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Parathyroid Adenoma Revealed by Acute Pancreatitis: Case Report

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Abstract | **Case Report**

The association between acute pancreatitis and primary hyperparathyroidism (PHPT) has widely been reported in literature, but a causal relationship remains controversial. This case report is about a patient admitted with acute pancreatitis as a first manifestation of a parathyroid adenoma. The initial medical management followed by surgery permitted a total recuperation. The purpose of this paper is to keep in mind the metabolic etiology, by evaluating calcium and parathyroid hormone (PTH) in the differential diagnosis of non-biliary, non-alcoholic acute pancreatitis.

Keywords: Acute pancreatitis, Primary Hyperparathyroidism, Case Report.

INTRODUCTION

Primary hyperparathyroidism (PHPT) is caused by excessive, incompletely regulated secretion of parathyroid hormone (PTH) from one or more of the four parathyroid glands. Usually asymptomatic, it's one of the most common causes of hypercalcaemia and thus should be considered in anyone with an elevated calcium concentration [1].

Acute pancreatitis (AP) is an inflammatory disease affecting the exocrine part of the pancreatic gland. It's associated with high morbidity and mortality. The most frequent causes are gallstones and alcohol abuse, in up to 75% of the cases. Metabolic conditions giving rise to pancreatitis are less common, accounting for 5%-10% cases. The causes include hypertriglyceridemia, hypercalcemia, diabetes mellitus, porphyria, and Wilson's disease [2].

The coexistence of the two diseases has widely been reported in literature, but a causal relationship remains controversial. We report a case of hypercalcemia-induced acute pancreatitis as the first manifestation of a benign parathyroid adenoma in a female patient.

CASE PRESENTATION

A 78 years old woman with a history of hypertension treated by amlodipine, obese (BMI at 32kg/m2), and no alcohol consumption or smoking was admitted to the emergency ward on October 23, 2021. She was complaining from acute epigastric pain radiating to the back evolving for 1 week, associated to biliary vomiting for 2 days, in a context of general asthenia evolving for months.

On admission, medical statement found a conscious (GCS 15/15), apyretic and eupneic patient with a 99% of O2 saturation in ambient air, blood pressure was at 16/9 cmHg and a cardiac pulse at 72. The abdominal examination found no scars of prior interventions or abnormal mass, but acute epigastric tenderness.

Acute pancreatitis was suspected, and routine blood examination revealed white blood cell count of leukocytosis 15400/mm3 (NR = 4000-10000/mm3) with neutrophilia, 12g/dL of hemoglobin and platelets count at 190000/mm3. The biochemistry investigation objective high serum lipase at 660U/L (10-60U/L), an elevated C-Reactive Protein at 172mg/L (<5mg/L), with liver transaminases, alkaline phosphatase and gamma-glutamyltransferase within normal ranges, also a hypokalemia at 2,60 mEq/L (NR = 3,50-5,10mEq/L). The renal function was normal. Then a complementary analysis found hypercalcemia at 142 mg/L (85-105mg/L), hypophosphatemia at 18mg/L (NR = 23-47mg/L) and a high parathormone level at 392 pg/mL (NR = 6-40 pg/mL).

We noted an elevation of serum calcium levels during the first days attaining 178mg/ml.

After a fast echo that noted only a peri pancreatic inflammation with no signs of gallstones, a contrast-enhanced CT was performed revealing acute pancreatitis with a glandular necrosis of 30-50% (Grade C of Balthazar).

In order to confirm the diagnosis, cervical echography noted a nodule in the right inferior pole of the thyroid with a mediastinal extension; then was confirmed by a cervical MRI that shows the presence of right paratracheal formation suggesting a parathyroid adenoma. Thus, the diagnosis of PHPT associated to acute pancreatitis was made.

The therapeutic management included, during the 3 first days, a suspension of enteral nutrition, intravenous rehydration and bisphosphonates associated to 40mg per day of furosemide. The patient was still altered.

The surgical indication was made and the patient was sent to our department. An elective cervical exploration found a normal right thyroid lobe. A gentle medially retraction found a 2x1cm parathyroid adenoma. The levels of parathyroid hormone and calcium decreased within the first days, and the patient was discharged at the third post- operative day. At the next consultation 03 months later, the patient was feeling better and PTH and calcium levels with normal ranges.

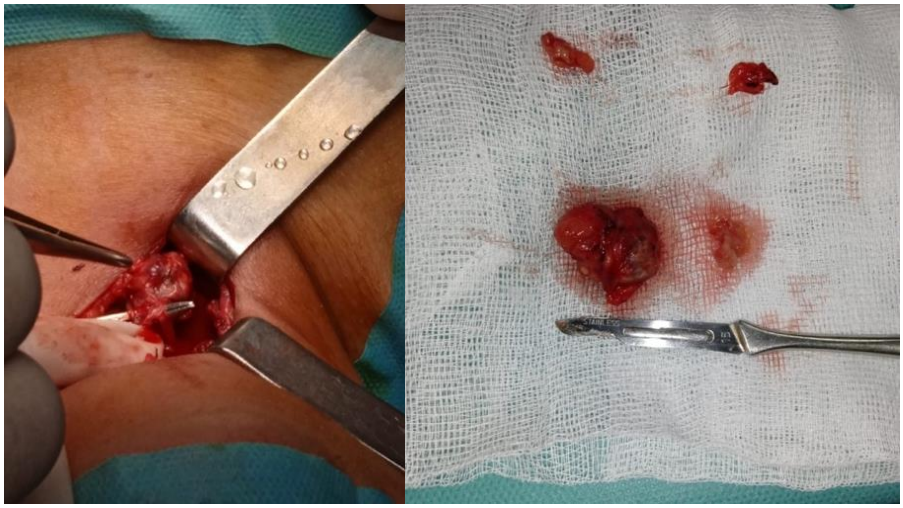
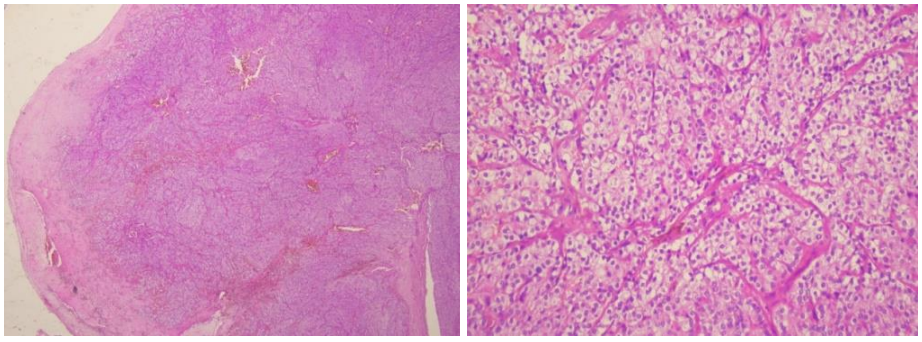


Figure 1: Intraoperative finding and after excision image of the adenoma



(a) (b)

Figure 2 : Anatomopathological aspect of the adenoma [(a) x100; (b) x 200]

DISCUSSION

Eirdheim was the first to report a case of the association of PHPT and acute pancreatitis in 1903 when he noted necrotizing pancreatitis in a patient with parathyroid adenoma [3]. However, it's only on 1957 that Cope and his colleagues wrote an original paper on the pancreatitis as a presenting manifestation of primary hyperparathyroidism [4].

PHPT represents a non-physiological overproduction of parathyroid hormone (PTH). It is most commonly caused by a single adenoma of the parathyroid gland (80-85%), but less common causes include parathyroid hyperplasia, carcinoma, and multiple endocrine neoplasia (MEN) types 1 and 2A.

Compared with young individuals, postmenopausal women over the age of 50 years have a greater probability of developing PHPT [5].

Acute pancreatitis due to PHPT-induced hypercalcemia is rare. In patients with PHPT, acute pancreatitis is reported in 3%-15% of patients. Nonetheless, hypercalcemia due to other causes can also induce acute pancreatitis [6].

Three mechanisms are involved in the development of PHPT-induced acute pancreatitis. One is PHPT-induced high serum calcium level, which can lead to acceleration of the conversion of trypsinogen to trypsin in the pancreas resulting in pancreatic auto digestion and subsequent acute pancreatitis [7]. Secondly, the accumulation of calcium can promote the formation of ductal obstruction, pancreatic calculi, and subsequent attacks of acute pancreatitis [4]. Thirdly, genetic variants in serine protease inhibitor Kazal type 1 and cystic fibrosis trans membrane conductance regulator genes in combination with hypercalcemia markedly increase the risk of developing acute pancreatitis in patients with PHPT [8].

Similarly, experimental models in rats have suggested that acute hypercalcemia induces dose dependent morphological alterations of acute pancreatitis, acute hyperamylasemia, and early ectopic trypsinogen activation [9].

In a study that included a total of 1435 patients, Carnaille *et al*, concluded that acute pancreatitis is the consequence and not the cause of PHPT; and hypercalcemia seems to be a major factor in the development of the pancreatitis in PHPT [10].

However, a cumulative review of the available large case series or cohorts with PHPT and pancreatitis over the last 30 years suggests that patients with PHPT develop a higher rate of pancreatitis than hospitalized patients without PHPT, this was limited by the fact that data are confounded in many instances by measurement bias and thus lack appropriate control subjects who have universally undergone serum calcium testing [11].

CONCLUSION

According to the literature review and studying this case report of acute pancreatitis caused by PHPT-induced hypercalcemia, it seems important to measure the levels of calcium, phosphor and parathyroid hormone (PTH) in the cases of acute pancreatitis, where there is no evidence of gallstones of alcohol consumption.

Establishing good cooperation between various hospital departments is also critical in treating this rare phenomenon.

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