Case report

Hypercalcemia, necrotizing pancreatitis and bone lesions: a benign cause

In August 2016 a 23-year-old patient was referred to our intermediate care station. He had presented in an affiliated clinic three days earlier for abdominal pain and vomiting that had developed over several days. His medical record did not show any significant comorbidities. Initial laboratory studies showed significant hypercalcemia of 3.7 mmol/l, lipase of 1342 U/l and leukocytosis of 18/nl. Abdominal sonography showed no cholestasis but perihepatic and perisplenic free liquid. Abdominal computer tomography at the day of admission showed non-enhancing low attenuating regions within the pancreas corpus an tail and peripancreatic fluid collection. Diagnosis of necrotizing pancreatitis was done (Figure 1).

The CT scan also revealed multiple osteolytic bone lesions in his pelvis of which one conferred relative pelvic instability (Figure 2).

Malignant hypercalcemia was suspected and subsequently the patient was referred to a tertiary care center. Intravenous bisphosphonate was administered for hypercalcemia. However subsequent laboratory studies showed a parathyroid hormone (PTH) level of 810 ng/l and low 25-hydroxy-Vitamin D2 levels of 9.7 ug/l, tumor markers (CEA, CA19-9, AFP) were negative. Primary parathyroidism was suspected and calcitonin administered, subsequently hypercalcemia resolved. On thoracic CT imaging an osteolytic lesion and pathological fracture was identified in his right humerus head. His right arm was immobilized in a Gilchrist sling. Thyroid sonography showed an enlarged parathyroidal le-
Surgical parathyroidectomy was performed and confirmed the specimen to be consistent with parathyroid adenoma. We refrained from performing a biopsy of the bone lesions because a brown tumor was clinically and radiographically highly likely. Our final diagnosis was brown tumors associated with long standing primary hyperparathyroidism and necrotizing pancreatitis due to hypercalcemia.

Discussion and conclusion

PHPT is a known cause of osteitis fibrosa cystica. Brown tumors are caused by excess osteoclast activity combined with demineralized fibrous bone tissue resulting in its brown appearance (1). Its development is rare in mild PHPT and associated with parathyroid carcinoma (2). Dissolution of bone structure and cessation of bone production results in a release of stored calcium leading to serum hypercalcemia. Accompanying low Vitamin-D levels are associated with higher rates of bone resorption and fracture risk (3). PHPT has been linked to pancreatitis in several retrospective studies even though its link remains controversial (4). Pancreatitis risk has been related to serum calcium, but data remains conflicting (5). Calcium serum level might predispose pancreatitis due to activation of pancreatic calcium sensors (6). Prognosis of pancreatitis has been described as benign compared to alcoholic or biliary cause (4).

We present a patient with osteolytic brown tumors as a result of PHPT and necrotizing pancreatitis due to accompanying hypercalcemia. Our patient was initially misdiagnosed for malignant osteolytic bone lesions. Even though usually presenting as a mild disease (7) PHPT can result in significant morbidity in patients affected. During follow-up the patient's symptoms resolved and we saw a remineralization of bone lesions.

References