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Acute Pancreatitis Secondary to Primary Hyperparathyroidism: A Case Report and Literature Review

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Abstract: Acute pancreatitis may have a great variety of etiologies, but the two main causes, responsible of 80% of acute pancreatitis, are alcohol and biliary lithiasis. The other causes more rare, are drugs, genetic, malformative, cells tumor, medication and metabolic (hyperlipidemia, hypercalcaemia) hypercalcaemia secondary to primary hyperparathyroidism (P-HPT), is a rare cause of acute pancreatitis. We report à case of a patient whose initial presentation of the primary hyperparathyroidism was an acute pancreatitis.
Keywords: Acute pancreatitis, Hypercalcemia, Hyperparathyroidism.

INTRODUCTION

Acute pancreatitis is an inflammatory condition of the pancreas which is painful and at times deadly. The two main causes, responsible of 80% of acute pancreatitis, are alcohol and the biliary lithiasis. hypercalcaemia secondary to primary hyperparathyroidism (P-HPT), is a rare cause of acute pancreatitis, oscillating between 1.5 and 7% in the different series [1].

CASE PRESENTATION

A 35 year-old woman was in her usual state of health until four days prior to presentation when she began experiencing severe epigastric abdominal pain that radiates to the back, accompanied by nausea and vomiting. The pain improves when the patient leans forwards and worsens with deep inspiration and movement. she had no major medical history.

On physical examination: her heart rate was 93/min, blood pressure 120/60 mm/hg and body temperature 37C. There was tenderness and guarding in the upper abdomen, however, there was no lump or organomegaly and bowel sounds were sluggish. Examination of other systems did not reveal any abnormality. Her admission laboratory finding were:

- Hemoglobin: 12 g/dl; hematocrit: 40%.
- White blood count: 5430 (50% neutrophils).
- Glucose: 90 mg/dl; urea: 33 mg/dl; creatinine: 1.03 mg/dl.
- lipase: 528 U/L.
- Cholesterol: 138 mg/dl; triglycerides: 99 mg/dl.
- Total protein: 7 g/dl; albumin: 4.1 g/dl.
- Serum calcium: 10 mg/dl.

Acute pancreatitis was diagnosed based on clinical and biological data, and abdominal ultrasonography did not demonstrate any pathological finding. An abdominal CT scan showed an edematous, unstructured pancreas with no other findings.

As all these explorations were negative, elevated serum calcium levels were proposed as a potential etiology for this disease. New serum and urinary calcium and intact parathyroid hormone (iPTH) measurements were obtained: serum calcium: 12.5 mg/dl; urinary calcium 66 mg/dL iPTH: 951 pg/ml (normal < 65 pg/ml). Hypercalcemia due to hyperparathyroidism was thus confirmed. The patient was treated with bowel rest, hydratation and pain control ; She improved in the following days, with lipase level back to normal, correct oral feeding, and no abdominal pain. When the disease had disappeared completely imaging tests were requested. Cervical ultrasonography and scintigraphy with Tc-99 showed images consistent with a right lower parathyroid adenoma (Fig-1).

In september 2016, right lower parathyroidectomy was performed with selective access(Fig-2 and 3). During the procedure, immediately after parathyroidectomy, a significant decrease in iPTH levels was confirmed. Afterwards, histological study of the surgical piece confirmed that the mass was a parathyroid adenoma. After surgery serum calcium went back to normal, and the patient had no more episodes of abdominal pain during follow-up.

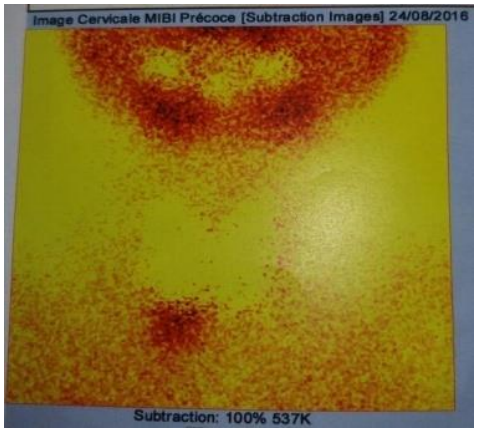

Fig-1: Tc-99m-sestambi scan showing hot focus in the inferior pole of right lobe of thyroid

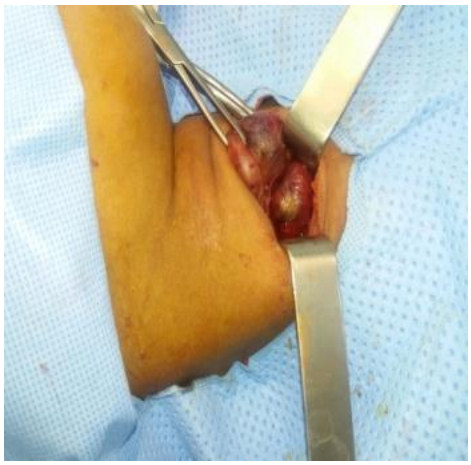

Fig-2: per operative aspect

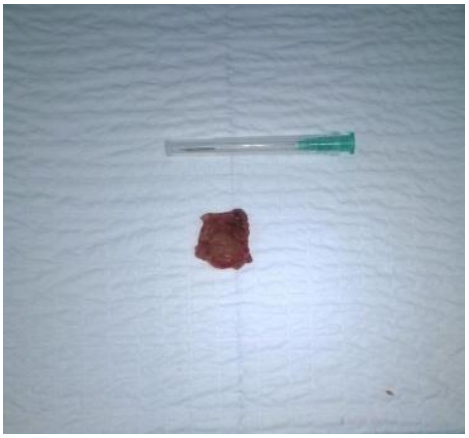

Fig-3: Parathyroid adenoma resected

DISCUSSIONS

Pancreatitis related to hyperparathyroidism (HPT) was first reported in 1947 by Martin and Canseco in the Journal of American Medical Association. But it was 10 years later that pancreatitis as a feature of primary HPT became well known through the writing of Cope in the Annals of Surgery in 1957 [2].

The pathophysiology has given rise to multiple controversies, which are still of topicality. It is currently admitted that pancreatitis is a consequence of the hyperparathyroidism, and not the contrary, as this had been mentioned in the1950s [3]. Two main hypotheses have been proposed to demonstrate the causal link : On the one hand, hypercalcemia could cause a local DIC causing necrotizing and haemorrhagic lesions, leading to pancreatitis ; On the other hand, hypercalcemia could lead to a higher concentration of ionized calcium in the pancreatic juice. The latter, by precipitating in this alkaline medium, could activate the transformation of inactive trypsinogen into active trypsin, and thus trigger a

autodigestion of the gland. These hypotheses are all the more plausible, since pancreatitis has been reported in patients with hypercalcemia secondary to multiple myeloma or vitamin D intoxication [4] even after parenteral nutrition, inducing hypercalcemia [5]. More recently, Felderbauer *et al*. Were suggestive of a genetic risk factor such as mutations of serine protease inhibitor Kasal type 1 (SPINK 1) and CFTR [6].

SPINK 1 is a natural inhibitor of trypsinogen and mutations of this gene have been reported in some pancreatitis , Mutations in the CFTR gene are responsible for cystic fibrosis. Some mild CFTR mutations are responsible for attenuated forms of the disease with moderate pancreatic involvement. It is admitted that such genetic mutations may be favorable to the development of acute pancreatitis in the presence of another triggering factor such as hypercalcemia [6].

The Primary Hyperparathyroidism is often diagnosed fortuitously in the presence of hypercalcaemia with increased parathyroid hormone (PTH) and the condition is mostly asymptomatic. Acute pancreatitis as a revealing cause of primary hyperparathyroidism is rarely reported in the literature.

The main causes of primary HPT are single or double parathyroid adenoma (80%), hyperplasia of all four or more existing parathyroid glands (15-20%) and rarely cancer (2%) of the parathyroid gland [7-9]. The topographic diagnosis uses conventional imaging (cervical ultrasound, CT scan or cervical MRI), and functional imaging (MIBI scintigraphy) for ectopic and multiple sites.

In our case, the parathyroid adenoma was easily seen on ultrasound and the diagnosis was supported by cervical scintigraphy.

Nowadays, surgical treatment is recommended more frequently for symptomatic patients with P-HPT. Parathyroid surgery in patients with P-HPT, whether symptomatic or not, generally allows normalization of calcium and PTH levels. Other therapeutic options may be considered in patients with asymptomatic P-HPT such as Selective Estrogen Receptor Modulators (SERM), bisphosphonates and calcimimetics [10].

CONCLUSION

P-HPT, which is a curable cause of acute pancreatitis, must be systematically sought after any acute pancreatitis, by dosing calcemia.

REFERENCES

1. Valenzuela JE, Segura EB, Torres AS, Alvarez FC. Acute pancreatitis associated with hypercalcemia. A report of two cases. Rev Esp Enferm Dig. 2009;101:65-9.
2. Abdullah M. Pancreatitis in primary hyperparathyroidism. Medical Journal of Malaysia. 2003 Oct 1;58(4):600-3.
3. Curto C, Caillard C, Desurmont T, Sebag F, Brunaud L, Kraimps JL, Hamy A, Mathonnet M, Bresler L, Henry JF, Mirallié E. Pancréatite aiguë et hyperparathyroïdie primaire: étude multicentrique de l'Association francophone de chirurgie endocrinienne. Journal de Chirurgie. 2009 Jun 30;146(3):270-4.
4. Knackstedt C, Winograd R, Koch A, Abuzahra F, Trautwein C, Wasmuth HE. Acute necrotic pancreatitis induced by severe hypercalcaemia due to tacalcitol ointment. British Journal of Dermatology. 2007 Mar 1;156(3):576-7.
5. Izsak EM, Shike M, Roulet M, Jeejeebhoy KN. Pancreatitis in association with hypercalcemia in patients receiving total parenteral nutrition. Gastroenterology. 1980 Sep 1;79(3):555-8.
6. Felderbauer P, Karakas E, Fendrich V, Bulut K, Horn T, Lebert R, Holland-Letz T, Schmitz F, Bartsch D, Schmidt WE. Pancreatitis risk in primary hyperparathyroidism: relation to mutations in the SPINK1 trypsin inhibitor (N34S) and the cystic fibrosis gene. The American journal of gastroenterology. 2008 Feb 1;103(2):368-74.
7. Ewing P, Hardy JD. The mediastinum. In: Baue AB, Geha AS,Hammond GL, Lab H, Nannheim KS, editors. Glenn's Thoracic and Cardiovascular Surgery. Cormecticut: Appleton and Lange. 1991; 1: 569-94.
8. Richards ML, Bondeson AG. Thompson NW. Mediastinal parathyroid adenomas and carcinomas. In: Shields TW, LoCicero ill I, Ponn RB, editors. General Thoracic Surgery. Philadelphia: Lippincot, Williams and Willkins. 2000; 2: 2383-90.
9. Fraker DL. Update on the management of parathyroid tumors. Current opinion in oncology. 2000 Jan 1;12(1):41-8.
10. Babey M, Kopp P. Hyperparathyroïdie primaire. InForum Med Suisse 2009; 9(44), 791-797).