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Scientific Letters

Superior Mesenteric Artery Syndrome (Wilkie Syndrome): Analysis of a Series of 5 Cases☆

Síndrome de la pinza aorto-mesentérica (Sind. de Wilkie). Análisis de una serie de 5 casos

Superior mesenteric artery syndrome (SMAS) or Wilkie syndrome (WS) involves compression of the third part of the duodenum by the aortomesenteric angle, which can be caused by anatomical malformations or losses of perivascular adipose tissue after periods of hypercatabolism or malnutrition. Symptoms include postprandial pain, feeling of fullness, nausea with or without vomiting, and consequent weight loss. The initial treatment is conservative with hygienic-dietary measures and weight gain, although bypass surgery should be indicated in the event of failure.

We present a series of patients with SMAS who were treated surgically (Table 1).

Between 2009 and 2018, 7 patients were treated. In 5 (71.4%), duodenal derotation and anastomosis were performed (3 by duodenoduodenostomy and 2 by duodenojejunostomy), while duodenojejunostomy was performed in another 2 (28.5%) without derotation, and one of them laparoscopically.

All patients had a history of postprandial pain that improved after vomiting, as well as low body weight. The persistence of pain, difficult weight maintenance and risk of inadequate growth, despite dietary treatment, were indications for surgery.

Diagnostic testing was variable. In 85.7%, CT scan with oral contrast was performed, in 14.2% Doppler ultrasound was used, and another 14.2% underwent MRI. Barium contrast studies were used in 42.8% to confirm the suspected compression of the SMAS on the duodenal segment.

Out of the 5 patients who underwent duodenal derotation, 2 presented complications with the need for reoperation: one postoperative pancreatitis that required necrosectomy, cholecystectomy and choledochoduodenostomy; and one stenosis of the anastomosis on the tenth postoperative day due to perianastomotic fibrosis, treated with adhesiolysis and balloon dilation through the jejunostomy.

WS or SMAS is a rare syndrome[1] that is not often included in the differential diagnosis of recurrent abdominal pain. It should be considered in patients with malnutrition or hypercatabolism, postprandial abdominal pain and distension that characteristically improves with vomiting or reduced oral intake, as well as in food disorders that lead to low weight, like anorexia nervosa.[2,3]

The cause may be anatomical malformations[4] and adipose tissue loss that diminish the aortomesenteric angle. Other causes include postoperative complications that increase the pressure on the SMAS (postoperative weight loss, adhesions, scoliosis surgery, etc.).[5–7]

Conservative treatment with nutritional support to sustain weight gain can improve quality of life and avoid complications due to episodes of recurrent intestinal obstruction.

The radiological criteria for diagnosis have not been clearly established. However, in a suggestive clinical context, the presence of an aortomesenteric angle of less than 25° and/or a distance of less than 8 mm between the SMAS and the edge of the duodenum should lead us to suspect the possibility of a WS[4] (Fig. 1).

Nevertheless, there are false negatives, especially in asymptomatic or oligosymptomatic periods when it may be difficult to objectify duodenal compression or retrograde dilation.

Acute treatment consists of decompression using a nasogastric tube, placement of the patient in the left lateral decubitus position, fluid and electrolyte supplementation, and hypercaloric nutritional therapy, preferably enteral (oral or through a nasogastric tube).[8]

Given the failure of conservative treatment or clinical persistence, surgical treatment is indicated, and a duodeno-

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Table 1 – Patients Treated Surgically for SMAS.

Year	Sex	Age (Yrs)	Diagnosis	Technique	Complications
2009	Female	32	Doppler ultrasound Barium study	Duodenal derotation+duodenoduodenostomy E-E	Anastomotic stenosis
2009	Male	15	CT	Duodenal derotation+duodenojejunostomy S-S	–
2010	Female	20	CT Barium study	Duodenal derotation+duodenojejunostomy S-S	–
2010	Male	6	CT angiography Barium study	Duodenojejunostomy S-S	–
2012	Female	35	MRI CT	Duodenal derotation+duodenoduodenostomy E-E	–
2015	Female	15	CT	Duodenal derotation+duodenoduodenostomy E-E	Acute pancreatitis
2016	Female	24	CT	Laparoscopic duodenojejunostomy S-S	–

ECO: ultrasound; MRI: magnetic resonance imaging; SMAS: superior mesenteric artery syndrome; CT: computed tomography.

jejunostomy, gastrojejunostomy or division of the Treitz ligament can be performed to mobilize the duodenum (Strong technique[9]). The best results are obtained with duodenojejunal anastomosis,[3,4,10] as in our case series.

Laparoscopic duodenojejunostomy is an effective, minimally invasive treatment, with an acceptable rate of postoperative complications and favorable long-term results, which is why it is considered the treatment of choice.

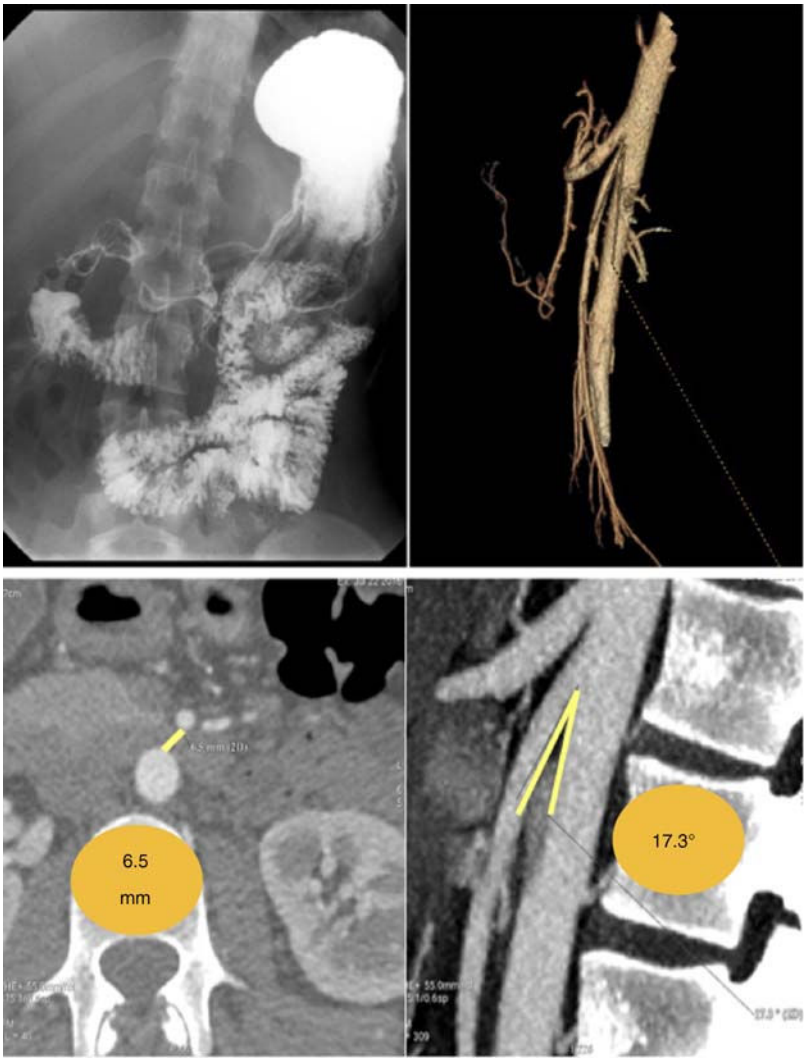

Fig. 1 – Intestinal tract, difficult passage of contrast material in the third part of the duodenum. Aortomesenteric angle (vascular, axial and sagittal reconstruction); diagnostic criteria for SMAS.

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Pulmonary Amyloidosis: A Diagnostic Challenge☆

Amiloidosis pulmonar: un desafío diagnóstico

Amyloidosis is a heterogeneous group of multisystem disorders, characterized by extracellular deposition of amyloid protein fibrils in organs and systems[1]; all show green birefringence under polarized light when stained with Congo red dye.[1,2]

The 2 most common types of amyloidosis are primary or immunoglobulin light chain amyloidosis and secondary or AA amyloidosis.[1] The etiology of the disease is unknown and symptoms are variable.[3]

The finding of a pulmonary mass corresponding with an amyloid tumor is uncommon. These are isolated lesions confused with malignant masses, both radiologically and metabolically, as on PET they can be hypermetabolic.[3] Surgery is diagnostic and therapeutic.[2]

The first case was a 60-year-old male whose medical history included surgery for a rectal neoplasm (pT3N0). During follow-up, two millimetric lesions were observed on thoracic CT: one in the right upper lobe measuring 8 mm, and another in the left upper lobe (LUL) measuring 6 mm, suggestive of metastases.

A PET/CT scan was performed, which determined that the nodules had a normal metabolic profile. Atypical resection of both pulmonary nodules was performed in 2 stages by video-assisted thoracoscopy. The pathology study reported nodules of amorphous material and positive Congo red, which was compatible with pulmonary amyloidoma.

The second case was a 77-year-old male, who consulted for left thoracic pain that had been progressing for one month with pleuritic characteristics. CT scan revealed a spiculated nodule with calcifications in the apical segment of the LUL (24 × 21 × 18 mm). Fiber-optic bronchoscopy and percutaneous core-needle biopsy did not

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