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Laparoscopic treatment of celiac axis compression syndrome (CACS) and hiatal hernia: Case report with bleeding complications and review

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ABSTRACT

INTRODUCTION: Median arcuate ligament (MAL) malposition is a rare cause of celiac axis compression syndrome (CACS) or Dunbar syndrome.
PRESENTATION OF CASE: A 26-year-old female presented with severe postprandial epigastric pain, weight loss, heartburn and regurgitation unresponsive to medical therapy. CT angiography and duplex ultrasound demonstrated the MAL crossing anterior to the celiac artery (CA). Reconstructions demonstrated CA compression, while the superior mesenteric artery (SMA) was normal. The MAL was laparoscopically divided, releasing the celiac axis. A concomitant Nissen fundoplication was performed. At 3-months follow-up, the CT-scan demonstrated no evidence of CACS with complete symptom resolution.
DISCUSSION: Dunbar's syndrome can be treated with endovascular surgery, laparoscopic MAL division or vascular surgery.Six anatomical and morphologic variations of aortic and esophageal hiatus are described. The result of the analysis of these anatomical data leads to the conclusion that hiatus hernia, Dunbar's syndrome and GERD have a common etiopathogenesis and physiopathology.
CONCLUSION: Laparoscopic treatment is useful and feasible in centers with experience in majorlaparoscopic surgery with reduced invasiveness, better cosmetic effect and shorter postoperative course.

1. Introduction

Celiac artery compression syndrome (CACS) was first described by Harjola.[1] In 1965, Dunbar reported the first surgically treated patients.[2] Several pathogenetic hypotheses have been proposed. The origin of the celiac artery may be abnormally high, leading to compression by an anatomically normal median arcuate ligament (MAL). Alternatively, an exceedingly long MAL could compress a normal celiac artery. The blood steal hypothesis suggests that large collateral vessels can and will acquire a greater percentage of total blood flow postprandially, depriving smaller vessels of the flow they require.[3] The last hypothesis stems on a neurogenic cause. There is a wide range of variation regarding the location, morphology, and neural interconnections of the plexus.[4] Reports in the literature have shown that this plexus, when fibrotic, can restrict blood flow to the celiac artery.[5,6] The pathology is more common in young women with low BMI, with a clinical history of chronic postprandial abdominal pain, diarrhea, vomiting, epigastric bruit and weight loss. CACS diagnosis is radiological in all patients by digital subtraction angiogram, computed tomogram scan, MRI or duplex ultrasound.[7] Computed tomography (CT) angiography allows visualization not only of the stenosed vessel but also the underlying median arcuate ligament and adherent tissue using 3D imaging. Inspiratory and expiratory MRI may discriminate between normal variations of the celiac trunk or pathological narrowing of the proximal celiac segment.[8]

2. Case report

A 26-year old, female patient with a body mass index of 21 complained of 8-kg weight loss in a year associated with post-prandial abdominal pain. The pain which was predominantly localized to the upper and mid-abdomen; there was no evidence of organic or functional pathology. Color-flow duplex sonography showed an "ascending" course of the celiac trunk, with reduction of the ostial

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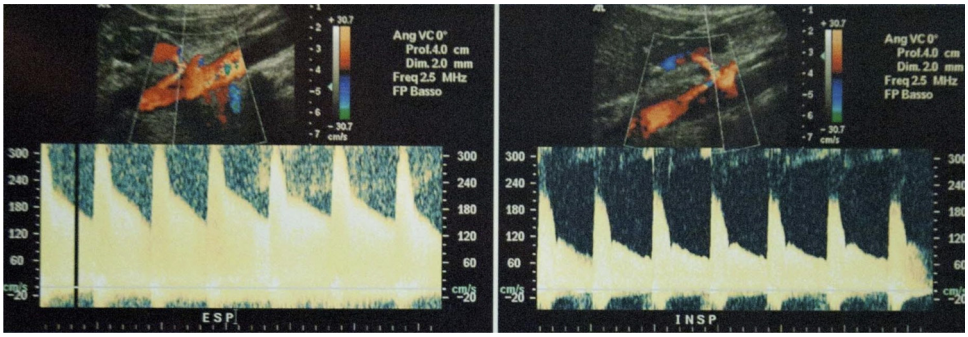

Fig. 1. Preoperative Eco-color-doppler of aorta and celiac trunk preoperative during inspiration and expiration.

segment's caliber, in expiration, and mild post-stenotic ectasia with systo/diastolic blood flow acceleration (>300 cm/s). These data were consistent with a significant stenosis (Fig. 1). There was also evidence of an increase of the systolic-peak in the initial portion of the superior mesenteric artery (200 cm/s). CT angiography confirmed the diagnosis of Dunbar's syndrome, showing that the celiac trunk was constricted during expiration (Fig. 2). Her symptoms were also consistent with a degree of gastro-oesophageal reflux disease confirmed on oesophageal pH studies. Given the clinical presentation the patient was offered laparoscopic MAL division and a concomitant Nissen–Rossetti fundoplication.

Following pneumoperitoneum induction, four ports were inserted (one 10 mm port for the camera, three 5 mm operating ports). The pars flaccida of the lesser omentum was opened and the right crus of the diaphragm was isolated. The MAL superficial fibers were identified. The common hepatic artery and the left gastric artery were identified and followed to the origin of the celiac trunk. The MAL fibers were disected and divided and the celiac trunk was skeletonized. During skeletonization, a small puncture occurred in the proximal celiac trunk. An additional 5 mm port was placed in the left periumbilical space. The bleeding was controlled with a combination of a haemostatic mixture (Human Trombin + Gelatin Matrix) and one polypropylene suture. A routine Nissen[19] fundoplication was performed. Her postoperative course was unremarkable. Abdominal CT with and without contrast and duplex ultrasound were performed on the sixth postoperative day and were repeated three months post-operatively. There was a clear and progressive widening of the aorto-celiac angle (Fig. 2). Her postprandial cramping pain disappeared and she had gained 2 kg in weight three months after the operation.

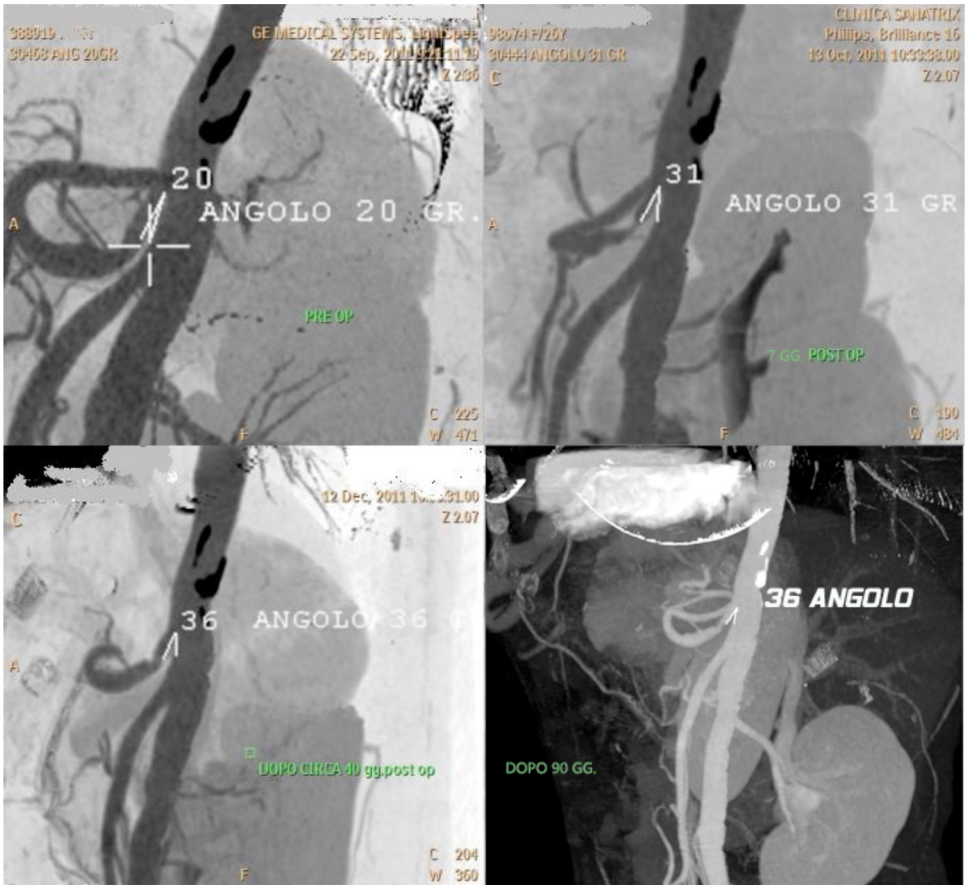

Fig. 2. AngioTc showing the preoperative angle formed by aorta and celiac trunk and its postoperative increase during follow up. *Abbreviations*: MAL, medium arcuate ligament; CACS, celiac artery compression syndrome; GERD, gastroesophageal reflux disease.