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World J Clin Cases 2022 February 26; 10(6): 1754-2052



Contents

Thrice Monthly Volume 10 Number 6 February 26, 2022

OPINION REVIEW

- 1754** Gut-brain axis: Focus on gut metabolites short-chain fatty acids
Guo C, Huo YJ, Li Y, Han Y, Zhou D

MINIREVIEWS

- 1764** Association between direct-acting antiviral agents in hepatitis C virus treatment and hepatocellular carcinoma occurrence and recurrence: The endless debate
Kamal A, Elsheaita A, Abdelnabi M

ORIGINAL ARTICLE

Retrospective Cohort Study

- 1775** Effects of bilirubin on perioperative myocardial infarction and its long-term prognosis in patients undergoing percutaneous coronary intervention
Li Y, Li DB, Zhao LD, Lv QB, Wang Y, Ren YF, Zhang WB
- 1787** Disease exacerbation is common in inflammatory bowel disease patients treated with immune checkpoint inhibitors for malignancy
Rubin SJS, Balabanis T, Gubatan J, Habtezion A
- 1795** Multidrug-resistant organisms in intensive care units and logistic analysis of risk factors
Han Y, Zhang J, Zhang HZ, Zhang XY, Wang YM

Retrospective Study

- 1806** Change and impact of left ventricular global longitudinal strain during transcatheter aortic valve implantation
Zhang H, Xie JJ, Li RJ, Wang YL, Niu BR, Song L, Li J, Yang Y

Observational Study

- 1815** Early detection of noise-induced hearing loss
Meng ZL, Chen F, Zhao F, Gu HL, Zheng Y
- 1826** Empathetic nursing with mindful cognitive therapy for fatigue, depression, and negative emotions in leukemia patients undergoing long-term chemotherapy
Lu YY, Lu XM, Shao CY, Wang CC, Xu TT, Zhang BL

Prospective Study

- 1834** Superior pancreatic lymphadenectomy with portal vein priority *via* posterior common hepatic artery approach in laparoscopic radical gastrectomy
Zhang YJ, Xiang RC, Li J, Liu Y, Xie SM, An L, Li HL, Mai G

Randomized Controlled Trial

- 1843** Systematic nursing interventions in gastric cancer: A randomized controlled study
He F, He RX

META-ANALYSIS

- 1852** Impact of adding opioids to paravertebral blocks in breast cancer surgery patients: A systematic review and meta-analysis
Chen MH, Chen Z, Zhao D

CASE REPORT

- 1863** Multiple different remote epidural hematomas after craniotomy: A case report
He Q, Tao CY, Fu RH, You C
- 1869** Tuberculous pericarditis-a silent and challenging disease: A case report
Lucero OD, Bustos MM, Ariza Rodríguez DJ, Perez JC
- 1876** Transileocolic endovascular treatment by a hybrid approach for severe acute portal vein thrombosis with bowel necrosis: Two case reports
Shirai S, Ueda T, Sugihara F, Yasui D, Saito H, Furuki H, Kim S, Yoshida H, Yokobori S, Hayashi H, Kumita SI
- 1883** Efficacy of EGFR-TKI sequential therapy in patients with EGFR exon 19 insertion-positive non-small-cell lung cancer: A case report
Shan BB, Li Y, Zhao C, An XQ, Zhang QM
- 1889** Novel compound heterozygous variants in the TAF6 gene in a patient with Alazami-Yuan syndrome: A case report
Lin SZ, Feng JH, Sun LP, Ma HW, Wang WQ, Li JY
- 1896** Asymmetric limb weakness in Guillain-Barré syndrome: Three case reports
Hu M, Li X, Wong HY, Feng XG, Wang YZ, Zhang GR
- 1903** Modified treatment of knee osteoarthritis complicated with femoral varus deformity: A case report
Xu SM, Li W, Zhang DB, Bi HY, Gu GS
- 1909** Novel HNF1A gene mutation in maturity-onset diabetes of the young: A case report
Xu Q, Kan CX, Hou NN, Sun XD
- 1914** Cerebral corridor creator for resection of trigone ventricular tumors: Two case reports
Liu XW, Lu WR, Zhang TY, Hou XS, Fa ZQ, Zhang SZ
- 1922** Left abdominal wall proliferative myositis resection and patch repair: A case report
Xing RW, Nie HQ, Zhou XF, Zhang FF, Mou YH
- 1929** Concurrent ankylosing spondylitis and myelodysplastic syndrome: A case report
Xu GH, Lin J, Chen WQ

- 1937** Life-threatening subclavian artery bleeding following percutaneous coronary intervention with stent implantation: A case report and review of literature
Shi F, Zhang Y, Sun LX, Long S
- 1946** Cryptogenic organizing pneumonia associated with pregnancy: A case report
Lee YJ, Kim YS
- 1952** Eosinophilia complicated with venous thromboembolism: A case report
Su WQ, Fu YZ, Liu SY, Cao MJ, Xue YB, Suo FF, Liu WC
- 1961** Neck and mediastinal hematoma caused by a foreign body in the esophagus with diagnostic difficulties: A case report
Wang LP, Zhou ZY, Huang XP, Bai YJ, Shi HX, Sheng D
- 1966** Therapeutic endoscopy of a Dieulafoy lesion in a 10-year-old girl: A case report
Chen Y, Sun M, Teng X
- 1973** Cavernous hemangioma of an intrapancreatic accessory spleen mimicking a pancreatic tumor: A case report
Huang JY, Yang R, Li JW, Lu Q, Luo Y
- 1981** Surgery and antibiotics for the treatment of lupus nephritis with cerebral abscesses: A case report
Hu QD, Liao LS, Zhang Y, Zhang Q, Liu J
- 1991** Median arcuate ligamentum syndrome: Four case reports
Kim JE, Rhee PL
- 1998** Novel ABCB4 mutations in an infertile female with progressive familial intrahepatic cholestasis type 3: A case report
Liu TF, He JJ, Wang L, Zhang LY
- 2007** Primary duodenal dedifferentiated liposarcoma: A case report and literature review
Kim NI, Lee JS, Choi C, Nam JH, Choi YD, Kim HJ, Kim SS
- 2015** Implant site development using titanium plate and platelet-rich fibrin for congenitally missed maxillary lateral incisors: A case report
Zhang TS, Mudalal M, Ren SC, Zhou YM
- 2023** Successful embolization of an intrahepatic portosystemic shunt using balloon-occluded retrograde transvenous obliteration: A case report
Saito H, Murata S, Sugihara F, Ueda T, Yasui D, Miki I, Hayashi H, Kumita SI
- 2030** Bilateral pneumothorax and pneumomediastinum during colonoscopy in a patient with intestinal Behcet's disease: A case report
Mu T, Feng H
- 2036** Acute kidney injury due to intravenous detergent poisoning: A case report
Park S, Ryu HS, Lee JK, Park SS, Kwon SJ, Hwang WM, Yun SR, Park MH, Park Y

- 2045** Vaginal enterocele after cystectomy: A case report

Liu SH, Zhang YH, Niu HT, Tian DX, Qin F, Jiao W

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Cavernous hemangioma of an intrapancreatic accessory spleen mimicking a pancreatic tumor: A case report

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Abstract

BACKGROUND

Intrapaneatic accessory spleen (IPAS) is an uncommon condition, with the majority of cases presenting as solid lesions. Thus, this condition is frequently misdiagnosed as pancreatic solid neoplasm. Moreover, splenic cavernous hemangioma is a rare disorder, whereas lesions with a cystic appearance arising from IPAS have not been reported.

CASE SUMMARY

Herein, we present a case involving a 32-year-old male who had a complex cystic lesion in the tail of the pancreas revealed by conventional ultrasound. The lesion was misdiagnosed as a pancreatic cystadenoma because of its confusing anatomic location, as well as due to its peripheral nodular and internal septal enhancement patterns on contrast-enhanced ultrasound. After multidisciplinary discussion, the patient finally underwent laparoscopic pancreatic body and tail resections. Postoperative pathology demonstrated the lesion to be a cavernous hemangioma arising from the IPAS.

CONCLUSION

Cavernous hemangioma in the intrapancreatic accessory spleen may mimic pancreatic cystadenoma, which is a condition with the potential to be malignant. Imaging follow-ups or surgical interventions may be helpful for the exclusion of malignant risks in complicated cystic lesions, especially those with parietal and septal enhancements.

Key Words: Intrapaneatic accessory spleen; Pancreas; Diagnosis; Contrast enhanced ultrasound; Case report

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Core Tip: Intrapaneatic accessory spleen (IPAS) is an uncommon condition; however, overlapping imaging manifestations of IPAS and pancreatic tumors may lead to unnecessary surgery. Cystic splenic cavernous hemangioma is a rare disorder, whereas lesions with a cystic appearance arising from IPAS have not been reported. Herein, we report a cavernous hemangioma in the IPAS that was misdiagnosed as being a pancreatic cystadenoma *via* contrast-enhanced modalities. The diagnosis of cystic lesions in IPAS can be challenging. Imaging follow-ups or surgical interventions may be needed for the possible malignancy risk of a complicated cystic lesion, especially those with parietal and septal enhancements.

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INTRODUCTION

An intrapancreatic accessory spleen (IPAS) is an uncommon condition, with a prevalence ranging from 1.1%-3.4% in individuals[1,2]. An IPAS is typically asymptomatic and has an innocuous nature. However, overlapping imaging manifestations of an IPAS and primary pancreatic tumors may lead to unnecessary surgery[3]. A typical IPAS demonstrates a solid lesion with a round, oval or triangular shape, which is similar to the spleen on both precontrast and contrast-enhanced images. Therefore, this disorder is frequently confused with adenocarcinomas, neuroendocrine tumors or other solid pancreatic entities. When compared with a solid IPAS, cystic lesions arising from an IPAS are rare but necessitate a differential diagnosis with pancreatic cystic neoplasms, especially those possessing the potential to be malignant. Moreover, when considering the high likelihood of false-negative results, biopsy of cystic pancreatic lesions is seldom performed, and surgery is ultimately performed in most patients.

Herein, we report such a case involving a patient who underwent laparoscopic pancreatic body and tail resections because of an indeterminate pancreatic cystic lesion. Postoperative pathology confirmed this lesion as being a cavernous hemangioma arising from an IPAS. Furthermore, the clinical and imaging characteristics of IPAS and pancreatic cystic neoplasms (according to the previous literature) were also reviewed (Table 1).

CASE PRESENTATION

Chief complaints

A 32-year-old male was referred to our hospital because of a suspicious lesion neighboring the hilum of the spleen, which was detected *via* conventional grayscale ultrasound in a local community hospital. The patient did not complain of obvious discomfort.

History of past illness

The patient had a history of chronic hepatitis B.

Physical examination

The patient did not complain of abdominal pain or any remarkable discomfort during the physical examination.

Laboratory examinations

In addition to a slightly increased albumin-globulin ratio (2.96) and glutamine transpeptidase level (63 IU/L), no abnormal laboratory test results, including those of related tumor markers, were found.

Imaging examinations

The patient underwent contrast-enhanced ultrasound (CEUS) in our department. Before the CEUS, a baseline ultrasound illustrated a complicated cystic nodule measuring 2 cm, with a well-defined border in the tail of the pancreas without salient blood supply on color Doppler ultrasound (Figure 1). For the CEUS, a bolus injection of the US contrast agent SonoVue (Bracco, Milan, Italy) was administered through the antecubital vein, followed by a flush of 5 mL of 0.9% normal saline. The lesion demonstrated peripheral nodular and internal septal isoenhancement in the arterial phase, followed by slight hyperenhancement of the enhanced area in the venous phase. The predominant cystic area of the lesion did not show any enhancement in either phase. According to the aforementioned enhancing pattern in the CEUS, the lesion was suspected to be a pancreatic cystadenoma *via* CEUS (Figure 1).

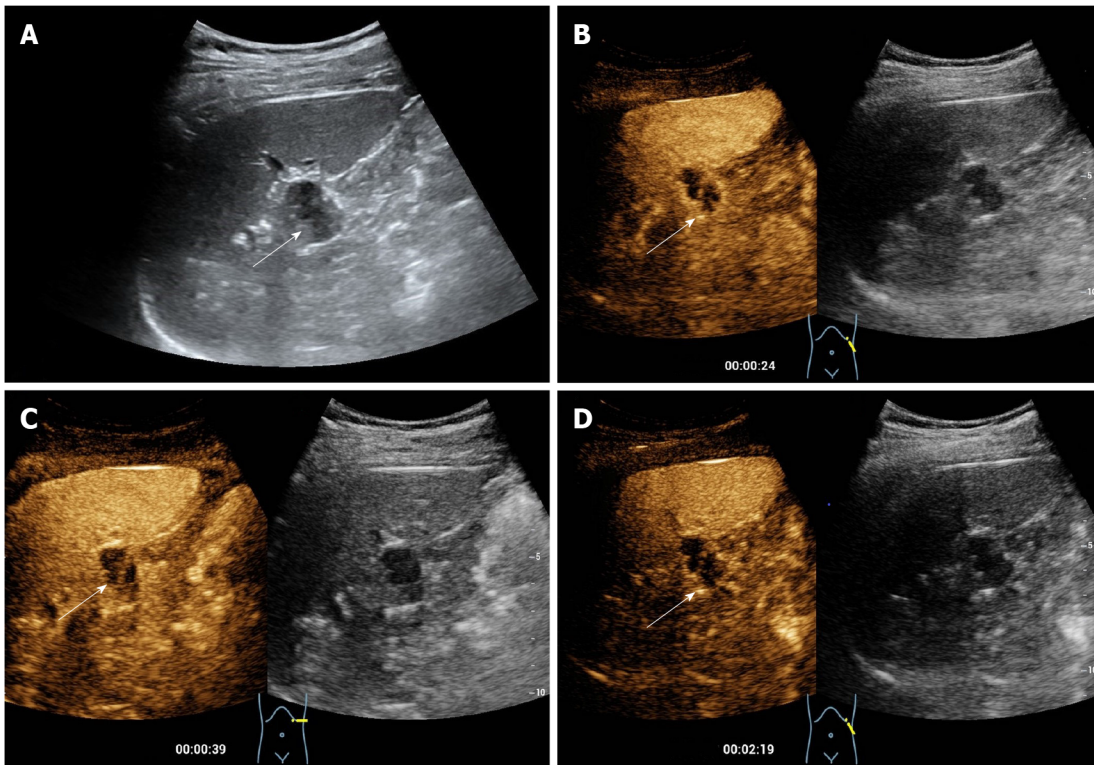


Figure 1 Pre-contrast and contrast enhanced ultrasound of the pancreatic lesion. A: A complicated cystic lesion (arrow) measuring 2 cm was detected in the tail of the pancreas by grayscale ultrasound in a 32-year-old male patient; B: Peripheral nodular and internal septal isoenhancement (arrow) in the arterial phase was shown on contrast-enhanced ultrasound; C and D: The enhanced part of the lesion exhibited mild hyperenhancement in the early venous phase without definite washout in the late venous phase. The cystic component did not show any enhancement through either phase.

Contrast-enhanced computed tomography (CECT) was performed to further examine the lesion. On the unenhanced CT, a nodule with a diameter of 2.2 cm and slightly low density was identified in the tail of the pancreas. Septa were observed, whereas no significant enhancement was presented within the lesion (Figure 2). The nodule was diagnosed as being a pancreatic cystic lesion *via* the CECT. Moreover, no salient abnormalities were found in the liver, kidney, spleen or biliary system *via* imaging evaluations.

FINAL DIAGNOSIS

The lesion was misdiagnosed as pancreatic cystadenoma by CEUS and CECT.

TREATMENT

After multidisciplinary discussion and communication with the patient, as well as with his family, laparoscopic pancreatic body and tail resections were performed.

OUTCOME AND FOLLOW-UP

Postoperative pathology demonstrated that the lesion was a splenic cavernous hemangioma in the pancreas (Figure 3). After an uneventful postoperative course, the patient was discharged on postoperative day 5. No obvious abnormality was found in a follow-up abdominal US one month later (Timeline of diagnosis and treatment of the pancreatic lesion is presented in Supplementary Figure 1).

DISCUSSION

Intrapancreatic accessory spleen is a rare congenital condition, compared with an accessory spleen

Table 1 Clinical and radiological characteristics of intrapancreatic accessory spleen and pancreatic cystic neoplasms

Pancreatic cystic neoplasms						
	IPAS	Pseudocyst	SCA	MCA	SPN	IPMN
Clinical features[1, 17,18]						
Age (mean: year)	40 to 65	At any age	60	40 to 50	30	65
Gender	Slightly higher in males	Males > females	Older females	Females > males	Young females	Males > females
Incidence	11%-17% of AS	5%-40% after pancreatitis	16% of PCN	29% of PCN	2% and 3% of PCN	20%-50% of PCN
Benign/malignant	Benign	Benign	Benign	Low malignant potential	Low malignant potential	Malignant potential
Anatomic location	Tail > head/body	1/3 near the head	Head > body/tail	Body/tail > head	Body/tail > head	Arising from the pancreatic ducts
Size (mean: cm)	≤ 2	Depending on the duration of disease	5-8	7-10	6	0.8
Potential mimickers	NET and PDAC	MCA	MCA and IPMN	MCA: IPMN and MCAC	MCA: IPMN and MCAC	SCA: MCA and MCAC
Radiological diagnosis						
Ultrasound[7,17, 19-21]						
Baseline US	Hypoechoic lesion with well-defined border	Transonic: net separation: irregular internal outline: fluid-containing lesion	Small transonic lesions with thin septa inside	Unilocular or septated cystic lesions with thickened walls and well-defined margins	Encapsulated mixed mass (solid and cystic)	Lesions developed inside the main/branch pancreatic ducts: parietal nodules and septa can be seen in the cysts
Doppler US	Blood supply may from the splenic vessels	No obvious blood flow encompass or inside the lesion	No obvious blood flow encompass or inside the lesion	No obvious blood flow encompass or inside the lesion	Blood flow signal around the tumor	No obvious blood flow encompass or inside the lesion
CEUS[19,21]	Inhomogeneous hyperenhancement followed by homogeneous hyperenhancement	Iso- or hyperenhancement of the cystic wall: without definite washout	Isoenhancement of the cystic walls and septa: without definite washout	Iso-enhancement of the cystic walls and nodules: without definite washout	Rim hyperenhancement in the capsule:centripetal hyperenhancement followed by mild washout in the solid part: no enhancement in the cystic components	Iso-enhancement in the cystic wall and nodules
CECT[18,21-23]	Inhomogeneous hyperenhancement followed by homogeneous hyperenhancement	Round or oval fluid collection with a thin: hardly perceptible wall or enhancing thick wall	Well-defined: polycystic or honeycomb lesions showing enhancing internal septa and cyst walls	Well-circumscribed round/oval macrocystic lesions with enhancement of the walls	Hypo-attenuating on pancreatic phase followed by homogeneous gradual enhancement to iso-attenuating on the hepatic venous phase	Dilated main/side pancreatic ducts: nodules arising from the ducts manifest hyperattenuating at contrast-enhanced CT
CEMRI[22,24]						
T1-W	Inhomogeneous hypointensity	Blood products and necrotic components commonly present intrins-	High intensity fluid in the cysts	Homogeneous low t1 signal intensity	Low signal intensity: SPN with hemorrhage presents t1 hyperintensity	Loss of t1 signal and delayed uptake of contrast material

		ically increased t1 signal intensity: the thickened wall shows a rim hyperintensity				
T2-W	Homogeneous hyperintensity	The hyperintensity in tissues surrounding the pseudocyst represents the inflammation on t2 fat-suppressed images	Honeycomb pattern (microcysts) or macrocysts manifest signal intensity of simple fluid	Homogeneous high t2 signal intensity	Predominantly solid show mildly increased t2 signal intensity: cystic-dominated present t2 signal intensity closer to that of fluid	Papillary excrescences or nodules in the walls of the dilated ducts present hypointense on t2-weighted images
Management	Usually require no treatment	Serial imaging follow-up	Follow-up or resection depending on the size of the tumor	Surgical resection	Surgical resection	Recommended to be surgically resected

AS: Accessory spleen; IPAS: Intrapaneatic accessory spleen; PCN: Pancreatic cystic neoplasm; SCA: Serous cystadenoma; MCA: Mucinous cystadenoma; SPN: Solid pseudopapillary neoplasm; IPMN: Intraductal papillary mucinous neoplasm; MCAC: Mucinous cystadenocarcinoma; US: Ultrasound; CEUS: Contrast enhanced ultrasound; CECT: Contrast enhanced computerized tomography; CEMRI: Contrast enhanced magnetic resonance imaging; T1-W: T1-weighted; T2-W: T2-weighted.

located at the hilum of the spleen[2,4]. Due to its innocuous nature and infrequent induction of symptoms, IPAS seldom requires therapy unless they cause symptoms as a result of the compression, torsion or spontaneous rupture of a hemorrhage[5,6].

Typical IPAS presents as a solid lesion and demonstrates similar manifestations to the spleen on both precontrast and contrast-enhanced ultrasound[7,8]. However, cystic neoplasm development in IPASs is rare. Sporadic cases of epidermoid cysts in IPASs (known as ECIPASs) have been reported[6,9-11]. The walls of ECIPASs are irregularly thickened and thicker than those of mucinous cystic neoplasms (MCNs) and intraductal papillary mucinous neoplasms (IPMNs)[9]. Moreover, the evident contrast enhancement of the partially thickened wall of ECIPAS (which is similar to that of the spleen) makes it possible to distinguish ECIPASs from MCNs or IPMNs.

The differential diagnosis was even more considerable in our case. The cystic cavernous hemangioma in the IPAS (known as CHIPAS) presented peripheral nodular and internal septal enhancements, which are frequently observed in pancreatic mucinous cystadenomas (MCAs). Furthermore, the majority of MCAs are located in the tail of the pancreas, where IPASs are also frequently discovered[12]. Therefore, this increases the difficulty of an accurate diagnosis. However, the ancillary features of a fibrous pseudocapsule or calcified contents inside of the MCNs have also been reported[13]. Another pancreatic cystic lesion that warrants vigilant discrimination from the CHIPAS is an IPMN. An IPMN in the main duct possesses a high risk of malignancy, with 38%–68% being confirmed as high-grade dysplasia or pancreatic cancer in postoperative specimens[14]. Fortunately, CEUS is sensitive in being demonstrated in the dilated main pancreatic duct and the polycystic lesion connecting to the pancreatic duct or in developing within the duct in cases of IPMNs[15].

To our knowledge, there is only one case report of solid cavernous hemangioma detected in both the spleen and the IPAS[16]. In this case, the CHIPAS was accurately identified by the investigators because of a similar enhancement pattern of the pancreatic lesion and the splenic lesions on CECT and contrast-enhanced magnetic resonance imaging. An accurate diagnosis was more difficult, as in our patient, because there was no lesion in the spleen for comparison. Moreover, a splenic hemangioma typically shows a hyperechoic and solid appearance. The atypical cystic appearance in our patient increased the

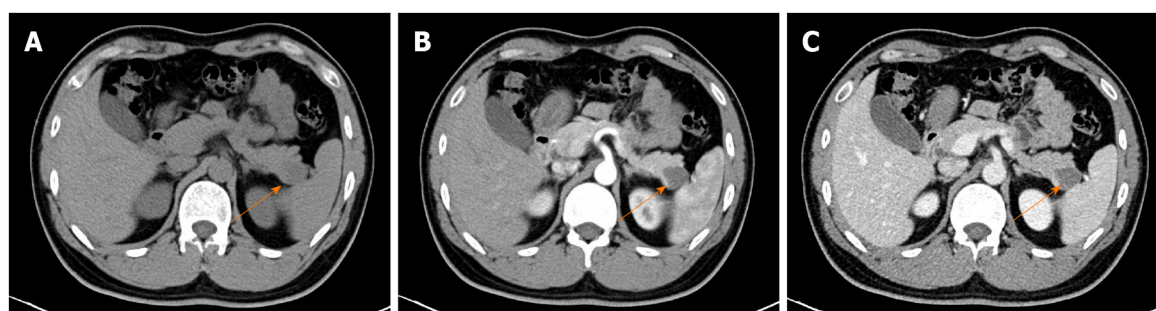


Figure 2 Pre-operative computed tomography scan of the pancreatic lesion. A: A slightly low-density nodule measuring 2.2 cm (arrow) was found in the tail of the pancreas on unenhanced computed tomography (CT); B and C: Septa were faintly visible whereas no salient enhancement was presented within the lesion (arrows) in either the arterial or the venous phases on axial contrast-enhanced CT.

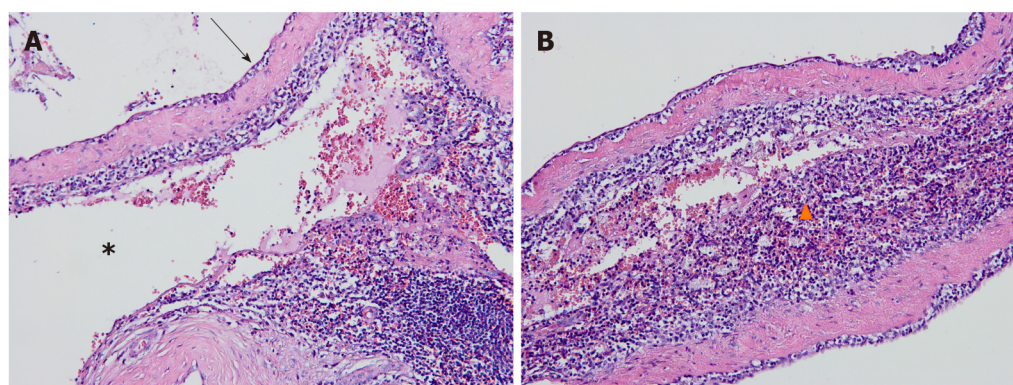


Figure 3 Hematoxylin-eosin staining of the cavernous hemangioma arising from the intrapancreatic accessory spleen. A: Large dilated vascular spaces (asterisk) separated by fibrous septa and endothelial cells (arrows) lining on the surface of the vascular spaces were observed in the intermediate-power view (original magnification, 200×); B: A high-powered photomicrograph (original magnification, 400×) illustrated splenic tissues (triangles) adjacent to the vascular spaces.

difficulty of making an accurate diagnosis.

Herein, we presented on an extremely rare case of a cystic cavernous hemangioma arising from an IPAS. Contrast-enhanced ultrasound is sensitive in demonstrating the enhancements of the septa and the parietal nodule. However, an accurate diagnosis of cystic cavernous hemangioma arising from an IPAS *via* imaging tools is challenging. Imaging follow-ups or surgical interventions may be needed, due to the possible malignancy risk of a complicated cystic lesion with parietal and septal enhancements.

CONCLUSION

Cavernous hemangioma in the intrapancreatic accessory spleen may mimic pancreatic cystadenoma, which is a condition with the potential for malignancy. Imaging follow-ups or surgical interventions may be helpful for the exclusion of malignant risks in complicated cystic lesions, especially those with parietal and septal enhancements.

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FOOTNOTES

Author contributions: Luo Y performed the contrast-enhanced ultrasound examination for the patient and proposed writing it up as a case report; Huang JY collected the clinical information of the patient, reviewed the literature and contributed to manuscript drafting; Yang R provided the pathological data and helped with creating the figures; Li JW contributed to revising the grammar of the manuscript; Luo Y and Lu Q were responsible for the revision of the

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