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Laparoscopic management of median arcuate ligament syndrome: A case report

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Abstract
Median Arcuate Ligament syndrome is a rare disorder usually occurring in female with thin body habitus in the 4th or 5th decade. The classical triad consists of postprandial abdominal pain, epigastric bruit, presence of extrinsic celiac artery compression revealed by vascular imaging and usually diagnosed very late after years. Here we report a 60 year old male patient who had come with pain in abdomen for the past 2 years with vomiting for the past 1 month and has been investigated in multiple centres for his pain in abdomen and finally in recent Contrast enhanced CT abdomen he was found to have Median Arcuate Ligament Syndrome. Patient was managed surgically by Laparoscopic Median Arcuate ligament release. Patient had an uneventful recovery.

Keywords: median arcuate ligament syndrome, celiac artery compression, laparoscopic median arcuate ligament release

Introduction

Median arcuate ligament syndrome (MALS) or Dunbar syndrome is a rare clinical entity characterized by celiac trunk compression by median arcuate ligament and variable gastrointestinal symptoms (postprandial epigastric pain, nausea, weight loss, anorexia and diarrhoea [1]. However, some degree of radiographic compression is observed in 10%-24% of asymptomatic patients. Besides the extrinsic vascular compression, MALS has a multifactorial etiology and it has been suggested as a neurogenic disease resulting in altered sensation and pain from the somatic nerves in the splanchnic plexus. MALS is a diagnosis of exclusion, so other causes must be excluded. Currently, no group consensus agreement as to the diagnostic criteria for MALS exists; duplex ultrasound, angiography, and gastric exercise tonometry are used in different combinations and with varying diagnostic values throughout the literature [2].

Case report

A 60 year old male came with chief complains of pain in abdomen for 2 years, insidious in onset, gradually progressive which was aggravated post prandially, Vomiting multiple episodes for the past 1 month containing food and history of loss of weight where the patient had significant loosening of his garments. The patient was evaluated in multiple centres and had undergone multiple radiological investigations and was referred to our centre. The patient did not have any comorbidities and had no past surgical history as well. He also denied any form of addiction. On per abdomen examination, the abdomen was soft and on auscultation Epigastric Bruit was heard.

Ultrasound abdomen was done along with doppler which showed Focal narrowing of the celiac trunk at the origin along with post-stenotic dilatation(stenosed segment diameter-3.6mm,Post stenotic diameter -11.7mm).Post Systolic Velocity of post stenotic dilated segment was raised-121cm/sec. Then CT Abdomen Angiography was done Focal Narrowing (60-70%) of the superior aspect of the proximal celiac trunk forming a hooked or **J** appearance along with post stenotic dilatation (stenosed segment diameter-3mm, Post stenotic Diameter-11mm). There was no evidence of Collateral Formation or associated atherosclerotic changes (Figure 1).

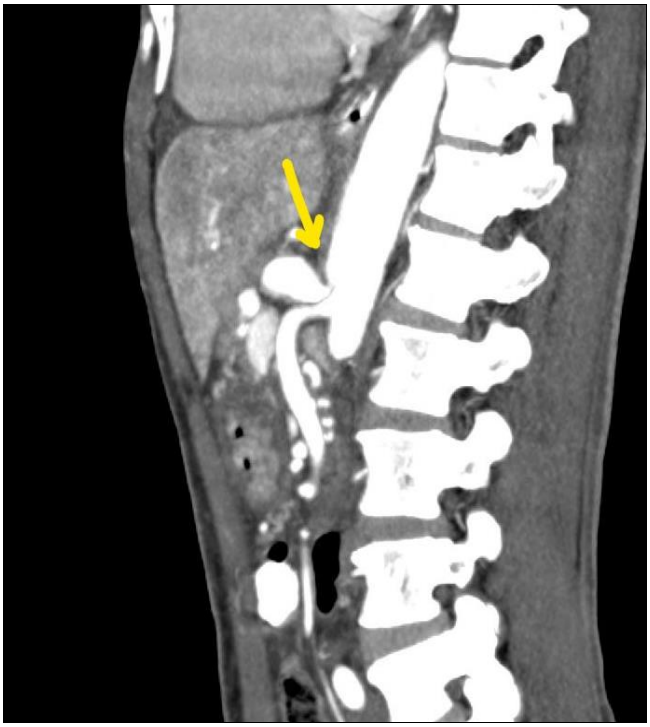

Fig 1: CT abdomen Angiography showing focal Narrowing (60-70%) of the superior aspect of the proximal celiac trunk

Our Patient was operated for Laparoscopic release of Median Arcuate Ligament. Patient was given a Head up Position and Port Placements are as shown below (Fig 2).

Fig 2: Port Placement for Laparoscopic Median Arcuate Ligament Release

Lesser sac was entered by dissecting along the Greater Omentum. Left gastric Artery and Vein were identified. The Left Gastric artery delineated completely to reach the Celiac Artery. All the branches from the Celiac Trunk was identified (Figure 3). Bands were found at the origin of Celiac Trunk. These bands were released using Harmonic Scalpel and Celiac Artery was skeletonised till its origin from Abdominal Aorta. Drain was placed and all the Ports removed.

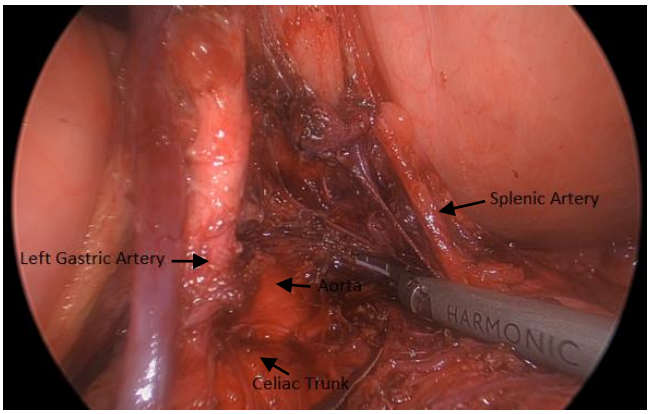

Fig 3: Celiac Trunk with its branches with the Band of adhesions around the vessel and abdominal aorta

Post Operatively Patient had decrease in the intensity of pain. Postoperative repeat Ultrasonography was done which did not show any significant change in the diameter and velocity of blood in the stenosed segment. Patient was followed up after 3 months when he had complete resolution of symptoms and repeat Ultrasonography showed increase in the diameter of the celiac trunk and also significant decrease in the peak velocity.Patient also showed some weight gain of around 3kgs after 3 months.

Discussion

The median arcuate ligament is a fibrous arch that unites the diaphragmatic crura on either side of the aortic hiatus [3]. The ligament usually passes superior to the origin of the celiac axis. However, in some people, the ligament inserts low and thus crosses the proximal portion of the celiac axis, causing compression and sometimes resulting in abdominal pain. The diagnosis of clinically significant celiac axis compression, referred to as median arcuate ligament syndrome, is traditionally made with conventional angiography

The MALS is a rare vascular disorder with an incidence of about 2 cases per 100,000 patients, with diffused and non-specific abdominal pain. In literature, it was found that the incidence of MALS in asymptomatic patients ranges between 7.3 and 27%.[4]. Such high incidence in asymptomatic patients could raise the issue whether surgical release of the compression would relieve the symptoms of non-specific abdominal pain, which could be related to other causes. It is known that many patients have minimal asymptomatic celiac trunk compression; hence, it is of major importance to discriminate those having a pathological compression [5]. The first anatomic description of MALS dates back to 1917, given by Lipshutz,[6] but only in 1965 the radiologist J.D. Dunbar reported the first case series focusing on diagnosis and treatment.[7] This syndrome is due to the celiac trunk compression by the median arcuate ligament in association with ganglionic periaortic tissue.[8] Although 47 years have elapsed, the pathogenesis of MALS is still uncertain. Some authors support the theory based on a higher origin of the celiac trunk from aorta, others maintain the exuberant growth of neurofibrous tissue originating from the celiac plexus causing compression.[9] The symptoms are the typical postprandial pain associated with nausea, vomiting, and unintentional weight loss.[10] The pathophysiology of MALS is still unknown. The first hypothesis is based on mesenteric ischemia even if the superior and inferior mesenteric arteries are normal. It could be due to postprandial “theft” of blood, which causes pain. The second, instead, is the consequence of direct or indirect overstimulation of the celiac plexus, caused by chronic inflammation, resulting in splanchnic vasoconstriction with ischemia.[5,10] Despite several tests, the diagnosis is based on the exclusion of other abdominal diseases. Lateral aortic angiography is the gold standard but there are other less invasive techniques such as US-Doppler, CT or MRA. In every case it is important to correlate abdominal symptoms with radiological data [10]

Most of the patients had extensive workup done for abdominal pain like Ultrasonography abdomen, endoscopy, colonoscopy, motility studies, CT, MRI and others have undergone previous surgery (appendectomy, cholecystectomy, vagotomy, and pyloroplasty), without relief [11].

Typically a diagnosis of exclusion, MAL syndrome involves a vague constellation of symptoms including epigastric pain, postprandial pain, nausea, vomiting, and weight loss which is due to foregut ischemia [12].The most common symptom the patient presents with is the epigastric pain. Extrinsic compression of the vasculature and surrounding neural

ganglion has been implicated as the cause of these symptoms. Multiple imaging techniques can be used to demonstrate celiac artery compression by the MAL including mesenteric duplex ultrasonography, computed tomography angiography, magnetic resonance angiography, gastric tonometry, and mesenteric arteriography.

Focal narrowing of the proximal celiac artery, a characteristic hooked appearance of the narrowed segment without calcification and atherosclerotic changes are diagnostic of MAL syndrome. Poststenotic dilatation are also typically seen [3]. Post stenotic dilatation is probably to the increased velocity and the force through which the blood exits the narrow segment.

Surgical intervention involves open, laparoscopic, or robotic ligament release; celiac ganglionectomy; and celiac artery revascularization [12]. There remains a limited role for angioplasty because this intervention does not address the underlying extrinsic compression resulting in symptoms, although angioplasty with stenting may be used in recalcitrant cases.

Usually the surgical procedure is uneventful. The postoperative complication that usually occur is urinary retention in a male. At three-month follow-up, usually the patients are asymptomatic.

The role of interventional radiology is limited to angioplasty and stenting to open the stenosis rather than addressing the underlying compression of celiac trunk which has resulted in the symptoms [13]

Conclusion

So MALS is a rare disorder but should be always kept in mind as a diagnosis of exclusion. Classical thin built patients with epigastric pain, nausea, vomiting and weight loss should be evaluated for MALS before planning for any intervention so that unnecessary intervention can be avoided in such patients. In our case there was no immediate postoperative relief but it took 3 months for the dynamics to change to a normal physiological one. After 3 months the patient had complete relief of symptoms. It is just a single case report study so nonetheless,further studies with large numbers and longer follow up duration are recommended to ascertain the conclusion.

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