Bisphosphonates: To Give or Not to Give, that is the Question – A case of prolonged hypercalcemia in a patient with severe rhabdomyolysis complicated by bilateral foot drop

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Rhabdomyolysis has an initial oliguric stage characterized by hypocalcemia and a recovery stage characterized by hypercalcemia. The latter can be severe, and cause altered mental status, arrhythmias and even death. Immobilization can also contribute to hypercalcemia, as in this patient with severe rhabdomyolysis with complication of bilateral foot drop and prolonged hypercalcemia after renal function recovered. We present a 19-year-old male with history of sickle cell trait and G6PD deficiency, admitted to ICU for acute renal failure and hemolytic crisis after intense exercise training. Labs were pertinent for: K 6.9 mmol/L (n=3.5-4.7), lactate 34.8 mmol/L (n=0.5-2.2), phosphorus 17.5 mg/dL (n=2.5-4.9), CPK 5420 U/L (n=39-308), magnesium 5.0 mg/dL (n=1.7-2.4), calcium 5.3 mg/dL (n=8.5-10.1), albumin 3.1 g/dL (n=3.4-5), ionized calcium 0.86 mmol/L (n=1.13-1.32), Cr 1.9 mg/dL (n=0.67-1.17), AST 15064 U/L (n=10-37), ALT 3687 U/L (n=10-65), myoglobin 240000 mcg/L (n=<95). Creatine peaked to 7.9 mg/dL. He required 8 weeks of hemodialysis, multiple pRBC transfusions, calcium supplementation, and high dose steroids. Once off hemodialysis, he steadily became hypercalcemic over the span of 40 days (10.1-11.9 mg/dL). Endocrinology was consulted: PTH was 2 pg/mL (n=18.4-80.1); Calciotriol <8 pg/ml (n=18-72); 25-OH vit D <4.2 ng/mL (n=30-100). IVFs were started and the cause of hypercalcemia was thought to be calcium release from recovering muscles. Notably, patient had bilateral foot drop from rhabdomyolysis-associated muscle edema and mobility was limited. Due to prolonged hypercalcemia of >30 days, which became worse after IVFs were discontinued (peaked to 12.9 mg/dL corrected calcium), endocrinology was consulted: A whole body bone scan showed stress-related bone changes but no pockets of calcium deposit in muscle. C-telopeptide was 1603 pg/mL (n=87-1200). Immobilization-related hypercalcemia was then considered, and IVFs were restarted. As calcium remained <12 after discontinuing IVFs for one week and as patient was participating in frequent physical therapy, it was decided to hold off on bisphosphonate therapy. Rare causes of hypercalcemia were also excluded [IGF-1 240 ng/mL (n=10-548), AM-cortisol 12.92 ug/dL (n=5.3-22.5), ACTH 42 pg/mL (n=0-47), PTHrP 13 pg/mL (n=11-20)]. In rhabdomyolysis, cell death due to various stress insults (eg crush injury, ischemia, infection) causes release of phosphorus contributing to initial hypocalcemia from formation of calcium phosphate deposits. In the recovery phase of AKI, these deposits mobilize from muscle causing hypercalcemia. The average duration of hypercalcemia phase is 10 days. However, in our case the patient had prolonged hypercalcemia of more than one month, which caused concern for immobilization hypercalcemia. A bone scan was helpful in differentiating between the two entities as rhabdomyolysis-related hypercalcemia may have revealed calcium pockets in muscle. The elevated C-telopeptide also reinforced this diagnosis.

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