16.7), anemia, history of hypovitaminosis D, secondary hyperparathyroidism, and severe osteoporosis, was advised to present to the emergency room for hypocalcemia and hypophosphatemia after routine lab draw. His initial ionized calcium was 0.89 mmol/L (1.09–1.29 mmol/L) and phosphorus was 0.9 mg/dL (2.3–4.4 mg/dL), with a 25-hydroxyvitamin D level of 62 ng/mL (20–50 ng/mL), PTH of 132 pg/mL (11–51 pg/mL), and normal renal function. Hemoglobin was stable between 8–9 g/dL (13.5–17.1 g/dL). He endorsed ongoing fatigue, oral ulcers, and perioral numbness, which had been attributed to Humira infusion 2 months prior. He denied other paresthesias, carpopedal spasms, seizures, bone pain, or confusion. He reported receiving his second annual infusion of zoledronic acid 10 days prior in Turkey. He was started on high-dose oral calcium, calcitriol, and phosphate, which were continued on discharge 5 days later. Several days into his hospitalization, the patient spoke with his wife in Turkey who confirmed that his calcium and phosphorus were both within normal limits immediately prior to his infusion. A 1,25-hydroxyvitamin D level obtained during his workup was normal at 42.5 pg/mL (19.9–79.3 pg/mL), however an FGF23 level, which returned 2 weeks later, was elevated at 287 RU/mL (<=180 RU/mL). Dotatate PET to rule out oncogenic osteomalacia was negative. One month later, after normalization of calcium and phosphorus levels, repeat PTH and FGF23 levels were both within normal limits.

Conclusion: This case demonstrates that a transient increase in FGF23 levels may accompany and exacerbate hypophosphatemia following zoledronic acid infusion. The mechanism for this elevation is unclear, though we speculate that in the setting of acute hypocalcemia and hypophosphatemia due to zoledronic acid infusion, secondary hyperparathyroidism may upregulate FGF23 production, which further decreases phosphorus levels. If this scenario is accurate, the transient FGF23 elevation seen is not pathologic, but physiologic. Indeed, in the patient above, calcium and phosphorus repletion lead to normalization of both PTH and FGF23.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Severe Osteomalacia and Fractures Secondary to Vitamin D Deficiency

Justin Mathew, MD, David Berger, MD, Vafa Tabatabaie, MD.
Montefiore Medical Center, New York, NY, USA.

Background: Vitamin D deficiency is a common entity among the elderly. Low vitamin D levels can lead to poor bone mineralization, in addition to elevations in PTH levels with resultant increases in bone turnover. However, severe Vitamin D deficiency causing osteomalacia has become uncommon in the United States due to increased screening and treatment. Vitamin D supplementation is a mainstay of therapy for osteoporosis, yet its effect on bone density is generally thought to be modest. We present here an extreme case of vitamin D deficiency leading to severe secondary hyperparathyroidism and bone demineralization, with excellent response to supplementation.

Clinical Case: Patient was a 73-year-old woman with hypertension who presented to the ER with acute on chronic back and lower extremity pain. She had these pains for about a year, but they had worsened over the last 4 days. She had been homebound for the past 1–2 years due to severe pain while ambulating, reported a five-inch loss of height and 50 pounds weight loss, and maintained a vegan diet. She had not had medical care in 15 years. Imaging studies demonstrated a displaced left femoral neck fracture, a nondisplaced right femoral neck fracture, multilevel thoracolumbar compression fractures, and a nondisplaced right scapular fracture. Blood tests revealed normal renal function, calcium 8.6mg/dL (nl 8.5–10.5), phosphorus 2.6mg/dL (nl 2.5–4.5), and alkaline phosphatase 2,821U/L (nl 45–164). Secondary osteoporosis workup was negative for hypercalciuria or multiple myeloma, but was notable for a PTH level of 2,190 pg/mL (nl 10–65) and 25-OH Vitamin D level of <5ng/mL (nl >30). C-telopeptide was measured at 3,346 pg/mL (nl <1000) and osteocalcin >300ng/mL (nl 8–32). DEXA scan showed T-scores of -4.2 at the lumbar spine and -6.8 at the distal forearm. She was started on high-dose vitamin D supplementation, with serum Vitamin D level rising to 42.1ng/mL after 6 months of treatment. This corresponded to a decrease in PTH to 141.1pg/mL and alkaline phosphatase to 375U/L. Repeat DEXA two years later showed 52.8% increase in bone mineral density at the lumbar spine, and 27.1% increase at the forearm. The patient's body pains have significantly improved and she is now ambulatory again.

Conclusions: Vitamin D deficiency is an uncommon cause of severe bone demineralization in the United States. However, in certain high-risk populations, it can present with debilitating osteomalacia and numerous pathologic fractures. Even in cases of osteoporosis with severe PTH elevation, Vitamin D deficiency must be ruled out as a potential secondary etiology, as it can be easily treated with potentially dramatic response.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Severe Refractory Hypocalcemia After Administering Zoledronic Acid for Osteoporotic Fracture in Primary Hyperparathyroidism That Is Complicated Into Hungry Bone Syndrome: A Case Report

Ali Saleh Alhamdan, MBBS[1], NAJAH YOUNES DOUBA, MRCP[2], Abdulaziz Aljamaan, MD[1], Abdulrahman Aidh Alghamdi, Endocrine Fellow[1], Zahrah Abbas Alhammad, MD[1], Khalid Alanazi, MD[1].
[1]KING SAUD MEDICAL CITY, Riyadh, Saudi Arabia,
[2]MINESTRY OF HEALTH, RIYADH, Saudi Arabia.

Zoledronic acid is a very effective (IV) amino bisphosphonate which is indicated in osteoporosis, hypercalcemia of malignancy, multiple myeloma, Paget's disease, and bone metastases from solid tumors. Bisphosphonate inhibits bone resorption through actions on osteoclast activity resulting in increasing bone density. Unfortunately, there are side effects associated with zoledronic acid one of those is mild to moderate hypocalcemia. Hungry Bone Syndrome (HBS) is defined as a severe drop in calcium levels less than 2.1 mmol/L and/or prolonged hypocalcemia for more than 4 days post parathyroidectomy. Most seen in patients' who have secondary hyperparathyroidism compared to primary