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CLINICAL CASE

Acute pancreatitis associated with hypercalcaemia☆

Mauro Enrique Tun-Abraham*, Gabriela Obregón-Guerrero, Larry Romero-Espinoza, Javier Valencia-Jiménez

Servicio de Gastrocirugía, Hospital de Especialidades de Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México D.F., México

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KEYWORDS
Acute pancreatitis;
Hypercalcaemia;
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Abstract
Background: Hypercalcaemia due to primary hyperparathyroidism is a rare cause of acute pancreatitis, with a reported prevalence of 1.5–8%. There is no clear pathophysiological basis, but elevated parathyroid hormone and high serum calcium levels could be responsible for calcium deposit in the pancreatic ducts and activation of pancreatic enzymes, which may be the main risk factor for developing acute pancreatitis. The aim of this report is to describe four cases.
Clinical case: Four cases are reported of severe pancreatitis associated with hypercalcaemia secondary to primary hyperparathyroidism; three of them were with complications (two pseudocysts and one pancreatic necrosis). Cervical ultrasound, computed tomography, and scintigraphy, using 99mTc-Sestambi, studies showed the parathyroid adenoma. Surgical resection was the definitive treatment in all four cases. None of the patients had recurrent acute pancreatitis events during follow-up.
Conclusions: Acute pancreatitis secondary to hypercalcaemia of primary hyperparathyroidism is rare; however, when it occurs it is associated with severe pancreatitis. It is suspected in patients with elevated serum calcium and high parathyroid hormone levels. Imaging techniques, such as cervical ultrasound, computed tomography, and scintigraphy, using 99mTc-Sestambi, should be performed, to confirm clinical suspicion. Surgical resection is the definitive treatment with excellent results.

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* Corresponding author at: Servicio de Gastrocirugía, Hospital de Especialidades de Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social. Av. Cuauhtémoc, 3° Piso Colonia Doctores, Del. Cuauhtémoc, México D.F. C.P. 06725, México.
Tel.: +52 5627 6900 Ext 21436.
E-mail address: jlmo1968@hotmail.com (M.E. Tun-Abraham).

PALABRAS CLAVE
Pancreatitis aguda;
Hipercalcemia;
Hiperparatiroidismo primario

Pancreatitis aguda asociada con hipercalcemia

Resumen
Antecedentes: La hipercalcemia secundaria a hiperparatiroidismo primario es una causa rara de pancreatitis aguda, con una prevalencia documentada de 1.5 a 8%. La pérdida del mecanismo regulador de paratohormona y la hipercalcemia favorecen el depósito de calcio en los conductos pancreáticos y la activación de enzimas pancreáticas son probablemente los factores precipitantes. El objetivo de este informe es describir 4 casos.
Caso clínico: Cuatro pacientes con diagnóstico de pancreatitis aguda severa asociada con hipercalcemia secundaria a hiperparatiroidismo primario, tres de ellos con complicaciones asociadas a pancreatitis (dos con pseudoquiste y uno con necrosis pancreática). El ultrasonido de cuello, la tomografía de cuello y la gammagrafía con Tc-99 sestamibi confirmaron la presencia de un adenoma de paratiroides. La resección quirúrgica del adenoma fue el tratamiento definitivo, con excelentes resultados sin nuevos episodios de pancreatitis o hipercalcemia durante su seguimiento.
Conclusiones: La pancreatitis aguda por hipercalcemia secundaria a hiperparatiroidismo primario es infrecuente; sin embargo, cuando ocurre se asocia a pancreatitis severa. Su sospecha es en pacientes con elevación sostenida de calcio y paratohormona cuando se han descartado otras causas frecuentes. La confirmación diagnóstica se realiza con ultrasonido de cuello, tomografía de cuello o gammagrafía con sestamibi. La resección quirúrgica del adenoma es el tratamiento definitivo con resultados excelentes y cese de recurrencias de pancreatitis aguda.

Background

Primary hyperparathyroidism is a disorder of calcium metabolism that results from an abnormally high level of serum calcium, and an increase in parathyroid hormone. Primary hyperparathyroidism is the most common cause of hypercalcaemia. It can appear at any age; however, in most cases, it is reported over the age of 45, with predominance in females in the ratio of 2:1.[1]

Hypercalcaemia caused by primary hyperparathyroidism is a rare cause of acute pancreatitis, with a documented predominance of 1.5–8%. However, less than 1% of the patients who suffer from acute pancreatitis have primary hyperparathyroidism.[1,2] According to Egea,[1] the association between primary hyperparathyroidism and acute pancreatitis was described by Erdheim in 1903, while the association with chronic pancreatitis was published by Martin and Canseco in 1947. However, it was not until 1957 when Cope et al. considered it as another sign of primary hyperparathyroidism.[1,2]

Patients with primary hyperparathyroidism and hypercalcaemia have up to 10 times more risk of suffering from acute pancreatitis episodes (approximately 2%). The association between hypercalcaemia and certain genetic mutations (gene SPINK 1 (Kazal-type serine protease inhibitor type 1), CFTR (cystic fibrosis transmembrane conductance regulator gene) and CASR (calcium receptor gene)) could be responsible for this predisposition in some patients with hyperparathyroidism.[1–4]

Although the association between these entities is well described and many theories about their pathogenesis have been proposed, there are few references on acute pancreatitis cases associated with hypercalcaemia and hyperparathyroidism in medical literature. The most common scenario is when they appear in an already known hyperparathyroidism context or during a parathyroidectomy postoperative period; it is more infrequent as an initial sign of a patient suffering from hyperparathyroidism.[1–4]

We report four cases of acute pancreatitis caused by hypercalcaemia due to primary hyperparathyroidism that was not known previously.

Clinical cases

Case 1

A 28-year-old male patient with a history of bronchial asthma, diabetes mellitus and amygdalectomy during infancy. He presented with recurrent abdominal pain, which led to three hospital admissions where he was diagnosed with severe acute pancreatitis, with no aetiologic diagnosis. A pancreatic pseudocyst was documented during his last admission; he was sent to our unit for this reason. During physical examination, he was dehydrated and had abdominal pain located at the epigastrium. Laboratory reports at the time of admission showed: haemoglobin 12.1 g/d, leukocytes 13,000 μl (64.5% neutrophils), plasma amylase 108 U/l, lipase 199 U/l, glucose 209 mg/dl, calcium 14.2 mg/dl and phosphorus 2.0 mg/dl. The abdominal tomography and the endoscopic ultrasound reported a pseudocyst in the tail and head of the pancreas, which was drained endoscopically through a transgastric method. Patient evolved adequately and was discharged due to improvement (Fig. 1A).

He was readmitted five days later due to fever, anorexia, asthenia, adynamia, nausea, vomiting and abdominal pain at the epigastrium, which irradiated to the right hypochondrium. Laboratory analysis reported normal levels