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The two sides of superior mesenteric artery compression treatment: conservative or surgical management?

Las dos caras del tratamiento de la compresión de la arteria mesentérica superior: ¿manejo conservador o quirúrgico?

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ABSTRACT

Introduction: Superior mesenteric artery syndrome (SMAS) is a rare condition characterized by vascular compression of the duodenum. There is controversy regarding the optimal treatment.

Case report: In case 1, we describe the case of a 21-year-old woman (body mass index [BMI] 16.9 kg/m^2) with high-level obstructive symptoms three months prior, with computed tomography scan (TC) showing a superior mesenteric artery aorta angle (SMAA)

of 13° and compression of the third portion of the duodenum (D3), for this reason a nasojejunal tube was placed for enteral feeding. In case 2, enteral nutrition was initiated for feeding a 17-year-old female with anorexia nervosa (BMI 8.3 kg/m^2). She presented macrohematuria, vomiting, epigastralgia, abdominal distension and acute abdomen when oral feeding was reinitiated. TC reported a SMAA of 15°, in addition to compression of the left renal vein (Nutcracker syndrome) and gastro duodenal expansion, surgical management was necessary.

Discussion: Both cases had favorable evolution, being the nutritional support fundamental. SMAS should be suspected in all people with high-level obstructive symptoms and recent weight loss.

Keys words: Mesenteric clamp. Superior mesenteric artery syndrome. Obstructive symptoms. Nutritional support. Surgical treatment.

RESUMEN

Introducción: el síndrome de la arteria mesentérica superior (SAMS) es una condición rara caracterizada por la compresión vascular del duodeno, y existe controversia acerca de su tratamiento.

Caso clínico: en el caso 1, presentamos a una mujer de 21 años (índice de masa corporal [IMC] 16,9 kg/m^2) con datos de obstrucción alta y epigastralgia durante los tres meses previos, con tomografía computarizada (TC) que reporta ángulo de la arteria mesentérica superior (AAMS) de 13° y compresión de D3. Se colocó sonda nasoyeyunal para alimentarla. En el caso 2, se inició alimentación enteral por sonda nasogástrica en una mujer de 17 años con anorexia nerviosa (IMC 8,3 kg/m^2). Al reiniciar la vía oral presentó hematuria, vómito, epigastralgia, distensión abdominal y abdomen agudo. La TC reportó AAMS 15°, compresión de la vena renal izquierda (síndrome de Nutcracker) y distensión gastroduodenal. Requirió tratamiento quirúrgico.

Discusión: ambas pacientes tuvieron evolución favorable, siendo fundamental el soporte nutricional. Se debe sospechar el SAMS en los pacientes con datos de obstrucción intestinal alta y pérdida de peso reciente.

Palabras clave: Pinza mesentérica. Síndrome de la arteria mesentérica superior. Síntomas obstructivos. Soporte nutricional. Tratamiento quirúrgico.

BACKGROUND

Superior mesenteric artery syndrome (SMAS) is a condition characterized by extrinsic compression of the third portion of the duodenum (D3), caused by the superior mesenteric artery (SMA) and the aorta. It was described for the first time by Von Rokitanski in 1842, but in 1927 Wilkie first described its physiopathological mechanism (1). It is also known as Wilkie syndrome, Cast syndrome (2), arteriomesenteric duodenal obstruction, duodenum vascular compression syndrome, and chronic duodenal ileus (3). It is a rare disorder, with an incidence of 0.013-1%. It is infrequently diagnosed and affects chronically ill patients; lower-grade duodenal compressions that are asymptomatic may also exist (4). SMAS occurs more frequently in adolescents and young adults, with a modest predominance in women (1), as observed in the cases presented. Weight gain can help to resolve the compression; thus, nutritional management is vital in this context. However, it is unknown whether nutritional or surgical management is preferred for this condition.

CASE REPORT 1

We present the case of a 21-year-old woman with a long history of low intake, with the purpose of maintaining a “good physical state”. She began having nausea, postprandial vomiting, and epigastralgia three months prior to her admission. Based on her medical records, she did not report intentional weight loss, surgeries, or chronic diseases. She was admitted clinically stable: weight, 40.3 kg (normally 45 kg); height, 1.55 m; and body mass index (BMI), 16.9 kg/m^2; she lacked physical exam data. There were no remarkable results

from her laboratory exams, and a computed tomography scan (TC) of the abdomen was performed, showing a superior mesenteric artery aorta angle (SMAA) of 13°, an aortic mesenteric distance of 3.3 mm, and compression of the D3 (Fig. 1). For these reasons, a nasojejunal (NJ) tube was placed for enteral feeding, progressing towards 35 kcal/kg, without suspending oral feeding (approximate consumption 38 kcal/kg). No complications were presented, and no prokinetics were required.

She was discharged two weeks later with mixed feedings (oral and enteral) and completed six weeks with outpatient enteral nutrition, adequately tolerating the nutrition and gaining 3 kg. She had a follow-up TC performed, during which an improvement in mesenteric compression was observed (Fig. 2). The NJ tube was removed, and oral nutritional support of 45 kcal/kg was continued, with good results. During follow-up, she continued to increase in weight (45 kg and BMI of 18.7 kg/m^2) and reported being asymptomatic, with a psychiatric evaluation diagnosing anxiety and depression.

Ethics

Written informed consent was obtained from both patients, authorizing publication, reproduction and dissemination on paper and the internet. The authors have followed the protocols established in our health center to access the data of the clinical records in order to be able to carry out this type of publication for the purpose of research/dissemination for the scientific community.

CASE REPORT 2

We present the case of a 17-year-old female patient with a diagnosis of anorexia nervosa one year before. She had a usual weight of 43 kg, a current weight of 20.5 kg, a height of 1.57 m, and a BMI of 8.3 kg/m^2. She had an electrolyte imbalance at hospital admission (hypokalemia), hypoglycemia, and an alteration in liver function tests (associated with extreme malnutrition), without reporting gastrointestinal symptoms. Enteral nutritional support was initiated through a nasogastric (NG) tube for continuous feeding (exclusive)

starting with 5 kcal/kg/day. She had prior administration of thiamine IV and correction of serum electrolytes because of the risk of refeeding syndrome.

On day 12 of hospitalization, she presented with macrohematuria and required transfusion of a globular package. Her urine exam showed erythrocyturia, nitrites, bacteriuria, and leukocyturia, without proteinuria. Her treatment began with intravenous antibiotics and hydration, showing clinical improvement. She was given enteral nutrition progressing over the next two weeks to 38 kcal/kg, which was adequately tolerated. Oral feedings were reinitiated on day 21, but on the fifth day after initiation, she presented vomiting, epigastralgia, abdominal distension without data regarding acute abdomen, and a new episode of macrohematuria. A TC was performed, reporting an SMAA of 15° and an aorto-mesenteric distance of 4.3 mm, in addition to compression of the left renal vein and gastro duodenal expansion (Fig. 3).

The worsening of the patient's condition after receiving four weeks of enteral nutritional support was managed with total parenteral nutrition and surgery, where the Treitz ligament and the left renal vein were liberated. However, she continued to be intolerant to oral feedings and had abdominal distension, for which she required another surgical intervention: partial gastrectomy and duodenal and jejunal anastomosis. After this intervention, she experienced improvement, and on the fifth day post-surgery, oral feeding was reinitiated with adequate tolerance. However, because her oral intake was suboptimal due to an underlying pathology, an NJ tube was placed, through which she began complementary enteral feeding. She currently continues oral feeding with outpatient management, her liver function tests are normal, she does not present an electrolyte imbalance, and she weighs 32 kg, with a BMI of 13 kg/m^2. She was followed by a psychiatrist during this entire period.

DISCUSSION

Although the exact etiology of SMAS is unknown, it has been proposed that the principal mechanism involved is weight loss with a reduction in mesenteric fat reserve, which increases the compression in the space through which the duodenum passes (Table I).

SMAS is stimulated by other internal factors related to body anatomy, such as a hypertrophic or short Treitz ligament, lower origin of the SMA (1), diseases related to poor absorption, psychiatric disorders, traumatic aneurism of the AMS, familial SMAS, prolonged prostration, post-spinal correction surgery for scoliosis (6) and post-gastric bypass surgery, for which the diagnosis is much more difficult (7). Two physiopathological mechanisms have been described: the first mechanism is related to surgery and has an acute presentation, and the second mechanism is a result of severe weight loss, insidious in nature, and with progressive symptoms (3). The presentation of both patients' symptoms was gradual, in agreement with the second mechanism.

The angle between the AMS and the aorta measures between 38-65° (6,8). An angle < 25° is more likely to cause obstruction and gastric distension (the angle in the first case measured 11.7° and in the second case, 14°). The reduction in the aorto-mesenteric distance from the normal value of 10-28 mm to 2-8 mm causes a compression of the D3 and can also compress the left renal vein (6). There remains some controversy surrounding a diagnosis of SMAS since symptoms do not always correlate well with abnormal anatomic findings on radiologic studies (3).

The clinical findings are compatible with high-level intestinal obstruction, including abdominal pain during a meal, early satiety, nausea, bilious vomiting, and/or reflux with weight loss (9). This occurred in both patients, with these symptoms turning into a vicious cycle; because the patients no longer tolerated oral feedings, they lost even more weight. This condition should be differentiated from conditions that cause obstruction and/or intestinal dysmotility such as pseudointestinal obstruction (3). As the symptoms are not specific, diagnosis can be delayed and complicated by a gastric perforation and severe electrolyte imbalances (3).

Management of this condition can be conservative or surgical. The objective of the treatment is to alleviate the symptoms associated with intestinal compression and correct the precipitating factor by restoring the patient's nutritional state through enteral or parenteral support. The choice of the type of support will depend on the grade of the obstruction and the patient's tolerance. Adequate nutritional support and gastric

decompression contribute to the improvement of the AMSA (10), for which reason nutritional support is required in the initial stages of treatment. The adoption of postural therapy (left lateral decubitus, genu-pectoral) during feeding increases the AMSA (9), improving symptoms in half of patients (1).

Because weight gain is associated with an increase in adipose tissue with the consequent freeing of the pressure on the D3, conservative management can become the definitive treatment in many cases (6). In fact, conservative management has been shown to be successful in 83% of cases (10), and the first patient described forms part of this successful percentage. In patients with chronic symptoms, the likelihood of improvement is minimal; thus, a course of nutritional support to prepare for surgery should be considered (6). In these cases, nutritional support should be maintained until the nutritional status has improved sufficiently to not require support.

To begin nutritional support, enteral feeding via a nasal tube distal to the obstruction is usually chosen (3). Sometimes gastric decompression in patients with gastric and duodenal expansion is necessary (3). In these cases, when a gastric decompression tube with a jejunal extension is used for feeding, the use of prokinetics such as metoclopramide could be considered because this improves motility and aids gastric emptying. Parenteral nutrition is an alternative if the enteral pathway is contraindicated, and could even be considered as an adjunct therapy (10), as occurred in the second case.

Once weight gain has been noted, the diet should progress slowly (3) until reaching the caloric goal. However, those cases in which there is no improvement after 3-4 weeks with conservative management should be considered as unmanageable, particularly in patients with chronic SMAS with duodenal stasis or complicated peptic acid disease (10). In this type of situation, surgical procedures such as duodenal-jejunal anastomosis or division of the Treitz ligament with mobilization of the duodenum (Strong's operation) could be required (8). The patient in case 2 required two surgical procedures that included partial gastrectomy and gastrojejunal anastomosis. However, in the context of highly malnourished patients, such as in the second case, nutritional support is fundamental not only for their improvement but also as pre-surgical support if surgery is necessary.

In the first case, enteral/oral nutrition allowed the vascular space to be increased, as demonstrated on a TC; this could have potentially eliminated the need for surgery. However, in the second case, it was not sufficient, and surgery was required. Both cases presented SMAS, but only the second presented with nutritional support and had an extremely low BMI, which increased the case's complexity. Nutritional management is fundamental during the entire course of the disease and in recovery, although unmanageable cases do exist where surgery should be considered as an option.

Exclusive nutritional support should be the therapy of choice in cases of rapid development (< 4 weeks), and surgery should be considered in longer duration cases with nutritional support. We suggest evaluating each case individually.

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Table I. Physiopathological mechanisms of superior mesenteric syndrome

Composition	Related to body composition, anatomical abnormalities, exaggerated spinal curvature, rapid linear growth without a compensatory weight gain
Medical conditions	Poor absorption, catabolic state, emaciation, cancer, prolonged bed rest, trauma
Psychiatric conditions	Eating disorders, drug abuse
Surgeries	Correction of scoliosis, bariatric surgery, spinal procedures, vertebral column fixation with plaster, post-operatory weight loss
Unusual causes	Diabetes, retroperitoneal space abscesses, pancreatitis, lymphoma, traumatic aneurysm from AMS, familial mesenteric artery syndrome and recurrent mesenteric artery syndrome, unusually high insertion of the Treitz ligament

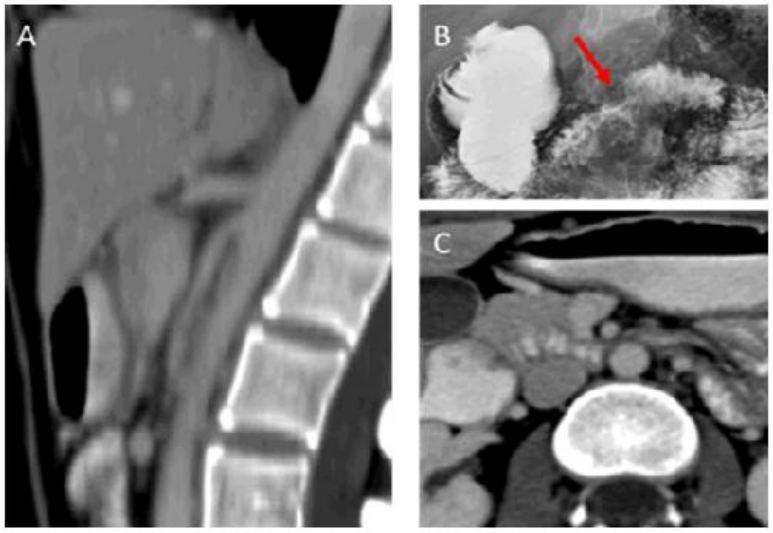

Figure 1. Tomography in venous phase and initial gastroduodenal overlay in patient 1. A and B. Aorto-mesenteric angle and distance with an approximate measurement of 13 and 3.3 mm, respectively. In figure B, duodenal proximal expansion of up to 3.8 cm is shown. C. Extrinsic compression of the third portion of the duodenum (red arrow) that restricts the threading movement of barium and proximal expansion of the duodenal bulb.

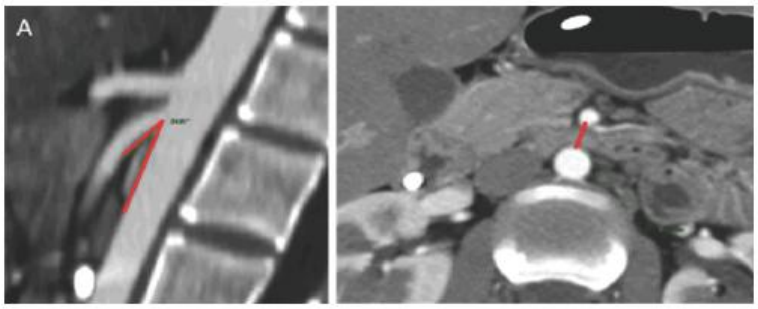

Figure 2. Follow-up computed tomography for patient 1. A and B. Increase in the aorto-mesenteric angle and distance from 23 and 7.4 mm, respectively (red lines).

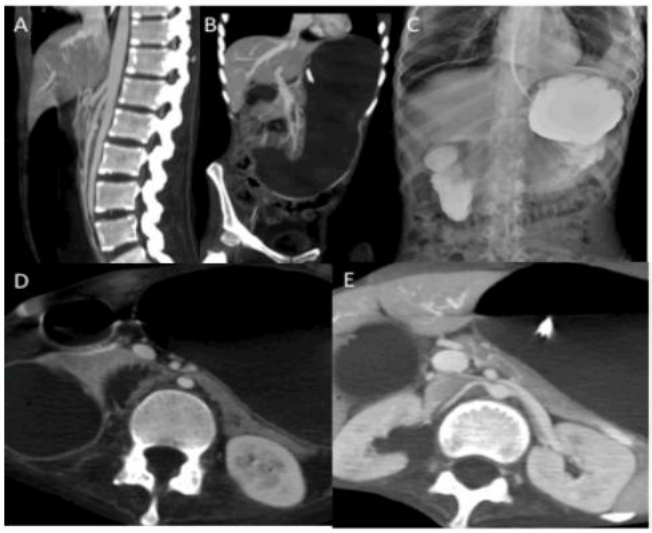

Figure 3. Maximum intensity multiplane reconstruction of CT in arterial and venous phase and esophagogastroduodenal overlay of patient 2. A. Sagittal reconstruction of CT in arterial phase where an aorto-mesenteric angle of 15° is observed. B. Coronal reconstruction showing gastric and proximal duodenal expansion. Esophagogastroduodenal overlay with retention of barium contrast on a gastric camera, first and second portion of the duodenum. D and E. Aorto-mesenteric distance of 4.5 mm that restricts duodenal expansion and compression of the left renal vein. The left kidney adequately concentrates contrast IV.