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(RESEARCH ARTICLE)

Diagnosis of Solid Pseudo-Papillary Neoplasm of the Pancreas by Endoscopic Ultrasound-Guided Fine Needle Aspiration Cytology: Case Report and Brief Literature Review

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Abstract

Pancreatic solid pseudopapillary neoplasm (SPN), often called Frantz tumor, is a rare, low-grade malignant tumor with an excellent prognosis after surgical removal. We report the case of a 32-year-old woman who presented to her primary care physician with intermittent epigastric discomfort and early satiety for several months. CT of the abdomen with contrast revealed a 6.5 cm mass in the body and tail of the pancreas with a mixed solid and cystic appearance and a thin peripheral capsule. MRI demonstrated areas of hemorrhage and confirmed a fibrous pseudo-capsule. Immunocytochemistry (IHC) demonstrated characteristic pseudopapillary architecture, and molecular analysis confirmed a CTNNB1 mutation encoding beta-catenin, consistent with SPN of the pancreas. The patient underwent distal pancreatectomy with splenectomy,

SPN can be diagnostically challenging due to its vague clinical presentation and overlapping features with other solid and cystic tumors of pancreatic origin. This case highlights the importance of timely recognition and multidisciplinary surgical workup and management to achieve optimal care and a favorable prognosis.

Keywords: Pancreas; Solid Pseudopapillary Neoplasm; Low-Grade Malignant Tumor; Immunocytochemistry; Multidisciplinary; Ultrasound-Guided Fine-Needle Aspiration; CTNNB1 Mutation

1. Introduction

Pancreatic SPN is a rare low-grade malignant tumor, typically with an excellent prognosis after surgical excision. It comprises 2% of all pancreatic exocrine tumors. These tumors primarily affect young female patients ranging in their teens to their thirties. This neoplasm predominantly occurs in the body or tail of the pancreas in adults, whereas pediatric cases occur in the pancreatic head. [1]

SPNs of the pancreas are classified as low-malignant potential tumors and are unlikely to metastasize. Presentation varies and is often asymptomatic, leading to a delay in diagnosis. As this neoplasm advances, symptoms such as abdominal pain, bloating, palpable mass, and nausea arise from compression of adjacent tissues. [2] Diagnostic imaging usually reveals a well-encapsulated mass with cystic or solid morphology, calcifications, and hemorrhage. [3] IHC studies are typically positive for vimentin, CD10, and β-catenin. Prognosis is generally favorable with high survival rates. [1]

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In this report, we highlight the clinical presentation, differential diagnosis, pathological traits, and management of a young female patient with a pancreatic SPN.

2. Case Presentation

2.1. Clinical Presentation and Physical Examination

A 32-year-old woman with no significant past medical history presented to her primary care physician with a several-month history of vague, intermittent epigastric discomfort and early satiety. She denied nausea, vomiting, jaundice, or significant weight loss, though she noted occasional bloating after meals. There was no family history of pancreatic disease or hereditary cancer syndromes, and she was otherwise in excellent general health. Her symptoms had been gradually worsening, prompting her physician to pursue further evaluation.

On physical examination, she appeared well-nourished and in no acute distress. Vital signs were within normal limits. Abdominal examination revealed mild epigastric fullness without guarding or rigidity. A faint, smooth, non-tender mass was palpable in the upper abdomen on deep palpation. There was no hepatosplenomegaly, lymphadenopathy, or jaundice.

2.2. Initial Investigations, Radiographic Studies, and Differential Diagnosis

Initial evaluation was largely unremarkable. Complete blood count, comprehensive metabolic panel, and coagulation studies were all within normal limits. Tumor markers, including CA 19-9 and CEA, were not elevated. Serum lipase and amylase were normal. Given the palpable epigastric mass and her symptoms, cross-sectional imaging was promptly obtained. Contrast-enhanced computed tomography (CT) of the abdomen and pelvis revealed a well-circumscribed, heterogeneous, 6.5 cm mass in the body and tail of the pancreas with a mixed solid and cystic appearance and a thin peripheral capsule. There was no pancreatic ductal dilation, vascular invasion, or distant metastasis. Magnetic resonance imaging (MRI) confirmed the encapsulated nature of the mass, demonstrating areas of hemorrhage and a characteristic fibrous pseudo capsule, findings that were highly suggestive of a solid pseudopapillary tumor of the pancreas.

The differential diagnosis included SPN of the pancreas as the leading consideration, given the patient's age, sex, and imaging characteristics. Other entities included a mucinous cystic neoplasm, a non-functional pancreatic neuroendocrine tumor, and, less likely, a pancreatic acinar cell carcinoma. Serous cystadenoma was considered but felt less consistent with the solid-cystic morphology and degree of hemorrhage observed on imaging.

2.3. Multidisciplinary Tumor Board Discussion and Endoscopic Ultrasound-Guided Fine Needle Aspiration Cytology

The case was subsequently presented at the multidisciplinary tumor board, attended by surgical oncology, gastroenterology, diagnostic radiology, medical oncology, and pathology. The board's consensus favored proceeding with endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) to obtain tissue for cytopathologic and ancillary evaluation prior to definitive surgical planning. It was agreed that if cytology confirmed the suspected diagnosis, the patient would be an excellent candidate for a distal pancreatectomy with splenectomy, given the tumor's location.

EUS-FNA was performed without complication. Sufficient diagnostic material was obtained utilizing the Rapid On-Site Evaluation (ROSE), and a paraffin cell block was prepared to be used for IHC and molecular studies. The cytologic preparations were richly cellular and demonstrated characteristic findings. Loosely cohesive clusters and single cells were arranged around delicate fibrovascular cores, forming the hallmark pseudopapillary architecture. The tumor cells were monomorphic, oval to round, with fine nuclear chromatin, small, inconspicuous nucleoli, and pale, eosinophilic to clear cytoplasm. Intracytoplasmic hyaline globules and foamy macrophages were noted in the background. Nuclear grooves, another well-recognized feature of this entity, were also identified. There was no significant atypia, necrosis, or mitotic activity. (Figure 1 A, B, C, D) The overall cytomorphology was consistent with SPN of the pancreas.

IHC studies performed on the cell block material provided critical confirmatory evidence. The tumor cells demonstrated strong, diffuse nuclear and cytoplasmic positivity for beta-catenin, reflecting the characteristic aberrant nuclear accumulation associated with CTNNB1 mutation. The cells were also positive for CD10, vimentin, CD56, and progesterone receptor, and showed loss of membranous E-cadherin expression. Synaptophysin showed focal positivity, while chromogranin was negative, helping to distinguish this tumor from a well-differentiated pancreatic neuroendocrine tumor. The tumor cells were also positive for vimentin and NSE. Ki-67 proliferative index was low, estimated at less than 3%. (Figure 2 A, B, C, D, E, F) Molecular analysis confirmed a somatic activating mutation in exon

3 of the CTNNB1 gene, encoding beta-catenin, which is identified in the vast majority of solid pseudopapillary tumors and represents a defining molecular hallmark of this entity.

2.4. Surgical Management, Outcome, and Follow-Up

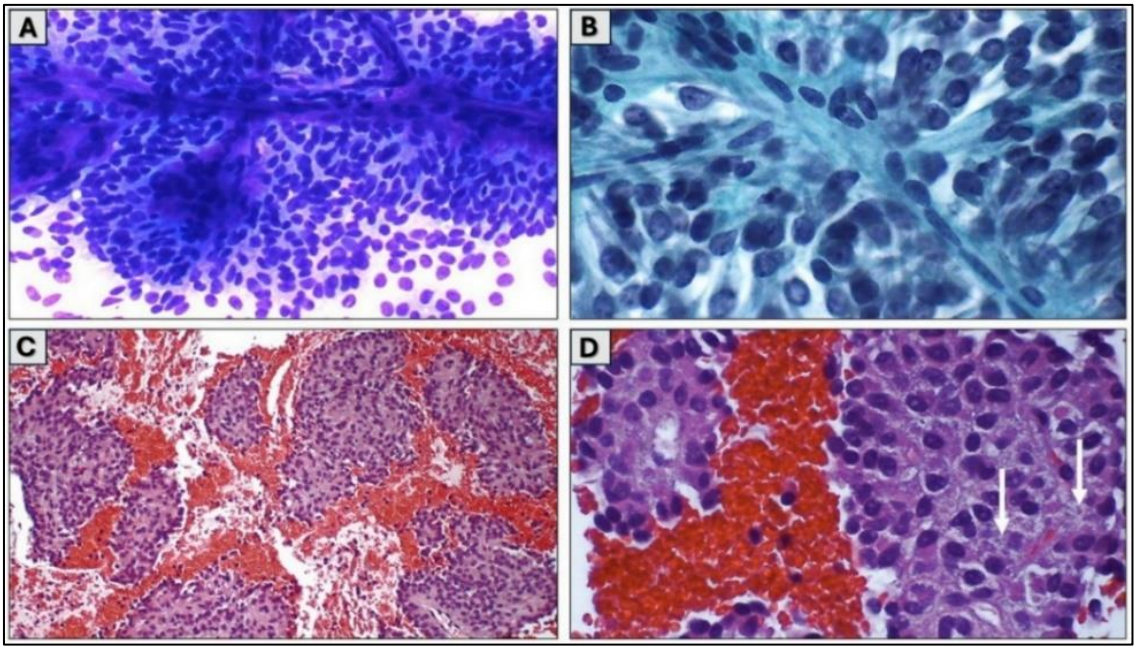

Figure 1 Cytomorphologic features of solid pseudo-papillary neoplasm (Cytology smears and cell block preparation)

1A: High power view showing loosely cohesive clusters and single cells arranged around delicate fibrovascular cores (Cytology smear, DQ stain X40).; 1B: High-power view showing monomorphic oval to round nuclei with fine chromatin, small inconspicuous nucleoli, and pale eosinophilic to clear cytoplasm (Cytology smear, Pap stain, ×60). ; 1C: Low power view showing clusters of tumor cells in a hemorrhagic background (Cell block, H&E stain X20); 1D: High power view showing the nuclear features of the neoplasm in a different stain (Cell block, H&E stain X40)

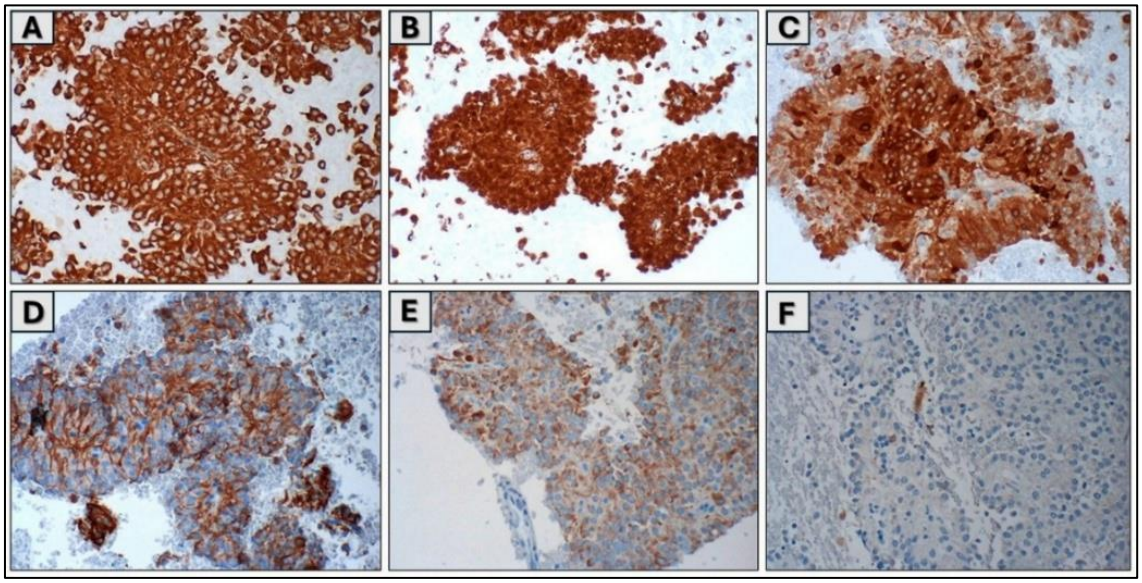

Figure 2 Immunohistochemistry results of solid pseudo-papillary neoplasm (Cell block preparation)

2A: Tumor cells positive for vimentin; 2B: Tumor cells positive for B-catenin; 2C: Tumor cells positive for NSE; 2D: Tumor cells positive for CD56; 2E: Tumor cells positive for CD10; 1F: Tumor cells negative for cytokeratin

The patient subsequently underwent elective distal pancreatectomy with splenectomy. The resection specimen confirmed a 6.8 cm encapsulated solid pseudopapillary tumor with focal areas of hemorrhage and cystic degeneration.

Surgical margins were negative, and all fifteen sampled regional lymph nodes were free of tumor. The final pathologic stage was pT2N0M0.

Her postoperative course was uneventful. On her six-month follow-up visit, she was doing well with no clinical or radiologic evidence of recurrence. The multidisciplinary team recommended annual surveillance imaging for at least 5 years, given the low but recognized potential for late recurrence, even in completely resected cases.

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

SPN of the pancreas was first described by Frantz in 1959 as a cavity lacking an epithelial lining that contains fluid. [4] SPNs are rare, accounting for approximately 2% of all exocrine pancreas neoplasms, 5% of cystic pancreas neoplasms, and primarily affect women with an average age range of 20 to 30 years. [5,6] No clear risk factors have been identified, it is widely accepted that the Wnt/Beta-catenin signaling pathway plays a critical, driving role in the tumorigenesis and pathogenesis of pancreatic SPN. [6]

SPNs exhibit different locational preferences by age. In adults, SPN most frequently arises in the tail or body of the pancreas, while in children and young adolescents, it is more commonly found in the head of the pancreas. [7] They are described as low-grade malignancies, with less than 10% of patients experiencing local recurrence. [7] One case study demonstrated that SPN with an incomplete capsule had exogenous growth patterns and were more likely to invade vasculature or organs. [8]

3.2. Pathogenesis, Pathophysiology

The origin of SPNs has been difficult to pinpoint, but some studies suggest it may be pancreatic embryonic stem cells that have undergone differentiation immaturely as the pancreas forms. [8] One study suggests that SPNs may originate from genital ridge cells that incorporate into the pancreas during embryogenesis, given their proximity during week 7 and similar IHC staining. [9] Given the uncertainty of origin, a clear molecular or genetic alteration has not yet been identified.

SPNs are nonfunctioning tumors that often cause symptoms due to mass effect, with the most common symptom being abdominal pain/discomfort, and approximately 40% presenting without any symptoms. [8] Studies have found that these tumors are diffusely positive for neuron-specific enolase (NSE), CD10, CD56, SYN, lu5, Cam5.2, nestin, vimentin, S-100, and alpha-1-antitrypsin [8,9]. This demonstrates that SPNs contain cells originating from various cell lines, suggesting an immature differentiation during embryogenesis. [8]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical, radiology, pathology, Lab, diagnosis, management, and outcome)

3.3.1. Clinical Presentation

SPN has a strong female predominance of over 85%, and its peak incidence occurs in women aged 20–40 years. In a recent 2024 systematic review of 1384 patients, the mean age was reported as 30 years; therefore, the 32-year-old patient is archetypal for SPN. [2] Clinical presentation includes nonspecific abdominal pain, discomfort, or early satiety lasting several months because of the slow growth of SPN tumors. The patient's complaints of epigastric pain and early satiety are similar to those in contemporary case series. [10] The average tumor size among 6651 patients in a pooled study in 2025 was 5.8 cm. [10] However, our patient's tumor size is larger (6.5 cm or 6.8 cm after pathology), but within the reported range.

The palpable abdominal mass, as seen in this case, is reported less often in contemporary studies due to earlier imaging detection, a significant finding that indicates changes in clinical approach. None of the atypical signs, like male gender, age above 50 years, obstructive jaundice, or aggressive symptomatology, were found. If these features are detected, they are associated with a high malignancy potential and a poor prognosis. [2,10]

3.3.2. Diagnostic Workup

Contrast-enhanced CT and MRI showed a well-circumscribed, encapsulated pancreatic lesion with mixed solid and cystic components and areas of hemorrhage. There was no pancreatic ductal dilatation, vascular invasion, or metastasis. These findings are highly characteristic of SPN and are consistently reported in recent imaging studies. [3,11] MRI, with

its superior soft-tissue resolution, better demonstrated the pseudocapsule and hemorrhagic areas, which helped distinguish SPN from other pancreatic neoplasms. [11]

EUS-FNA with ROSE is considered the evidence-based standard for preoperative tissue diagnosis. A 2023 study found that SPNs often yield radiographic false positives, whereas EUS-FNA with IHC enhances diagnostic precision, particularly for atypical or small tumors. [12] The patient's cytology results, IHC analysis, and molecular studies conducted on the cell block indicated aberrant Wnt/β-catenin signaling caused by a CTNNB1 mutation, a characteristic marker of SPN, a finding present in >90% of SPNs. [13]

Differential diagnoses considered mucinous cystic neoplasm, pancreatic neuroendocrine tumor (PanNET), and acinar cell carcinoma, which align with the differential diagnosis in the current literature. [11] Exclusion of PanNET was based on chromogranin negativity, with only focal synaptophysin positivity and positive β-catenin providing a strong basis for SPN diagnosis.

3.3.3. Management

Surgical resection is the optimal management strategy for SPN cases. In patients with tumors situated in the body/tail, distal pancreatectomy with or without splenectomy is the standard for treatment. Distal pancreatectomy with splenectomy can be considered based on the large tumor mass and location. According to a 2024 systematic review, 5-year survival was 98.1%, and the recurrence rate after complete resection was 2.8%. [2] Although sparing the spleen is becoming a popular management strategy for small cancers, it can be challenging and relatively unsafe, particularly if the tumor abuts the spleen vessels or is larger than 5 cm.

In selected cases with small SPNs (<2 cm), EUS-RFA (Radiofrequency ablation) has recently been described as an alternative minimally invasive treatment modality that yielded favorable short-term outcomes, including no recurrence at a median follow-up of 18.5 months. [10] However, the current tumor size (6.8 cm) necessitated surgery. One advantage in this case is the multidisciplinary tumor board consultation prior to EUS-FNA and surgery, which aligns with best practice by promoting diagnostic certainty and surgical planning.

3.3.4. Outcome and follow-up

The patient achieved R0 resection without nodal involvement (0/15) and an uneventful postoperative course, both of which are good prognostic indicators. Pooled data from 6651 patients revealed that 5-year survival was >95%, while 10-year disease-free survival was around 94%. Although recurrence is uncommon (2.8%; mean time to recurrence, 4.2 years) [2], it remains an important consideration. Reported risk factors for recurrence include tumor size greater than 5 cm, a Ki-67 index greater than 3%, and necrosis. [2] In our case, the large tumor size was offset by a very low Ki-67 index and absence of necrosis, suggesting a low overall risk of recurrence.

The planned 5-year surveillance imaging is consistent with current recommendations. However, the current follow-up period of 6 months is relatively short, as late recurrences have been reported up to 5 years after complete resection. Some authors now advocate surveillance for as long as 10 years, particularly for larger tumors. [14]

3.3.5. Literature Summary and Lessons Learned

The case is consistent with the relevant literature on demographics, imaging modalities, cytology, IHC, and surgical management. It offers distinctive contributions, including: (a) comprehensive diagnostics (CT scan, MRI, EUS-FNA with ROSE, IHC, and CTNNB1) [14]; (b) a multidisciplinary approach to decision making as a standard; and (c) recognition of decreased incidence of palpable lump as a sign of early detection and diagnosis. The only outlier is the short-term follow-up. In summary, this case represents a prototypical SPN while illustrating recent evidence-based diagnostics. Unlike recent literature, this case supports a favorable outcome of SPN following complete resection, while demonstrating a shift towards multidisciplinary management. Future follow-up would be required to rule out late recurrence.

3.3.6. What Have We Learned from This Case?

This case reinforces that SPN should be the leading diagnostic consideration in any young woman presenting with a well-circumscribed, mixed solid-cystic pancreatic mass, even in the absence of elevated tumor markers or systemic symptoms. It demonstrates that EUS-FNA, when performed with ROSE and supplemented by a cell block for immunocytochemistry and molecular analysis, can yield a definitive preoperative diagnosis.

The cytomorphologic findings, pseudopapillary architecture, monomorphic oval nuclei, nuclear grooves, and intracytoplasmic hyaline globules are highly characteristic and, when combined with the IHC profile (diffuse nuclear

beta-catenin positivity, CD10, vimentin, CD56, progesterone receptor, and loss of E-cadherin), leave little diagnostic ambiguity. Molecular confirmation of a CTNNB1 exon 3 mutation further solidifies the diagnosis.

The case also highlights the indispensable role of the multidisciplinary tumor board in integrating clinical, radiologic, cytopathologic, and molecular data to guide surgical planning and long-term surveillance.

Abbreviations

- Solid pseudopapillary neoplasm (SPN);
- Immunocytochemistry (IHC);
- Endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA);
- Rapid On-Site Evaluation (ROSE)

4. Conclusion

SPN of the pancreas is a rare but clinically important entity that disproportionately affects young women and carries a favorable prognosis when diagnosed and resected at an early stage. This case illustrates that EUS-FNA, augmented by ROSE, IHC, and CTNNB1 molecular analysis, provides a reliable and minimally invasive means of establishing a definitive preoperative diagnosis. The characteristic cytomorphologic features, combined with a confirmatory IHC panel and molecular profiling, allow confident distinction from other pancreatic neoplasms, particularly pancreatic neuroendocrine tumors, and support well-informed surgical decision-making.

Our patient underwent successful distal pancreatectomy with splenectomy, achieving a pT2N0M0 resection with negative margins, and remained disease-free at six-month follow-up. Given the recognized potential for late recurrence, continued annual surveillance imaging is warranted. Awareness of this entity among clinicians and pathologists is essential to ensure timely diagnosis, appropriate surgical management, and structured long-term follow-up.

Compliance with ethical standards

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Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

Informed consent for the publication of this case was obtained from the patient.

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