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WEIGHT LOSS: A CAUSE OR A CONSEQUENCE OF WILKIE'S SYNDROME

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ABSTRACT

The Superior mesenteric artery syndrome (SMAS) known as Wilkie's syndrome is a rare cause of duodenal obstruction. It is a disorder in which acute angulation of the SMA causes compression of the third part of the duodenum between the SMA and the aorta, following the disappearance of perivascular fatty tissue after advanced malnutrition. The diagnosis requires a high degree of clinical suspicion of upper gastrointestinal obstruction plus progressive weight loss, with radiographic studies demonstrating compression of the third portion of the duodenum. The severity of the symptoms depends on the aortomesenteric angle and the degree of the compression. The treatment is medical, but in case of failure, surgery is necessary.

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INTRODUCTION

A 27-year-old female presented to our institution with persistent epigastric pain, severe bloating, postprandial vomiting, irregular bowel movements and a 20 kg weight loss in the past 2 years. Upon admission to the hospital, vital signs were within normal limits. Physical examination was unremarkable except for distension of the abdomen and stomach, with diffuse abdominal pain on palpation. Laboratory evaluation at admission was normal. Blood urea level 28 mg/dl and creatinine 1.05 mg/dl, White blood cell count was 5,000/mm3, hemoglobin 12 g/dl, and sodium level 145 mmol/l. Abdominal X ray demonstrated distension of the stomach (Fig 1). Endoscopy was done with difficulty due to liquid and nutriment stagnation in the stomach. A nasogastric tube was inserted. Endoscopy was repeated the second day, and demonstrated the presence of external pulsatile compression at the 3rd part of the duodenum. (Fig 2). A CT scan of the abdomen and pelvis with contrast injection showed an acute angle of 10° between the SMA and the aorta, anterior compression upon the third part of the duodenum (Fig. 3) with severe dilatation of the stomach and proximal duodenum, and absence of any intraluminal mass (fig.4). Diagnosis of Wilkie's syndrome was made based on these findings. Laparotomy was done and showed an important dilatation of the stomach and duodenum up to D3. A duodenojejunostomy was done. Control barium swallow exam on the fourth postoperative day showed normal passage of the contrast. The postoperative period was uneventful, and the patient was discharged on day five.

DISCUSSION

Superior mesenteric artery (SMA) syndrome also known as Wilkie's syndrome, is a rare digestive condition that is caused by compression of the third portion of the duodenum between the aorta and the superior mesenteric artery. Some studies report that its incidence ranges between 0.01 and 0.3 % (Welsch, 2007). SMA syndrome was first described in 1861 by the Austrian professor Von Rokitansky, who suggested that this disease is caused by an obstruction of the third part of the duodenum as a result of arteriomesenteric compression, based on an autopsy finding (Von Rokitansky, 1861).

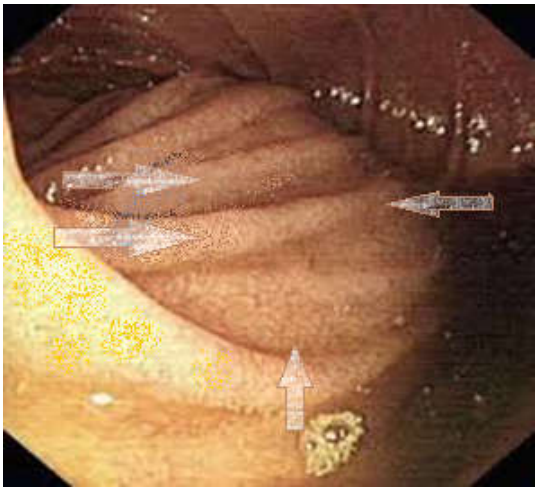

Fig .1. Extrinsic compression of the 3rd part of the duodenum

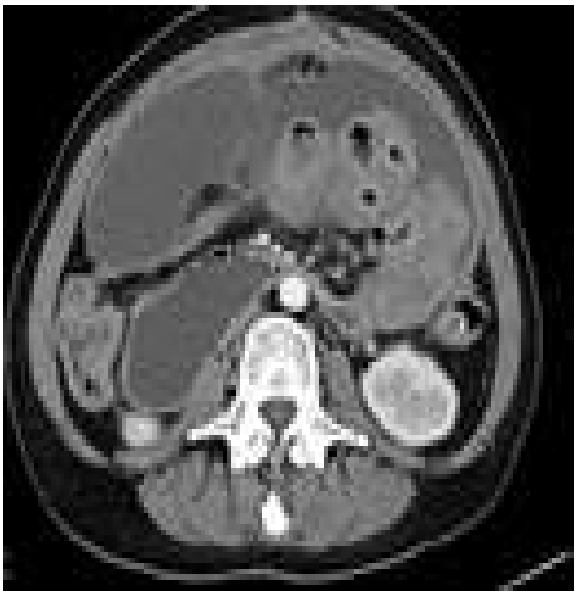

Fig 2: distension of the duodenal bulb and stomach due to compression of the third part

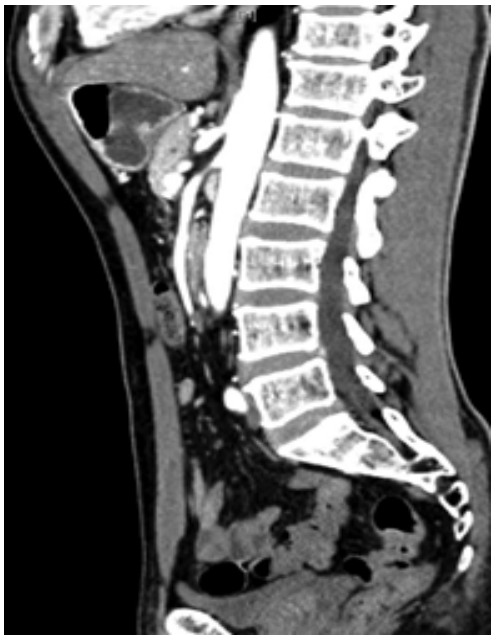

Fig 3: The angle between the abdominal aorta and superior mesenteric artery was 10°

In humans, the superior mesenteric artery usually forms an angle of approximately 45° (range, 38-65°) with the abdominal aorta, due to erect posture (Ahmed, 1997). Any factor that sharply narrows the aortomesenteric angle to <25% can cause entrapment and compression of the third part of the duodenum as it passes between the superior mesenteric artery and aorta, resulting in SMA syndrome, and as a result, the aortomesenteric distance is decreased to <10 mm (normal is 10-20 mm) (Merrett *et al*., 2009). SMA syndrome is caused primarily by any process that causes narrowing of the aortomesenteric angle, most commonly seen with conditions causing severe weight loss leading to a depletion of the mesenteric fat pad (Ozkurt *et al*., 2007). Burns (1972), malabsorption syndromes, malignancy, eating disorders such as anorexia nervosa (Verhoef, 2009), and most recently, bariatric surgery (Goitein *et al*., 2004) are the most common reported reasons of significant weight loss (Merrett *et al*., 2009).

Etiological factors also include congenital or acquired anatomical abnormalities. Congenital etiologies include high insertion of the duodenum at the ligament of Treitz or a low origin of the SMA (Merrett *et al*., 2009). Acquired anatomical abnormalities can occur following spinal surgery such as scoliosis surgical repair (Tsirikos *et al*., 2009), spinal trauma, and after surgery of the abdomen such as ileal J-pouch anastomosis after total proctocolectomy (Ballantyne *et al*., 1987). Signs and symptoms of SMA syndrome can be nonspecific. Patient often presents with chronic upper abdominal symptoms such as epigastric pain, nausea, eructation, post-prandial discomfort, early satiety and voluminous bilious vomiting (Matherge, 2013). Patients have also reported improvement of symptoms when lying supine in a knee-to-chest or left lateral position. This maneuver reduces the small bowel mesenteric tension at the aortomesenteric angle (Ahmed *et al*., 1997). High index of clinical suspicion combined with radiographic evidence can aid the diagnosis. Contrast-enhanced abdominal CT or magnetic resonance angiography has shown the most sensitivity for diagnosing SMA syndrome; it visualizes the vascular compression of the duodenum and enables the evaluation of the aortomesenteric angle and distance (Unal *et al*., 2005).

Dilation of the duodenum in its first and second part, anti-peristaltic flow of the barium proximal to the obstruction, and a delay of gastroduodenojejunal transit time with relief when the patient is placed in knee-chest or left lateral position, are suggestive of SMA syndrome on barium radiography (Welsch *et al*., 2007). Other causes of obstruction must be ruled out by endoscopic examination, which in this case may show a pulsatile extrinsic compression suggestive of SMA syndrome (Gustafsson *et al*., 1984). Initially treatment is conservative and should focus on nutritional support such as parenteral nutrition and/or ideally through post-pyloric enteric feeding via a nasojejunal tube placed distal to the obstruction (Welsch *et al*., 2007; Matherge, 2004). Prokinetics and postural maneuvers during meals may be helpful in some patients. Patients with severe symptomatology or those who do not respond to nutritional support may require surgical management (Mandarry *et al*., 2010). Duodenojejunostomy is the operation of choice with high success rates up to 90% (Mandarry *et al*., 2010). Gastrojejunostomy, has an increased postoperative complication rate due to the presence of a blind loop and the non-decompression of the duodenum which may cause recurrence of symptoms.

Strong's procedure, another surgical option, consists of mobilizing the duodenum by lysing the ligament of Treitz; however, it has a 25% failure rate (Merrett *et al*., 2009).

Conclusion

SMA syndrome is an atypical cause of proximal intestinal obstruction. The symptomatology depends on the degree of the obstruction. Patients can present with abdominal pain, post prandial vomiting, bloating and progressive weight loss that could be the major cause of Wilkie syndrome. Conservative therapy with nutritional supplementation is the initial approach in case of partial obstruction. Duodenojejunostomy is indicated in case of medical treatment failure.

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