her last result (1426 U/L from 933 U/L) and contrast-MRI now showed the development of areas of walled-off necrosis in the body and tail of the pancreas. Serum ionized calcium was again elevated (2.17 mmol/L); she also had an elevated intact PTH level (780.6 pg/ml, RI: 18.5–88.0 pg/ml). Parathyroid SPECT-CT showed a sestamibi-avid lesion inferoposterior to the right thyroid lobe, indicating a probable parathyroid adenoma. She was given 1 dose of Denusomab 120 mg subcutaneously and started on Calcitonin nasal spray 200 IU twice a day. She underwent ultrasound-guided drainage of the pancreatic necrosis and was subsequently discharged. Due to difficulty in procurement of the Calcitonin nasal spray, she was maintained on Cinacalcet 30 mg/tablet, 1 tablet once a day upon discharge with plans to undergo parathyroidectomy once she was had fully recovered from her pancreatitis. **Conclusion:** Primary hyperparathyroidism should always be considered when hypercalcemia is noted during the clinical management of acute pancreatitis. **Reference:** Misgar RA, Bhat MH, Rather TA, Masoodi SR, Wani AI, Bashir MI, Wani MA, Malik AA. Primary hyperparathyroidism and pancreatitis. J Endocrinol Invest. 2020 Oct;43(10):1493–1498.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

An Interesting Case of Isolated Bone Marrow Sarcoidosis

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Introduction: About 30% cases of sarcoidosis have extrapulmonary manifestations but only 7% of patients present without any lung involvement. Among those 7%, most of the patients have manifestations on the skin but isolated bone marrow sarcoidosis has not been commonly reported. This case represents an unusual manifestation of isolated bone marrow sarcoidosis presenting with very high calcium levels.
Case Presentation: A 58-year-old female presented to us with fatigue, poor appetite, and nausea. She did not report any weight changes. Her cancer screening was up to date. On examination, she appeared dehydrated. No neck swelling was appreciated. Cardiac, respiratory, abdominal, and neurological examinations were normal. Complete blood count showed hemoglobin of 10.6 mg/dL, white blood cell count of 3.8 k/dL, and platelet count of 87 $x10^9$/L. Metabolic panel revealed hypercalcemia with corrected calcium levels as high as 12.6 mg/dL. Ionized calcium was 8.1 mg/dL (normal 4.8 - 5.6). Her parathyroid hormone (PTH) level was elevated up to 64.6 mg/dL and then further increased to 134.3 mg/dL. A 24-hour urinary calcium level was normal. 1, 25-dihydroxy (1,25-OH) and 25-OH vitamin D levels were 97 mg/dL (normal 18–72) and 31.2 mg/dL, respectively. Serum protein electrophoresis and light chain analysis were normal. Hyperparathyroidism was suggested as a cause of hypercalcemia. Ultrasound of the neck and sestamibi scan showed a right lower pole parathyroid adenoma. Paraneoplastic hypercalcemia was also one of the differentials. Parathyroid hormone related peptide (PTHrP) was 9 pg/mL (normal 14 - 27). Colonoscopy was normal. Computerized tomography showed normal lungs, liver and spleen. No masses and lymphadenopathy was seen. A bone marrow biopsy was done for pancytopenia. Patient underwent parathyroid adenoma removal followed by a drop in serum calcium level (8.2 mg/dL). Patient was discharged on calcium carbonate and vitamin D tablets. Upon outpatient follow-up, calcium level started to rise again up to 9.8 mg/dL. Despite discontinuation of supplemental calcium and vitamin D, calcium continued to uptrend (11.5 mg/dL 4 weeks later). Angiotensin converting enzyme (ACE) level came back as high as 129 (normal level < 40 mcg/L). Meanwhile, the bone marrow biopsy results showed that 40% of bone marrow was occupied by non-caseating granulomas suggesting sarcoidosis. Patient was started on steroids for isolated bone marrow sarcoidosis, and eventually her serum calcium level normalized.
Conclusion: An isolated bone marrow sarcoidosis is an extremely rare manifestation of extrapulmonary sarcoidosis. It can present with pancytopenia and should be sought in patients with persistent hypercalcemia. In addition, our case was challenging due to the presence of a concurrent hyperparathyroidism which was initially thought to be the only explanation of our patient's hypercalcemia.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

An Uncommon Case of Hypophophatasia Presenting With Galactorrhea, Diagnosed at a Later Age Than Expected

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Hypophosphatasia is a rare genetic disorder which causes accumulation of inorganic pyrophosphate that in turn inhibits mineralization. It is caused by a mutation in the non-specific alkaline phosphatase gene (TNSALP). We will review the diagnosis during adulthood when the presentation is characterized by poor healing, recurrent metatarsal stress fractures and bone pain. We are reporting the case of a 38-year-old white female with history of Melanoma referred to the Pituitary Clinic due to galactorrhea. During the encounter it was noted that she had history of disseminated joint pain, myalgias, periodontal complications as well as biochemical evidence of low alkaline phosphatase. At age three she had premature teeth loss, and clumsy gait, which later resolved. She was evaluated for this and diagnosed with odontohypophophatasia at which time no treatment was initiated. As an adult patient had two non-traumatic ankle fractures with associated frail teeth, chipping, and cracking without significant trauma. At around 36 years of age, patient started developing worsening joint pain, stiffness, myalgias and 35 pounds weight gain. Family History is remarkable for her father, having history of low alkaline phosphate and odontogenic infections with no joint or muscle pain. Paternal grandmother had history of dental infections and frail teeth. The patient has four sons; her oldest son has low alkaline phosphate with no clinical symptoms. Patient's biochemical analysis was unremarkable except for low alkaline phosphate range between 25–38 U/L (40–125 U/L) and elevated Vitamin B6