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Anatomopathology

Acute Pancreatitis Revealing Primary Hyperparathyroidism Due to Unilateral Parathyroid Hyperplasia Associated with Parathyroid Adenoma

Fatima Ezzahra Mennani[1*], Eleazar Niyogushima[1], Sara Ijdda[1], Sana Rafi[1], Ghizlan El Mghari[1], Nawal El Ansari[1], Chafiaa Benallal[1], Hanane Rais[1]

[1]Department of Anatomopathology, Mohammed VI University Hospital Marrakech Morocco

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***Corresponding author:** Fatima Ezzahra Mennani
Department of Anatomopathology, Mohammed VI University Hospital Marrakech Morocco

Abstract **Case Report**

Primary hyperparathyroidism is a common endocrine pathology characterized by elevated levels of parathyroid hormone, the most common clinical manifestations being renal or skeletal damage accompanied by moderate or severe hypercalcemia. Around 80-85% of primary hyperparathyroidism cases are caused by a single parathyroid adenoma, 4-5% by multiple adenomas, 10-15% by parathyroid hyperplasia and less than 1% by parathyroid carcinoma. We report the case of a patient with primary hyperparathyroidism presenting with acute pancreatitis. After cooling of the pancreatitis, the patient underwent surgery. Post-operative follow-up showed definitive hypocalcemia, and pathological examination revealed the association of parathyroid hyperplasia and a unilateral parathyroid adenoma.

Keywords: Primary Parathyroidism, Hypercalcaemia, Parathyroidectomy, Parathyroid Adenoma, Parathyroid Hyperplasia, Pancreatitis.

INTRODUCTION

Primary hyperparathyroidism is a disorder of phosphocalcic metabolism linked to an inappropriate increase in parathyroid hormone (PTH) secretion. It is more common after the age of 50, and occurs more frequently in women. It is due to a parathyroid adenoma in around 80-85% of cases, and to hyperplasia of all four parathyroid glands in around 15% [1]. In this article, we review the diagnostic and therapeutic approach to primary hyperparathyroidism, based on a complex clinical case of parathyroid adenoma associated with unilateral parathyroid hyperplasia revealed by acute pancreatitis.

CASE REPORT

A 52-year-old hypertensive patient on dual therapy for 2 years, admitted to the emergency department with acute onset abdominal pain, was found to have chronic bone pain for 8 years, accentuated over the past 1 year and predominantly in the legs and back, a polyuro-polydipsic syndrome, digestive disorders such as chronic constipation and chronic epigastralgia, aggravated 3 days prior to admission. Clinical examination revealed a normotensive, normocardic patient with epigastric tenderness. Biological workup showed lipasemia 240 U/l, corrected calcium 126 mg/l, phosphorus 26 mg/l and PTH 3858 pg/ml.

Abdominal CT showed stage C pancreatitis. Abdominal MRI showed a hypotrophic pancreas with dilatation of the Wirsung with no visible obstruction, infiltration of the left pararenal fascia and fat in the posterior cavities of the epiplons. Cervical CT showed a lesion formation measuring 28× 24.5 mm at the inferior pole of the left parathyroid lobe, possibly related to a parathyroid adenoma. Parathyroid scintigraphy showed hyperfixation in the left lower parathyroid lobe.

After the pancreatitis had cooled down, the patient underwent surgery to resect the 2 left upper and lower parathyroids. Pathological examination showed a left parathyroid adenoma associated with a left upper parathyroid hyperplasia, with no sign of malignancy.

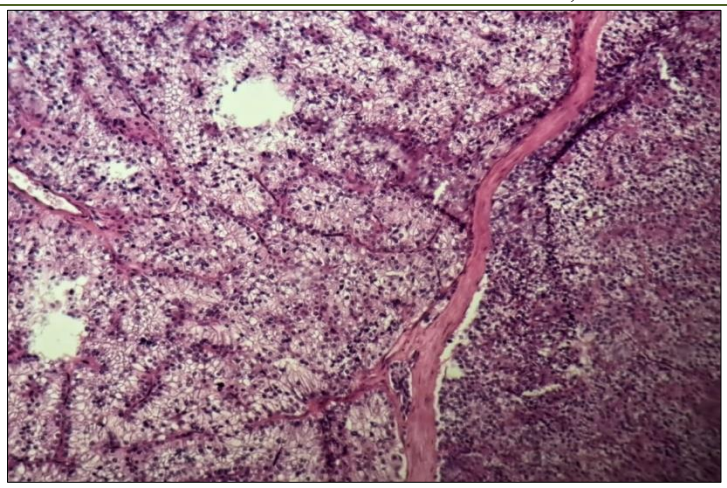

Figure 1: Left inferior parathyroid adenoma without capsular invasion or vascular emboli

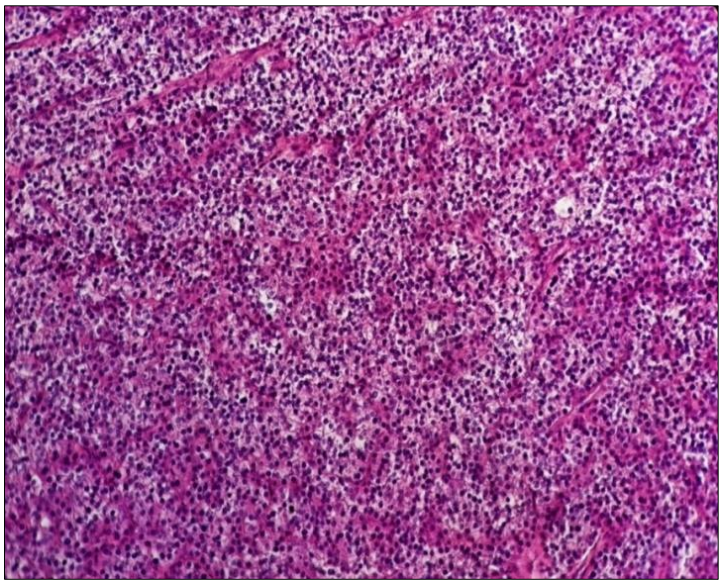

Figure 2: Left upper parathyroid hyperplasia without signs of malignancy

In the immediate post-operative period, the patient presented symptoms of hypocalcemia such as perioral and extremity paresthesias, muscle cramps, and Chvostek and Trousseau clinical signs were positive. Immediate postoperative calcemia was 73 mg/l and postoperative PTH was 71pg/ml. The patient was put on a protocol to correct acute hypocalcemia, with a favorable outcome.

DISCUSSION

Primary hyperparathyroidism is a common endocrine disorder affecting calcium metabolism due to hypersecretion of parathyroid hormone (PTH) by the parathyroid glands. Abnormal elevation of PTH leads to an increase in serum calcium [2]. The clinical manifestations of hyperparathyroidism are dominated by the general signs of hypercalcemia, such as asthenia and weight loss, bone signs, renal signs and, exceptionally, acute pancreatitis [8]. The most common cause of primary hyperparathyroidism is single parathyroid adenoma, accounting for 80-85% of cases, double adenomas for 4%, multiglandular hyperplasia for 10-15% and, more rarely, parathyroid carcinoma for less than 1% [3].

The prevalence of acute pancreatitis in hyperparathyroidism ranges from 1.5% to 13%. Such a low prevalence may be explained by the fact that pancreatitis occurs only in advanced stages of parathyroid disease. However, in our patient, acute pancreatitis was the first clinical manifestation [4, 5]. The association between primary hyperparathyroidism and acute pancreatitis is explained by an auto-activation of pancreatic enzymes such as trypsinogen in the pancreatic parenchyma by hypercalcemia, causing autodigestion of the pancreas with calcium deposition in the pancreatic duct, causing its obstruction, which in turn triggers enzyme activation [7-9].

Once primary hyperparathyroidism has been confirmed, the next step is to locate the abnormally secreting gland. Imaging methods include cervical ultrasound, Tc99m sestamibi imaging, single-photon

emission computed tomography (SPECT), magnetic imaging, cervical computed tomography and single-photon emission computed tomography (SPECT), magnetic resonance imaging (MRI), positron emission tomography combined with computed tomography (PET/CT) [6].

The treatment of choice is surgery; prior to surgery, it is essential to correct hypercalcemia with adequate hydration, forced calciuresis with loop diuretics and prevention of further bone resorption with bisphosphonates; hemodialysis may be a faster way to control hypercalcemia when forced calciuresis is not possible. This is coordinated simultaneously with the management of acute pancreatitis. In our case, hypercalcemia was corrected by rehydration, forced calciuresis and biphosphonates. At the same time, the acute episode of pancreatitis was managed conservatively, and definitive treatment by surgical excision was carried out once the patient's general condition had stabilized [10, 11].

CONCLUSION

Causes of pancreatitis are largely dominated by gallstones and alcohol. Primary hyperparathyroidism is exceptionally associated with the development of pancreatitis. It is important to check serum calcium levels for pancreatitis, and to consider pancreatitis in patients with primary hyperparathyroidism who present with abdominal symptoms. Combined medical and surgical treatment remains the cornerstone of the management of acute pancreatitis associated with primary hyperparathyroidism**.**

Competing Interests: The authors declare that there are no competing interests regarding the publication of this paper.

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