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Case Report

Parathyroid carcinoma presenting as severe necrotising pancreatitis: early recognition and cure

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ABSTRACT

Parathyroid carcinoma (PC) is rare and typically presents with severe hypercalcemia. Acute pancreatitis as the initial manifestation is uncommon and may delay diagnosis. A 50-year-old lady with no comorbidities presented to the medical Gastroenterology Department with acute severe necrotising pancreatitis (CTSI 9, BISAP 2), complicated by multiorgan dysfunction. Laboratory evaluation revealed marked hypercalcemia (corrected calcium 12.3–11.3 mg/dl), hypophosphatemia (1.5 mg/dl), and a significantly elevated parathyroid hormone (PTH) level (217 pg/ml). No gallstones were identified on imaging. Further localisation with neck ultrasound and sestamibi scan highlighted a right superior parathyroid lesion. Following optimisation, the lesion was excised. Intraoperative PTH fell to 9.7 pg/ml at 30 minutes postoperatively and serum calcium normalised (POD1 9.9 mg/dl, POD2 8.4 mg/dl). Histopathology confirmed parathyroid carcinoma (25×20×15 mm) with capsular and vascular invasion. In severe or "idiopathic" pancreatitis, concurrent hypercalcemia with elevated PTH should prompt evaluation for primary hyperparathyroidism, including rare parathyroid carcinoma. Timely surgery can be curative; vigilant postoperative surveillance is essential.

Keywords: Parathyroid carcinoma, Hypercalcemia, Pancreatitis, Primary hyperparathyroidism

INTRODUCTION

Parathyroid carcinoma accounts for a small fraction of primary hyperparathyroidism, but often presents with profound hypercalcemia and target-organ involvement.[1] Pancreatitis is an infrequent initial presentation and may be overlooked when more common aetiologies dominate. However, there is a gap in current guidelines and clinical awareness regarding the recognition of parathyroid carcinoma as an underlying cause of acute or unexplained severe pancreatitis, particularly in the absence of classic risk factors. This case highlights the need for increased vigilance in identifying rare but clinically significant associations to support timely diagnosis and intervention. Our findings also suggest that current guidelines for the evaluation of acute pancreatitis and primary hyperparathyroidism could be strengthened by recommending routine assessment of serum calcium and PTH in cases of idiopathic or severe pancreatitis. Incorporating such recommendations may facilitate earlier identification of underlying parathyroid carcinoma and improve patient outcomes.

Learning objectives

This report highlights the early clinical and laboratory features that can assist in the prompt recognition of parathyroid carcinoma presenting as pancreatitis. It also

reviews key aspects of surgical management, emphasizing the importance of early diagnosis and timely intervention to improve patient outcomes in such cases. We report a case of parathyroid carcinoma revealed by severe necrotizing pancreatitis, emphasizing diagnostic cues, perioperative management, and oncologic considerations.

CASE REPORT

A 50-year-old woman with no history of gallstones, diabetes, or thyroid disease presented with acute severe necrotising pancreatitis (CTSI 9, BISAP 2), complicated by acute kidney injury and type 2 respiratory failure.

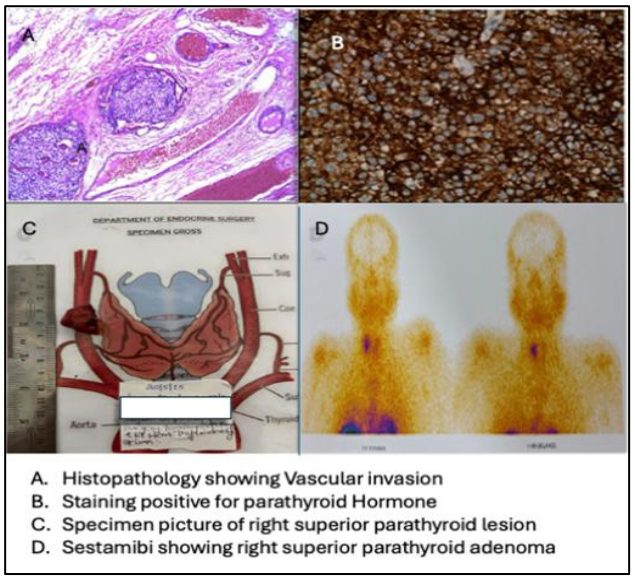

Figure 1 (A-D): Histopathology.

Intensive phase

She required admission to the intensive care unit for the organ support, including the intravenous fluids, electrolyte management, renal monitoring, and respiratory support. Aggressive supportive therapy led to the gradual resolution of acute kidney injury and respiratory compromise.

Serial laboratory evaluation revealed marked hypercalcemia (corrected Ca up to 12.3 mg/dl), hypophosphatemia (1.5 mg/dl), and very high pancreatic enzymes (lipase 17,224 u/l; amylase 1,552 u/l), with normal triglycerides and no cholelithiasis on ultrasound. Workup for hypercalcemia revealed an elevated intact PTH of 217 pg/ml.

Elective phase

After stabilization and transfer out of intensive care, further localisation identified a right posterior parathyroid lesion on ultrasound (approximately 16×14 mm) and sestamibi (approximately 22×14×26 mm) (Figures 1A and B). Following medical optimisation, she underwent focused excision of a right superior parathyroid mass (approximately 30×15×20 mm) adjacent to the RLN (Figure 1C); the right inferior gland appeared normal. Intraoperative PTH fell from 217 pg/ml to 9.7 pg/ml at 1 hour, and serum calcium normalized postoperatively (9.9 mg/dl on POD1, 8.4 mg/dl on POD2). Histopathology confirmed parathyroid carcinoma (approximately 25×20 ×15 mm) with capsular and vascular invasion; IHC was PTH-positive and thyroglobulin-negative (Figure 1D). She was discharged well on calcium supplementation with plans for biochemical surveillance.

At 12 months of follow-up, she remained asymptomatic with no evidence of recurrence clinically or on imaging. Laboratory testing showed normocalcemia (serum calcium 9.5 mg/dl) and PTH within the reference range. No complications or signs of disease relapse were observed during this period, highlighting a favourable prognosis with ongoing surveillance.

DISCUSSION

From a pathophysiology perspective, although uncommon, elevated calcium levels can precipitate acute pancreatitis. Among the various causes of hypercalcemia, primary hyperparathyroidism (PHPT) remains the leading contributor. Other sources, though less frequent, include drug-induced hypercalcemia, malignancies such as multiple myeloma, lymphomas, and promyelocytic leukemia, as well as granulomatous diseases like sarcoidosis and tuberculosis.[2,3] Hypercalcemia can trigger pancreatitis, likely via premature zymogen activation, ductal obstruction, and increased viscosity. At the bedside, recognition of these features, such as observing rising enzymes without a clear cause or noting ductal changes on ultrasound or CT in the context of hypercalcemia, can guide early consideration of parathyroid disease. In this patient, persistent hypercalcemia, markedly elevated PTH, and imaging findings pointed to PHPT as a reversible cause of severe necrotising pancreatitis.

Preoperative differentiation between parathyroid carcinoma and adenoma is challenging, but certain features may suggest malignancy. Clinically, carcinoma is more likely in the setting of very severe hypercalcemia (serum calcium typically greater than 14 mg/dl) and markedly elevated PTH. Although the diagnostic thresholds vary somewhat in the literature, a preoperative intact PTH level significantly above the upper reference limit (commonly greater than 300 pg/ml, and often exceeding 500 pg/ml in carcinoma) should raise a strong suspicion, particularly when accompanied by a palpable neck mass or evidence of end-organ involvement, such as renal or skeletal complications. Imaging may reveal a large, irregular, or invasive-appearing parathyroid lesion, local adherence to adjacent structures, or suspicious lymphadenopathy. These concrete cut-offs may help clinicians rapidly stratify risk and make decisions in acute

or high-pressure scenarios. While these clues are not definitive, their presence should heighten suspicion for carcinoma and prompt consideration of more extensive surgical planning.

Table 1: Reported cases of parathyroid carcinoma presenting with pancreatitis.

Castleman and Keating, 1969	49 F	Acute pancreatitis, bone pain	14.8	NA	Left inferior	Parathyroidectomy	Recovered
Shane et al, 1974	58M	Recurrent pancreatitis, nephrolithiasis	15.6	NA	Right inferior	Parathyroidectomy	Recovered
Akerström et al, 1980	51 F	Pancreatitis with bone pain	14.2	NA	Left superior	Excision + neck dissection	Alive at 2 years
Heath et al, 1984	55M	Pancreatitis with renal stones	15.1	NA	Right inferior	Parathyroidectomy	Recovered
Obara et al, 1989	62M	Acute abdomen, pancreatitis	16.3	980	Ectopic mediastinal	Sternotomy + excision	Recovered
Calandra et al, 1995	47 F	Pancreatitis with skeletal symptoms	13.9	1560	Right inferior	Parathyroidectomy	Disease-free at 3 years
Iihara et al, 2003	41M	Recurrent pancreatitis	15.0	920	Left inferior	Parathyroidectomy + neck dissection	Alive at 5 years
Pandey et al, 2007	52M	Pancreatitis with renal calculi	15.2	1500	Right inferior	Total parathyroidectomy	Alive at 2 years
Erbil et al, 2010	46 F	Acute pancreatitis, vomiting	14.7	980	Left inferior	Parathyroidectomy + hemithyroidectomy	Recovered
Păun et al, 2014	59 F	Acute pancreatitis, bone pain	15.8	1050	Left inferior	EN BLOC resection	Alive at 2 years
Yüceyar et al, 2018	54M	Pancreatitis, vomiting	16.1	1120	Retro-oesophageal	Parathyroidectomy	Alive at 4 years
Ramírez et al, 2021	45 F	Pancreatitis with renal stones	14.5	760	Left inferior	Parathyroidectomy	Disease-free at 1 year
Puzhakkal et al, 2026 (under review)	75 F	Acute pancreatitis with hypercalcaemic crisis	16.2	731	Right superior	Parathyroidectomy + hemithyroidectomy	Normocalcaemic at follow-up
Puzhakkal et al, 2026 (present case)	50 F	Severe necrotizing pancreatitis	12.3	217	Left inferior	Parathyroidectomy	Normokalaemia on follow up

If carcinoma is suspected intraoperatively based on gross findings such as a firm, grey-white, adherent mass or local invasion, surgeons should perform EN BLOC resection of the involved parathyroid gland, the ipsilateral thyroid lobe, and any adjacent soft tissues to ensure complete removal and minimize the risk of local recurrence. Care should be taken to avoid capsular rupture or tumour spillage. In cases of lymphadenopathy, selective lymph node dissection may also be appropriate. In cases where suspicion arises only intraoperatively, immediate adaptation of the surgical approach can improve local control and long-term outcomes.

Considering the epidemiology, the relationship between PHPT and pancreatitis remains debated. Differential diagnoses for acute pancreatitis included gallstone disease, alcohol, hypertriglyceridemia, drug-induced and rarer causes, but in the absence of these, PHPT was deemed causal or contributory.[4] The incidence of pancreatitis in individuals with PHPT varies among studies.[5] While large cohorts, such as those from the Mayo Clinic, have reported similar rates of pancreatitis in patients with and without PHPT, other reports suggest higher rates. Notably, severe hypercalcemia (calcium greater than 14 mg/dl) is correlated with increased risk of pancreatitis.[6] In some instances, as in our case, acute pancreatitis may be the first sign of unrecognized PHPT. Reports of pancreatitis directly associated with parathyroid carcinoma are exceedingly rare, with only about a dozen cases documented from 1969 to 2021, including both cervical and ectopic lesions. Most patients described in the literature had features of classical PHPT, such as renal or skeletal involvement.

For management, timely parathyroidectomy is important and is associated with decreased pancreatitis recurrence

and improved outcomes once the acute phase is managed. Our patient experienced a dramatic decline in PTH and normalization of serum calcium postoperatively, supporting the diagnosis of PHPT as a reversible cause. While parathyroid carcinoma typically presents with very high PTH, marked hypercalcemia, a palpable neck mass, and invasive features, operative findings in this case initially suggested adenoma. Histopathology, however, confirmed carcinoma with capsular and vascular invasion. Red flags for carcinoma include very high PTH and calcium, large or cystic lesions, local adherence, and end-organ damage.[7] The definitive diagnosis is based on invasive features, as seen here. When carcinoma is suspected preoperatively or intraoperatively, EN BLOC resection with ipsilateral thyroid lobectomy and soft-tissue excision offers optimal local control and reduces the risk of tumour seeding.

In our case, carcinoma was diagnosed after surgery, which is common.[8] If margins or surrounding tissues are involved in final pathology, early multidisciplinary discussion regarding completion of oncologic surgery is vital. Long-term surveillance is essential, as recurrence can be biochemical or structural and may be local or distant. For follow-up, current recommendations include monitoring serum calcium and PTH levels every 3 months for the first year postoperatively, then every 6 months for the next 2 years, and annually thereafter.[9] Neck imaging, such as ultrasound, may be considered annually or when biochemical evidence of recurrence is present.

More frequent assessments may be individualized based on initial tumour features and postoperative findings. Monitoring calcium and PTH is crucial; reoperation remains the mainstay for resectable relapses, and agents such as calcimimetics, bisphosphonates, or denosumab may help control hypercalcemia in unresectable cases.[10]

CONCLUSION

In cases of severe or “idiopathic” pancreatitis, concurrent hypercalcemia with elevated PTH should prompt evaluation for primary hyperparathyroidism, including rare parathyroid carcinoma. A recommended stepwise diagnostic approach includes:

(1) confirm hypercalcemia with repeat measurement of corrected serum calcium; (2) assess PTH levels to distinguish PTH-dependent from non-PTH-dependent causes; (3) evaluate for other causes of pancreatitis, such as gallstones, alcohol, and hypertriglyceridemia; (4) undertake focused parathyroid imaging, such as neck ultrasound and sestamibi scan, if PTH is elevated; and (5) consult endocrinology or endocrine surgery for further management. Timely surgery can be curative; vigilant postoperative surveillance is essential.

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