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Orthopedic Consequences of Uncontrolled Tertiary Hyperparathyroidism

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Introduction

- Secondary hyperparathyroidism (SHPT) is common in chronic kidney disease.
- When the kidneys can no longer adequately convert vitamin D3 into its active form (1,25 dihydroxy-vitamin D4), calcium and phosphorus homeostasis is disrupted. Consequently, there is an increase in parathyroid hormone (PTH) secretion. PTH via RANK ligand indirectly stimulates osteoclasts to reabsorb bone in an effort to maintain calcium levels.
- When left untreated in chronic dialysis patients, SHPT can lead to metabolic bone disease such as osteomalacia (vitamin D deficiency) and osteitis fibrosa cystica (bone demineralization).
- Advanced SHPT can transition to tertiary HPT based on hypertrophic glands that become recalcitrant to optimized medical therapy.
- In this report we present the orthopedic consequences of a young, non-adherent patient with dialysis-dependent chronic kidney disease and tertiary hyperparathyroidism.

Case

- A 26-year-old male with peritoneal dialysis-dependent end-stage renal disease.
- The patient presented at 20-years-old with vague abdominal pain. Evaluation demonstrated a serum creatinine 8.0 mg/dl.
- The etiology of his renal failure was consistent with congenital abnormality of the kidneys and urinary tract.
- Renal ultrasound demonstrated an absent right kidney and atrophic left kidney. His PTH level was elevated at 1844 pg/ml (normal= 14 to 65 pg/mL) and ranged consistently between 2,000 and 5,000 during the past three years.
- The patient was otherwise healthy with a normal physical exam. During the past year the patient has developed severe renal osteodystrophy and atraumatic fractures.
- He developed bilateral patellar fractures after a ground level fall. He subsequently developed atraumatic bilateral pubic ramus fractures. Knee, pelvis and spine radiographs demonstrated abnormal density of visualized bony structures in addition to cortical thickening.
- Progressive, well-demarcated lucencies are seen throughout the femurs. Spine films demonstrate sclerotic areas along the endplates from all visualized vertebral bodies.

Figure 1.AP Pelvis demonstrating atraumatic bilateral pubic ramus fractures, Brown tumors

Figure 2. Ap Spine demonstrating “rugger jersey spine”

Discussion

- Although hyperparathyroidism is common in advanced kidney disease, the dramatic consequences seen in this report are rare. Adherence to dialysis and medical therapy was suboptimal since end stage kidney disease was diagnosed.
- Treatment includes vitamin D sterols (calcitriol), calcimimetic compounds (cinacalcet) and phosphorus control. Calcimimetics increase the sensitivity of calcium sensing receptors and reduce excess PTH secretion without raising calcium levels.
- The patient consistently declined surgical intervention and underwent unsuccessful attempts at alcohol ablation of the parathyroid glands.
- Our patient demonstrates a variety of the orthopedic consequences seen in uncontrolled hyperparathyroidism of renal origin. Findings include atraumatic factures, looser zones (transverse lucencies traveling part way through a bone), Brown tumors (focal demineralization of bone), and “rugger jersey spine” which describes prominent endplate densities at contiguous vertebral levels producing an alternating sclerotic-lucent-sclerotic appearance.

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