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 diffusely 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

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Hypercalcemia 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 hypercalcemia 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 supplementations prior to treatment.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Immobilization Hypercalcemia (IH) in an Adult After Spinal Cord Injury

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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 cervical 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 f. 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 hypercalciuria, 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 infusion. **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

(2–4), Proposed mechanism includes decreased mechanical stress on the bone which leads to increase bone resorption and remodeling and demineralization. To establish the diagnoses of IH, an exhaustive evaluation is warranted to rule out other causes of PTH dependent and independent hypercalcemia(5). Multiple treatment approaches have been used over the past few decades including early mobilization, hydration., saline diuresis, subcutaneous Calcitonin, glucocorticoids (6), IV sodium sulphate and phosphate (7). Bisphosphonates are the preferred pharmacological treatment of IH. Nonnitrogen containing bisphosphonates including clodronate and etidronate (8–9),. are not preferred due to concern for bone demineralization and osteomalacia. (10). Pamidronate has been used to treat IH due to its efficacy, safely and cost (11). Caution to be exercised in those patients with kidney disease.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORT

In Search of a Missing Adenoma

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Background: The most common cause of primary hyperparathyroidism (PHPT) is overproduction of PTH by a parathyroid gland adenoma. While definitive therapy is parathyroidectomy, 4% of patients develop persistent PHPT - a sustained hypercalcemic state that is detected within six months of parathyroidectomy. A missed parathyroid adenoma is the most common cause of persistent PHPT, and accurately locating these glands presents a diagnostic challenge. We describe a rare case of persistent PHPT due to a missed mediastinal parathyroid adenoma.
Case: A 54-year-old woman with a history of PHPT presented with abdominal pain, nausea, and decreased oral intake. She underwent parathyroidectomy six months ago with reimplantation of one parathyroid gland into the right sternocleidomastoid muscle (SCM). She was now hypercalcemic to 13.9 mg/dL (8.5–10.5) with intact PTH 1273 pg/mL (15.0–65.0), vitamin D 25-OH 31.4 ng/mL (>30.0), and normal PTHrP. She was not taking calcium, and other causes of hyperparathyroidism were excluded. Sestamibi scintigraphy localized only to the right SCM, and the initial impression was recurrent HPT due to the previously implanted gland. Follow-up CT neck with and without contrast failed to localize any regrowth in the SCM, but did reveal a 1.4 cm mediastinal soft tissue mass, suspicious for an ectopic parathyroid adenoma. She subsequently underwent video-assisted thoracoscopic excision, and pathology was consistent with ectopic hypercellular parathyroid tissue. Post-operatively, her PTH down-trended and calcium normalized.
Conclusion: This case describes a small yet biochemically aggressive mediastinal adenoma causing persistent PHPT. While sestamibi scans have ~90% sensitivity for localization of ectopic adenomas, they can fail to identify a small culprit lesion in 12% of patients, whereas CT imaging with and without contrast has increased sensitivity for adenomas <2 cm. Thus, diagnosing persistent PHPT requires sestamibi scan in combination with other imaging modalities for accurate diagnosis of missed adenomas.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORT

Intravenous Iron Induced Severe Hypophosphatemia

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Introduction: Hypophosphatemia is a recently recognized adverse effect of certain intravenous iron formulations. It was previously thought to be asymptomatic and transient, however it can cause severe hypophosphatemia associated with renal phosphate wasting. We present a case of severe hypophosphatemia following intravenous ferric carboxymaltose administration.
Clinical Case: A 40-year-old female with history of iron deficiency anemia, Hashimoto's hypothyroidism and multinodular goiter status post thyroidectomy and obesity status post gastric sleeve presents to the hospital due to dizziness, generalized weakness, bone pain and severe myalgia right after 2nd dose of 750 mg of ferric carboxymaltose administration. She received 1st dose of 750 mg of ferric carboxymaltose a week ago prior to the presentation. She was found to have severe hypophosphatemia with a phosphorus level 1.0 mg/dl (2.4 - 4.8 mg/dl), hypocalcemia with calcium level at 8.7 mg/dl, normal PTH at 40 pg/ml and normal 25-OH Vitamin D level at 59.1 ng/ml. The fractional excretion of filtered phosphate (FEPO4) was 19.5%, supporting renal phosphate wasting. She was hospitalized for 5 days and received intravenous phosphate 45 mmol on hospitalization day 1, 15 mmol on day 2 and 30 mmol on day 3. She was discharged on K-phos Neutral 250 mg TID, Calcium carbonate/Vitamin D3 600 mg/500 IU 2 tablets TID and Calcitriol 0.5 mcg daily. While she was taking K-phos Neutral, Calcitriol and Calcium carbonate/Vitamin D3, her phosphorous level was down to 1.3 mg/dl, which was 4 weeks after 1st dose of ferric carboxymatose administration. She presented to the ED and received intravenous phosphate 15 mmol. She is currently taking K-phos Neutral 250 mg QID, Calcitriol 0.5 mcg daily and Calcium carbonate/Vitamin D3 600 mg/500 IU 2 tablets TID. Her phosphorous levels ranged at 2.5 - 3.0 mg/dl. Her bone pain and myalgia have gradually improved.
Conclusion: Clinician should be aware of the side effect of hypophosphatemia following ferric carboxymaltose administration. Recent studies showed that incidence of hypophosphatemia associated with intravenous iron administration was significantly higher in ferric carboxymatose group compared with either iron isomaltoside group or ferumoxytol group. The impact of severe hypophosphatemia should be considered when choosing an intravenous iron medication.