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A Clinical Case of Laparoscopic Treatment of Median Arcuate Ligament Syndrome

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Abstract:

Median arcuate ligament syndrome (MALS), also referred to as Dunbar syndrome, is a rare vascular compression disorder characterized by extrinsic compression of the celiac artery by the median arcuate ligament. It commonly presents with vague gastrointestinal symptoms such as postprandial epigastric pain, nausea, anorexia, weight loss, and occasionally diarrhea. However, radiologic evidence of celiac artery compression is not uncommon in asymptomatic individuals, reported in up to 10–24% of cases, indicating that anatomical compression alone is insufficient for diagnosis. The pathophysiology of MALS is multifactorial, with a possible neurogenic component involving altered pain processing from the splanchnic plexus. As MALS is a diagnosis of exclusion, thorough evaluation is necessary to rule out other potential causes. Surgical decompression of the median arcuate ligament—via open, laparoscopic, or robot-assisted approaches—is the primary treatment, with endovascular therapy reserved for persistent stenosis following surgery.

This article presents a clinical case exemplifying the laparoscopic treatment of MALS. A 28-year-old female patient with a 7–8 year history of nonspecific abdominal symptoms experienced worsening pain and significant weight loss (18 kg) over the past month. Diagnostic imaging with duplex ultrasonography and computed tomography revealed a 75% proximal stenosis of the celiac artery. The patient underwent successful laparoscopic release of the median arcuate ligament. Postoperatively, all gastrointestinal symptoms resolved, peristalsis resumed within 12 hours, and the patient was able to eat normally the following day. Additionally, the abdominal bruit resolved. She was discharged one day after surgery and remained symptom-free during a 5-month follow-up period.

Keywords: Median arcuate ligament, Dunbar syndrome, celiac artery, laparoscopic decompression, vascular compression syndrome

Background

Median Arcuate Ligament Syndrome (MALS), also known as Dunbar syndrome, is a rare cause of chronic abdominal pain, resulting from external compression of the celiac artery and/or celiac plexus by the median arcuate ligament — a fibrous band connecting the right and left crura of the diaphragm. In some individuals, the ligament inserts lower than normal, leading to mechanical compression of the proximal celiac artery, especially during expiration.

The syndrome most commonly affects young, thin women and presents with nonspecific gastrointestinal symptoms such as postprandial epigastric pain, nausea, vomiting, bloating, diarrhea, and unintentional weight loss. These symptoms often mimic more common gastrointestinal disorders, leading to delayed or missed diagnosis.

Although imaging may reveal celiac artery compression in a portion of the healthy population (10–24%), only some individuals become symptomatic. This discrepancy suggests that neurogenic mechanisms — specifically, irritation of the celiac plexus — may play a central role in the development of pain and gastrointestinal symptoms.

Given the overlap with other GI conditions, MALS is considered a diagnosis of exclusion. Diagnostic workup includes Doppler ultrasound, computed tomography angiography (CTA), and magnetic resonance angiography (MRA). Treatment is primarily surgical, involving decompression through open, laparoscopic, or robotic approaches. Endovascular procedures are generally reserved for cases with persistent stenosis after decompression.

Case Presentation

The patient is a 28-year-old female with a 7–8 year history of chronic abdominal discomfort. Over the past year, the abdominal pain progressively worsened, becoming constant throughout the day and night. The pain intensified significantly after food intake and was accompanied by nausea and vomiting. As a result, the patient experienced a dramatic weight loss of 18 kg over the past month. She also reported a sensation of heaviness in the epigastric region, heartburn, persistent nausea, vomiting, constipation, marked general weakness, and severe fatigue even with minimal physical activity. Additional symptoms included headaches, insomnia, dizziness, and a subjective sensation of increased pulsation in the abdominal aorta. She also complained of coldness in her extremities.

Diagnostic Findings:

Duplex ultrasound revealed a stenotic lesion exceeding 70%. Peak systolic velocity (PSV) during expiration reached 240 cm/s, decreasing to approximately 200 cm/s during inspiration.

CT angiography (Figures A and B) demonstrated a localized 75% stenosis of the proximal celiac trunk.

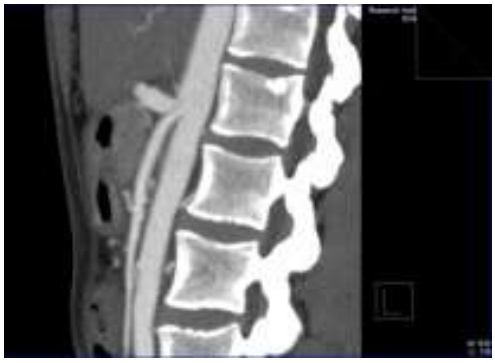
Surgical Treatment:

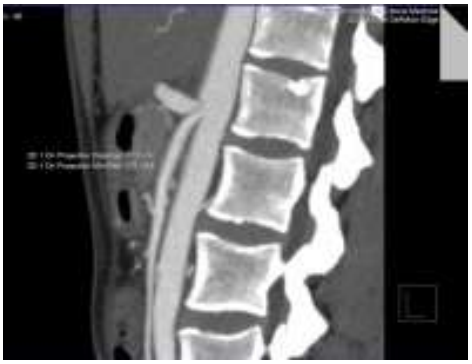
The patient underwent laparoscopic surgery: **"Dissection and release of the median arcuate ligament from the abdominal aorta."** This procedure is also referred to by some authors as a **celiac trunk ganglionectomy**, due to the presence of neurofibrous infiltration [1].

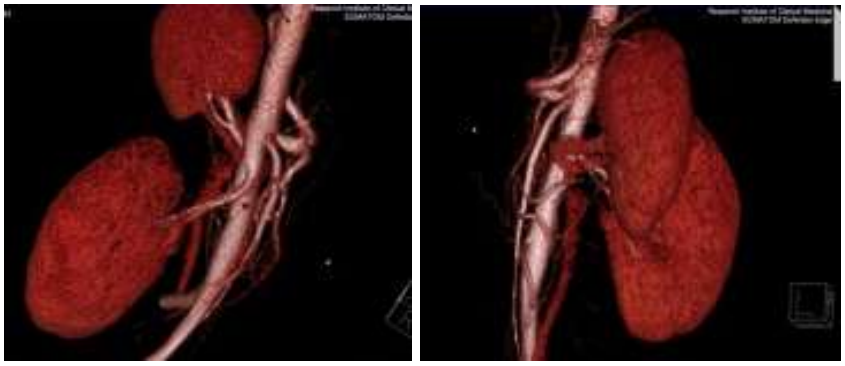
Postoperative Outcome:

The patient was discharged 24 hours after surgery. Remarkably, she reported complete resolution of abdominal pain immediately upon awakening from anesthesia. All pathological abdominal symptoms disappeared postoperatively, and she noted a warming sensation in her extremities. Intestinal peristalsis returned within 12 hours. On the morning following surgery, the patient was able to tolerate oral intake without recurrence of symptoms. The disturbing sensation of abdominal aortic pulsation was no longer present.

At follow-up one week post-surgery, the patient reported feeling very well, with no specific complaints. It is noteworthy that she continued to exhibit low blood pressure (approximately 80/40 mmHg), which occasionally caused mild discomfort in the chest area. As of five months postoperatively, the patient remains symptom-free, with no recurrence of her preoperative complaints. She is currently under periodic follow-up by a cardiologist and angiologist. A control CT angiography is planned six months after the procedure.







Discussion

Median Arcuate Ligament Syndrome (MALS), also known as Dunbar Syndrome or celiac artery compression syndrome, is a rare and often underdiagnosed vascular condition characterized by the external compression of the celiac trunk by the median arcuate ligament of the diaphragm. The syndrome was first described clinically by Harjola in 1963, who associated abdominal symptoms with compression of the celiac artery [2]. Subsequently, in 1965, Dunbar and colleagues analyzed 13 patients and confirmed that the clinical manifestations of postprandial abdominal pain and weight loss were due to ischemia caused by extrinsic compression of the celiac trunk by the median arcuate ligament [3]. Since then, the condition has frequently been referred to as "Dunbar Syndrome."

MALS predominantly affects young individuals, typically between the ages of 20 and 40, with a higher prevalence in women. In many cases, the syndrome remains asymptomatic or manifests with vague gastrointestinal complaints, leading to misdiagnosis or delayed diagnosis. Common symptoms include postprandial epigastric pain, nausea, vomiting, weight loss, early satiety, and sometimes diarrhea or constipation. Due to the nonspecific nature of these symptoms, MALS is considered a diagnosis of exclusion.

Advances in imaging modalities such as **duplex ultrasound** and **CT angiography** have improved the ability to detect MALS. Duplex sonography may reveal elevated peak systolic velocities (PSV) of the celiac artery, especially during expiration, which normalizes during inspiration—a dynamic feature characteristic of MALS [4,5]. In the clinical case presented here, duplex ultrasound revealed >70% stenosis with a PSV of 240 cm/s during expiration and 200 cm/s during inspiration. CT angiography confirmed a 75% localized stenosis of the proximal segment of the celiac trunk [6].

The primary goal of treatment is to restore normal blood flow in the celiac artery. While traditional open surgical decompression was historically the mainstay of treatment, **minimally invasive approaches** such as laparoscopic and robot-assisted techniques are now preferred due to their numerous advantages. These include better visualization of the aortic region, reduced intraoperative blood loss, lower rates of cardiopulmonary complications, less postoperative pain, shorter hospital stay, and reduced risk of adhesions [2,7]. In our case, laparoscopic release of the median arcuate ligament was

successfully performed. Notably, this was the **first such laparoscopic intervention reported both in Georgia and in the post-Soviet region**.

Although some authors propose **percutaneous angioplasty with stenting** as an alternative or adjunctive treatment—particularly in cases where traditional surgery is contraindicated or fails to relieve residual stenosis—this remains a subject of ongoing debate. Endovascular techniques are generally considered secondary interventions after surgical decompression.

Despite increasing recognition, many questions about MALS remain unanswered due to the rarity of the condition and the lack of large-scale clinical studies. The variability in clinical presentation, combined with the frequent presence of radiological compression in asymptomatic individuals, complicates the establishment of standardized diagnostic and therapeutic guidelines.

In conclusion, Dunbar Syndrome is a rare but potentially debilitating condition that requires high clinical suspicion and thorough radiological assessment for diagnosis. Surgical intervention, especially through minimally invasive techniques, remains the cornerstone of treatment and often leads to significant symptom relief and quality-of-life improvement in carefully selected patients.

Conclusion

Dunbar Syndrome (MALS) is a rare and often overlooked cause of chronic abdominal symptoms, primarily affecting young women. Timely diagnosis through advanced imaging and a high index of clinical suspicion is crucial. Minimally invasive surgical treatment, particularly laparoscopic release of the median arcuate ligament, has shown excellent outcomes and remains the treatment of choice. Continued clinical awareness and further research are needed to better understand the pathophysiology and optimize management strategies for this complex condition.

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ფაშვის ღეროს კომპრესიული სინდრომის ლაპარასკოპიული მკურნალობის კლინიკური შემთხვევა

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ივანე ჯავახიშვილის სახელობის თბილისის სახელმწიფო უნივერსიტეტი,მედიცინის ფაკულტეტი

აბსტრაქტი

ფაშვის ღეროს კომპრესიული სინდრომი, რომელიც საერთაშორისო სახელწოდებით ცნობილია როგორც Median Arcuate Ligament Syndrome (MALS) ან დანბარის სინდრომი, წარმოადგენს იშვიათ ვასკულურ პათოლოგიას, რომელსაც ახასიათებს მედიანური რკალისებური იოგის მიერ ფაშვის ღეროს კომპრესია. კლინიკურად ვლინდება მუდმივი და ძლიერი ტკივილი მუცელში ანუ „მუცლის ანგინა“გულისრევა, მადის დაქვეითებას, წონის კლება და ზოგჯერ დიარეა. პაციენტების 10-24% მიმდინარეობს უსიმპტომოდ.დიაგნოსტიკა ეფუძნება დუპლექს სკანირებასა და ანგიოგრაფიულ კვლევებს (CT/MR), რომლებიც აფასებენ სტენოზის ხარისხსა და ჰემოდინამიკურ დარღვევებს.

სტატიაში, მოყვანილია კლინიკური შემთხვევის მაგალითი, ფაშვის ღეროს კომპრესიული სინდრომის ლაპაროსკოპიული გზით ქირურგიულ მკურნალობას. პაციენტი — 28 წლის ქალი — წლების განმავლობაში უჩიოდა პერიოდულ მუცლის ტკივილს. დაავადების ხანგრძლივობა შეადგენდა დაახლოებით 7–8 წელს, თუმცა ბოლო ერთ წელში ტკივილის ინტენსივობა საგრძნობლად გაიზარდა, ამ პერიოდში პაციენტმა დაიკლო 18 კგ. დიაგნოსტიკური კვლევებიდან ჩატარდა დუპლექს ულტრასონოგრაფია და კომპიუტერული ანგიოგრაფია, რომლებმაც აჩვენა ფაშვის ღეროს 75%-მდე ლოკალური სტენოზი პროქსიმალურ სეგმენტში. პაციენტს ჩაუტარდა ლაპაროსკოპიული ოპერაცია -მედიანური რკალისებური იოგის გაკვეთა-განცალკევება მუცლის აორტიდან ოპერაციის შემდგომ კლინიკური სიმპტომები პაციენტს მოეხსნა

პაციენტი გაეწერა ოპერაციიდან 24 საათში. ხუთთვიანი დაკვირვების პერიოდში პაციენტი თავს გრძნობს კარგად და სიმპტომები არ განმეორებულა.

საკვანძო სიტყვები: შუა რკალისებრი იოგი, დანბარის სინდრომი, ფაშვის ღეროს კომპრესიული სინდრომი, მუცლის ანგინა.