Endovascular repair and management of a ruptured inferior pancreaticoduodenal artery aneurysm: A case report and literature review

Pavel Kibrik, DO, Jerry Zhu, BS, Ajit Rao, MD, Daniel Han, MD, Peter Faries, MD, *and* James Cornwall, MD, *New York, NY*

ABSTRACT

Inferior pancreaticoduodenal artery aneurysm (IPDA) with the stenosis of the celiac axis is rare and may cause rupture. A unique etiology of IPDAs with celiac stenosis is median arcuate ligament syndrome. These aneurysms develop as a result of the dilation of the arteries from the retrograde blood flow into the pancreaticoduodenal arches because of celiac artery compression by the median arcuate ligament. We describe a 39-year-old man whose ruptured IPDA was associated with celiac artery stenosis and who was managed with coil embolization. The patient has agreed to have their case details and images published. (J Vasc Surg Cases Innov Tech 2025;11:101650.)

Keywords: Endovascular repair; IPDA; Celiac artery compression; Median arcuate ligament; Coil embolization

CASE REPORT

A 39-year-old man with a history of childhood asthma and psoriatic arthritis was referred from urgent care to the emergency department with nausea, vomiting, syncope, loss of appetite, and pain in the abdomen for 1 day. At the time of admission, his blood pressure was 102/71 mm Hg, and he had a pulse of 120/min. Blood panels showed decreased serum hemoglobin (9.3 g/dL), decreased hematocrit (25.7%), and a slightly decreased platelet count (143,000/μL). Physical examination was notable for voluntary abdominal guarding and pain upon movement. Purpura was noted bilaterally over the lower quadrants. Upon imaging, a computed tomography angiogram of the abdomen and pelvis revealed a hematoma in the retropancreatic space and free fluid in the abdomen layering above the liver (Fig 1). Active extraversion was not identified. General surgery was consulted, and the patient was taken to the operating room for an exploratory laparotomy to control bleeding and for an abdominal washout. We evacuated 1050 mL of blood and clots. No active areas of bleeding were found throughout the mesentery, but a retroperitoneal hematoma around the base of the transverse mesocolon was noted.[1] Vascular surgery was consulted intraoperatively. Upon close review of prior imaging, an IPDA was identified with celiac axis occlusion (Fig 2). Given that the retroperitoneal hematoma seemed to be stable and was not expanding, the decision was made to close the abdomen and prepare the patient for coil embolization on an angiography table, because there were no fluoroscopic capabilities on the operating room table.

Right common femoral artery access was obtained with a 5F sheath. A selective angiogram of the superior mesenteric artery (SMA) confirmed occlusion of the celiac axis with retrograde filling via the SMA. Subsequent opacification of the gastroduodenal, common hepatic, proper hepatic, and splenic arteries was also noted on angiography. A cystic pseudoaneurysm was noted 8 to 10 mm distal to its origin from the proximal SMA. A 3F microcatheter was placed in the proximal pancreatic arcade to obtain a selective angiogram of the pancreaticoduodenal arcade. Multiple views were taken to ensure the IPDA could be cannulized and to ensure the gastroduodenal artery remained patent, because this was the primary blood supply to the celiac axis (Fig 3). Angiography also revealed hypertrophy of the vertical branch of the dorsal pancreatic artery; that is, the arch of Beuhler. Coil embolization was performed by deploying six microcoils (Nester embolization coil, Cook Medical, Bloomington, IN) in the IPDA pseudoaneurysm from distal to proximal. Onyx liquid embolization (ONYX, Medtronic, Minneapolis, MN) was then performed to cease flow to the aneurysm. A repeat angiogram showed satisfactory embolization of the IPDA (Fig 4). Median arcuate ligament syndrome (MALS) was suspected based on a combination of clinical symptoms and selective angiography showing focal narrowing of the proximal celiac artery with poststenotic dilation.[2] Celiac artery revascularization was considered but not performed because MALS was suspected as the underlying etiology of the aneurysm, and median arcuate ligament decompression was not undertaken at the time. The patient was extubated in the intensive care unit on postoperative day 3. The patient's pain was stabilized, and he was discharged on postoperative day 9 with no complications.

DISCUSSION

IPDAs occur infrequently and are thought to account for only 2% of all visceral aneurysms. Despite their rarity,

From the Division of Vascular Surgery, Department of Surgery, The Mount Sinai Medical Center.

Correspondence: Pavel Kibrik, DO, Division of Vascular Surgery, Icahn School of Medicine at Mount Sinai, 1425 Madison Ave, 4th Floor Box 1273, New York, NY 10029 (e-mail: pkibrik@gmail.com).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2468-4287

https://doi.org/10.1016/j.jvscit.2024.101650

Journal of Vascular Surgery Cases, Innovations and Techniques
■■■ 2025

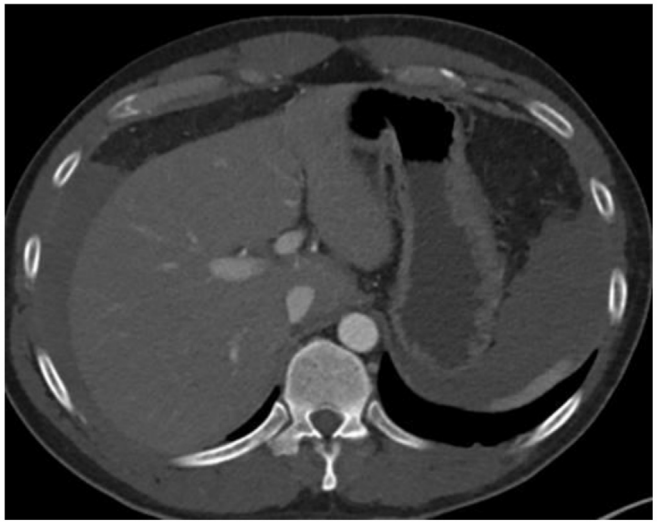

Fig 1. Contrast-enhanced computed tomography images showed a hematoma in the retropancreatic space and free fluid in the abdomen, layering above the liver.

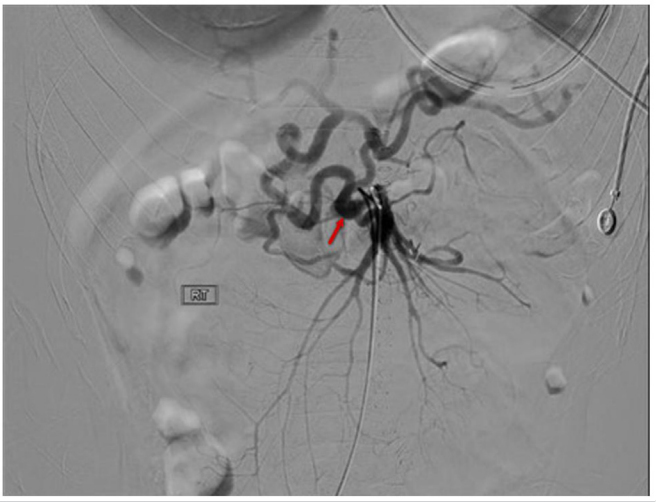

Fig 3. Superior mesenteric arterial angiography shows marked dilation of the gastroduodenal artery, pancreaticoduodenal arcades, and the arch of Beuhler. A cystic aneurysm can be seen 8 to 10 mm distal from the origin of the proximal superior mesenteric artery (SMA).

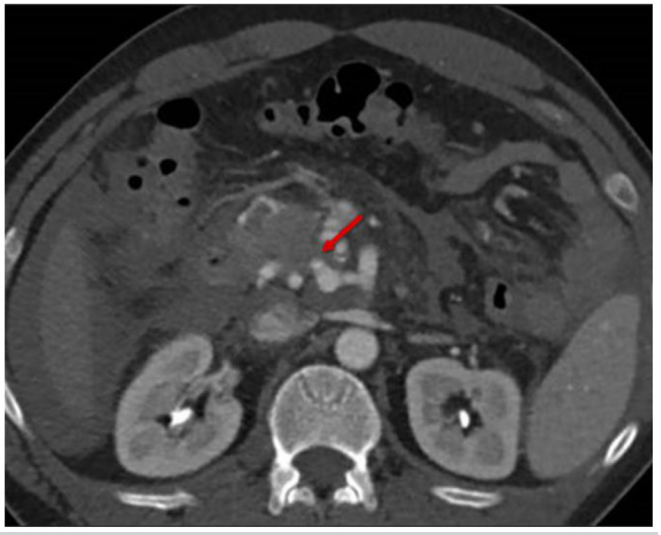

Fig 2. Initial computed tomography angiography revealed an inferior pancreaticoduodenal artery aneurysm. Active extravasation could not be determined based on imaging.

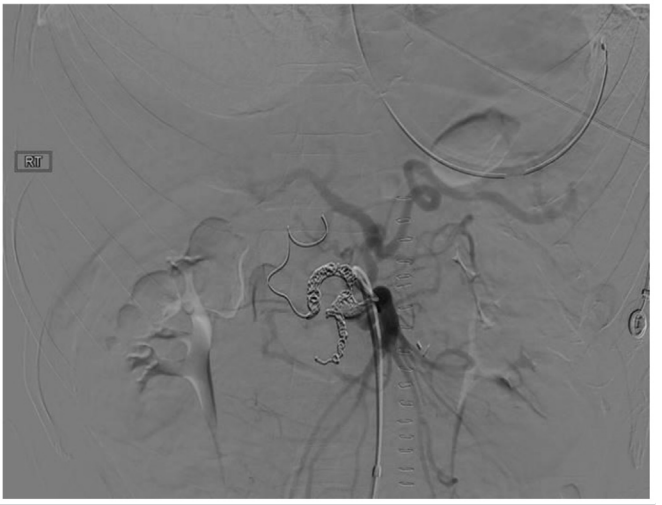

Fig 4. Successful embolization of the inferior pancreaticoduodenal artery aneurysm was performed using 6 Nester embolization coils.

IPDAs carry a high risk of rupture, independent of size, and the mortality rate has been reported as high as 80%.[3] The most common causes of IPDAs are thought to be the result of periarterial inflammation, which may occur as a consequence of pancreatitis.[4] Other causes of IPDAs include trauma, surgery, congenital lesions (eg, MALS), and secondary arteriosclerosis. Celiac axis stenosis or occlusion, which has been reported in 63% of cases of IPDA, may precipitate the development of pancreaticoduodenal artery aneurysm owing to increased retrograde blood flow through the pancreaticoduodenal arcades.[5] The high flow and increased intra-arterial pressures through collateral vessels result in arterial dilation and aneurysm formation. Symptoms of chronic IPDA may include abdominal angina owing to reduced flow through the stenotic lesion.[6] Ruptured IPDAs may present acutely with abdominal pain, hypotension, and gastrointestinal bleeding.

MALS is an uncommon cause of IPDA with celiac axis stenosis and is characterized clinically by weight loss, nausea, vomiting, and postprandial epigastric pain. Anatomic risk factors are hypothesized to predict the development of MALS.[7] The median arcuate ligament is composed of the right and left diaphragmatic crura

and forms the attachment between the diaphragm and vertebrae. It normally passes superior to the origin of the celiac artery at the level of the first lumbar vertebrae, but in one-quarter of individuals, the median arcuate ligament passes anterior to the celiac trunk, which can result in compression of the celiac axis.[8] Diagnosis is reached based on exclusion; however, angiogram or computed tomography imaging can be supportive, which would typically show findings of focal narrowing of the celiac artery origin with poststenotic dilation, indentation of the superior aspect of the celiac artery, and a hook-shaped appearance of the celiac artery.

No clear guidelines currently exist for the treatment of IPDAs. Amid varying viewpoints on treating IPDAs, however, it is evident that early detection and prompt intervention are necessary given the high risk of rupture that exists independent of aneurysm size.[9] Endovascular treatment is preferred owing to the increased mortality rate associated with open surgery. The endovascular approach for ruptured IPDAs involves selective catheterization of the SMA and the branches feeding the aneurysm sac.[10] Targeted embolization of the aneurysm sac can be achieved using platinum or steel microcoils. If access to the aneurysm cannot be obtained owing to challenging anatomy, the proximal and distal portions of the parent artery can be occluded with the use of microcoils.[11] In particular, utmost care must be taken to maintain collateral flow from the SMA, because it provides flow to the stomach, spleen, and liver in the absence of celiac axis patency. In our patient, for example, multiple views were taken with selective angiography to ensure the gastroduodenal artery remained patent before and after embolization (Fig 3). Angioplasty and stenting of the celiac artery have also been considered to improve blood flow to the celiac axis.[12] However, various case series have shown no recurrence of IPDA after initial embolization of the aneurysm sac alone.

It is unclear what the optimal treatment strategy is for patients with ruptured IPDAs secondary to MALS. In these patients, the cause of the celiac stenosis is extraluminal, and median arcuate ligament division may be necessary to relieve compression of the celiac axis if the stenosis is severe.[13] Laparoscopic release of the median arcuate ligament is becoming increasingly accepted in surgical practice for patients with symptoms of MALS; however, it carries the risk of hemorrhage, injury to the abdominal aorta, and the potential for incomplete release.[14] Some authors have also suggested celiac revascularization as the primary treatment for IPDAs in these patients and reported favorable outcomes. Their approach hypothesizes that slow flow in the aneurysm sac after celiac artery revascularization induces thrombosis of the aneurysm, but this point remains controversial. Simultaneous treatment of the IPDA and celiac revascularization is more widely accepted, but it remains unknown whether treatment prevents aneurysm recurrence.[15] Autologous venous bypass between the supraceliac aorta and the common hepatic artery may also be an option in patients with complete obstruction of the celiac artery.[16] In our patient, celiac revascularization was considered but not performed, because median arcuate ligament decompression was not attempted. Future follow-up is planned with this patient, and division of the median arcuate ligament and celiac artery revascularization may be performed if warranted by the recurrence of symptoms.

At the time of the procedure, the exact diagnosis was uncertain. Hemoperitoneum was suspected owing to the presence of blood in the abdomen, prompting the decision to proceed with surgery. Although blood was detected, the source remained unidentified. As a result, an exploratory laparotomy was performed. It was only after further examination that an aneurysm was suspected, leading to the discovery of an IPDA. Initially, there was no clear indication that the bleed was related to the celiac artery, and it was believed that the aneurysm was fully occluded. Consequently, releasing the ligament was not considered possible if the aneurysm was indeed completely occluded.

ANALYSIS AND REVIEW

One of the complexities faced in the treatment of IPDAs is the presence of conditions such as MALS that lead to celiac axis stenosis or occlusion. It cannot be solved by a single approach and requires a multidisciplinary approach. Even though endovascular techniques are now considered the gold standard, they remain somewhat invasive and may not be the best solution as compared with open surgical repair.[17] The success in the management of these patients depends not only on preprocedural planning and meticulous endovascular technique, but also on their early intervention and timely surgical intervention.[18]

In the given situation, the endovascular method involving coil embolization and Onyx liquid embolization irreversibly excluded the IPDA from the circulation. It allowed for the preservation of the collateral pathways for visceral perfusion attainment. The successful completion of the aneurysm was initiated by meticulous angiographic examination. It enabled the detection pathways of the collaterals and the subsequent cannulation of the sac, keeping the gastroduodenal artery undamaged, which was critical for the continued blood flow to the celiac artery system.

The ongoing management of this patient remains a challenge. This is because the etiology of MALS is perseverance, which may lead to either an aneurysm recurrence or ischemic complications owing to persistent stenosis of the celiac artery.[19] Tight clinical and imaging follow-up are crucial to catch disease

relapse or the appearance of additional visceral artery aneurysms or ischemic manifestations.

In situations where MALS originates from the IPDA and celiac artery stenosis, invasive interventions may be imperative to straighten the celiac artery from the compression performed by the median arcuate ligament. Median arcuate ligament release (MALR) in either open or laparoscopic approaches is envisaged to decompress the compression on the celiac axis.[7] It is achieved by disrupting the ligament and improving the antegrade flow, which might decrease the risk of aneurysm formation and ischemic complications in recurrent events. Nonetheless, MALR does also have risks such as hemorrhage, insult to adjacent structures, and incomplete decompression of the compression.

Celiac artery revascularization, either through angioplasty, stenting, or a bypass graft, may be considered in cases of severe or complete occlusion of the celiac axis. MALR is insufficient to restore the flow to an adequate degree. It is in this way that the blood flow to the territory served by the celiac trunk is reestablished, which may decrease the risk of any future complications. The determination of the process for MALR and celiac artery revascularization together with an endovascular aneurysmal embolization requires thorough clinical evaluation and a comprehensive comparison of the potential benefits and risks of therapy. Teamwork between vascular surgeons, interventional radiologists, and other specialists is necessary to design a tailored treatment that addresses the incidental aneurysm through release and the underlying vascular occlusion or compression.

CONCLUSIONS

The endovascular management of the IPDAs, which rupture together with the celiac axis stenosis or occlusion, is the most complicated and challenging process. Although endovascular techniques turn out to be a much less invasive and effective approach to aneurysm exclusion, long-term management may demand additional interventions that address the underlying cause of the condition and help to prevent recurrences. It is key for successful outcomes as well as patient care to apply careful preprocedural planning, apply the endovascular technique with a great deal of accuracy, and rely on a multidisciplinary approach involving various specialists in rare and complicated cases.

DISCLOSURES

None.

REFERENCES

1. Stanley JC, Wakefield TW, Graham LM, Whitehouse WM, Zelenock GB, Martin Lindenauer S. Clinical importance and management of splanchnic artery aneurysms. *J Vasc Surg*. 1986;3:836–840.
2. Kalva SP, Athanasoulis CA, Greenfield AJ, et al. Inferior pancreaticoduodenal artery aneurysms in association with celiac axis stenosis or occlusion. *Eur J Vasc Endovasc Surg*. 2007;33:670–675.
3. Murata S, Tajima H, Fukunaga T, et al. Management of pancreaticoduodenal artery aneurysms: results of superselective transcatheter embolization. *AJR Am J Roentgenol*. 2006;187:W290–W298.
4. Gama Sosa MA, De Gasperi R, Pryor D, et al. Low-level blast exposure induces chronic vascular remodeling, perivascular astrocytic degeneration and vascular-associated neuroinflammation. *Acta Neuropathol Commun*. 2021;9:167.
5. Klonaris C, Psathas E, Katsargyris A, Lioudaki S, Chatziioannou A, Karatzas T. Multiple aneurysms of the inferior pancreaticoduodenal artery: a rare complication of acute pancreatitis. *Case Rep Vasc Med*. 2013;2013:1–5.
6. Balachandran VP, D'Angelica MI. *Intraoperative injury to hepatic arterial structures*. Springer EBooks; 2015:217–228.
7. Horton KM, Talamini MA, Fishman EK. Median arcuate ligament syndrome: evaluation with CT angiography. *Radiographics*. 2005;25:1177–1182.
8. Okobi OE, Afuda BA, Boms M, et al. Median arcuate ligament syndrome: management and literature review. *Cureus*. 2022;14:e2888..
9. Kumari D, Funaki B. Recurrent IPDA aneurysm from celiac occlusion treated by a flow diverting stent. *J Clin Interv Radiol ISVIR*. 2022;07:194–196.
10. Kim EN, Lamb K, Relles D, Moudgill N, DiMuzio PJ, Eisenberg JA. Median arcuate ligament syndrome—review of this rare disease. *JAMA Surg*. 2016;151:471–477.
11. Kok HK, Asadi H, Sheehan M, Given MF, Lee MJ. Systematic review and single-center experience for endovascular management of visceral and renal artery aneurysms. *J Vasc Interv Radiol*. 2016;27:1630–1641.
12. Doita S, Aoki H, Kajioka H, Tanakaya K, Kawamoto K. The combination of preoperative celiac axis stenting and neoadjuvant chemotherapy in an elderly patient with pancreatic cancer: a case report. *Surg Case Rep*. 2024;10:59.
13. Lu XC, Pei JG, Xie GH, Li YY, Han HM. Median arcuate ligament syndrome with retroperitoneal haemorrhage: a case report. *World J Clin Cases*. 2022;10:7509–7516.
14. Goodall R, Langridge B, Onida S, Ellis M, Lane T, Davies AH. Median arcuate ligament syndrome. *J Vasc Surg*. 2020;71:2170–2176.
15. Venturini M, Marra P, Colombo M, et al. Endovascular treatment of visceral artery aneurysms and pseudoaneurysms in 100 patients: covered stenting vs transcatheter embolization. *J Endovasc Ther*. 2017;24:709–717.
16. Foertsch T, Koch A, Singer H, Werner L. Celiac trunk compression syndrome requiring surgery in 3 adolescent patients. *J Pediatr Surg*. 2007;42:709–713.
17. Ogino H, Sato Y, Banno T, Arakawa T, Hara M. Embolization in a patient with ruptured anterior inferior pancreaticoduodenal arterial aneurysm with median arcuate ligament syndrome. *Cardiovasc Intervent Radiol*. 2002;25:318–319.
18. 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;46:799–802.
19. Chen AJ, Yeh S, Dhindsa Y, Lawrence PF, Woo K. Outcomes of median arcuate ligament release: a single institution retrospective review. *Ann Vasc Surg*. 2023;94:296–300.

Submitted Jul 7, 2024; accepted Sep 30, 2024.