Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Prolonged Hypercalcemia After Recovery From Severe Rhabdomyolysis and Acute Renal Failure

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Introduction: Rhabdomyolysis is characterized by elevated creatine kinase, electrolyte abnormalities and acute renal failure. After the initial hypocalcemic phase, serum calcium levels can normalize or become significantly elevated during the recovery phase. We report a case of persistent hypercalcemia during the recovery phase of rhabdomyolysis-induced acute renal failure. **Case Description:** A 26-year old woman with Cornelia de Lange syndrome initially presented with septic shock, complicated by severe rhabdomyolysis and acute renal failure requiring hemodialysis. On admission, creatine kinase level was >30,500 U/L [29–168], BUN 95 mg/dL [6–22], Creatinine 3.39 mg/dL [0.57–1.11], and Corrected Calcium 7.1 mg/dL [8.6–10.2]. Approximately 1 month after admission, the patient developed hypercalcemia with corrected calcium values ranging from 10.4 to 14.5 mg/dL with PTH 10.5 pg/mL [8.7–77.1] and 1,25-Dihydroxyvitamin D and 25-OH Vitamin D levels <8.0 pg/dL [18–78] and 21.20 ng/mL [30–80], respectively. Her renal function improved and dialysis was discontinued seven weeks after initial presentation, although her GFR remained impaired (22 mL/min/BSA). She remained hypercalcemic and was treated with intermittent doses of Calcitonin when the level rose above 12.0 mg/dL. Spot urinary calcium to creatinine ratio was low at 0.13. Improvement in her calcium was seen with a level of 10.8 mg/dL eleven weeks after she was initially noted to be hypercalcemic and she was able to be discharged. Two weeks after discharge, an outpatient calcium improved further to 10.4 mg/dL.
Discussion: Rhabdomyolysis is a clinical syndrome that results from severe muscle damage with release of the breakdown products from injured muscle cells leading to acute renal failure and electrolyte abnormalities. The initial hypocalcemic phase is due to entry of calcium into damaged myocytes and deposition of calcium salts in damaged muscle. After recovery, most patients have normalization of calcium levels. However, in up to one-third of patients, a rebound hypercalcemia can ensue. The proposed mechanism is massive muscle calcium release during renal recovery. In the cases reported, the hypercalcemia phase resolves by an average of 8 days with the longest reported recovery being 3.5 months. We report a case of prolonged hypercalcemia in the setting of continued renal impairment. It appears that the patient's low GFR impaired urinary calcium excretion resulting in persistent hypercalcemia which eventually resolved over time with some improvement in renal function. Other case reports have described treatment with IV fluids, Calcitonin, Pamidronate and hemodialysis, although the efficacy of these interventions is unclear. Rhabdomyolysis is an under-recognized cause of hypercalcemia. Our case highlights the potential for prolonged hypercalcemia after recovery from rhabdomyolysis in the setting of renal impairment.

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Prolonged Hypocalcemia After a Single Dose of Denosumab in Chronic Kidney Disease

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Introduction: Denosumab, a monoclonal antibody that inhibits RANK L (receptor activator nuclear factor-kappa beta ligand), is one of the few medications that can be used to treat osteoporosis in patients with chronic kidney disease (CKD). However, its use is associated with a much higher incidence of hypocalcemia in this patient population. What remains unclear is the duration of hypocalcemia after denosumab use. We describe a case of prolonged hypocalcemia of 9 months in a patient with CKD after a single dose of denosumab.
Case: A 64-year-old Caucasian man with a history of bilateral lung transplant for interstitial pulmonary fibrosis and CKD Stage IV was referred to the Endocrinology clinic for evaluation of steroid-induced osteoporosis. Bone density scan was consistent with osteoporosis with the lowest T-score of -2.8 at the left femoral neck, which showed a 25.3% decline from a previous one two years prior. His labs upon initial visit: 25 hydroxy Vitamin D: 36.5 ng/mL (30–100), 1, 25 hydroxy vitamin D 32 pg/ml (19.9–79.3), corrected Serum Calcium 8.9 mg/dL (8.5–10.5), Serum Cr 4.38 mg/dL (0.6–1.4), PTH 157 pg/mL (10–65), Serum Alkaline Phosphatase 61 Units/L (25–125), Urine NTX 39 nM BCE/mM creatinine (21–83). After discussing risks and benefits, he was given a dose of subcutaneous denosumab 60 mg. He had been started on Calcium/Vitamin D (600 mg/400 IU BID) prior to receiving his dose. Keeping in mind the increased risk of hypocalcemia given his history of CKD, his corrected serum calcium was checked one week later, and it was 6.5 mg/dL. The patient was asymptomatic. However, given the severity of his hypocalcemia, he was started on calcitriol 0.25 mcg oral BID and calcium carbonate 1200 mg daily. He did show mild improvement in three days to a corrected calcium of 7.0 mg/dL. His calcitriol was briefly increased to 0.5 mcg BID and calcium carbonate was increased to 1800 mg daily. The regimen was weaned to calcitriol 0.25 mcg daily and previous calcium/Vitamin D dosing later that month. Thereafter, his labs were monitored regularly and there were several unsuccessful attempts made to decrease the calcitriol/calcium carbonate. Given persistent hypocalcemia, other bloodwork including a bone specific alkaline phosphatase and celiac screen were checked which were unremarkable. Finally, nine months after his denosumab dose, calcitriol was discontinued safely. Serum calcium levels have remained stable thereafter. Given prolonged hypocalcemia, it was decided not to administer another dose of denosumab.
Conclusion: Patients with CKD who receive denosumab are not only at risk for developing severe, but also prolonged hypocalcemia. Therefore, it is imperative to monitor serum calcium levels, not only immediately after receiving a dose, but serially.