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CASE REPORT

Recurrent acute pancreatitis due to tertiary hyperparathyroidism manifesting as pancreatic calcification in a patient receiving continuous ambulatory peritoneal dialysis

Chen-Yi Liao [a], Chao-Wen Hsueh [b], Ching-Chang Lee [b], Wu-Hsien Kuo [b], Ren-Jy Ben [a], En-Hua Huang [c], Chih-Chiang Wang [a], I-Hung Chen [a], Ming-Kai Tsai [a,*]

[a] *Division of Nephrology, Department of Internal Medicine, Kaohsiung Armed Forces General Hospital, Kaohsiung City, Taiwan, ROC*
[b] *Division of Gastroenterology, Department of Internal Medicine, Kaohsiung Armed Forces General Hospital, Kaohsiung City, Taiwan, ROC*
[c] *Department of Radiology, Kaohsiung Armed Forces General Hospital, Kaohsiung City, Taiwan, ROC*
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KEYWORDS
acute pancreatitis;
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Abstract Chronic alcohol use and gallstones are the most frequent causes of acute pancreatitis (AP), a life-threatening medical emergency. Above-normal calcium levels in these patients indicate potential malignancy or hyperparathyroidism (HPT). Whereas primary HPT has been associated with AP, tertiary HPT has seldom been linked with AP. Herein we present a case of peritoneal dialysis presenting with recurrent AP and incidental pancreatic duct calcification due to tertiary HPT.

急性胰炎是致命的緊急疾病,最常見的病因為長期飲酒及膽結石。當這些病人的血鈣過高,意味著癌症或副甲狀腺功能亢進的可能性。急性胰炎可能與原發性副甲狀腺功能亢進有關,但鮮少見於三發性副甲狀腺功能亢進患者。以下是一宗三發性副甲狀腺功能亢進所致復發性急性胰炎的個案,患者為腹膜透析接受者,並以偶發性胰管鈣化呈現。

* Corresponding author. Kaohsiung Armed Forces General Hospital, Number 2, Zhongzheng 1st Road, Lingya District, Kaohsiung City 802, Taiwan, ROC.
E-mail address: tmk802@gmail.com (M.-K. Tsai).

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Introduction

Acute pancreatitis (AP) is a medical emergency; chronic alcohol use and gallstones are the most common etiologies, accounting for 60–70% of AP cases. Hypercalcemia resulting from a variety of conditions, including malignancy in the setting of bone metastases or multiple myeloma, vitamin D toxicity, sarcoidosis, familial hypocalciuric hypercalcemia, total parenteral nutrition, perioperative high-dose calcium infusions during cardiopulmonary bypass, and hyperparathyroidism (HPT), can also lead to AP. Specifically, HPT usually presents with hypercalcemia and high parathyroid hormone (PTH) levels; however, HPT-mediated hypercalcemia is not a commonly associated etiology of AP; it is reported only in 1.5–7% of AP cases in different series.[1] AP is most commonly regarded as a complication of primary HPT (PHPT) and is rarely reported in tertiary HPT patients.[2] Studies to date, suggest that patients with end-stage renal disease (ESRD) have an increased risk of developing AP,[3] which may further be influenced by the dialysis method used, including peritoneal dialysis (PD).[4] A number of studies have reported an increase in the incidence of AP in PD patients compared with those undergoing hemodialysis[4,5]; however, the underlying mechanism is yet to be established. Pancreatic calcification is an occasional feature of chronic alcoholic pancreatitis and is a rarely reported epiphenomenon in patients with tertiary HPT. Here we report a patient with recurrent AP and tertiary HPT in whom imaging studies revealed pancreatic calcifications. Our findings will aid clinicians in achieving a better understanding of the underlying pathogenesis.

Table 1 Laboratory parameters during acute pancreatitis attack.

	First	Second	Third	This admission
Months after 4 y of PD	29	38	42	43
Calcium (8.4–10.2 mg/dL)	10.7	10.2	10.6	10.4
Phosphate (2.4–4.5 mg/dL)	4.3	6.0	6.6	6.1
Parathyroid hormone (3.4–7 mg/dL)	794.5	835	1324.3	1298.8
Serum amylase (29–103 U/L)	61	21	195	140
Serum lipase (11–82 U/L)	1630	905	2585	1872
Peritoneal amylase (U/L)	N/A	N/A	N/A	1
Peritoneal lipase (U/L)	N/A	N/A	N/A	18
Peritoneal white blood cell count ($<$100 cells/mm^3)	8	5	N/A	5
Peritoneal culture	No growth	N/A	N/A	No growth
Alkaline phosphatase (34–104 U/L)	105	168	164	144
Triglyceride ($<$150 mg/dL)	124	141	97	136
BUN (7–20 mg/dL)	46	72	69	46
Creatinine (0.7–1.2 mg/dL)	9.4	9.7	10.6	11.1

BUN = blood urea nitrogen; N/A = none available; PD = peritoneal dialysis.

Case Report

A 60-year-old male with a history of hypertension and ESRD presented to the emergency department with profound epigastric pain radiating to the back; this pain was aggravated on sitting up and while drinking water. Eight years ago, the patient had been admitted to the hospital with complaints of fever, fatigue, and bruising in the extremities for 2 weeks. At that time, abdominal ultrasonography was performed, which revealed the right kidney size to be 8.0 cm and the left kidney size to be 7.4 cm. Based on a positive blood culture for *Salmonella* group D1, the patient was tentatively diagnosed with *Salmonella* group D1 bacteremia and probable hypertension-related Stage IV acute-on-chronic kidney injury; however, the patient had denied renal biopsy. Although bacteremia improved with 10 days of intravenous ciprofloxacin treatment, due to the progression of uremia, temporary hemodialysis was needed. Since then, the patient had been on regular continuous ambulatory PD with 4000 mL of 1.5% dextrose solution and 4000 mL of 2.5% dextrose solution every day for the last 8 years.

The patient did not have any previous complications due to PD such as infectious peritonitis. However, he had experienced three AP attacks in the last 2 years (Table 1). His medication consisted of a maintenance therapy for the prevention of renal osteodystrophy for the past 4 years with 0.5 g calcitriol (activated vitamin D) once a day, 400 g magnesium aluminum silicate two times a day as a phosphate binder, and 1.0 g peptidine [consisted of aluminum magnesium hydrate (400 mg) and magnesium trisilicate (300 mg)] three times a day also as a phosphate binder. He was an ex-smoker and denied alcohol abuse and a family history of AP. The patient's weight, height, and body mass index (BMI) were 53 kg, 169 cm, and 18.5 kg/m^2, respectively. On admission, his temperature was 36.4°C, pulse rate was 78 bpm, and blood pressure was 210/104 mmHg. Abdominal examination revealed moderate distension with generalized tenderness on palpation, but not on rebound, and auscultation revealed hypoactive bowel sounds. He was empirically treated with cefoxitin (1.0 g), intravenously administered every 8 hours, with an initial diagnosis of AP and suspected infectious peritonitis. Dialysate effluent fluid analysis showed a white blood cell count of 5/L, and microbiology culture revealed no growth, thus excluding the possibility of peritonitis. Despite antibiotic therapy for 5 days, his epigastric pain persisted, and the patient failed to improve; thus, he was diagnosed with AP based on the clinical presentation and laboratory findings. Abdominal computed tomography (CT) done to investigate recurrent AP revealed inflammation of the pancreas, fat stranding with moderate free intra-abdominal fluid, and no evidence of cholelithiasis. Satellite calcifications over the pancreatic body and duct, splenic artery, and abdominal aorta were noted; no other metastatic calcification sites were observed. (Figures 1A and 1B). Treatment