Solid pseudopapillary neoplasm of the pancreas: A case report

LEI ZHANG, DONGHUI JI and KUOPENG LIANG

Department of Ultrasound, Xingtai People's Hospital, Xingtai, Hebei 054001, P.R. China

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Abstract. Solid pseudopapillary neoplasm of the pancreas (SPN) is a rare exocrine tumor, accounting for <3% of all pancreatic neoplasms. SPN predominantly occurs in young females and typically presents with nonspecific clinical symptoms, including abdominal discomfort, bloating, abdominal pain and palpable abdominal masses. In certain cases, the tumor is detected incidentally during routine physical examinations. This article reports a case of SPN located in the pancreatic tail, which was initially identified during a routine health examination, subsequently diagnosed using contrast-enhanced ultrasound and enhanced computed tomography (CT), and ultimately confirmed by surgical pathology. Given that SPN is commonly diagnosed using CT and magnetic resonance imaging, the use of ultrasound for the initial diagnosis in this case renders it particularly noteworthy.

Introduction

Solid pseudopapillary neoplasm of the pancreas (SPN) is a rare low-grade malignant tumor, accounting for ~0.2-2.7% of all pancreatic neoplasms (1-3). As morphological and pathological studies on SPN have advanced in both domestic and international research settings, the lesion has been referred to using several different names, including pancreatic papillary-solid tumor, pancreatic papillary-cystic tumor, pancreatic cystic-solid tumor and pancreatic cystic-solid papillary acinar cell tumor (4-6). In 1996, the World Health Organization (WHO) officially designated SPN based on its characteristic solid pseudopapillary architecture and classified it as a borderline malignant tumor with indeterminate biological behavior (7). By 2010, the WHO further defined SPN as a low-grade malignant neoplasm (8). SPN predominantly occurs in young females and its clinical presentation is typically nonspecific. Preoperative diagnosis primarily depends on imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI). Complete surgical resection remains the treatment of choice. The postoperative prognosis for SPN is generally favorable, with the reported 5-year survival rate exceeding 95% (1).

Case report

The patient was a 20-year-old female with no significant clinical symptoms. A mass in the pancreatic tail was incidentally detected during a routine physical examination and the patient was subsequently admitted to Xingtai People's Hospital (Xingtai, China) in March 2025 for further evaluation and management. The patient's laboratory findings, including complete blood count, cytokine analysis, comprehensive biochemical assays, neutrophil apolipoprotein measurement and thymidine kinase 1 testing, demonstrated no clinically significant abnormalities. Conventional ultrasound demonstrated an irregularly shaped, well-circumscribed cystic-solid lesion measuring ~48x44 mm in the tail of the pancreas (Fig. 1A). Color Doppler flow imaging revealed no detectable blood flow within the lesion (Fig. 1B). Contrast-enhanced ultrasound showed synchronous enhancement between the lesion and the pancreas beginning at 10 sec, characterized by heterogeneous moderate enhancement with visible non-enhancing areas, followed by persistent hypo-enhancement (Fig. 1C and D). The ultrasound diagnosis indicated a cystic-solid mass in the pancreatic tail, likely benign and of pancreatic origin, with SPN not entirely excluded. Enhanced CT scan revealed an irregular mass measuring ~49x42 mm in the pancreatic tail, showing CT attenuation values of 28-55 HU in the arterial phase (Fig. 2A) and 36-68 HU in the venous phase (Fig. 2B). The lesion exhibited gradual mild enhancement, with visible calcifications and internal septations, as well as an intact capsule and well-defined margins. Radiological diagnosis suggested a space-occupying lesion in the pancreatic tail, with SPN being a probable diagnosis. Given the definitive diagnosis and absence of comorbid conditions, tumor marker testing was not performed. Following complete surgical resection, histopathological analyses confirmed the diagnosis of SPN with focal cystic degeneration and hemorrhage (Fig. 3A and B). H&E staining was performed as follows: The tumor tissues were fixed in 10% formalin for >24 h at 25˚C (Beijing BioDee Biotechnology Co., Ltd.), followed by gradient alcohol dehydration and embedding in paraffin. Subsequently, 3-μm thick sections were prepared and stained with H&E. The ICD-10 classification code for this disease is D37.700x003 (7). According to the ICD-10 classification system, borderline tumors are assigned codes within the range D37 to D48. Within

Correspondence to: Dr Lei Zhang, Department of Ultrasound, Xingtai People's Hospital, 818 Xiangdu North Road, Xingtai, Hebei 054001, P.R. China
E-mail: 495318153@qq.com

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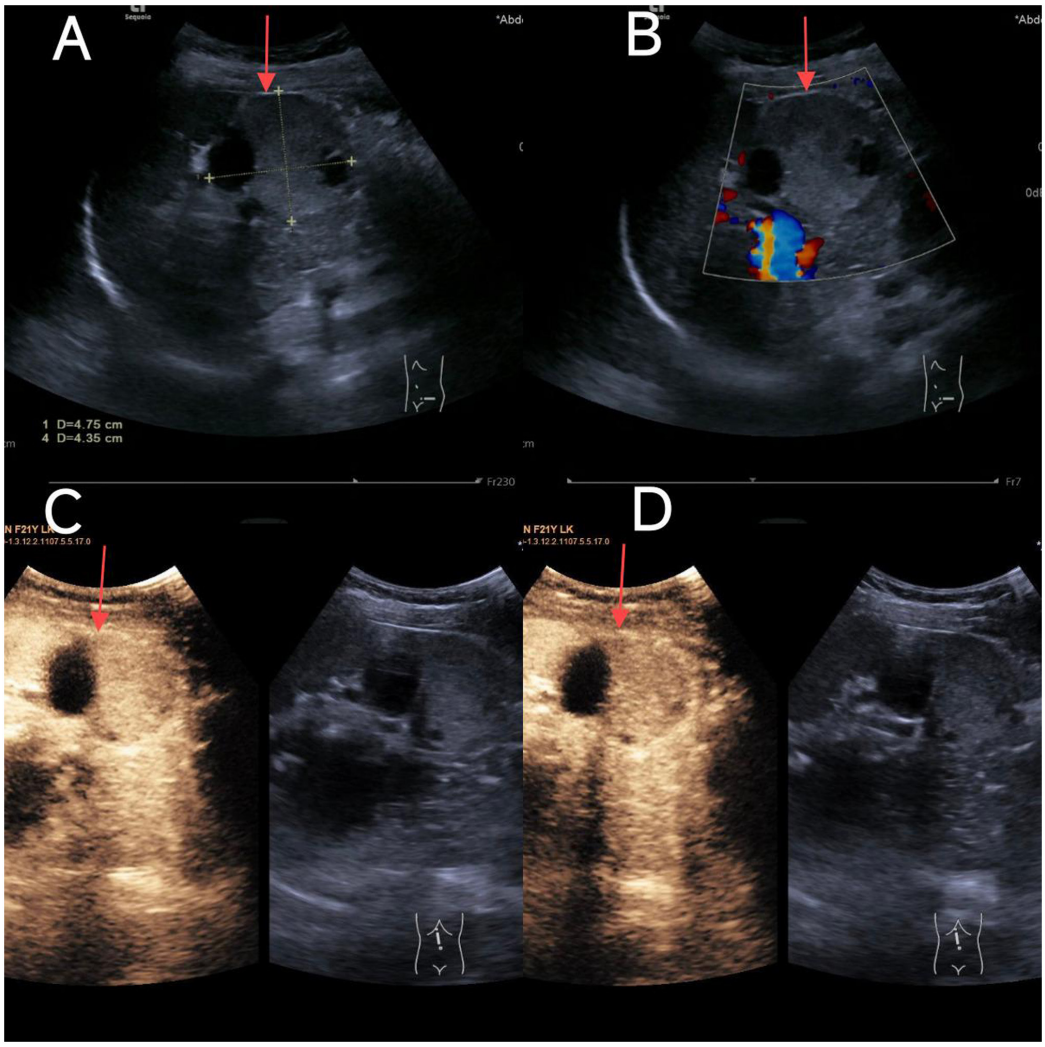

Figure 1. Ultrasonographic findings. (A) Conventional ultrasound examination identifying a cystic-solid lesion measuring ~48x44 mm located in the tail of the pancreas (arrow). (B) Color Doppler flow imaging revealing no detectable intratumoral blood flow (arrow). (C) The lesion demonstrates moderate enhancement, with clearly delineated non-enhancing regions during the arterial phase on contrast-enhanced ultrasound (arrow). (D) Enhanced ultrasound showing persistent low enhancement of the lesion in the venous phase (arrow).

this range, codes D37 to D44 are used for tumors categorized as 'uncertain malignant potential' (dynamic undetermined), whereas codes D45 to D48 are assigned to tumors classified as 'unspecified behavior' (dynamic unknown). The specific ICD-10 code for borderline pancreatic tumors is D37.7, which is classified under the category 'tumors of other digestive organs, uncertain malignant potential or unspecified behavior'. The patient completed the initial follow-up examination within one month following surgery, with clinical observations confirming satisfactory wound healing and the restoration of normal gastrointestinal function. Subsequent follow-up evaluations should be performed at regular intervals of 3 to 6 months. Postoperative follow-up demonstrated no abnormal abdominal masses or symptoms and enhanced CT imaging revealed no radiological evidence of tumor recurrence or metastasis. It is essential to closely monitor the patient's psychological condition and provide timely psychological counseling and support to alleviate negative emotions, such as anxiety and depression. Simultaneously, the patient should be encouraged to maintain a positive and optimistic outlook in order to strengthen their confidence in the recovery process.

Discussion

SPN is a rare low-grade malignant tumor characterized by solid and pseudopapillary architectural features (4). Since its initial description by Frantz (9) in 1959, the understanding of SPN has progressively advanced. In recent years, with advancements in imaging modalities such as CT and MRI, along with improvements in clinical diagnostic and therapeutic capabilities, the reported incidence of SPN has increased (10). The histogenesis and underlying pathogenetic mechanisms of SPN remain incompletely understood. Kosmahl *et al* (11) proposed that SPN originates from primordial cells of the reproductive ridge and ovarian anlagen, which may integrate with the developing pancreatic primordium during embryogenesis. This hypothesis aligns with the clinical observation that SPN predominantly affects young females (2). Current evidence suggests that SPN development is associated with mutations in exon 3 of the β-catenin gene and reduced E-cadherin-mediated signal transduction within the Wnt signaling pathway (12,13). Furthermore, emerging studies indicate potential involvement of other molecular pathways,

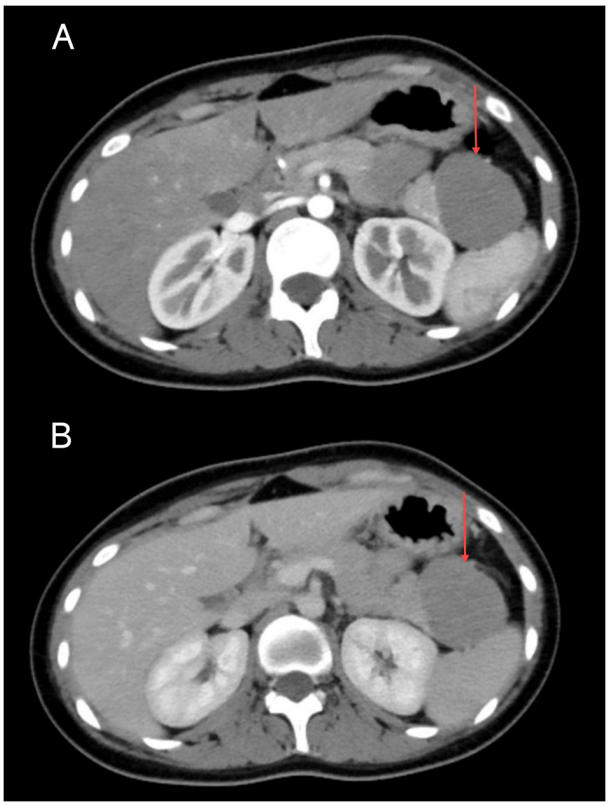

Figure 2. Radiological findings. (A) Enhanced CT showing low enhancement of the lesion in the arterial phase (arrow). (B) Enhanced CT showing low enhancement of the lesion in the venous phase (arrow). CT, computed tomography.

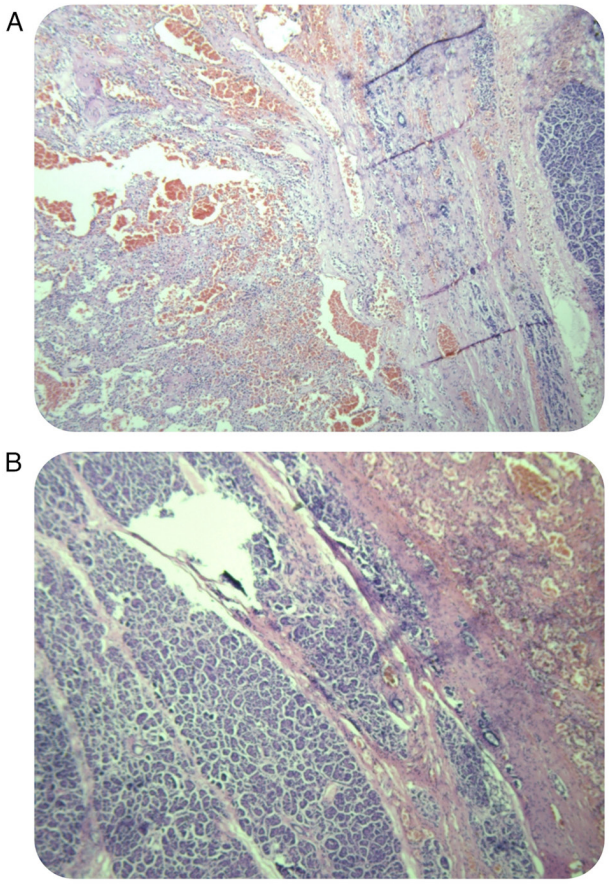

Figure 3. Representative images of pathological findings. (A) The cellular morphology presents a relatively uniform pattern, characterized by oval to elongated nuclei and distinct nuclear grooves (H&E staining; magnification, x100). (B) The tumor cell nuclei within a mucinous background exhibit a relatively uniform morphology, display reduced intercellular cohesion and are arranged in papillary patterns around small blood vessels (H&E staining; magnification, x100).

including the Notch and Hedgehog signaling pathways, in the pathogenesis of SPN (14,15). A study conducted by Law *et al* (16) enrolled a total of 2,744 patients diagnosed with SPN. The findings indicated that the mean age of the patient cohort was 28.5 years, with females comprising as high as 87.8% of the study population. Although SPN can arise in any region of the pancreas, it most frequently occurs in the pancreatic body and tail. Yu *et al* (17) reported a mean tumor diameter of 7.87 cm, with 54.8% of cases located in the body and tail of the pancreas, while Song *et al* (18) observed a mean tumor size of 6.4 cm, with 60.4% of tumors arising in the same anatomical region.

SPN lacks specific clinical manifestations. Approximately one-third of patients are asymptomatic and may be identified incidentally during routine physical examinations (6). The most frequently observed clinical presentations include abdominal pain and discomfort, while other common symptoms comprise nausea, vomiting, back pain and the detection of palpable abdominal masses. Laboratory findings in patients with SPN typically fall within normal ranges and the majority of tumor markers yield negative results (19,20). Although tumor markers demonstrate limited diagnostic specificity for SPN, they serve a valuable role in differential diagnosis, particularly in distinguishing SPN from malignancies such as pancreatic cancer.

Given the lack of specificity in laboratory tests for SPN, imaging modalities such as CT, MRI and ultrasound play a crucial role in its diagnosis (11). With continuous advancements in imaging technologies, the accuracy of the preoperative diagnosis of SPN has significantly improved. CT remains the most widely utilized imaging method for the preoperative evaluation of SPN, typically revealing a well-defined, heterogeneous cystic-solid mass that may be associated with hemorrhage or calcification (6,21). A limitation of the present study is the absence of MRI evaluation. Compared to CT, MRI offers superior soft-tissue contrast, enabling more precise visualization of the tumor's relationship with adjacent bile ducts and pancreatic ducts, which is of considerable importance for surgical planning (22). Pancreatic duct dilation is uncommon in SPN (17). Endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) serves as a valuable technique for obtaining histological confirmation prior to surgery. Studies have demonstrated that EUS-FNA achieves a preoperative diagnostic accuracy exceeding 80% for SPN (23). Nevertheless, due to its invasive nature and potential complications, including bleeding, infection and tumor seeding along the needle tract, its clinical application remains limited. A nationwide study based on Japanese SPN cases indicated that fluorodeoxyglucose positron emission tomography may provide additional diagnostic value for small-diameter tumors (<2 cm) (24). SPN can be differentiated from pancreatic cancer, pancreatic

cystadenoma, intraductal papillary mucinous neoplasm (IPMN), and pancreatic pseudocyst based on patient age at onset and CT imaging findings. Pancreatic cancer typically presents as a hypodense lesion during the contrast-enhanced phase of CT imaging, with poorly defined margins relative to adjacent normal pancreatic parenchyma, and is commonly associated with hepatic and lymph node metastases. Mucinous cystadenoma is characterized by multilocular cystic structures with low attenuation, frequently accompanied by eggshell-like peripheral calcifications, which constitute a hallmark radiological feature. Serous cystadenoma typically appears as unilocular or multilocular cystic lesions with fluid-equivalent density on CT scans, featuring a pathognomonic central stellate scar and calcification. IPMN of the pancreas manifests as a low-attenuation mass, often associated with pancreatic ductal dilatation. Pancreatic pseudocysts are usually depicted as well-circumscribed, homogeneously attenuated, round or ovoid lesions on CT imaging, with enhancement limited to the cyst wall during the contrast-enhanced phase (25).

Surgical resection remains the primary and preferred treatment modality for SPN (26). The selection of the surgical approach should be based on tumor size, anatomical location and intraoperative rapid pathological evaluation of frozen sections. Several studies have indicated that surgical resection should still be considered even in cases with preoperative evidence of local organ invasion or distant metastasis (27-29). Emerging evidence suggests that gemcitabine-based chemotherapy regimens may offer a clinical benefit in patients with metastatic or unresectable SPN (30,31). The patient of the present study underwent radiotherapy or chemotherapy. Considering the limited evidence regarding the application of radiotherapy or chemotherapy in the treatment of SPN, which is primarily derived from case reports, the effectiveness of these therapeutic modalities in managing SPN remains to be fully elucidated and corroborated. The definitive diagnosis of SPN primarily depends on histopathological and immunohistochemical analyses. The characteristic pathological features of SPN include the presence of solid, pseudopapillary and cystic components, with tumor cells arranged around fibrovascular cores in a pseudopapillary configuration. Currently, no specific immunophenotypic profile is unique to SPN; however, several markers demonstrate consistent expression patterns. In most studies, high expression levels of β-catenin, progesterone receptor (PR), synaptophysin (Syn), α-1-antichymotrypsin (AACT), vimentin (Vim) and CD56 have been observed, whereas chromogranin A (CgA) typically shows low or negative expression (32,33). The overexpression of β-catenin aligns with the molecular mechanism involving mutations in the β-catenin gene and its role in SPN pathogenesis via the WNT signaling pathway (34). As an immunophenotype associated with sex hormones, the high expression of PR suggests a potential involvement of hormonal factors in the development of SPN (35,36). Wang *et al* (37) reported that loss of PR expression was significantly associated with poorer recurrence-free survival and disease-specific survival. Immunohistochemical profiling enables differentiation of SPN from pancreatic ductal adenocarcinoma and neuroendocrine tumors. Markers such as β-catenin, lymphoid enhancer-binding factor 1 (LEF-1) and transcription factor E3 (TFE3) have demonstrated utility in distinguishing SPN from these mimics (34,38). Several studies recommend the use of combined immunophenotypic panels to enhance diagnostic accuracy for SPN (38,39). Kim *et al* (39) demonstrated that a panel consisting of β-catenin, LEF-1 and TFE3 achieved a sensitivity of 100% and a specificity of 91.9% in differentiating SPN from pancreatic cancer and neuroendocrine tumors. Ki-67 serves as a proliferation marker reflecting tumor cell growth activity. Given that SPN is a low-grade malignant tumor with an indolent biological behavior, Ki-67 expression is generally low. Yang *et al* (40) indicated that a Ki-67 labeling index ≥4% was associated with adverse postoperative outcomes in patients with SPN.

Given the low-grade malignant potential of SPN and its generally favorable prognosis, definitive conclusions regarding risk factors for benign vs. malignant behavior, recurrence or metastasis remain elusive. Several studies have investigated the association between tumor size and malignant potential. Kang *et al* (41) reported that a tumor diameter >5 cm may indicate malignant transformation in SPN. By contrast, De Robertis *et al* (42) observed no significant association between tumor size and malignancy grade. Additionally, the presence of an incomplete tumor capsule and calcification has been linked to poorer clinical outcomes in patients with SPN (43,44).

Patients diagnosed with SPN generally demonstrate a favorable prognosis following surgical resection. A study by Liu *et al* (45) followed 243 patients with SPN for an average of almost four years and their five-year survival rate was even higher at 98.4%. Even if SPN recurred or spread after the first surgery, numerous individuals still survived for a long time if they had another operation.

In summary, SPN is a low-grade malignant tumor of the pancreas that predominantly affects young women between the ages of 20 and 30 years. It is most frequently located in the pancreatic body or tail. Due to its indolent clinical course, SPN often presents without specific symptoms and is commonly detected incidentally during routine physical examinations. When symptoms occur, they may include nonspecific abdominal discomfort such as pain or distension. Laboratory findings are typically unremarkable, with minimal abnormalities observed in routine blood tests. Preoperative diagnosis primarily depends on imaging modalities, particularly contrast-enhanced CT, while definitive diagnosis relies on histopathological evaluation combined with immunohistochemical profiling. Accumulating evidence suggests that an incomplete tumor capsule serves as an independent prognostic indicator for more aggressive biological behavior in SPN. Complete surgical resection remains the cornerstone of treatment, offering favorable long-term outcomes in the majority of cases. Notably, even in instances of preoperative metastasis or postoperative recurrence, aggressive surgical intervention can still yield prolonged survival. Postoperatively, regular follow-up is strongly recommended for all patients with SPN, particularly those with high-risk features, to enable early detection and timely management of potential recurrences or metastases.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

DJ conceived and designed the study. LZ analyzed and summarized the data and wrote the manuscript. KL, LZ and DJ collected the laboratory examination data and images of the case. LZ critically revised the manuscript. KL and LZ confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

This study was conducted in accordance with the principles expressed in the Declaration of Helsinki.

Patient consent for publication

The patient involved in the present study was subjected to standard clinical practice and provided written informed consent for the publication of medical data and images.

Competing interests

The authors declare that they have no competing interests.

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