have been treated with fluconazole, which has a favourable side effect profile and yields good results. Adjusted calcium reduced to 2.62 nmol/L, 25 OH Vitamin D normalised to 111 nmol/L and 24:24,25 dihydroxyvitamin D ratio is now 17. Patient's liver functions and full blood count has been monitored regularly during the course of treatment and the drug was well tolerated. **Conclusion:** Genetic causes of hypercalcemia can be left undiagnosed for long periods and there is a lack of proven or definitive therapeutic agents for correction of elevated calcium. Here fluconazole has been shown to reduce the hypercalcaemic burden and effectively lowered the Vitamin D levels in this case of a *CYP24A1* mutation. This study augments fluconazole use in these cases but further studies are needed to elucidate the long term safe usage.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Delayed Diagnosis of Congenital Hypoparathyroidism in a Kindred of Three Patients With Autosomal Dominant Deafness

Karel Dandurand, MD, Dalal Ali, MD, Susan Tran, MD, MSc, FRCPC, Tina Zhou, MD, MSc, FRCPC, Aliya Aziz Khan, MD,FRCP,FACP, FACE.
McMaster University, Hamilton, ON, Canada.

Background: Congenital hypoparathyroidism can be related to autosomal dominant mutations or deletions in GATA-binding protein 3 gene on chromosome 10[1,2]. Affected patients present with a triad of hypoparathyroidism, renal dysplasia and neurosensorial deafness. We hereby present the case of a patient with the rare Barakat syndrome, also known as HDR syndrome. **Clinical Case:** A 11-year-old girl, diagnosed with deafness at birth, was brought to medical attention because of menorrhagia requiring blood transfusions two months after menarche. A pelvic ultrasound demonstrated a septate uterus as well as right multicystic dysplastic kidney with solitary left kidney and ovary. As her maternal grandmother, mother and older sister suffered from congenital deafness and her mother also had a kidney cyst, the patient was referred to genetics to identify a unifying cause of the autosomal dominant pattern of deafness and urogenital anomalies. Chromosome microarray analysis revealed a copy number change on chromosome 10p14 of 1925 kb predicted to result in the deletion of a single protein coding gene, GATA3. Embryonically, GATA3 is involved in the development of the inner ear, kidneys and parathyroid glands. The patient was lost to follow up so that a serum calcium was drawn three years later, revealing low ionized calcium of 1.06 mmol/L (N 1.16–1.29), low corrected total calcium of 2.11 mmol/L (N 2.30–2.62) along with PTH of 1.1 pmol/L (N 2.0–9.4), PO4 of 1.73 mmol/L (N 1.03–1.78) and creatinine of 64 umol/L (N 50–71). She was started on calcium carbonate 1000 mg TID and calcitriol 0.5 mcg BID and genetic analysis of the mother and sister revealed the same mutation compatible with Barakat syndrome. Compliance has been difficult, and when the patient transitioned to adult endocrinology three years later, she was on alfacalcidiol 2 mcg daily along with calcium carbonate 1500 mg daily and her labs were still suboptimal with a total corrected calcium of 1.82 mmol/L (N 2.22–2.54) and ionized calcium 0.98 mmol/L (N 1.16–1.29). Renal function determines the prognosis, and reassuringly her creatinine remains normal. Upon further questioning of the mother, she recalls that the patient had to be intubated for respiratory failure as a newborn, she had delayed milestones and also had seizure like activity during her infancy and early childhood. She had brought these symptoms to her family physician's attention however no further investigations were completed and serum calcium was not checked.

Conclusion: Early recognition of hypocalcemia symptoms is critical in identifying patients with congenital hypoparathyroidism, even more so when associated with other features that are part of complex familial syndrome such as Barakat syndrome.

1. Barakat, AJ. Barakat syndrome revisited. Am J of Med Genet A. 2018, Jun; 176(6):1341–13482. Barakat, AY. Familial nephrosis, nerve deafness and hypoparathyroidism. J Pediatri. 1977;9(1):61

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORT

Denosumab Induced Severe Hypocalcemia in a Patient With Metastatic Prostate Cancer

Kamrun Naher, MD, Komandur Thrupthi, MD, Kazi Samsuddoha, MD, Sneha Galiveeti, MD, Vivien Leung, MD
Bronx Care Health System, The Bronx, NY, USA.

Background: Denosumab can lead to severe hypocalcemia in patients with underlying risk factors such as vitamin D deficiency, low PTH, hypomagnesemia, and CKD. Denosumab is a monoclonal antibody against RANKL, reducing the activity of osteoclasts and thus reducing the release of calcium in the bloodstream causing hypocalcemia. Hypocalcemia can range from mild to severe symptoms requiring prolonged hospitalization. Medications such as zoledronic acid and Denosumab are known to reduce the occurrence of Skeletal related events (pathological fracture, spinal cord compression, and radiation to bone). For metastatic prostate cancer, about 90% can develop bone metastasis with significant morbidity and mortality [1]. Our patient presented with severe hypocalcemia after denosumab use without any above risk factors. **Clinical Case:** A 66-year-old male with a medical history of Prostate cancer with Metastasis to chest and bone presented to ER with syncope. Patient-reported poor oral intake, nausea, and vomiting for the last few days. In the ER, the patient was found afebrile, bp 116/76, HR 92, saturating 100% on room air. On examination, the patient was found lethargic, malnourished, foley in place due to chronic urinary retention. The abdomen was soft and non-tender. Laboratory findings were significant for Hb 9.1, Na 133, K3.6, bicarb 21, total calcium (Ca) 4.2, ionized Ca 0.63 and corrected Ca 5.4, magnesium 1.6, phosphorus 2.1, albumin 3.3, ALT 218, AST 229, ALP 1607. Lipase 82, Total bilirubin 1.5, direct bilirubin 0.8. Spot Urinary Ca 0.7, Vitamin D 25 OH 36.9, serum PTH 225 pg/mL. Serum cortisol AM 20.9, BUN 16, and serum Creatinine 1.0. The patient was started on 11g calcium gluconate in 1L dextrose @ 50c/hr and calcitriol 0.25mcg twice daily. Serum Ca level was monitored every 6 hours and reached 6.7. Later was started on Ca carbonate