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Celiac artery occlusion from median arcuate ligament compression complicating a hemorrhagic duodenal ulcer repair

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ABSTRACT

We present a case of a hemorrhagic duodenal ulcer complicated by occlusion of the celiac artery (CA) by acute median arcuate ligament (MAL) compression. Angiography revealed retrograde flow through the gastroduodenal artery (GDA) to the hepatic artery, with occlusion at the CA origin. This unique presentation required coordinated release of the MAL to reestablish antegrade CA flow before pyloroplasty and GDA ligation. The presence of preexisting MAL compression of the CA should be considered during the repair of bleeding duodenal ulcers through embolization or ligation of the GDA, because impaired CA perfusion could result in foregut ischemia. (J Vasc Surg Cases Innov Tech 2024;10:101371.)

Keywords: Acute median arcuate ligament syndrome; Celiac artery stenosis; Duodenal ulcer; Gastroduodenal artery; Median arcuate ligament release

The median arcuate ligament (MAL) is a fibrous arch that connects the right and left crura of the diaphragm at the aortic hiatus. Anatomic variations can lead to direct compression of the proximal celiac artery (CA) by the MAL. Significant compression often presents as MAL syndrome (MALS) and its characteristic clinical features of foregut ischemia such as postprandial epigastric pain, weight loss, and food aversion.[1] Significant CA compression might not be recognized because many individuals remain asymptomatic, likely due to collateral circulation via the gastroduodenal artery (GDA). Therefore, the diagnosis and surgical release of preexisting MAL compression (MALC) of the CA is important in surgical cases that require ligation of the GDA. We present a case of a hemorrhagic duodenal ulcer with concomitant CA occlusion by MALC that required coordinated MAL release (MALR) before pyloroplasty and GDA ligation. The patient provided written informed consent for the report of her case details and imaging studies. She also provided written informed consent for her treatment, which includes consent for educational activities and publications in accordance with the Cleveland Clinic Department of Research, because this case report does not include protected health information (ie, HIPAA [Health Insurance Portability and Accountability Act of 1996] identifiers).

CASE REPORT

A 36-year-old woman presented to an outside hospital emergency department with massive hematemesis and hypovolemic shock. At presentation, her hemoglobin was 3.0 g/dL and her hematocrit was 11.3%. She quickly went into cardiac arrest with pulseless electrical activity arrest and underwent 15 minutes of cardiopulmonary resuscitation. A massive transfusion protocol was rapidly initiated, and she received 9 U of packed red blood cells. The patient arrived at the intensive care unit intubated and sedated with vasopressor support. Her significant laboratory values included hemoglobin of 9.8 g/dl, hematocrit of 30.3%, and lactate of 7.5 mmol/L. An orogastric tube placed at the outside facility had frank bloody output of 550 mL.

Upper gastrointestinal tract bleeding was suspected, and a high-dose proton pump inhibitor and intravenous octreotide were initiated. Esophagogastroduodenoscopy revealed blood clots throughout the entire stomach and proximal duodenum; however, the source of the bleeding was not identified. Subsequently, the patient again became unstable and again required initiation of a massive transfusion protocol. As a temporizing measure, a second esophagogastroduodenoscopy was performed, showing active extravasation from a 3-cm ulcer extending from the pylorus to the duodenal bulb. Two hemostatic clips were placed over the ulcer. At this time, the patient was hemodynamically stable.

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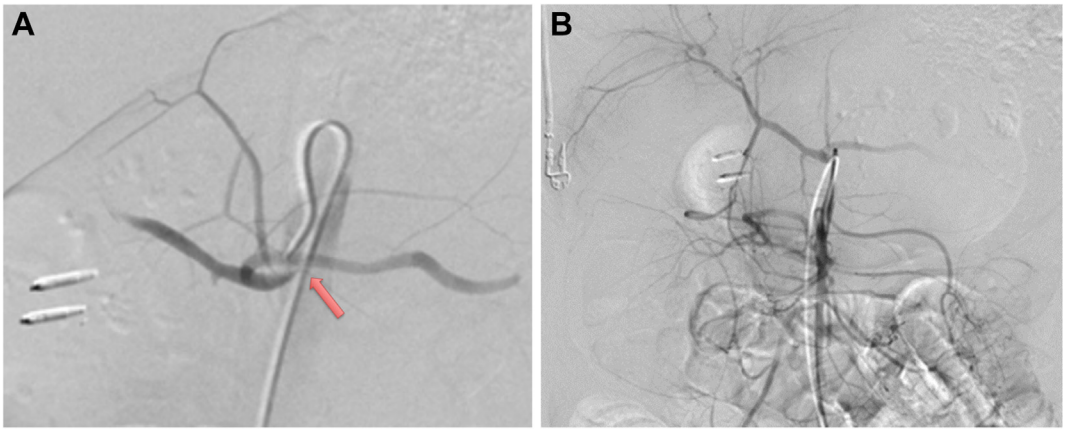

Fig 1. Angiogram revealing near complete occlusion (*red arrow*) at the origin of the celiac artery (CA) suspicious for median arcuate ligament (MAL) compression (**A**) and subsequent retrograde flow through the gastroduodenal artery (GDA) and flowing into the common hepatic artery and splenic artery (**B**).

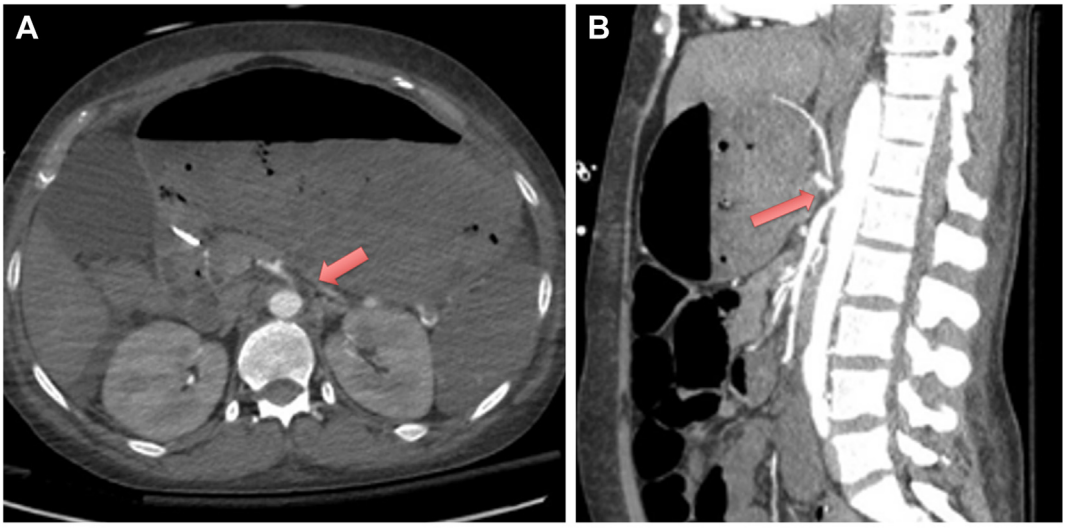

Fig 2. Transverse (**A**) and sagittal (**B**) sections of computed tomography angiography showing >90% stenosis of the celiac artery (CA) origin (*red arrow*) with "trace string-like attenuation."

A mesenteric angiogram was performed by interventional radiology in an attempt to control the bleeding through GDA embolization. Angiographic imaging revealed high-grade CA stenosis at the origin with retrograde filling through the GDA into the common hepatic and splenic arteries suspicious for MALC (Fig 1). The procedure was then aborted to preserve flow to the celiac branches and minimize the risk of hepatic infarction. Computed tomography angiography revealed >90% stenosis of the proximal CA with "trace string-like attenuation" (Fig 2).

At this point, vascular and general surgery decided to take the patient to the operating room for an exploratory laparotomy. When entering the abdominal cavity, the stomach was massively distended, obstructing the operative field. Therefore, a small gastrostomy was performed along the greater curvature of the stomach to allow for clot evacuation. The distal CA was exposed, which had a nonpalpable pulse and weak monophasic signals on intraoperative Doppler ultrasound. The supraceliac aorta was exposed via the lesser sac, and the origin of the CA appeared thickened and inflamed, with complete compression by the MAL. The MAL was divided, and the celiac plexus was lysed, restoring antegrade flow through the CA. The GDA was then temporarily clamped to assess the inline flow through the CA, which now exhibited a strong Doppler signal and palpable pulse. Next, general surgery performed a longitudinal enterotomy, extending from the distal gastric antrum across the pylorus onto the first portion of the duodenum. This facilitated exposure of the bleeding ulcer, which was controlled, and the GDA was ligated with 3-0 Prolene suture. After GDA ligation, we confirmed that the CA and its associated branches

still had a palpable pulse. On achieving hemostasis, pyloroplasty using a Heineke-Mikulicz technique was performed to close the enterotomy.

The patient was discharged on postoperative day 8. At her 1-month follow-up, the patient remained asymptomatic. Duplex ultrasound at follow-up confirmed a widely patent CA without overlying compression.

DISCUSSION

Hemorrhage remains the most common cause of death related to peptic ulcer disease. Emergent operative repair is indicated for hemorrhagic peptic ulcers that require >4 to 6 U of blood and that continue bleeding after attempted endoscopic techniques. For massive hemorrhagic posterior duodenal ulcers, emergent surgical ligation or angiographic embolization of the GDA should be considered in an attempt to control the bleeding. However, it is important to first visualize adequate flow through the CA and its branches to avoid ischemia to organs of the foregut.[2]

In our patient, angiographic embolization or surgical ligation of the GDA before MALR would likely have resulted in catastrophic ischemia to the celiac region. The presence of preexisting CA stenosis should be considered before sacrificing the GDA. This is especially important when there is not a preoperative diagnosis of MALS due to a patient's unknown medical history or lack of symptoms despite significant CA compression. Our patient arrived as an emergent transfer and did not have known diagnosis of MALS. However, the high-volume retrograde flow through the GDA as a result of CA occlusion likely contributed to the significant amount of hemorrhage from the ulcer.

In patients with CA stenosis, flow to the celiac region is dominantly preserved by collateral flow through the GDA and the pancreaticoduodenal arcade from the superior mesenteric artery. When GDA division is required, patients with preexisting CA occlusion can be at risk of ischemia to the celiac region. Therefore, revascularization of the occluded CA was required to preserve arterial flow to the liver and biliary tree in our patient. The initial operative plan was to perform aortic–celiac bypass. However, MALC of the CA was found and released to restore antegrade flow through the CA.

Pancreaticoduodenectomy (PD) commonly requires GDA division. Ischemic complications such as hepatic cytolysis, pancreatic tail necrosis, and anastomotic rupture have been reported after PD when preexisting MALC was not released.[3-5] These potential ischemic complications can be prevented when preexisting MALC is diagnosed and released before GDA division during PD, as described by Yamamato et al.[6] Similarly, Rolff and Storkholm[7] reported a case in which MALR was performed immediately after emergent GDA ligation during repair of a traumatic penetrating pancreatic injury, decreasing the risk of foregut ischemia. The diagnosis of MALC should be suspected when computed tomography angiography shows a characteristic "hooked" appearance at the CA origin.[6] Temporary intraoperative clamping of the GDA resulting in weakened hepatic arterial signals via Doppler ultrasound also suggests MALC of the CA and might indicate the need for MALR before GDA ligation. Immediately after MALR, the hepatic artery should be palpable during temporary GDA clamping, such as was seen in our case. This maneuver is important in confirming that the hepatic artery is supplied via antegrade flow from the CA.

CONCLUSIONS

The presence of preexisting CA occlusion and MALC of the CA should be considered during surgical procedures that require sacrificing the GDA, because impaired CA perfusion can result in catastrophic ischemia to the liver and foregut. Our case demonstrates successful coordinated MALR followed by pyloroplasty and GDA ligation in a patient presenting with a hemorrhagic duodenal ulcer complicated by preexisting CA compression by the MAL.

DISCLOSURES

None.

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