

PROSPERI PORTA Ilaria (1), PAGANI Daniela (1), BARICOCCHI Denise (1), LUNARDI Gianmarco (2), NATRELLA Massimiliano (2), BALDASSARRE Emanuele (3), PODIO Stefano (1)

CASE REPORT

A 50-year-old woman presented to our ED with a complaint characterized by severe epigastric pain and vomiting since 12 h. Past medical history was negative. In the last 6 months she had similar episodes with appetite loss and significant weight decrease. 3 months before, CT scan excluded obstructive pathology or intestinal ischemia and she was referred to GI unit for dyspepsia.

Physical examination showed extreme thinness. Abdomen was flat and palpable, with tenderness. US was negative. ECG was normal. Laboratory findings showed only leukocytosis (WBC 26,000). COVID and flu swab negative. Chest and abdominal X-ray were negative. We gave crystalloids, intravenous PPIs, antiemetics, and analgesics (paracetamol and morphine via IV). The complaint rapidly worsened with agitation, significant restlessness, repeated squatting and self induced vomit. A new CT scan revealed a reduced angle at the junction between the Superior Mesenteric Artery (SMA) and the Aorta (approximately 17°), with compression of the renal artery (nutcracker syndrome) (Fig. 1,2) and the third portion of the duodenum (superior mesenteric artery syndrome, SMAS), resulting in upstream dilation of the gastric cavity. The patient was sent to surgery.

DISCUSSION

SMAS can cause proximal intestinal obstruction with compression of the third portion of duodenum, due to the narrowing of the space between SMA and aorta. The incidence is 0.013-0.3%, with a higher prevalence in females. The onset is primarily attributed to mesenteric fat pad loss between the 2 arteries. Risk factors are: significant weight loss (malignancies, malabsorption syndrome, immunodeficiencies, major trauma, burns, bariatric surgery, prolonged bed rest, anorexia nervosa), congenital or acquired anatomical variants (short Treitz ligament, anomalous SMA origin, Nissen fundoplicatio). Due its rarity, SMAS is often misunderstood, leading to diagnostic delays. Moreover symptoms like postprandial abdominal pain, nausea and vomiting, early satiety, anorexia, weight loss are too aspecific, mimicking a nervous anorexia. SMAS should be suspected in case of epigastric pain that worsens in supine position and ameliorates with lateral decubitus (knees to chest). A missed or delayed diagnosis could lead to life-threatening complications such as hypovolemic shock, aspiration pneumonia, sudden death, especially in young individuals. The death generally is related to arrhythmias with electrolyte imbalances, severe IVC compression and important pulmonary depression induced by alkalosis and increased intra-abdominal pressure.

Thus the diagnosis is based on both clinical presentation and radiological evidence of duodenal obstruction. CT scan allows to calculate the angle between SMA and aorta, identifying possible complications such as gastric necrosis and acute pancreatitis. US scan is a rapid and non-invasive diagnostic tool, with high sensitivity. In ED endoscopy can reveal complications and indicate extrinsic duodenal compression too. Laboratory tests are aspecific but helpful to find electrolyte alterations.

Treatment is predominantly conservative with via nasogastric tube, postural changes, lateral decubitus. Parenteral nutrition and metoclopramide are usually recommended. to promote intestinal motility. Surgery is reserved only in selected cases, after failure of medical therapy.



Fig. 1



Fig. 2

CONCLUSION

SMAS is a severe condition that requires rapid diagnosis and early treatment to prevent long-term complications. A correct diagnosis could be a challenge, but SAMS should be suspected in an history of rapid weight loss, previous abdominal surgery (typically bariatric), trauma, or congenital anomalies.

Such in our case, the differential diagnosis with dyspepsia should be considered. The role of urgentist is to early consider this syndrome to help the radiologist, particularly focusing their attention on the aortomesenteric angle to reach an early diagnosis, avoiding complications.

Affiliazioni

(1) SC MECAU, ASL Valle d'Aosta, (2) SC Radiologia, ASL Valle d'Aosta, (3) SC Urologia, ASL Valle d'Aosta

Contatti

PROSPERI PORTA Ilaria, SC MECAU, ASL Valle d'Aosta, Via Guido Rey 1 11100 Aosta
iprospериporta@ausl.vda.it Tel +39 0165 543923

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