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A RARE ASSOCIATION OF PEPTIC ULCER DISEASE RELATED GASTRIC OUTLET OBSTRUCTION WITH SUPERIOR MESENTERIC ARTERY SYNDROME IN AN ADOLESCENT MALE

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ABSTRACT

Introduction: Superior mesenteric artery (SMA) syndrome is a rare clinical entity caused by acute angulation of SMA and abdominal aorta resulting in duodenal obstruction in its third part. Its association with complicated peptic ulcer disease is rare. Although the global incidence of peptic ulcer disease and its complications has decreased in the last few decades, it is still prevalent in low or middle socioeconomic countries like India. We present a rare association of gastric outlet obstruction (GOO) caused by complicated peptic ulcer disease (PUD) and SMA syndrome in an adolescent patient. **Case presentation:** An 18-year-old boy presented with mild upper abdominal pain, recurrent non-bilious vomiting, and severe weight loss for two years. The esophago-gastro-duodenoscopy (EGD) revealed pin-point pylorus, suggesting GOO. Contrast-enhanced computed tomography showed a decrease in aorto-mesenteric angle with compression of the third part of the duodenum and simultaneous stricture in the first part of duodenum giving a dumbbell appearance of dilated duodenum and stomach. He underwent duodeno-jejunostomy and Roux-en-Y gastrojejunostomy.

Conclusion: Though SMA syndrome is rare, it should be considered in cases of upper intestinal obstruction. The coexistence of PUD related GOO with SMA syndrome is extremely rare and is the first of its kind reported in the literature.

Keywords: Superior mesenteric artery syndrome, Gastric outlet obstruction, Peptic ulcer disease, Duodenal obstruction

1. INTRODUCTION

Superior mesenteric artery syndrome also known as Wilkie's syndrome is a rare but well-defined entity obstructing the third part of the duodenum **[1]**. Although its true incidence is unknown, approximately 0.013-0.78% of upper gastrointestinal barium studies and 0.005% of esophagogastroduodenoscopies show evidence of SMA syndrome **[1, 2]**. Contrast-enhanced computed tomography (CECT) with arterial reconstruction is the best modality for the establishment of diagnosis and evaluation of severity **[3]**. The third part of the duodenum passes through the angle between the origin of SMA and abdominal aorta, which is approximately 45˚ (range, 38-60˚), and is supported by a pad of fat **[2, 3]**. The loss in the pad of fat due to various reasons like severe weight loss secondary to malabsorption syndromes, trauma, anorexia nervosa, and cancer cachexia, burns, and application of abdominal cast predispose to duodenal compression between SMA and aorta **[1, 4, 5]**. Anatomical variations like the high insertion of the duodenum at the ligament of Treitz, low origin of SMA, and rarely familial causes also predispose to SMA syndrome **[6, 7]**.

Although malignancy remains the most common cause of GOO globally, complicated PUD remains a predominant cause of GOO in India especially in young patients **[8, 9]**. Besides, people staying in rural areas, in unhygienic conditions are exposed to recurrent *Helicobacter pylori* infection and subsequent complicated PUD **[10, 11]**. The SMA syndrome may mimic GOO, but the true association of PUD related GOO and SMA has not been reported in the literature. We report a rare case of coexistent GOO and SMA syndrome in a young boy belonging to eastern India.

1. Case report

An 18-year-old male belonging to low socioeconomic status presented with mild upper abdominal pain, recurrent non-bilious vomiting which was aggravated in the last three months, and severe weight loss (of 15 kilograms) for two years. History did not suggest any traumatic event. On examination, he was thin built with a body mass index of 17.5 kg/m^2. Laboratory parameters were within normal limits except for microcytic hypochromic anemia with hemoglobin of 9.5 gm/dL.

The EGD showed a dilated stomach with a pinpoint pyloric opening, which could not be negotiated. The rest of the gastric mucosa was erythematous, and the mucosal biopsy revealed a positive rapid urease test

suggesting *Helicobacter pylori* infection. CECT scan with oral contrast showed grossly dilated stomach and the first and second part of the duodenum in dumbbell fashion with stricture at the first part of the duodenum **(shown in Figure 1, 2)**.

The arterial reconstruction showed narrowing of the aortomesentric angle to 12.4˚, and the aorto-mesenteric distance was 4mm causing compression of the third part of the duodenum **(shown in Figure 3, 4)**.

He underwent infra-colic duodeno-jejunostomy and a Roux-en-Y gastro-jejunostomy, with the length of Roux limb of approximately 40 cm and was prepared 20 cm distal to the duodeno-jejunostomy site **(shown in Figure 5, 6)**. The postoperative period was uneventful. *Helicobacter pylori* eradication was continued for 21 days after discharge.

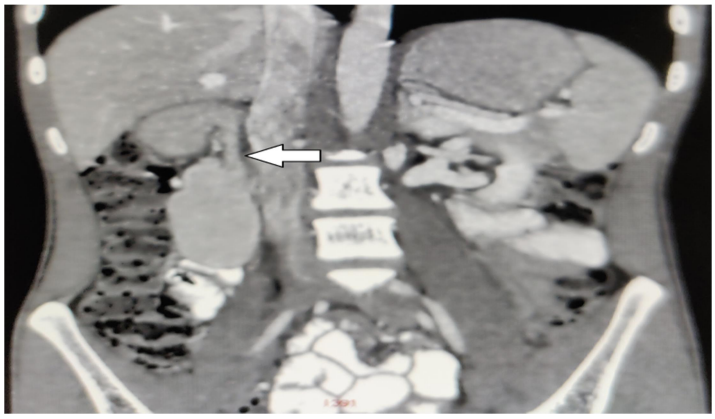

Figure 1: CECT abdomen, coronal section, showing stricture at first part of duodenum (marked with arrow) with dumbbell shaped dilation of stomach and duodenum.

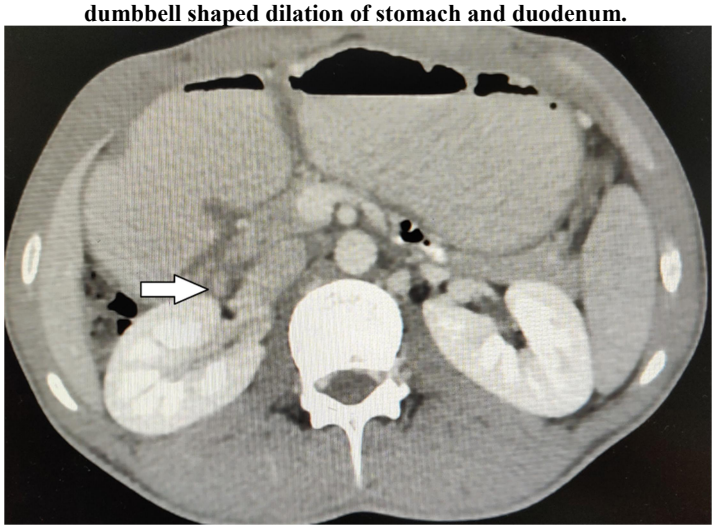

Figure 2: CECT abdomen, axial section, showing duodenal stricture (marked with arrow) with proximal dilated stomach.

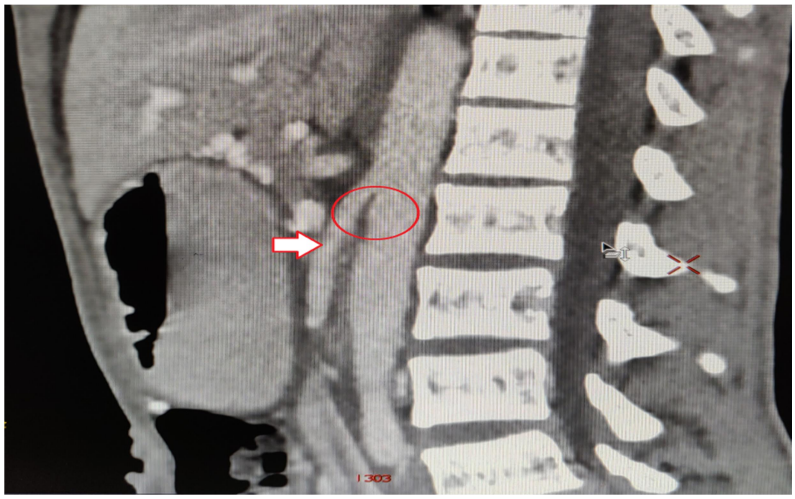

Figure 3: CECT with arterial reconstruction, sagittal view, showing marked narrowing of aorto-mesenteric angle (marked by circle), superior mesenteric artery marked with arrow

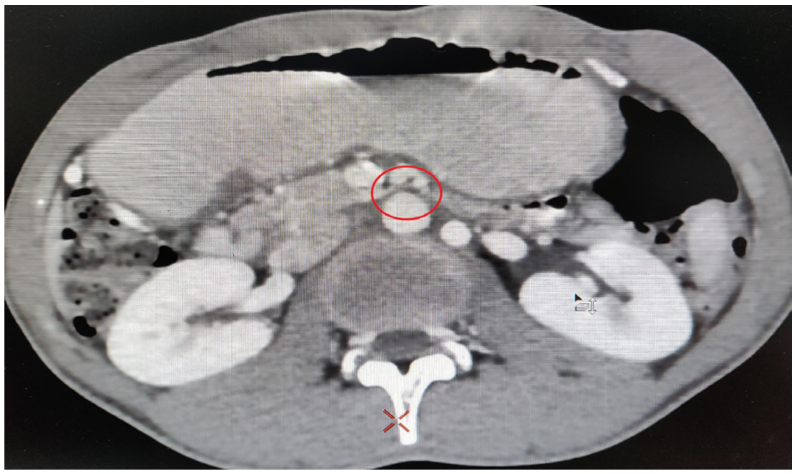

Figure 4: CECT with arterial reconstruction, axial view, showing shortening of aorto-mesenteric distance (marked with circle)

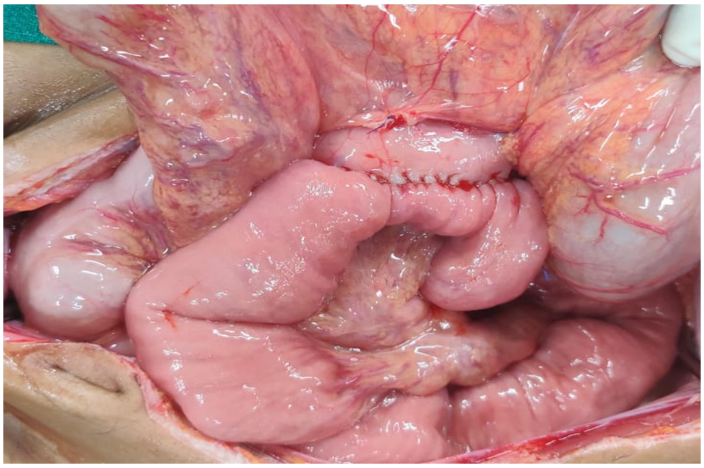

Figure 5: Intraoperative picture showing completed infra-colic duodeno-jejunostomy

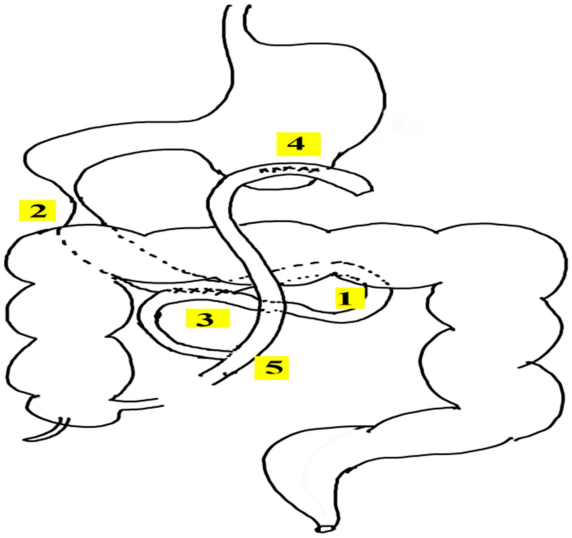

Figure 6: Schematic diagram of reconstruction 1) narrowed duodenum because of arterial compression, 2) duodenal stricture because of peptic ulcer disease, 3) site of duodeno-jejunostomy, 4) site of Roux-en-Y gastro-jejunostomy, 5) site of jejuno-jejunostomy

2. DISCUSSION

Anatomically, the third part of the duodenum and left renal vein pass through the angle created by the origin of SMA and abdominal aorta, which normally ranges between 38 and 60˚ [1, 2]. The angle is supported by a pad of fat which is essential to maintain the space for duodenal and venous patency. Loss of this pad of fat, anatomical variations of SMA origin, or abnormal duodenal attachments lead to a decrease in the aorto-mesenteric angle, and thereby predispose to duodenal obstruction and occasionally left renal vein occlusion **[12, 13]**. Although barium study and ultrasonography have been used to diagnose SMA syndrome, CECT with arterial reconstruction remains the best tool to accurately measure the aorto-mesenteric angle and distance **[3]**. When the aorto-mesenteric angle decreases below the critical level, usually less than 15˚, the duodenal compression ensues **[2, 12]**. Another radiological parameter determining the manifestation of SMA syndrome is the aorto-mesenteric distance, which is critical when less than 8mm **[2, 7]**. In our case, the aorto-mesenteric angle and distance were 12.4˚ and 4mm respectively.

SMA syndrome has been linked with several etiologies. Severe cachexia secondary to burn, malignancies, psychiatric illness like anorexia nervosa have shown to predispose to SMA syndrome **[1, 4]**. The low origin of SMA from the aorta, high insertion of the ligament of Treitz, severe lumbar lordosis, duodenal adhesions have also been

associated with this condition **[5, 7]**. Although most of the patients present with weight loss, in one study 50% of pediatric patients did not have weight loss **[1, 14]**. The growth spurt in adolescents has been associated with SMA syndrome, especially when exacerbated by other triggers like extra weight loss by dieting or infection **[14, 15]**. The present case was an adolescent boy who attained growth spurt that predisposed SMA syndrome.

The incidence of chronic duodenal ulcer has decreased due to improvement in socioeconomic status, awareness, and hygiene **[10, 11]**. Furthermore, the availability of antisecretory agents, *Helicobacter pylori* eradication therapy has decreased the complications relating to PUD **[11]**. However, the scenario is less altered in the population belonging to low socioeconomic status. In India, the incidence of PUD and its complications, though decreased, are still highly prevalent **[10, 11]**. The GOO presents with a gradual increase in non-bilious vomiting episodes, thereby decreasing the food intake and aggravating weight loss. Moreover, the *Helicobacter pylori* itself, by causing gastritis, further aggravates the situation **[10]**. In our case, the symptoms persisted for two years causing significant weight loss. The growth spurt exacerbated by decreased food intake and weight loss secondary to PUD associated GOO led to SMA syndrome.

The treatment for SMA syndrome is varied. Few cases have been successfully managed conservatively with dietary supplementation either by the enteral or parenteral route **[7]**. The surgery essentially consists of open or minimally invasive duodeno-jejunostomy **[7]**. In our case, Roux-en-Y gastro-jejunostomy was added to duodeno-jejunostomy because of associated GOO. The present case emphasizes the rare association of PUD related GOO with SMA syndrome in an adolescent boy, in which the former triggered the later.

2. CONCLUSION

SMA syndrome is a rare clinical entity, predisposed by growth spurt in the adolescent age group and aggravated by significant weight loss. Although it may present with features of GOO, its true association with PUD related GOO is extremely rare, and this report is the first of its kind in the literature.

Statement of Ethics

Informed consent was obtained for the publication of the case details from the patient and his relatives.

Disclosure Statement

The authors have no conflict of interest to disclose.

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Author Contributions

Sumit Subhadarshi Mohanty and Satyaprakash Ray Choudhury designed, wrote, edited, and approved the final version of the manuscript.

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