SHORT COMMUNICATION

ACUTE SEVERE HYPERCALCEMIA AFTER TRAUMATIC FRACTURE AND IMMOBILIZATION IN AN ADULT WITH HYPOPHOSPHATASIA COMPLICATED BY RENAL FAILURE

Whyte MP,1,2 Leelawattana R,1 Reinus WR,3 Novack DV1,4

1Division of Bone and Mineral Diseases, Washington University School of Medicine at Barnes-Jewish Hospital; St. Louis, MO, 63110, USA 2Center for Metabolic Bone Disease and Molecular Research, Shriners Hospital for Children; St. Louis, MO, 63131, USA 3Department of Musculoskeletal Radiology, Temple University School of Medicine; Philadelphia, PA, 19140, USA 4Department of Pathology, Washington University School of Medicine at Barnes-Jewish Hospital; St. Louis, MO, 63110, USA

Calcium homeostasis is tightly regulated by the direct or indirect effects of parathyroid hormone (PTH) and biologically active vitamin D (calcitriol) on the skeleton, gastrointestinal tract, and kidney. We describe a 55-year-old man with hypophosphatasia (HPP) together with adultonset chronic kidney failure necessitating hemodialysis who developed acute and severe hypercalcemia after traumatic fracture and immobilization. HPP features impaired skeletal mineralization from endogenous accumulation of inorganic pyrophosphate (PPi), an inhibitor of mineralization, due to deficient activity of the “tissue-nonspecific” isoenzyme of alkaline phosphatase (TNSALP). Severely affected infants and young children with HPP can develop hypercalcemia and have hyperphosphatemia from this block in mineralization. Our patient manifested signs and symptoms of HPP in early childhood, and had osteomalacia documented by bone histopathology in adult life. Renal failure in middle-age was attributed to hypertension and NSAID use. Compliance for oral calcium carbonate to bind dietary inorganic phosphate (Pi) was poor, and he had not taken aluminum hydroxide or vitamin D supplementation. Before acute immobilization, serum calcium ranged from 8.4 - 10.7 mg/dl (8.6 - 10.3 Nl), Pi 5.8 - 6.4 mg/dl (2.5 - 4.5 Nl), and intact PTH 63 - 75 pg/ml (10 - 55 Nl). Then, a fall fractured multiple large bones. After several hours, he became confused. Serum calcium was 14.9 mg/dl, ionized calcium 7.4 mg/dl (4.5 - 5.1 Nl), PTH 16 pg/ml, and calcitriol 18 pg/ml (15 - 60 Nl). Hemodialysis rapidly corrected his hypercalcemia and confusion. The unique osteomalacia of HPP represents a type of inactive skeletal remodeling. Our HPP patient demonstrated that sufficient calcium can nevertheless emerge from the HPP skeleton during acute immobilization to cause sudden hypercalcemia if renal excretion cannot compensate for the mineral efflux.