LAPAROSCOPIC TREATMENT OF CELIAC AXIS COMPRESSION SYNDROME: CASE REPORT

Tratamento laparoscópico da síndrome de compressão do tronco celiaco: relato de caso

Júlio Cezar Uili COELHO, Jean Carlos da SILVA, Micheli Fortunato DOMINGOS, João Augusto Nocera PAULIN, Guilherme Figueiró FERRONATO

From the Hospital Nossa Senhora das Graças (Nossa Senhora das Graças Hospital), Curitiba, Paraná, Brazil

Correspondence:
Julio Coelho
E-mail: coelhjcu@yahoo.com.br

INTRODUCTION

Celiac axis compression syndrome, also known as median arcuate ligament syndrome or Dunbar syndrome, is a rare condition. This syndrome was first reported by Harjola in 1963. Dunbar described it as a clinical syndrome in his memorial paper in 1965. It is characterized by compression of the celiac axis by the median arcuate ligament of the diaphragm.

The median arcuate ligament is a fibrous arch formed at the base of the diaphragm at the level of the 12th thoracic vertebra, where the left and right diaphragmatic crura join. This fibrous arch forms the anterior aspect of the aortic hiatus, through which the aorta, thoracic duct, andazygous vein pass. The median arcuate ligament usually comes into contact with the aorta above the origin of the celiac axis. However, in some individuals, the aorta may be abnormally low and passes in front of the celiac axis, causing its compression, which is named median arcuate ligament syndrome.

Some patients with this syndrome refer severe clinical manifestations such as postprandial abdominal pain, weight loss, and vomiting. The primary treatment modality for this condition is surgical division of its fibers. The traditional surgical approach has been through an upper abdominal laparotomy incision. Roayaie et al. in 2000 reported the first patient with celiac axis compression syndrome treated by laparoscopy access. Afterwards, several authors have demonstrated that the laparoscopic access may be employed with success to treat this condition. To best of our knowledge, this is the first report of laparoscopic treatment of the celiac axis compression syndrome in Brazil.

CASE REPORT

A 60-year-old woman presented with a three-year history of intermittent postprandial epigastric pain, and weight loss of 6 kg. The abdominal pain was relieved with fasting. She denied nausea, vomiting and diarrhea. Physical examination was normal. Several exams, including abdominal ultrasonography, upper gastrointestinal endoscopy, colonoscopy, small bowel radiographic study, tomography failed to reveal any abnormality. Finally, an angiotomography showed high-grade stenosis of the anterior wall of the proximal celiac axis caused by extrinsic compression of the median arcuate ligament (Figure 1A).

The patient underwent laparoscopic section of the ligament and celiac ganglionectomy. The patient was placed in reverse Trendelenburg position with the legs abducted and supported on cushioned spreader bars. The operation was performed through five trocars inserted in the upper abdomen, similar to that of Nissen-Rossetti procedure. A right subcostal retractor was used to retract the left lobe of the liver laterally and the stomach was retracted to the patient’s left side with a Babcock clamp. After dividing the gastrohepatic omentum and identifying the right crus of the diaphragm inferiorly to the cardia, the junction of both crus was carefully separated to expose the anterior surface of the aorta and identify the median arcuate ligament and celiac plexus. The median arcuate ligament that was compressing the proximal celiac axis was sectioned and all neural tissue overlying the celiac axis was resected. The operation was uneventful and lasted 70 min. The patient was discharged from the hospital 12 h after the operation completion and had an uneventful recovery. At two-month follow-up, she referred only two episodes of mild abdominal pain and gained 3 kg. An angiotomography obtained at that time showed no celiac axis stenosis (Figure 1B).

DISCUSSION

Since the first report of the celiac axis compression syndrome several decades ago, controversy still remains regarding the pathophysiology and clinical implications of this condition. The observation of celiac axis compression in asymptomatic patients leads to questions about the real existence of the syndrome. Some authors suggested that the clinical manifestations are caused by ischemia secondary to the reduction of blood flow through the stenotic celiac axis. However, others claimed that pain originates from direct compression of celiac ganglia.

In the past, celiac axis compression syndrome was diagnosed by conventional angiography. Lateral projection of aortography was the first choice to identify the celiac axis stricture. Nowadays thin-section multidetector CT scanners, associated with three-dimensional reconstruction, have become the best method to obtain high-resolution images of the aorta and its branches. Angiotomography, especially during expiration, has a high precision to identify celiac axis compression syndrome. In addition, this method also allows visualization not only of the stenosed vessel but also the underlying median arcuate ligament and adherent tissue using three-dimensional imaging. Angiotomography is also important to exclude the presence of celiac axis calcifications, an important cause of arterial stricture.

The angiotomography of this patient showed a severe stricture of the celiac axis caused by extrinsic compression of the median arcuate ligament. The stricture was successfully treated by laparoscopic section of the median arcuate ligament. Postoperative angiotomography demonstrated absence of residual stenosis of
REFERENCES


MC’S, man, 32, with a history of progressive and painless increased abdominal size four weeks ago, no other complaints. He had also several congenital malformations including: pectus excavatum, congenital dislocation of the hip and clubfoot (operated in childhood). Physical examination revealed ascites, bilateral pleural effusion and a hardened mass located in flank and the right iliac fossa of about 20 cm. Was detected the absence of the right testicle in scrotum. No abnormal laboratory tests were present. Computed tomography and magnetic resonance imaging of the abdomen and pelvis showed ascites and heterogeneous pelvic mass (Figure 1). The cytological study of ascites and pleural effusion showed no neoplastic cells.

MC’S was subjected to four cycles of chemotherapy with bleomycin, etoposide and cisplatin, keeping normalization of markers and improvement of ascites. He is currently with eight years of evolution, and in that period maintained regular outpatient follow-up showing no measurable disease to blood tests and imaging.

FIGURE 2 - Tumor mass with smooth outer surface, opaque, sometimes lobed, with brown bleeding areas in A. When cut, in B, whitish, firm and elastic tumor, permeated by areas of bleeding and yellowish and softened areas.

The patient was discharged on the sixth day after surgery. The left testicle was evaluated and was normal. The staging was completed and once considered the patient in stage III (ascites), was subjected to four cycles of chemotherapy with bleomycin, etoposide and cisplatin, keeping normalization of markers and improvement of ascites. He is currently with eight years of evolution, and in that period maintained regular outpatient follow-up showing no measurable disease to blood tests and imaging.