Case Reports in Gastroenterology

Case Rep Gastroenterol 2023;17:294–301
DOI: 10.1159/000533617
Received: November 1, 2022
Accepted: August 8, 2023
Published online: October 11, 2023

© 2023 The Author(s).
Published by S. Karger AG, Basel
www.karger.com/crg

OPEN ACCESS

This article is licensed under the Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC) (http://www.karger.com/Services/OpenAccessLicense). Usage and distribution for commercial purposes requires written permission.

Case Report

Ruptured Gastroduodenal Artery Pseudoaneurysms as a Complication of Pancreatitis

Haider Ghazanfar[a] Abhilasha Jyala[a] Sameer Datta Kandhi[a]
Dongmin Shin[a] Kazi Samsuddoha[b] Harish Patel[a]

[a]Department of Gastroenterology, Bronxcare Health System, Bronx, NY, USA; [b]Department of Pathology, Bronxcare Health System, Bronx, NY, USA

Keywords
Pseudoaneurysms · Pancreatitis · Gastroduodenal artery · Coil embolization

Abstract
Visceral artery pseudoaneurysms is a known vascular complication of pancreatitis that can lead to life-threatening hemorrhages with a high mortality rate if left untreated. We present a case of ruptured gastroduodenal artery pseudoaneurysm in a 68-year-old male with acute pancreatitis presenting with fatal gastrointestinal and retroperitoneal bleeding that was successfully managed with endovascular coil embolization of the involved vasculature. Patients with hemorrhagic pancreatitis or those presenting with unexplained retroperitoneal or gastrointestinal bleeding in the setting of pancreatitis with an unexplained drop in hematocrit or sudden expansion of pancreatic fluid collection should be screened in a timely manner for pseudoaneurysm using CT angiogram of the abdomen, which is the gold standard imaging modality to identify pseudoaneurysms. Once pseudoaneurysm is diagnosed, it should be treated immediately. Endovascular treatment options are now favored over surgical options in most cases.

© 2023 The Author(s).
Published by S. Karger AG, Basel

Introduction

Visceral artery pseudoaneurysms are rare with an estimated prevalence of less than 10% [1]. They usually develop following an insult to the arterial vessel wall that is in close proximity to the pancreas. A pseudoaneurysm is different from a true aneurysm in which it does not contain all the components of the arterial wall. Instead, it is consisted of a single layer

Correspondence to:
Sameer Datta Kandhi, sameerdattakandhi@yahoo.com

Case Rep Gastroenterol 2023;17:294–301
DOI: 10.1159/000533617
© 2023 The Author(s). Published by S. Karger AG, Basel
www.karger.com/crg

of fibrous tissue containing leaked blood which may continue to enlarge until it becomes symptomatic and/or rupture. Inflammation from pancreatitis causes autodigestion and erosion of peripancreatic vessels, causing vessel wall weakening and subsequent aneurysmal dilation [2]. Visceral arterial pseudoaneurysms can lead to life-threatening complications such as rupture and bleeding, with increased morbidity and mortality up to 90% in untreated patients and 12.5% even with treatment. Prompt diagnosis and intervention is essential for better outcome. With the rapid growth in the field of interventional radiology, there has been a paradigm shift in managing these vascular lesions with endovascular procedures rather than conventional surgical options [3, 4]. Here, we present a case of an elderly male with multiple comorbidities presenting with altered mental status and hypovolemic shock secondary to unexplained gastrointestinal and retroperitoneal hemorrhage. A focused clinical examination and timely imaging led to the diagnosis of a life-threatening ruptured pancreatic pseudoaneurysm that was successfully treated with endovascular coil embolization, a case that could have otherwise resulted in death.

Case Presentation

A 68-year-old male was brought to the emergency department of our hospital after being found confused on the floor of his apartment by his neighbor. As per the neighbor, patient was last seen in his usual state of health the day before. A review of patient's previous electronic medical records revealed a past medical history of uncontrolled hypertension, human immunodeficiency virus infection on antiretroviral therapy, morbid obesity, chronic obstructive pulmonary disease on two liters of ambulatory home oxygen, and treated hepatitis C infection. He was a former heavy smoker, non-alcoholic along with history of recreational drug (cocaine and heroin) used in the past. He used to work as a dispatcher in the truck business. Family history was significant for hypertension and thyroid abnormalities in the father. There was no documented history of allergies. He was not under any medications used for prophylaxis of AIDS-related opportunistic diseases. His antiretroviral regimen consisted of emtricitabine-tenofovir alafenamide along with dolutegravir-rilpivirine.

While in the emergency room, the patient was noted to be afebrile, with a blood pressure of 131/82 mm Hg, heart rate of 150 beats per minute, saturating 94% on 2 L/min supplemental oxygen. He appeared cachectic with bitemporal wasting on general physical examination. No stigmata of cirrhosis were noted. He was not oriented to time, place, and person. His pupils were dilated bilaterally. No signs of head or neck trauma were noted. Cardiovascular and pulmonary examinations were within normal limits. The abdomen was noted to be markedly distended, soft with no palpable masses, and normal bowel sounds heard on auscultation, with tympanic note on percussion. Patient was eventually intubated for airway protection. Initial electrocardiography from the triage showed sinus tachycardia to 149 beats per min with no abnormal heart rhythm or signs of ischemia. Urine analysis was unremarkable. Urine drug screen was positive for cocaine and methadone. Severe acute respiratory syndrome coronavirus 2 polymerase chain reaction test came back negative. Other significant initial laboratories drawn on admission have been listed in Table 1.

Computed tomographic (CT) imaging of the head was unremarkable. CT chest without contrast showed bilateral ground opacities. CT abdomen and pelvis without intravenous contrast showed evidence of hemorrhagic pancreatitis centered at pancreatic head along with large volume of blood in retroperitoneal space, reactive inflammatory changes of the adjacent duodenum, para-aortic and common iliac lymphadenopathy, and fluid-filled distention of the stomach and esophagus suspicious for gastric outlet obstruction (Fig. 1). An orogastric tube inserted following CT abdomen, revealed coffee ground aspirate. Repeat laboratories showed

Case Rep Gastroenterol 2023;17:294–301
DOI: 10.1159/000533617
© 2023 The Author(s). Published by S. Karger AG, Basel
www.karger.com/crg

Table 1. Laboratory test results of the patient

Laboratory tests	Day 1 of hospitalization	Day 5	At time of discharge	Reference range
WBC count	24.3	14.3 k/μL	7 k/μL	4.8–10.8 k/μL
RBC Count	4.77	2.69 MIL/μL	3.19 MIL/μL	4.50–5.90 MIL/μL
Hemoglobin	15.8	8.5 mg/dL	10.2 mg/dL	12.0–16.0 g/dL
Hematocrit	47.6	25.7%	30.5%	42–51%
Platelet count	171	155 k/μL	215 k/μL	150–400 k/μL
Sodium, serum	132	139 mEq/L	140 mEq/L	135–145 mEq/L
Potassium, serum	3.0	3.2 mEq/L	3.9 mEq/L	3.5–5.0 mEq/L
Blood urea nitrogen, serum	41	17 mg/mL	10 mg/mL	8–26 mg/dL
Creatinine, serum	1.2	0.5 mg/dL	0.6 mg/dL	0.5–1.5 mg/dL
Bilirubin, serum total	0.5	0.7 mg/dL	0.3 mg/dL	0.2–1.1 mg/dL
Serum direct bilirubin	0.3	0.3 mg/dL	<0.2 mg/dL	0.0–0.3 mg/dL
Albumin level, serum	3.6 g/dL	3.2 g/dL	3.4 g/dL	3.2–4.6 g/dL
Alkaline phosphatase	51	48 units/L	73 units/L	56–155 unit/L
Aspartate transaminase	125	21 units/L	18 units/L	9–48 unit/L
Alanine aminotransferase	81	30 units/L	12 units/L	5–40 unit/L
Lactic acid level	8.2	0.8 mmoles/L	1.1 mmoles/L	0.5–1.6 mmoles/L
Prothrombin time	15.2	13.9 s	10.5 s	9.9–13.3 s
International normalized ratio	1.31	1.20	0.92	0.85–1.14
Serum calcium	6.6 mg/dL	7.6 mg/dL	9.2 3 mg/dL	8.5–10.5 mg/dL
Serum lipase	24 U/L			≤61U/L
Hepatitis C viral load, serum	Undetectable			
Absolute CD4 count, serum	137 cells/μL			490–1,740 cells/μL
Serum triglyceride	163 mg/gL			55–150 mg/dL
Serum ethanol	<10			≤10 mg/dL
Urine toxicology	Cocaine and methadone			Negative

a significant drop in hemoglobin level from 15.5 gm/dL to 12.2 gm/dL. Rectal examination showed light brown stool. The patient received two units of packed red blood cell transfusion. His bedside index of severity in acute pancreatitis (BISAP) score was noted be four and was started on nothing per oral diet (NPO) along with intravenous fluid and proton pump inhibitor.

Emergent CT angiography was obtained given the clinical picture which showed an enlarging retroperitoneal and right-sided intraperitoneal hematoma with suspicion for active arterial extravasation or punctate pseudoaneurysms in the pancreatic head (Fig. 2). Celiac angiogram was done under fluoroscopic guidance, which showed gastroduodenal and superior pancreaticoduodenal artery pseudoaneurysms (Fig. 3). Gastroduodenal artery (GDA) embolization was carried out using microcoils and gel foam via the right common femoral

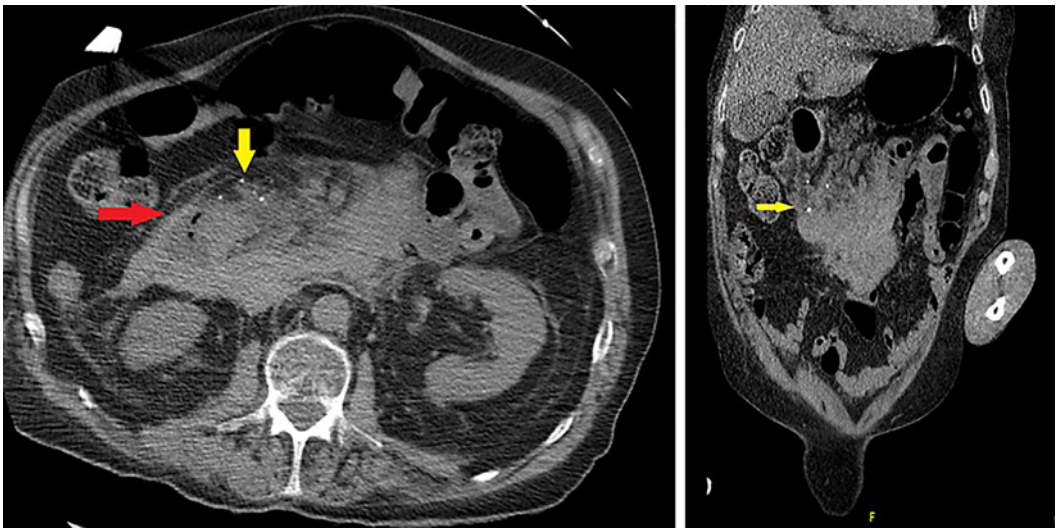

Fig. 1. CT abdomen without intravenous contrast (axial and coronal sections) showing peripancreatic inflammation surrounding the pancreatic body and head along with retroperitoneal bleed (red arrow).

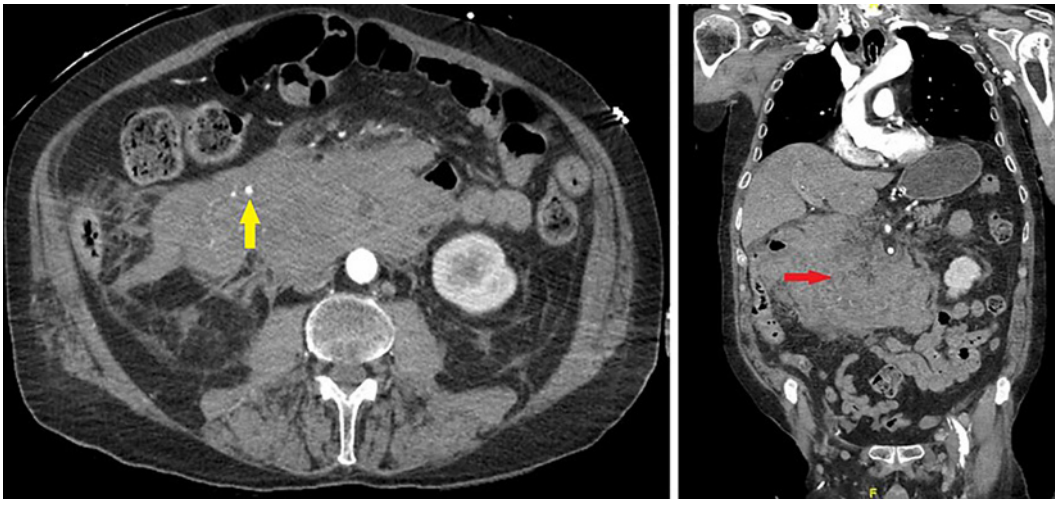

Fig. 2. CT angiography of abdomen (axial and coronal) in arterial phase showing retroperitoneal and right-sided intraperitoneal hemorrhage (red arrow) with active contrast extravasation (yellow arrows) suspicious for arterial bleeding versus punctuate pseudoaneurysm.

artery approach (Fig. 4). Patient was then monitored closely in the medical intensive care unit for rebleeding episodes.

A sonogram of the abdomen was obtained on day 3 of hospitalization, showing liver with normal echotexture and an 8-mm dilated common bile duct along with gallbladder sludge and pericholecystic fluid. Patient was diagnosed with acute hemorrhagic pancreatitis secondary to gallstones. Repeat CT abdomen with intravenous contrast on day 4 of hospitalization showed resolving retroperitoneal hematoma with no signs of active arterial extravasation. Patient's clinical status gradually improved over the course of hospitalization with near normalization of serum lactic acid levels and white cell counts. Hemoglobin levels continued to remain stable

Case Rep Gastroenterol 2023;17:294–301
DOI: 10.1159/000533617
© 2023 The Author(s). Published by S. Karger AG, Basel
www.karger.com/crg

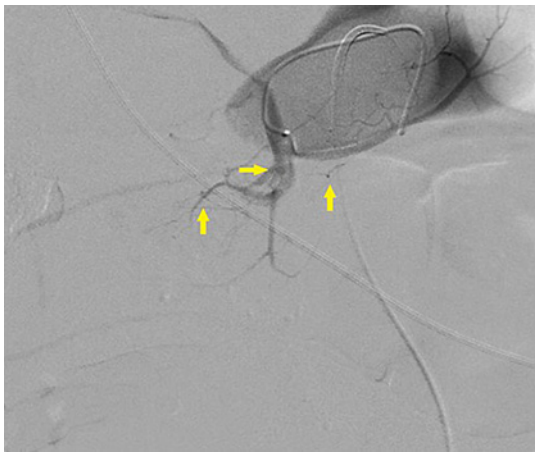

Fig. 3. Celiac artery angiogram (pre-embolization) showing pseudoaneurysm (yellow arrows) involving gastroduodenal and superior pancreaticoduodenal artery.

by day 5 with no further drop and patient was extubated to high flow nasal cannula on day 6 of hospital stay. Thereafter, his clinical course was complicated COPD exacerbation and new onset pneumonia, while in the hospital following which patient clinical status gradually improved and was subsequently discharged to a skilled nursing facility in stable condition on day 32 of hospitalization.

Discussion

Visceral artery pseudoaneurysm is a rare vascular complication of pancreatitis, with an estimated prevalence of less than 10%. If left untreated, it can lead to fatal hemorrhage with a high mortality rate [5, 6]. Unlike a true aneurysm that involves all three layers of the vessel wall, a pseudoaneurysm results from a disrupted vessel wall and consists of leaked blood contained by a single layer of the fibrous capsule. Pseudoaneurysm can result from trauma, inflammation (e.g., pancreatitis), infection, and iatrogenic causes (e.g., vascular interventions, intra-abdominal surgeries). Due to its weak structural support, the risk of rupture is higher for pseudoaneurysm than that of a true aneurysm of similar size [7–9]. Our patient developed visceral arterial pseudoaneurysms secondary to inflammation from his severe acute pancreatitis likely due to gallstones given the sonographic findings and was unlikely related to his alcohol, human immunodeficiency virus, or antiretroviral medication use. Though it is no longer advocated to keep the patient on "nothing per oral diet (NPO)" in acute pancreatitis due to risks of superinfection, our patient was initially placed on NPO given the fact that he was intubated and also had an ongoing active overt bleed as noticed with orogastric aspirates.

In the case of pancreatitis, severe pancreatic inflammation can cause leakage of the exocrine pancreatic enzymes to the peripancreatic space and erode into the nearby vessels that can lead to pseudoaneurysm formation. Hence, vascular complications of acute pancreatitis usually tend to occur in the second phase of evolution, after an initial phase of systematic inflammatory response. Pseudoaneurysm can also arise from erosion of pancreatic pseudocyst into a nearby artery [10, 11].

Splenic artery (30–50%) is the most commonly involved artery due to its proximity to the pancreas, followed by gastroduodenal (30%), pancreaticoduodenal (20%), left gastric, hepatic, superior mesenteric, and small intrapancreatic arteries [5, 11]. Our patient was found to have gastroduodenal and superior pancreaticoduodenal artery pseudoaneurysms.

A GDA pseudoaneurysm most often presents with gastrointestinal or intraperitoneal bleeding due to rupture, abdominal pain, pulsatile abdominal mass, anemia, or even shock but

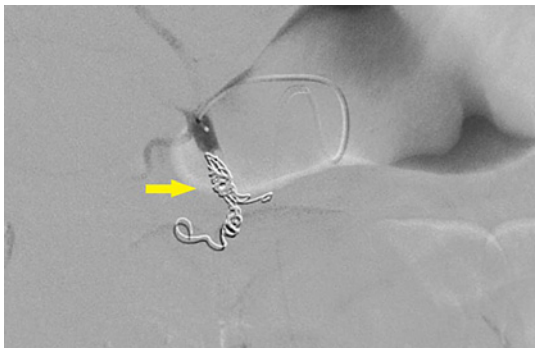

Fig. 4. Celiac artery angiogram post embolization showing microcoils (yellow arrow) in the GDA, resulting in complete stasis of flow to the artery and its branches.

can be asymptomatic in 7.5% of cases [5, 8, 12]. A rare case of hemorrhage from the ampulla of Vater, also known as hemosuccus pancreaticus, can also occur when the pseudoaneurysm communicates with the pancreatic duct. Rare cases of obstructive jaundice caused by extrahepatic biliary compression by the GDA pseudoaneurysm have also been reported [7].

CT of the abdomen and pelvis with contrast has a sensitivity of 67% for diagnosing pseudoaneurysms, but CT angiogram is the gold standard for diagnosis, with a sensitivity of 100% [5]. Abdominal color Doppler ultrasound can help identify a pseudoaneurysm with a sensitivity of 50% [8, 11]. Endoscopy is useful to exclude other sources of gastrointestinal bleedings when the etiology of bleeding from arterial pseudoaneurysm is not very clear. Although visualization of bleeding from the ampulla of Vater is characteristic for hemosuccus pancreaticus, it is often missed during esophagogastroduodenoscopy due to its intermittent nature of bleeding [13, 14]. In our case, we were able to diagnose GDA pseudoaneurysm by CT angiogram.

Due to its high mortality rate with rupture, pseudoaneurysms should be treated immediately once it is identified, regardless of their size or whether it is symptomatic or not [6, 9]. Endovascular treatment with various embolization techniques (e.g., coil, stent, transcatheter thrombin injection) has become the favored approach in stable patients due to its advantage over conventional surgical treatments in terms of lower morbidity and mortality rate, less postoperative pain, decreased hospital length of stay, and early return to daily life activities [7, 8, 11, 12]. Surgery should be reserved for those who are hemodynamically unstable or those who have failed endovascular embolization [8, 11, 12]. However, the prolonged length of stay for our patient was primarily due to development of nosocomial pneumonia and exacerbation of underlying pulmonary conditions of the patient rather than the procedure related recovery time.

Despite the high success rate of embolization, rebleeding can occur in 20–40% of patients [5, 14], and observation with close follow-up after embolization is necessary [12]. The overall mortality rate remains high at 7.4–16% even after endovascular therapy and 20–30% after surgery [5, 11]. After embolization, our patient did not experience rebleeding and was discharged to a skilled nursing facility in a clinically stable condition.

Conclusion

Physicians should be aware of visceral arterial pseudoaneurysms as rare vascular complication associated with pancreatitis that may lead to fatal gastrointestinal or retroperitoneal bleeding with a high mortality rate. Patients with hemorrhagic pancreatitis or those presenting with unexplained retroperitoneal or gastrointestinal bleeding in the setting of pancreatitis with an unexplained drop in hematocrit or sudden expansion of pancreatic

fluid collection should be screened in a timely manner for arterial pseudoaneurysm using CT angiogram of the abdomen, which is the gold standard imaging modality to identify pseudoaneurysms. Once pseudoaneurysm is diagnosed, it should be treated immediately. Endovascular treatment options are now favored over surgical options in most cases. The CARE Checklist has been completed by the authors for this case report and attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000533617).

Statement of Ethics

The patient has given written informed consent to publish this case including publications of images. Research complies with the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. This study protocol was reviewed, and the need for approval was waived by the BronxCare Health System Ethics Committee.

Conflict of Interest Statement

The author(s) of this manuscript do(es) not have any conflict of interest to declare.

Funding Sources

There was no external source of funding to support this case report.

Author Contributions

The first author (Haider Ghazanfar) and the coauthors (Abhilasha Jyala, Sameer Datta Kandhi, Dongmin Shin, Kazi Samsuddoha, Harish Patel) were all involved in summarizing this case report, writing the manuscript, and proofreading the final version of the manuscript).

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

References

1 Carr JA, Cho JS, Shepard AD, Nypaver TJ, Reddy DJ. Visceral pseudoaneurysms due to pancreatic pseudocysts: rare but lethal complications of pancreatitis. J Vasc Surg. 2000 Oct;32(4):722–30.
2 Hoilat GJ, Mathew G, Ahmad H. Pancreatic pseudoaneurysm. Treasure Island, FL: StatPearls; 2021.
3 Sagar S, Soundarajan R, Gupta P, Praveen Kumar M, Samanta J, Sharma V, et al. Efficacy of endovascular embolization of arterial pseudoaneurysms in pancreatitis: a systematic review and meta-analysis. Pancreatology. 2021 Jan;21(1):46–58.
4 Shrikhande GV, Khan SZ, Gallagher K, Morrissey NJ. Endovascular management of superior mesenteric artery pseudoaneurysm. J Vasc Surg. 2011 Jan;53(1):209–11.

Case Rep Gastroenterol 2023;17:294–301
DOI: 10.1159/000533617
© 2023 The Author(s). Published by S. Karger AG, Basel
www.karger.com/crg

5 Gurala D, Polavarapu AD, Idiculla PS, Daoud M, Gumaste V. Pancreatic pseudoaneurysm from a gastroduodenal artery. Case Rep Gastroenterol. 2019 Oct 30;13(3):450–5.
6 White AF, Baum S, Buranasiri S. Aneurysms secondary to pancreatitis. Am J Roentgenol. 1976;127(3):393–6.
7 Chapman BM, Bolton JS, Gioe SM, Conway WC. Gastroduodenal artery pseudoaneurysm causing obstructive jaundice. Ochsner J. 2021;21(1):104–7.
8 Awada Z, Al Moussawi H, Alsheikh M. Gastroduodenal artery pseudoaneurysm rupture post-billroth II surgery: case report. Cureus. 2019;11(1):e3833. Published 2019 Jan 7.
9 Kasirajan K, Greenberg RK, Clair D, Ouriel K. Endovascular management of visceral artery aneurysm. J Endovasc Ther. 2001 Apr;8(2):150–5.
10 Lakin R, Kashyap V. Splanchnic artery aneurysms. In: Cronenwett J, Johnston KW, editors. Rutherford's vascular surgery. 8th ed. Philadelphia, PA: Saunders; 2014. p. 2220–35.
11 Mallick IH, Winslet MC. Vascular complications of pancreatitis. JOP. 2004 Sep 10;5(5):328–37.
12 Saqib NU, Ray HM, Al Rstum Z, DuBose JJ, Azizzadeh A, Safi HJ. Coil embolization of a ruptured gastroduodenal artery pseudoaneurysm presenting with hemosuccus pancreaticus. J Vasc Surg Cases Innov Tech. 2020;6(1):67–70. Published 2020 Feb 12.
13 Obara H, Matsubara K, Inoue M, Nakatsuka S, Kuribayashi S, Kitagawa Y. Successful endovascular treatment of hemo- succus pancreaticus due to splenic artery aneurysm associated with segmental arterial mediolysis. J Vasc Surg. 2011;54(5):1488–91.
14 Pang TC, Maher R, Gananadha S, Hugh TJ, Samra JS. Peripancreatic pseudoaneurysms: a management-based classification system. Surg Endosc. 2014 Jul;28(7):2027–38.