



# Dunbar syndrome—a reappraisal

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## Abstract

Dunbar syndrome also called as median arcuate ligament syndrome is a rare cause of angina abdominis. Diagnosis can be made with CT angiography or magnetic resonance imaging (MRI). Surgery is the gold standard treatment. This is an instance of Dunbar syndrome who was assessed for abdominal torment and was alluded from gastroenterology department for vascular and surgical intervention.

**Keywords** Dunbar syndrome · Median arcuate ligament syndrome · MRI

## Introduction

One of the rare causes of angina abdominis is constriction of the celiac trunk by median arcuate ligament. Release of compression with or without revascularization is the treatment of choice. This is an appraisal of one such case which was referred to the vascular department for vascular intervention.

## Case report

A 42-year-old male presented with post-prandial epigastric pain for a span of 6 months. On evaluation, USG Doppler showed focal narrowing at the origin of the celiac trunk with normal caliber of the distal vessel. There was incremental peak systolic velocity (PSV) of the celiac artery, prominent on inspiration (PSV of celiac artery—inspiration 196 cm/s and expiration 104 cm/s). Contrast enhanced computed tomography (CECT) of the abdomen revealed a hook-like narrowing of proximal celiac trunk, caused by superior indentation of thickened median arcuate ligament (MAL thickness 4.3 mm). MRI affirmed the diagnosis (Fig. 1a). Surgical treatment with relinquishment of compression with or without revascularization was orchestrated.

## Surgery

Laparotomy with looping of the celiac trunk vessels was done. Release of compression of celiac vessel was accomplished with division of taut median arcuate ligament (Fig. 1b, c) The tissues circumventing the celiac artery and encompassing ganglion was abstracted. Since narrowing was not rigorous, revascularization (Fig. 1d) was not done. Post-operative period was uneventful. The patient was discharged. He was symptom-free when followed-up at 6 months and 1 year.

## Discussion

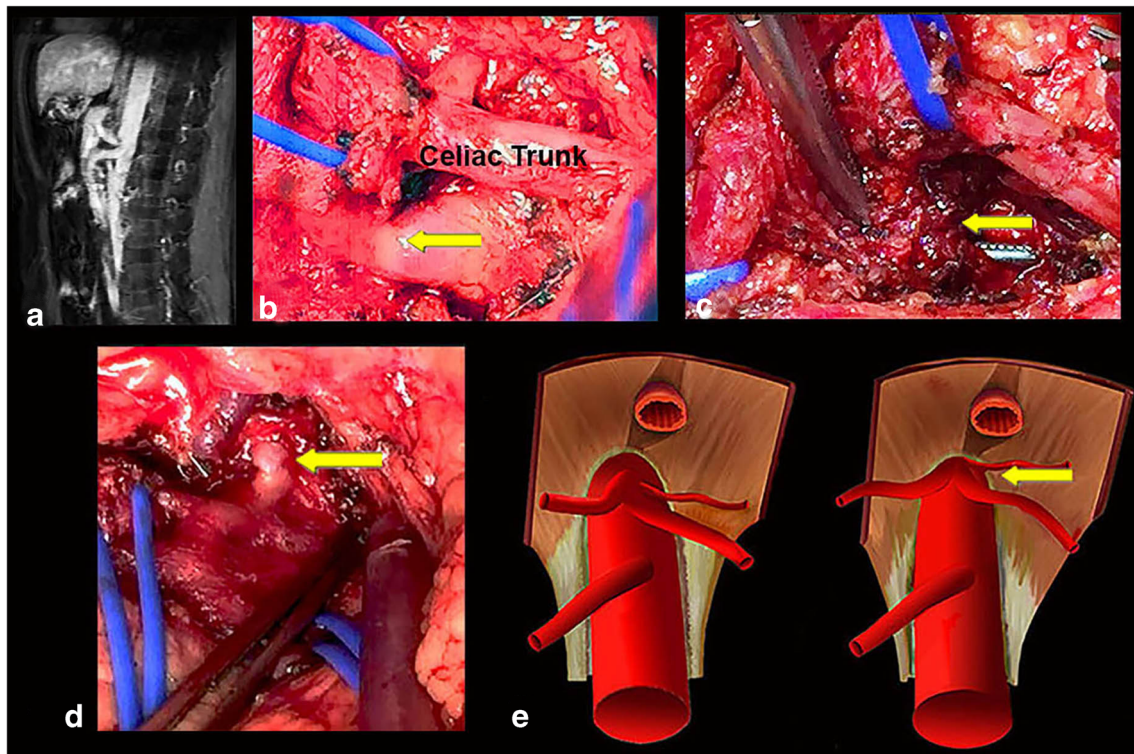
At T12 L1 level lies the MAL which is a fibrous arch that amalgamates the diaphragmatic crura on either side of the aortic hiatus. The celiac artery arises from the anterior part of the aorta, conventionally below the level of MAL (Fig. 1e). When MAL inserts lower than customary or when celiac artery arises above than mundane (arrow in Fig. 1e), the artery gets compressed, causing symptoms. This condition is called as MAL syndrome or celiac artery compression syndrome or celiac axis syndrome or Dunbar syndrome.

MALS is an infrequent entity which was first described by Harjola with an incidence of two out of 100,000 [1]. The first MALS clinical study was by Dunbar et al. The etiology of Dunbar's syndrome has been found as hereditary, vascular, and neurogenic [2].

It especially occurs in young patients and is common in thin women. The classic triad of Dunbar includes post-prandial abdominal pain, weight loss, and epigastric bruit. Atypical presentation includes nausea and vomiting [3]. Pain is aggravated in supine position and relieved in standing

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**Fig. 1** a Representation of compression of celiac trunk by the diaphragm. b MRI showing fish hook deformity appearance. c Compression of celiac artery. d Compressing ligament hooked up. e After release of ligament the constricted part of artery is seen

position. Epigastric bruit, however, sometimes may be the only clinical sign in Dunbar syndrome, although it can be seen in 30% of normal healthy adults. Doppler ultrasound has been the best initial test for Dunbar syndrome which shows an increased PSV. Selective angiography and computed tomography angiography abdomen (CTA) are the gold standard diagnostic methods. MRI can substantiate the diagnosis (Fig. 1b). CTA will show a characteristic hook-like narrowing of proximal celiac trunk, caused by superior indentation of thickened median arcuate ligament. The compression is more apparent in the expiratory phase. Absence of arterial calcification and presence of characteristic hook sign (arrow in Fig. 1b) differentiate Dunbar's syndrome from atherosclerosis.

The management strategies include interventional angioplasty and surgery. Endovascular celiac artery stenting alone without release of the ligament is dismised because of clinical failure due to slippage of stents caused by persisting extrinsic compression [4].

Surgical release of MAL and revascularization of celiac artery with celiac ganglionectomy is the treatment of choice. Surgical release can be accomplished by open laparoscopy or by robotic approach [5, 6]. One of the approaches is retrograde, with division of gastrohepatic ligament and exposure of the right crus of the diaphragm. The coronary vein and left gastric artery are mobilized and looped. The left gastric is further traced till the trifurcation of the celiac artery. Median arcuate ligament could be seen compressing the artery. Surgery aims at precise identification of

the celiac trunk with complete division of muscular bands surrounding the celiac artery. Once release is completed, assessment of celiac artery is made. Vascular damage or significant stenosis mandates artery revascularization. Perivascular sympathectomy and celiac ganglionectomy can relieve vasospasm and increase blood flow.

Tracci [7] found that MAL release alone were more likely to have symptom recurrence. According to some study, when 53% remain asymptomatic with release of compression alone, addition of revascularization increases the long-term results to about 80% [4].

## Conclusion

Dunbar syndrome is a rare entity. Clear comprehension of celiac trunk anatomy is fundamental for surgical management. Surgical release of compression with revascularization is the treatment of choice; however, the need for revascularization should be assessed predicated on intra-operative assessment of stenosis and vascular damage.

## Compliance with ethical standards

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national

research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Conflicts of interest** The authors declare that they have no conflict of interest.

**Informed consent** Informed consent was taken.

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