Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Radiotherapy-Associated Pelvic Insufficiency Fracture Treated by Romosozumab: Course of L1 and L5 Vertebral Body CT Attenuation

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Background: Radiotherapy is a risk factor for osteoporosis and insufficiency fractures via osteoblast apoptosis and vascular injury. PTH analogs teriparatide and abaloparatide are contraindicated in patients with prior exposure to radiotherapy crossing bone due to the increased risk of osteosarcoma. Patients with radiotherapy-associated fractures or osteoporosis were eligible only for antiresorptive agents until romosozumab was recently FDA-approved. Current International Society for Clinical Densitometry (ISCD) guidelines include assessment of "opportunistic CT" as a surrogate for DXA scan using L1 vertebral body attenuation: >150 Hounsfield units (HU) is normal and <100 HU signifies osteoporosis.
Clinical Case: A 60 year old female patient with history of endometrial cancer diagnosed at age of 57 and treated with hysterectomy and bilateral salpingo-oophorectomy, chemotherapy, then pelvic radiotherapy, was referred to endocrinology for pelvic insufficiency fractures evaluation. Two years after completing chemoradiotherapy, she complained of right groin and low back pain with difficulty walking. MRI pelvis showed bilateral sacral ala and right pubic ramus insufficiency fractures. She had normal serum mineral concentration, 25-OH vitamin D sufficiency, normal PTH, eGFR, liver function tests and 24-hour urine calcium excretion. Screening for celiac disease and multiple myeloma was negative. DXA scan BMD T-score showed osteoporosis, -3.0 at the right femoral neck. L1-L4 T-score was +0.4 but unreliable due to presence of degenerative changes. Four months after onset of pain, patient started romosozumab 210mg SQ monthly for a total of 12 doses, after which she started oral alendronate. Pain essentially resolved within 6 months of romosozumab therapy. C-telopeptide (CTX) and procollagen type 1 N-terminal propeptide (P1NP) were obtained at baseline, 3 and 12 months after romosozumab initiation. CTX was 362, 247 and 258 pg/mL (reference range, >49 years: not established), and P1NP was 82, 178 and 62 mcg/L (reference range, 20 - 108), respectively. Attenuation of L1 and L5 vertebral body was measured using CT abdomen and pelvis scans before and 5 months after radiotherapy, and before and after completion of romosozumab therapy. L1 attenuation measured 161, 132, 127 and 179 HU, and L5 measured 150, 46, 50 and 86 HU, respectively.
Conclusion: Pelvic radiotherapy was associated with a decline in L1 CT attenuation and even greater magnitude of decrease at L5. Romosozumab was associated with clinical improvement, restoration of L1 CT attenuation and diminishment of regain at L5. Although L5 attenuation has not been previously assessed for osteoporosis, this site may be of predictive value in patients who receive pelvic radiotherapy.

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Rare Case of Severe Hypocalcemia Due to PTH Resistance Related to Diet Pill

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Introduction: The production of parathyroid hormone (PTH) is essential for calcium the maintenance of normal mineral metabolism. Parathyroid cells have cell-surface calciumsensing receptors, even small changes in extracellular Ca induce rapid changes in PTH secretion. Hypocalcaemia is a well-recognized manifestation of magnesium deficiency. We present rare case of severe hypocalcemia due to PTH resistance caused by hypomagnesemia related to diet pill. **Case Report:** 61 year old Hispanic obese female with chronic gastritis on omeprazole 20 mg daily, no other significant past medical and surgical history, not taking any prescribed medications presents to ED with complaints of tingling and numbness around the mouth, abdominal cramps, chest pain, shortness of breath and anxiety. On arrival calcium was found to be 6.0mg/dl(8.2–10.2mg/dl) with ionized calcium of 0.60mmol/l(1.13-1.32mmol/l) and EGFR>90ml/min, Albumin-4.0g/l, Magnesium-1.1mg/dl(1.6–2.3mg/dl), Phosphorus-6.1mg/dl(2.4–4.5mg/dl),rest of the electrolytes were normal. Patient was given IV calcium gluconate 2g and magnesium which helped improving her symptoms. PTH was 1700pg/ml(23-73pg/ml), low Vitamin D 25-hydroxy 20ng/dl(30-100ng/dl). Urinary calcium was <1mg/dl(2.0–17.5mg/dl). Vitamin D1,25- dihydroxy 34pg/ml(18-78pg/ml), PTH-like peptide levels 0.6pmol/l(<4.2pmol/l). EKG was normal no QT interval changes. For 4 weekspatient was taking weight loss medication was given to her by her brother called nucific-bio-x4. Patient had lost 4 pounds while taking the medication and had suppression of appetite. Physical features of pseudohypoparathyroidism were not seen. Patient had poor dietary intake of calcium, denied taking vitamin D supplements. Patient was given calcium acetate 2001 mg TID with meals, calcitriol 0.5mcg daily and calcium gluconate 2g IV intermittently was given. That improved her calcium levels to 6.8mg/dl with ionized calcium-0.90mmol/l. Magnesium was replaced IV and discharged on magnesium oxide 400mg BID to maintain magnesium the normal range. The patient was given loading dose of ergocalciferol 50,000 IUfor 8 weeks. At the time of discharge repeat PTH levels were 1600 pg/ml and calcium levels were 8.2mg/dl. 3D CT of the neck did not parathyroid mass. After discharge patient continued on oral calcium and magnesium to maintain calcium levels between 8.4–10.2mg/dl. After discharge calcium levels were 8.6mg/dl, vitamin D 25-hydroxy levels were 32ng/dl and maintenance dose of vitamin D3 2000 IU daily also continued. Nucific-bio-x4 pill was discontinued. **Conclusion:** In conclusion, severe life threatening hypocalcemia can occur with unsupervised weight lossmedications due to malabsorption of magnesium in patient with likely mutation in magnesium receptors causing PTH resistance. Vitamin D deficiency worsens hypocalcemia resulting into secondary hyperparathyroidism.