

Yemen Journal of Medicine

Yemen
Journal of Medicine
April reviews, type annum stores
Additional Strongly of the Conference of th

https://yemenjmed.com

Article information

DOI: 10.63475/yjm.v4i2.0163

Article history:

Received: 02 July 2025 Accepted: 11 July 2025 Published: 25 September 2025

Correspondence to:

Sajad Ahmad Salati Email: docsajad@gmail.com; S.SALATI@qu.edu.sa ORCID: 0000-0003-2998-7542

How to cite this article

Salati SA, Malik FR, AlSulaim LS. Dunbar syndrome – a narrative review. Yemen J Med.2025;4(2):269-278

Copyright License: © 2025 authors. This scholarly article is disseminated in accordance with the provisions of the Creative Commons Attribution License, thereby permitting unrestricted utilization, distribution, or reproduction across any medium, provided that credit is given to the authors and the journal

Review Article

Dunbar Syndrome - A Narrative Review

Sajad Ahmad Salati¹, Faiza Riaz Malik², Lamees Sulaiman AlSulaim³

- 1 Professor, Department of Surgery, College of Medicine, Qassim University, Kingdom of Saudi Arabia
- 2 Assistant Consultant, Department of Surgery, Qassim University Medical City, Buraydah, Kingdom of Saudi Arabia
- 3 Assistant Professor, Department of Surgery, College of Medicine, Qassim University, Buraydah, Kingdom of Saudi Arabia

ABSTRACT

Dunbar syndrome (DS) is a rare condition, predominantly in females. It arises due to the compression of the celiac trunk and celiac plexus by the fibrous attachments of the median arcuate ligament of the diaphragm. DS mimics many common gastrointestinal disorders due to the postprandial nature of symptoms and is technically a diagnosis of exclusion, confirmed by various imaging modalities. The goal of treatment is decompression of the celiac trunk, and surgery is either the mainstay of therapy, along with adjuvant interventional procedures like percutaneous transluminal angioplasty and stenting. In this review article, we revisit DS to enhance awareness among healthcare providers and discuss this disorder in terms of its historical background, epidemiology, etiopathogenesis, clinical presentation, diagnosis, and management.

Key words: Dunbar syndrome, median arcuate ligament syndrome, celiac artery compression syndrome, diaphragmatic crura, angiogram

INTRODUCTION

Dunbar syndrome (DS) (synonyms: median arcuate ligament (MAL) syndrome; celiac artery (CA) compression syndrome; celiac axis syndrome) is a rare anatomoclinical entity characterized by vague symptoms. Most of the time, this condition is underestimated, resulting in delayed diagnosis and a significant reduction in quality of life (QOL) for patients. [1] The available literature is still limited, and hence many aspects of etiopathogenesis and management are still not fully clear. The syndrome is generally believed to result from compression of the celiac axis by the MAL. [2] Since imaging modalities are frequently utilized for diagnostic purposes in common abdominal disorders, greater numbers have been documented, and newer treatment strategies have been explored in recent years. [3] It is in this backdrop that this article has been compiled to review the historical background, epidemiology, etiopathogenesis, diagnostic evaluation, and treatment of DS and thereby increase awareness about this rare disorder.

METHODS

A search of PubMed was conducted using the key terms "Dunbar syndrome," "median arcuate ligament syndrome," and "celiac artery compression syndrome." No time frame was defined, but the literature from 2014 to 2024 was preferred to analyze the recent trends in evaluation and treatment.

Historical background

In 1917, Lipshutz [4] was the first researcher to describe DS. Based on 62 cadaveric dissections, he postulated that the diaphragmatic crura may occasionally overlap the

celiac trunk's origin. George [5] conducted 38 cadaveric dissections and found that the diaphragm overlapped the CA in eight of the cases. He noted that in these situations, the diaphragm's constrictive action resulted in a decrease in the diameter of the celiac trunk at its origin. A 57-year-old man's postprandial epigastric pain was relieved by surgical decompression of the CA, according to a 1963 report by Harjola. [6]

In 1965, Dunbar et al. [7] made history when they showed, by angiography, that the celiac trunk was compressed in a cohort of 15 patients who experienced postprandial stomach pain and weight loss. After the diaphragm's MAL was sectioned to surgically release the constricted CA, 12 (80%) of the cases had their clinical symptoms resolved. Subsequently, numerous additional writers concurred with Dunbar et al. and linked SA compression to the MAL.

Epidemiology

With an incidence of two cases per 100,000 people, DS is considered a rare disorder. Females with thin habitus in their third to fifth decade of life are most affected, with a 4:1 female-to-male ratio. [8] Besides gender, the literature also mentions smoking, hypertension, hyperlipidemia, malnourishment, and a history of previous abdominal surgery as risk factors. [9]

The increased use of multiplanar contrast-enhanced imaging technologies for the assessment of abdominal disorders is responsible for the rise in the number of cases reported in the literature in recent years. [9,10] There have been reports of a 10% to 24% incidence of radiographic compression of the celiac axis in certain groups; [11] however, only a small percentage of patients experience symptomatic CA compression.

Etiopathogenesis

The exact etiology and pathophysiology of DS have not been fully elucidated. The two medial borders of the diaphragmatic

crura are joined by MAL, which is a fibrous band, often located at the anterior and superior aspects of the CA, close to the level of the 12th thoracic or 1st lumbar vertebra. Similarly, CA generally arises from the abdominal aorta, usually between the 11th thoracic and the 1st lumbar vertebra. [12]

This ligament can potentially create external compression of the CA (**Figure 1**), if congenitally: [12,13]

- 1. The aortic origin of the CA is an abnormally cephalad, or
- 2. The insertion of the MAL is abnormally caudad, due to severe lumbar lordosis or accelerated growth.

The compression of the CA by the MAL is believed to cause intermittent visceral ischemia. During the expiration phase of breathing, the diaphragm moves upwards, thereby stretching the crura, which in turn worsens the degree of compression of CA. [14,15] However, this explanation alone may not completely explain the condition and the clinical features because:

- a) There is usually a rich collateral network of mesenteric vessels between the CA and the superior mesenteric artery (SMA). [16,17]
- b) The anatomic compression of the CA by MLA is also seen in asymptomatic patients. Park et al. [18] performed a CA angiogram in 400 cases of hepatic tumors planned for chemoembolization and incidentally found that 7.3% of the cases had significant celiac stenosis (>50% stenosis; >a 10-mmHg pressure gradient), though they were asymptomatic.

Hence, several other pathophysiological mechanisms have been suggested, and a multifactorial genesis for the disease is preferred. [2,19] The factors include:

 a) Overstimulation of the underlying celiac ganglion and altered neural control, resulting in abnormal

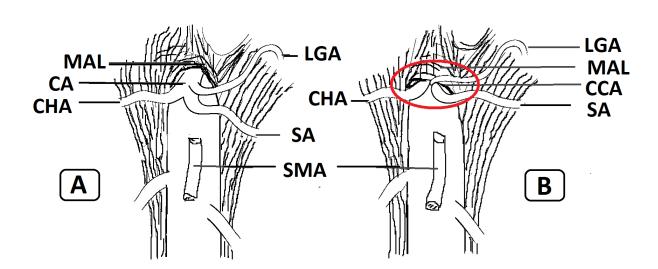


Figure 1: Schematic diagram comparing the relationship of the median arcuate ligament (MAL) and celiac artery (CA). (A) Normal anatomical relationship. (B) Abnormal anatomy with compression of the CA by the MAL. [Image credits: Sajad Ahmad Salati - Author]

- gastric electrical rhythm and delayed gastric emptying. [20,21]
- b) Histological changes in the CA, such as hyperplasia of the intima, proliferation of elastic fibers in the media, and disorganization of the adventitia. [20]
- c) Constriction of the celiac trunk as a ligature or in an annular manner by an abnormally dense and fibrosed celiac ganglion. [22]

Role of lumbar lordosis: Sahm et al., [3] after a study of 18 patients (12 female, 6 male) aged between 15 and 65 years, suggested that an exaggerated lumbar lordosis triggers the development of a DS. This mechanism also tends to explain the co-existence of other vascular compression disorders, predominantly nutcracker syndrome, in patients with DS. In contrast, a kyphotic posture of the lumbar spine decreases the traction of the MAL towards the coeliac trunk and thereby tends to relieve its compression. The mechanics of the lumbar spine explain the relief of the postprandial pain attacks that most patients achieve by adopting a crouching embryonic position.

Role of genetic factors: Numerous published case reports imply that the responsible anatomic linkages of DS could be inherited genetically. Bech et al., [23] reported DS in two identical twin sisters, ages 27 years. One twin had severe CA stenosis, and the other twin had full CA occlusion, as detected on angiography. Furthermore, in both, proximal SMA narrowing with retrograde filling from a meandering mesenteric artery was demonstrated. Division of the MAL and CA revascularization completely relieved symptoms in both patients.

Using multidetector computed tomography (CT) scans, Okten et al., [24] identified DS in members of one family, including a father, a daughter, and three brothers. A 16-year-old DS patient who had laparoscopic MAL release along with CA skeletonization was described by Said et al., [25] The patient's mother likewise had a history of DS and had been treated via an open surgical approach with complete MAL division followed by patch angioplasty of the CA, owing to persistent stenosis.

Synchronous disorders of the SMA: There have been reports of SMA's simultaneous involvement in a variety of vascular diseases in DS patients. While undergoing surgery for SMA dissection, a 43-year-old man was found to have DS by Okada et al., [26] Following SMA lesion treatment, a laparoscopic MAL release and ganglionectomy were carried out. Rama et al., [27] described a case in which the SMA and the CA were compressed by MAL. Following a laparoscopic release of MAL, the patient's symptoms resolved.

Clinical features

Patients with DS experience vague symptoms comprising postprandial upper abdominal pain, nausea, vomiting, diarrhea, and unintended weight loss. In a series by Sahm et al., [3] 18 patients (12 female, 6 male), aged between 15 and 65 years, presented with abdominal pain (100.0%), postprandial pain (66.7%), nausea/vomiting (16.7%), and weight loss averaging 12.2 kg ± 4.5 kg (55.6%). Similarly, in a series of 36 patients, Cusati et al., [28] documented abdominal pain (94%), postprandial abdominal pain (80%),

weight loss (50%), bloating (39%), nausea and vomiting (55.6%), and abdominal pain triggered by exercise (8%). On auscultation of the abdomen, there may be tenderness on deep palpation. Furthermore, bruits may be audible in up to 35% of cases. [29]

Asymptomatic DS: The majority of patients with DS are asymptomatic. This is attributed to the compensation of the arterial flow to the foregut from an extensive collateral circulation, formed of various vessels including the pancreaticoduodenal arteries, common hepatic artery, splenic artery, and dorsal pancreatic artery. The patients do not experience the symptoms associated with DS, but the elevated flow of blood through these small collateral arteries has been found to significantly increase the probability of aneurysm formation and life-threatening bleeding due to rupture. [30]

Psychiatric comorbidities in DS: Many psychiatric disorders, including anxiety disorders, depression, panic disorder, and post-traumatic stress disorder, have an increased incidence in DS. Skelly et al., [31] conducted psychological assessments of 51 patients with DS (80% female) with a mean age of 30.5 (±12.4) years before and 6 months after surgical operation. Psychiatric diagnoses were found in 28% of patients, and there were no differences in the number of patients with psychiatric diagnoses between presurgical and postsurgical evaluations. The study suggested that having a psychiatric diagnosis in the presurgical phase may predict significantly lower QOL after surgery.

In pediatric patients, concurrent psychological problems are particularly common. Stiles-Shields et al., [32] undertook psychological assessments of 32 pediatric patients before surgery for DS and 6 months after surgery. They found that more than 50% of the pediatric patients had a comorbid psychological disorder that continued to persist even after the surgical operation.

Mak et al., [33] suggested that the psychosocial profiles of children suffering from DS and other gastrointestinal disorders resulting in chronic abdominal pain (CAP) are similar. The overlap of physical and psychosocial symptoms led to a recommendation that patients with CAP should be evaluated for DS. Certain other disorders characterized by physical abnormalities have been found to have a higher prevalence in patients with DS, which include Ehlers-Danlos syndrome, visceral vasculopathy, joint hypermobility, and postural orthostatic tachycardia syndrome. [34,35]

DS in athletes

Exercise-related transient abdominal pain (ETAP) is a common entity in young athletes, mostly occurring due to cramps. But in recent literature, it has been reported that the initial presentation of DS in athletes may be exercise-induced pain rather than the common postprandial symptoms. In a case series published by Harr et al., [36] three athletes had presented with ETAP. Imaging studies, including CT scans and magnetic resonance angiography (MRA), were undertaken when conservative treatments failed to alleviate the symptoms, and a diagnosis was made. They underwent laparoscopic MAL release without any complications, resolved their symptoms, and subsequently returned to

athletics. Haskins et al., [37] have presented an 18-year-old field hockey player who presented with a 1-year history of ETAP. Despite a trial of preventative strategies, the patient's pain persisted, prompting an uneventful laparoscopic MAL release. The patient's symptoms disappeared completely.

DS in children

DS is reported in the pediatric age group, also with a clinical presentation that mimics chronic functional abdominal pain (CFAP). Mak et al., [38] prospectively studied 46 pediatric (<21 years of age; 91% females) patients diagnosed with DS over four years from 2008 to 2012. All the patients had been misdiagnosed with CFAP and had poor QOL. The patients underwentlaparoscopic release of the MAL and celiac neurolysis, resulting in a significantly enhanced blood flow through the CA. Overall improvement in the QOL was documented in 83%, while secondary procedures were required in 17% of patients.

Joyce et al., [39] reported six patients with DS (83.3% females) with a mean age of 15.7 years. They were managed successfully with laparoscopic release for MAL, and a significant improvement from pre- to post-surgical scores was observed in the physical functions, mental health, self-esteem, and QOL. The study found that laparoscopic release for MALS, in carefully chosen patients, normalizes blood flow in the CA without the need for subsequent reconstruction. This enhances the child's and parents' mental and physical QOL. Stiles-Shields et al., [40] enrolled 12 pediatric patients of DS with a mean age of 15.2 years (91.7% female) and their parents (91.7% mothers) in a study, and based on their observations, they proposed the use of a pre-operative cognitive behavioral therapy regimen with an emphasis on psychoeducation and coping methods.

Differential diagnosis

DS mimics several common disorders, owing to the postprandial nature of its symptoms. The simultaneous co-existence of these conditions might also present a significant diagnostic challenge. [1,12,20] The prominent differential diagnoses include:

Peptic ulcer: Postprandial epigastric pain is an important symptom. Upper gastrointestinal endoscopy confirms the diagnosis, and proton pump inhibitors typically result in improvement.

Willkie syndrome (SMA syndrome): This is a rare vascular compression disorder in which acute angulation of the SMA results in compression of the third part of the duodenum, leading to obstruction. Patients with SMA syndrome may present with chronic symptoms like recurrent episodes of abdominal pain, vomiting, early satiety, and loss of appetite. CTA/MRA enables visualization of vascular compression of the duodenum and measurement of the aortomesenteric distance. The normal magnitudes of the aortomesenteric angle and aortomesenteric distance are 28° to 65° and 10 to 34 mm, respectively. In Willkie syndrome, both parameters are reduced, with values of 6° to 22° and 2 to 8 mm.

Gallbladder disorders: These include biliary dyskinesia and chronic cholecystitis, and the patient typically presents with postprandial abdominal pain. Ultrasonogram (USG) and hepatobiliary iminodiacetic acid scans usually clinch the diagnosis.

Chronic mesenteric ischemia (CMI): CMI usually results from long-standing atherosclerotic disease of two or more mesenteric vessels and is generally characterized by postprandial abdominal pain. A CT angiogram (CTA) of the abdomen and pelvis with intravenous (IV) contrast or MRA without and with IV contrast usually confirms the diagnosis.

Investigations

DS is often diagnosed by exclusion; before any clinical suspicions of DS are expressed, more prevalent gastrointestinal and hepatobiliary illnesses are ruled out. [2] The following imaging modalities, when combined with clinical characteristics, are crucial in establishing the diagnosis when a suspicion is raised:

Duplex ultrasonography (DUS): DUS is the preferred initial screening tool (**Figure 2**) due to its non-ionizing nature and the lack of requirement for contrast. The sensitivity and specificity of DUS in making a diagnosis of DS are about 75% and 89%, respectively. [42] It can display the deflection angle greater than 50°, post-stenotic dilatation, and elevated peak systolic velocities (>200 cm/s) exaggerated during expiration. [43,44]

In the series by Sahm et al., [3] the average flow velocities recorded in the CA in the mid-position of the diaphragm were 285.9 cm/s at expiration and 199.0 cm/s at inspiration, whereas in the series by Klimas et al., [45] the reported flow velocities ranged between 190 and 450 cm/s.

However, this modality is constrained by:

- a) Operator-related factors: DUS is an operatordependent tool, and experienced operators are required to recognize the findings.
- **b) Patient-related factors:** body habitus (obesity) and overlying bowel gas can hinder image capture.

Computed tomography angiography (CTA): CTA works by visualization of the degree of stenosis via the arterial phase during expiration and the venous phase during inspiration (Figure 3). By using multiple scanners, a three-dimensional image of the arterial planes with detailed resolution is produced using multiplanar reconstruction. CTA is non-invasive, easily reproducible, and has lower interobserver variation; however, adverse effects due to contrast-induced nephropathy and radiation exposure restrict the type of patients that can be subjected to this imaging modality.

Magnetic resonance angiography (MRA): This is an imaging modality (Figure 4) with no risks of ionizing radiation and allergy to IV contrast agents. This modality delineates the anatomy and is highly effective in diagnosis as well as preoperative planning.

Management

Treatment modalities in symptomatic patients include surgical and non-surgical procedures [46,47] as depicted in (**Figure 5**).

Surgical procedures: Surgical intervention is undertaken through open or minimally invasive approaches, which include either laparoscopic or robot-assisted techniques.

The aims of the operation are:

 Decompression of the celiac trunk to restore its lumen and normal blood flow; intraoperatively, the

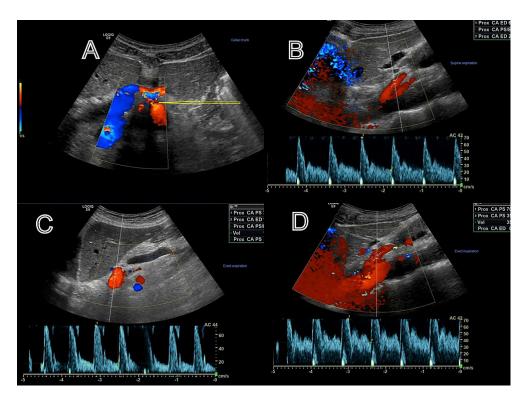


Figure 2: Doppler ultrasound with the arrow showing the point at the constriction of the different variations of flow through the celiac trunk: (A) constricted portion of the celiac artery. (B) supine expiration. (C) erect expiration. (D) erect inspiration. Image Source: Okobi et al. [41] Reused under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

adequacy of decompression can be assessed with D-USG by confirming that there is minimal elevation of CA velocity and no change with deep respiration. [48] Whatever the approach adopted, the operation steps involve the opening of the lesser sac, division of the hepato-gastric ligament, and dissection of the crura until the anterior wall of the aorta is exposed. After proper identification of the celiac trunk due to its three branches, it is carefully skeletonized and circumferentially freed of fibrous, lymphatic, and nerve attachments. [49] After CA decompression, revascularization may or may not be undertaken.

- Release of the coeliac plexus and prevention of its postoperative strangulation by appropriate enlargement of the aortic hiatus.
- Laparotomy: Laparotomy (open surgical approach) is the traditional approach, and entry into the peritoneal cavity is achieved through a midline incision. The safety of attaining complete release of CA and control of hemorrhage under direct visual guidance represents an important advantage of an open surgical approach. However, the main drawback of this technique is the need for a longer exposure time and a bigger incision. [50]

To assess the long-term effects of treating DS with an open surgical technique, Reilly et al., [51] investigated potential variables that might affect long-term symptom alleviation. Operative treatment comprised of CA decompression only, CA decompression and dilatation, or CA decompression

with reconstruction (primary re-anastomosis or interposition vessel grafting). At a mean follow-up of 9 years, a positive correlation with clinical improvement was demonstrated more often with a postprandial pain pattern, age between 40 and 60 years, and weight loss of 20 pounds or more. CA decompression plus some form of celiac revascularization led to sustained relief in 73% cases in comparison to only 53% with celiac decompression alone. In 70% of patients without symptoms, late follow-up angiograms revealed a broadly patent CA, but in 75% of patients with symptoms, the celiac axis was stenosed or occluded.

• Minimally invasive approach: The utilization of robot-assisted operations [52] and laparoscopic procedures [53] that are minimally invasive has increased in recent years. Comparable outcomes to laparotomy have been shown, along with the advantages of fewer incisions, enhanced peroperative vision, less pain following surgery, reduced length of hospital stay, faster recuperation, and enhanced cosmetic outcomes. [54]

Do et al., [55] presented the analysis of 16 cases of DS with similar characteristics and comorbidities: 12 cases treated via a laparoscopic approach and four patients via a robot-assisted approach. No intraoperative or perioperative conversions, complications, or deaths were encountered, and the mean operative time for the laparoscopic approach was significantly shorter than for the robotic approach (101.7 vs. 145.8 min; p = 0.02). There was no significant difference in the length of hospital stay. 67% and 50% of cases in the laparoscopic group and the robotic group, respectively,



Figure 3: CTA (computed tomography angiogram) showing compression of the celiac trunk as indicated by the arrow. Image Source: Okobi et al. [41]. Reused under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

attained full resolution of their symptoms. Van Petersen et al., [56] demonstrated satisfactory results in a series of DS patients managed with a retroperitoneal endoscopic MAL release, with 83% of operative patients experiencing symptom improvement.

Birkhold et al., [57] conducted a retrospective chart review of 11 patients who underwent a laparoscopic MAL release from January 2015 to January 2020. The patients were further surveyed for physical functioning, emotional well-being, and social functioning aspects by using the 36-item Short Form and Visick Questionnaires. In 10 (73%) cases, they found that the symptoms improved with minimal morbidity, and only one patient underwent additional interventions for symptom resolution due to residual CA stenosis. The study concluded that laparoscopic MAL release is a safe and effective surgery for patients suffering from DS.

Belluzzi et al., [58] retrospectively analyzed the data related to the demographics, preoperative evaluation, postoperative adverse outcomes, and the resolution of presenting symptoms in 30 patients (77% females) who underwent a primary laparoscopic MAL release from June 2021 to July 2023. Duplex mesenteric doppler and CTA during inspiration and expiration, and 3D reconstruction were undertaken in all the patients to establish the diagnosis. In 96.6% of cases, the procedure was successful, and conversion to laparotomy was required in only one case. There were no major complications, and there were no requirements for reintervention or reoperation. Celiac plexus/splanchnic block injection was, however, required in one case to alleviate pain. The study concluded that DS can be effectively and safely managed using a laparoscopic approach, but cautioned that the long-term effectiveness of this approach would need further studies with longer follow-ups.

Butz et al., [59] retrospectively analyzed 20 patients with DS who underwent MAL release through laparoscopic (n = 3) or robotic (n = 17) approaches between 2014 and 2023. They found that there was no statistically significant difference between the two approaches with respect to the mean operation time, intraoperative blood loss, and the mean

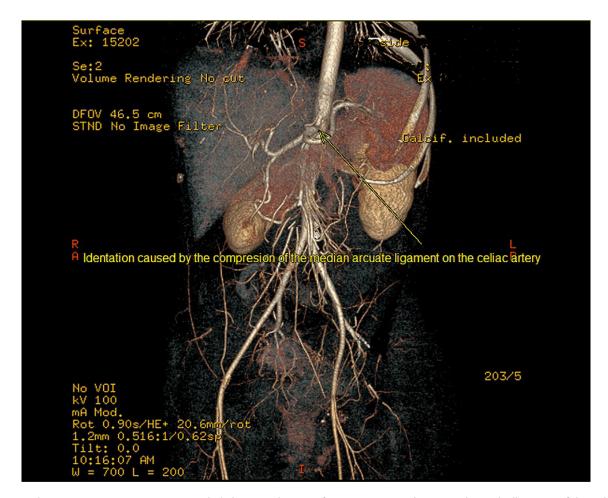


Figure 4: MRA (magnetic resonance angiography) showing the site of compression with pre- and post-ballooning of the celiac trunk. Image Source: Okobi et al. [41]. Reused under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

hospital stay. Significant reduction in the peak systolic velocity in the CA was documented in 17 (85%) patients with symptom improvement. Four (20%) patients had no symptom relief, and in 3 cases, follow-up imaging showed evidence of respiratory-related CA stenosis. The study concluded that despite complexity, laparoscopic and robotic-assisted MAL releases are safe procedures with good long-term outcomes.

Hybrid approach: Minimally invasive endovascular therapy (EVT) in the form of percutaneous angioplasty with stenting is a recent innovation for the management of DS, although its effectiveness has not yet been determined. Sunohara et al. [60] reported a 59-year-old male in whom the lumen of the compressed CA was well dilated using intravascular ultrasound and then followed by the placement of a bare metal stent. After 15 months, the patient was symptom-free with a patent stent. EVT provides an alternate treatment option for selected cases of MALs when the traditional surgical modalities are contraindicated or have failed.

The hybrid method, combining laparoscopy and EVT, has been proposed as a satisfactory long-term solution for DS with minimum risk of restenosis. Michalik et al. [61] reported five patients, in whom laparoscopic release of MAL was followed at one month with Doppler-aided percutaneous angioplasty of the CT with stent implantation. All patients reported complete relief of symptoms.

Prognosis

A cure rate of up to 60% to 90% has been achieved through different treatment approaches. [12] Van Petersen et al. [56] treated 46 patients through an endoscopic retroperitoneal approach, and at follow-up of 20 months, 41 (89%) patients were free of symptoms or significantly improved. Grotemeyer et al. [53] attained complete freedom from symptoms in 11 of the 15 (73.33%) surgically operated patients. Metz et al., [62] conducted a systematic analysis of 44 studies related to the management of DS by various available modalities, thereby including 880 adult and 195 pediatric patients. Overall, 70% of adult and pediatric patients reported long-term relief in symptoms and an improvement in QOL. Woestemeier et al., [63] analyzed 20 DS patients who underwent surgical CA decompression and found that 12 patients (60%) attained postoperative relief in their symptoms and decreased their analgesic intake.

Various factors have been postulated that may influence prognosis, and these include age of the patient, treatment modality, surgical technique, postsurgical follow-up facilities, and associated comorbidities. [64,65] Sun et al., [66] have highlighted that the abdominal pain in DS is sympathetically mediated via the celiac plexus, and hence, celiac plexus block can serve as a diagnostic tool as well as a predictor of surgical

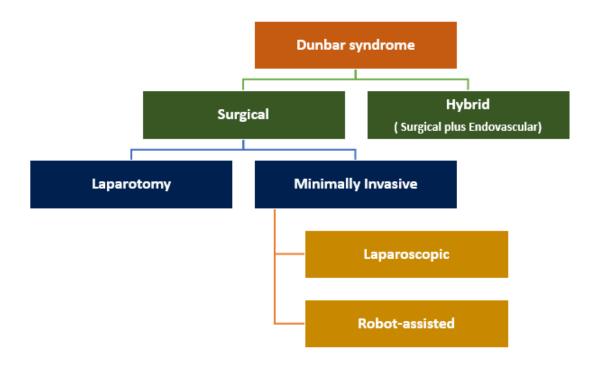


Figure 5: Management options of Dunbar Syndrome [Image credits: Sajad Ahmad Salati - Author]

outcomes. The patients having a comorbid psychological disorder in the presurgical phase generally tend to persist even after the surgical operation, thereby significantly lowering the QOL. [31,32]

CONCLUSIONS

DS is a rare disorder caused by compression of the CA and celiac plexus by the MAL and manifests as gastrointestinal ischemic symptoms. The disease mimics many common abdominal disorders and adversely affects the QOL. Clinicians must be aware of this entity so that the disease can be suspected and promptly diagnosed. Treatment is predominantly surgical. Prognosis and risk factors are still being studied; further studies are required to attain further clarification.

ACKNOWLEDGEMENTS

The authors thank the Cureus Journal of Medical Sciences for having allowed the reusage of images under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

AUTHORS' CONTRIBUTION

All authors have significantly contributed to the work, whether by conducting literature searches, drafting, revising, or critically reviewing the article. They have given their final approval of the version to be published, have agreed with the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

SOURCE OF FUNDING

None.

CONFLICT OF INTEREST

None.

REFERENCES

- Sghaier A, Jelassi MA, Fradi K, Mraidha MH, Hamila F, Youssef S. Median arcuate ligament syndrome (Dunbar syndrome): A diagnosis not to be underestimated. Radiol Case Rep. 2023;19(2):636-641.
- Kim EN, Lamb K, Relles D, Moudgill N, DiMuzio PJ, Eisenberg JA. Median arcuate ligament syndrome-Review of this rare disease. JAMA Surg. 2016;151(5): 471-477.
- 3. Sahm M, Otto R, Pross M, Scholbach T, Mantke R. Laparoscopic therapy of the coeliac artery compression syndrome: A critical analysis of the current standard procedure. Ann R Coll Surg Engl. 2020;102(2):104-109.
- 4. Lipshutz B. A composite study of the coeliac axis artery. Ann Surg. 1917;65:159-169.
- 5. George R. Topography of the unpaired visceral branches of the abdominal aorta. J Anat. 1934;69:196-205.
- 6. Harjola PT. A rare obstruction of the coeliac artery. Report of a case. Ann Chir Gynaecol Fenn. 1963;52:547-550.
- Dunbar JD, Molnar W, Beman FF, Marable SA. Compression of the celiac trunk and abdominal angina. Am J Roentgenol Radium Ther Nucl Med. 1965;95(3): 731-744.
- Trinidad-Hernandez M, Keith P, Habib I, White JV. Reversible gastroparesis: Functional documentation of celiac axis compression syndrome and postoperative improvement. Am Surg. 2006;72(4):339-344.
- Khrucharoen U, Juo YY, Sanaiha Y, Finn JP, Jimenez JC, Dutson EP. Factors associated with symptomology of celiac artery compression and outcomes following

- median arcuate ligament release. Ann Vasc Surg. 2020;62: 248-257.
- 10. Khrucharoen U, Juo YY, Sanaiha Y, Chen Y, Jimenez JC, Dutson EP. Robotic-assisted laparoscopic median arcuate ligament release: 7-year experience from a single tertiary care center. Surg Endosc. 2018;32(9):4029-4035.
- Koc M, Artas H, Serhatlioglu S. The investigation of incidence and multidetector computed tomography findings of median arcuate ligament syndrome. Turk J Med Sci. 2018;48(6):1214-1218.
- 12. Loukas M, Pinyard J, Vaid S, Kinsella C, Tariq A, Tubbs RS. Clinical anatomy of celiac artery compression syndrome: A review. Clin Anat. 2007;20(6):612-617.
- 13. Horton KM, Talamini MA, Fishman EK. Median arcuate ligament syndrome: Evaluation with CT angiography. Radiographics. 2005;25(5):1177-1182.
- Den B, Ignashov AM, Perlei VE, Gichkin Alu, Ustiuzhaninov AS. [The significance of respiratory and orthostatic tests in duplex scanning in diagnostics of celiac artery compression syndrome.] Vestn Khir Im I I Grek. 2013; 172(2):28-31.
- 15. Reuter SR, Bernstein EF. The anatomic basis for respiratory variation in median arcuate ligament compression of the celiac artery. Surgery. 1973;73(3):381-385.
- 16. van Petersen AS, Kolkman JJ, Meerwaldt R, Huisman AB, van der Palen J, Zeebregts CJ, et al. Mesenteric stenosis, collaterals, and compensatory blood flow. J Vasc Surg. 2014;60(1):111-119.e1-2.
- van Petersen AS, Kolkman JJ, Gerrits DG, van der Palen J, Zeebregts CJ, Geelkerken RH; Dutch Mesenteric Ischemia Study Group. Clinical significance of mesenteric arterial collateral circulation in patients with celiac artery compression syndrome. J Vasc Surg. 2017; 65(5):1366-1374.
- 18. Park CM, Chung JW, Kim HB, Shin SJ, Park JH. Celiac axis stenosis: Incidence and etiologies in asymptomatic individuals. Korean J Radiol. 2001;2(1):8-13.
- Sultan S, Hynes N, Elsafty N, Tawfick W. Eight years experience in the management of median arcuate ligament syndrome by decompression, celiac ganglion sympathectomy, and selective revascularization. Vasc Endovascular Surg. 2013;47(8):614-619.
- 20. Brandt LJ, Boley SJ. Celiac axis compression syndrome. A critical review. Am J Dig Dis. 1978; 23(7):633-640.
- 21. Balaban DH, Chen J, Lin Z, Tribble CG, McCallum RW. Median arcuate ligament syndrome: A possible cause of idiopathic gastroparesis. Am J Gastroenterol. 1997; 92(3):519-523.
- 22. Harjola PT, Lahtiharju A. Celiac axis syndrome. Abdominal angina caused by external compression of the celiac artery. Am J Surg. 1968;115(6):864-869.
- 23. Bech F, Loesberg A, Rosenblum J, Glagov S, Gewertz BL. Median arcuate ligament compression syndrome in monozygotic twins. J Vasc Surg. 1994;19(5):934-938.
- Okten RS, Kucukay F, Tola M, Bostanci B, Cumhur T. Is celiac artery compression syndrome genetically inherited?: A case series from a family and review of the literature. Eur J Radiol. 2012;81(6):1089-1093.
- 25. Said SM, Zarroug AE, Gloviczki P, Shields RC. Pediatric median arcuate ligament syndrome: first report of familial pattern and transperitoneal laparoscopic release. J Pediatr Surg. 2010;45(12):e17-20.
- 26. Okada H, Ehara K, Ro H, Yamada M, Saito T, Negami N, et al. Laparoscopic treatment in a patient with median

- arcuate ligament syndrome identified at the onset of superior mesenteric artery dissection: A case report. Surg Case Rep. 2019;5(1):197.
- 27. Rama M, Nasser W, Palvannan P, Belko S, DiMuzio P, Palazzo F. Supradiaphragmatic origin of the celiac trunk leading to median arcuate ligament syndrome with superior mesenteric artery involvement. J Vasc Surg Cases Innov Tech. 2023;10(1):101315.
- 28. Cusati DA, Noel AA, Gloviczki P. Median arcuate ligament syndrome: a 20-year experience of surgical treatment. 60th Annual Meet Soc Vascul Surg. 2006;5:1-4.
- 29. Jimenez JC, Harlander-Locke M, Dutson EP. Open and laparoscopic treatment of median arcuate ligament syndrome. J Vasc Surg. 2012;56(3):869-873.
- Heo S, Kim HJ, Kim B, Lee JH, Kim J, Kim JK. Clinical impact of collateral circulation in patients with median arcuate ligament syndrome. Diagn Interv Radiol. 2018;24(4): 181-186.
- 31. Skelly CL, Stiles-Shields C, Mak GZ, Speaker CR, Lorenz J, Anitescu M, et al. The impact of psychiatric comorbidities on patient-reported surgical outcomes in adults treated for the median arcuate ligament syndrome. J Vasc Surg. 2018;68(5):1414-1421.
- 32. Stiles-Shields C, Skelly CL, Mak GZ, Speaker C, Boyd H, O'Brien S, et al. Psychological factors and outcomes in the surgical treatment of pediatric patients with median arcuate ligament syndrome. J Pediatr Gastroenterol Nutr. 2018;66(6):866-871.
- 33. Mak GZ, Lucchetti AR, Drossos T, Fitzsimmons-Craft EE, Accurso EC, Stiles-Shields C, et al. Pediatric chronic abdominal pain and median arcuate ligament syndrome: A review and psychosocial comparison. Pediatr Ann. 2016;45(7):e257-e264.
- 34. Huynh DTK, Shamash K, Burch M, Phillips E, Cunneen S, Van Allan RJ, et al. Median arcuate ligament syndrome and its associated conditions. Am Surg. 2019;85(10): 1162-1165.
- 35. Kakavand B, Burns RC, Centner A, Casas-Melley A. Median arcuate ligament syndrome in children: A single-center experience. Cureus. 2024;16(3):e57184.
- 36. Harr JN, Haskins IN, Brody F. Median arcuate ligament syndrome in athletes. Surg Endosc. 2017;31(1):476.
- Haskins IN, Harr JN, Brody F. Exercise-related transient abdominal pain secondary to median arcuate ligament syndrome: A case report. J Sports Sci. 2016;34(13):1246-1249.
- 38. Mak GZ, Speaker C, Anderson K, Stiles-Shields C, Lorenz J, Drossos T, et al. Median arcuate ligament syndrome in the pediatric population. J Pediatr Surg. 2013;48(11):2261-2270.
- 39. Joyce DD, Antiel RM, Oderich G, Gloviczki P, Tung J, Grothe R, et al. Pediatric median arcuate ligament syndrome: Surgical outcomes and quality of life. J Laparoendosc Adv Surg Tech A. 2014;24(2):104-110.
- 40. Stiles-Shields C, Osos S, Heilbrun A, Feldman ECH, Mak GZ, Skelly CL, et al. Targeting coping to improve surgical outcomes in pediatric patients with median arcuate ligament syndrome: Feasibility study. Front Psychol. 2021; 12:695435.
- 41. Okobi OE, Afuda BA, Boms M, Ekpemiro CU, Umeh NJ, Nnaji CG, et al. Median arcuate ligament syndrome: Management and literature review. *Cureus*. 2022;14(9): e28889.

- 42. Narwani N, Khanna N, Rajendran I, Kaawan H, Al-Sam R. Median arcuate ligament syndrome diagnosis on computed tomography: What a radiologist needs to know. Radiol Case Rep. 2021;16(11):3614-3617.
- 43. Scholbach T. Celiac artery compression syndrome in children, adolescents, and young adults: Clinical and color duplex sonographic features in a series of 59 cases. J Ultrasound Med. 2006;25(3):299-305.
- 44. Skeik N, Cooper LT, Duncan AA, Jabr FI. Median arcuate ligament syndrome: A nonvascular, vascular diagnosis. Vasc Endovascular Surg. 2011;45(5):433-437.
- 45. Klimas A, Lemmer A, Bergert H, Brodhun M, Scholbach T, Grober K. Laparoscopic treatment of celiac artery compression syndrome in children and adolescents. Vasa. 2015;44(4):305-312.
- Upshaw W, Richey J, Ravi G, Chen A, Spillers NJ, Ahmadzadeh S, et al. Overview of median arcuate ligament syndrome: a narrative review. Cureus. 2023; 15(10):e46675.
- 47. Iqbal S, Chaudhary M. Median arcuate ligament syndrome (Dunbar syndrome). Cardiovasc Diagn Ther. 2021;11(5):1172-1176.
- 48. Skelly CL, Mak GZ: Median arcuate ligament syndrome Current state of management. Semin Pediatr Surg. 2021;30:151129.
- 49. Lu LY, Eastment JG, Sivakumaran Y. Median arcuate ligament syndrome (MALS) in hepato-pancreato-biliary surgery: A narrative review and proposed management algorithm. J Clin Med. 2024;13(9):2598.
- Antony A, Ravindran SK, Jayan NP, Yadukrishna S, Sebastian R, Kumar A, et al. Performing median arcuate ligament release surgery in celiac artery compression syndrome: Insights from a tertiary care hospital. J Minim Access Surg. 2024;20(3):318-325.
- 51. Reilly LM, Ammar AD, Stoney RJ, Ehrenfeld WK. Late results following operative repair for celiac artery compression syndrome. J Vasc Surg. 1985;2(1):79-91.
- 52. Jaik NP, Stawicki SP, Weger NS, Lukaszczyk JJ. Celiac artery compression syndrome: Successful utilization of robotic-assisted laparoscopic approach. J Gastrointestin Liver Dis. 2007;16:93-96
- 53. Grotemeyer D, Duran M, Iskandar F, Blondin D, Nguyen K, Sandmann W. Median arcuate ligament syndrome: Vascular surgical therapy and follow-up of 18 patients. Langenbecks Arch Surg. 2009;394(6):1085-1092.
- 54. Roayaie S, Jossart G, Gitlitz D, Lamparello P, Hollier L, Gagner M. Laparoscopic release of celiac artery compression syndrome facilitated by laparoscopic ultrasound scanning to confirm restoration of flow. J Vasc Surg. 2000; 32(4):814-817.
- 55. Do MV, Smith TA, Bazan HA, Sternbergh WC, Abbas AE, Richardson WS. Laparoscopic versus robot-assisted

- surgery for median arcuate ligament syndrome. Surg Endosc. 2013; 27(11):4060-4066.
- 56. van Petersen AS, Vriens BH, Huisman AB, Kolkman JJ, Geelkerken RH. Retroperitoneal endoscopic release in the management of celiac artery compression syndrome. J Vasc Surg. 2009; 50(1):140-47.
- 57. Birkhold M, Khalifeh A, Nagarsheth K, Kavic SM. Median Arcuate Ligament Syndrome is effectively relieved with minimally invasive surgery. JSLS. 2022;26(4): e2022.00067.
- Belluzzi A, Salame M, Abi Mosleh K, Rasmussen TE, Kendrick ML, Ghanem OM. Laparoscopic median arcuate ligament release: surgical technique and clinical outcomes. Surg Laparosc Endosc Percutan Tech. 2024; 34(1):74-79.
- Butz F, Haase O, Martin F, Hillebrandt KH, Knitter S, Schoning W, et al. Short- and long term outcome of minimally invasive therapy of median arcuate ligament syndrome. Langenbecks Arch Surg. 2024;409(1): 322.
- Sunohara D, Miura T, Nomoto F, Itagaki T, Komatsu T, Mochidome T, et al. Effectiveness of endovascular therapy using stents in a patient with celiac artery compression syndrome due to the median arcuate ligament: A case report. J Cardiol Cases. 2023;28(3):128-131.
- Michalik M, Dowgiałło-Wnukiewicz N, Lech P, Majda K, Gutowski P. Hybrid (laparoscopy + stent) treatment of celiac trunk compression syndrome (Dunbar syndrome, median arcuate ligament syndrome (MALS)). Wideochir Inne Tech Maloinwazyjne. 2016;11(4):236-239.
- 62. Metz FM, Blauw JTM, Brusse-Keizer M, Kolkman JJ, Bruno MJ, Geelkerken RH; Dutch Mesenteric Ischaemia Study Group. Systematic review of the efficacy of treatment for median arcuate ligament syndrome. Eur J Vasc Endovasc Surg. 2022;64(6):720-732.
- 63. Woestemeier A, Semaan A, Block A, Arensmeyer J, Dohmen J, Kania A, et al. Prognostic factors for the long-term outcome after surgical celiac artery decompression in MALS. Orphanet J Rare Dis. 2023;18(1):334.
- 64. Sun Z, Zhang D, Xu G, Zhang N. Laparoscopic treatment of median arcuate ligament syndrome. Intractable Rare Dis Res. 2019;8(2):108-112.
- 65. Bayati IA, Gajendran M, Davis BR, Diaz JR, McCallum RW. Median arcuate ligament syndrome: Clinical presentation, pathophysiology, and management-description of four cases. Gastrointest Disord. 2021;3(1): 44-50.
- 66. Sun Z, Fritz DA, Turner S, Hardy DM, Meiler SE, Martin DC, et al. Celiac plexus block as a predictor of surgical outcome for sympathetically mediated abdominal pain in a case of suspected median arcuate ligament syndrome: A case report. A A Pract. 2018;11(3):76-78.