urinary calcium. Symptoms of generalised body pain resolved, and her bone mineral density (BMD) improved over 47 months of medical therapy. Power improved to 4/5, and she was able to ambulate with assistance. BMD at the femoral neck, total hip and lumbar spine increased by 68.2%, 44.6% and 65.2% respectively.

Conclusion: This is a challenging case of TIO which has failed to localise despite best efforts. One must consider FGF23-independent and dependent causes of osteomalacia when patients present with severe frailty and hypophosphatemia as substantial morbidity results from delayed diagnosis and treatment. TIO-related PMTs can be difficult to localise, even with a combination of functional and anatomical imaging. With medical therapy, bone mineralisation and symptoms can improve significantly. Patients need to be monitored for complications of long-term phosphate and calcitriol replacement.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Successful Parathyroidectomy May Not Resolve Hypercalciuria in Patients With Primary Hyperparathyroidism

Neriy Izkhakov, MD, MPH, Heranmaye Prasad, MD, Nicholas John Vernetti, MD, Samer Nakhle, MD.
HCA Healthcare; Sunrise Health GME Consortium; Endocrinology, Diabetes and Metabolism; MountainView Hospital, Las Vegas, NV, USA.

Background: Hypercalciuria, with twenty-four-hour urinary calcium of >400 mg/day, is one of the indications for parathyroidectomy in patients with primary hyperparathyroidism. We report five cases where hypercalciuria is not resolved following a successful parathyroidectomy (normalization of serum calcium) in such patients. Here resolution of hypercalciuria is defined as twenty-four-hour urinary calcium of less than 200 mg/day. **Clinical Case:** This is a case series of five patients who remained hypercalciuric at 6 to 19 months after successful parathyroidectomy. Pre-parathyroidectomy, average PTH was 95 pg/dL (Min 69 pg/dL, Max 120 pg/dL), average serum calcium was 11.0 mg/dL (min 10.3 mg/dL, max 12.0 mg/dL), and average twenty-four-hour urinary calcium was 455 mg/day (min 386 mg/day, max 551 mg/day). Calcium levels were corrected to normal range post-parathyroidectomy and remained in normal range. However, hypercalciuria did not resolve. Post-parathyroidectomy, average PTH was 44 pg/dL (min 25 pg/dL, max 69 pg/dL), average serum calcium was 9.6 mg/dL (min 9.3 mg/dL, max 9.8 mg/dL), and average twenty-four-hour urinary calcium was 284 mg/day (min 201 mg/day, max 376 mg/day). Two patients who had history of nephrolithiasis prior to parathyroidectomy continued to develop nephrolithiasis at six and sixteen months after successful parathyroidectomy. **Conclusions:** This case series showed that hypercalciuria may not resolve following a successful parathyroidectomy in patients with primary hyperparathyroidism and elevated twenty-four-hour urinary calcium at 6 to 19 months after surgery. Further observations to evaluate long term effects of parathyroidectomy on hypercalciuria is needed.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

The Importance of Interpreting Urine Calcium:Creatinine Ratio in PHPT Within Ethnic Context

Mariana Abdel-Malek, BSc (Hons), MBBS, MRCP[1], Shamaila Zaman, MBBS, MRCP[1], George Tharakan, MBBS, BSc, MRCP, PhD[1], Edouard G. Mills, MBChB, MRCP[2], Adam John Buckley, MA MBBS MRCP PhD[1], Jeannie Todd, MBBS, BSc, MD, FRCP[1], Preeshila Behary, MBChB, MRCP, PhD[1], Jeremy Philip Cox, MBBS,MD[3], Alexander N. Comninos, BSc (Hons), MBBS, MRCP, PhD[4].
[1]Imperial College Healthcare NHS Trust, London, United Kingdom, [2]Section of Investigative Medicine, Imperial College, London, United Kingdom, [3]St Mary's Hospital Imperial College, London, United Kingdom, [4]Imperial College, London, United Kingdom.

A 56-year-old Afro-Caribbean lady was found to have incidental hypercalcaemia on routine GP investigations. Tiredness was the only elicitable hypercalcaemic symptom and aside from early menopause at age 40, she had no significant past medical or family history. Examination was unremarkable. Blood results showed a raised adjusted calcium 2.68mmol/L (2.2–2.6), normal phosphate 1.06mmol/L (0.80–1.50), raised parathyroid hormone (PTH) 14.1pmol/L (1.6–7.2) and low 25-hydroxyvitamin D 28.1nmol/L (70–150).

She had osteopaenia of the femora and left radius on DEXA scan but no nephrocalcinosis on renal ultrasound. On initial investigation, her urinary calcium output was low at 1.55mmol/day resulting in a 24h calcium:creatinine ratio (UCCR) of 0.0065. Although suggestive of Familial Hypocalciuric Hypercalcaemia (FHH), her notable Vitamin D deficiency was considered to contribute to the observed hypocalciuria. After Vitamin D repletion, a repeat UCCR improved to 0.012, however, remained in the indeterminate range. No known pathogenic variant was identified on genetic analysis for FHH.

Her PTH and Calcium levels remained persistently high within 9.7–17.1pmol/L and 2.65-2.82mmol/L respectively, suggestive of Primary Hyperparathyroidism (PHPT) given the end organ damage and negative genetic studies. Based on her symptom of fatigue, osteopaenia at a young age and hypercalcaemia, localisation studies were arranged which showed no definitive evidence of a parathyroid adenoma and explorative surgery was planned.

The negative genetic testing, PTH level 17.1pmol/L, osteopaenia, low-normal magnesium and phosphate level collectively support a diagnosis of PHPT in this case, despite a low UCCR which however is observed in some PHPT patients. Indeed, a lower UCCR ratio has been reported in the healthy Afro-Carribean population across all age groups, as well as in Afro-Carribean patients with PHPT [1]. The underlying mechanism for this is yet to be determined but may be due to increased renal sensitivity to PTH or altered activity of the tubular calcium reabsorptive pathways. One can further speculate regarding an evolutionary reason behind a protective homeostatic system favouring renal calcium reabsorption over excretion in this frequently vitamin D deficient population.

Clinical practice relies heavily on the use of UCCR in aiding the biochemical differentiation of PHPT and FHH. However, as this case highlights, its use can be misleading in Afro-Carribean patients and therefore should be interpreted within an ethnic context.

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Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

The Unusual Suspect: Hypocalcemia in Preeclampsia After Magnesium Infusion

Basma Abdulhadi, MD[1], Monica Agarwal, MD[2].
[1]UNIVERSITY OF ALABAM-BIRMINGHAM, Birmingham, AL, USA, [2]University of Alabama at Birmingham, Birmingham, AL, USA.

Background: Magnesium plays a vital role in calcium homeostasis. Magnesium sulfate is used in obstetric populations for the management of preeclampsia and eclampsia. Hypocalcemia secondary to iatrogenic hypermagnesemia is an uncommon complication. We report a case of symptomatic hypocalcemia due to hypermagnesemia. **Clinical Case:** A 26-year-old female with type 1 diabetes at 33 weeks gestation was admitted for preeclampsia. She had severe hypertension and proteinuria, and the decision to induce labor was made. She was started on a magnesium sulfate drip and remained on the drip for 33 hours. Magnesium levels peaked at 7.5 mg/dL (1.7–2.5 mg/dL). The magnesium drip was suspended after delivery. She complained of mild tingling and numbness in her hands, feet, and perioral area. Shortly after delivery, she had a pre-syncopal event. The laboratory evaluation revealed a calcium of 5.9 mg/dL (8.4–10.4) with an albumin of 2.2 g/dL (3.7–5.5), magnesium of 4.2 mg/dL, phosphorus 2.2 mg/dL (2.3–4.6) and GFR above 60 mL/min/1.73m2 . Spot urine calcium to creatinine ratio was 0.17 mg/dL. The 25-hydroxy vitamin D level was low at 12 ng/mL (20–100). The parathyroid hormone (PTH) level was inappropriately normal at 21.7 pg/mL (12–88). She had no history of hypocalcemia, and calcium level obtained six months before the presentation was normal. She received one calcium gluconate infusion, was then started on a continuous calcium gluconate infusion for symptomatic hypocalcemia. She also received oral supplementation of calcium and vitamin D. The calcium levels normalized within 24 hours as the hypermagnesemia resolved from the discontinuation of the magnesium drip. The subsequent PTH level was 38.8 pg/mL. She was discharged on vitamin D supplements. She had no recurrence of hypocalcemia. **Conclusion:** Our case highlights the effect of magnesium infusion on the parathyroid gland leading to profound symptomatic hypocalcemia. Magnesium plays an essential role in the secretion of PTH. Transient hypoparathyroidism can occur due to hypomagnesemia and hypermagnesemia (tocolytic therapy). In hypomagnesemia, the parathyroid gland secretes insufficient PTH, and the renal and skeletal response to PTH is reduced. In hypermagnesemia, the magnesium activates the extracellular calcium-sensing receptor, subsequently causing inhibition of the parathyroid gland. Amelioration of hypermagnesemia leads to the normalization of parathyroid function. Health care providers should be aware of this phenomenon, especially in the obstetric population receiving magnesium infusion. The monitoring of calcium levels may be necessary for this patient population. The laboratory testing should include magnesium in the evaluation of hypocalcemia.

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Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Transient Osteoporosis

Caroline A. Poku, MD[1], Pauline M. Camacho, MD[2], Zubair Ilyas, MD[3].
[1]LOYOLA UNIVERSITY OF CHICAGO, Elmhurst, IL, USA, [2]LOYOLA University Medical Center, Maywood, IL, USA, [3]Loyola University, Maywood, IL, USA.

Background: Transient osteoporosis is an uncommon and self-limited clinical syndrome characterized by acute joint pain with evidence of bone marrow edema on MRI. It predominantly affects healthy middle-aged men or women in the third trimester of pregnancy. The hips, knee, foot and ankle are affected in decreasing order of frequency. Pathophysiology is unknown but multiple etiologies such as ischemia, neurogenic compression or impaired venous return have been proposed. Classically, it is unilateral and bilateral in only 20%-40% of cases. It has been reported to periodically involve different joints over time with one report showing the progression to regional migratory osteoporosis in at least 20% of patients. There are no specific biomarkers to aid with diagnosis, MRI shows diffuse bone marrow edema sometimes associated with joint effusion with infrequent subchondral microfractures. Other etiologies to consider for bone marrow edema include osteomyelitis, avascular necrosis, trauma, tumors and inflammatory arthropathy. Transient osteoporosis can be self- limiting however, bisphosphonate use has been associated with shortened recovery time. In our patient given lack of access to his previous records to review and ascertain his previous diagnosis, his diagnosis of record was transient osteoporosis rather than regional migratory osteoporosis. **Clinical Case:** A 47 yo male presented to clinic with complaint of left ankle pain. Pain initially noted when he tripped and fell one year ago. Initial x-rays did not reveal any fractures. He was unable to weight bear due to pain although he had full range of motion at the ankle with a normal neurological and vascular exam of the foot. Due to persistence of pain, an MRI was done which showed cutaneous edema around the medial and lateral aspects of the ankle, trace tibiotalar joint effusion, marrow edema in the distal tibia and navicular with no acute fracture or definite evidence of avascular necrosis. On further questioning he reported a previous history of hip pain at age 32 and 41 with no preceding trauma. X-rays were negative for fracture and MRI showed marrow edema. Symptoms resolved after a few weeks with possible treatment with Alendronate. With the current presentation biochemical work up including Vitamin D, PTH, 24-hour urine calcium,