the treatment of scoliosis or vertebral fractures, traumatic brain injury, spastic quadriplegia, and prolonged recumbency[6-7]
- Anatomical anomalies such as abnormally high and fixed position of the ligament of Treitz, or an unusually low origin of the SMA[8]
- Constitutional factors such as tall thin body build which is reported in 80% of patients,[9] exaggerated lumbar lordosis, visceroptosis, and rapid linear growth without compensatory weight gain, particularly during adolescence.

Familial SMA syndrome also has been documented suggesting the possibility of genetic predisposition.[10]

Clinical symptoms can be acute or chronic with intermittent exacerbations. The patient usually presents with symptoms of gastrointestinal obstruction, which includes postprandial nausea, epigastric pain, bilious vomiting, or vomiting of partially digested food. The symptoms are typically relieved when the patient is lying prone, in the left lateral decubitus, or in the knee-chest position; and they are aggravated when the patient is lying in the supine position.

Principal complication are electrolyte imbalance, gastric perforation, and duodenal bezoar formation.[11,12] A patient can present with complications like shock, respiratory failure, and acute gastric necrosis. Massively dilated stomach can cause compression of inferior vena cava and can result in shock. Massive gastric dilatation may compromise the ventilation and cause respiratory failure due to diaphragmatic splitting. Ischemic necrosis of the stomach can also occur if the intra-gastric pressure exceeds 20 cm H2O.[13,14] Delayed onset SMA syndrome even can cause gastric dilatation and hypomotility with chronic acid reflux followed by esophageal stenosis, thus peptic stricture of esophagus.[15]

Diagnosis depends on high index of suspicion since symptoms can be non-specific.

The SMA syndrome should be distinguished from a SMA-like syndrome. Sometimes, duodenal dilatation proximal to compression by the SMA may essentially be part of generalized dilatation of duodenum of variable etiology and not secondary to obstruction by a compressing structure. The most notable of these conditions is scleroderma; however, other causes of reduced duodenal peristalsis such as diabetes, pancreatitis, dermatomyositis, lupus erythematosus, myxoedema, amyloidosis, myotonic dystrophy, or chronic idiopathic intestinal pseudo-obstruction can cause this SMA-like syndrome.[16]

The clinical diagnosis can be confirmed by radiologic studies in 95% of cases.[17] Historically, barium meal and arteriography were used as diagnostic tools,[18] but more recently, CT, CT-angiography, and magnetic resonance imaging (MRI) have been used and showed higher diagnostic sensitivity.[19]

Upper gastrointestinal contrast series will show the following characteristic criteria.[20]

- Dilatation of the first and second parts of the duodenum with or without gastric dilatation
- Abrupt, partial or complete, vertical obstruction of barium flow at the site where SMA crosses the third part of the duodenum (duodenal clamp)

Figure 1: Acute angulation of SMA from aorta

Figure 2: Dilated stomach, first and second part of duodenum
Arteriographic criteria include a significantly decreased aorto-SMA angle of 6° to 25° (normal = 45°) and a shortened aortomesenteric distance of 2 to 8 mm (normal = 10 to 20 mm) according to Konen et al. He reported that CT angiography with 3D reconstructions allows more accurate measurements of the aorta-SMA angle and distance than angiography alone since the SMA is anterolateral to the aorta; as a result, this is currently considered the technique of choice, as it is both non-invasive and more precise.

Conservative treatment is recommended in all patients with SMA syndrome and it aims at identifying and correcting reversible predisposing factors and restoring retroperitoneal fat to relieve the obstruction. It involves decompression of the stomach and correction of dehydration and electrolyte imbalance by intravenous fluids. Then feeding starts with small meals of liquids and proper positioning of the patient after meals either prone or in the left lateral decubitus position. Furthermore, prokinetics like metoclopramide administration may be used to enhance emptying of the stomach. Patients with severe rapid weight loss may benefit from total parenteral nutrition or enteral feeding by the insertion of a nasojejunal tube, which is passed distal to the site of obstruction.

Medical treatment has a high success rate in cases with an acute presentation of SMA syndrome but most chronic cases require surgery.

Indications for surgery:
- Failure of conservative medical therapy
- A long history of indigestion, progressive weight loss, and pronounced dilatation of the duodenum with stasis
- Complicated peptic ulcer disease

The various surgical techniques which have been described are ligament of Treitz amputation, gastrojejunostomy, subtotal gastrectomy and Billroth 2 gastrojejunostomy, duodenojejunostomy, anterior reposition of the duodenum, duodenal circular drainage operation, etc.

Historically, gastrojejunostomy was used to treat SMA syndrome. This procedure has largely been abandoned due to associated complications including dumping syndrome, blind loop syndrome, and marginal ulceration.

Cleavage of the ligament of Treitz, first described by Strong in 1958, is another option, enabling the duodenum to drop away from the apex of the sharpened aortomesenteric angle and it has been used successfully in many cases. The major advantage of the procedure is the avoidance of a gastrointestinal anastomosis but this produces an unacceptably high failure rate. Recurrence after ligament liberation occurs simply as a result of post-operative adhesions tethering the bowel into a similar position as the ligament of Treitz had done previously.

The most common operation for SMA syndrome, duodenojejunostomy, was first proposed in 1907 by Bloodgood. This open surgery involves the creation of an alternate route between the duodenum and the jejunum, bypassing the compression caused by the abdominal aorta and the SMA. This is the most effective of the procedure with success rate of 90%.

Laparoscopic duodenojejunostomy offers a new minimally invasive therapeutic approach for the management. It is feasible, safe, and effective. It gives the same results as open surgery with all the advantages of minimally invasive surgery.

Laparoscopic lysis of duodenum and jejunum has also been tried. A laparoscopic approach without any anastomosis or suture possesses this “simple and safe” advantage. Other than Strong's procedure which also has the advantage but only applies to some selected patients whose ligament of Treitz and the fourth part of the duodenum were conspicuously lifted high, this approach probably has more indications.

On July 30, 2008, the world’s first robotically-assisted intestinal bypass surgery for a patient with SMA syndrome was announced by the London Health Sciences Centre in a 16-year-old girl.

If reversed peristalsis exists continually and chronically, the reversed peristalsis will be hardly eliminated. Releasing from obstruction by operation cannot alleviate clinical symptoms because the strongly reversed peristalsis still exists. Therefore, emphasis should be laid on eliminating the reversed peristalsis to alleviate frequent vomiting. This can be done by Duodenal circular drainage procedure. The operation is complicated as compared with duodenojejunostomy, and the patients may suffer from more damages as a result of the operation; small stomach syndrome and anemia can be induced by resection of most gastric body; the mucous membrane of alimentary tract cannot be nourished by gastrin after resection of gastric antrum; and bilious reflux gastritis may occur after resection of sphincter of pylorus.

Gastroparesis after correction surgery is a frequently encountered problem related to gastric and duodenal atony. Although the presence of such persistent symptoms has been described in the literature, there is little information on their management. Prokinetics have been tried with some success in patients with refractory symptoms after surgery. The other available treatment options for gastroparesis include a gastric pacemaker and gastric volume reduction surgery, but there is a lack of real evidence for their role in treating gastroparesis after corrective surgery.

CONCLUSION

SMAsyndrome is a rare condition and the diagnosis depends on
high index of suspicion as symptoms being nonspecific. MRA, being not invasive, is rapidly replacing the arteriogram in confirming the diagnosis. Conservative management may be sufficient in early cases. Open or laparoscopic duodenojenunostomy is the surgical treatment of choice.

REFERENCES


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