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*RESEARCH ARTICLE*

# PRIMARY HYPERPARATHYROIDISM REVEALED BY ACUTE ALITHIASIC PANCREATITIS: A RARE CASE

**Fadoua Idrissi Zaki, Ahmed Mougui and Imane El Bouchti**
Rheumatology Department, ARRAZI Hospital, CHU Mohammed VI, Marrakech, Morocco.

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## *Abstract*

Acute pancreatitis is a rare presentation of Primary hyperparathyroidism (PHPT), its prevalence during PHPT is about 1%. The physiopathology of the implication of PHPT's hypercalcemia is not completely elucidated. Several hypotheses plead for the direct or indirect role of hypercalcemia via the activation of pancreatic proteases. Acute alithiasic pancreatitis can be a revealing feature of PHPT wich is a curable disease with the mastery of parathyroid surgery and hypercalcemia treatment. Herein, the authors report a rare case of a patient with PHPT revealed by acute alithiasic pancreatitis with a dreaded complication.



## Introduction:-

Primary hyperparathyroidism (PHPT) corresponds to inappropriate hypersecretion of parathyroid hormone (PTH) by one or more parathyroid glands. It is responsible for hypercalcemia, which may be complicated by acute pancreatitis (Amjoud et al. 2016a), which may exceptionally be the revealing mode of this endocrinopathy (Rabehi et al. 2017).

We report a rare case of a patient with PHPT revealed by acute alithiasic pancreatitis.

**Case report:-**

This was a 53-year-old female patient with a history of peptic ulcer disease (PUD) and low-energy fractures (clavicle,humerus and two costal fractures) treated traditionally, who came to the emergency room with an abdominal pain and uncontrollable vomiting. Clinical examination found a conscious patient with a stable hemodynamic and respiratory statusandsigns of malnutrition, dehydration, and abdominal tenderness without defensiveness or contracture. The biological check-up showed a high lipasemia (234 IU/L with a normal <130 IU/L). An urgent abdominal CT scan revealed anacute alithiasic pancreatitis stage C of Balthazar(Fig 1). The diagnosis of acute pancreatitis was retained and controlled by medical treatment: rapid rehydration, analgesia and parenteral nutrition.

After eliminating other classical causes of pancreatitis, the etiological assessment revealed hypercalcemia at 135 mg/L with a normal albumin range at 36.2g/L. The patient was transferred to the rheumatology department where a complete phosphocalcicassessment revealed a PHPT array with a hypercalcemia at 124mg/L, a normal albuminemia at 36g/L, a hypophosphatemia at 23 mg/L, a high level of parathormone at 1389.3 pg/mL, a normal 24-hour calciuria at 235mg/24H, a normal range of vitamin D at 34ng/mL, and a correct renal function. The radiological evaluation showed brown tumors and distal phalanx tufts (fig 2, 3 and 4).

**Corresponding Author:- Fadoua Idrissi Zaki**
Address:- Rheumatology Department, ARRAZI Hospital, CHU Mohammed VI, Marrakech, Morocco.

A bone densitometry was performed and showed osteoporosis with a Lumbar spine T-score of -4.3 (0.675g/cm2), -4.1 (0.480g/cm2) in the hip and -5 (0.440g/cm2) in the forearm. As part of the pre-surgical assessment of PHPT, a cervical ultrasound scan was performed and showed a thyroiditis aspect with a right cyst classified EU-TIRADS 2, and a left lobe nodular goiter classified EU-Tirads 3, without individualization of any parathyroid process. The cervical CT scan showed a left parathyroid lesion locally infiltrating the left thyroid lobe. The patient underwent a total thyroidectomy with left parathyroid adenomectomy. Extemporaneous examination revealed a diffuse nodular dystrophic goiter with a largely remodeled parathyroid adenoma without histological evidence of malignancy.

The first day after surgery, the patient presented with hypocalcemia that was maintained beyond day 4, with hypophosphatemia at 20 mg/L and a normal parathyroid hormone level at 75 pg/mL. The diagnosis of hungry bone syndrome (HBS) was retained and an intravenous calcium treatment (6g of calcium gluconate) with vitamin D supplementation (3 micrograms of Un-Alpha) was administered until stabilization of the calcemia over a period of 44 days with an oral relay of 4g/d then progressive degression to 2g/d.

## Discussion:-

PHPT is the 3rd most common endocrinopathy after diabetes mellitus and thyroid pathology (Bouchenna et al. 2017). It is the main etiology of hypercalcemia in outpatient clinics. Its clinical expression is highly variable. It depends on the level of hypercalcemia and its rate of onset. About 70 to 80% of patients showed up today at an asymptomatic stage thanks to the systematic calcium blood levels measurementand the diagnostic facilities inherent in the techniques for exploring the parathyroid glands.

When it is symptomatic, the manifestations are dominated by the involvement of the skeletal and renal systems, responsible for a decrease in bone mineral density (BMD), fragility fractures, as well as renal insufficiency, nephrocancinosis or kidney lithiasis.

Digestive manifestations of PHPT are dominated by constipation, epigastralgia, nausea, and the PUD that our patient presented (Dandurand et al. 2021).

Pancreatitis is a rare presentation of PHPT, its prevalence during PHPT is about 1% (Abouzahir et al. 2006), with a male predominance (Bouchenna et al. 2017). Their clinical symptomatology is intricate and dominated by abdominal pain and vomiting.

The association between PHPT and pancreatitis is not entirely coincidental. Nevertheless, the pathophysiology of this association is not completely elucidated (Rabehi et al. 2017). Some authors suggest that in PHPT, there is an accumulation of calcium in the pancreatic juice which increases the conversion of trypsinogen to trypsin leading to an aggression of the parenchyma and pancreatic ducts responsible for pancreatitis. Other authors believe that the pancreatic secretion is lower than normal with habituel enzymatic activity which results to the formation of protein plugs in the pancreatic ducts leading to their obstruction and the autodigestion of the pancreas. This obstruction could also be due to calcifications (Abouzahir et al. 2006). Also, parathyroid hormone is thought to play a role in the pathogenesis of pancreatitis by inhibiting pancreatic vascularization or by inducing the formation of microthrombi leading pancreatic parenchyma necrosis (Curto et al. 2009).

In all cases of PHPT, after treatment of hypercalcemia, parathyroid surgery is clearly the therapeutic option of choice (Dandurand et al. 2021), but the latter may rarely be complicated by HBS, secondary to exaggerated bone avidity for calcium, especially in situations of deep demineralization and bone remodeling (Amjoud et al. 2016b). Its prevalence is about 13%, characterized by rapid, profound and prolonged hypocalcemia beyond the 4th day of surgery, associated with hypophosphatemia and hypomagnesemia (GuillénMartínez et al. 2020).

The prevention of HBS is very controversial, some use vitamin D or even zoledronic acid preoperatively to prevent the risk of HBS (Salman et al. 2021- Mayilvaganan et al. 2017), but its treatment relies on the treatment and stabilization of hypocalcemia.

## Conclusion:-

Acute pancreatitis can be a revealing feature of PPH. Phosphocalcic assessment should be performed systematically in the presence of acute pancreatitis.

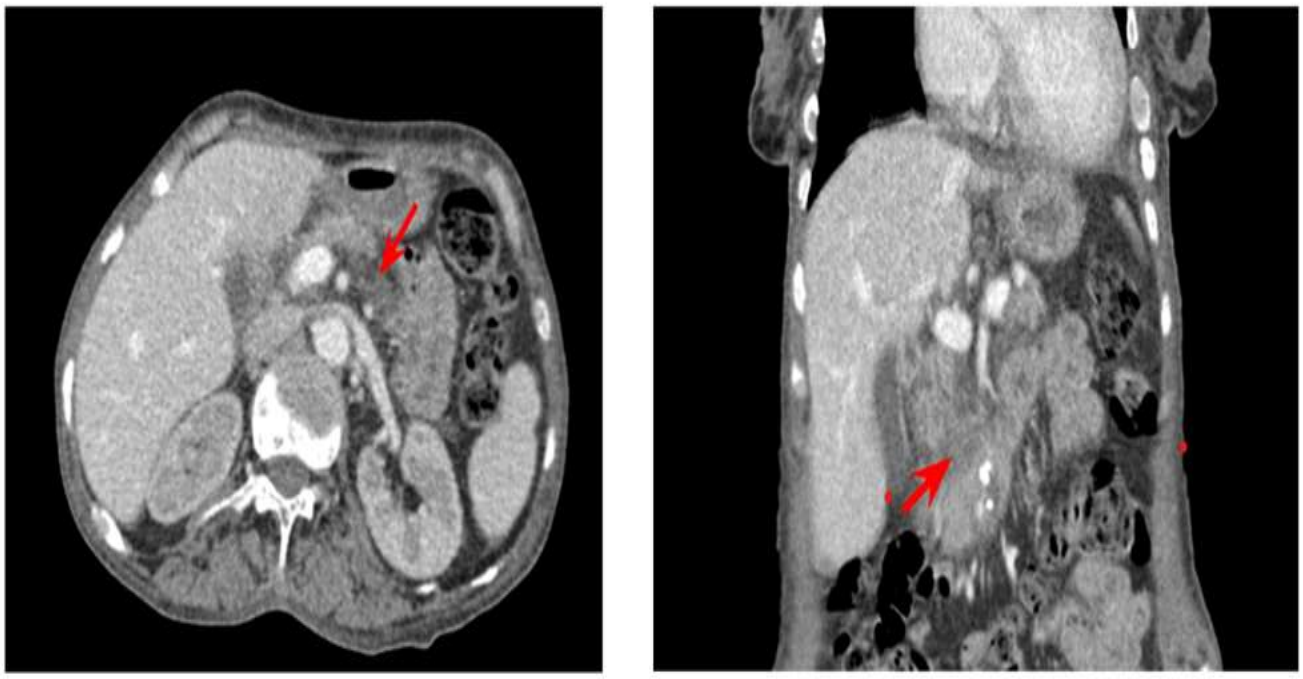

**Figure 1:-** Abdominal CT revealing alithiasic pancreatitis stage C with infiltration of the peri and subpancreatic fat (red arrow).

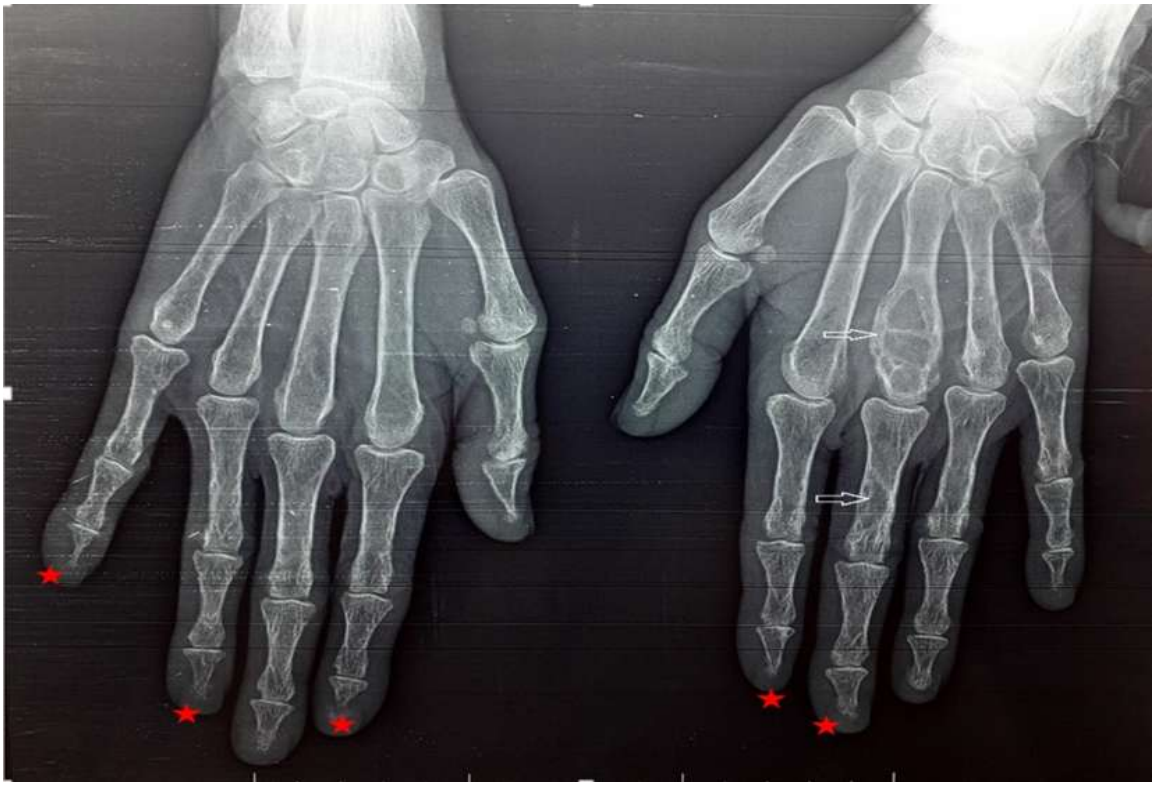

**Figure 2:-** X-rays of both hands in AP view showing diffuse bone demineralization, brown tumors (white arrows) with resorption of the phalangeal tufts (red stars).

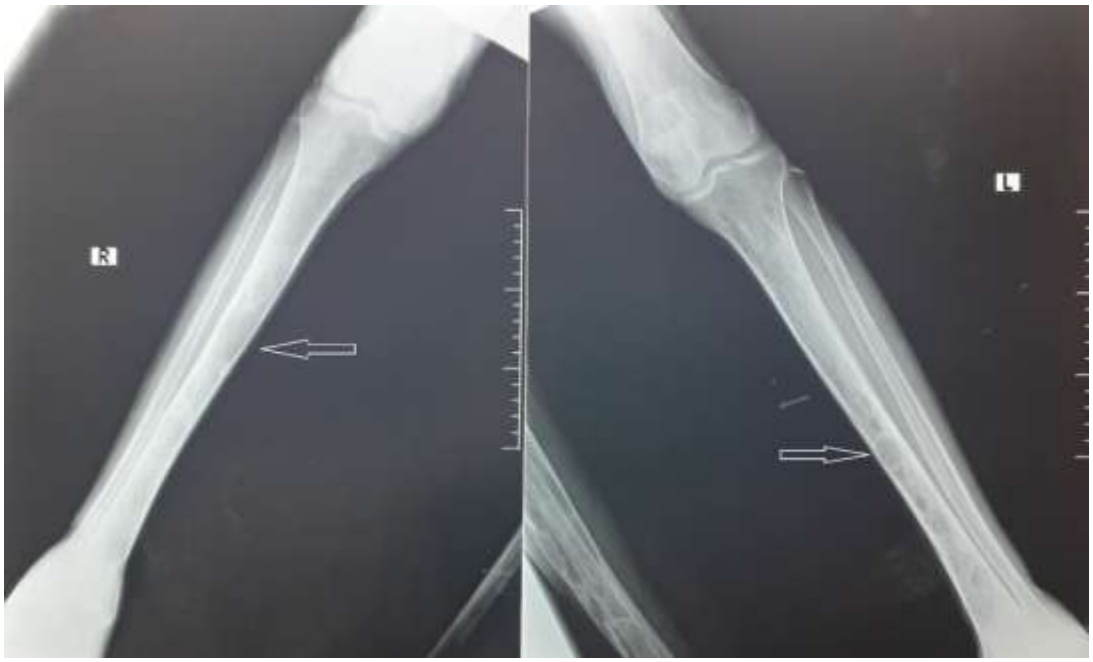

**Figure 3:-** X-ray of the legs in AP view showing brown tumors (arrows).

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