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CASE REPORT

Celiacomesenteric Trunk as a Cause of Median Arcuate Ligament Syndrome

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A 28-year-old man presented with a constellation of symptoms with 6 months of duration, most prominent being unexplained weight loss of 35 pounds and postprandial abdominal discomfort. Multiple specialist consultations, laboratory and instrumental studies did not yield a unifying diagnosis. The patient had been treated with multiple medications without improvements. On examination, an epigastric bruit that increased with expiration and decreased with inspiration raised suspicion for median arcuate ligament syndrome (MALS). Ultrasonography of the mesenteric arteries revealed a rare anatomical variant of abdominal vessels known as celiacomesenteric trunk (the celiac artery and superior mesenteric artery sharing a common origin from the aorta). There was also a median arcuate ligament impression on the celiac artery and marked respiratory variations of Doppler velocities in support of the diagnosis of MALS. This is a case report of celiacomesenteric trunk clinically associated with MALS.

Introduction

Median arcuate ligament syndrome (MALS), or celiac axis compression syndrome, is a rare condition, typically presenting as chronic postprandial abdominal pain, weight loss, and epigastric bruit [1,2]. Pathophysiology is generally explained by either compression of the splanchnic nerve plexus causing neurogenic pain, or compression of the proximal celiac trunk by the median arcuate ligament (ventral arch of aortic hiatus formed by tendinous fibers between both diaphragmatic cruras) causing chronic mesenteric ischemia [1,3].

We present an illustrative case of a patient with MALS who underwent exhaustive evaluation before the diagnosis of MALS was entertained. In this patient, a rare anatomical variant of abdominal vessels known as celiacomesenteric trunk (where the celiac artery and superior mesenteric

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artery share a common origin from the aorta) probably accounted for the clinical symptoms. Both celiacomesenteric trunk and MALS are rare entities and this is one of the very first cases of their association to be reported.

Case report

A 28-year-old man presented with a chief complaint of unexplained weight loss of 35 pounds within 6 months (his body mass index decreased from 20 to 15). His symptoms included postprandial abdominal pain, early satiety, regurgitation, nausea, and generalized fatigue without an unifying diagnosis. Multiple consultations prior to and during this admission included gastrointestinal, nutrition, endocrine, medical genetics, physical medicine and rehabilitation, neurology, ophthalmology, psychiatry, and vascular surgery specialists. There was no evidence of a psychiatric illness or eating disorder. He had been treated with multiple medications, including proton-pump inhibitors, antidepressants, anxiolytics, androgens, and parenteral supplements without improvement.

Gastroduodenal manometry, gastric emptying and accommodation studies, malabsorption studies, and repeat esophagogastroduodenoscopy with biopsies were performed. Imaging included computed tomography (CT) scans of the chest, abdomen, and pelvis, hepatobiliary scintigraphy. Findings were unremarkable except for mild chronic gastritis. Other unrevealing tests included serum levels of vitamins and iron; toxicology and allergy panels; autonomic nervous system dysfunction tests and nerve conduction studies; paraneoplastic panel; and genetic tests for inborn errors of metabolism and mitochondrial disorders, including mitochondrial neurogastrointestinal encephalopathy disease.

During admission at our institution, on examination we noticed an epigastric bruit that increased with expiration and decreased with inspiration. Otherwise, except for body mass index, the physical examination was unremarkable. There were no signs of elemental deficiencies and no stigmata of eating disorders, endocrine abnormalities, or heritable diseases.

The finding of an epigastric bruit on examination prompted a concern that the patient might have MALS. Duplex ultrasonography of the mesenteric arteries with color Doppler was performed. It revealed a likely common origin of the celiac artery and the superior mesenteric artery from the aorta (Figs. 1 and 2). The celiac artery was seen with a degree of median arcuate ligament impression; peak systolic velocity increased to 236 cm/s during expiration from a normal velocity of 152 cm/s during inspiration (Figs. 3 and 4). The velocity during expiration was approximately the institutional cut-off (250 cm/s) for 70% stenosis. However, the celiac and the superior mesenteric arteries were seen to have a common origin. This finding is the least frequently reported anatomic variation of all abdominal vascular anomalies, known as celiacomesenteric trunk [4]. This anatomic configuration likely accounted for a flow compromise in both abdominal vessels with resultant clinical symptoms. The patient declined confirmatory CT angiography.

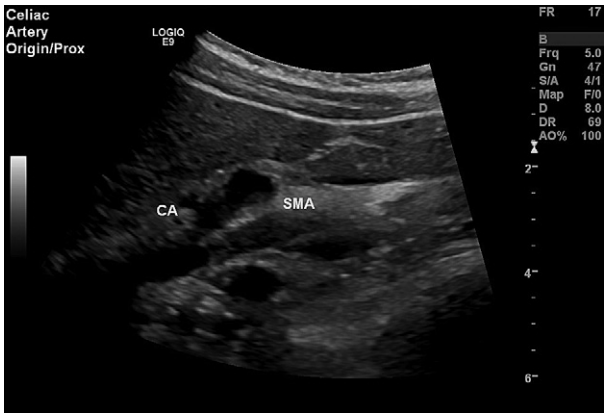

Fig. 1 Grayscale image of the common origin of the celiac and superior mesenteric arteries from the aorta (celiacomesenteric trunk).

The patient's condition stabilized with supportive treatment with a well-balanced diet administered in small frequent meals. He slowly began regaining weight. Vascular surgeons did not recommend surgical treatment at the time of their consultation, favoring continuous supportive treatment and a plan for further imaging with possible intervention if the symptoms persisted. Three months after discharge, the patient continued to improve gradually with the prescribed interventions and regained several kilograms of weight.

Discussion

Since its first description by Harjola in 1963, MALS, or celiac axis compression syndrome, has remained an incompletely understood, often illusive diagnosis [1,5]. The fact that imaging evidence of dynamic celiac artery compression during expiration does not consistently predict resolution of symptoms after surgical release or celiac artery revascularization often leads to hesitance to intervene invasively. It has also raised questions regarding the very existence of this clinical entity and its pathophysiology [3].

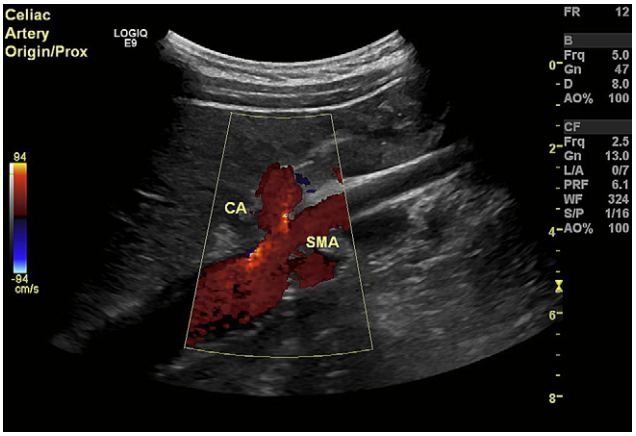

Fig. 2 Color duplex of the common origin of the celiac and superior mesenteric arteries from the aorta (celiacomesenteric trunk).