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Case Report

The Reappearing Pancreas: A Case of Acute Pancreatitis on a Background of Diffuse Fatty Infiltration

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INTRODUCTION

Acute pancreatitis is a common cause of hospitalization in the United States, while fatty infiltration of the pancreas (FIP) is among the most frequent benign pancreatic conditions, affecting nearly one-third of the population.[1] The extent of FIP varies, with diffuse involvement of the entire gland occurring in only a minority of cases.[2] Total fatty replacement of the pancreas can be associated with exocrine insufficiency.[3-5]

This report presents a case of diffuse FIP with exocrine insufficiency complicated by acute pancreatitis, highlighting key imaging findings and their potential pitfalls. A broader discussion of the underlying pathophysiology of pancreatitis also is included, informed by the unique combination of clinical factors in this patient.

CASE REPORT

A 55-year-old woman presented to the emergency department with generalized body aches, fever, and acute worsening of abdominal pain accompanied by intractable nausea and vomiting. She also reported a recent history of intermittent abdominal pain, nausea, and loose stools. She had experienced multiple prior episodes of pancreatitis, most recently three weeks earlier during a hospitalization at an outside facility. Her past medical history was notable for obesity, celiac disease, adrenal insufficiency, and rheumatoid arthritis, and her surgical history included a cholecystectomy. She denied alcohol use.

On physical examination, she had abdominal tenderness. Her body mass index (BMI) was 31 kg/m^2. Initial laboratory studies revealed mild normocytic anemia (hemoglobin 11.0 g/dL), a low serum lipase level (5 U/L), hypoalbuminemia (2.9 g/dL), and hyponatremia (129 mmol/L). White blood cell count and levels of total bilirubin, calcium, and triglycerides were normal. During the hospitalization, her International Normalized Ratio (INR) and prothrombin time (PT) were found to be markedly elevated, 9.0 (reference 0.9-1.2) and 95 seconds (reference 9.9-14.2), respectively, suggesting vitamin K deficiency. A fecal pancreatic elastase-1 level was extremely low (<10 µg/g), indicating severe exocrine insufficiency.

Contrast-enhanced computed tomography (CT) of the abdomen and pelvis showed extensive edema and fat stranding throughout the pancreas and adjacent retroperitoneal fat (Figure 1), consistent with interstitial edematous pancreatitis. When compared with a CT scan from two months earlier (Figure 2), the pancreatic parenchyma had markedly changed. The earlier CT demonstrated extensive fatty infiltration of the entire pancreas with minimal residual normal parenchyma; the average density at the pancreatic body was -83 Hounsfield units (HU). More remote abdominal magnetic resonance imaging (MRI) from three years prior (Figure 3) also showed near-complete fatty infiltration, evidenced by pancreatic isointensity relative to retroperitoneal fat and loss of signal on fat-saturated sequences. Notably, a small region of fatty sparing within the anterior pancreatic body had been present at that time, appearing as an island of normal parenchyma. By the time of the current presentation, this area had become fully infiltrated by fat.

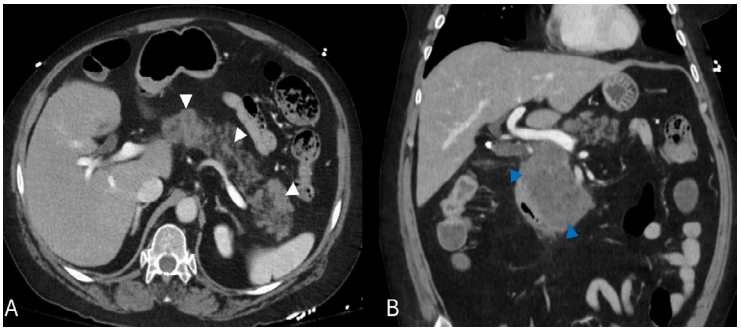

Figure 1. Computed tomography (CT) of the abdomen and pelvis with contrast. (A) Axial image demonstrates diffuse stranding and edema throughout the pancreatic body and tail (white arrowheads). (B) Coronal reformatted image shows stranding with mass-like thickening of the pancreatic head (blue arrowheads). Attenuation of the inflamed tissue appears similar to that of normal pancreatic parenchyma.

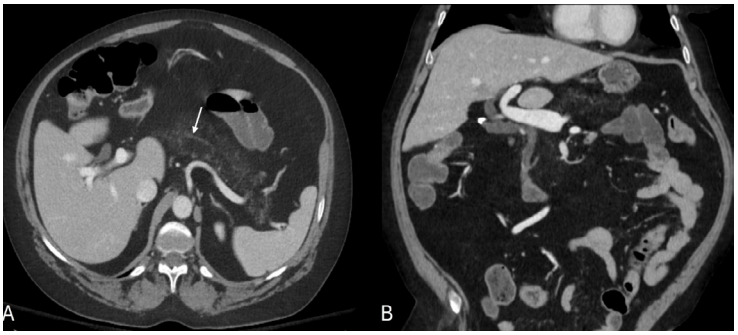

Figure 2. Computed tomography (CT) of the abdomen and pelvis with contrast obtained two months before Figure 1. (A) Axial and (B) coronal reformatted images demonstrate near-complete fatty replacement of the pancreas. The normal, non-dilated main duct is seen in the pancreatic body (white arrow).

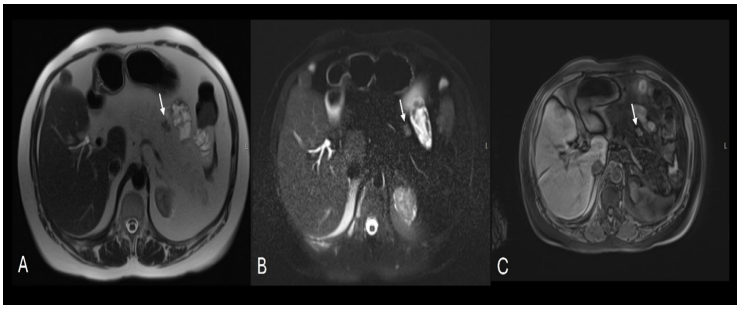

Figure 3. Magnetic resonance imaging (MRI) of the abdomen obtained three years before Figure 1. Axial (A) T2-weighted, (B) T2-weighted fat saturated, and (C) T1-weighted fat saturated pre-contrast images. (A) Macroscopic fat signal throughout the pancreas. (B, C) Diffusely hypointense pancreatic signal on fat saturated sequences. Small focus of tissue within the anterior pancreatic body (white arrow) with signal characteristics of normal parenchyma indicating an area of fatty sparing.

DISCUSSION

FIP is one of several terms used in the literature to describe the same process. Other synonyms include *fatty replacement, fatty pancreas, pancreatic lipomatosis, pancreatic steatosis, intrapancreatic fat deposition, and non-alcoholic fatty pancreas disease*.[3,6] The pathogenesis of FIP is not fully understood but is considered multifactorial. Age, obesity, diabetes, diet, and alcohol consumption are known associations, and FIP generally is viewed as a manifestation of metabolic syndrome. Rare hereditary pediatric disorders, such as cystic fibrosis, Shwachman-Diamond syndrome, and Johanson-Blizzard syndrome, also can cause FIP and are invariably accompanied by pancreatic exocrine insufficiency.[6,7]

Histologically, FIP is characterized by adipocyte infiltration between pancreatic lobules as well as intracellular fat deposition within acinar and islet cells.[7] FIP typically is heterogeneous, with uneven fat deposition more common than the diffuse pattern seen in this case. Matsumoto et al.[2] developed a classification system for uneven fatty infiltration and found that the anterior pancreatic head is most frequently affected, while the posterior head and uncinate process often are spared.[2] Most patients are asymptomatic, with FIP discovered incidentally on imaging. However, marked fatty infiltration can lead to exocrine insufficiency, resulting in malabsorption of dietary fat and fat-soluble vitamins, with symptoms including chronic diarrhea, steatorrhea, and abdominal pain. Diabetes and low serum lipase levels also may be present.[3-6]

Only a few reports describe diffuse fatty replacement complicated by acute pancreatitis. In Coulier's pictorial review, two such cases were included.[6] In this patient, the diffusely edematous fatty pancreas demonstrated CT attenuation similar to normal parenchyma, giving the impression that the pancreas had "reappeared" (Figure 1). This highlights the importance of comparison imaging. Without prior studies showing extensive fatty replacement, the degree of inflammation could easily have been underestimated. Clinically, there were few specific indicators of pancreatitis, the lipase level was low, and the abdominal pain could have been attributed to her comorbidities. Thus, the temporal change in pancreatic appearance combined with peripancreatic inflammatory stranding was important for accurate diagnosis.

Two recent studies by Sbeit et al.[8,9] found that FIP is associated with both increased occurrence and greater severity of acute pancreatitis. Proposed mechanisms include persistent tissue stress and low-grade inflammation within the fatty pancreas, punctuated by episodic inflammatory surges that precipitate acute pancreatitis.[3,8]

In general, acute pancreatitis results from autodigestion of the pancreas following inappropriate activation of digestive enzymes. Under normal conditions, enzymes are stored as inactive proenzymes in acinar cells, with activation occurring only in the duodenum. These safeguards prevent autodigestion. When they fail, a cascade of intrapancreatic enzyme activation leads to acinar injury, ischemia, fat necrosis, proteolysis, and inflammatory edema.[10]

This patient's extremely low fecal elastase-1 level is specific for exocrine pancreatic insufficiency,[11] and the low serum lipase level further supports this diagnosis. Additional laboratory abnormalities, including anemia, hypoalbuminemia, and vitamin K deficiency (elevated PT/INR), indicated generalized malabsorption. Her malabsorption likely is due to both celiac disease and exocrine insufficiency, though their relative contributions cannot be distinguished.

Some evidence suggests that FIP may be partially reversible through lifestyle modification and pharmacotherapy.[3,7,12] However, given this patient's degree of exocrine insufficiency, she likely has passed the threshold of reversibility, with substantial loss of acinar cells responsible for proenzyme production. In such cases, pancreatitis may be driven predominantly by non-autodigestion mechanisms. Local immune factors may provoke a leukocyte infiltrate that drives inflammation. Alternatively, as suggested by Caldart et al.,[12] intracellular fat within residual acinar cells may impair trafficking and exocytosis of proenzymes, leading to inappropriate intrapancreatic activation. The abundant interlobular adipocytes may then fuel a lipase-mediated cascade of rapid, diffuse fat necrosis, essentially allowing fat to amplify the pancreatitis.

Conclusions

This case illustrates how a common but often overlooked condition, fatty infiltration of the pancreas, can lead to serious clinical consequences. While FIP frequently is asymptomatic and incidentally detected, some patients, such as the one described here, may develop acute pancreatitis and profound exocrine insufficiency with resulting malabsorption. This report highlights the pathophysiologic links between FIP, pancreatitis, and exocrine dysfunction, and emphasizes the important role of comparative imaging in evaluating these dynamic processes. As clinical recognition and scientific understanding of FIP continue to expand, this case offers a clear example of its relevance to patient care.

ARTICLE INFORMATION

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REFERENCES

1. Singh RG, Yoon HD, Wu LM, Lu J, Plank LD, Petrov MS. Ectopic fat accumulation in the pancreas and its clinical relevance: A systematic review, meta-analysis, and meta-regression. Metabolism. 2017 Apr;69:1-13. Epub 2016 Dec 29. PMID: 28285638.
2. Matsumoto S, Mori H, Miyake H, et al. Uneven fatty replacement of the pancreas: evaluation with CT. Radiology. 1995 Feb;194(2):453-8. PMID: 7824726.
3. Ye J, Wang JG, Liu RQ, Shi Q, Wang WX. Association between intra-pancreatic fat deposition and diseases of the exocrine pancreas: A narrative review. World J Gastroenterol. 2025 Jan 14;31(2):101180. PMID: 39811515.

4. Anand R, Narula MK, Chaudhary V, Agrawal R. Total pancreatic lipomatosis with malabsorption syndrome. Indian J Endocrinol Metab. 2011 Jan;15(1):51-3. PMID: 21584169.
5. Kumar R, Bhargava A, Jaiswal G. A case report on total pancreatic lipomatosis: An unusual entity. Int J Health Sci (Qassim). 2017 Sep-Oct;11(4):71-73. PMID: 29085272.
6. Coulier B. Pancreatic Lipomatosis: An Extensive Pictorial Review. J Belg Soc Radiol. 2016 Feb 23;100(1):39. PMID: 30151451.
7. Mahyoub MA, Elhoumed M, Maqul AH, et al. Fatty infiltration of the pancreas: a systematic concept analysis. Front Med (Lausanne). 2023 Sep 22;10:1227188. 1227188. PMID: 37809324.
8. Sbeit W, Khoury T. Fatty Pancreas Represents a Risk Factor for Acute Pancreatitis: A Pilot Study. Pancreas. 2021 Aug 1;50(7):990-993. PMID: 34629451.
9. Sbeit W, Abu Elheja F, Msheiil B, et al. Fatty pancreas was associated with a higher acute pancreatitis Systemic Inflammatory Response Syndrome score at hospital admission. Eur J Gastroenterol Hepatol. 2023 Sep 1;35(9):980-984. Epub 2023 Jul 3. PMID: 37395190.
10. Hruben R, Iacobuzio-Donahue C. The pancreas. In: Kumar V, Abbas AK, Aster JC, editors. Robbins and Cotran pathologic basis of disease. 9th ed. Philadelphia (PA): Elsevier; 2015. Chapter 19.
11. Lindkvist B. Diagnosis and treatment of pancreatic exocrine insufficiency. World J Gastroenterol. 2013 Nov 14;19(42):7258-66. PMID: 24259956.
12. Caldart F, de Pretis N, Luchini C, Ciccocioppo R, Frulloni L. Pancreatic steatosis and metabolic pancreatic disease: a new entity? Intern Emerg Med. 2023 Nov;18(8):2199-2208. Epub 2023 Jul 18. PMID: 37462859.

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