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Median Arcuate Ligament Syndrome and Recurrent Colitis With Hematochezia: Persistent Symptoms After Technically Successful Robotic Release

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Abstract

Median arcuate ligament syndrome (MALS) is an uncommon diagnosis caused by extrinsic compression of the celiac artery and surrounding celiac plexus by the median arcuate ligament. The diagnosis is challenging because celiac artery compression may be incidental and presenting symptoms may overlap with those of more common gastrointestinal pathologies. We report a 55-year-old woman with recurrent abdominal pain, nausea, vomiting, hematochezia, and 30-40 pounds of unintentional weight loss after multiple admissions for presumed ischemic colitis. Mesenteric duplex ultrasound demonstrated greater than 70% celiac artery stenosis without superior mesenteric artery stenosis, and computed tomography angiography (CTA) showed narrowing of the celiac ostium with a hooked configuration. Esophagogastroduodenoscopy (EGD) was normal. She underwent robotic release of the median arcuate ligament, dissection of celiac plexus neural tissue, and repair of an incidental hiatal hernia, and the celiac artery was decompressed from its origin to the trifurcation. Over the following nine months, she was readmitted repeatedly with abdominal pain, diarrhea, and hematochezia, including episodes of left-sided and transverse colitis and one episode of descending colon diverticulitis, while serial CTA showed widely patent mesenteric arteries. Her weight remained stable, but chronic abdominal pain persisted. Colonic bleeding in a patient with celiac compression should not be attributed to MALS, and persistent symptoms after technically adequate release should prompt evaluation for coexisting ischemic, inflammatory, infectious, diverticular, and functional causes.

Categories: Radiology, Pain Management, General Surgery
Keywords: celiac artery compression, celiac plexus block, ischemic colitis, median arcuate ligament syndrome (mals), recurrent hematochezia, robotic surgery procedures

Introduction

Median arcuate ligament syndrome (MALS) is a rare neurovascular compression syndrome in which a low-lying median arcuate ligament compresses the proximal celiac artery and the adjacent celiac plexus [1,2]. Its pathophysiology remains incompletely understood. Both vascular and neurogenic mechanisms have been proposed, including reduced celiac artery flow, irritation of the celiac plexus, and sympathetically mediated splanchnic vasoconstriction [1,2]. Patients classically present with chronic or recurrent epigastric pain, postprandial pain, nausea, vomiting, early satiety, food avoidance, and weight loss [1,2].

The diagnosis is difficult because these symptoms are nonspecific and the characteristic imaging findings also occur in asymptomatic individuals [2,3]. Computed tomography angiography (CTA) can demonstrate focal narrowing of the proximal celiac artery with a hooked configuration, which favors extrinsic compression over atherosclerotic stenosis [4]. Duplex ultrasound typically shows elevated celiac artery velocities that increase in expiration, and current guidelines recommend measurement in both inspiration and expiration [5]. Imaging alone, however, does not establish symptomatic MALS, and conventional imaging findings do not reliably predict clinical response after release [3,6].

We present a patient with severe epigastric pain, nausea, intolerance of oral intake, and substantial unintentional weight loss in the setting of radiographic and intraoperatively confirmed median arcuate ligament compression. Her clinical course was further complicated by recurrent hematochezia and episodes of left-sided colitis before and after robotic release. The coexistence of these distinct clinical manifestations illustrates the difficulty of establishing the symptomatic significance of celiac artery compression in patients with concurrent gastrointestinal pathology.

Case Presentation

A 55-year-old woman with a surgical history notable for two cesarean deliveries and a laparoscopic myomectomy presented with recurrent episodes of severe abdominal pain followed by bloody diarrhea. She

had no known peripheral vascular disease, hypercoagulable disorder, inflammatory bowel disease, or autoimmune disease, and she had never smoked. Her family history was notable for a mother who reportedly died of ischemic bowel disease.

Her symptoms began approximately four months before operative intervention. She described abrupt, severe abdominal cramping with nausea, vomiting, and bright red blood per rectum. During an early admission, flexible sigmoidoscopy demonstrated sigmoid diverticulosis with mucosal ulceration and continuous linear ulcerations compatible with ischemic colitis. Histopathological examination of the nonulcerated mucosa demonstrated preserved crypt architecture without significant inflammation, favoring a self-limited process. Ischemic colitis remained a presumptive diagnosis based on the clinical presentation and endoscopic findings, as the available histopathological findings did not specifically establish ischemic injury. She was managed supportively but continued to experience recurrent episodes.

During a recurrent presentation, CTA of the abdomen and pelvis demonstrated patent mesenteric vessels without active bleeding and mild colonic wall thickening from the splenic flexure to the sigmoid colon. Given the absence of peritonitis, active extravasation, or mesenteric arterial occlusion, nonoperative management was recommended. The working differential diagnosis included ischemic colitis, infectious colitis, and diverticular bleeding.

Mesenteric duplex ultrasonography demonstrated elevated celiac artery velocities, with a peak systolic velocity (PSV) of 234 cm/s and an end-diastolic velocity (EDV) of 69 cm/s, interpreted by the radiologist as consistent with greater than 70% stenosis (Figure *1*). No significant atherosclerotic plaque was identified. The superior mesenteric artery (SMA) did not meet duplex criteria for significant stenosis, and the inferior mesenteric artery (IMA) could not be visualized because of overlying bowel gas and patient discomfort. A separate visceral venous duplex demonstrated patent hepatic, portal, and splenic veins with normal flow direction.

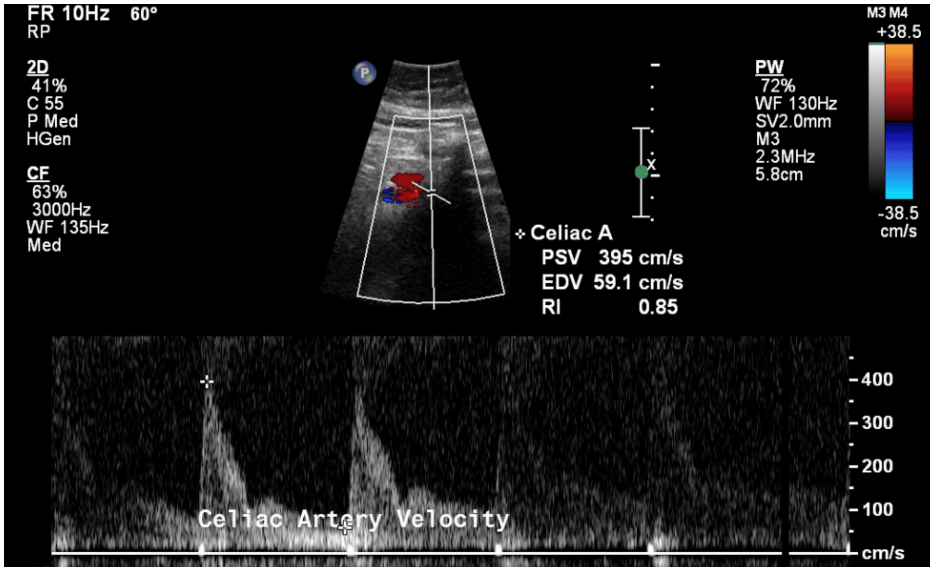


FIGURE 1: Mesenteric arterial duplex

Duplex ultrasound showing elevated celiac artery velocities (peak systolic velocity 234 cm/s, end-diastolic velocity 69 cm/s) consistent with greater than 70% stenosis but without evidence of superior mesenteric artery stenosis.

Several weeks later, she re-presented with severe epigastric and periumbilical pain, lightheadedness, nausea, and dry heaving, without hematemesis, hematochezia, or melena. She reported persistent intermittent abdominal pain, decreased appetite, and poor oral intake, with approximately 30 pounds of weight loss over the preceding month and up to 40 pounds over several months. An outpatient colonoscopy after the prior admission had shown mild sigmoid diverticulosis, small internal hemorrhoids, and two 5-mm tubular adenomas, which were removed. Biopsies of mildly erythematous right and left colonic mucosa showed no acute inflammation, chronic mucosal injury, or microscopic colitis. On examination, she was afebrile and hemodynamically stable. Her abdomen was soft and nondistended, with mild epigastric, left upper quadrant, and suprapubic tenderness and no rebound or guarding. Preoperative labs were notable for metabolic acidosis and mild acute renal injury, with a normal serum lactate level of 0.8 mmol/L. Renal function and bicarbonate returned to normal after surgery; however, there was a slight hypokalemia and a drop in hemoglobin from 14.2 to 11.7 g/dL. Serum lactate was raised to 2.7 mmol/L, although it dropped to 0.7 mmol/L after intravenous fluid resuscitation. Table *1* summarizes perioperative laboratory results.

Laboratory Test	Preoperative	Postoperative	Reference range
Blood urea nitrogen (mg/dL)	22	14	7-20
Creatinine (mg/dL)	1.29	0.64	0.50-1.10
Bicarbonate (mmol/L)	15	23	22-29
Potassium (mmol/L)	3.7	3.3	3.5-5.1
Hemoglobin (g/dL)	14.2	11.7	12.0-15.5
Hematocrit (%)	40.7	33.5	36.0-46.0

TABLE 1: Pertinent abnormal laboratory values before and after median arcuate ligament release

Repeat CTA before surgery demonstrated prominent narrowing of the celiac artery ostium with a hooked configuration consistent with median arcuate ligament compression (Figure *2*). There was also focal narrowing of the post-ostial proximal SMA of unclear etiology, with patent distal SMA and celiac branches, and no bowel wall thickening, pneumatosis, portal venous gas, or other acute abnormality. Although the patient had a history of recurrent hematochezia and left-sided colitis, her symptoms during the admission leading to surgery were predominantly epigastric and periumbilical pain, nausea, poor oral intake, and significant unintentional weight loss. She denied hematochezia, hematemesis, and melena during this presentation. These clinical findings, together with elevated celiac artery velocities on duplex ultrasonography and characteristic proximal celiac artery narrowing on CTA, supported the clinical suspicion of symptomatic MALS. A diagnostic celiac plexus block was considered; however, given the severity of her symptoms and impaired oral intake, the patient elected to proceed with operative decompression during the same admission.

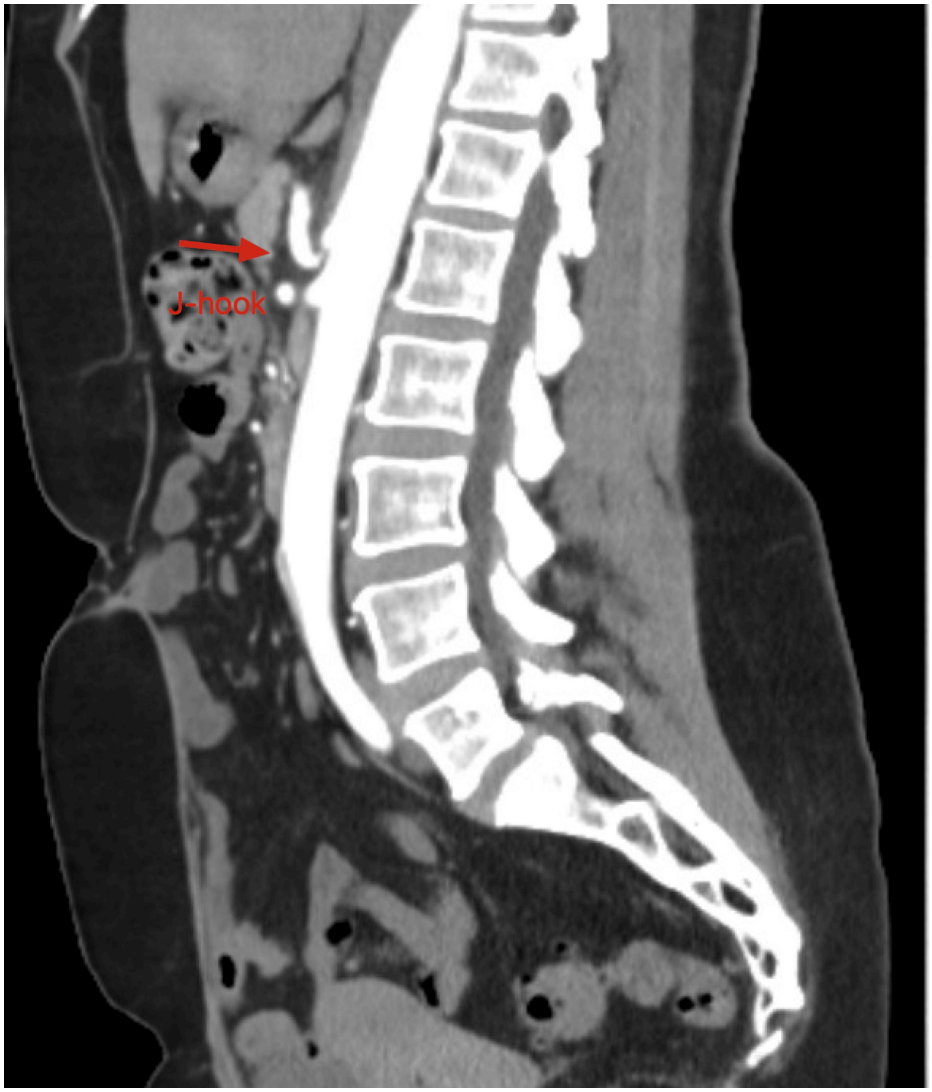


FIGURE 2: Pre-operative CTA abdomen and pelvis

Prominent narrowing of the celiac artery ostium with appearance of classic median arcuate ligament compression with a J-hook appearance.

The patient underwent esophagogastroduodenoscopy (EGD), robotic-assisted release of the median arcuate ligament with dissection of celiac plexus neural tissue, and repair of an incidental small hiatal hernia. EGD showed no significant abnormality of the esophagus, stomach, or duodenum. The gastrohepatic ligament was opened to access the celiac axis. A small hiatal hernia was encountered, and the herniated portion of the stomach was reduced from the chest. The esophagus was circumferentially dissected with preservation of the anterior and posterior vagus nerves. The left gastric and hepatic arteries were identified, the overlying tissue was dissected to the adventitial layer, and the celiac artery was followed proximally to the aorta.

A prominent median arcuate ligament was found compressing the celiac trunk and was divided. Dissection was carried proximally along the abdominal aorta for approximately 5 cm, releasing the surrounding tissue to the adventitial layer, and inferiorly along the aorta beyond the celiac origin. Neural tissue of the celiac plexus and surrounding tissue were dissected away from the celiac artery and adjacent vascular structures. At completion, the celiac artery was free from its origin to the trifurcation, and the common hepatic, left gastric, and splenic arteries were each dissected for approximately 3 cm to confirm that no residual compression remained.

The patient reported complete relief of abdominal pain for several days following surgery. Approximately two weeks postoperatively, she presented with recurrent postprandial epigastric and left lower quadrant pain, nausea, vomiting, and nonbloody diarrhea. She described the pain as different from her preoperative symptoms but was unable to further characterize this distinction. Subsequent readmissions were associated with recurrent abdominal pain and hematochezia. CTA demonstrated a widely patent celiac trunk, SMA, and IMA without radiographic evidence of mesenteric ischemia, colitis, or enteritis (Figure *3*). An upper

gastrointestinal series approximately four weeks after surgery was normal, with no recurrent hiatal hernia or gastroesophageal reflux and normal fluoroscopic gastric emptying. Gastroenterology, surgery, and internal medicine teams were unable to establish a definitive etiology for her recurrent symptoms. Supportive care, proton pump inhibitor therapy, and outpatient rheumatologic evaluation for possible vasculitis were recommended. Pain medicine recommended multimodal analgesia, and an outpatient celiac plexus block was arranged.

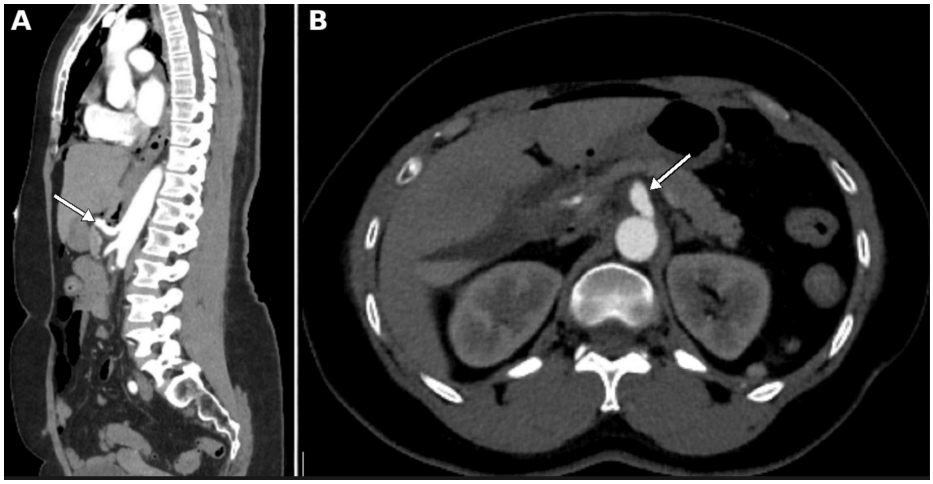


FIGURE 3: Postoperative computed tomography angiography after median arcuate ligament release

(A) Sagittal view showing the celiac artery (arrow) with significant improvement of the stenosis and improved morphology compared with the preoperative imaging. (B) Axial view at the level of the celiac artery origin showing a patent celiac trunk arising from the aorta (arrow).

Approximately three months after surgery, she was admitted to an outside hospital for colitis. CT there showed sigmoid-predominant colitis with lesser transverse colon involvement, of possible infectious, inflammatory, or ischemic etiology, and sigmoid wall thickening possibly related to longstanding diverticular disease. Magnetic resonance imaging showed colitis of the sigmoid and descending colon and a focal area of circumferential mid-transverse colonic wall thickening with restricted diffusion. EGD and colonoscopy were reported to show diverticulosis, with biopsy results pending. She was discharged on amoxicillin-clavulanate for possible diverticulitis but developed vomiting, diarrhea, hematochezia, and abdominal pain after one day of therapy and presented to our institution three days after discharge. CTA showed a fluid-filled colon suggestive of diarrheal illness, with unremarkable celiac, SMA, renal, and IMA origins. A multiplex enteric pathogen polymerase chain reaction (PCR) panel was negative. *Clostridioides difficile* testing was positive for glutamate dehydrogenase antigen and negative for toxin by enzyme immunoassay, and the toxin gene was reportedly detected by PCR. Infectious disease consultants considered this colonization rather than active infection and recommended observation without directed therapy. Because the mesenteric vessels were patent, no further surgical intervention was recommended, and she improved with supportive care. The outpatient celiac plexus block was not performed because of recurrent hospitalizations and referral for further vascular evaluation.

In contrast to the preoperative CTA showing a hooked celiac artery, CTA on three occasions after release demonstrated widely patent celiac, SMA, and IMA origins. Taken together, the postoperative laboratory and imaging findings made residual celiac compression or large-vessel mesenteric arterial obstruction unlikely as the explanation for her recurrent hematochezia and abdominal pain. The clinical course is summarized in Table *2*.

Timing relative to release	Event	Key findings
About four months before	Onset of abrupt abdominal pain and hematochezia	Flexible sigmoidoscopy: sigmoid diverticulosis with linear ulcerations; biopsy favored a self-limited process
Before surgery	Recurrent admission; mesenteric duplex	CTA: patent vessels, wall thickening from splenic flexure to sigmoid. Duplex: celiac PSV 234 cm/s, EDV 69 cm/s; SMA normal
About two months before	Outpatient colonoscopy	Mild sigmoid diverticulosis; two tubular adenomas removed; random biopsies without inflammation
Index admission	Epigastric pain, poor intake, 30-40 lb weight loss	AKI and metabolic acidosis with normal lactate. CTA: hooked celiac origin; focal proximal SMA narrowing
Day 0	EGD; robotic median arcuate ligament release; hiatal hernia repair	Normal EGD; prominent ligament compressing celiac trunk divided; artery free to trifurcation
Within two months after	Two readmissions for pain and bloody stools	CTA: celiac, SMA, IMA widely patent. Upper GI series normal. Rheumatology referral; celiac plexus block arranged
About three months after	Outside admission for colitis; readmission	CT and MRI: sigmoid, descending, and mid-transverse colitis. Lactate 2.7 to 0.7 mmol/L with fluids; CTA patent; C. difficile antigen positive, toxin negative
About five months after	Left lower quadrant pain, diarrhea	CTA: distal descending colon diverticulitis; normal mesenteric vessels
Five to eight months after	Outside multidisciplinary evaluation	Symptoms not attributed to MALS by vascular surgery; concern for Crohn's disease; eosinophilic esophagitis
About nine months after	One day of hematochezia	CT: diverticulosis without colitis; stool burden. Symptoms resolved; weight stable

TABLE 2: Timeline of the Clinical Course

AKI: acute kidney injury; CTA: computed tomography angiography; EDV: end-diastolic velocity; EGD: esophagogastroduodenoscopy; GI: gastrointestinal; IMA: inferior mesenteric artery; MALS: median arcuate ligament syndrome; MRI: magnetic resonance imaging; PSV: peak systolic velocity; SMA: superior mesenteric artery

Discussion

The indication for operative intervention in this patient was based primarily on her recurrent epigastric pain, nausea, impaired oral intake, and substantial unintentional weight loss in conjunction with concordant vascular imaging findings, rather than her history of hematochezia and left-sided colitis. Although these findings supported a clinical diagnosis of MALS, the possibility of incidental celiac artery compression with an alternative cause of abdominal pain could not be excluded. Intraoperative confirmation of ligamentous compression established the anatomical abnormality but did not establish a causal relationship between the compression and all of her gastrointestinal symptoms.

Her presentation, however, was not classic. MALS typically causes foregut symptoms such as epigastric pain, nausea, vomiting, food avoidance, and weight loss [1,2]. Hematochezia and left-sided colitis are not typical features. The celiac artery supplies foregut structures, whereas the descending and sigmoid colon depend on the IMA, with collateral supply from the SMA through the marginal artery and the arc of Riolan. When the celiac origin is compressed, its branches are perfused largely by retrograde flow from the SMA through the pancreaticoduodenal arcades [5]. Whether this redistribution can compromise perfusion at the colonic watershed remains speculative, and her clinical course after decompression did not support this mechanism. Although the initial endoscopic findings were suggestive of ischemic colitis, the available histopathological findings did not specifically demonstrate ischemic injury. The microscopic description characterized the nonulcerated mucosa, limiting histopathological assessment of the observed ulcerations. Ischemic colitis therefore remained a presumptive diagnosis. Her recurrent hematochezia was more suggestive of coexisting colonic pathology than a direct consequence of celiac artery compression, although its underlying etiology was not definitively established.

The initial episodes of abrupt abdominal cramping followed by bloody diarrhea and segmental left-sided colonic involvement were consistent with the clinical presentation of colonic ischemia, which occurs more frequently in women and often develops in the absence of major mesenteric arterial occlusion [7]. Recurrent episodes of poor oral intake, dehydration, and transient renal dysfunction raise the possibility that intravascular volume depletion contributed to episodic colonic hypoperfusion, particularly in watershed regions. Although serial CTA demonstrated patent mesenteric vessels, these findings do not exclude transient nonocclusive colonic ischemia. The focal proximal SMA narrowing observed on preoperative CTA warrants consideration, as concomitant SMA narrowing and celiac artery compression could potentially limit mesenteric perfusion reserve and collateral circulation. However, duplex ultrasonography did not demonstrate hemodynamically significant SMA stenosis, and subsequent postoperative CTA demonstrated a patent SMA without significant proximal narrowing. The clinical significance of this finding therefore remains undetermined. Her maternal history of fatal ischemic bowel disease is also noteworthy, although the underlying etiology was unknown.

The initial sigmoidoscopic biopsies favored a self-limited process, while random colonic biopsies obtained two months before surgery showed no chronic mucosal injury, arguing against established inflammatory bowel disease at that time. Following release, the patient experienced recurrent episodes of colitis involving the sigmoid, descending, and transverse colon, an episode of descending colon diverticulitis, and subsequent hematochezia without radiographic evidence of colitis. The interpretation of C. difficile testing further complicated the diagnostic evaluation. A positive glutamate dehydrogenase assay and toxin gene PCR with a negative toxin assay indicate the presence of a toxigenic organism but do not independently distinguish colonization from active infection, supporting the use of multistep diagnostic algorithms that incorporate toxin detection [8]. Although transient nonocclusive colonic ischemia may account for some of her clinical presentations, the findings across multiple admissions suggest a potentially multifactorial process for which no definitive unifying diagnosis was established.

The patient's serial imaging studies reflect the diagnostic challenges associated with the dynamic nature of celiac artery compression. Initial CTA obtained during evaluation for gastrointestinal bleeding demonstrated patent mesenteric vessels, whereas subsequent duplex ultrasonography revealed elevated celiac artery velocities, and repeat CTA demonstrated the characteristic hooked configuration of the proximal celiac artery. Duplex velocity thresholds for grading celiac artery stenosis vary, and respiratory variation is particularly relevant in MALS, as compression is accentuated during expiration and diminishes during inspiration [5]. Although respiratory variation may account for the differences between imaging studies, the available duplex measurements were insufficient to establish this relationship. Moreover, celiac artery compression is well documented in asymptomatic individuals, and conventional imaging findings do not reliably predict symptomatic improvement following surgical release [3,6]. Careful correlation of clinical and radiographic findings therefore remains fundamental to appropriate surgical patient selection.

Celiac plexus block has become an important adjunct in the evaluation of suspected MALS. Pain relief after a block supports a neurogenic component and may identify patients more likely to benefit from release [9,10]. In a multicenter cohort of 516 patients, absence of relief after a block was associated with a higher likelihood of treatment failure [11]. In this case, a block was considered before surgery, but the patient proceeded directly to release because of symptom severity and her preference for definitive treatment. A postoperative block was arranged but was not performed because of recurrent hospitalizations. In retrospect, a preoperative block would have helped distinguish neurogenic foregut pain from the lower abdominal pain associated with her colitis.

Operative treatment focuses on decompression of the celiac artery and the surrounding neural tissue. Open, laparoscopic, and robotic approaches have all been described, and a review of 400 open and laparoscopic cases reported immediate symptom relief in 85% [12]. The robotic approach provides stable visualization and fine instrument control around the aorta, the celiac branches, and the celiac plexus [13]. In this patient, the operation confirmed a prominent median arcuate ligament compressing the celiac trunk; the artery was released from its origin to the trifurcation, and each branch was dissected for several centimeters to confirm complete decompression.

The patient's initial postoperative course was notable for complete relief of abdominal pain for several days, followed by recurrent postprandial epigastric and left lower quadrant pain that she described as qualitatively different from her preoperative symptoms. This transient improvement suggests that celiac artery compression or associated neural irritation may have contributed to her initial symptoms. However, the recurrence of both upper and lower gastrointestinal symptoms limited the assessment of sustained MALS-specific symptom relief. CTA on three occasions after release demonstrated widely patent celiac, SMA, and IMA origins, and an upper gastrointestinal series showed no recurrent hiatal hernia or reflux. These findings argue against residual fixed celiac artery compression or large-vessel mesenteric obstruction as the primary cause of her recurrent symptoms. Persistent symptoms after median arcuate ligament release should prompt further diagnostic evaluation rather than automatic attribution to failed decompression. In this patient, continued multidisciplinary evaluation was appropriate, as the differential diagnosis remained broad and included recurrent ischemic colitis, Crohn's disease, diverticular disease, infectious colitis, C. difficile colonization versus infection, vasculitis, constipation, functional bowel disorders, postoperative adhesions, and chronic neurogenic abdominal pain.

This report has several limitations. The patient did not return for the recommended outpatient rheumatologic evaluation or celiac plexus block, limiting further characterization of the etiology of her persistent symptoms. Although she reported transient postoperative pain relief, the absence of longitudinal, symptom-specific assessment precluded determination of sustained improvement in MALS-related symptoms independently of her recurrent lower gastrointestinal complaints. Furthermore, the operation combined arterial decompression with dissection of periarterial neural tissue without formal celiac ganglionectomy, making it difficult to distinguish the relative contributions of vascular and neurogenic mechanisms to symptom relief. Nevertheless, this case illustrates that MALS may coexist with gastrointestinal pathology outside the celiac vascular territory and emphasizes the importance of distinguishing anatomic decompression from clinical improvement while maintaining a broad differential diagnosis when symptoms persist after surgical release.

Conclusions

The diagnosis of MALS requires careful clinical and radiographic correlation, as anatomic celiac artery compression alone does not establish symptomatic disease. In this patient, robotic median arcuate ligament release achieved anatomic decompression and transient symptomatic relief; however, recurrent abdominal pain, diarrhea, and hematochezia persisted despite patent mesenteric vessels on serial imaging. The underlying etiology of her recurrent symptoms remained undetermined, and a causal relationship between celiac artery compression and her colonic manifestations could not be established. This case illustrates the distinction between anatomic success and clinical outcomes and emphasizes the importance of investigating alternative or coexisting gastrointestinal pathology when symptoms persist after surgical decompression.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mohamad Ballan, Richard Marino, Anne Akharoh, Josh Spada, Brian Nam

Acquisition, analysis, or interpretation of data: Mohamad Ballan, Josh Spada, Brian Nam

Drafting of the manuscript: Mohamad Ballan, Richard Marino, Anne Akharoh, Josh Spada, Brian Nam

Critical review of the manuscript for important intellectual content: Mohamad Ballan, Anne Akharoh, Josh Spada, Brian Nam

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors used OpenEvidence (Florida, USA), ChatGPT (OpenAI, California, USA), and Claude (Anthropic, California, USA) as follows: language polishing, structure or formatting in Introduction, Case Presentation, Discussion, Tables 1 & 2, and figure legends. AI was used to assist with manuscript organization and language editing. The authors reviewed the clinical information and verified the references and interpretations. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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