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In Case of Acute Pancreatitis; think of Hyperparathyroidism! A Case Report

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Abstract **Case Report**

***Introduction*:** The revelation of primary hyperparathyroidism by acute pancreatitis is an exceptional situation. ***Materiel and Methods*:** A case report. ***Case Report*:** We report the case of a 73 years old patient, hypertensive, having the antecedent of an ischemic cardiopathy complicated by cardiac insufficiency and atrial fibrillation, admitted to the urgencies for an abdominal pain evolving since 10 days, associated with vomiting, the initial biological assessment showed lipasemia at 2543IU/l, kidney failure and malignant hypercalcemia at 142mg/l. The etiological work-up showed primary hyperparathyroidism with double localization, the patient benefited from a parathyroidectomy with a good clinicobiological evolution. ***Discussion*:** The association hyperparathyroidism - acute pancreatitis was most often explained by the link of hypercalcemia, most of the above theories have not presented scientific evidence, and the current challenge is to seek a direct link between these two pathologies, the genetic theory remains an option, but its role is not clear.

Keywords: Acute pancrétitis, hypercalcemia, hyperparathyroidism, F-Choline Pet-scanner, calcimetrical treatment.

INTRODUCTION

Primary hyperparathyroidism is most often asymptomatic, revealed by hypercalcemia which is often discovered fortuitously in a routine check-up or in the context of exploration of a complication, which is frequently chronic. The occurrence of acute pancreatitis which reveals hyperparathyroidism is an exceptional situation. We report the case of severe acute pancreatitis (Balthazaar Stage E) revealing primary hyperparathyroidism.

CASE REPORT

Mrs. Z. Halima, 73 years old, presented to the emergency room with sudden onset pancreatic pain associated with vomiting, evolving for 10 days in a context of altered general condition and apyrexia.

Her history included hypertension complicated by ischemic heart disease at the stage of heart failure, treated with furosemide, spironolactone, a beta-blocker and an antiplatelet agent, and a cardiac arrhythmia under anti- coagulation therapy. She did not drink alcohol and had never undergone an intervention on the biliary-pancreatic tract.

The examination in the emergency room showed a patient in good general condition, her GCS was 15/15, hemodynamically (BP: 12/06 cm Hg) and respiratory (SpO2: 96%) stable, abdominal palpation showed epigastric pain with posterior radiation and slight defensiveness. and pulmonary auscultation showed signs of decompensation of the left heart failure.

The biological assessment showed: a lipasemia at 2543 IU/l, a kidney failure (Urea: 0,7g/l and Creatinine: 20) and a malignant hypercalcemia (Calcemia corrected at 142mg/l).

The diagnosis of acute pancreatitis was retained. The pancreatic CT scan, performed after 72 hours was in favor of a Balthazar E stage.

The patient was put on a calcimetrical treatment, associated with a hydration whose flow was adapted to the cardiac function, which allowed to obtain a biological improvement of the calcemia which passed to 98mg/l, and a normalization of the renal function.

The patient was transferred to the Gastroenterology Department where an etiological assessment showed a PTH level of 679.

Abdominal ultrasound and MRI scan did not reveal any lithiasis or tumor cause, and the lipid profile was normal.

On the basis of these arguments, the diagnosis of acute pancreatitis stage E complicating a primary hyperparathyroidism was retained.

The renal-vesical ultrasound, performed as part of the workup, did not find any renal lithiasis and the bone densitometry was in favor of osteoporosis.

Cervical ultrasound performed as part of the localization workup revealed a right parathyroid adenoma.

The MIBI-T scan did not show any scintigraphic evidence of pathological parathyroid tissue but an intense fixation of the left lobe left a doubt on its inferior pole as to the possibility of a parathyroid focus (figure 1).

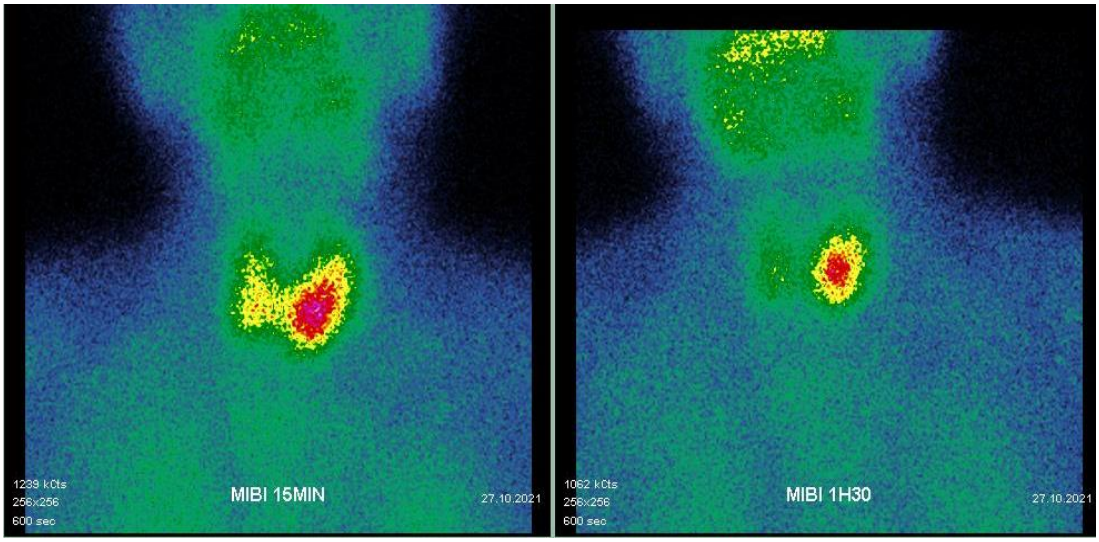

Figure 1: TC- Mibi scintigraphy of Mrs Z. Halim

The F-Choline PET-Scanner showed a right inferior thyroid hypermetabolism with a parathyroid appearance and a left posterior para-laryngeal hypermetabolism (figure 2, 3 and 4).

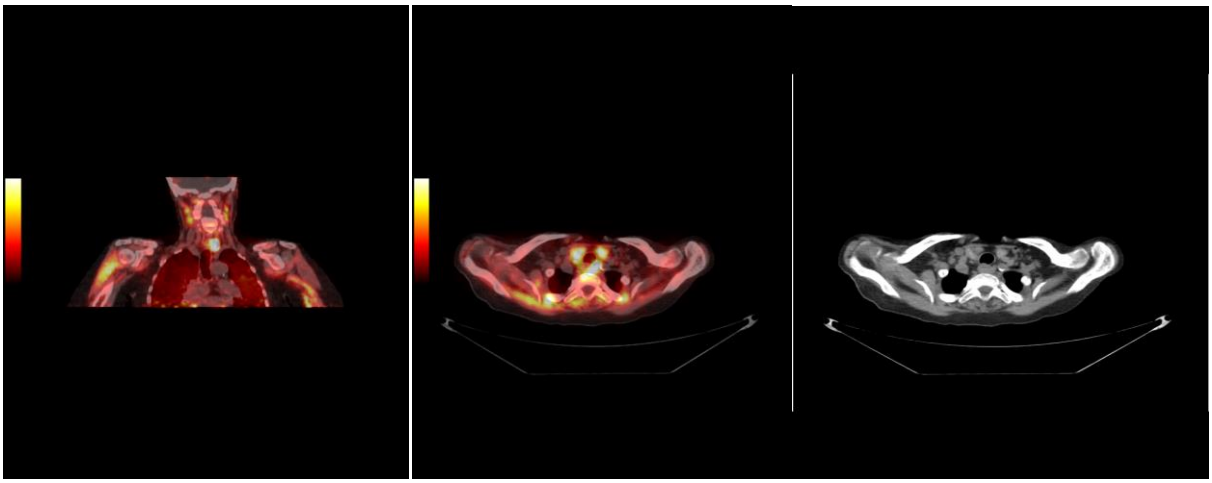

Figure 2 **Figure 3** **Figure 4**

Our patient benefited, after correction of electrolyte disorders, a dissection of the 2 pathological parathyroids (figure 5), whose anatomopathological study was in favor of a parathyroid nodule. The PTH was raised from 679pg to 70pg intra-operatively.

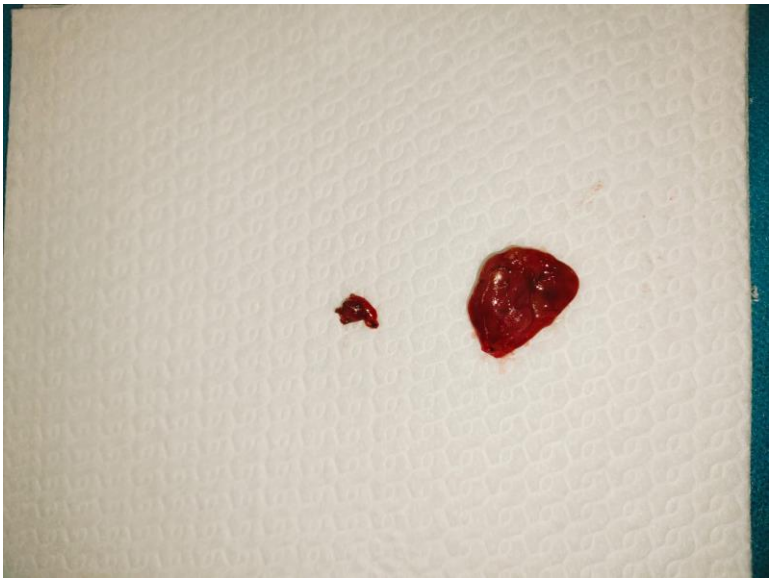

Figure 5: Surgical specimen

The evolution was marked by a clear clinicobiological improvement.

DISCUSSION

Hypercalcemia is a rare cause of acute pancreatitis, even more so if it is secondary to primary hyperparathyroidism; its prevalence in the literature varies, according to studies, from 1.5 to 5% [1-3]. Authors suggest that this association is not coincidental, and several pathophysiological explanations have been proposed, but none has been experimentally proven to date; the highest prevalence of acute pancreatitis in patients with hyperparathyroidism has been observed in those with hypercalcemia [4] and the hypercalcemia-acute pancreatitis link is currently well established. Indeed, significant evidence has accumulated supporting a pivotal role for intracellular calcium levels in the pathogenesis of acute pancreatitis [5], one explanation; suggests that mitochondrial calcium overload and the resulting decrease in ATP production results in dysfunction of calcium extrusion pumps. If calcium overload persists, complete ATP depletion ensues, preventing the acinar cell from entering the apoptotic pathway. This process leads to necrosis and subsequent destruction of the cell, with activated proteases in the interstitial space, affecting surrounding acinar cells and initiating a vicious cycle that ends in macroscopic inflammation (acute pancreatitis) and systemic disease (systemic inflammatory response syndrome) [6]. For Prinz and his team [7], acute pancreatitis is the consequence of a deposit of lithiasis secondary to an accumulation of calcium in the gastric juice. The third explanation is that of intra-pancreatic trypsinogen activation, which is widely accepted as the lever leading from acinar cell injury to acute pancreatitis [8]. Toxic intracellular calcium concentrations are consistently linked to premature activation of trypsinogen, although the exact mechanisms are not yet clear.

Recently, the role of a genetic substrate has been suggested; mutations in the SPINK1 (Kazal type I serine protease inhibitor) and CFTR (cystic fibrosis) genes have been detected in hyperparathyroid patients who developed acute pancreatitis [9]. Mutations have also been found in the CTRC (chymotrypsin C) gene [10]. SPINK1 is a trypsin- specific gene whose mutated variant has a reduced ability to inhibit trypsin that may be prematurely activated in the pancreas [9]. There are also variants in the anionic trypsinogen gene PRSS2, which appear to exert some protective effect against the development of AP in patients with hyperparathyroidism [11].

Our observation is particular, since severe acute pancreatitis is an exceptional mode of revelation of hyperparathyroidism, and hyperparathyroidism represents a rare etiology of acute pancreatitis. Our patient had a cardiac insufficiency whose treatment included a diuretic (furosemide), and we suggest that the latter played an important role in the worsening of hypercalcemia secondary to hyperthyroidism, which ultimately led to the installation of acute pancreatitis.

CONCLUSION

The association hyperparathyroidism - acute pancreatitis was most often explained by the link of hypercalcemia, most of the above theories have not presented scientific evidence, and the current challenge is to seek a direct link between these two pathologies, the genetic theory remains an option, but its role is not clear.

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CONFLICT OF INTEREST

We know of no conflicts of interest associated with this publication.

Ethical Approval

We declare that our work respects the ethical principles of medical publications.

Authors Contribution

AB carried out the bibliographic research, AAG and YE did the patient's medical follow-up. AH wrote initial and final draft of article. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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