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Contents

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REVIEW

ORIGINAL ARTICLE

Retrospective Study

CASE REPORT

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Contents

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CASE REPORT

Cystic ductal adenocarcinoma of pancreas complicated with neuroendocrine tumor: A case report and review of literature

Dong-Mei Zou, Zeng-Yi Shu, Xu Cao

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Abstract

BACKGROUND

Difficulties in making an accurate preoperative diagnosis of cystic pancreatic lesions pose a challenge for radiologists. It would be helpful to report rare cases and review the literature.

CASE SUMMARY

In the present report, a case of a patient with a pancreatic cystic lesion initially misdiagnosed as a pseudocyst by radiologist was documented, which was later pathologically confirmed as pancreatic ductal adenocarcinoma with neuroendocrine tumor. However, subsequent literature review yielded no previous reports of pancreatic ductal adenocarcinoma with neuroendocrine tumors and cystic lesions. Therefore, literature on the imaging diagnosis of pancreatic cystic lesions was instead reviewed and discussed.

CONCLUSION

Careful evaluation of the characteristics revealed by multimodal imaging techniques, medical history, laboratory examination data and follow-up observations, is critical to the diagnosis and treatment of pancreatic cystic disease. We provide valuable insights into the diagnosis of pancreatic cystic disease through a rare case report and literature review.

Key Words: Pancreas; Ductal adenocarcinoma; Cystic; Neuroendocrine tumor; Case report

Core Tip: A case of cystic pancreatic ductal adenocarcinoma with neuroendocrine involvement that was initially misdiagnosed as a pseudocyst was reported. The lack of specific symptoms and distinctive radiological features poses challenges in the preoperative diagnosis of pancreatic cystic lesions, particularly in differentiating between atypical and rare lesions.

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INTRODUCTION

Pancreatic neoplasms typically have severe health implications. They can be categorized as either exocrine or endocrine tumors based on their cellular origin and histopathology. Exocrine tumors account for > 95% cases of pancreatic neoplasms, of which ductal adenocarcinoma of the pancreas is the most common type. This type of malignancy is associated with poor prognosis[1]. By contrast, pancreatic neuroendocrine neoplasms are relatively rare, constituting 1%-2% of all pancreatic neoplasms[2]. With advancements in imaging technology, the detection rate of pancreatic tumors is progressively improving. However, at present, the complex anatomical location, secretory function and various pathological subtypes of the pancreas can lead to different imaging manifestations and misdiagnosis, especially when the lesions exhibit cystic features. Pancreatic cystic lesion (PCL) is a term used to describe malignant, potentially malignant and benign lesions[3]. The management and treatment strategies of such cystic lesions vary greatly, which depend on the different pathological types. Therefore, making accurate differential diagnoses will have important clinical implications.

In the present case report, a patient with pancreatic ductal adenocarcinoma with neuroendocrine tumor showing a cystic lesion was documented. Combined with the naked eye findings of the resected specimens and the pathological changes under the microscope, it seems difficult to explain the imaging and pathological features of this case through the well-known cystic mechanism of pancreatic ductal adenocarcinoma. On this basis, discussions are provided and related literature was reviewed to improve understanding into the pathophysiology of PCLs. This may be particularly helpful in providing more comprehensive insight into pancreatic ductal adenocarcinoma.

CASE PRESENTATION

Chief complaints

A 66-year-old male patient was referred to the hospital due to persistent abdominal distension for > 1 months.

History of present illness

Previous gastroscopy examinations in other hospitals had indicated a large submucosal projection in the gastric body.

History of past illness

No special notes.

Personal and family history

No special notes.

Physical examination

Physical examination revealed no abnormalities.

Laboratory examinations

Laboratory tests revealed elevated levels of carbohydrate antigen 19-9 (CA 19-9) in the serum (51.49 U/mL; normal value, < 37 U/mL), whilst the levels of lipase (34 U/L; normal value, 0-60 U/L) and amylase (38 U/L; normal value, 0-140 U/L) were within the normal range.

Imaging examinations

Ultrasonography revealed the presence of heterogenous hypoechoic cystic lesions (Figure 1). Computed tomography (CT) scans of the abdomen, both non-enhanced and three-phase enhanced, revealed irregular cysts located in the tail of the pancreas. These cysts protruded outside the border of the pancreas, were in close proximity to the gastric wall and partially encased the blood vessels at the hilum of the spleen (Figure 1). magnetic resonance imaging (MRI) revealed a high signal intensity in the T1-weighted images (T1WI) and clear high signal intensity in T2-weighted images (T2WI). Additionally, delayed enhancement of the cyst wall was observed during the enhanced scan (Figure 2).

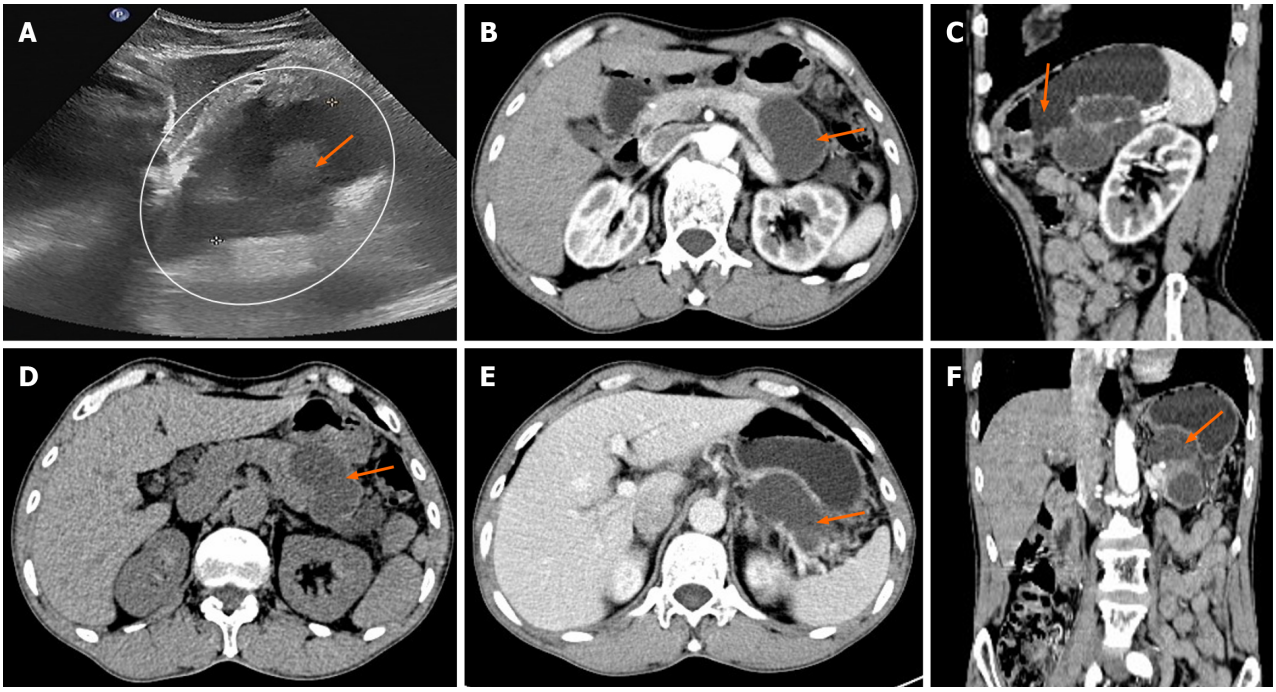

Figure 1 Preoperative ultrasound and computed tomography imaging results. A: Ultrasound imaging showed that the cystic lesion of the body and tail of the pancreas (within the ellipse) was irregular and heterogeneous hypoechoic (shown by the arrow); B: On the non-enhanced axial image, the low-density cystic mass (indicated by the arrow) in the tail of the pancreas protruding outside the outline; C: On the axial image in the arterial phase of contrast enhanced scan, the cystic mass (indicated by the arrow) does not communicate with the main pancreatic duct and the cyst wall is not exactly enhanced; D: On the axial image of the portal vein phase of contrast enhanced scan, the cystic mass (arrow) compresses the posterior wall of the gastric body and is not clearly separated from the blood vessels of the spleen; E: On the sagittal images of the arterial phase of contrast enhanced scan, the cystic mass surrounds the splenic vessels and narrows its lumen (as indicated by the arrow); F: The shape of the cystic mass on the coronal image of the arterial phase is irregular (indicated by the arrow).

FINAL DIAGNOSIS

Pathological examination of the resected mass from the body and tail of the pancreas, spleen and regional lymph nodes revealed a moderately differentiated ductal adenocarcinoma with a G1 neuroendocrine tumor. The neuroendocrine tumor component accounted for approximately 10% of the tumor (Figure 3). No tumor involvement was observed in the pancreatic capsule, the stump end of the pancreas or the spleen. Additionally, no tumor metastasis was detected in the lymph nodes. Immunohistochemistry confirmed the origin of the tumor cells from epithelial and neuroendocrine cells (Figure 4). Ultimately, the clinical diagnosis was established as moderately differentiated ductal adenocarcinoma, stage IIB (T3N1M0pm), with neuroendocrine tumor involvement.

TREATMENT

The patient underwent resection of the body and tail of the pancreas and splenectomy followed by two cycles of chemotherapy.

OUTCOME AND FOLLOW-UP

Subsequent CT examinations conducted 3 months later revealed no residual tumor, recurrence, or complications. Currently, the patient has been followed up for 7 months and remains in good health.

DISCUSSION

PCL with malignant or malignant potential include cystic ductal adenocarcinoma, intraductal papillary mucinous neoplasms (IPMN) and mucinous cystic neoplasm (MCN), solid pseudopapillary tumors (SPT) and cystic neuroendocrine tumors (CNET). By contrast, benign pancreatic lesions can include serous cystadenoma (SCA), pseudocysts and squamous epithelium lined PCL [such as pancreatic lymphoepithelial cyst (PLEC) and pancreatic epidermoid cyst (EDC)][3].

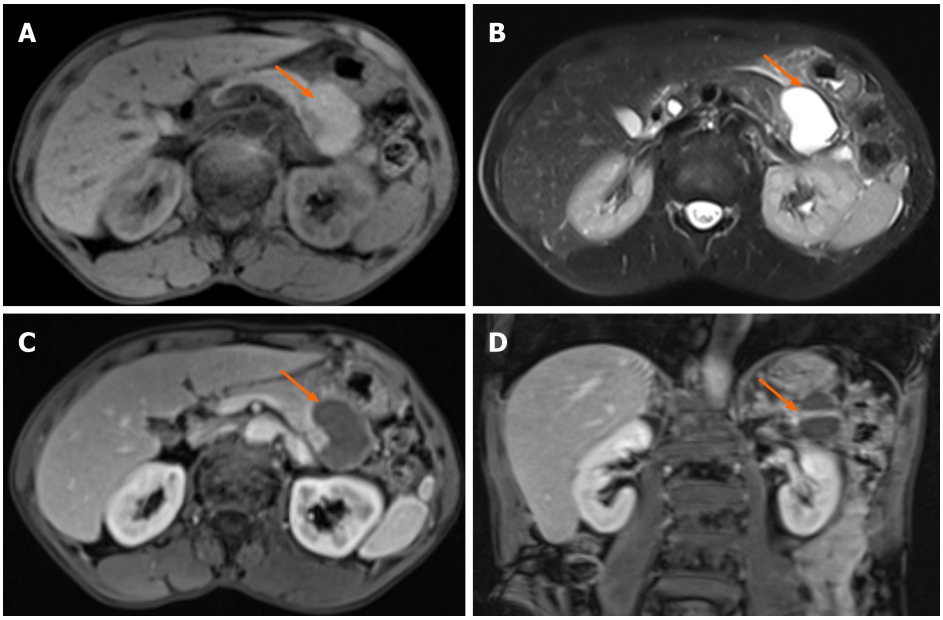

Figure 2 Preoperative magnetic resonance imaging results. A: The cystic lesions (arrow) showed high signal intensity in the cyst and low signal intensity in the cyst wall on the T1-weighted axial images; B: The cystic lesions (arrow) showed obvious high signal intensity and the cystic wall showed low signal intensity on the T2-weighted axial images; C: Contrast-enhanced axial images showed enhancement of the cyst wall (arrow); D: The shape of the cyst (arrow) was irregular and the cyst wall was enhanced on the contrast-enhanced coronal images.

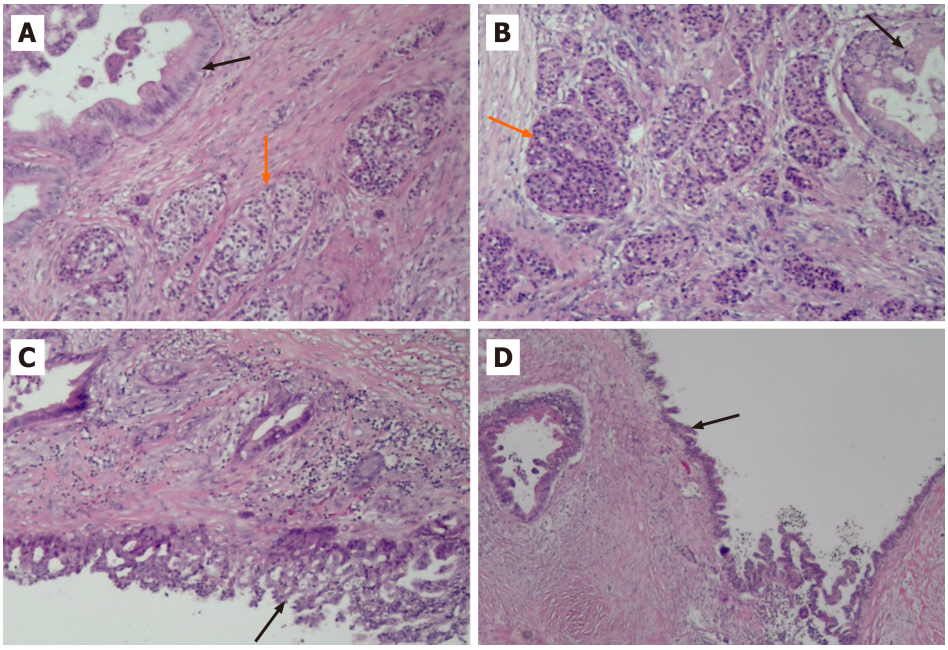

Figure 3 Ductal adenocarcinoma with neuroendocrine tumor. A and B: The black arrow indicates ductal adenocarcinoma and the orange arrow indicates neuroendocrine tumor. H&E staining, magnification, ×100; C and D: The black arrow indicates the atypical epithelial cells lined with the cyst wall. H&E staining, magnification, x100.

In total, approximately 7% pancreatic ductal adenocarcinoma (PDAC) exhibit cystic lesions on imaging[4-7]. According to the histopathological subtypes, cystic lesions caused by PDAC can be divided into neoplastic and non-neoplastic subtypes[8]. Neoplastic cysts in PDAC include large-duct type cysts, neoplastic mucin cysts, colloid carcinoma and degenerative cystic changes. Non-neoplastic cystic lesions in PDAC include retention cyst changes caused by duct obstruction and pseudocysts caused by tumor-associated pancreatitis[4,9-11]. PDAC with large-duct type cysts tend to remain solid, with several small intratumoral cysts, which can exhibit the 'honeycomb' appearance in the images. Therefore, they are prone to misdiagnosis as SCA or branched pancreatic duct type IPMN[9,12]. PDAC with neoplastic mucin cysts are caused by mucin-producing tumor cells that are typically solitary with cysts ≤ 7 cm in size. However, they do occasionally present in multiple cysts. PDAC with neoplastic mucin cysts should be differentiated from IPMN with invasive carcinoma, since the former will more likely present as a solid mass, whilst the latter will more likely

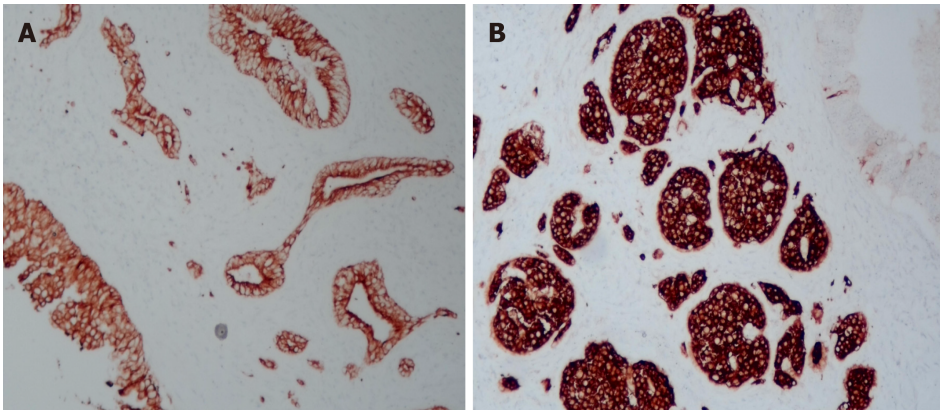

Figure 4 Immunohistochemistry results. A: Cytokeratin 8 immunohistochemical staining was positive as a marker of glandular epithelium in ductal adenocarcinoma. Magnification, ×200; B: As a neuroendocrine tumor marker, synaptophysin was positive in immunohistochemical staining. Magnification, ×200.

appear around the solid mass[10]. Colloid carcinoma is a histological variant of PDAC and has a superior prognosis compared with that of other PDAC subtypes. Because of its rich extracellular mucus, it frequently presents as high-signal intensity on T2WI and internal reticular and marginal progressive enhancement on MRI[13]. The high-signal intensity on both T1WI and T2WI in our case may be caused by intracapsular hemorrhage[14], which can be inferred from the blood clots found after operation. Pathologically, PDAC with degenerative cystic changes are formed by tumor necrosis, with necrotic and hemorrhagic tissues in the cavity. It typically presents as a large single cystic lesion in the tumor, where the edge and center of the tumor is irregular[4,10]. PDAC with retention cysts is caused by tumor obstruction of the pancreatic duct. Thorough evaluation of the area around the cyst may reveal poorly enhanced ductal adenocarcinoma near the reserved cyst[12,15]. PDAC can cause pancreatitis due to obstruction of ducts, which may lead to the formation of pseudocysts. If there is no indication of acute pancreatitis in elderly patients, the possibility of occult PDAC with pseudocyst should be considered. In the present case, despite the absence of solid components in the cystic lesions, the cystic fluid was non-colloid, where pathologically, atypical epithelial cells were observed on the lining of the cyst wall. These findings do not appear to be fully explained by the aforementioned cystic PDAC. Therefore, it was hypothesized that the mechanism underlying lesion formation in the present case may initially be ductal intraepithelial neoplasia originating from ductal retention cysts, before finally developing into invasive carcinoma.

CNET account for 13%-17% of all pancreatic neuroendocrine tumors[15]. The mechanism of neuroendocrine tumor cyst formation is proposed to be caused by the slow and expansive growth of the tumor, leading to the formation of fibrous capsules and cystic degeneration, characterized by hemorrhage and necrosis in the tumor or tumor growth in the duct[16-18]. Although cystic changes do occur, their solid components will typically show a strong arterial enhancement, enabling its distinguishment from PDAC[19]. The present case does not satisfy this diagnosis of mixed tumor because the neuroendocrine tumor accounted for only 10% of the total tumor. It may also be because the enhanced scan showed delayed enhancement of the cyst wall, which is different from the features shown by the early enhancement of CNET. Although a number of reports of mixed tumors of the pancreas have been previously documented[20-22], completely cystic lesions lined with epithelium remained scarce, which was the case in the present report.

IPMN accounts for 21%-33% of all PCL and typically occurs in the head of pancreas[23,24]. This form of lesions will progress into invasive cancer 27.6%-68% of the time, which is the highest potential of all precancerous lesions of PDAC known. It can be subdivided into main duct, branch duct and mixed type[25-27]. Thin-slice enhanced CT and 3D-magnetic resonance cholangiopancreatography are beneficial techniques for determining the relationship between cystic masses and the main pancreatic duct, in addition to the solid components of intramural nodules or adherent growth, in turn facilitating diagnosis. MCN account for 10% of all pancreatic cystic lesions[28]. They are frequently located in the body or tail of the pancreas, especially in female patients aged approximately 40 years. Imaging findings will typically reveal a well-encapsulated cystic mass, accompanied by septal mucus and hemorrhagic fluid contents, where peripheral calcification can be observed on CT[29]. SPT are rare, commonly observed in female patients aged 30 years. Intratumoral bleeding components are frequently observed in SPT cases. MRI would beneficial in this case for analyzing the extent of hemorrhagic degeneration in the lesions[30]. SCA is a cystic cavity consisting of glycogen-rich monolayer cuboidal epithelial cells. In terms of size, number and arrangement of the cyst, serous cystic tumor can be divided into single cyst type, polycystic type, honeycomb type and mixed type[31]. Among them, the honeycomb type is comprised of numerous tiny 'sacs' (≤ 1 cm) arranged in a dense honeycomb shape, where the intervesicular fibrous septum condenses into a central scar and may calcify was seen as the characteristic imaging manifestation[32]. In addition, rare cases may appear solid on CT, with obvious enhancements at arterial phase, meaning that they are prone to misdiagnosis as neuroendocrine tumors. MRI T2WI shows the characteristics of cystic signals in the tumor, which is helpful for differentiation[33,34].

PLEC is a rare cystic lesion of the pancreas, which mainly originates from the residual epithelium of the peripancreatic lymph nodes and is most frequently observed in middle-aged and elderly male patients[35,36]. PLEC can present as unilocular or multilocular cystic lesions. On CT plain scans, highly invasive cysts that are thin and uniform with high density protruding beyond the outline of the pancreas and septum of the cyst wall can be seen, coupled with rare calcification[37]. By contrast, MRI typically reveals low signal on T1WI and high signal on T2WI. Contrast-enhanced scan

will show slight enhancement of the cyst wall or septum[38]. A small number of cases have previously reported that their cellular morphological features may overlap with other benign and malignant pancreatic lesions[39]. EDC is a rare non-neoplastic true cyst of the pancreas. Pancreatic EDC originates from an intrapancreatic accessory spleen, normally as a cystic lesion at the edge of the pancreatic tail[40]. If imaging features similar to normal spleen are identified in the pancreas, such as superparamagnetic iron oxide-enhanced MRI and the presence of residual spleen tissue can be confirmed, then the radiological diagnosis of pancreatic EDC can be provided[41].

Due to the overlapping imaging features among the various pancreatic cystic lesions, non-invasive diagnosis based solely on imaging examination is both critical and challenging. Accurate diagnosis requires the consideration of multiple complex factors, such as patient age, tumor location, composition of cystic lesions, relationship with the pancreatic duct and findings around the cystic lesions. Additional cellular and molecular investigations will be required to obtain precise diagnostic, treatment and prognostic information.

In the present particular case, the radiologist initially misdiagnosed the lesion as a pseudocyst. This could be attributed to similarities in imaging features between pseudocysts and the absence of solid components. Furthermore, uncertainty regarding whether the patient had experienced pancreatitis prior to admission and the lack of a significant increase in CA199 levels may have contributed to the misdiagnosis. Subsequent postoperative diagnosis confirmed pancreatic ductal adenocarcinoma with a neuroendocrine tumor which, to the best of our knowledge, is a rarely reported pathological diagnosis for pancreatic cystic lesions.

CONCLUSION

The lack of specific symptoms and distinctive radiological features poses challenges in the preoperative diagnosis of pancreatic cystic lesions, particularly in differentiating between atypical and rare lesions. The present case, along with a review of the literature, highlights the importance of carefully assessing the characteristics of each type of these lesions by using multimodal imaging techniques and integrating them with the patient's medical history, laboratory examinations and follow-up observations. This comprehensive approach is crucial in determining the necessity for surgical resection and obtaining accurate diagnostic and prognostic information. Our report on a rare case provides valuable insights into the diagnosis of pancreatic cystic disease.

FOOTNOTES

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