

Significance of radiology in the diagnosis and management of ruptured left gastric artery aneurysm associated with acute pancreatitis

Case report

Bimbadhar Valluru, MD^a, Bin Yang, MS^b, Kalyan Sharma, MD^b, Ahmed Abdullahi Adam, MD^b, Du Wei, PhD, MD^{a,*}, Zhou Zhou, MD^{b,*}, Mahamed Osman Ali, MD^b

Abstract

Rationale: Left gastric artery aneurysms are very rare which progresses into hemorrhagic shock and diagnosis is very challenging particularly in patients with acute pancreatitis and cholecystitis whose vitals become unstable suddenly.

Patient concerns: A 72-year-old female has presented with severe progressing abdominal pain was treated with total parenteral nutrition for acute pancreatitis based on preliminary work up, but suddenly became unstable with dropping vitals over the ensuing 48 hours. Physical examination has a positive Murphy sign and appeared lethargic. She has no past history of any chronic systemic illness or malignancy.

Diagnoses: Ruptured left gastric artery aneurysm and left hepatic artery aneurysm with intraperitoneal hemorrhage associated with acute pancreatitis and cholecystitis.

Interventions: Emergency interventional surgery was performed to embolize both the aneurysms and the giant aneurysmal sac of the left gastric artery was secured with a micrometallic occluding coil which eventually controlled the active hemorrhage.

Outcomes: The patient became stable and was discharged after 15 days without any recurrence or complications during the 6-month follow-up.

Lessons This case is a peculiar example of a missed diagnosis of left gastric artery aneurysm associated with acute pancreatitis and cholecystitis with deteriorating clinical condition. Exhaustive radiological investigations are necessary for early diagnosis correlating with presenting clinical situations. Radiologists should be familiar with the challenges in diagnosis and management.

Abbreviations: APACHE = Acute Physiology and Chronic Health Evaluation, BISAP = bedside index of severity in acute pancreatitis, CT = computed tomography, DSA = digital subtraction angiography.

Keywords: acute pancreatitis, CT angiography, DSA, left gastric aneurysm, pseudoaneurysm, Seldinger technique, TAE

Editor: N/A.

BV and BY contributed equally to this work.

First author—BV

Co-first author—BY

Second author—KS, AA

Third author—MOA

This work was supported by the Medical Imaging Department, The First Affiliated Hospital of Dali University, key subjects for medical imaging.

The authors have no conflicts of interest to disclose.

^a Department of Radiology, The First Affiliated Hospital of Dali University, Dali University Institute of Materia Medica. ^b Department of Radiology, The First Affiliated Hospital of Dali University, Dali, Yunnan, People's Republic of China.

* Correspondence: Du Wei, and Zhou Zhou, Department of Radiology, The First Affiliated Hospital of Dali University, Dali 671000, Yunnan, China (e-mails: duweifsk@qq.com [WD], zhouzhoufsk@qq.com [ZZ]).

Copyright © 2019 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Medicine (2019) 98:10(e14824)

Received: 27 October 2018 / Received in final form: 25 January 2019 /

Accepted: 5 February 2019

<http://dx.doi.org/10.1097/MD.00000000000014824>

1. Introduction

Gastric artery aneurysms (GGAs) are very rare, typically asymptomatic presenting as an acute condition. The incidence of all visceral—GGAs is as follows: splenic artery is the one that is mostly involved (60%) which have unusually the high incidence of rupture when associated with physiological medial changes during pregnancy,^[1] followed by hepatic arteries (40%), superior mesenteric artery (5.5%), and aneurysms of gastric and gastroepiploic arteries (4%),^[2] while the right GGAs are extremely rare.^[3] Though there were several case reports of hemoperitoneum from rupture of gastroepiploic arteries, very few cases of hemorrhagic shock due to the rupture of left gastric artery aneurysm have been published in English medical works (recent being Nishimura et al and Nissim et al).^[4,5] The patients can insidiously become unstable due to spontaneous rupture resulting in intraperitoneal hemorrhage, so they are frequently occult and often fatal. Because of the high mortality rate, early diagnosis and management are very important by prompt radiological examinations while correlating with laboratory investigations.

We present a case of a ruptured left gastric artery aneurysm (LGAA) and hemorrhagic shock that is associated with acute pancreatitis, cholecystitis who was successfully treated with trans-arterial embolization using the Seldinger technique. In this case report, we also summarized the etiology, clinical presenta-

tion, challenges, diagnostic protocols, and the interventional surgical management we followed for a ruptured LGAA. We anticipate that our case report could provide more insight to clinicians and radiologists in following a systemic approach to early diagnosis and management of GGAs.

2. Materials and methods

2.1. Patient information

A 74-year-old woman initially presented to a general physician at a local hospital with severe pain in the left upper quadrant of the abdomen for a week that was associated with intermittent nausea, bilious vomiting, and general fatigue. She was admitted into our hospital for further evaluation and management as the pain was sharp and progressing, unrelieved by empirical prescription. On admission, she was alert though appeared to be in distress. Her past medical history denies hypertension and diabetes, nonalcoholic, any long-standing systemic illness or recent abdominal trauma/surgeries.

On the initial day, her vital signs were slightly unstable, her blood pressure was 151/83 mm Hg, heart rate was 94 bpm, the respiratory rate was 19 breaths per minute with oxygen saturation over 94% and nonpyretic. On physical examination, she revealed a positive Murphy sign and no palpable abdominal mass was found.

A provisional diagnosis of acute pancreatitis was made based on a preliminary ultrasound, so further was recommended for complete blood work. The patient was kept fasting; was suggested TPN to observe if clinical or biochemical pancreatitis can be attained. Based on the complete blood work-up, BISAP score-4 and APACHE-II score- 18; it was concluded as Acute Pancreatitis/Cholecystitis/ Electrolyte imbalance as Hypokalemia/ Hyponatremia/ Hypophosphatemia with an Anion gap of 1 mmol/L. She also tested positive for Occult Blood Test and showed mild uremia. She was initiated with antibiotics and corrected accordingly with the intravenous fluids. During the ensuing 48 hours, her blood pressure dropped to 77/55 mm Hg, elevated hs C-reactive protein (CRP)>10:42 mg/L, D-Dimer levels >5000 ng/mL which is suggestive of active hemorrhagic hypovolemic shock. Hemoglobin levels and hematocrit levels dropped to 6.3 g/dL and 19.3% (severely anemic) respectively. A hypovolemic shock protocol was initiated to correct anemia and hypovolemia with 1500 cc of whole blood transfusion, intravenous fluids respectively, and stabilized accordingly until HGB-9.5 g/dL and HCT-34.8% was attained.

2.2. Imaging examination

All the material and methods were out in accordance with the guidelines and regulations set by the Department of Radiology and Medical Imaging of the First Affiliated Hospital of Dali University. All the protocols were approved by the Organization for Clinical Drug trials/scientific research division of Dali University-Ethical Approval number-FSK20181024. Informed written consent was obtained from the patient for publication of this case report and accompanying images. After stabilization, a complete radiological assessment was performed with an Emergency Bedside Ultrasound (USG), Plain and contrast-enhanced dynamic computed tomography (CT) of the abdomen followed by visceral angiography.

2.3. Diagnostic assessment

Emergency USG revealed signs of pancreatitis along with a well-defined hypoechoic lesion near to the lesser curvature of the stomach suggesting focal dilatation of an artery—a suspected

aneurysm. Progressive ascites and bilateral pleural effusions were also present.

Plain CT demonstrated a well-defined focal hypodense lesion measuring 2.54 cm×1.74 cm along the lesser curvature of the stomach. There was also hyper-attenuated irregular hematoma collection surrounding the lesion extending into the gastric pyloric antrum, intraperitoneum to retroperitoneum and in the surrounding area of the spleen (Fig. 1A). Plain CT abdomen also revealed acalculous cholecystitis and acute pancreatitis, the former is evident by gallbladder wall thickening, pericholic fat stranding, and pericholic fluid collections; latter is evident by pancreatic enlargement, peripancreatic fat stranding, and fluid collections (Fig. 1B); stratification of pancreatitis severity is also assessed by Balthazar score which is consistent with Level-D and 0 for pancreatic necrosis and modified CT severity index (CTSI) has a significant score of 6 out of 10 suggesting moderate acute pancreatitis.

Triphasic contrast study was done which demonstrated a well-defined round hyperdense lesion attenuating homogeneously with the aorta and eccentric contrast-filling of the aneurysmal sac originating from the celiac axis. There is no prompt extravasation of contrast material due to chronic thrombus more peripherally, suggesting a ruptured left gastric artery pseudoaneurysm (Fig. 2).

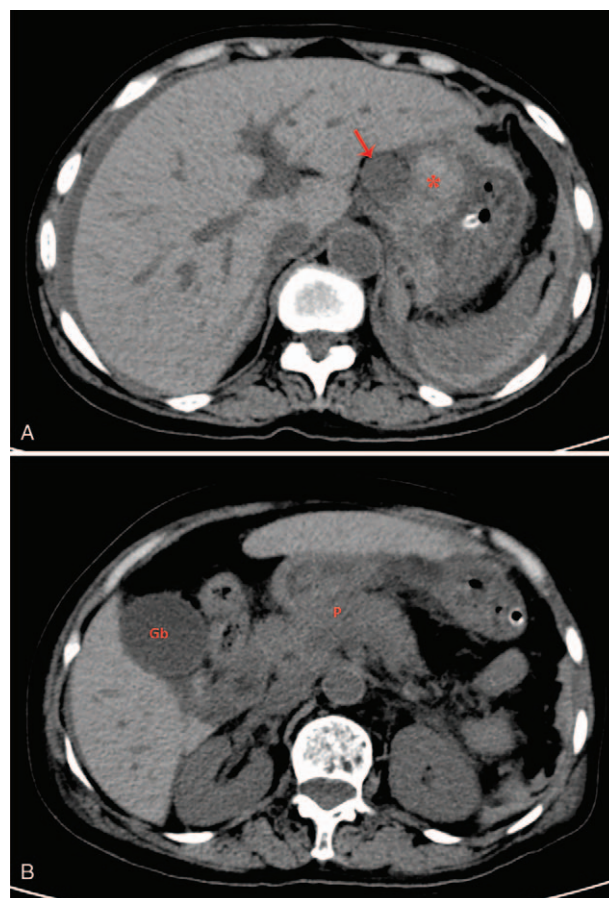


Figure 1. A, Plain CT abdomen showing hypodense mass lesion around the coeliac axis measuring 2.54 cm × 1.74 cm (arrow) near to the lesser curvature of the stomach with mild hyperdense shadow near to the primary lesion (asterisk). There is evidence of intraperitoneal collection as well. B, Plain CT abdomen showing acute pancreatitis (P) and acalculous cholecystitis (Gb). CT=computed tomography.

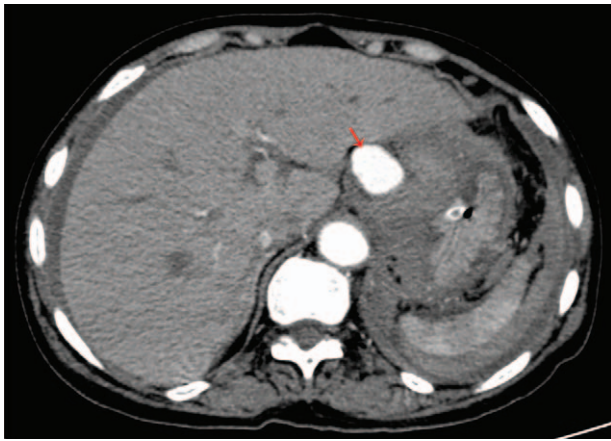


Figure 2. Contrast-enhanced CT arterial phase (axial view) demonstrating pseudoaneurysm (arrow) of the left gastric artery. CT = computed tomography.

3. Results

3.1. CT angiography

The CT angiography also revealed the lumen filled with contrast and mural thrombus enhancement along the lesser gastric curvature and arterial 3D reconstruction showed the site clearly suggesting an aneurysm in the LGAA (Fig. 3A–C).

3.2. Interventional surgery

Complete celiac axis was visualized under fluoroscopic guidance by inserting 5F introducer-seldinger wire through the right femoral artery. Digital subtraction angiography (DSA) revealed a tortuous expansion of a giant pseudoaneurysm in the left gastric artery and a small aneurysm in the left hepatic artery (Fig. 4). The left gastric and left hepatic arteries proximal to aneurysmal sites were embolized using 710 to 1000 μm , 1000 to 1400 μm gelatin sponge microspheres respectively. The micro metallic occluding coils 2 \times 5 mm (Tornado embolization micro coil by Cook

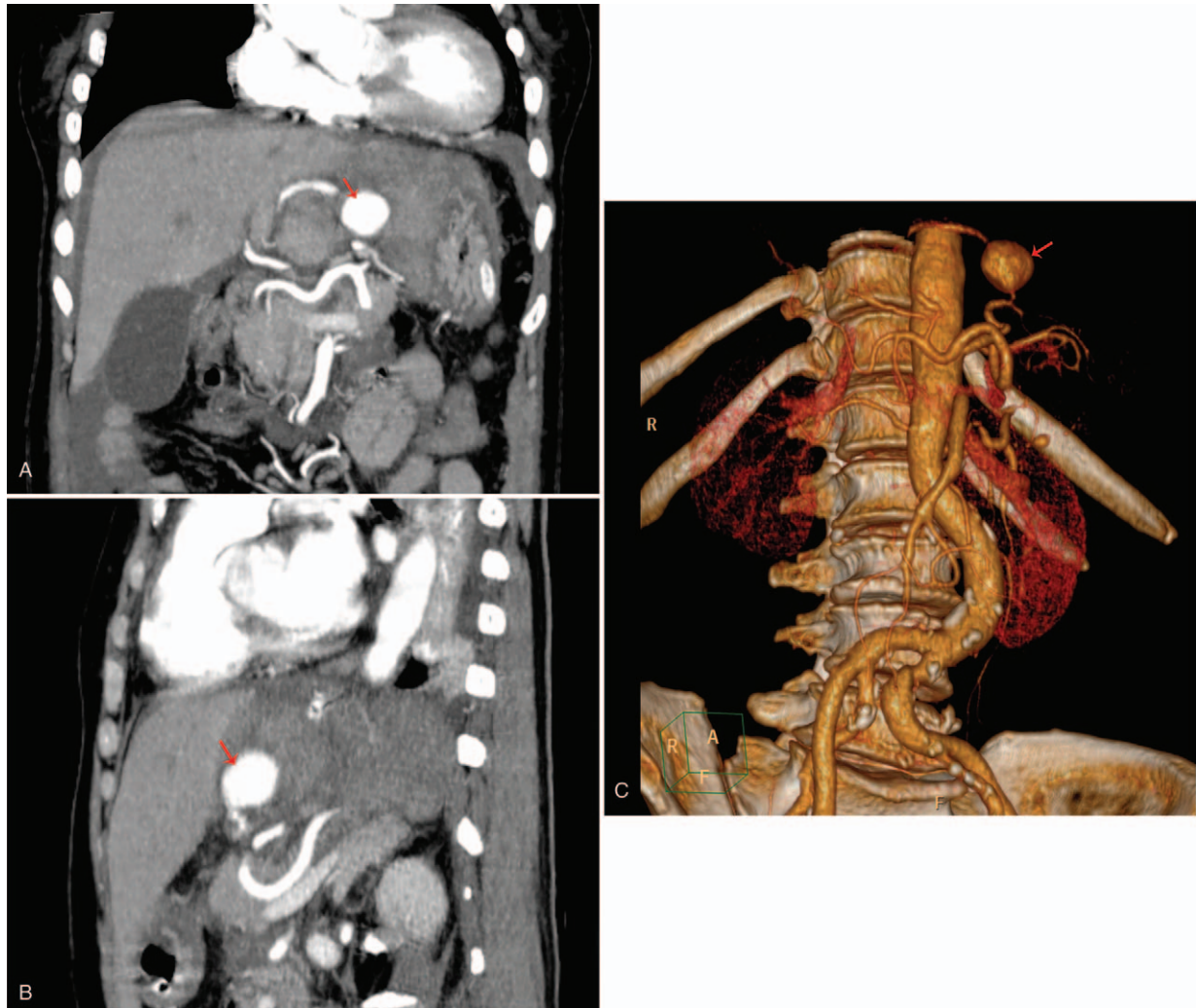


Figure 3. A, Coronal CT angiogram of the abdomen showing eccentric contrast filling of aneurysmal sac (arrow) of the left gastric artery. B, Sagittal CT angiogram showing aneurysmal sac (arrow) with no extravasation of contrast material. C, Three-dimensional volume rendering of the abdominal aorta in a patient with left gastric artery aneurysm (arrow).

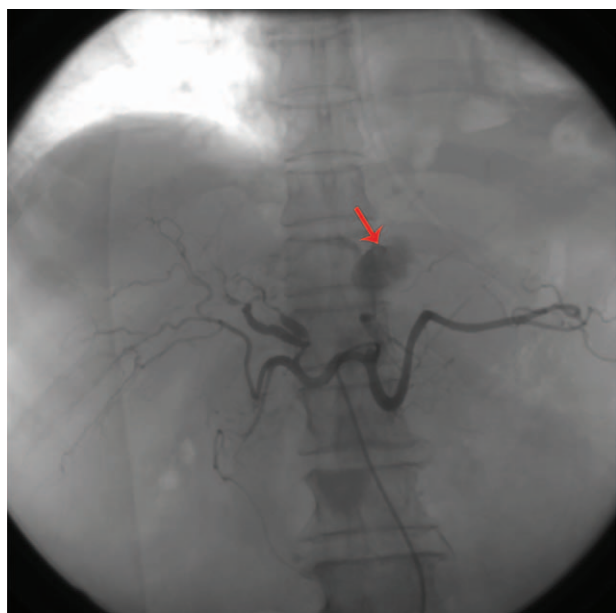


Figure 4. Fluoroscopic image in a patient showing left gastric artery aneurysm.

Medical, Bloomington, Indiana) 4 and 5 mm are advanced to the aneurysmal sacs and released, springing into position until the blood flow from both the arteries was completely interrupted. The operation was smooth, vital signs and hemoglobin remained stable; the patient was discharged home after 15 days when there is no further episode of bleeding or complaint about any special abdominal discomfort.

4. Discussion

4.1. Etiology and pathophysiology

Aneurysms of gastric arteries are rare, typically asymptomatic; especially left GGAs are of greater clinical importance because of the risk of intragastric or intraperitoneal spontaneous and massive hemorrhage, patients become unstable suddenly. LGAAs typically present as an acute condition with progressive long-standing unexplained upper quadrant abdominal pain and are incidentally found during evaluation of pre-existing clinical conditions until spontaneous rupture occurs. About 19 case reports of left or right GGAs with different clinical histories were published in the last 35 years in PubMed (<https://www.ncbi.nlm.nih.gov/pubmed>) contributing to just a handful of cases that are reported worldwide.^[4] Predisposing factors like old age, hypertension, and marked arteriosclerosis/atherosclerosis were commonly associated with pre-existing conditions in aneurysms involving left and right gastric arteries. In most of the cases reviewed, such an association was made only during an exploratory laparotomy; in more than half of the cases, the diagnosis could not be made until autopsy.^[6]

There are reported cases of GGAs (left>right) with different etiologies such as bacterial and viral infections like Tuberculosis (Rasmussen aneurysm), mycotic embolization in the subacute bacterial endocarditis, Syphilis etc., Copper deficiency (lysol oxidase enzyme),^[7] or cystic degenerative disorders like Behcet disease, polyarteritis nodosa, systemic lupus erythematosus^[3] and segmental artery mediolysis,^[8] trauma per abdomen,^[5] abdominal surgeries, prior congenital presence (cirroid) etc. Case

studies presented by Baker LePain et al differentiate segmental artery mediolysis from the above-mentioned conditions suggesting that inflammatory response, anatomical location, vascular integrity, size could possibly be the other major factors interacting to form these aneurysms.^[9]

But, in patients with no specific vasculopathies, infections or any other risk factors, the incidence, male-female predilection, age distribution, natural history/risk factors, determinant etiology, pathophysiological mechanisms in forming left or right GGAs still remain inconclusive and should further be investigated.

Being rare and unusual, the pathogenesis of aneurysm formation in left gastric artery associated with pancreatitis is poorly understood. Especially, subacute, unmanaged pancreatitis often present with mucosal ulcerations, intraluminal hemorrhages, peritonitis etc., where elastase plays a part in the development of pancreatitis by enhancing superoxide production of neutrophils by binding on high and low-affinity sites.^[10] Then the pancreatic enzymes (pancreatin) like amylase, lipase, and protease liberated in pancreatitis lead to focal activation of periarterial inflammatory processes in the corresponding blood vessels; this causes partial digestion of the arterial wall and results in focal dilatation forming a true aneurysm or autodigestion of arterial walls (lysis of tunica intima and media) incorporated into walls of a pseudocyst and ruptures into it, resulting in focal dilation of the vessel forming a pseudoaneurysm.^[11] Given its proximity to the pancreas, when there is the 3-fold rise of pancreatic enzymes in the setting of pancreatic inflammation or injury, rupture of left gastric artery pseudoaneurysm should be suspected clinically when vitals become unstable rapidly, expanding hematoma in the lesser sac or vice versa.^[12,13]

Severe cholecystitis or cholangitis, on the other hand, indicated by raised markers of inflammation (neutrophils, CRP), bilirubin, and aminotransferases have an association with vasculitis and could lead to peritonitis that could boost the periarterial inflammatory process which may stem an aneurysm.^[14]

Our case has no history of trauma, hypertension, or atherosclerosis and any other risk factors that can be associated; radiological signs are consistent with acute acalculous cholecystitis, acute pancreatitis, and intraperitoneal hemorrhage. So, in this scenario, we assume that an intercomplex mechanism of hepato-biliary and pancreatic origins have contributed to stem an aneurysm and could have complicated the condition by rupturing the artery leading to hemorrhagic shock.

4.2. Challenges in the diagnosis of LGAA

In patients with unexplained abdominal pain and unstable vitals, diagnostic physicians often fail to investigate differentials further because of very low incidence rates, poorly understood etiology and unusual location gives no suspicion of LGAA; an unaccounted aneurysm formation leads to failure of recognition; due to progressive expansion, an aneurysm may rupture into gastrointestinal tract or peritoneal cavity causing frequent unexpected massive spontaneous intraperitoneal hemorrhage that often leads to failure of localization of active bleeding site, the patients rapidly deteriorate toward shock insidiously. This could happen when there are delays in forming a diagnosis or missed diagnosis that often results in the death of the patients despite resuscitation. Though gastric artery aneurysm remains an important clinical condition with high lethal potential,^[15] they are never included in the differential diagnosis which also happened in our case. It was concluded as acute subclinical pancreatitis as we did not overcome the challenges based on

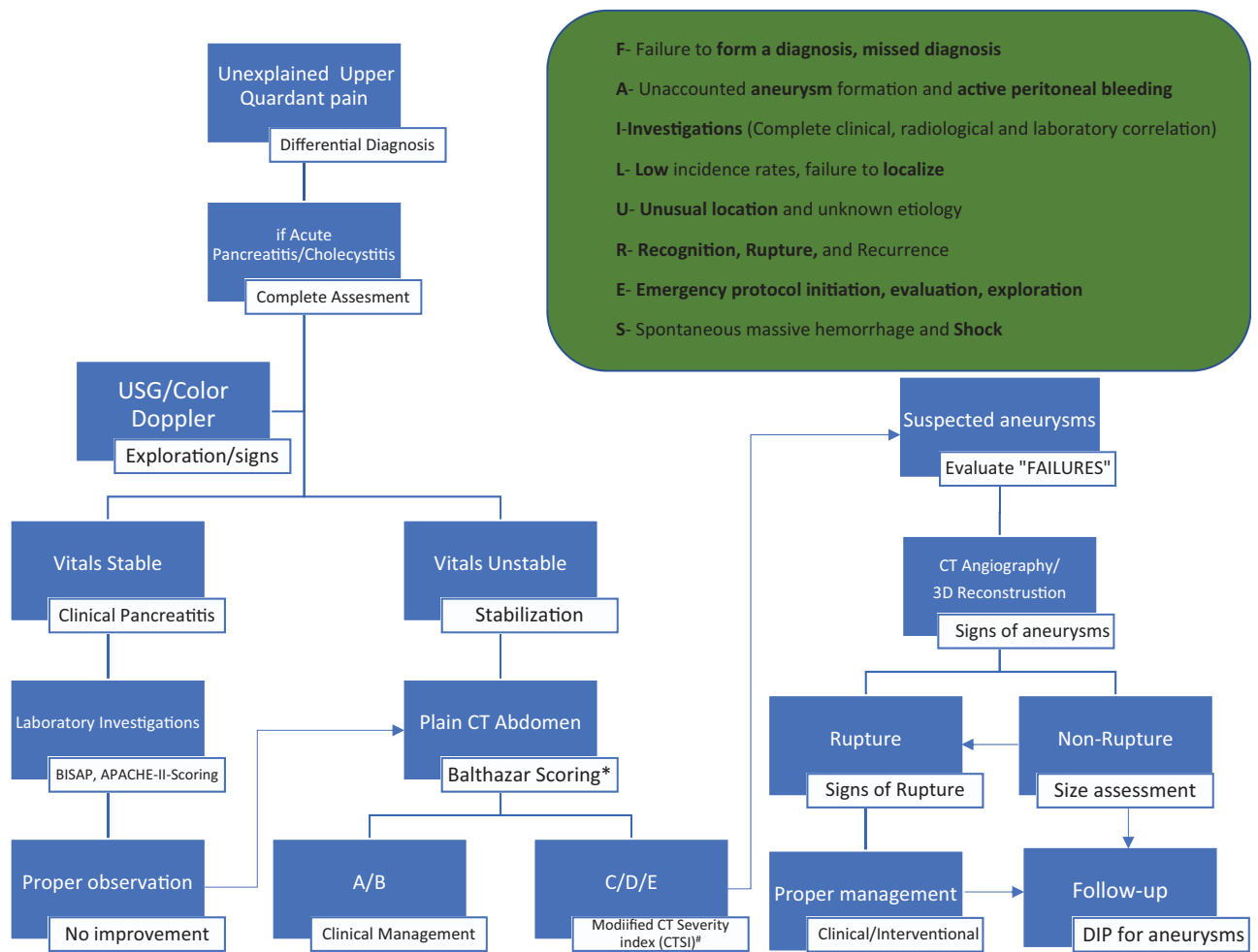


Figure 5. Flowchart and challenges followed in the diagnosis and management of LGGAs. APACHE=Acute Physiology and Chronic Health Evaluation, BISAP= bedside index of severity in acute pancreatitis, DIP=diagnostic investigatory protocol. *Balthazar Scoring for grading of pancreatitis: A: normal pancreas: 0, B: enlargement of pancreas: 1, C: inflammatory changes in pancreas and peripancreatic fat: 2, D: ill-defined single peripancreatic fluid collection: 3, E: 2 or more poorly defined peripancreatic fluid collections: 4 and pancreatic necrosis: 0–6. #Modified CTSI for pancreatic inflammation: 0–4, pancreatic necrosis: 0–4 and extra pancreatic complications: 2—maximum score: 10.

initial evaluation and emergency radiological investigations were started only when the vitals became unstable suddenly.

4.3. Role of radiology

Diagnosis of LGAA is challenging and should be included in the differential diagnosis in patients presenting as a typical situation like ours, that is, acute pancreatitis, cholecystitis, rapidly deteriorating vitals. Abrupt emergence of hemorrhagic/septic shock or severe intragastric or intraperitoneal hemorrhage often prompts for a complete exploratory protocol, and initiation of a complete diagnostic investigation protocol (DIP) through advanced radiological interventions correlated with biochemical investigations could help to have a systematic approach to prevent all such consequences. We quote these factors as “FAILURES” mnemonic, mentioned in the flowchart (Fig. 5).

4.4. Radiological presentation of LGAA

4.4.1. Ultrasound presentation. Ultrasound is of greater value as a bed-side assessment tool for making a provisional diagnosis of cholecystitis and pancreatitis for it is simple, safe, and

inexpensive. It has reported a sensitivity of 95% and specificity close to 100% making it usually the preferred choice for monitoring small aneurysms as the aneurysms never decrease in size.^[16] LGGAs typically present as a hypoechoic lesion around the lesser curvature of the stomach. On Doppler study, “To and fro” or “Ying and Yang” sign can be seen with the bidirectional flow due to the swirling of blood at the pseudoaneurysm neck and within.^[17] As there were reported cases associated with cholecystitis, erosion into the biliary tree causing hemobilia, ruling out other complications like adenomyomatosis, cholangitis, or Rokitansky-Aschoff sinuses is necessary.^[14]

4.4.2. CT and contrast CT—recognize, localize, and evaluate rupture.

In our case, plain CT abdomen revealed significant acalculous cholecystitis and acute pancreatitis and a round soft tissue structure about 2.54 cm × 1.74 cm in size attenuating with ill-defined heterogeneous slight to iso-dense shadow is seen near to the lesser curvature of the stomach. A soft tissue attenuating lesion along the lesser curvature is unusual and should lead to suspicion when there is no significant history of peptic ulcer disease or malignancies. Moreover, stratification of pancreatitis

by Balthazar scoring^[18]/CTSI^[19] can not only help us to evaluate the clinical management but should also allow us to estimate unanticipated outcomes and to recommend further investigations accordingly, if necessary, when vitals become unstable.^[20]

Contrast CT with nonionic iodinated contrast agents has not only a great value in ruling out most malignancies, but also to narrow down other abnormalities in the lesser omentum.^[21] During tri phasic studies, analysis of attenuation changes and expansion of contrast material in the surrounding viscera helps us to recognize, localize the site of active bleeding. Intraparenchymal pseudoaneurysms have a similar appearance to active hemorrhage on initial scanning but do not decrease in size on delayed phases and follow the blood pool on all phases. Enhancement of a lesion homogeneous to aortic attenuation should allow us to recognize it as the vascular origin (pseudo > true). Nonvascular mass lesions may show central-peripheral filling like enhancement with Hounsfield units lower than aorta and do not extravasate. Contrary to this, active hemorrhage appears as a high density (> 80–95 Hounsfield units)^[22] due to extravasation, that is, blushing out of IV contrast media that increases in size on delayed imaging. So, an acute ruptured gastric artery aneurysm is more likely to show extravasation of contrast material into the peritoneum which should suggest intraperitoneal hemorrhage while chronic cases show a mural thrombus peripherally.

4.4.3. CT angiography (CTA) and 3D reconstruction—exploration and presurgical individualization. CTA also has a great diagnostic value in recognizing, localization, enhanced exploration by visualizing complete celiac axis and aids to rule out lesions like vascular neoplasms, etc. Three-dimensional (3D) vascular reconstruction rendered from multiplanar CTA-dynamic imaging provides a real-time presurgical assessment of individualized anatomy by visualizing complete vascular structures in the celiac axis. The left gastric artery is the first and smallest of the coeliac axis that arises from the abdominal aorta. At the cardia, it divides into small branches that supply the anterior and posterior gastric surfaces. One of these branches turns sharply downward to follow the lesser curvature of the pylorus, which usually anastomoses with the terminal branches of the right gastric artery. Although other visceral aneurysms are metachronous, GGAs, on the other hand, tend to be solitary and very rare.^[1] Aneurysms of the gastric arteries can arise in the portion of the artery lying within the gastric wall (intramural or intragastric) or outside of it (extramural or extragastric); if ruptured mortality is high from these lesions. However, the incidence of gastroduodenal bleeding with pseudoaneurysm is low (approximately 2%), most commonly, bleeding is into the bowel followed by massive hemorrhage into the peritoneal cavity, pancreatic duct, or biliary tree.^[11]

When there is no significant history, depending on these basic presentations, ruling out all other potential conditions and given the possibility of LGAA, we believe that preassessment with such interventions is necessary for both stable and unstable patients with inflammatory conditions.

Recently, due to advances in diagnostic imaging modalities, there is an increase in the number of reported cases of GGAs. USG, plain CT/contrast CT for initial evaluation; DSA, CTA/super selective visceral angiography remain “Gold Standard” for detection and evaluation/recognition and anatomical localization of GGAs as it provides the ability for early diagnosis without exploratory laparotomy. DIP has given us a chance to narrow diagnosis in a short time and take timely action to save the patient efficiently rather than making a diagnosis at the time of autopsy.

4.4.4. Interventional surgery—management. Ultraemergency exploratory laparotomy has been replaced and can effectively be treated by interventional surgery procedures such as super-selective embolization with microcoils for true aneurysms or polyvinyl alcohol/gelatin sponge, trans gastric CT, or endoscopic ultrasound guided thrombin injected directly into the pseudoaneurysm.^[11] When such facilities are not available, surgical repair is a better option. Whenever an aneurysm of the gastric artery is discovered it should be embolized or excised.

5. Conclusion

We experienced a case of ruptured left gastric artery aneurysm with spontaneous hemorrhagic shock associated with acute pancreatitis. Although rare, left GGAs are yet the leading cause of death due to spontaneous rupture causing intra-abdominal and retroperitoneal hemorrhage. They often are asymptomatic and solitary. Diagnostic clinicians should be aware of its etiology, diagnosis, and management. Due to the unusual location of an aneurysm in the visceral arteries, the emergency personnel should be aware of its possibility, radiological presentation, and laboratory investigations as complications like rupture, spontaneous hemorrhage, and recurrence are more likely. Rapid hemostatic stabilization (shock protocol) and resuscitation are very important, due to high mortality even during interventional surgery. Radiologists should be familiar with manifestations, challenges in diagnosis and presentation, and recommend DIPs, especially if they encounter an asymptomatic patient with unexplained severe epigastric pain that rapidly becomes unstable.

Acknowledgments

The authors thank all the personnel from the Department of Radiology for their collective efforts on interventions, image postprocessing, clinical management, and follow-up.

Author contributions

Conceptualization: Bimbardhar Valluru, Kalyan Sharma.

Data curation: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Du Wei, Zhou Zhou.

Formal analysis: Bimbardhar Valluru, Kalyan Sharma, Ahmed Abdullahi Adam, Zhou Zhou.

Funding acquisition: Bin Yang, Du Wei, Zhou Zhou.

Investigation: Bimbardhar Valluru, Kalyan Sharma, Ahmed Abdullahi Adam, Du Wei, Mahamed Osman Ali.

Methodology: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Ahmed Abdullahi Adam, Zhou Zhou, Mahamed Osman Ali.

Project administration: Bimbardhar Valluru, Ahmed Abdullahi Adam, Du Wei.

Resources: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Du Wei, Mahamed Osman Ali.

Software: Ahmed Abdullahi Adam, Du Wei.

Supervision: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Ahmed Abdullahi Adam, Du Wei.

Validation: Bin Yang, Ahmed Abdullahi Adam, Du Wei, Zhou Zhou.

Visualization: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Ahmed Abdullahi Adam, Du Wei, Zhou Zhou, Mahamed Osman Ali.

Writing – original draft: Bimbardhar Valluru.

Writing – review & editing: Bimbardhar Valluru, Bin Yang, Kalyan Sharma, Ahmed Abdullahi Adam, Du Wei, Zhou Zhou, Mahamed Osman Ali.

References

- [1] Stanley JC, Thompson NW, Fry WJ. Splanchnic artery aneurysms. *Arch Surg* 1970;101:689–97.
- [2] Sessa C, Tinelli G, Porcu P, et al. Treatment of visceral artery aneurysms: description of a retrospective series of 42 aneurysms in 34 patients. *Ann Vasc Surg* 2004;18:695–703.
- [3] Chughtai T, Pascual JL, Partap V, et al. Left gastric artery aneurysm: case report and review of the literature. *Int J Angiol* 2000;9:156–8.
- [4] Nishimura T, Sakata H, Yamada T, et al. Ruptured gastric artery aneurysm. *Acute Med Surg* 2016;3:39–42.
- [5] Nissim L, Diven C, Baun A. Embolization of left gastric artery pseudoaneurysm after blunt trauma. *Imaging Med* 2017;9:15–7.
- [6] Neil RT, John EY, Edwin JS. Aneurysms of gastric arteries as a cause of intraperitoneal hemorrhage: review of literature. *Ann Surg* 1968;168:294–7.
- [7] Smith-Mungo LI, Kagan HM. Lysyl oxidase: properties, regulation and multiple functions in biology. *Matrix Biol* 1998;16:387–98.
- [8] Pillai AK, Iqbal SI, Liu RW, et al. Segmental arterial mediolysis. *Cardiovasc Intervent Radiol* 2014;37:604–12.
- [9] Baker LePain JC, Stone DH, Mattis A, et al. Clinical diagnosis of segmental arterial mediolysis: differentiation from vasculitis and other mimics. *Arthritis Care Res* 2010;62:1655–60.
- [10] Tsuji N, Watanabe N, Okamoto T, et al. Specific interaction of pancreatic elastase and leucocytes to produce oxygen radicals and its implication in pancreatitis. *Gut* 1994;35:1659–64.
- [11] Niraj Jani, McGrath D, Kevin M. Left gastric artery aneurysm. *Gastrointest Endosc* 2007;67:154–5.
- [12] Mathew AJ, Raju RS, Vyas FL, et al. Left gastric artery pseudoaneurysm following traumatic pancreatic transection. *Trop Gastroenterol* 2007;28:133–4.
- [13] Smith RE, Fontanez-Garcia D, Plavsic BM. Gastrointestinal case of the day. Pseudoaneurysm of the left gastric artery as a complication of acute pancreatitis. *Radiographics* 1999;19:1390–2.
- [14] Saluja SS, Ray S, Gulati MS, et al. Acute cholecystitis with massive upper gastrointestinal bleed: a case report and review of the literature. *BMC Gastroenterol* 2007;7:12.
- [15] Khan S, Cheatle T. Left gastric artery aneurysm—a case report. *Eur J Vasc Endovasc Surg* 1997;14:413–4.
- [16] Singh K, Bønaa KH, Solberg S, et al. Intra- and interobserver variability in ultrasound measurements of abdominal aortic diameter. The Tromsø Study. *Eur J Vasc Endovasc Surg* 1998;15:497–504.
- [17] Lupattelli T. The yin-yang sign. *Radiology* 2006;238:1070–1.
- [18] Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: value of CT in establishing prognosis. *Radiology* 1990;174:331–6.
- [19] Bollen TL, Singh VK, Maurer R, et al. Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. *AJR Am J Roentgenol* 2011;197:386–92.
- [20] Jáuregui-Arrieta LK, Alvarez-López F, Cobián-Machuca H, et al. Effectiveness of the modified tomographic severity index in patients with severe acute pancreatitis. *Rev Gastroenterol Mex* 2014;73:144–8.
- [21] Pivawer G, Lee JL, Nardi PM. Case 105: left gastric artery aneurysm. *Radiology* 2007;242:312–4.
- [22] Agrawal GA, Johnson PT, Fishman EK. Splenic artery aneurysms and pseudoaneurysms: clinical distinctions and CT appearances. *AJR Am J Roentgenol* 2007;188:992–9.