Parathyroid Calcification as a Complication of Secondary Hyperparathyroidism

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Dear Sir,

Metabolic consequences of chronic irreversible renal failure include hyperphosphatemia, hypocalcemia, and secondary hyperparathyroidism. Persistent hyperphosphatemia not only contributes to abnormal vitamin D balance, but is also responsible for metastatic calcification. The following is an example of such a perturbation.

A 40-year-old white male with end-stage renal disease had been on maintenance hemodialysis for 8 years. Renal disease was secondary to radiation nephritis following therapy for testicular neoplasia. His course on hemodialysis was complicated by severe, but controllable hypertension, severe hyperphosphatemia and hypocalcemia, despite the use of oral and intravenous phosphate binders. He also had secondary hyperparathyroidism as evidenced by elevated alkaline phosphatase (fig. 1), and a serum parathyroid hormone level of 100 pmol/l drawn 2 months prior to admission to the hospital (normal less than 6.1, PTH bioassay, Mayo Clinic, Rochester, Minn.). Renal osteodystrophy was diagnosed on the basis of erosive lesions on radiographs of hands, 'Rugger-Jersey' spine and multiple rib fractures.

Physical examination revealed a pale, young male with blood pressure of 148/90. There was calcification at sclerocorneal junction, neck was supple without adeno-

Parathyroidectomy

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Fig. 1. Profile of serum calcium, phosphorus and alkaline phosphatase through the observation period.

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Fig. 2. Photomicrograph of excised parathyroid tissue showing acini (open arrow) and subcapsular calcification (closed arrow).

pathy or palpable mass, cardiac examination revealed a murmur consistent with mitral insufficiency. Soft-tissue calcification was apparent in the second digits of both hands. Pertinent laboratory testing revealed hemoglobin 7.3 g/dl, microcytosis, BUN 95 mg/dl, creatinine 14.2 mg/dl, calcium 9.3 mg/dl, phosphorus 7.8 mg/dl. Chest x-ray showed calcification in acromioclavicular and sterno-clavicular joints.

The patient underwent parathyroidectomy with auto-transplantation of a part of parathyroid tissue into left forearm. Pathologic examination showed four enlarged hyperplastic parathyroid glands, one with calcification (fig. 2). Postoperatively, an expected fall in serum calcium was treated with oral and intravenous calcium supplementation and 1,25-dihydroxy vitamin D₃. Uremic soft-tissue calcification is a well-described complication of chronic renal failure, occurring in 38–81% of chronic dialysis patients [1, 2]. Visceral organ calcification has been described [3] involving heart, lungs, stomach and kidneys. The relationship between duration of dialysis, serum calcium phosphate product and soft-tissue calcification is unclear. Kuzella et al. [4] found no relationship between patient age, presence of metabolic bone disease, radiographic findings and serum calcium phosphate product and the incidence or severity of soft-tissue calcification. Others have reported a high incidence of metastatic calcification when serum calcium phosphate product exceeds 70 mg/dl [5]. The patient described here manifested evidence of soft-tissue calcification as well as bony changes of renal osteodystrophy. Persistent hyperphosphatemia resulting from advanced renal disease and dietary noncompliance was present with elevated serum calcium phosphate product over 3.5-year period. Lineaweaver et al. [6], using CT imaging of hypercalcemic patients originally described calcified parathyroid tissue, being present in parathyroid adenoma as well as adenocarcinoma. A chronic hemodialysis patient described by Krishna et al. [7] who had parathyroid carcinoma did not have parathyroid calcification. The case we are describing is unique in the sense that calcification of parathyroid glands when it occurs in the context of end-stage renal disease is a complication of metastatic calcification and does not suggest carcinoma.

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