

stabilized (corrected 8.2 mg/dL, albumin 3.4 g/dL) and her associated symptoms had resolved. She was discharged on Ocal 2 tabs 4 times daily and Calcitriol 1 mcg twice daily.

Conclusion: Given these biochemical results and imaging evidence of bone metastases, this case highlights the uncommon findings of hypocalcemia in the setting of malignancy. With the combination of low 24-hour urine calcium, elevated PTH, normal vitamin D25, low PO₄, normal kidney function and increased alkaline phosphatase, these findings most likely indicate hypocalcemia secondary to osteoblastic bone metastasis, resulting from deposition of calcium in osteoblastic lesions.

References: Schattner A, Dubin I, Huber R, Gelber M. Hypocalcaemia of malignancy. *Neth J Med.* 2016 Jul;74(6):231–9. PMID: 27571720

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Hypomagnesemia-Induced Hypocalcemia Successfully Treated With Sodium-Glucose Cotransport Inhibitor (SGLT-2i), a Case Report

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Background: Severe hypomagnesemia can result in hypocalcemia as magnesium is a co-factor necessary for PTH production in the parathyroid glands. Calcium replacement alone can prove difficult in the setting of hypomagnesemia, therefore optimal treatment should also include repletion and normalization of magnesium levels. Sodium-glucose cotransporter 2 inhibitors (SGLT-2i) are a class of oral medications used in the treatment of type 2 diabetes and recently in heart failure which inhibit the sodium-glucose transporter in the proximal tubule of the kidney. A lesser known effect of SGLT-2i is renal tubular resorption of magnesium leading to increased serum magnesium levels. We report a case of refractory diuretic-induced hypomagnesemia and subsequent hypocalcemia which was successfully treated with SGLT-2i. **Clinical Case:** A 74-year-old male with uncontrolled type 2 DM, vitamin D deficiency, NASH cirrhosis, chronic lower extremity edema on furosemide therapy, and CKD stage III presented with unexplained hypocalcemia at 7.4–8.4 mg/dL (n: 8.8 – 10.4 mg/dL) and hypomagnesemia at 1.0–1.3 mg/dL (n: 1.5 – 2.5 mg/dL) for the past two years. He had been followed by his PCP who performed a workup revealing 25-OH vitamin D level 22.6 ng/mL, PTH 43 pg/mL (n: 10–55 pg/mL) with corresponding calcium 8.1 mg/dL. He was started on ergocalciferol 50,000IU, magnesium oxide 400mg daily, titrated up to 400mg BID, and calcium carbonate 400mg BID with no improvement in magnesium or calcium level. Later, he was admitted to the hospital with volume overload. During admission, magnesium was repleted with IV magnesium

sulfate to a level of 2.1 mg/dL and serum calcium level normalized to 8.8 mg/dL without supplementation once magnesium level was replete. He was discharged with resumption of oral magnesium sulfate 400mg BID, but within 10 days his serum magnesium level dropped to 1.0 mg/dL. Endocrinology was consulted, and due to the severe hypomagnesemia, empagliflozin 10mg daily was started for the purpose of increasing the serum magnesium level and secondarily for improvement in glucose and edema control. After one week of treatment, the magnesium level increased to 1.4mg/dL and calcium level to 8.7mg/dL. Six months later, magnesium and calcium levels remained stable at 1.7 mg/dL and 9.1 mg/dL, respectively. The patient continues to now remain normocalcemic and normomagnesemic on empagliflozin 12.5mg daily. **Conclusion:** SGLT-2i represent a potent class of anti-hyperglycemic agents with the secondary effect of renal tubular absorption of magnesium which may help achieve appropriate PTH secretion in cases of treatment refractory hypocalcemia. This is one of the first cases to report the off-label use of SGLT-2i for refractory hypocalcemia due to hypomagnesemia. SGLT-2i should be considered in these cases especially in the setting of suboptimally controlled type 2 DM when other interventions have been unsuccessful.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Hypophosphatasia Misdiagnosed as Osteoporosis in a Young Girl

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Introduction: Hypophosphatasia (HPP) is a rare inherited disease of mineral metabolism characterized by low activity of the tissue-nonspecific isoenzyme of alkaline phosphatase (TNALP), which causes an inability to liberate inorganic phosphate for hydroxyapatite crystal propagation as well as toxic accumulation of inorganic pyrophosphate, pyridoxal-5'-phosphate and urinary phosphoethanolamine. It has a prevalence of 1/100,000 to 1/900,000, although milder forms have an estimated prevalence of 1/6,370. Variable mutations in TNALP cause clinical expressions ranging from a severe perinatal form, which is often fatal after birth from pulmonary complications, to an infantile form, which can cause vitamin B6-responsive seizures, to an asymptomatic adult form.

Case: A 27-year-old, ventilator-dependent female with osteoporosis, hypothyroidism, cerebral palsy with previous spinal fusion, seizure disorder and nephrocalcinosis presented with surgical site infection from a right femur ORIF she underwent a month ago. She had a history of microfractures and low-impact fractures of both femurs requiring several surgeries. Osteoporosis was diagnosed at age 5 and she had been on Fosamax ever since. She did not meet any developmental milestones as a baby and never reached menarche. Various diagnoses by multiple specialists did not fully explain her clinical presentation. Her medications included alendronate 5 mg, calcium carbonate 600 mg, ergocalciferol 400 U and levothyroxine 50 mcg.