

magnesium of 1.6, and a low-normal PTH of 18 which was the highest value ever recorded for this patient.

Conclusion: To our knowledge, this is the second reported case of spontaneous remission of idiopathic hypoparathyroidism in adulthood.

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A Remission of Hypocalcemia in Idiopathic Hypoparathyroidism

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Background: Acquired hypoparathyroidism is known to be transient when caused by surgical trauma but unlikely in idiopathic etiology. Herein we describe a unique presentation of idiopathic hypoparathyroidism that was diagnosed in early adolescence and remitted in adulthood.

Clinical Case: A male patient was diagnosed with idiopathic hypoparathyroidism at 13 years old after presenting with severe hypocalcemia resulting in generalized convulsive seizure. At diagnosis, his corrected calcium (cCa) was 6.7 (8.5-10.2 mg/dL) and PTH was 8.5 (15-65 pg/mL). Patient was noted to have oral candidiasis raising concerns for autoimmune regulator mutations associated with autoimmune polyendocrinopathy. Adrenal function was normal. Genetic testing was not done due to cost. Calcium citrate of 500 mg twice daily and calcitriol, gradually increased to 1.5 mcg daily, were initiated with normalization of calcium level. At age 24, he presented with altered mental status and was admitted with hypercalcemia and subarachnoid hemorrhage. He had cCa of 12.6 compared with previous calcium levels ~ 7 (8.5-10.2 mg/dL), ionized calcium of 1.57 mmol/L, magnesium of 1.8 (1.7-2.3mg/dL), hypophosphatemia of 0.4 (2.7-4.8 mg/dL) and undetectable PTH. Further testing showed AKI with creatinine of 2.1, from baseline of 0.8 (0.7-1.22 mg/dL). Vitamin D 25 OH and 1,25 dihydroxy vitamin D, ACE level and PTHrP were all unremarkable. Workup for multiple myeloma was negative. Adrenal and thyroid function tests were normal. CT brain showed diffuse subarachnoid blood with CTA showing an elongated saccular aneurysm measuring 2.57×1.72×1.85 mm. Hypercalcemia and AKI resolved with hydration. Interestingly, corrected calcium remained in the low-normal range between 8.6 - 8.8 (8.5-10.2 mg/dL), throughout 10 days of hospitalization despite holding all calcium citrate and calcitriol supplements, which were also held on discharge. PTH level prior to discharge was 7 (15-65 pg/mL) with cCa of 9.1 and magnesium of 1.7. At one week follow up, a reduced dose of calcium citrate of 250 mg twice a day and calcitriol 0.5 mcg daily were resumed which resulted in recurrent hypercalcemia and AKI with normalization by holding treatment. Repeated testing after one month showed cCa of 8.6, creatinine of 1.3, PTH of 17, magnesium of 1.7, and 25 OH vitamin D of 27.2 (31-80 ng/mL). Calcium citrate of 250 mg daily along with weekly ergocalciferol of 50,000 units and Magnesium Oxide 400 mg TID were initiated. Testing 8 months after the hospitalization revealed cCa of 8.6, creatinine of 1.2,