stabilized (corrected 8.2 mg/dL, albumin 3.4 g/dL) and her associated symptoms had resolved. She was discharged on Oscal 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**

Anand Gandhi, M.D.1, Michael Mortensen, DO2, Mahmoud Alsayed, MD3, Jerome H. Targovnik, MD4, Karyne Lima Vinales, MD3, Ricardo Rafael Correa, MD, EsD, FACP, FACE, FAPCR, CMQ6.

1University of Arizona College of Medicine - Phoenix, Phoenix, AZ, USA, 2PHOENIX VA HEALTHCARE SYS, Flagstaff, AZ, USA, 3University of Arizona COM-Phoenix, Phoenix, AZ, USA, 4Phoenix VA Healthcare Sys, Paradise Valley, AZ, USA, 5Phoenix VA Healthcare System, Phoenix, AZ, USA, 6University of Arizona College of Medicine Phoenix, Phoenix, AZ, USA.

**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**

Tiffany Tsang, MD1, Maya P. Raghuwanshi, MD, MPH, FACP, FACE2.

1Rutgers New Jersey Medical School, Newark, NJ, USA, 2Rutgers-NJMS, Clark, NJ, USA.

**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 did not fully explain her clinical presentation. Her medications included alendronate 5 mg, calcium carbonate 600 mg, ergocalciferol 400 U and levothyroxine 50 mcg.
Physical exam showed poor dentition, a misshapen skull and bowed legs with contractures of her extremities. Her labs revealed Ca 9.1 mg/dL (8.4–10.2 mg/dL), albumin 3.2 gm/dL (3.5–5.2 gm/dL), phosphorus 5.1 mg/dL (2.5–4.5 mg/dL), alkaline phosphatase 32 U/L (35–105 U/L), PTH 28 pg/mL (15–65 pg/mL), vitamin D 33.5 ng/mL (30–100 ng/mL), C-telopeptide 509 pg/mL (34–635 pg/mL). A right knee X-Ray reported gracile and demineralized bones with muscular atrophy. She recently transitioned care from a pediatric endocrinologist to an adult endocrinologist, who tested her positive for heterozygous ALPL pathogenic variant hypophosphatasia and was considering her for asfotase alfa enzyme replacement therapy.

Discussion: Our patient had infantile HPP, but due to misdiagnosis as osteoporosis, she was inappropriately treated with a bisphosphate for over 20 years. Treatment of HPP had been supportive until the approval of asfotase alfa (Strensiq) in October 2015. It is a bone-targeted human recombinant enzyme replacement therapy approved for infantile- and juvenile-onset HPP and has been shown to decrease mortality from 73% to 16% at age 5. With improvement in life-sustaining technology, more HPP patients are able to survive into adulthood. Awareness of the complex and polymorphic presentation of HPP by adult endocrinologists is paramount for accurate diagnosis, thus avoiding inappropriate treatments.

Bone and Mineral Metabolism

Bone and Mineral Case Report

Iatrogenic Hypocalcemia With Treatment of Milk-Alkali Syndrome
Sharan D. Parikh, DO, Geetha K. Bhat, MD.
Cooper University Hospital, Camden, NJ, USA.

Hypocalcemia is common disorder with the most likely etiology being primary hyperparathyroidism in the outpatient setting and malignancy in the hospitalized. With emergence of proton pump inhibitors and histamine blockers, milk-alkali syndrome has become a rarity. We report a unique case of hypocalcemia secondary to milk-alkali syndrome overtreated with bisphosphate therapy resulting in hypocalcemia.

A 77-year-old woman with a past medical history of hypertension, gastroesophageal reflux disease presented with slurring of speech for 2 days with nausea and vomiting. Labs showed a calcium of 15.4 mg/dL, with an albumin of 4.0 g/dL. Other pertinent labs showed an ionized calcium of greater than 7.3 mg/dL, pH of 7.49, PTH of 15 pg/mL, PTHrP of 9 pg/mL, vitamin D 25-OH of 16 ng/mL, TSH of 2.16 IU/mL and acute kidney injury. Patient was started on intravenous fluids and given both calcitonin and pamidronate on presentation by the admitting team. When seen in consultation, history revealed that patient was consuming more than eight calcium carbonate antacid tablets daily and was also on hydrochlorothiazide. The calcium level decreased to 8.7 mg/dL within 48 hours. There was a concern for potential hypocalcemia due to pamidronate. Patient was advised to restart calcium carbonate 500 mg twice daily upon discharge with close follow up. However, supplementation was not started and repeat calcium was 6.7 mg/dL twelve days later. The calcium normalized within a week after starting temporary calcium supplementation.

A now rare cause of hypercalcemia, milk-alkali syndrome is often overlooked in the differential diagnosis resulting in overtreatment and potentially dangerous hypocalcemia. Emergent management of intravenous hydration and bisphosphonate therapy is often immediately given by clinicians. Bisphosphonate therapy is not immediately effective and demonstrates calcium lowering effects by the second to fourth day. However, patients with milk-alkali syndrome generally improve with intravenous hydration and cessation of the causative agent. This case demonstrates the importance of obtaining a proper history with a complete list of medications and over the counter supplemenations prior to treatment.

Bone and Mineral Metabolism

Bone and Mineral Case Report

Immobilization Hypercalcemia (IH) in an Adult After Spinal Cord Injury
Zakaria Sibai, MD1, Robert H. Wendthrob, Medical student2, Swati M. Daftary, MD3, Sathya S. Krishnasamy, MD3.
1University of Louisville Hospital, Louisville, KY, USA, 2University of Louisville, Louisville, KY, USA.

Background: Immobilization hypercalcemia (IH) is an uncommon diagnosis and little has been published about management of this condition. We present a case of IH and discuss clinical aspects of this unique bone metabolism state. Case: 32-year-old female with history significant for IV drug use, presented to the hospital with features of septic shock, Work up showed MRSA bacteremia associated with tricuspid endocarditis and septic pulmonary emboli. MRI of the spine done for progressive weakness of extremities revealed a cerebral epidural abscess leading to spinal cord compression and myelomalacia. She underwent spinal abscess drainage and corpectomy, remained quadriplegic. 8 weeks later we were consulted to see her at inpatient rehabilitation facility for hypercalcemia. Labs showed Creat(0.6 mg/dL), hypercalcemia with corrected Calcium of 12.9 mg/dL. Further workup showed suppressed PTH (< 6), V D-25 at (< 10 mg/dl). Phosphate (normal). ACE levels, PTHrp, Vit. D 1,25, SPEP, UPEP,and TSH were all normal. 24 hours urine calcium showed hypercalcuiuria, 400 mg/ml. Fasting Bone markers were elevated with serum N-telopeptide > 40 nM BCE, ref range (6.2–19.0) and C-Telopeptide is 2060 pg/ml ref range (60–650), which indicates increase bone turnover. IH was diagnosed and hydration attempted for few weeks. Serum calcium remained at 13 mg/dl, so she received a single slow infusion of pamidronate 30 mg. 4 days after infusion, calcium levels decreased to normal value of 10.2 mg/dl without any side effects. She remained normocalcemic 5 weeks post infu. 

Discussion: IH is an uncommon condition but early recognition and intervention will minimize secondary long-term complications such as kidney stones, Osteoporosis or low bone mass, renal failure and acute pancreatitis. Albert, Fuller described IH in 1941(1). Most cases of IH had been reported in children and adolescents with high bone turn over following recent spinal cord injury(2–3). Very few reports have been published in young adults such as our patient, IH is attributed to prolonged immobilization from paralysis due to spinal cord injury, and other etiology...