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CASE REPORT | PANCREAS

Synchronous Pancreatic Masses

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ABSTRACT

Any mass lesion in the pancreas usually raises the possibility of undiagnosed pancreatic cancer. With the advancement of imaging modalities, we are seeing an increasing number of incidental findings, some of which may be clinically significant. When dealing with incidental pancreatic findings, it is critical to keep a broad differential in mind in addition to ruling out pancreatic malignancy. We present 3 rare cases of patients with 2 or more synchronous solid masses in the pancreas caused by pancreatic cancer, type 1 autoimmune pancreatitis, and sarcoidosis.

KEYWORDS: pancreatic ductal adenocarcinoma; autoimmune pancreatitis; pancreatic sarcoidosis; synchronous pancreatic masses

INTRODUCTION

Any mass lesion in the pancreas usually raises the possibility of undiagnosed pancreatic cancer (PC). With the advancement of imaging modalities, incidental findings are increasing. There have only been a few reports of synchronous pancreatic masses.[1–5] We present 3 rare cases of synchronous solid pancreatic masses caused by PC, type 1 autoimmune pancreatitis (AIP), and sarcoidosis. Depending on the pathology, each patient's clinical course varied greatly. This series highlights the plurality of solid pancreatic masses and comparable imaging characteristics with PC.[6]

CASE REPORTS

Case 1: A 65-year-old woman presented with abdominal pain and an unintentional weight loss of 20 pounds over 4 months. Her medical history included coronary artery bypass, congestive heart failure, atrial fibrillation, and pulmonary hypertension. She drank

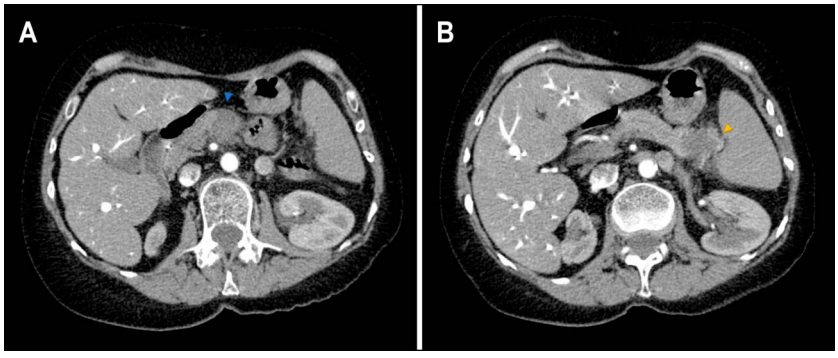

Figure 1. Abdominal contrast-enhanced computed tomography demonstrating 2 solid masses in the body (blue arrow, panel A) and tail of the pancreas (yellow arrow, panel B).

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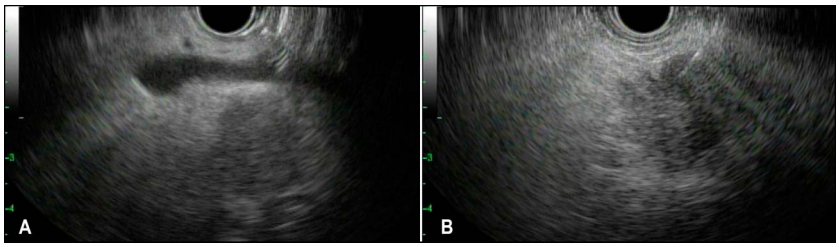

Figure 2. Endoscopic ultrasound demonstrating the 2 irregular mass-like processes, hypoechoic in the pancreatic body (panel A) and heterogenous in the pancreatic tail (panel B).

alcohol occasionally but denied smoking or previous pancreatitis. The abdominal computed tomography (CT) scan demonstrated 2 hypodense masses in the body (2 cm) and tail (2.5 cm) of the pancreas with peripancreatic lymphadenopathy but no vascular involvement. Main pancreatic duct (MPD) was normal (Figure 1). The carbohydrate antigen 19-9 level was elevated at 75 U/mL (normal: 0–37 U/mL). The immunoglobulin G4 (IgG4) level was normal. Endoscopic ultrasound (EUS) confirmed the 2 irregular mass-like processes, hypoechoic in the pancreatic body (2 cm) and heterogenous in the pancreatic tail (3 cm) (Figure 2). EUS-guided fine needle biopsy (FNB) of both pancreatic masses was performed separately using a transgastric approach and confirmed infiltrating pancreatic ductal adenocarcinoma (PDAC). Owing to the significant comorbidities, she was deemed unfit for surgery and underwent chemoradiation and palliative care.

Case 2: A 76-year-old man presented with postprandial abdominal pain and unintentional weight loss. He had a history of coronary artery disease, uncontrolled diabetes, 80+ pack-years smoking history, and alcohol use disorder. He had elevated liver enzymes, alanine transaminase: 136 U/L (normal: 10–54 U/L); aspartate transaminase: 79 U/L (normal: 14–40 U/L); total bilirubin: 1.8 mg/dL (normal: 0.2–1.3 mg/dL); and alkaline phosphatase: 489 U/L (normal: 38–113 U/L). Adbominal CT revealed infiltrative changes throughout the pancreas, primarily along the pancreatic body and tail. No MPD dilatation was observed (Figure 3). EUS revealed several hypoechoic heterogenous mass-like lesions in the head and tail of the pancreas (Figure 4), and FNB showed that areas of fibrosis and edema with lymphoplasmacytic infiltration on histopathology and immunohistochemistry demonstrated IgG4+ plasma cells. He was diagnosed with type 1 AIP and started on prednisone 40 mg daily for 4 weeks. This was gradually tapered down to a maintenance dose of 5 mg every other day over the following year. He was successfully weaned off of prednisone after 2 years and did not experience recurrence.

Case 3: A 54-year-old man with a medical history of sarcoidosis with pulmonary and extrapulmonary involvement underwent a positron emission tomography scan for sarcoidosis surveillance. It demonstrated focal increased uptake in the head/tail of the pancreas. The CT scan did not show any mass or MPD dilation. EUS demonstrated several heterogenous, infiltrative masses with ill-defined borders involving the pancreatic head and the tail (Figure 5). FNB was performed, and histopathology demonstrated nonnecrotizing granulomas (Figure 6). After excluding infectious granulomatous diseases, these findings were attributed to pancreatic involvement of his sarcoidosis. Owing to side effects, he did not receive systemic therapy and was kept under clinical observation.

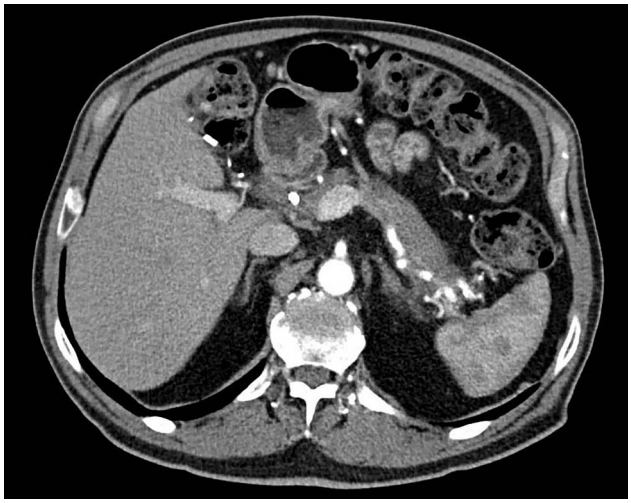

Figure 3. Abdominal contrast-enhanced computed tomography demonstrating infiltrative changes throughout the pancreas and the classic "sausage-shaped" appearance in autoimmune pancreatitis type 1.

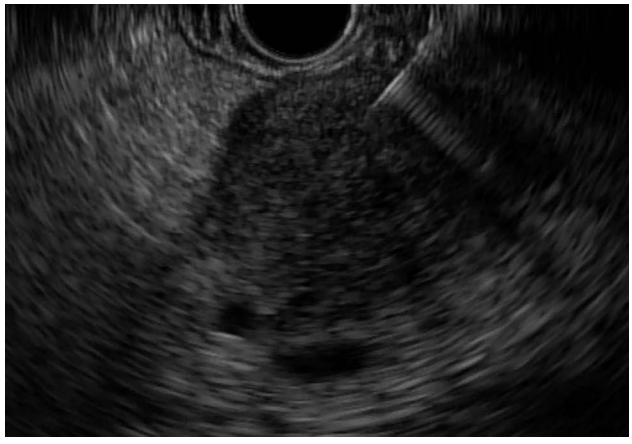

Figure 4. Endoscopic ultrasound with fine needle biopsy of one of the hypoechoic heterogenous mass-like lesions.

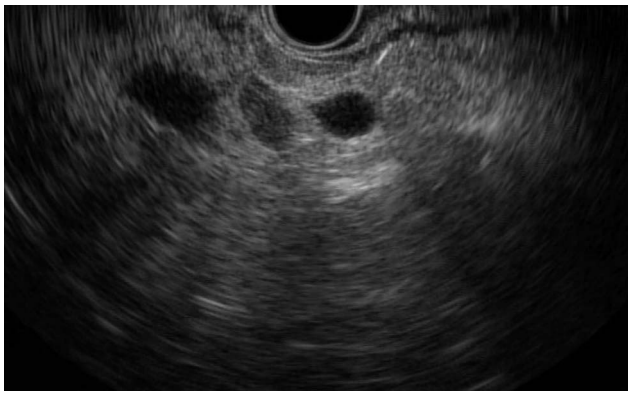

Figure 5. Endoscopic ultrasound demonstrating several heterogenous, infiltrative masses with ill-defined borders.

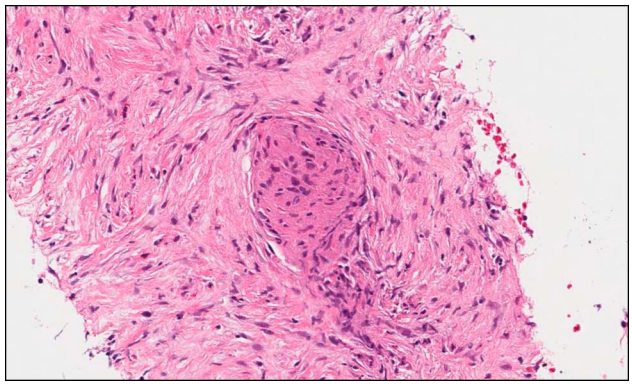

Figure 6. Hematoxylin and eosin, 200× magnification. High-power view of a sarcoidal granuloma.

DISCUSSION

We concentrated on 3 rare cases of synchronous solid masses in the pancreas caused by PC, type 1 AIP, and sarcoidosis. The clinical course of each patient differed greatly depending on the pathology (Table 1). Typically, synchronous tumors are diagnosed within 6 months of the index tumor.[7] A very few cases have been reported in the medical literature of synchronous pancreatic tumors.[1–5] Differential diagnosis for multiple synchronous pancreatic masses includes pancreatic neuroendocrine tumors in multiple endocrine neoplasia type 1, primary pancreatic lymphoma, AIP, and renal cell carcinoma.[8–12] In a Chinese study, 8,096 patients underwent CT scans over a period of 40 months; the etiology and prevalence of the multiple solid pancreatic masses were found to be benign (19.8%), most common being AIP (17.4%). Among malignant etiologies, pancreatic neuroendocrine tumors (51.2%), PDAC (12.4%), metastasis (7.4%), and lymphoma (5.8%) were reported.[13]

PC is the seventh leading cause of death in both men and women, with a 5-year survival rate of 5.0%.[14] Presentation includes weight loss, anorexia, abdominal pain, nausea, vomiting, jaundice, and Courvoisier sign.[15] Classic imaging findings include hypovascular mass with proximal MPD dilatation.[16] Our first patient presented with unintentional weight loss, abdominal pain, and fatigue with multiple pancreatic masses and was diagnosed with PDAC. This patient did not have surgery because of significant cardiac comorbidity and was referred for

Table 1. Summary of the reported cases

Case	Clinical presentation	Medical history	Pancreatic lesions	Pathology facilitated by EUS-FNB*	Management
Case 1 65-year-old woman	Abdominal pain, unintentional weight loss, fatigue ↑Ca19-9	Coronary artery bypass, heart failure, mitral and tricuspid regurgitation, atrial fibrillation, pulmonary hypertension, renal thrombosis	Two (pancreatic body and tail)	Ductal adenocarcinoma	Follow-up with hematology/oncology, radiation oncology, and palliative care
Case 2 76-year-old man	Postprandial gastric discomfort, unintentional weight loss	Coronary artery disease, uncontrolled diabetes, previous smoker (80+ pack-years), former alcoholic	Several (pancreatic head, and tail)	Areas of fibrosis and edema with lymphoplasmacytic infiltration and immunohistochemistry were positive for IgG4+ plasma cells consistent with type 1 autoimmune pancreatitis	Responded to steroids
Case 3 54-year-old man	Incidental findings on imaging	Complicated pulmonary and extrapulmonary sarcoidosis	Multiple	Noncaseating granulomas consistent with sarcoidosis	Not on any sarcoidosis medications due to side effects Periodic clinical monitoring

CA 19-9, carbohydrate antigen 19-9; EUS-FNB, endoscopic ultrasound-guided fine needle biopsy; IgG4, immunoglobulin G4.

palliative radiation chemotherapy. Fujimori et al[5] described a case with synchronous pancreatic masses that required total pancreatectomy which revealed 3 lesions with identical carcinoma cells that communicated with each other through the intraductal component, indicating a single large tumor.

AIP has been categorized into 2 types based on the International Consensus Diagnostic Criteria. Type 1 AIP can present with mild abdominal symptoms, acute pancreatitis, or obstructive jaundice, with histopathology consistent with IgG4 infiltration.[17] In our second patient, irregular heterogeneous masses were identified in the pancreatic head and tail on CT/EUS, immunohistochemistry was positive for IgG4+ plasma cells, and he was diagnosed with type 1 AIP. Shikowa et al reported an AIP case with several masses affecting the head/body of the pancreas that resolved after 1 month of systemic steroid treatment.[4]

Pancreatic sarcoidosis is usually the incidental finding found during autopsy.[18] It is sporadic and usually presents with symptoms similar to PC, including weight loss and obstructive jaundice.[19] However, our third patient was asymptomatic. He had imaging findings of pancreatic masses with a clinical history of complicated sarcoidosis involving pulmonary and extrapulmonary organs. Pancreatic sarcoidosis has a good overall prognosis, with cardiac and pulmonary disease being the most common causes of death.[20]

In conclusion, synchronous pancreatic masses are rare and a broad differential should be considered while encountering such patients. The entire pancreas should be evaluated with multimodal imaging along with EUS-guided acquisition histopathology to make a definitive diagnosis because the clinical course varies based on the disease.

DISCLOSURES

Author contributions: A. Chatterjee and N. Sharma drafted the article. M. Franklin acquired data and images. A. Singh and R. Garg acquired data. P. Chahal drafted and approved the final manuscript, and is the article guarantor.

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Informed consent was obtained for this case report.

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REFERENCES

1. Bittorf B, Kessler H, Merkel S, et al. Multiple primary malignancies: An epidemiological and pedigree analysis of 57 patients with at least three tumours. *Eur J Surg Oncol*. 2001;27(3):302–13.
2. Mehdi I, Shah AH, Moona MS, et al. Synchronous and metachronous malignant tumours expect the unexpected. *J Pak Med Assoc*. 2010;60(11):905–9.
3. Sastry A, Wayne M, Steele J, Iskandar M, Yuan S, Cooperman AM. Three synchronous, sporadic and separate periampullary and pancreatic tumors: More than a coincidence? *World J Surg Oncol*. 2014;12(1):382.
4. Shiokawa M, Kodama Y, Hiramatsu Y, et al. Autoimmune pancreatitis exhibiting multiple mass lesions. *Case Rep Gastroenterol*. 2011;5(3):528–33.
5. Fujimori N, Nakamura T, Oono T, et al. Adenocarcinoma involving the whole pancreas with multiple pancreatic masses. *Intern Med*. 2010;49(15):1527–32.
6. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2020. *CA Cancer J Clin*. 2020;70(1):7–30.
7. Chinn SB, Spector ME, Prince ME, Sun GH. Synchronous and metachronous tumors in head and neck cancer: Analysis of the SEER Database. *Otolaryngol Neck Surg*. 2013;149(2 Suppl):P198.
8. Kawakami H, Kuwatani M, Yamato H, et al. Pancreatic metastasis from renal cell carcinoma with intraportal tumor thrombus. *Intern Med*. 2008;47(22):1967–70.
9. Metz DC, Jensen RT. Gastrointestinal neuroendocrine tumors: Pancreatic endocrine tumors. *Gastroenterology*. 2008;135(5):1469–92.
10. Nakamura T, Ito T, Abe Y, et al. Primary pancreatic low-grade mucosa-associated lymphoid tissue lymphoma presenting with multiple masses. *Clin J Gastroenterol*. 2008;1(4):168–73.
11. Inoue D, Gabata T, Matsui O, Zen Y, Minato H. Autoimmune pancreatitis with multifocal mass lesions. *Radiat Med*. 2006;24(8):587–91.
12. Kajiwara M, Kojima M, Konishi M, et al. Autoimmune pancreatitis with multifocal lesions. *J Hepatobiliary Pancreat Surg*. 2008;15(4):449–52.
13. Zhu L, Dai Mh, Wang St, et al. Multiple solid pancreatic lesions: Prevalence and features of non-malignancies on dynamic enhanced CT. *Eur J Radiol*. 2018;105:8–14.
14. Ilic M, Ilic I. Epidemiology of pancreatic cancer. *World J Gastroenterol*. 2016;22(44):9694–705.
15. Porta M, Fabregat X, Malats N, et al. Exocrine pancreatic cancer: Symptoms at presentation and their relation to tumour site and stage. *Clin Transl Oncol*. 2005;7(5):189–97.
16. Gandhi NS, Feldman MK, Le O, Morris-Stiff G. Imaging mimics of pancreatic ductal adenocarcinoma.
17. Shimosegawa T, Chari ST, Frulloni L, et al. International consensus diagnostic criteria for autoimmune pancreatitis: Guidelines of the international association of pancreatology. *Pancreas*. 2011;40(3):352–8.
18. Garcia C, Kumar V, Sharma OP. Pancreatic sarcoidosis. *Sarcoidosis Vasc Diffus Lung Dis*. 1996;13(1):28–32.
19. Mayne AIW, Ahmad J, Loughrey M, Taylor MA. Sarcoidosis of the pancreas mimicking adenocarcinoma. *BMJ Case Rep*. 2013;2013(1):bcr2013009118.
20. Ebert EC, Kierson M, Hagspiel KD. Gastrointestinal and hepatic manifestations of sarcoidosis. *Am J Gastroenterol*. 2008;103(12):3184–92; quiz 3193.