

Recurrent Acute Pancreatitis Secondary to Untreated Hyperparathyroidism: A Case Report and Literature Review

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Cristina Patoni ^{1, 2}, Stella Ioana Popescu ³, Cristian Gheorghe ^{1, 4}

¹. Gastroenterology, Carol Davila University of Medicine and Pharmacy, Bucharest, ROU ². Gastroenterology, “Dr. Carol Davila” Central Military Emergency University Hospital, Bucharest, ROU ³. Ophthalmology, “Dr. Carol Davila” Central Military Emergency University Hospital, Bucharest, ROU ⁴. Gastroenterology, Fundeni Clinical Institute, Bucharest, ROU

Corresponding author: Cristina Patoni, patonicristina@gmail.com

Abstract

Acute pancreatitis (AP) is a common gastrointestinal disease with diverse etiologies, among which hyperparathyroidism represents a rare but well-established cause. While a single episode of AP can often be managed supportively, failure to definitively treat the underlying hyperparathyroidism significantly increases the risk of recurrence.

We present the case of a 63-year-old woman admitted for moderate AP with documented hypercalcemia (total calcium 11.11 mg/dL). She was subsequently referred to endocrinology, where parathyroid hormone (PTH) was found to be 238 pg/mL, and parathyroid scintigraphy with 99mTc-sestamibi showed equivocal focal uptake at the upper pole of the right thyroid lobe, consistent with a possible right superior parathyroid adenoma. Despite confirmed biochemical hyperparathyroidism and a recent history of AP, the clinical recommendation was watchful waiting, with follow-up at three months, without surgical referral. Three years later, at the age of 66, she was re-admitted with a second episode of mild AP. PTH had risen to 777.5 pg/mL with total calcium 12.12 mg/dL, supporting hyperparathyroidism as the likely underlying etiology. Given the patient's underlying chronic kidney disease, tertiary hyperparathyroidism could not be excluded, although the patient's relatively stable renal function favored primary hyperparathyroidism.

This case highlights two important clinical lessons for the management of hyperparathyroidism-associated pancreatitis. First, it is essential to routinely assess serum calcium and PTH levels in all patients with AP of unknown origin. Second, the decision to perform a parathyroidectomy should be based on clinical and biochemical evidence and should not be postponed due to inconclusive imaging results. A review of the current literature emphasizes that parathyroidectomy is the only definitive treatment for symptomatic hyperparathyroidism. Early detection, prompt surgical referral, and effective communication with patients are vital for preventing recurrence in this manageable condition.

Categories: Internal Medicine, Endocrinology/Diabetes/Metabolism, Gastroenterology

Keywords: acute pancreatitis, hypercalcemia, parathyroidectomy, primary hyperparathyroidism, recurrent pancreatitis, tertiary hyperparathyroidism

Introduction

Acute pancreatitis (AP) is a common and potentially life-threatening condition marked by inflammation of the pancreas. Although gallstones and alcohol consumption are the primary causes, a smaller yet significant number of cases are caused by metabolic issues such as hypertriglyceridemia and hypercalcemia [1,2]. Primary hyperparathyroidism (PHPT) is characterized by excessive parathyroid hormone (PTH) secretion, leading to elevated blood calcium levels. This condition is a known factor in both acute and chronic pancreatitis, with the reported incidence of pancreatitis in individuals with PHPT varying between 1.5% and 23%, depending on the study [3,4].

Various mechanisms explain the connection between hypercalcemia and pancreatitis. High levels of calcium in pancreatic acinar cells lead to the early activation of trypsinogen inside the cells, which starts the self-digestion of the pancreatic tissue [5]. Moreover, elevated calcium levels in pancreatic juice can lead to the formation of protein plugs and blockages in the ducts and may also cause the sphincter of Oddi to spasm [6]. Importantly, not all patients with PHPT develop pancreatitis, which has led to investigations into additional genetic susceptibility factors, including mutations in the SPINK1 trypsin inhibitor gene (N34S) and the cystic fibrosis transmembrane conductance regulator (CFTR) [7].

PHPT predominantly affects women, with a higher incidence in postmenopausal women. Female predominance varies by region but is consistently reported across all populations [8]. Typical signs include nephrolithiasis, osteoporosis, and neurocognitive symptoms. Although pancreatitis is uncommon, it is a

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well-known complication that can potentially be avoided [3]. One notable clinical pattern is the recurrence of pancreatitis in individuals whose PHPT remains either undetected or poorly managed [9,10].

We report the case of a 63-year-old woman who experienced two episodes of acute pancreatitis over three years. Upon initial admission, the patient's serum calcium was elevated, but PTH was not measured. Later, an endocrinological assessment confirmed PHPT through parathyroid imaging, although parathyroidectomy was not advised, leading to recurrence of pancreatitis. This case includes a review of the existing literature on pancreatitis associated with PHPT, highlighting the impact of delayed diagnosis and delayed surgical intervention.

Case Presentation

First episode

A 63-year-old woman with a significant past medical history, including arterial hypertension, chronic obstructive pulmonary disease (COPD), stage 4 chronic kidney disease (CKD, KDIGO classification) [11], recurrent urinary tract infections, vesical lithiasis, and deep vein thrombosis of the right lower limb, presented to the emergency department with epigastric pain radiating to the back in a band-like pattern.

Physical examination revealed an afebrile patient with a BMI of 27 kg/m², with tenderness in the upper abdominal quadrants. Abdominal ultrasound excluded cholelithiasis as an etiological factor. Abdominal CT confirmed acute pancreatitis (Figure 1).

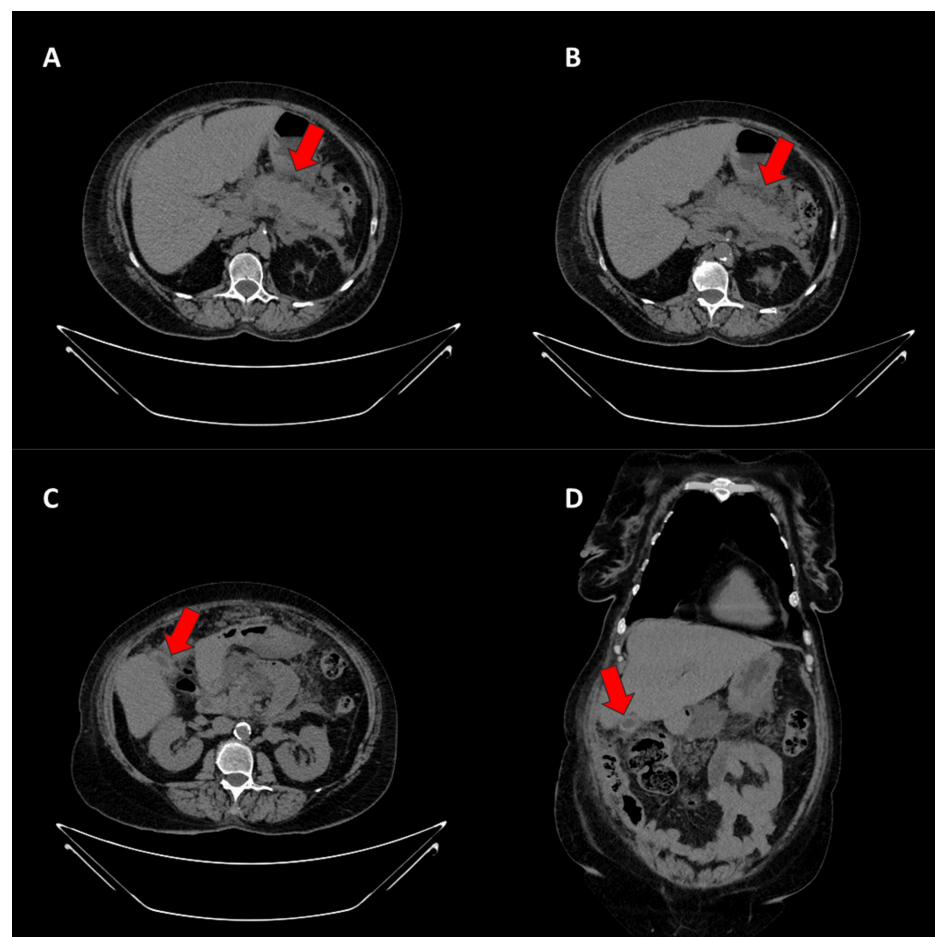


FIGURE 1: First episode of acute pancreatitis

Images A, B: CT images demonstrating an enlarged pancreas with indistinct margins and homogeneous native parenchymal attenuation. Peripancreatic fluid collections with near-fluid densities are noted in the omental bursa and in the infragastric space, consistent with acute interstitial edematous pancreatitis.

Images C, D: Partially distended gallbladder with diffusely thickened walls, without evidence of cholelithiasis.

Based on the presence of transient organ dysfunction (transiently elevated creatinine), the episode was classified as moderate acute pancreatitis according to the revised Atlanta classification [1]. Laboratory

findings are summarized in Table 1. Of note, total serum calcium was elevated at 11.11 mg/dL; serum creatinine was 1.80 mg/dL, broadly consistent with the patient's known baseline CKD G4 status. The patient was not taking calcium-containing phosphate binders, vitamin D supplementation, or calcium-containing antacids at the time of this admission. Serum PTH was not measured at this admission, and serum phosphorus was not measured at any point during the patient's subsequent evaluation. Alcohol consumption was denied by the patient.

Parameter	Normal range	Episode 1 (age 63)	Episode 2 (age 66)
Amylase (U/L)	28-100	350 ↑	283 ↑
Lipase (U/L)	8-78	284 ↑	171 ↑
Total calcium (mg/dL)	8.8-10.60	11.11 ↑	12.12 ↑
Ionized calcium (mmol/L)	3.82-4.82	Not measured	5.64 ↑
PTH (pg/mL)	15-68.3	Not measured	777.5 ↑
CRP (mg/L)	0-5	319 ↑	211 ↑
Leukocytes (/mm ³)	4,100-10,900	14,280 ↑	11,230 ↑
Urea (mg/dL)	20.97-43	146 ↑	118 ↑
Creatinine (mg/dL)	0.50-1.20	1.80 (transient ↑)	2.4 ↑ (baseline CKD)
GGT (U/L)	0-38	102 ↑	51
Alkaline phosphatase (U/L)	46-122	146 ↑	47
ALT (U/L)	0-34	15	11
AST (U/L)	11-34	21	13
Triglycerides (mg/dL)	30-150	152	167
Cholesterol (mg/dL)	<200	132	236 ↑
Fibrinogen (mg/dL)	276-471	1,163 ↑	760 ↑
D-dimers (ng/mL)	0-250	1,026 ↑	1,245 ↑
Procalcitonin (ng/mL)	<0.5	0.75	0.38
Urine culture	Positive/negative	Negative	E. coli positive
CA-125 (U/mL)	0-35	485.2 ↑	35.4
CA 19-9 (U/mL)	0-37	10.36	35.16
CEA (U/mL)	0-5	1.86	<1.73
AFP (U/mL)	0-8.78	<2	3.02
Abdominal ultrasound	Not applicable	No cholelithiasis	Not repeated

TABLE 1: Comparative laboratory values and clinical parameters at both admissions

↑ = above reference range

AFP = Alpha-Fetoprotein; ALT = Alanine Aminotransferase; AST = Aspartate Aminotransferase; CA-125 = Cancer Antigen 125; CA 19-9 = Cancer Antigen 19-9; CEA = Carcinoembryonic Antigen; CKD = Chronic Kidney Disease; CRP = C-reactive Protein; E. coli: Escherichia coli; GGT = Gamma-Glutamyl Transferase; N/A = Not Available; PTH = Parathyroid Hormone

The patient was managed conservatively with intravenous fluid resuscitation (Ringer's lactate), bowel rest, analgesics, antiemetics, and electrolyte correction. She also received intravenous iron and allopurinol. Inflammatory markers improved progressively, and she was discharged with recommendations to undergo gynecological, nephrological, and endocrinological evaluation, given the elevated calcium and markedly elevated CA-125 (485.2 U/mL).

Endocrinological workup between episodes

Following discharge, the next endocrinological assessment documented in our institution's records took place approximately one and a half years later. At that assessment, laboratory testing showed a markedly elevated parathyroid hormone level of 1406 pg/mL, total serum calcium of 15 mg/dL, triglycerides of 422 mg/dL, high-density lipoprotein (HDL)-cholesterol of 235 mg/dL, and serum creatinine of 1.53 mg/dL.

Approximately one month later, at a further follow-up visit, serum creatinine had risen to 2.22 mg/dL, while total calcium had decreased to 12 mg/dL.

Approximately two months after this visit, parathyroid scintigraphy was performed using 99mTc-sestamibi with planar cervical acquisition and single-photon emission computed tomography (SPECT)-CT, using a wash-out protocol (administered dose: 555 MBq; effective dose: 5 mSv). At this assessment, serum PTH had decreased to 238 pg/mL (reference range 15–65 pg/mL), still confirming biochemical hyperparathyroidism. The scintigraphic study demonstrated discrete inhomogeneity of thyroid uptake with near-complete wash-out on delayed acquisition, with the exception of minimal focal retention projected at the upper pole of the right thyroid lobe, raising the possibility of a right superior parathyroid adenoma, though findings remained inconclusive (Figure 2). An incidental 7 mm subpleural pseudonodular density in the posterobasal segment of the left lower lobe was noted on native CT sections, without uptake of the radiotracer. Cervical ultrasound for parathyroid localization was not performed.

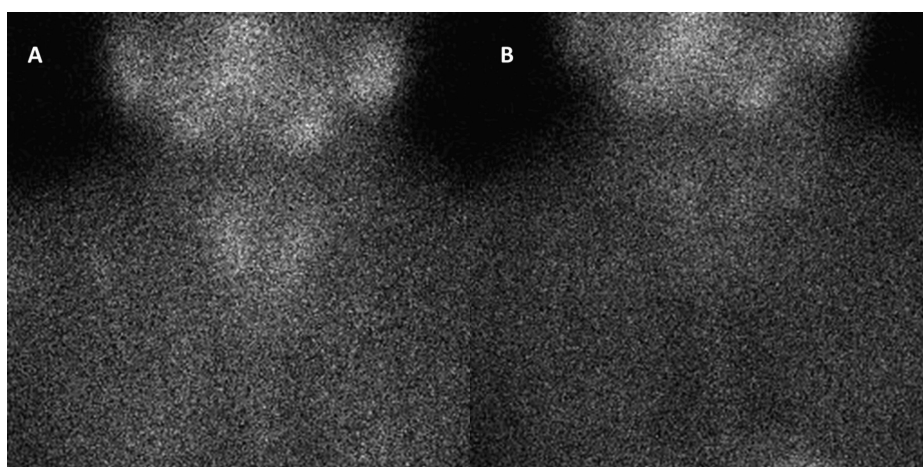


FIGURE 2: Tc-99m sestamibi parathyroid scintigraphy (wash-out protocol); planar cervical acquisition

Early phase image (A) demonstrates diffuse radiotracer uptake in the thyroid gland. Delayed phase image (B) shows near-complete wash-out of thyroid activity, with only minimal residual focal uptake projected at the upper pole of the right thyroid lobe, raising the possibility of a right superior parathyroid adenoma, though planar findings remained inconclusive.

Despite confirmed biochemical evidence of hyperparathyroidism and despite the recent history of acute pancreatitis, which constitutes a symptomatic indication for parathyroidectomy according to current guidelines [12], the endocrinological recommendation at this stage was watchful waiting, with follow-up at three months. No surgical referral was made, and no pharmacological treatment was initiated. No record of a specific pharmacological or surgical intervention for hyperparathyroidism is available from this interval, and it is not established from the retrospective record whether further endocrinology or nephrology evaluation occurred between this assessment and the second hospital admission, described below.

Second episode

Three years later, the patient presented again with band-like epigastric pain, nausea, and vomiting. Physical examination revealed abdominal distension and diffuse tenderness in the upper abdomen, without signs of peritoneal irritation. The episode was classified as mild acute pancreatitis according to the revised Atlanta classification, with no organ dysfunction or local complications [2]. Serum PTH was now 777.5 pg/mL, more than three times the value recorded at the endocrinological assessment, with total calcium 12.12 mg/dL and ionized calcium 5.64 mg/dL, confirming progressive PHPT as the etiology of recurrent pancreatitis. Laboratory results for both episodes are compared in Table 1.

Imaging revealed inhomogeneous pancreatic parenchyma with edematous hypodense areas consistent with acute pancreatitis, pulmonary emphysema, chronic pyelonephritis, and a 1.3 cm hypodense lesion with irregular margins in the posterior wall of the uterine body (Figure 3).

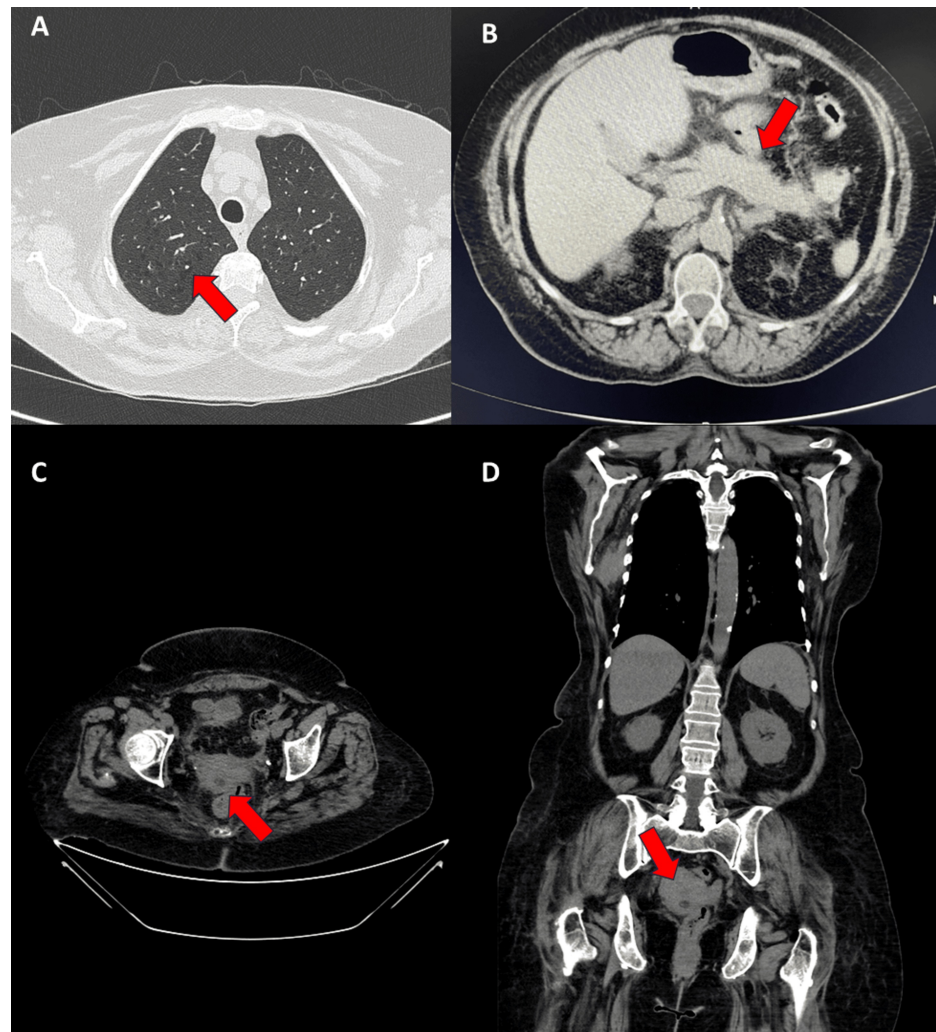


FIGURE 3: Second episode of acute pancreatitis with incidental findings

Image A: Multiple small emphysematous blebs in the upper lobe of the lung; Image B: Heterogeneous pancreatic parenchyma with increased density and heterogeneity of the peripancreatic fat; Images C, D: A hypodense lesion with irregular margins, measuring 1.3 cm, is identified in the posterior wall of the uterine body.

Concurrent urinary tract infection with *Escherichia coli* was documented. Management included intravenous Ringer's lactate, analgesics, antiemetics, antibiotic therapy (cefotaxime), and a single dose of zoledronic acid for hypercalcemia control. Clinical improvement was achieved, and the patient was discharged with recommendations for endocrinological, gynecological, and cardiological follow-up.

Discussion

Serum calcium and PTH as part of the standard etiological workup in acute pancreatitis

One of the key takeaways from this case is the importance of routinely including serum calcium and, if elevated, PTH in the diagnostic evaluation of acute pancreatitis. During the initial episode, the total calcium level was 11.11 mg/dL, gallstones were ruled out via abdominal ultrasound, and the patient denied alcohol use. These combined findings justified further metabolic assessment. However, PTH was not measured during this hospital stay, and the hypercalcemia was not further investigated until the patient was referred to endocrinology post-discharge. This scenario is not rare in clinical settings. Lanitis et al. reported a series of patients with recurrent acute pancreatitis where elevated calcium levels had been noted in several previous admissions without triggering additional investigation [13]. The practical implication is evident: serum calcium should be routinely measured as part of the laboratory assessment for all patients experiencing acute pancreatitis, especially when typical causes have been ruled out. This aligns with the current American College of Gastroenterology guidelines, which recommend including calcium in the initial evaluation of unexplained pancreatitis cases [14].

Another factor to consider in this situation is the presence of accompanying metabolic risk factors. During the second episode, cholesterol was 236 mg/dL, and triglycerides were 167 mg/dL. Although these figures do not meet the criteria typically linked to pancreatitis caused by hypertriglyceridemia, they do indicate additional metabolic influences. According to Bai et al., about 65% of PHPT patients who experienced pancreatitis had at least one other known risk factor [3]. This overlap has clinical significance: the presence of additional risk factors should not discourage a thorough investigation of hypercalcemia as a possible underlying cause, especially in cases of recurrence.

Diagnostic considerations: primary, secondary, or tertiary hyperparathyroidism

PHPT is classically characterized by autonomous PTH secretion resulting in hypercalcemia, with serum phosphate typically low or low-normal. Although hypophosphatemia is a recognized rather than a defining feature of PHPT, it is not part of the formal diagnostic or surgical criteria [15]. The biochemical picture in our patient: persistent, markedly elevated PTH (238-1406 pg/mL) together with hypercalcemia (total calcium 11.11-15 mg/dL), is broadly consistent with this pattern. However, the patient's chronic kidney disease (CKD G4, KDIGO) raises a second possibility that needs to be considered before PHPT can be accepted as the most likely explanation.

Secondary hyperparathyroidism (SHPT), the compensatory response to chronic hypocalcemia most often driven by CKD, can be ruled out with reasonable confidence: serum calcium in SHPT is expected to be low or normal rather than elevated, reflecting a parathyroid response that remains coupled to the calcium-sensing feedback loop [16]. This is not the pattern observed throughout this patient's course.

More difficult is the distinction between PHPT and tertiary hyperparathyroidism (THPT), in which parathyroid tissue chronically stimulated by CKD eventually becomes autonomous, producing a biochemical picture represented by elevated PTH with hypercalcemia that can closely resemble PHPT. Distinguishing SHPT from THPT in patients with CKD is often challenging due to overlapping biochemical and clinical features [17], and the same overlap complicates distinguishing established THPT from PHPT.

Phosphorus is one of the parameters that help distinguish these two entities: in THPT, end-stage renal disease is complicated by parathyroid disease characterized by elevated PTH, calcium, and phosphate concentrations [18], in contrast to the low or low-normal phosphate more typical of PHPT [15]. No serum phosphorus measurement was obtained at any point in this patient's recorded history, thereby removing the parameter that could most directly have supported or refuted either diagnosis.

The parathyroid scintigraphy obtained in this patient was equally inconclusive. It is important to note that Sestamibi scintigraphy is significantly less sensitive for detecting multiglandular hyperplasia than for detecting a solitary adenoma. Therefore, an inconclusive scan cannot, on its own, differentiate between a small or atypically located adenoma (which would suggest primary hyperparathyroidism, or PHPT) and multiglandular hyperplasia, which is typically associated with tertiary hyperparathyroidism (THPT) [19,20]. Where first-line scintigraphy is inconclusive, complementary localization with cervical ultrasound, 4D-CT, or 18F-fluorocholine PET/CT may add diagnostic value, the latter shown to be more sensitive than conventional imaging both generally [21] and specifically in CKD-associated hyperparathyroidism [22]. None of these were performed in our patient.

This diagnostic overlap is not unique to our patient: published cases of PHPT versus THPT in CKD have generally only been resolved with confidence after surgical and histopathological examination. Lunn et al. described a patient with CKD and PTH exceeding 2400 pg/mL whose presentation was explicitly framed around this same question, attributing it to PHPT only after surgery confirmed a large adenoma [23]. Strakosha et al. similarly confirmed a primary adenoma by surgery and histopathology in a patient initially managed for presumed SHPT [24]. THPT itself, while classically associated with dialysis or transplantation, has also been reported in non-dialysis, non-transplant patients with advanced CKD, with severe consequences when diagnosis is delayed [25]. The one feature lending modest support to PHPT over THPT is the relatively stable renal function (creatinine approximately 1.5-2.4 mg/dL) rather than progression toward dialysis-dependent disease, the setting in which THPT is most consistently described.

Surgical indication in symptomatic PHPT: a clinical and biochemical decision

Perhaps the most instructive aspect of this case concerns the relationship between imaging findings and the surgical indication in PHPT. Following the first episode of pancreatitis, the patient underwent parathyroid scintigraphy with 99mTc-sestamibi and SPECT-CT, with the equivocal result described above; cervical ultrasound was not performed. In this context, the decision to recommend watchful waiting rather than immediate surgical referral was clinically understandable on imaging grounds alone.

However, this case underscores a principle clearly outlined in the guidelines from the Fifth International Workshop on the Evaluation and Management of Primary Hyperparathyroidism: the decision to perform

parathyroidectomy in PHPT is based on clinical and biochemical criteria, rather than on imaging results [26]. Parathyroid imaging identifies the location of an abnormal gland for surgical planning, but does not determine whether surgery should be performed. For patients with a biochemical picture consistent with PHPT who also have symptoms such as AP, surgery is warranted regardless of imaging outcomes. This case highlights that inconclusive parathyroid imaging should prompt further localization studies, such as cervical ultrasound, 4D-CT, or 18F-fluorocholine PET/CT, rather than deferring the surgical decision [27,28].

Additionally, in their respective studies, Misgar et al. and Chowdhury et al. reported that parathyroidectomy normalized calcium and PTH levels, and no subsequent pancreatic episodes were observed during follow-up [4,29].

Disease progression and the importance of a clear discharge plan

The biochemical changes observed across this patient's course are significant: PTH rose to 1406 pg/mL with total calcium of 15 mg/dL at an outpatient visit prior to scintigraphy, decreased to 238 pg/mL at the time of the (equivocal) scintigraphic assessment, and subsequently rose again to 777.5 pg/mL, with total calcium of 12.12 mg/dL, by the time of the second pancreatitis episode. This fluctuating but overall progressive trajectory illustrates the potential for substantial biochemical instability and progression in untreated symptomatic PHPT/THPT and underscores the importance of timely, definitive management [17,26].

The available medical records do not explain why structured follow-up was not maintained between the two episodes. It remains unclear whether the patient missed scheduled appointments, whether follow-up was not organized beyond the initial three-month suggestion, or whether other factors played a role. This uncertainty highlights a crucial clinical point: upon discharge after diagnosing symptomatic PHPT (or a biochemical picture in which THPT cannot be excluded), it is essential to provide the patient with a clear and detailed management plan, including the natural progression of untreated disease, the risk of recurrent pancreatitis without definitive treatment, and the necessity of surgical referral as the only curative option [26].

Multidisciplinary management and additional considerations

This case highlights the critical role of coordinated multidisciplinary efforts for patients with PHPT who present with complex comorbidities. The patient had stage 4 CKD, COPD, a history of deep vein thrombosis, and, during the second hospital stay, an incidental uterine lesion together with persistently elevated CA-125 levels first noted at the initial episode. In this scenario, gastroenterology, endocrinology, endocrine surgery, nephrology, and gynecology each offered vital perspectives, and a unified, multidisciplinary management approach, explicitly addressing the CKD-related differential discussed above, would ideally have guided this patient's care.

Limitations

This report has several limitations: serum phosphorus was never measured, removing a key parameter for distinguishing PHPT from SHPT/THPT in CKD. As a retrospective report, additional evaluation outside our institution's records cannot be excluded. No localization study beyond a single equivocal scintigraphy was performed, and, most importantly, no surgical exploration or histopathological confirmation was obtained, leaving the distinction between PHPT and THPT unresolved.

Conclusions

Hyperparathyroidism, though uncommon, is a significant and potentially treatable cause of repeated AP episodes. This case, along with the related literature review, highlights three essential clinical points. First, it is crucial to measure serum calcium in all AP patients, and any increase should immediately lead to a PTH evaluation. Second, the decision to perform parathyroidectomy in symptomatic PHPT is based on clinical and biochemical evidence. Uncertain parathyroid imaging should not prevent surgery but rather prompt further localization studies. Third, if parathyroidectomy is necessary but postponed for any reason, a detailed follow-up plan must be clearly communicated to the patient, including specific criteria for urgent reassessment. Effective collaboration among gastroenterology, nephrology, endocrinology, and endocrine surgery specialists is vital to ensure that the need for surgery is identified, clearly conveyed, and addressed promptly.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Cristina Patoni, Stella Ioana Popescu, Cristian Gheorghe

Acquisition, analysis, or interpretation of data: Cristina Patoni

Drafting of the manuscript: Cristina Patoni

Critical review of the manuscript for important intellectual content: Stella Ioana Popescu, Cristian Gheorghe

Supervision: Cristian Gheorghe

Disclosures

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