



Case Report

## MALS as an Unusual Cause of Chronic Abdominal Pain: Case Report

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### ABSTRACT

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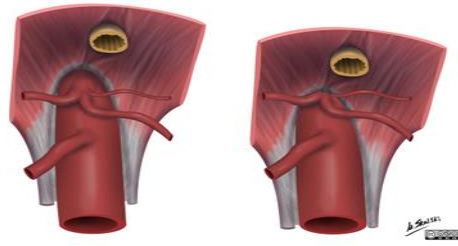
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Median arcuate ligament syndrome (MALS) is a rare cause of chronic mesenteric ischaemia (CMI) which is caused by extrinsic compression of the celiac axis by the fibrous attachments of the diaphragmatic crura, specifically the median arcuate ligament. Despite advances in diagnostic modalities, MALS continues to be difficult to qualify, quantify, and diagnose. MALS is typically a diagnosis of exclusion and presents with a constellation of symptoms such as epigastric pain, postprandial pain, nausea, vomiting, weight loss, and “food fear”. The pathophysiologic mechanism remains poorly understood, and symptom severity and treatment response are highly variable, contributing to ongoing controversy regarding the diagnosis. Anatomically, despite having up to 24% of the population with MAL compression, only up to 1% are symptomatic. This report presents a case of MALS, emphasising the clinical presentation, diagnostic evaluation, and management.

**Keywords:** MALS, Dunbar’s syndrome, vascular compression

### INTRODUCTION

MALS, also referred to as celiac artery compression syndrome or Dunbar’s syndrome, results from an abnormally low insertion of the diaphragm or an abnormally high origin of the celiac artery from the aorta (Fig. 1) (1). Compression may also be exacerbated by fibers of the celiac ganglion. Although the exact prevalence of MALS is uncertain, it is estimated to occur in approximately 2 per 100,000 individuals annually, with a higher incidence in females (4:1 ratio), most commonly affecting those aged 30 to 50 years (2). Diagnosis is typically by exclusion, following extensive evaluation for alternative causes, including upper endoscopy, abdominal ultrasound, and abdominal computed tomography. Gradually, the etiology has shifted from a vascular disease to a neurogenic illness with compression of the surrounding celiac plexus and ganglion. Management focuses on relieving celiac artery compression to restore blood flow and performing neurolysis to address chronic pain.

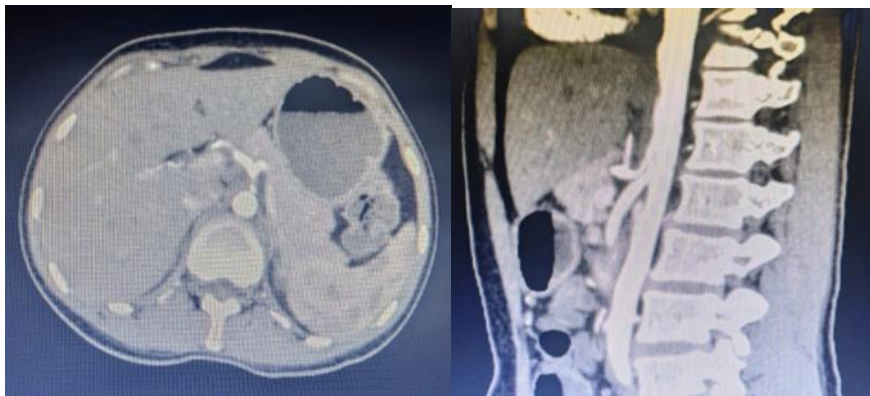


**Fig 1 - Anatomy of Median Arcuate Ligament Syndrome**

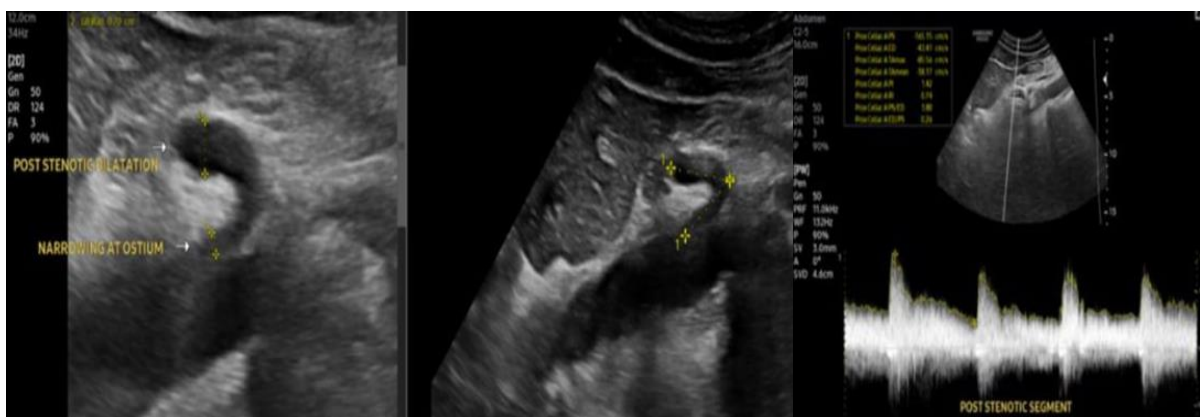
### CASE REPORT

A 26-year-old tall, thin female (BMI 18 kg/m<sup>2</sup>) with no significant past medical history presented with upper abdominal pain and progressive weight loss. The pain was accompanied by nausea, non-bilious vomiting, and bloating, and was exacerbated by the consumption of fatty foods. Nausea worsened with any oral intake and improved with bowel rest. The patient reported reduced food intake due to her symptoms and recurrent episodes of nausea and vomiting. Dietary modifications did not alleviate her symptoms. She was not taking any medications, had no allergies, and denied smoking or heavy alcohol consumption. She had previously been managed as a case of acid peptic disease and had undergone extensive evaluation elsewhere, including ECG, chest X-ray, abdominal ultrasound, and upper gastrointestinal endoscopy. Physical examination was unremarkable except epigastric bruit, more prominent on expiration. All laboratory results were within normal limits.

Contrast-enhanced computed tomography (CECT) of the abdomen demonstrated thickening of the median arcuate ligament and diaphragmatic crura abutting the abdominal aorta and celiac trunk, with a characteristic J-shaped or hooked configuration, mild stenosis, and post-stenotic dilatation (Fig. 2). Dynamic respiratory phase Doppler of the celiac artery revealed increased velocity (Fig. 3).

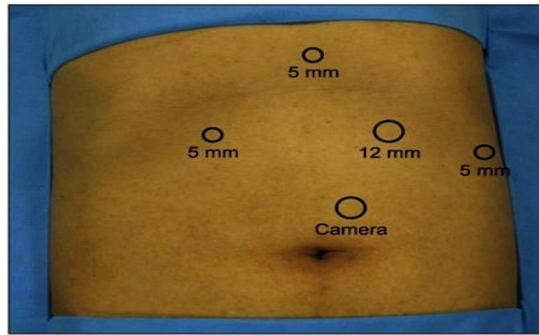


**Fig 2 - Contrast-enhanced computed tomography (CECT) of the abdomen**



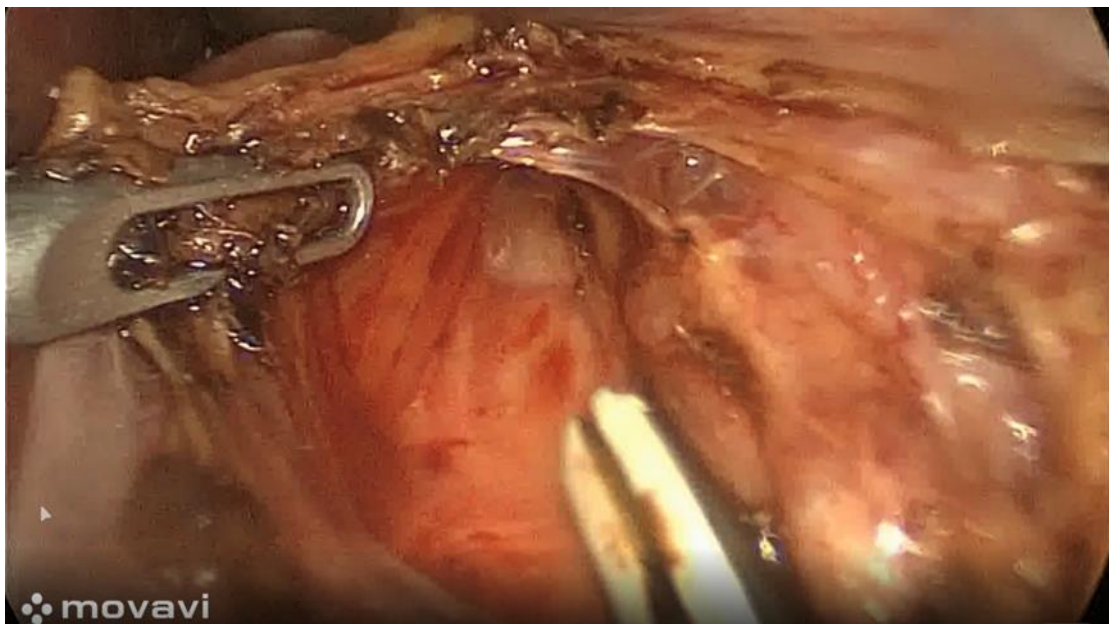
**Fig 3 - Dynamic respiratory phase Doppler of the celiac artery**

The diagnosis of MALS was established based on the patient's clinical history, radiological findings with pressure measurements, and exclusion of other pathologies. Laparoscopic release of the median arcuate ligament was recommended and performed under general anaesthesia following informed consent.



**Fig 4 - Ports position**

During surgery, laparoscopic ports were placed in a manner similar to that used for Nissen fundoplication (Fig 4). The right diaphragmatic crus was identified and divided to expose the anterior surface of the aorta. Dissection proceeded inferiorly along the aorta until the origin of the celiac trunk was visualised. The fibrous bands of the median arcuate ligament and elements of the celiac plexus were divided circumferentially (Fig. 5). The celiac artery was Completely skeletonized, thereby relieving extrinsic compression (Fig. 6).



**Fig 5 - Division of fibrous bands of Median Arcuate Ligament**



**Fig 6 - Skeletonized Celiac Artery**

The operative time was 210 minutes, and blood loss was minimal. The patient was discharged on the fifth postoperative day, able to tolerate orally, and was symptom-free. At follow-up, she experienced complete resolution of symptoms and regained weight.

## DISCUSSION

Anatomically, MALS was first described by Lipshutz in 1917 during cadaveric dissections, in which the celiac artery was sometimes overlapped by the diaphragmatic crura (3). Clinical resolution of postprandial epigastric pain and epigastric bruit following operative decompression of the celiac artery from a fibrosed celiac ganglion was published by Harjola in 1963 (4). Dunbar et al reported a case series involving surgical treatment of MAL syndrome in 1965(5) The stenosis of the celiac trunk occurs either in a too cranial emergence of the celiac artery from the aorta, or in a too caudal insertion of the left crus of the diaphragm on the lumbar vertebral column. 10% to 24% of the general population, the MAL crosses the aorta at a lower level and subsequently compresses the celiac artery (6), (7). However, it is clinically significant in only a small subset of patients, contributing to the controversy surrounding MAL syndrome as a pathologic entity.

In patients affected by median arcuate ligament syndrome, celiac artery compression occurs during expiration and is more evident with the patient in the erect position. At expiration, in fact, the aorta and its major branches, including the celiac artery, move cephalad, and this causes worsening of compression, while with inspiration, the celiac artery descends lower in the abdominal cavity, resulting in a more vertical orientation, which often relieves compression. (8)

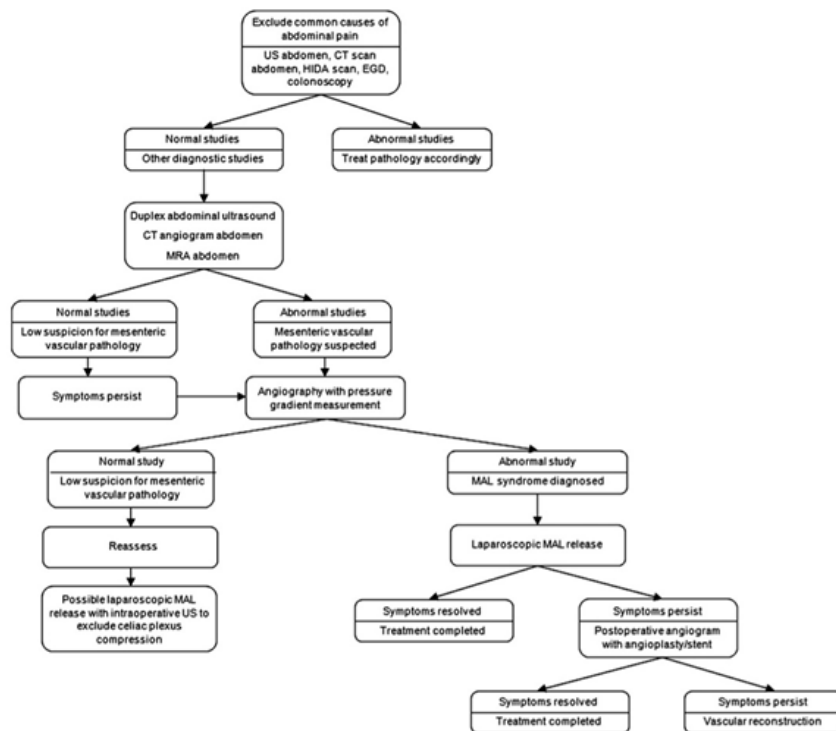
The pathophysiologic mechanisms underlying the clinical manifestations of MALS are multifactorial, involving both vascular and neurogenic components. Foregut ischemia due to compromised celiac artery blood flow was considered the primary reason for symptoms like postprandial epigastric pain. However, increasing evidence suggests that mesenteric collateral circulation often compensates adequately, thus challenging the ischemic hypothesis. As per the neurogenic hypothesis, chronic compression, and irritation of the celiac plexus, which lies close to the celiac artery, contribute to pain generation and autonomic disturbances. (9)

Patients are usually young, thin women between the ages of 30 and 50, and typically have had extensive workups for other sources of abdominal pain, which is located in the epigastric area and worsens after meals, with exercise, or with leaning forward. The pain is also associated with nausea, emesis, bloating, and diarrhoea. Patients may also experience sitophobia (food fear) because of these symptoms. Physical examination findings are typically nonspecific and overlap with those of other upper-abdominal gastrointestinal disorders. Therefore, abdominal ultrasound, EGD, and gastric emptying studies are usually performed to rule out other sources of pain. (10)

A mesenteric Doppler ultrasound is a good screening tool for patients with suspected median arcuate ligament syndrome. The ultrasound should show post-stenotic dilatation of the celiac trunk and elevated blood velocities, exaggerated during expiration. Computed tomography angiography (CTA) or magnetic resonance angiography (MRA) allows a special visualization of the compressed celiac trunk with a hooked appearance and post-stenotic dilatation.

Conventional angiography remains “the gold standard to show dynamic compression of the celiac artery”. Breathing maneuvers can be very helpful for diagnosis.

The aims of the treatment of MALS are (1) decompression of the celiac trunk so that normal blood flow is restored, and (2) pain management by celiac ganglionectomy. (11) Surgery is the treatment of choice, and laparoscopic surgery has shown promising results. A common procedure is to separate the ligament fibers and surrounding tissue at the origin of the celiac trunk to relieve compression. Compared with laparotomy, laparoscopic surgery can reduce surgical trauma and patient hospitalization, improve operative safety, and ultrasound can be used to confirm the opening of the celiac trunk. (12) Endovascular treatment has shown potential in certain cases, particularly for patients with significant vascular involvement or those who have not responded to other treatment options.



**Fig 6 - Suggested algorithm for evaluation and management of MALS (13)**

## CONCLUSION

MALS is a rare but significant cause of chronic abdominal pain and weight loss, requiring a high index of suspicion for diagnosis. Laparoscopic median arcuate ligament release and celiac plexus transection have demonstrated effectiveness, as illustrated by this case.

This report emphasizes the importance of considering MALS in the differential diagnosis of unexplained abdominal symptoms and highlights the favourable outcomes associated with surgical management. Future research should focus on elucidating the underlying pathophysiology, improving diagnostic accuracy, and advancing minimally invasive treatment approaches.

## CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

## CONFLICT OF INTEREST

The author declares no conflicts of interest.

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