Case report

Computed tomography features and surgical treatment of superior mesenteric artery syndrome:
A case report

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\textbf{A R T I C L E  I N F O}

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\textbf{A B S T R A C T}

Introduction: Superior mesenteric artery (SMA) syndrome is described as compression of the third part of the duodenum between SMA and aorta with resultant obstruction and dilatation of proximal duodenum and stomach. Virtually, any condition associated with weight reduction may predispose the patient to SMA syndrome. Case presentation: A 17-year-old boy complaining from persistent vomiting, dull abdominal pain, anorexia, and weight loss for long time presented to the pediatric surgery department. Computed tomography (CT) of the abdomen was prescribed to look for the cause of persistent vomiting and bulging of the epigastrium. Contrast Enhanced CT revealed decreased aortomesenteric angle and aortomesenteric distance causing compression of third part of duodenum with resultant marked distension of proximal duodenum, stomach, and even esophagus. The patient underwent laparotomic gasterojejunostomy. Conclusion: SMA syndrome is a rare clinical entity. CT can well delineate this abnormality. SMA syndrome can be treated with both conservative and surgical approaches.

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Background

Superior mesenteric artery (SMA) syndrome was first described by Rokitansky in 1842 \cite{1}. SMA syndrome is described as compression of the third part of the duodenum between the SMA and aorta with resultant obstruction and dilatation of proximal duodenum and stomach. Virtually, any condition associated with weight reduction may predispose the patient to SMA syndrome. Cases of SMA syndrome associated with tuberculosis, brucellosis, diabetes mellitus, anorexia nervosa, blunt abdominal trauma, burns, and surgery have been reported \cite{1–7}. Goitein, David, et al reported 3 cases of SMA syndrome following laparoscopic Roux-en-Y gastric bypass surgery \cite{8}.
Authors present a case of this rare entity diagnosed with computed tomography and treated by laparoscopic duodeno-jejunostomy.

Case presentation

A 17-year-old male complaining from persistent vomiting, dull abdominal pain, anorexia, and weight loss for long time presented to the pediatric surgery department. On physical examination patient looked ill, pale, and cachectic. Epigastric area bulging was observed. Other systems were unremarkable. Unsuccessful attempt of endoscopy was performed but the inappropriate endoscopic device, could not go further than the second part of duodenum. Computed tomography (CT) of the abdomen was prescribed to look for the cause of persistent vomiting and bulging of the epigastrium. Contrast Enhanced CT of the abdomen was performed with 128 slice Siemens scanner after oral administration of 600 mL of water and 70 mL of intravenous nonionic water soluble contrast medium (Omnipaque 300). CT examination revealed decreased aortomesenteric angle ($6^\circ$) and aortomesenteric distance (8 mm) (Fig. 1a) causing compression over third part of duodenum with resultant marked distension of proximal duodenum, stomach, and even esophagus (Fig. 1b). The mesenteric fat was decreased. Rest of the abdominal organs appeared normal. Based on the CT images the diagnosis of SMA syndrome was made. As the case was associated with significant mechanical obstruction, therefore surgery was selected as preferred treatment. The patient underwent laparotomic

Fig. 1 – (a) Contrast enhanced abdominal CT, sagittal cut: Decreased aortomesenteric angle and distance. Significantly distended stomach extending down to the pelvis.

Fig. 1 – (b) Contrast enhanced abdominal CT, axial cut: Decreased aortomesenteric distance exerting pressure on duodenum with resultant significant dilatation of proximal duodenum and stomach. There is marked loss of mesenteric fat.

Fig. 1 – (c) Contrast enhanced abdominal CT, coronal cut: Distended esophagus, stomach and duodenum with caliber transition of duodenum at while it reaches the border of aorta (Black arrow).
gastrojejunostomy. Follow-up after 10 days and 1 month was performed. The patient did not have any complain.

Discussion and conclusion

Wilkie described the clinical and pathophysiological characteristics and management approach of SMA syndrome in a series of 64 patient and named it Wilkie’s syndrome [9-10]. Many other eponyms like chronic duodenal ileus, megaduodenum, aortomesenteric artery compression, arteriomesenteric duodenal obstruction, cast syndrome, and chronic duodenal pseudoobstruction have also been used for this entity [11].

SMA syndrome has a specific anatomic basis. The SMA originates from the anterior aspect of the aorta, at L1-L2 vertebral level and descends downwards at an acute angle of approximately 45° (range 38°-60°) and a distance of 10-28 mm [12]. The duodenum crosses the midline at the level of the L3 lumbar vertebra in the distance between the aorta and SMA. Thinning out of the fat pad between SMA and aorta, consequently upon weight loss, narrows the aortomesenteric angle as well as the distance between them, causing compression of the duodenum and thus producing the clinical manifestations of the syndrome. Clinically, the patients present with features of gastric outlet obstruction. Sense of satiety, post-prandial epigastric pain, belching, and vomiting are characteristic features.

Upper gastrointestinal contrast study can reveal dilatation of the stomach and proximal duodenum with an abrupt cutoff across its third part. CT can reveal decreased aortomesenteric distance as well as aortomesenteric angle which can easily be seen on sagittal reconstructed images (as in current case: Fig. 1a). In proper clinical setting, these findings virtually establish the diagnosis of SMA syndrome. Ultrasonography has also been used to aid the diagnosis. The ultrasonographic findings include to and fro movements across the duodenum with facilitation of the flow through the jejunum and elongation of the aortomesenteric distance when the patient assumes the right recumbent position [13]. These findings establish the role of positioning in providing a symptomatic relief in such cases. Endoscopy, if performed may reveal narrowing of the third part of the duodenum due to extrinsic compression [1], however in current case the inappropriate endoscopy device could not go further than the second part of duodenum.

Both medical and surgical options do exist for management of SMA syndrome. Usually the treatment starts with medical lines that include decompression of the stomach and duodenum with a nasogastric tube, correction of nutritional, and electrolytes deficiencies [1]. If possible, enteral feeding with a nasojugal tube passing the point of compression can also be tried to facilitate the nutritional management. When tolerated, oral feeding may be resumed. This helps to build up the fat cushion between the SMA and aorta. Additionally lying in prone or right recumbent position can relief the symptoms. In cases where medical approaches fail, surgical intervention may be considered. The mainstay of the surgical treatment is to bypass the site of obstruction by anastomosing the proximal and distal gastrointestinal tract, thus resuming the functional integrity of the bowel. This may be in the form of gastrojejunostomy or duodenojejunostomy, by laparotomic or laparoscopic means [14,15]. Dissection of the ligament of Treitz, with mobilization of the third and fourth parts of the duodenum, releasing the compression, has also been reported [16]. More recently, robotic duodenojejunostomy has been utilized with success [17].

The differential diagnosis for this condition can be diabetic gastroparesis, scleroderma with duodenal involvement, hereditary megaduodenum and megaduodenum due to aganglionosis [18,19]. The distinction between these entities and SMA syndrome is of utmost importance when embarking on treatment, especially the surgical option.

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