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Eight Years Experience in the Management of Median Arcuate Ligament Syndrome by Decompression, Celiac Ganglion Sympathectomy, and Selective Revascularization

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Abstract

We aim to review an 8-year experience of median arcuate ligament syndrome (MALS) with chronic gastrointestinal ischemia (CGI) and evaluate clinical outcomes of arcuate ligament decompression, celiac sympathectomy, and selective revascularization. Between December 2002 and March 2012, of 25 patients referred with symptoms of CGI, 11 patients (10 women and 1 man) had clinical signs of abdominal angina and radiological evidence of MALS. Mean age was 50 $\pm$ 20.4 years. Median symptom duration was 34 months. All patients had median arcuate decompression and celiac sympathectomy. In all, 8 did not require revascularization, 2 had retrograde celiac and/or superior mesenteric artery (SMA) stenting, and 1 had SMA bypass. There was no mortality. The 30-day morbidity was 9%. Mean follow-up was 60 months. Eight patients noted complete relief of abdominal pain, and 1 reported some improvement. The MALS is not solely a vascular compression syndrome. The neurological component requires careful celiac plexus sympathectomy in addition to arcuate ligament decompression.

Keywords

median arcuate ligament syndrome, celiac axis compression, celiac ganglion hyperstimulation, surgical decompression, mesenteric revascularization

Introduction

The median arcuate ligament syndrome (MALS)[1-3] is a rare vascular disorder caused by a variable combination of mechanical extrinsic compression of the celiac artery and/or superior mesenteric artery (SMA) together with overstimulation of celiac sympathetic plexus.[4-9]

The MALS is more common in young patients, especially women, but it can occur in more elderly patients in whom symptoms are more likely to be exacerbated by concomitant atherosclerotic arterial disease.[1-10]

Objectives

The aim of this study is to review our 8-year experience at a vascular tertiary referral center with MALS and to evaluate the technical and clinical success rates of arcuate ligament decompression with celiac sympathectomy and selective revascularization. Primary end point is symptom-free survival. Secondary end points include technical success and freedom from major adverse clinical events.

Patients and Methods

This study was a retrospective review of all patients who underwent a procedure for symptoms of MALS and chronic mesenteric ischemia from December 2002 through March 2012, at tertiary referral vascular center. Of the 25 patients referred with symptoms of postprandial pain $\pm$ weight loss, only 11 patients, 10 women and 1 man, had computed tomography angiography (CTA) evidence of eccentric celiac artery compression and MALS.

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Table 1. Presentation.

Onset		Presenting Symptoms	
Acute or chronic	3	Postprandial pain	2
Acute	2	Weight loss (unintentional loss of >5% of body weight over a 6-month period)	1
Gradual	6	Postprandial pain and weight loss	8
Course			
Progressive	7	Smokers	3
Regressive	1	Nonsmokers	8
Steady	3		

The mean age was 50 ± 20.4 years. The series included a mother and a daughter. The mother, aged 47 years at the time of her surgery, had arcuate ligament decompression without revascularization. Eight years later, her daughter, aged 22 years at the time of her surgery, had the same surgery as her mother. The most prevalent vascular risk factors in these patients were hypercholestrolemia (36%) and hypertension (36%). In all, 27% (n = 3) of the patients were either exsmokers or smokers at the time of procedure. In all, 18% (n = 2) had a history of ischemic heart disease. Chronic renal failure was present in 18% of the patients, and 1 patient had a history of chronic obstructive pulmonary disease. None of the patients were diabetic.

In all, 73% (n = 8) of the patients presented with postprandial pain and weight loss of at least 5% of body weight over a 6-month period, 18% with postprandial pain alone, and 1 patient presented with weight loss as their sole symptom. The onset and progression of symptoms are detailed in Table 1. The median duration of symptoms was 34 months (±36).

Diagnostic Workup

All patients had abdominal duplex ultrasound performed when the patient was fasting, and peak systolic velocity (PSV) and end-diastolic velocity (EDV) were measured in the celiac and SMA on both inspiration and expiration. For the SMA, a PSV > 275cm/s[11]and a EDV of 45cm/s[12] were deemed significant. For the celiac, a PSV > 200cm/s[11] and a EDV of 55cm/s[11] were considered diagnostic, with retrograde flow within the hepatic artery regarded as 100% predictive of a severe celiac stenosis or occlusion.[11-14] Additional findings considered to be suggestive of MALS were elevated peak systemic velocities on expiration, which normalize with inspiration[15] and standing erect[16]; abnormal images of celiac artery and SMA origins; and reverse flow in the hepatic artery.[5]

Every patient had angiographic imaging of their gastrointestinal (GI) arteries performed during a breath hold at maximal inspiration on a 64-slice CT scanner, with 1-mm cuts. Three-dimensional reconstructions and measurements were undertaken using 3-mensio software (3mensio Medical Imaging BV, Bilthoven, The Netherlands). The MALS was distinguished from other etiologies of celiac artery stenosis by the characteristic focal narrowing in the proximal celiac artery, with its hooked appearance, best seen on lateral projections (Figure 1).[2,17-20]

Endoscopy was performed on every patient to assess mucosal perfusion of both the upper and the lower GI tract and to out rule any distinct GI pathology that could account for the presenting symptoms. Multidector CT enterography was performed to assess the small bowel.

Four patients had magnetic resonance imaging performed in their referring hospital.

Operative Technique

Open surgical release of the arcuate ligament was performed with the patient under general anesthesia. A 10-cm long upper midline abdominal incision was made, and the aorta identified at the level of the attachments of the diaphragmatic crura after cutting the triangular ligament and mobilizing the left lobe of the liver upward. The celiac axis, SMA, and median arcuate ligament were identified. After dissecting out the branches of the celiac axis, the arcuate ligament and any fibrous and ganglionic tissues overlying the celiac axis were divided and dissected. We routinely perform celiac ganglion sympathectomy by circumferential external arteriolysis, peeling off all neurofibrous tissue around the artery from its origin at the aorta and down to the first major side branch. Three patients required concomitant revascularization procedures. One patient had an SMA bypass procedure, and 2 patients underwent retrograde angioplasty with stenting of SMA, both of whom had occluded celiac axis (Table 2).

All excised tissues, including periarterial ganglionic tissue, were sent for histological analysis and culture.

Follow-Up

Uneventful, symptom-free recovery and a reversion to normal PSV and EDV in the treated splanchnic arteries on follow-up Duplex ultrasonography (US) were indicative of technical success.

Follow-up time points were postoperatively at 6 weeks, at 6 months, and yearly thereafter. Duplex ultrasound was performed at each postoperative follow-up, and CTA was only performed if an abnormality was detected on Duplex US.

Data Collection

Patient demographics, risk factors, comorbidities, history of symptoms, physical findings, preoperative investigations, and postoperative complications were obtained from examining patient charts and the prospectively maintained Vascubase database.

Follow-up data were obtained from the Vascubase system, telephone interviews, and in 1 case, an interview with the patient. Statistical comparison of various variables was carried out using Systat vers. 17.0 software (SPSS Inc, Chicago, Illinois).

A paired *t* test was used to analyze the preoperative and postoperative celiac and SMA PSVs. Statistical significance of the results was defined as $P < .05$.

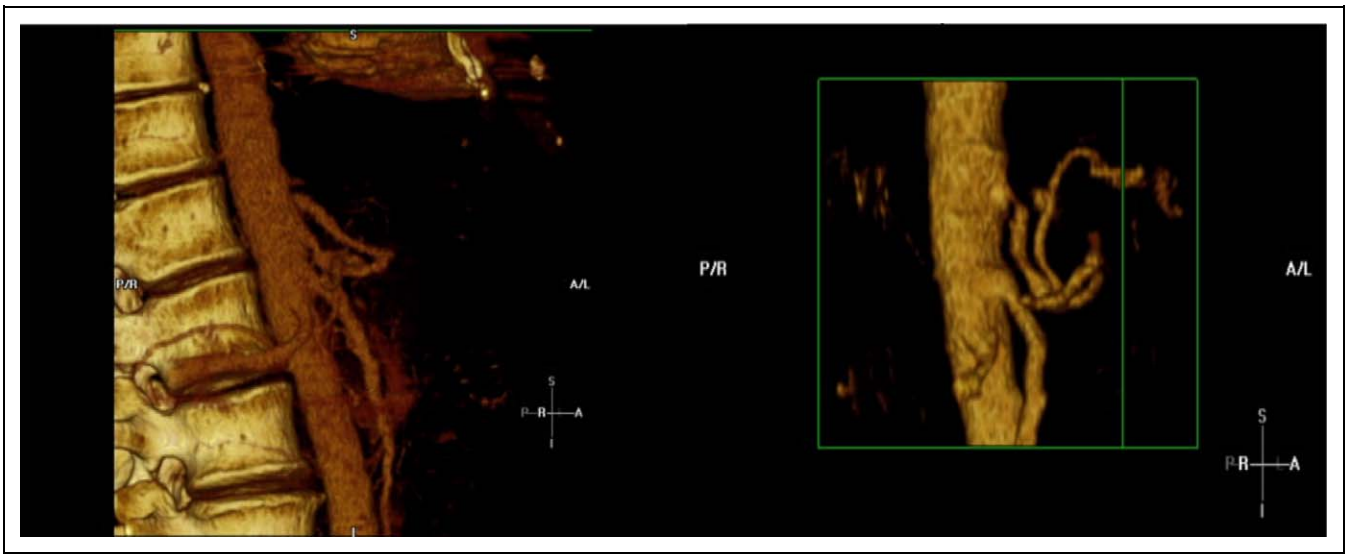

Figure 1. Three-dimensional multichannel computed tomographic angiography of 2 patients with postprandial epigastric pain. Both demonstrate the characteristic focal narrowing in the proximal celiac axis that has a distinct hooked appearance. Poststenotic dilatation of the artery is demonstrated.

Table 2. Procedure Performed and Frequency.

Procedure	Frequency	Percentage
Arcuate ligament decompression	11	100
Bypass of visceral vessels	1	9.1
Angioplasty + stenting	2	18.2
Angioplasty	0	0
Total	11	100.0

Table 3. Pathology.

Pathology	Frequency
Median arcuate ligament compression	11
SMA pathology	2
Both vessel pathology	1
Celiac pathology	2
Total	11

Abbreviation: SMA, superior mesenteric artery.

Results

Patients were followed up for a mean of 60 months ($\pm$32months).

All patients were found to have typical median arcuate ligament compression with "hook sign" demonstrated on lateral projections on CTA. Two patients had concurrent stenosis of the SMA.

In all, 36% (n = 4) of the patients had an magnetic resonance angiography (MRA). All 4 demonstrated celiac stenosis of which 3 had evidence of arcuate ligament compression on MRA. The SMA stenosis was also diagnosed on MRA in 2 of these patients. Two patients had SMA pathology (Table 3).

The mean preoperative celiac PSV was 390 cm/s ($\pm$ 81). The mean postoperative celiac PSV was 275 cm/s ($\pm$124). The mean difference was 115 cm/s, which is just shy of statistical significance ($P = .0567$).

The mean preoperative celiac EDV was 157 cm/s ($\pm$65). Postoperatively, the mean EDV dropped to 62 cm/s ($\pm$38). The difference between pre- and postoperative EDV was not statistically significant ($P = .0752$).

The mean preoperative SMA PSV was 284 cm/s ($\pm$102), and the mean postoperative SMA PSV was 223 cm/s ($\pm$166). The difference in pre- and postoperative SMA PSV values was not statistically significant ($P = .414$).

The mean preoperative SMA EDV was 66 cm/s ($\pm$38), and the mean postoperative SMA EDV was 63 cm/s ($\pm$10). The difference in pre- and postoperative SMA EDV values was not statistically significant ($P = .929$).

The average length of hospital stay was 9.2 days ($\pm$4.6.) The 30-day morbidity was 9% (n = 1). This patient developed acute renal failure and a chest infection 3 days postoperatively. All patients had a postoperative diarrhea denoting successful celiac sympathectomy that settled within 48 hours and disappeared before patients were discharged home. No other major adverse clinical event was reported in any other patient.

There was no mortality in this cohort of patients.

Histology

Four samples of ganglion tissue were reported as neurofibroma on histological analysis.

Intervention-Free Survival

Intervention Free survival was 100%.

Symptom-Free Survival

Over the course of the follow-up period, 8 of the patients noted complete relief of abdominal pain, and 1 patient reported some

improvement (SMA retrograde percutaneous transluminal angioplasty [PTA]). Two patients experienced recurrence and/or worsening of abdominal pain. It was noted that both the patients had a surgical history of adhesionolysis.

Discussion

Chronic gastrointestinal ischemia (CGI) is ubiquitously considered a rare disease entity. However, its prevalence is grossly underestimated, and a number of unchallenged misconceptions have led to misdiagnosis. Patients can be subjected to repeated and exhaustive evaluation of their symptoms with negative outcomes.[21,22] These studies have shown that presenting symptoms can vary considerably and that majority of the patients do not have the classic triad of postprandial pain, weight loss, and abdominal bruit. Furthermore, the generally accepted rule that CGI cannot occur unless at least 2 of the 3 major vessels are stenosed has been disproven, giving credence to the concept of CGI as a multifactorial entity with a neurological component to the pathophysiology. The persistence of these old theorems, until very recently and limited awareness of the spectrum of disease, has made it impossible to accurately determine the prevalence of CGI. However, modern less invasive imaging techniques and functional dynamic MRA testing will allow for more rapid, consistent, and objective diagnosis and enable clearer differentiation of the pathophysiology of various etiologies of CGI.

The median arcuate ligament is a musculofibrous arch uniting both sides of the diaphragmatic crura anterior to the aortic hiatus (T12). The diaphragmatic crura are attached to the anterior surfaces of vertebrae L1 to L4 on the right and on the left from vertebrae L2 to L3. The crura unite with the median arcuate ligament after passing upward and anterior to the aortic hiatus. The ligament is usually superior to the origin of the celiac axis, but as an anatomic variant in 10% to 24% of the population, a low-lying ligament may cause an extraluminal compression at the proximal portion of the celiac artery. The potential constrictive effect of the MAL, together with fibrous bands and periaortic ganglion irritation on the celiac trunk, become most pronounced in expiration.[1-3]

This condition was first described by Lipshutz in 1917, followed decades later by Harjola (in 1963) and Dunbar et al (in 1965).[7,23,24] The MALS as a specific cause of CGI has been much maligned, and its mere existence repeatedly disputed. The principle source of skepticism about MALS can be traced primarily to a publication in 1972 by a preeminent vascular surgeon of the time, Szilagyi, as well as one by Brandt and Boley published in 1978.[25] Szilagyi et al[4] reviewed all the literature on the syndrome and concluded that ''no objective facts have been uncovered and published to prove or even strongly suggest that isolated stenosis of the celiac artery has any pathophysiological effect on the function of the organs supplied by this vessel.'' They deemed treatment unnecessary on the basis of 3 main arguments. First, it was generally assumed that the abundant collateral splanchnic circulation prevents development of symptomatic ischemia in most patients. Second, surgical treatment consisted of dissecting the crural ligament, which probably resulted in neurolysis of the adjacent nerve plexus and thus pain relief. Third, many authors treated these patients and noted variable and often disappointing results.

It should also be borne in mind, as in our series, that the vascular component is not confined to the celiac artery, and studies have shown patients in which MALS has a multivessel component that can involve the SMA or even the renal arteries.[17] This multivessel pathophysiology was demonstrated in our series, in which patients with celiac occlusion had SMA revascularization combined with MAL decompression and ceoliac denervation which resulted in complete symptom relief.

On initial referral, one must be aware that neither patient demographics nor their presenting symptoms are pathognomonic. Although it peaks between the ages of 20 and 40, any age group can be affected. There is a perceived preponderance of this condition in younger women with asthenic habitus, and the female to male ratio is estimated at 3:1.[2,3,24] It is more common in the Asian population.[26] Pediatric patients of MALS have been reported,[27-29] which may have implications for an embryologic origin of this disease. Despite this embryology, many patients present only in their sixth or seventh decades, because the MAL and the celiac artery become slightly lower with advanced age,[30] and this, combined with the concomitant presence of atherosclerostic disease, may trigger symptoms and explain the late onset of the disease manifestations.

In the current study, patients were referred with a variable severity and combination of abdominal angina symptoms such as postprandial pain, unintentional weight loss, and/or abdominal bruit on physical examination. This reflects the erratic presentation of MALS. In a recent prospective study concerning 43 patients with MALS with a mean age of 38 years (range 14-73), the most predominant symptoms were postprandial pain (80%), weight loss (77%), and exercise-related pain (40%). An abdominal bruit on physical examination was found in only 23% of the patients.[21] This is reflective of the results found in our series.

In particular, the mechanism of pain is not sufficiently accounted for by celiac compression alone. Different theories exist, but the most accepted one is that increased blood demand through a compressed celiac artery leads to foregut ischemia and subsequent pain as a variant of a steal syndrome; other, less accepted theories suggest that the pain is caused directly or indirectly by overstimulation of the celiac plexus with subsequent splanchnic vasoconstriction and ischemia.[5,29]

Considering the extrinsic compression from the MAL, the neurological component of celiac plexus irritation and overstimulation and the risk of vessel damage and/or concomitant atherosclerotic disease, treatment must be directed at all 3 pathophysiological elements of MALS.

Open release of the median arcuate ligament[5] as a surgical intervention to manage MALS, as described by Grotemeyer et al,[31] and the removal of overlying periaortic ganglionic tissue relieves the compression of the celiac axis and restores blood flow through the artery.[32] It has been documented as a safe and reliable procedure with no mortality and low morbidity that

depicts our findings. However, long-term results are not favorable unless comprehensive treatment is undertaken to ensure arterial integrity. Several authors report that intraoperative ultrasound helps to monitor the decompressive effect of MAL.[9,33,34]

Reilly et al[18] found concomitant revascularization to have a positive effect on long-term symptom relief, which mirrors our results. Only 8 (53%) of the 15 patients treated by celiac decompression alone remained asymptomatic at late follow-up, in contrast to 22 (76%) of the 29 patients treated by celiac decompression plus celiac revascularization. A subsequent study on 43 patients managed with open surgical decompression, decompression with dilatation, or decompression with reconstruction demonstrated sustained symptom relief in 83% of the patients. Investigators identified patients eligible for celiac artery release using gastric exercise tonometry, which increased the success rate of the surgical intervention.[21]

A few groups have studied the use of laparoscopic treatment of MALS, both transabdominal[10] and retroperitoneal approaches.[33] However, although the experience at Johns Hopkins with a unique surgical series of 15 patients suggests that the celiac artery can be decompressed laparoscopically with minimal morbidity, there is potential for significant vascular injury.[9] This is especially pertinent in the context of multivessel disease.

Although Dasari et al[3] reported on a case of MALS that was treated successfully by way of PTA and stenting only, Sullivan et al[35] describe PTA as traumatic for the vessel in question and claim it renders the vessel wall more susceptible to collapse by intense extrinsic pressure such as the median arcuate ligament could exert.

Delis et al[6] argue that stent patency is compromised by slippage and mechanical stress and that stent insertion into the vessel without primary arcuate release first must only be considered as a temporary treatment. Silva et al[36] reported on 59 patients who had underwent stent placement in 79 (>70%) stenotic mesenteric arteries, but by 14 months, 29% had restenosis with half requiring reintervention. Baccari et al[37] and Delis et al[6] have demonstrated that this method alone has proved insufficient in eradicating the extrinsic pressure exerted by the ligament on the celiac axis and maintaining adequate perfusion through the vessel and should be performed as a complementary procedure only after release of the extrinsic compression of the CA. However, even with prior laparoscopic release of MAL, endovascular therapy is rarely suitable for patients with MALS, a sizeable proportion of who are young and have a arterial lesion that is an eccentric response to repetitive injury rather than atherosclerotic disease.

The decision to treat the complications of MALS is clear, but the decision to intervene when MAL variant anatomy is diagnosed incidentally is less so. It may not be the presence of the ligament sling alone but also the extent and severity of celiac compression that is related to both the phase of respiration and the phase of the cardiac cycle as well as the degree of ligament tension and elastic recoil upon the celiac axis. Poststenotic dilatation to any degree, vessel collateralization, gastroduodenal artery dilatation, and, indeed, pancreaticoduodenal artery aneurysm formation might be secondary features to indicate adverse hemodynamics, even in the absence of symptoms that should prompt consideration of surgical management.[38]

Reticence about open repair of MALS has stemmed from fear of performing a laparotomy, with its associated morbidity, without definitive proof of arterial or neurological compression; furthermore, in physicians who perform relatively few cases, the need for revascularization can be intimidating. The CTA has overcome many of these issues without the need for invasive angiography. Furthermore, for the treatment of MALS, it is best to refer to experienced tertiary referral centers for open surgical repair. Excellent results are possible in such centers, including a recent series from the Mayo Clinic, with mortality of 0.9% in low-risk patients.[39]

Conclusion

The MALS existence as a syndrome has been categorically proven by intraoperative findings supported by modern testing techniques. Contemporary imaging and functional testing allow us to make informed decisions on patient selection and advances such as electrocardiogram-gated CTA, and dynamic MRA will make these decisions even clearer. Successful treatment necessitates open surgical decompression and sympathectomy with selective revascularization.

Declaration of Conflicting Interests

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