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Pancreatic solid pseudopapillary neoplasm: Case report of an uncommon tumor and a brief review of the literature

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Abstract

Solid pseudopapillary neoplasm (SPN), also called Frantz tumor, is a low-grade pancreatic neoplasm with low malignant potential and is mostly found in young women. It may be a diagnostic challenge because it is rare and often presents with nonspecific symptoms.

A 22-year-old woman reported progressive pain in the left upper quadrant of her abdomen, early satiety, and slight weight loss in the last three weeks. The physical examination revealed a tender, palpable mass. Abdominal CT and MRI showed that a big, 8.5 cm, well-encapsulated, heterogeneous solid and cystic mass was present in the tail of the pancreas, which is typical of an SPN with hemorrhagic and necrotic components. This patient had a splenectomy and a distal pancreatectomy. The diagnosis was confirmed using histopathology, which revealed a neoplasm with solid and pseudopapillary structures. Immunohistochemistry was positive for β-catenin and CD10. The surgical margins were negative, and the Ki-67 index was low (2%). The patient managed to recover without issues and is still in the disease-free period 16 months after surgery.

The case is a typical example of SPN presentation, emphasizing the necessity of considering typical clinical and radiological features to diagnose it properly and perform a timely surgery. Most cases can be treated by performing an entire surgical resection, which provides a good long-term prognosis.

Keywords: Solid Pseudopapillary Neoplasm; Low Malignant Potential; Beta-Catenin; CTNNB1 Gene

1. Introduction

Of the uncommon pancreatic tumors, the solid pseudopapillary neoplasm (SPN) of the pancreas is a low-grade epithelial tumor that represents 0.9–2.7% of all pancreatic exocrine neoplasms [1]. The tumor shows a strong preference for female patients who develop SPN ten times more frequently than male patients, and most commonly affects women during their reproductive years. [2] The age distribution of male patients shows a single peak during their fifth decade, whereas female patients have bimodal peaks at ages 20-30 and 40-50. [2] [3]

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The slow growth of SPN tumors leads to silent clinical development until the tumor reaches a substantial size, which causes patients to experience nonspecific symptoms, including abdominal pain and early satiety, and detectable masses. [4] SPN most commonly develops in the body and tail sections of the pancreas, and rarely appears in the head of the pancreas, which can lead to obstructive complications. [3] The diagnostic value of serum tumor markers, including CEA and CA 19-9, is limited because these markers stay within normal ranges. [4] [5]

The diagnosis of SPN requires histopathological examination because imaging results do not provide specific diagnostic features. [6] The microscopic appearance of SPN typically displays poorly cohesive polygonal cells that create solid and pseudopapillary patterns and show β-catenin nuclear/cytoplasmic staining, and express CD10, vimentin, and progesterone receptor. [5] The CTNNB1 gene mutation leads to activation of the Wnt/β-catenin pathway, making SPN a unique molecular type of pancreatic tumor. [7]

The presence of atypical histological features in 10-15% of cases suggests potential recurrence or metastasis through vascular or perineural invasion, lymph node involvement, and elevated Ki-67 labeling. [8] Despite this, the prognosis for patients who receive complete surgical treatment remains excellent, even though some patients develop metastatic or recurrent disease. [1]

In this report, we describe a 22-year-old female patient exhibiting distinct imaging, histological, and IHC characteristics of SPN who underwent a distal pancreatic resection, splenectomy and experienced an uneventful recovery. This case adds to the existing literature and reinforces the favorable prognosis associated with timely surgical intervention.

2. Case presentation

A 22-year-old healthy female college student without prior history of abdominal complaints came to the emergency department with a three-week history of left upper quadrant abdominal pain that had been progressive over the past three weeks. The pain was characterized as dull, persistent, and spreading to her left shoulder, initially mild but gradually increasing in intensity. She complained of early satiety and a 5-pound unintended weight loss in the last month. The patient denied nausea, vomiting, fever, jaundice, and bowel habit disturbance. She did not have any significant medical history, no medical drugs were used, and no tobacco or alcohol use.

The patient presented with a good physical appearance despite being in minor distress because of abdominal discomfort. The vital signs were stable, with a normal temperature. On abdominal examination, mild left upper quadrant tenderness was present with a palpable, firm, 8 cm diameter mass below the left costal margin. To deep palpation, the mass was hard. No hepatosplenomegaly, lymphadenopathy, or peritoneal irritation was detected. The rest of the physical examination, including cardiopulmonary and neurological testing, was unremarkable. Laboratory preliminary testing revealed a normal complete blood count, an extensive metabolic panel, and liver function tests. There were normal levels of serum amylase and lipase. Tumor markers, including CA 19-9, CEA, and alpha-fetoprotein, were within normal limits.

A CT of the abdomen with contrast enhancement showed a well-circumscribed heterogeneous solid cystic 8.5 x 7.2 cm mass in the pancreatic tail. The lesion had peripheral enhancement and central low attenuation, likely due to hemorrhage and necrosis. No pancreatic ductal dilatation, vascularity, or lymphadenopathy was noted in the region. The spleen was observed to be compressed, but not affected. The CT was confirmed by MRI, which revealed a T1 hyperintense rim and heterogeneous internal signal intensity, consistent with a hemorrhagic cystic lesion. Caliber of the duct of the pancreas was unchanged along its path. Differential diagnosis at this stage was: Solid pseudopapillary neoplasm, pancreatic neuroendocrine tumor, mucinous cystic neoplasm, and pancreatic adenocarcinoma.

The case was discussed during the hepatobiliary multidisciplinary team meeting. Because of the young age of the patient, her female gender, and the typical appearance of the imaging of a well-encapsulated heterogeneous mass with hemorrhagic elements, a solid pseudopapillary neoplasm was deemed the most probable diagnosis. The group discussed the importance of preoperative tissue sampling or going directly to surgical resection. Although EUS-guided FNA may offer diagnostic confirmation, it was agreed that the imaging appearance was typical enough to be SPN, and the patient was an excellent surgical candidate.

According to the location and size of the tumor, the surgical team advised distal pancreatectomy with splenectomy. The surgical resection proceeded without a preoperative biopsy. The whole excision approach was done to provide a definite diagnosis as well as curative treatment of this probable benign yet locally aggressive neoplasm.

The surgical sample consisted of a distal pancreatectomy with the spleen. Sectioning showed an encapsulated, heterogeneous 8.5 x 7.0 x 6.5 cm mass in the tail of the pancreas. The cut sections showed variegated, solid, tan-pink areas intermixed with cystic spaces containing hemorrhagic and necrotic debris. Surgical margins were free of tumor. Microscopic examination revealed a cellular neoplasm consisting of homogenous cells organized in solid sheets and pseudopapillary structures surrounding thin-walled blood vessels. The tumor cells were homogeneous, polygonal to ovoid, with eosinophilic to clear cytoplasm and centrally located, round to oval, mostly grooved nuclei containing fine chromatin and without discernible nucleoli. The mitotic figures were rare (less than 1 per 10 high-power fields). Hemorrhage, necrosis, and cholesterol clefts were prominent. No lymph vascular or perineal invasion was detected. (Figure 1 A, B, C, D)

Immunohistochemistry (IHC) studies revealed that the tumor cells were positive for beta-catenin (in both nuclear and cytoplasmic patterns), CD10, vimentin, and cyclin D1. They had a negative result for chromogranin A, synaptophysin, trypsin, and cytokeratin's. The Ki-67 proliferation index was minimal at 2%. Molecular Investigations of the CTNNB1 gene sequencing revealed a point mutation in exon 3, corresponding to the aberrant Want activation features of solid pseudopapillary neoplasm, which confirmed the diagnosis. The final diagnosis rendered was a solid pseudopapillary neoplasm (SPN) of the pancreas, with no malignant transformation and negative margins.

SPN of the pancreas has an excellent prognosis after total surgical excision with a 95 percent plus cure rate of tumors that do not have signs of malignant transformation. This case had no lymph vascular invasion, negative margins, and a low Ki-67 index, indicating a low likelihood of recurrence. The patient recovered from the surgery uneventfully and was vaccinated against the usual post-splenectomy complications. Surveillance imaging using CT was planned at 6-month intervals during the first two years, followed by an annual interval. The patient was completely disease-free and totally asymptomatic at 16 months after surgery. The recent CT scan revealed that the post-surgical changes are normal without any local recurrence or metastatic disease.

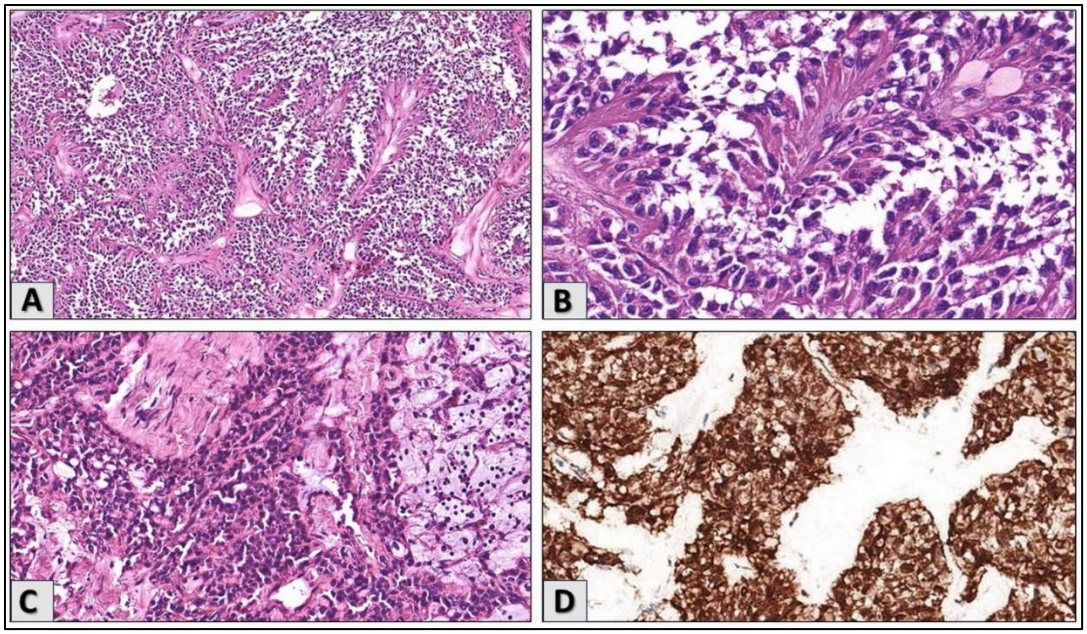

Figure 1 Microscopic features of an excised pancreatic solid pseudopapillary neoplasm

- **1A:** Low power view showing cellular neoplasm consisting of homogenous cells organized in solid sheets and pseudopapillary structures surrounding thin-walled blood vessels (H&E stain X20)
- **1B:** High power view showing homogeneous, polygonal to ovoid, tumor cells with eosinophilic to clear cytoplasm and centrally located, round to oval nuclei containing fine chromatin and without discernible nucleoli (H&E stain X60)
- **1C:** High power view showing clusters of histiocytic aggregates (Right side) within the tumor cells (H&E stain X40)
- **1D:** Tumor cells positive for beta-catenin (nuclear and cytoplasmic patterns)

3. Discussion

3.1. History, epidemiology, risk factors, and WHO classification

Frantz tumor or SPN of the pancreas is an uncommon and enigmatic tumor of the pancreas. It was referred to as a papillary pancreatic tumor in 1959 by Virginia Kneeland Frantz as either benign or malignant. [9] [10] The rarity and distinct appearance when microscopically viewed led to a variety of different names over many years, including solid and papillary epithelial neoplasm, papillary cystic neoplasm, and solid-cystic tumor. [7] [11] It was not officially called a solid pseudopapillary neoplasm of the exocrine pancreas by the World Health Organization (WHO) until 1996. In the 2019 WHO Classification of Tumors of the Digestive System, SPN was grouped with low-grade malignant epithelial neoplasms and recognized as having the potential to be an aggressive disease, with local invasion, recurrence, and distant metastasis, although these are rare. [12]

SPNs are rare, accounting for 1-2% of all exocrine pancreatic neoplasms. However, they contribute significantly to pancreatic tumors in young women, who are the most affected group. The tumor is highly female-specific, with a female-to-male ratio of 10:1. [1] [13] The mean age of diagnosis is typically between the second and third decades of life, as portrayed with our patient, with no racial or geographic inclination. Most of the tumors are found incidentally, on imaging for non-relevant reasons, but some patients may present with a mass or pain, as was the case in our patient. In adults, SPNs are more in the body and tail of the pancreas, and in children, they are more centered on the head of the pancreas. [4] The prognosis after resection is excellent, with a 5-year survival rate of 95-97%. However, 10-15% of patients will ultimately develop metastases, most commonly to the liver or lymph nodes. [3] [11] Risk factors for aggressive behavior are male sex, large tumor size, vascular or perineural invasion, and high Ki-67 index. [10] [14]

The etiology and risk of SPN are not well known. Unlike the other pancreatic neoplasms, smoking, alcohol consumption, or hereditary tendency towards pancreatic cancer have not been associated with the condition. [15] The overwhelming female predisposition has raised a hypothesis on the role of hormones in the pathogenesis of the disease; some studies have suggested that there may be a correlation between the use of oral contraceptives or pregnancy; however, this has not yet been proven. [11] [15]

3.2. Pathogenesis and pathophysiology

SPN has an unclear pathogenesis that is under active research, yet one of its key and main characteristics is the deregulation of the Wnt/b-catenin signal cascade. The pathway plays a crucial role in embryonic development and tissue homeostasis, and its inappropriate activation is linked to numerous cancers. A somatic point mutation in exon 3 of the CTNNB1 gene is detected in more than 90% of SPNs. [4] [17] This mutation can prevent the degradation and phosphorylation of the β-catenin protein. As a result, β-catenin is delivered to the nucleus and accumulates in the cytoplasm, where it creates a complex with transcription factors to activate target genes, including CCND1 (cyclin D1) and MYC. This leads to cell overgrowth and the formation of tumors. β-catenin in the nucleus and cytoplasm, as seen in our case, is the pathognomonic IHC evidence of SPN. [5] The histogenesis of SPN, or its cell of origin, remains a mystery. The IHC profile of the tumor cells is distinctive, with mesenchymal (vimentin) and epithelial (CD10) markers, as well as neuroendocrine markers, being co-expressed to different extents. This feature gives rise to various theories. [5] [7] These have been proposed to be based upon pluripotent embryonic cells of the pancreatic primordium, ductal epithelial cells, or even cells of the genital ridge, which were included in the pancreas at embryonic stages. [15] The tumor's strong predominance among females, and some cases having progesterone receptors, support the latter theory. [15] Nonetheless, none of the theories has been clearly established, and the exact cell of origin of SPN remains a contentious, debatable issue in the literature. [3]

3.3. Comparative analysis of our case with the existing literature

3.3.1. Clinical presentation and radiology

The patient exhibited a clinical picture that is consistent with the classical definition of SPN in the medical community. Most patients, including ours, are young women and in most cases display nonspecific abdominal symptoms. The most prevalent is imprecise pain or discomfort in the abdomen, usually involving the mass effect of the enlarged tumor. [4] [6] In a considerable percentage of cases, a palpable abdominal mass exists, as was the case with our patient. Early satiety and weight loss, which our patient presented, are also common descriptions associated with the size of the tumor and pressure on neighboring organs. [7] Notably, most SPNs are incidentally discovered during imaging performed for other reasons, as they are slow-growing. Our case also lacks jaundice and normal pancreatic enzyme levels (amylase and lipase), which are characteristic of SPNs, as they do not typically lead to ductal obstruction or parenchymal inflammation of the pancreas, unlike ductal adenocarcinomas. Tumor markers, including CA 19-9 and CEA, are usually

within the normal range, a finding similar to ours, and one of the crucial characteristics that distinguishes SPN from other pancreatic malignancies. [1] [4]

The radiological features in our case are pathognomonic of SPN. The SPNs in contrast-enhanced CT and MRI typically have a characteristic of a huge mass well encapsulated by a combination of solid and cystic formations. [6] The appearance is typical because internal hemorrhage and necrosis are commonly present, resulting in the heterogeneous solid cystic appearance seen in our patient's images. A fibrous capsule can frequently cause peripheral enhancement on imaging, as seen in our case. The site is also the pancreatic tail, but SPNs may be found at any site in the pancreas. [6] [11] The association of a young female patient with a massive, encapsulated, solid, cystic pancreatic mass of hemorrhagic debris is highly suggestive that even a confident preoperative diagnosis of SPN can often be reached by imaging alone, as our multidisciplinary team determined.

3.3.2. Laboratory, Pathological, and Molecular Findings

Our patient underwent the typical laboratory tests that led to the ultimate molecular confirmation of the diagnosis of SPN. The baseline laboratory results, including normal serum

The histopathological findings in our case are characteristic of SPN. The typical microscopic amylase and lipase levels, as well as tumor markers (CA 19-9 and CEA), are all in line with known information. [1] [4] Histological appearances featured the presence of solid sheets of homogeneous cells, mixed with pseudopapillary structures surrounding delicate blood vessels. Even the cellular features, which are monotonous, polygonal, eosinophilic cytoplasmic cells with grooved nuclei, are textbook features. [7] [18] The most specific and diagnostically important IHC reaction is the strong nuclear and cytoplasmic β-catenin positivity, which is a direct result of the underlying genetic mutation. Vimentin and CD10 co-expression, along with neuroendocrine negativity (chromogranin, synaptophysin) and most cytokeratins, is the ideal IHC profile of SPN. [10] Lastly, an eventual point mutation in exon 3 of the CTNNB1 gene provided excellent molecular confirmation, firmly establishing the diagnosis and placing our case squarely within the scope of more than 90 percent of the SPNs known to possess this characteristic genetic change. [17]

3.3.3. Management and outcomes

Our patient's management plan, which involved proceeding directly to surgical excision without a mass biopsy beforehand, is a common practice strategy among suspected SPNs. [18] Although EUS-guided FNA can provide the diagnosis cytologically, the typical imaging features in a young woman are deemed to be satisfactory for SPN diagnosis, as was the case with our multidisciplinary meeting. Complete surgical resection is the main and conclusive procedure used in the treatment of SPN. [3] The surgery procedure varies based on tumor size and location; in our case, the most common surgery would be a distal pancreatectomy with or without splenectomy since the tumor is in the pancreatic tail. The need to conduct a splenectomy may also depend on whether the tumor has been involved or encircled by the splenic vessels. The most important issue is achieving negative surgical margins to ensure a good long-term outcome. [4]

Our patient's postoperative recovery was uneventful. The patient received appropriate vaccinations to protect against encapsulated organisms after splenectomy, as recommended by the CDC. Following total surgical resection, SPN has a very good prognosis, particularly if it does not exhibit malignant characteristics. Tumors without vascular or capsular invasion, a high mitotic index, a high Ki-69 proliferation factor, or perineural invasion have a very low recurrence rate (less than 5%). [18] The probability of recurrence was extremely low in our case due to the low Ki-67 index (2%), negative margins, and the absence of aggressive features. In accordance with recommendations for low-risk pancreatic neoplasms, surveillance was scheduled using contrast-enhanced CT scans every six months for the first two years, followed by annual scans thereafter. [19] The patient was still asymptomatic and free of disease 16 months after surgery. Her overall quality of life had remained excellent, as evidenced by the most recent CT scan, which revealed no evidence of metastasis or recurrence.

SPN typically demonstrates a favorable prognosis upon complete resection, with 5-year survival rates surpassing 95% in non-metastatic instances. [3] This case highlights the significance of precise imaging interpretation, collaborative planning with clinic-pathological correlation, and timely surgical intervention in effectively managing this uncommon tumor.

3.4. What have we learned from this case?

This case is an excellent and historical reminder of the importance of maintaining a broad spectrum of differential diagnosis for pancreatic masses, particularly in patients who do not fit the typical demographics. The possibility of

pancreatic malignancy is not a typical differential diagnosis in a young and healthy female with nonspecific pain in the abdomen. Nevertheless, this case indicates that although SPN is a rare phenomenon, it can be a primary differential diagnosis in this population. The imaging appearance of a big, encapsulated, and complicated solid-cystic mass must be immediately suspicious of SPN.

Moreover, this case supports the existing knowledge regarding clinical, radiological, and pathological correlations, which characterize SPN. The patient's case history, the typical radiological appearances of a distinct mass with hemorrhagic and necrotic foci, and the unequivocal histopathological evidence of this disease, including the pseudopapillary structure and nuclear β-catenin, are all textbook images of this disease. It emphasizes the effectiveness of a multidisciplinary team approach, in which the unity of clinical suspicion, radiological/pathological interpretation, and surgical effectiveness resulted in the best possible outcome without involving a potentially complex preoperative procedure such as a surgical biopsy. Lastly, as demonstrated without patient, when proper surgery is performed with a total resection of the rare pancreatic tumor, it can be diagnostic as well as curative, ultimately, leading to a good prognosis of SPN.

4. Conclusion

By sharing this case with the medical community, we hope that more awareness will be raised, and the classical characteristics of SPN will be reinforced, which is a rare pancreatic tumor with a distinct clinical and pathological appearance. Through the records of this textbook example, we hope that clinicians will appreciate SPN as one of the major differentials for pancreatic masses in young women, enabling timely and efficient management. As this case illustrates, although it is a rare occurrence, a high index of suspicion due to characteristic imaging can result in a definitive preoperative diagnosis and a curative surgical resection, providing an excellent prognosis. Reporting these cases is crucial for developing a comprehensive understanding of uncommon diseases, enabling patients presenting with unusual tumors to receive proper and timely attention, thereby avoiding misdiagnosis and unnecessary interventions.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

Consent for the publication of this case was obtained from the patient.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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