PANCREATIC NEUROENDOCRINE TUMOR PRESENTING WITH MALIGNANT HYPERCALCEMIA

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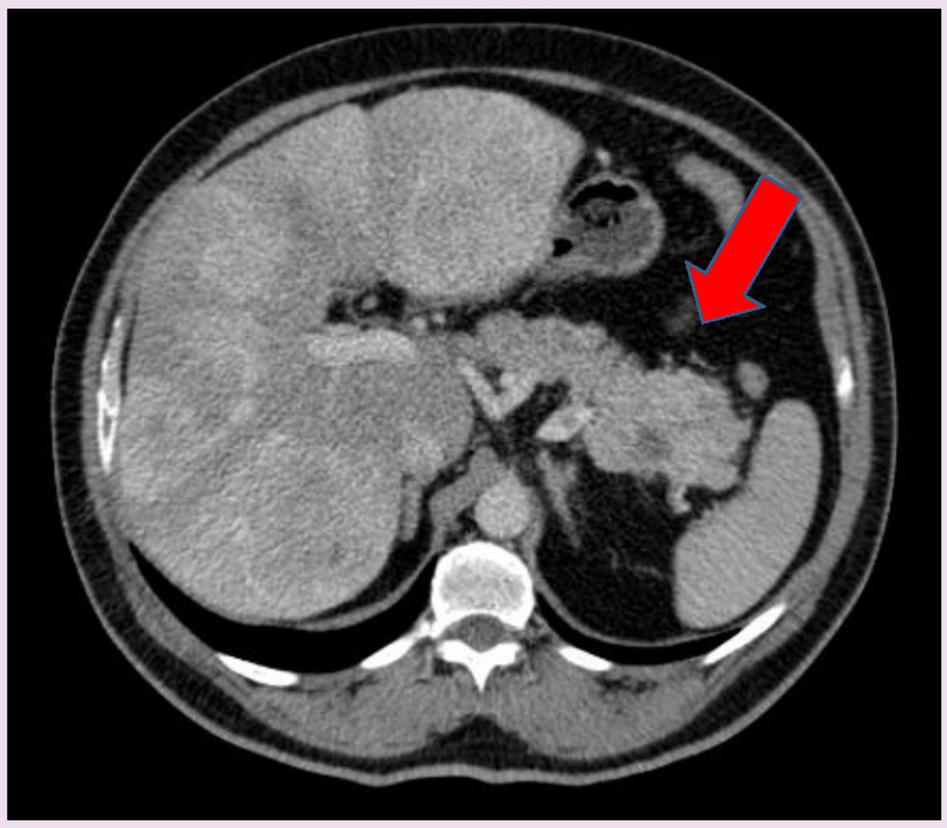
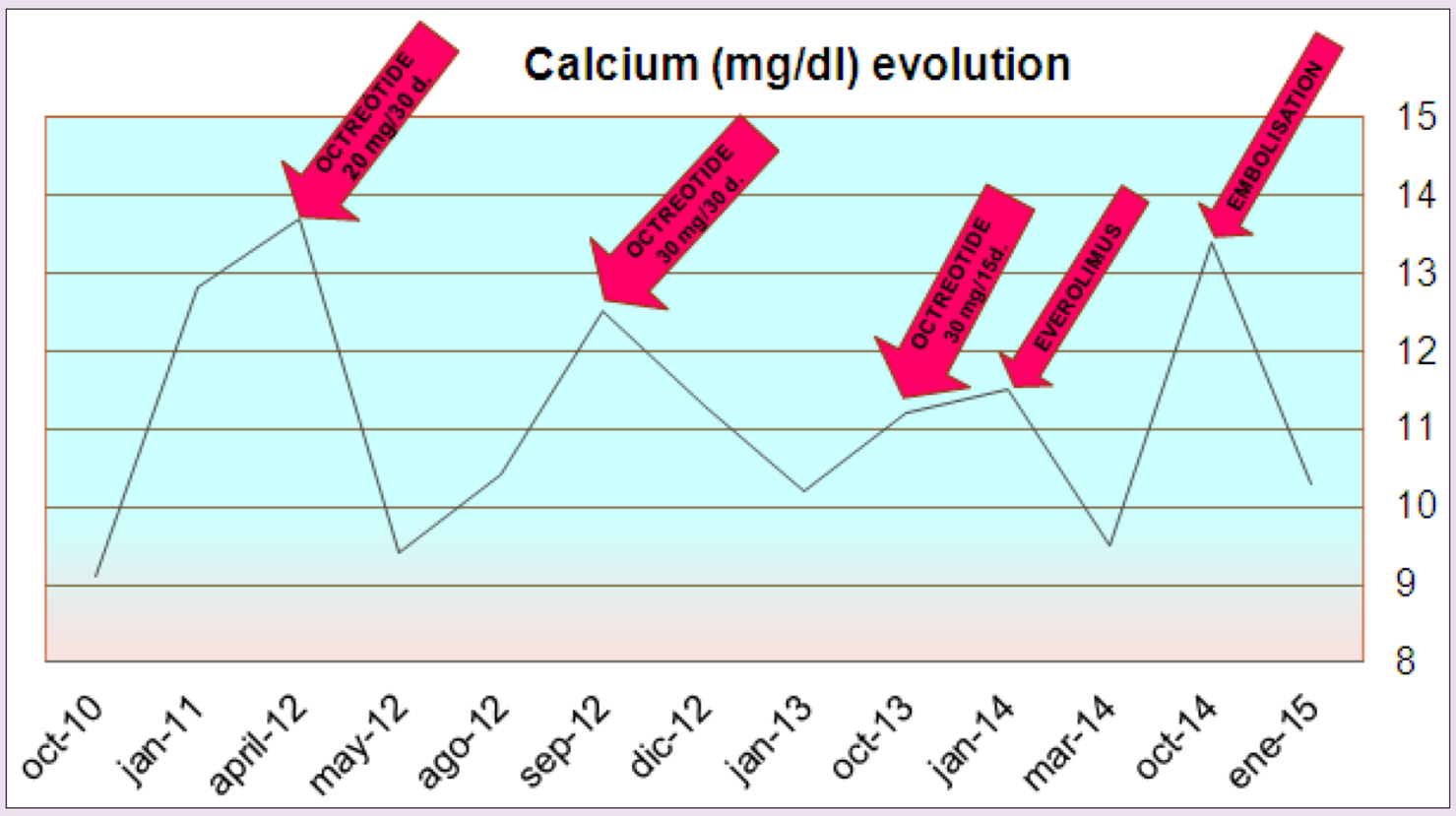
INTRODUCTION:

Although humoral hypercalcemia is one of the commonest paraneoplastic syndromes, and 5% of patients with malignant tumors may develop hypercalcemia, it has not been commonly described in patients with neuroendocrine tumors (NETs).

CASE REPORT:

A 55-yr-old man with personal history of hypertension, dyslipidemia and depression. He was referred to the outpatient for high levels of calcium in repeated assessments during the last year. He referred polyuria and polydipsia. Not clinical history of bone fractures, abdominal pain or another digestive symptoms. Initial biochemical assessment demonstrated: calcium 11,8 mg/dl (8,5-10,5), phosphate 2,4 mg/dl (2,5-4,5), creatinine 0,68 mg/dl, GGT 121 U/L, FA 182 U/L, iPTH 7 pg/ml (10-65), 25-OH-Vit D 8,78 ng/ml (>20), 1,25-OH-Vit D 98,8 pg/ml (16-56), ECA 26 U/L (8-52), calciuria 545 mg/per day (<400), chromogranin A 152 ng/ml (<100), PTHrp 1,5 pmol/l (<1). Proteinogram and bone scintigraphy negative. Abdominal CT revealed a 6.6-cm lobular mass at the tail of the pancreas with collateral circulation and multiple focal liver lesions, which occupied 70% of the hepatic parenchyma. Somatostatin receptor scintigraphy exhibited multiple lesions that overexpress somatostatin receptors suggestive of multiple hepatic metastases from neuroendocrine tumor, without pancreatic uptake. Percutaneous FNA (guided by endoscopy) of the pancreatic lesion revealed a well-differentiated NET. The diagnostic was parathyroid hormone-related peptide-secreting pancreatic neuroendocrine tumor. Initially, octreotide long-acting release (LAR) 20mg every 28 days was administrated and calcium levels decreased from 13,7 to 9,4 mg/dl in 3 months. After that, calcium levels increased again (12.2 mg/dl) so we increased octreotide LAR 30 mg every 28 days, and then 30 mg every 15 days; but due to biochemical progression everolimus treatment was initiated (20 months after octreotide treatment). After that, hypercalcaemia resolved and she remained clinically stable for a period of 10 months, but in october 2014 hypercalcaemia appeared again, so hepatic embolisation was used as therapy with the aim of reducing calcium. After 3 years treatment, there have been no radiological progression but biochemical deterioration, and hypercalcaemia has recurred many times despite medical treatment. Nowadays the patient is clinically asymptomatic and calcium levels are normal after last embolisation.





CONCLUSIONS:

The manifestation of paraneoplastic syndrome due to PTHrp hypersecretion, despite its rareness in NET, should be considered in the differential diagnosis of hypercalcemia in such tumors.

Upon reviewing the literature, there are just a few case reports describing PTHrp secretion occurring in pancreatic NETs.

Biochemical response following administration of octreotide has been documented with evidence of normalisation of calcium , but the duration of this effect is unclear.

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