Bone & Mineral Metabolism

Hypercalcemia In A Child On Ketogenic Diet For Intractable Epilepsy

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Introduction: Ketogenic diets have been shown to significantly reduce seizures in patients with intractable epilepsy refractory to common antiepileptic medications. There have been reports of hypercalcemia in diet-controlled epilepsy patients. The exact cause of hypercalcemia in ketogenic diets is unclear but decreased bone formation and impaired calcium excretion are suggested mechanisms.

Case: An 8-year-old male with Lennox-Gastaut syndrome was noted to have incidental hypercalcemia on admission (serum calcium 11.5 mg/dL, n = 8.8-10.1) for aspiration pneumonia, UTI, and increased seizure-like activity. Hypercalcemia persisted throughout his hospitalization (range: 10.8-12.4 mg/dL). Evaluation of hypercalcemia indicated normal serum phosphate (5.6 mg/dL, n = 3.0-6.0), appropriately low PTH (9.5 pg/mL, n = 10.0-65.0), undetectable PTHrP (<2.0 pmol/L), low 25 (OH) vitamin D (19ng/ml, n=30-100), low 1,25 (OH)2 vitamin D (7.1 pg/mL, n = 19.9-79.3), normal creatinine (0.3 mg/dL, n = 0.31-0.61), slightly elevated TSH (5.13 mIU/L, n = 0.5-4.5) with a normal free T4 (1.4 ng/dL, n = 0.8-2.0), and physiologic CBC. Alkaline phosphatase was low (88 U/L, n = 156-386) and skeletal survey showed diffuse demineralization. Urine calcium creatinine ratio was elevated at 0.6 when serum calcium was 11.6 mg/dL. Patient was 7 months into a ketogenic diet that successfully reduced seizure frequency and the plan was to further intensify the diet. On hospital day 11 when serum calcium was 12mg/dL, he was started on IV normal saline and furosemide up to a daily dose of 60 mg. On hospital day 12, patient was started on calcitonin (3 U/kg twice daily SQ). Serum calcium normalized (10 mg/dL), but calcitonin had to be discontinued due to severe nausea and vomiting. GI symptoms did not improve on a lower calcitonin dose. Calcium gradually corrected on fluids and furosemide, but hypercalcemia recurred (11mg/dL) after furosemide was weaned. The patient was then transferred to a higher level of neurologic care to continue treatment and increase the intensity of the ketogenic diet. There, on cumulative hospital day 26, he received zoledronic acid (0.0125 mg/kg) which was tolerated well and resulted in normalization of the serum calcium levels (10.9mg/dL, 10.2mg/dL and 9.8mg/dL on days 26, 28 and 29 respectively).

Conclusions: This case adds to the limited literature available on the evidence that management of refractory epilepsy with ketogenic diets may cause acute hypercalcemia. Although the underlying cause is unclear, decreased osteogenesis as marked by low alkaline phosphatase activity remains the likely explanation. Other risk factors such as immobilization also need to be explored. The successful implementation of protocols used to treat our patient and similar patients in the literature may help create screening protocols for early detection and management of the hypercalcemia to prevent complications.

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