Review

Median Arcuate Ligament Syndrome—Review of This Rare Disease

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IMPORTANCE Median arcuate ligament (MAL) syndrome is a rare disease resulting from compression of the celiac axis by fibrous attachments of the diaphragmatic crura, the median arcuate ligament. Diagnostic workup and therapeutic intervention can be challenging.

OBJECTIVE To review the literature to define an algorithm for accurate diagnosis and successful treatment for patients with MAL syndrome.

EVIDENCE REVIEW A search of PubMed (1995-September 28, 2015) was conducted, using the key terms *median arcuate ligament syndrome* and *celiac artery compression syndrome*.

FINDINGS Typically a diagnosis of exclusion, MAL syndrome involves a vague constellation of symptoms including epigastric pain, postprandial pain, nausea, vomiting, and weight loss. 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. Surgical intervention involves open, laparoscopic, or robotic ligament release; celiac ganglionectomy; and celiac artery revascularization. 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.

CONCLUSIONS AND RELEVANCE Median arcuate ligament syndrome is rare, and as a diagnosis of exclusion, diagnosis and treatment paradigms can be unclear. Based on previously published studies, symptom relief can be achieved with a variety of interventions including celiac ganglionectomy as well as open, laparoscopic, or robotic intervention.

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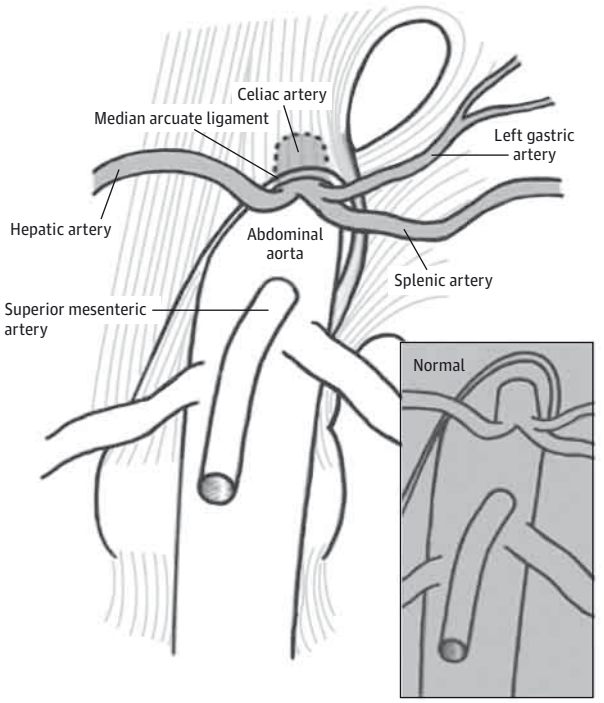
Median arcuate ligament (MAL) syndrome, also known as *celiac artery compression syndrome*, results from an anatomical compression of the celiac axis and/or celiac ganglion by the MAL and diaphragmatic crura (**Figure 1**). This extrinsic compression causes a constellation of symptoms including nausea, vomiting, weight loss, and postprandial epigastric pain. We conducted a search of PubMed (1995-September 28, 2015) using the key terms *median arcuate ligament syndrome* and *celiac artery compression syndrome*, to present a review of this rare disease.

Celiac artery compression was first described anatomically in 1917 by Lipshutz,[1] who noticed in cadaveric dissections that the celiac artery was sometimes overlapped by the diaphragmatic crura. In 1963, Harjola[2] reported on the clinical resolution of postprandial epigastric pain and epigastric bruit in a 57-year-old man following operative decompression of the celiac artery from a fibrosed celiac ganglion. In 1965, Dunbar et al[3] reported a case series involving surgical treatment of MAL syndrome. Despite its characterization several decades ago, MAL syndrome remains a diagnosis of controversy. Skepticism is rooted in an unclear pathophysiologic mechanism, although several theories have been proposed. One commonly accepted theory suggests that increased demand for blood flow through a compressed celiac artery leads to foregut ischemia resulting in epigastric pain, although development of collateral vessels usually prevents the development of ischemia. Another hypothesis is that the pain associated with MAL syndrome has a neuropathic component resulting from a combination of chronic compression and overstimulation of the celiac ganglion. This neuropathic compression may lead to direct irritation of sympathetic pain fibers and/or splanchnic vasoconstriction and ischemia.[4] In addition, vascular steal of blood flow by larger collateral vessels may lead to symptoms of celiac artery compression in patients with an occluded or compressed celiac trunk.[5]

The compressive component of MAL syndrome arises from the close relation of the celiac axis to the celiac plexus, MAL, and dia-

Figure 1. Median Arcuate Ligament in Relation to the Celiac Artery



Note the low-riding median arcuate ligament compared with normal anatomy.

phragmatic crura. Typically, the celiac axis branches off the abdominal aorta between vertebral levels T11 to L1, but wide variation in its origin has been reported.[5] The diaphragmatic crura typically arise from the anterior aspect of L1 to L4 and the anterior longitudinal ligament to join the anterior and superior to the celiac artery. The MAL is a band of fibrous tissue that anteriorly connects the diaphragmatic crura surrounding the aortic hiatus. Individuals with a high origin of the celiac artery or lower insertion of the diaphragm are more prone to compression of the celiac artery. It is proposed[6] that, in 10% to 24% of the population, the MAL crosses the aorta at a lower level and subsequently compresses the celiac artery (Figure 1). However, this infrequent finding is clinically significant in only a small subset of patients, contributing to the controversy surrounding MAL syndrome as a pathologic entity. In vivo, the compression of the celiac artery by the MAL has been demonstrated[7] to be relieved during inspiration as the MAL moves caudally while the compression increases with expiration, further constricting the celiac axis. Compression of the celiac artery has also been postulated[8] to arise in part from the surrounding celiac plexus, which can form thick, fibrous tissue at or near the celiac artery's origin.

Clinical Presentation

Although the incidence of MAL syndrome in the population is not well known, it is more prevalent in women (4:1 ratio) between the ages of 30 to 50 years and in patients with a thin body habitus.[9] The presentation of MAL syndrome is variable. It is most often characterized by chronic postprandial abdominal pain, nausea, vomiting, diarrhea, and unintentional weight loss. The pain is variable but is most often located in the epigastrium. This pain can occur at rest and can be either constant or intermittent. In addition, the pain may be positional, mitigated by leaning forward or drawing the knees to the chest.[10,11] In a Mayo Clinic study[12] of 36 patients, the symptoms of MAL syndrome included abdominal pain (94%), postprandial abdominal pain (80%), weight loss (50%), bloating (39%), nausea and vomiting (55.6%), and abdominal pain triggered by exercise (8%). The physical examination may reveal epigastric tenderness or bruit that is amplified with expiration. However, neither epigastric tenderness nor epigastric bruits are specific for MAL syndrome. Epigastric bruits have been detected in 16% of asymptomatic individuals and 30% of younger patients with MAL syndrome.[13]

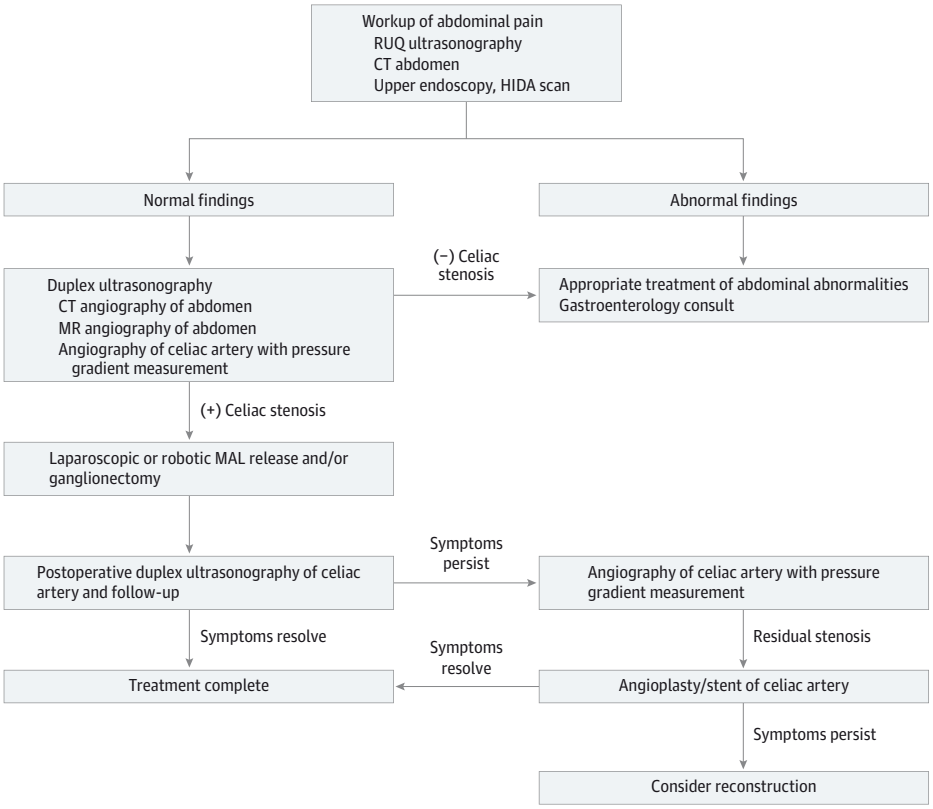
The pathophysiologic mechanism of MAL syndrome is further confounded by the high prevalence of asymptomatic patients exhibiting radiographic evidence of celiac compression. In a review[14] of 400 celiac artery angiograms conducted in asymptomatic patients for chemoembolization of hepatic tumors, 7.3% of these patients had significant celiac stenosis, defined as greater than 50% stenosis and greater than a 10-mm Hg pressure gradient. A factor further contributing to the controversy surrounding MAL syndrome is celiac artery stenosis, which has been documented as a common incidental finding on autopsy. In an autopsy study including 110 unselected patients, Derrick et al[15] found that stenosis of more than 50% was present in the celiac artery of 23 (21%) of the patients.

Diagnosis

Because the symptoms of MAL syndrome closely mimic those of other abdominal disorders, it is commonly considered a diagnosis of exclusion. Based on current literature and our institutional experience, an algorithm for diagnostic evaluation and intervention in patients with MAL syndrome is proposed (**Figure 2**). Patients typically undergo an extensive evaluation for other diagnoses including abdominal ultrasonography, abdominal computed tomography (CT), upper endoscopy, and hepatobiliary iminodiacetic acid scanning (Figure 2). Duplex abdominal ultrasonography during inspiration and deep expiration may be used as a preliminary anatomic and physiologic assessment of celiac compression (Figure 2 and **Figure 3**) with the understanding that the celiac axis tracks cephalad during expiration, leading to external compression and elevated velocities with poststenotic dilatation.[6,16-18] Using duplex ultrasonography in 364 patients, Gruber et al[19] were able to correlate a celiac artery end diastolic velocity of 350 cm/s or greater, a 210% change in pulse volume amplitude with inspiration and expiration, and a celiac artery deflection angle of 50° to a diagnosis of MAL syndrome with high sensitivity (83%) and specificity (100%) compared with findings in patients with angiographic evidence of MAL syndrome and asymptomatic controls.

Additional noninvasive imaging studies to aid in the diagnosis of MAL syndrome include CT angiography and magnetic resonance angiography (Figure 2 and **Figure 4**). Computed tomography angiography offers the advantage of 3-dimensional reconstruction and allows visualization of the compressed artery from different

Figure 2. Algorithm for Diagnosis and Management of Median Arcuate Ligament (MAL) Syndrome



CT indicates computed tomography; HIDA, hepatobiliary iminodiacetic acid; MR, magnetic resonance; and RUQ, right upper quadrant.

angles. Both CT and magnetic resonance angiography enable identification of concomitant abdominal pathology in addition to findings consistent with MAL syndrome. Magnetic resonance angiography can also be used in patients with intravenous contrast allergy and provides results similar to those of CT angiography. Lateral mesenteric angiography can be used to demonstrate celiac artery compression in MAL syndrome. As mentioned above, cephalad movement of the celiac axis during inspiration can reveal celiac artery compression and poststenotic dilatation on expiration. Angiography with breathing maneuvers is the criterion standard of diagnosis (Figure 4). Angiography can also be used for diagnosis if recurrent symptoms develop postoperatively.[11]

As an adjunctive modality, gastric exercise tonometry has been used[20] as a modality to identify gastric ischemia resulting from MAL syndrome. Elevated intramucosal and intraluminal $Paco_2$ levels suggest gastric ischemia. Measurements are taken before, during, and after 10 minutes of submaximal exercise. Criteria for a pathologic result include a gastric arterial $Paco_2$ difference greater than 0.8 kPa after exercise, an arterial lactate level less than 72 mg/dL (to convert to millimoles per liter, multiply by 0.111), and an increase in gastric $Paco_2$ levels after exercise. This modality has been demonstrated in limited studies to be an effective tool for both diagnosis and follow-up assessment in patients with MAL syndrome. Mensink et al[20] identified 29 patients with celiac artery compression using gastric exercise tonometry in a prospective cohort study. Twenty-two patients (76%) underwent celiac artery decompression, and 7 patients (24%) underwent revascularization. Of those revascularized, 5 patients had venous patching of celiac inflow and 2 patients had an antegrade aortoceliac bypass. With a mean follow-up of 39 months, repeat postoperative gastric exercise tonometry demonstrated normal gastric exercise tonometry in all asymptomatic patients and in 1 of 4 patients with persistent symptoms ($P < .001$).

Although imaging studies and gastric exercise tonometry help to identify patients with MAL syndrome, percutaneous celiac ganglion block may identify those who would respond well to surgical treatment.[10,21] The rationale for this procedure rests on the theory that the symptoms of MAL syndrome result from inflammation and compression of the celiac plexus, which serves as a relay center for abdominal visceral afferent fibers carrying pain sensation. The procedure involves percutaneous injection of the celiac ganglion with anesthetic agents (ie, lidocaine and bupivacaine) for short-term relief and ethanol for permanent block. Celiac ganglion block has traditionally been used for the relief of intractable pain typically associated with inoperable malignant disease but has also been used in benign disease, with a subjective 73% reduction in pain compared with a 37% reduction of pain in benign abdominal disease.[22] In a single-center case series[23] of 28 patients with chronic upper abdominal pain who underwent CT-guided celiac plexus block, 21 (75%) of the patients had some relief of pain and 17 patients (61%) of this subset had good relief of pain after the procedure. The Mayo Clinic experience also demonstrated good long-term pain relief in 9 patients who had good response to preoperative celiac ganglion block.[10]

Figure 3. Diagnostic Techniques for Median Arcuate Ligament (MAL) Syndrome

A, Ultrasonography of the celiac axis revealing elevated peak systolic velocity (PSV), end diastolic velocity (EDV), resistive index (RI), and more than a 200% change in pulse volume amplitude with inspiration. B, Computed tomographic angiography (CTA) of the abdomen demonstrating compression of the celiac axis (arrow) with expiration due to obstruction by MAL. C, Magnetic resonance angiography demonstrating stenosis of the celiac axis with poststenotic dilatation, consistent with MAL syndrome. Arrow indicates area of celiac axis occlusion with expiration and an area of poststenotic dilatation of the celiac artery that occurs with proximal obstruction. D, Aortogram with compression of the celiac axis by the MAL with poststenotic dilatation.

Management of MAL Syndrome

The treatment of MAL syndrome is aimed at relieving the compression of the celiac artery to restore adequate blood flow through the vessel and neurolysis to address chronic pain. As with the diagnosis, optimal treatment of MAL syndrome remains an area of controversy.

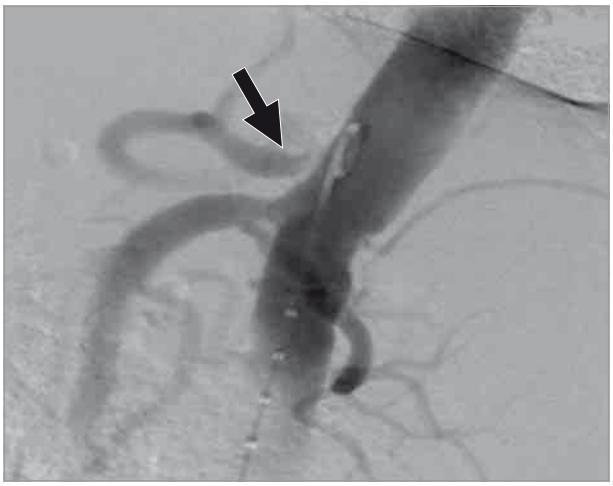
Open Decompression

The most traditional method of treatment of MAL syndrome is through an open approach. Harjola[2] and Dunbar et al[3] were among the first to publish their results following open removal of the celiac plexus and MAL, respectively. Open decompression involves an upper midline laparotomy to access and decompress the MAL and the diaphragmatic crura away from the celiac artery. The diaphragmatic fibers are incised for approximately 5 cm cephalad, exposing up to 4 cm of aorta. Confirmation of MAL release can be done via visual inspection or with intraoperative ultrasonography demonstrating a return to normal peak systolic velocities.[24,25] Neurolysis and wide excision of the involved celiac plexus is also recommended to address the neuropathic component of compression.[24,25] The rationales used for ganglionectomy are that resection compared with simple division may better inhibit reformation of a compressive band, and that ablation of the ganglion will address some of the pain associated with MAL syndrome.[4] The results of surgical interventions for MAL syndrome are summarized in the eTable in the Supplement.

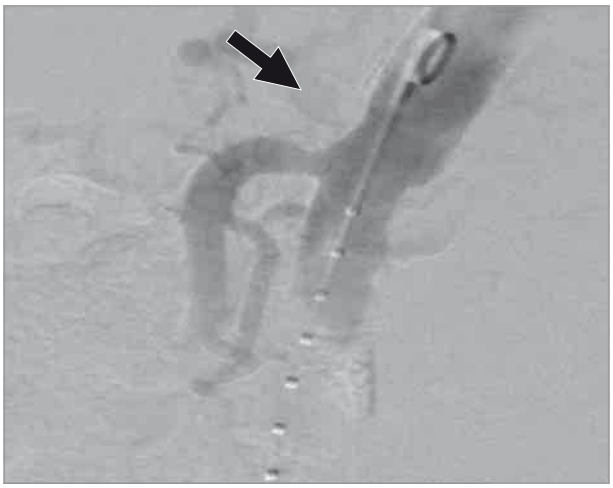
Options for open decompression of the MAL include exploratory laparotomy with decompression alone, decompression with graduated celiac dilatation via celiac or splenic artery arteriotomy, or decompression with reconstruction and bypass of the stenosed arterial segment.[4,25] In what appears to be the largest study to date, Reilly et al[24] report a case series of 51 patients (data collected in a single center from 1964 to 1980) who underwent open decompression alone, decompression with dilatation, or decompression with reconstruction. The study examined the late outcomes of patients who underwent open decompression, with mean follow-up time of 9 years. Regarding patient-reported symptom relief, MAL decompression alone was done in 16 patients, with symptom relief occurring in 9 (56%), and decompression and reconstruction or dilatation was performed in 35 patients, with symptom relief achieved in 27 (77%); however, the difference was not statistically significant. Twenty-eight postoperative arteriograms were performed in 25 patients: 10 patients (40%) were symptomatic and 7 (70%) of those showed persistent celiac stenosis. Eighteen arteriograms were performed in asymptomatic patients and 16 (89%) showed a widely patent celiac axis; the remaining 2 patients (11%) had some persistent

Figure 4. Mesenteric Arteriograms

A During expiration



B During inspiration



A, Mesenteric arteriogram during expiration; arrow indicates obstruction of celiac axis with expiration. B, Mesenteric arteriogram during inspiration; arrow indicates patency of celiac axis with inspiration.

stenosis. These outcomes are supported by a case series[26] of patients who underwent open decompression of the celiac artery without revascularization. In that series, 23 patients (50%) remained asymptomatic at follow-up (6-11 months), and 39 (82%) had partial or complete relief of symptoms. Furthermore, in a series of 18 patients, Grotemeyer et al[27] reported on the outcomes of open decompression of the celiac artery. Eleven of 15 patients (73%) (3 were lost to follow-up) had good resolution of symptoms; 6 of these patients (55%) had decompression of the celiac trunk alone, the other 5 (45%) had additional interventions performed on the celiac trunk.

Reconstruction

The decision for decompression with reconstruction depends on the intraoperative status of the celiac artery after release of the compressive fibers; compromised celiac artery flow after simple MAL release suggests the need for vascular reconstruction. It has been further suggested[28,29] that celiac artery reconstruction be performed in patients with a persistent malformation, thrill, or pressure gradient in the celiac artery despite initial decompression of the MAL fibers. The argument for vascular reconstruction is based on the histologic changes that occur in the celiac artery with chronic compression. The intimal and adventitial layers of the celiac artery undergo hyperplasia with proliferation of abnormal smooth muscle and elastic fibers.[4] Hyperplasia of the arterial wall can lead to a substantial narrowing of the artery lumen necessitating some form of revascularization once the extrinsic compression on the celiac artery is relieved. Options for vascular reconstruction include patch angioplasty of the celiac artery, reimplantation of the celiac artery on the aorta (with or without interposition grafting), and aortoceliac bypass of the stenosed segment with a saphenous vein or polyester (eg, Dacron) graft.

Laparoscopic MAL Release

Laparoscopic decompression of the celiac artery (with and without intraoperative ultrasonography) has become increasingly accepted as standard surgical management for MAL syndrome. Suggested benefits of laparoscopic treatment of MAL syndrome compared with open laparotomy include smaller incisions, decreased postoperative morbidity (including ileus, pain, blood loss, adhesions, and shorter recovery time), and improved view of the surgical field.[28] Disadvantages include difficulty in controlling hemorrhage, potential for incomplete release, and increased risk of injury to the abdominal aorta due to difficult laparoscopic dissection. In addition, fixed stenosis of the celiac artery may necessitate conversion to open or adjunct endovascular angioplasty or stenting as well as the inability to perform concomitant vascular reconstruction.

Roayaie et al[30] were the first to report on laparoscopic management of MAL syndrome. Several case reports[31,32] subsequently described the techniques used for laparoscopic decompression of the MAL. Laparoscopic treatment of MAL syndrome involves the use of 4 to 5 port sites to divide the MAL and skeletonize the celiac artery (with or without vascular intervention) with postoperative angiography to assess celiac artery flow. One consideration with a laparoscopic approach is whether to use intraoperative ultrasonography to assess celiac artery flow after decompression. Although there is no definitive consensus, resolution of the symptoms has been reported[31-33] both with and without the use of intraoperative ultrasonography. Advocates for its use in celiac artery decompression cite its ability to identify anatomy and verify adequate decompression via a decrease in celiac artery flow rate following MAL division. Several case reports[31,33] described the use of intraoperative ultrasonography to assess celiac artery flow and postoperative resolution of symptoms in all patients at 3 to 7 months. In contrast, successful postoperative resolution of symptoms has been achieved with only intraoperative visual inspection of the celiac artery after MAL release. Roseborough[32] reported subjective improvement of symptoms in 14 (93%) of 15 patients treated laparoscopically, with a mean follow-up period of 44.2 months.

Another consideration during laparoscopic MAL release is the presence of persistent stenosis in the celiac axis following division of the MAL. In this setting, the procedure can be converted to an

open approach followed by celiac artery revascularization to restore adequate flow. Alternatively, laparoscopic division of the MAL may be combined with intraoperative or postoperative percutaneous transluminal angioplasty (PTA). In a 1980 case series, Saddekni et al[34] reported the use of PTA in a patient with recurrent stenosis following open MAL decompression. The patient was symptom free at the 18-month follow-up visit. In the series reported by Roseborough,[32] of 15 patients treated laparoscopically, 4 (27%) underwent adjunctive intraoperative or postoperative PTA. Symptoms improved in 3 of these patients; the remaining patient achieved symptom relief only after a celiac artery bypass. In addition, in a series reported by Baccari et al,[35] of 16 patients with MAL syndrome treated laparoscopically, 14 patients (88%) remained asymptomatic (mean follow-up, 28.3 months); the other 2 individuals (12%) required further intervention for symptom relief (PTA and stenting of the celiac artery in one; aortoceliac bypass in the other). These limited studies suggest that PTA may be an adequate adjunct to MAL release in achieving good patient outcomes.

Endovascular Intervention

Although PTA has proved to be useful as an adjunctive therapy to prior surgical MAL division, when used as the sole therapy without MAL division, patient outcomes have been poor. These results may be due to the sustained extrinsic pressure that the intact MAL exerts on the celiac artery, causing a chronic process of intimal hyperplasia and intraluminal narrowing.[28] Multiple case reports[36-39] have demonstrated that endovascular angioplasty alone is unsuccessful at achieving long-term resolution of the symptoms. Several of these case reports describe failure of PTA, with long-term symptom resolution achieved after further surgical intervention. The common theme in these case reports is that each patient required open MAL decompression with reconstruction following failed attempts at endovascular therapy.

Although not a successful first-line therapy, arteriography and PTA with the addition of a balloon-expandable stent serve a role in the treatment algorithm of MAL; the procedure is a useful adjunct in patients with residual symptoms and/or stenosis after operative intervention.[18,40] If symptoms persist after PTA and stenting, mesenteric bypass can be performed (Figure 2).

Emerging Technologies

More recent developments in surgical treatment of MAL syndrome include a robotic-assisted technique for division of the MAL and celiac neurolysis. To our knowledge, Jaik et al[41] were the first to describe their use of a robotic-assisted laparoscopic approach in a 23-year-old woman who was symptom free at the 6-week follow-up visit. More recently, in our institutional experience, 6 patients with MAL syndrome underwent robotic-assisted MAL division with neurolysis of the celiac ganglion. Three of the patients had no recurrent pain at 3-, 11-, and 14-month follow-up visits. The other patients reported a recurrence of symptoms at 3-, 8-, and 13-month follow-up visits. Two of these symptomatic patients underwent subsequent celiac angioplasty with resolution of symptoms at 1 and 4 months. The suggested benefits of robotic-assisted surgery in the setting of MAL division include optic enhancements (increased magnification of structures, 3-dimensional view) and operator-based improvements (tremor elimination, added degrees of motion, and scaled operator movements). Optical enhancements and increased degrees of motion lead to enhanced microdissection at the base of the celiac trunk.[41,42] As with any robotic surgery, limitations of this modality include longer operating time, additional training for the surgeon, and increased cost. Although these limited studies suggest that robotic-assisted treatment of MAL syndrome is effective, further study is warranted.

Conclusions

Although diagnosis and treatment of MAL syndrome are unclear, symptom resolution has been achieved with multiple surgical modalities, including open, laparoscopic, or robotic ligament release as well as celiac ganglionectomy, which often requires celiac artery revascularization. Future work will focus on the fundamentals of better understanding of the pathophysiology, better diagnosis, and improving minimally invasive treatments. Current areas of development include the role of immediate or postoperative revascularization following laparoscopic MAL division. In addition, robotic-assisted techniques in MAL division have preliminarily produced good outcomes but warrant further study.

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REFERENCES

1. Lipshutz B. A composite study of the coeliac axis artery. *Ann Surg*. 1917;65(2):159-169.
2. Harjola PT. A rare obstruction of the celiac artery. *Ann Chir Gynaecol Fenn*. 1963;52:547-550.
3. 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.
4. Bech FR. Celiac artery compression syndromes. *Surg Clin North Am*. 1997;77(2):409-424.
5. 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.
6. Horton KM, Talamini MA, Fishman EK. Median arcuate ligament syndrome: evaluation with CT angiography. *Radiographics*. 2005;25(5):1177-1182.
7. 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.
8. Brandt LJ, Boley SJ. Celiac axis compression syndrome: a critical review. *Am J Dig Dis*. 1978;23(7):633-640.
9. 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.

10. Gloviczki P, Duncan AA. Treatment of celiac artery compression syndrome: does it really exist? *Perspect Vasc Surg Endovasc Ther*. 2007;19(3):259-263.

11. Duffy AJ, Panait L, Eisenberg D, Bell RL, Roberts KE, Sumpio B. Management of median arcuate ligament syndrome: a new paradigm. *Ann Vasc Surg*. 2009;23(6):778-784.

12. Cusati DA, Noel AA, Gloviczki P, et al. Median arcuate ligament syndrome: a 20-year experience of surgical treatment. Presented at: 60th Annual Meeting of the Society for Vascular Surgery; June 1-4, 2006; Philadelphia, PA.

13. Julius S, Stewart BH. Diagnostic significance of abdominal murmurs. *N Engl J Med*. 1967;276(21):1175-1178.

14. 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.

15. Derrick JR, Pollard HS, Moore RM. The pattern of arteriosclerotic narrowing of the celiac and superior mesenteric arteries. *Ann Surg*. 1959;149(5):684-689.

16. 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.

17. Wolfman D, Bluth EI, Sossaman J. Median arcuate ligament syndrome. *J Ultrasound Med*. 2003;22(12):1377-1380.

18. Skeik N, Cooper LT, Duncan AA, Jabr FI. Median arcuate ligament syndrome: a nonvascular, vascular diagnosis. *Vasc Endovascular Surg*. 2011;45(5):433-437.

19. Gruber H, Loizides A, Peer S, Gruber I. Ultrasound of the median arcuate ligament syndrome: a new approach to diagnosis. *Med Ultrason*. 2012;14(1):5-9.

20. Mensink PB, van Petersen AS, Kolkman JJ, Otte JA, Huisman AB, Geelkerken RH. Gastric exercise tonometry: the key investigation in patients with suspected celiac artery compression syndrome. *J Vasc Surg*. 2006;44(2):277-281.

21. Duncan AA. Median arcuate ligament syndrome. *Curr Treat Options Cardiovasc Med*. 2008;10(2):112-116.

22. Lee MJ, Mueller PR, vanSonnenberg E, et al. CT-guided celiac ganglion block with alcohol. *AJR Am J Roentgenol*. 1993;161(3):633-636.

23. Lee JM. CT-guided celiac plexus block for intractable abdominal pain. *J Korean Med Sci*. 2000;15(2):173-178.

24. 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.

25. Kohn GP, Bitar RS, Farber MA, Marston WA, Overby DW, Farrell TM. Treatment options and outcomes for celiac artery compression syndrome. *Surg Innov*. 2011;18(4):338-343.

26. Evans WE. Long-term evaluation of the celiac band syndrome. *Surgery*. 1974;76(6):867-871.

27. 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.

28. Takach TJ, Livesay JJ, Reul GJ Jr, Cooley DA. Celiac compression syndrome: tailored therapy based on intraoperative findings. *J Am Coll Surg*. 1996;183(6):606-610.

29. Loffeld RJ, Overtoom HA, Rauwerda JA. The celiac axis compression syndrome: report of 5 cases. *Digestion*. 1995;56(6):534-537.

30. 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.

31. Vaziri K, Hungness ES, Pearson EG, Soper NJ. Laparoscopic treatment of celiac artery compression syndrome: case series and review of current treatment modalities. *J Gastrointest Surg*. 2009;13(2):293-298.

32. Roseborough GS. Laparoscopic management of celiac artery compression syndrome. *J Vasc Surg*. 2009;50(1):124-133.

33. Carbonell AM, Kercher KW, Heniford BT, Matthews BD. Laparoscopic management of median arcuate ligament syndrome. *Surg Endosc*. 2005;19(5):729.

34. Saddekni S, Sniderman KW, Hilton S, Sos TA. Percutaneous transluminal angioplasty of nonatherosclerotic lesions. *AJR Am J Roentgenol*. 1980;135(5):975-982.

35. Baccari P, Civilini E, Dordoni L, Melissano G, Nicoletti R, Chiesa R. Celiac artery compression syndrome managed by laparoscopy. *J Vasc Surg*. 2009;50(1):134-139.

36. Wang X, Impeduglia T, Dubin Z, Dardik H. Celiac revascularization as a requisite for treating the median arcuate ligament syndrome. *Ann Vasc Surg*. 2008;22(4):571-574.

37. Cinà CS, Safar H. Successful treatment of recurrent celiac axis compression syndrome: a case report. *Panminerva Med*. 2002;44(1):69-72.

38. Delis KT, Gloviczki P, Altuwaijri M, McKusick MA. Median arcuate ligament syndrome: open celiac artery reconstruction and ligament division after endovascular failure. *J Vasc Surg*. 2007;46(4):799-802.

39. Matsumoto AH, Tegtmeyer CJ, Fitzcharles EK, et al. Percutaneous transluminal angioplasty of visceral arterial stenoses: results and long-term clinical follow-up. *J Vasc Interv Radiol*. 1995;6(2):165-174.

40. Columbo JA, Trus T, Nolan B, et al. Contemporary management of median arcuate ligament syndrome provides early symptom relief. *J Vasc Surg*. 2015;62(1):151-156.

41. 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(1):93-96.

42. Relles D, Moudgill N, Rao A, Rosato F, DiMuzio P, Eisenberg J. Robotic-assisted median arcuate ligament release. *J Vasc Surg*. 2012;56(2):500-503.