Superior Mesenteric Artery Syndrome – An Uncommon Complication After Surgical Corrections of Spinal Deformities

Síndrome da artéria mesentérica superior – Uma complicação incomum após correações cirúrgicas de deformidades da coluna

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Abstract

Vascular compression of the third part of the duodenum by the superior mesenteric artery results in an unusual occlusion of the duodenal transit known as superior mesenteric artery syndrome. This syndrome can occur after surgeries to correct spinal deformities in a rate ranging from 0.5% to 4.7%. It results from a positional alteration of the artery emergency point due to a change in trunk length after surgery. It is associated with risk factors such as low body mass index and weight loss. Patients usually present with intestinal occlusion, abdominal pain, nausea, bilious vomiting, and early satiety. Superior mesenteric artery syndrome must be recognized early to institute an adequate treatment, which can be clinical (with gastric tube for decompression and nutritional support) or require a surgical procedure. Secondary complications related to superior mesenteric artery syndrome include delayed surgical and nutritional recovery, healing problems, and prolonged hospitalization. The present study aims to report a case of superior mesenteric artery syndrome in a patient with neuromuscular scoliosis secondary to a transverse myelitis who underwent surgical treatment for spinal deformity correction.

Keywords

► superior mesenteric artery syndrome/complications
► intestinal obstruction
► spinal fusion
► scoliosis/surgery

Resumo

A compressão vascular da terceira parte do duodeno pela artéria mesentérica superior resulta no desenvolvimento de uma condição incomum de oclusão do trânsito duodenal conhecida como síndrome da artéria mesentérica superior. Este fenômeno pode acontecer após cirurgias de correção de deformidades da coluna, e sua taxa de
Superior Mesenteric Artery Syndrome

Araujo et al.

Introduction

Vascular compression of the third part of the duodenum by the superior mesenteric artery (SMA) results in an unusual condition of duodenal transit occlusion known as superior mesenteric artery syndrome (SMAS). Its etiology is linked to the anatomical relationship of the third part of the duodenum with the aortomesenteric angle. Obstruction is caused by an extrinsic compression created by the upper mesenteric neurovascular bundle and the base of the small intestinal mesentery root that intersect anteriorly to the duodenum.

The SMA exits the aorta at the L1 vertebral body level. It is surrounded by adipose and lymphatic tissue, forming a 45° to 60° angle with the aorta. The duodenum usually crosses the aorta at the L3 level, and it is suspended by the Treitz ligament. An imbalance between these structures can lead to SMAS

The incidence of SMAS after surgical procedures for spinal deformities correction ranges from 0.5 to 4.7%. Teenager, asthenic and tall patients are mostly affected. These patients are believed to have a lower amount of retroperitoneal fat, with no “fat pad” between the duodenum and the mesenteric artery.

Clinically, SMAS typically presents as abdominal pain, nausea, bilious vomiting, and early satiety. Its main differential diagnosis is paralytic ileus. Secondary SAMS-related complications include delayed surgical and nutritional recovery, healing problems, and prolonged hospitalization.

The present study aims to report a case of SMAS in a patient with neuromuscular scoliosis who underwent surgical treatment for spinal deformity correction.

Case Report

A 12-year-old male patient, admitted with viral transverse myelitis sequelae and diplegic sensory-motor impairment.

At follow-up, the patient presented with progressive neuromuscular scoliosis requiring surgical treatment. At the time, he had 63° scoliosis (T2 to L2) and 37° kyphosis at the thoracolumbar transition (T10 to L2). On admission, the patient had a body mass index (BMI) of 15.87 (thinness), and had lost 13 kg in the previous 2 years.

Posterior spinal T2-S1 arthrodesis was performed uneventfully, and the patient presented with progressive clinical improvement during the first postoperative days. On the 9th day after surgery, vomiting and fever started. On the 11th postoperative day, the patient presented with recurrent vomiting in addition to abdominal distension and loss of appetite. An abdominal tomography showed a large gastric distension extending to the third portion of the duodenum. An SMAS diagnosis was considered based on the anatomical region with reduced intestinal transit.

A nasogastric catheterization yielded an initial outflow of 1,200mL of bile-like fluid and partial relief of symptoms. However, the patient did not show any improvement in the following days, and a surgical intervention was indicated by the general surgery team. The patient underwent an upper digestive endoscopy, revealing a large amount of stasis fluid from the esophagus to the duodenum, in addition to a complete obstruction of the duodenal lumen in its third portion and a pulse attributed to the SMA, which compressed the duodenum. A supraumbilical median laparotomy was

Palavras-chave
- síndrome da artéria mesentérica superior/
- complicações
- obstrução intestinal
- fusão vertebral
- escoliose/cirurgia

Fig. 1 Illustration of the anatomical relationship of the third portion of the duodenum with the superior mesenteric artery and the obstruction observed in the superior mesenteric artery syndrome.
performed with a duodenojejunal shunt about 20 cm after the angle of Treitz (Figure 5). The postoperative evolution was satisfactory. Fluid intake was initiated on the 6th postoperative day, and the nasogastric tube was removed on the 8th day. On the 11th day, oral liquid feeding was started, slowly progressing to soft and solid foods. After 17 days, the patient was discharged from the hospital. At the first return visit, 7 days after discharge, he weighed 46.3 kg. After 4 months, he weighed 50 kg, with a normal intestinal rhythm and no food intolerance. At the 8th month, he was weighing 54 kg, totaling a weight gain of 7.7 kg, and did not present any gastrointestinal symptoms.

Discussion

Nausea and vomiting are frequent in patients undergoing spinal arthrodesis for scoliosis correction. These symptoms may result from analgesic medications such as opioids, or even may be due to paralytic ileus, which is common after major surgery. Paralytic ileus usually resolves within 5 to 7 days. The combination of surgical spinal deformity correction with previous weight loss increases the risk for SMAS. The symptoms of SMAS usually appear 6 to 8 days after surgery and, in contrast to paralytic ileus, often include air-fluid sounds. Later cases were also described, with onset of symptoms within 40 days.

Conservative treatment for SMAS consists of nasogastric decompression, nutritional support, and correction of fluid and electrolytic disorders. When tube drainage decreases to <100 mL in 8 hours, oral fluid administration can start, slowly progressing to soft foods in small amounts and more frequent feeds.

Most SMAS patients progress satisfactorily with conservative treatment. Children usually present a more favorable evolution than adults, with symptoms improvement in 2 to 3 days of gastric decompression. If the symptoms persist, surgical treatment must be considered, and the main options are gastrojejunostomy or duodenojejunostomy.

Superior mesenteric artery syndrome can occur both in surgical kyphoscoliosis correction and during the conservative treatment of deformities with plaster vests, when it is referred to as “plaster syndrome.” It is not clear why vest immobilization results in vascular compression of the duodenum. The first case of mesenteric vascular compression associated with a plaster vest was described by Willett, in 1878, using a Sayre plaster on a 17-year-old boy. Since then, new cases have been reported with different spinal deformities. Evarts et al. described 18 patients with SMAS during the treatment of spinal deformities. The authors warned for the need to suspect SMAS in symptomatic patients using plastered vests, Milwaukee-type vests, halo-femoral traction or submitted to surgical correction.

Altiok et al. conducted a retrospective study reviewing 2,939 records and found 17 SMAS cases. The authors evaluated the influence of vertebral translation and derotation maneuvers on SMAS incidence and concluded that the new correction procedures did not eliminate it. Spinal correction per se leads to its elongation, which changes the anatomical environment of the SMA.
Fig. 3  (a) Preoperative, lateral panoramic radiograph of the spine. (b) Postoperative radiograph.

Fig. 4  Coronal and axial computed tomography scans of the abdomen (white arrow – gastric distension; white asterisk – tapering of the third portion of the duodenum).
Braun et al. observed that patients with a BMI lower than the 25th percentile for age, those with a more rigid thoracic curve (< 60% correction on side-bending radiographs) and those with a laterally displaced lumbar curve (lumbar modifier of the Lenke classification, type B or C) are at greater risk for SMAS development after deformity correction.

Carelli et al. reported two cases of SMAS after sagittal spinal deformities correction, in which the conservative treatment of the intestinal occlusion was successfully for the resolution of symptoms. The authors believe that the incidence of SMAS must be higher than what is described, with most cases presenting spontaneous resolution.

Superior mesenteric artery syndrome, although uncommon, is a complication of spinal deformities correction. It is important that the medical team be aware of the syndrome and have a high degree of suspicion in patients with gastrointestinal symptoms to avoid delays in diagnosis and treatment that could result in unfavorable outcomes.

Note
Study developed at the Orthopedics Department, Rede Sarah de Hospitais de Reabilitação, Brasília, DF, Brazil.

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Conflict of Interests
The authors have no conflict of interests to declare.

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