of Covid-19 diagnosis. All Covid-19 symptoms subsided 14 days after onset. Repeat labs 1 and 3 months after Covid-19 infection showed persistently normal serum Ca (10.0 and 9.8 mg/dL), with low then normal PTH (13 and 43 pg/mL), compatible with spontaneous resolution of PHPT. Patient denied any neck discomfort before, during, or after Covid-19 infection. Spontaneous resolution of PHPT is rare and follows apoplexy of a large parathyroid adenoma. In our patient, imaging failed to localize a large parathyroid adenoma, making it less likely that resolution of her PHPT was caused by apoplexy. Resolution of PHPT temporally coincided with Covid-19 infection, although the link between the two conditions is unclear at this time. Hypothesized mechanisms include an imbalance in the normal PTH-Ca axis caused by SARS-CoV-2 mediated release of inflammatory cytokines (e.g. interferon, previously reported to lower serum calcium), or development of antibodies against the parathyroid or CaSR. RNA and protein expression of *ACE2,* the SARS-CoV-2 cell receptor gene, is not detected in normal parathyroid tissue. **Conclusion:** To our knowledge, this is the first reported case of spontaneous resolution of PHPT after Covid-19 infection. Further studies are needed to understand the frequency of this occurrence, and the underlying mechanism.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Sporadic Pseudohypoparathyroidism 1B in Monozygotic Twins: Insights Into the Pathogenesis of Methylation Defects

Yamato Keidai, MD[1], Yorihiro Iwasaki, MD, PhD[1], Kanako Iwasaki, MD, PhD[1], Sachiko Honjo, MD[1], Murat Bastepe, MD, PhD[2], Akihiro Hamasaki, MD, PhD[1].
[1]Tazuke Kofukai Medical Research Institute Kitano Hospital, Osaka, Japan, [2]MASSACHUSETTS GENERAL HOSPITAL, Boston, MA, USA.

Background: Pseudohypoparathyroidism (PHP) 1B is an imprinting disorder characterized by renal resistance to parathyroid hormone (PTH) without Albright Hereditary Osteodystrophy (AHO). PHP1B is associated with methylation defects at the *GNAS* differentially methylated regions (DMRs). In sporadic cases with PHP1B, the mechanistic basis of methylation defects remains to be solved, except in rare cases with uniparental disomy of chromosome 20. In addition, to date, monozygotic twin cases with sporadic PHP1B have not been reported.
Clinical Case: The patients were 26-year-old Japanese female monozygotic twins. They had been born to nonconsanguineous parents after an uneventful pregnancy. Both twins had common biochemical features, including hypocalcemia, hyperphosphatemia, elevated PTH levels, and impaired urinary excretion of phosphorus and cAMP in response to teriparatide. They showed no signs of AHO. The serum calcium levels of their parents and brother were within the normal range, and family history was unremarkable. Based on these findings, the twins were diagnosed with PHP1B. Targeted bisulfite sequencing of the *GNAS* DMRs in all family members revealed almost complete gain-of-methylation at the *NESP55* DMR, and loss-of-methylation at the *AS*, *XL*, and *A/B* DMRs in the twins, but not in other family members. Except for the *GNAS* locus, we did not find clear methylation defects in other imprinted genome loci in the twins. Methylation defects at the *GNAS* locus were further confirmed by methylation-sensitive restriction enzyme-qPCR. Whole-genome sequencing of the twins showed no pathogenic variants in the *GNAS* exons encoding the Gs alpha subunit. No large deletions or insertions were found at the *STX16* locus or in the region from AS exon 5 to XL. Based on the SNP genotyping results, large paternal isodisomies in the *GNAS* DMRs were unlikely. Collectively, these results suggested that the twins had concordant methylation defects that are seen in the sporadic form of PHP1b. We speculate that an early developmental event before the twin splitting is responsible for the abnormal methylation of the *GNAS* DMRs.
Conclusion: We report, for the first time, monozygotic twins with sporadic PHP1B who were phenotypically and epigenetically concordant. Our comprehensive molecular genetic analyses have thus far ruled out the previously described genetic defects underlying PHP1B. The current findings provide new insights into the mechanistic basis of the *GNAS* methylation defects in sporadic PHP1B.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Sternal Phosphaturic Mesenchymal Tumor-Induced Osteomalacia, Diagnosis and Management: A Case Report

Mopelola Adetola Adeyemo, MD, MPH, Aili Guo, MD
University of California Davis, Sacramento, CA, USA.

Background: Tumor induced osteomalacia (TIO) is a rare paraneoplastic disorder in which overproduction of fibroblast growth factor-23 (FGF-23) by mesenchymal tumor results in decreased renal phosphorus reabsorption and low to inappropriately normal 1,25-dihydroxyvitamin D, leading to hypophosphatemia and osteomalacia. Patients often present with bone pain, fractures, muscle weakness, and progressive decline in mobility. Due to the nonspecific nature of presenting symptoms of TIO diagnosis is often delayed. **Clinical Case:** A 55-year-old male presented with complaints of chest pain, shortness of breath, and generalized weakness following a ground level fall. Patient also reported a 10-year history of osteoarthritis with chronic back pain and 1-year history of generalized weakness, resulting in significant decline in functional status. On work-up, the initial CT scan of chest revealed multiple fractures including ribs, manubrium, scapula, and pubic rami. Subsequent biochemical evaluation was remarkable for hypophosphatemia to low of 1.3 mg/dL (2.4 - 5.0 mg/dL), low of 1,25-dihydroxyvitamin D of 13.1 pg/ml (19.9 - 79.3 pg/mL), reduced tubular phosphate reabsorption rate of 28% (normal > 80%) ratifying for renal phosphate wasting, normal iPTH level, and elevated serum FGF-23 level of 460 (normal < 180). Then, localization imaging for TIO was performed. After PET/CT scan showing increased uptake at the sternal area suggestive of lytic metastasis, subsequent CT angiogram of the chest identified mottled, irregular, mildly expansile appearance of the sternal manubrium. Sternal biopsy revealed phosphaturic mesenchymal tumor with positive FGF 23 mRNA expression. Surgical resection