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Case Report

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Hyperparathyroidism Secondary to Long-Term Use of Lithium Carbonate

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Background

Lithium carbonate has been used for over half a century in the management of many psychiatric conditions like persistent depression, self-mutilation behaviors and bipolar disorder, where it has been proven to have benefit in suicide prevention [1]. Nevertheless, many side effects have been linked to its use, some of which include chronic tubule-interstitial disease, nephrogenic diabetes insipidus, thyroid dysfunction and, the focus of this case report, hypercalcemia and hyperparathyroidism [2].

Objective: to describe a case of hyperparathyroidism secondary to chronic lithium use in a patient with normal sestambi scan.

Case Report

Our patient is a 35 year-old male with past medical history of depression schizoaffective disorder (Bipolar type) that presented our hospital complaining of one-night insomnia and depressive thoughts with suicidal ideation. Patient's examination was benign although he had disorganized thoughts with intense affect and behavior. He was started on Lithium one year ago and, at the time, basic laboratory, studies were unremarkable. On presentation, laboratory test results were relevant for a Calcium level of 11.8 mg/dL (8.4-11 mg/dL) PTH level of 213 pg/mL (7.5-53 pg/mL), Vitamin D total 25 of 25 ng/mL (30-100 ng/mL), Lithium level of 0.7 mmol/L (0.6-1.2 mmol/L) and phosphorous level of 2.9 mg/dL (2.5-4.5 mg/dL). The patient was diagnosed with hyperparathyroidism and Endocrinology was consulted.

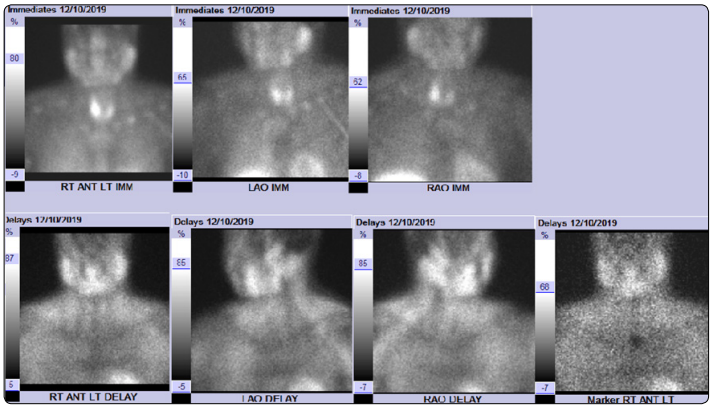

Image 1: Isotape is noted in thyroid and salivary glands in immediate images with washing out in delayed consistent with no evidence of parathyroid adenoma.

As treatment, generous amounts of Intravenous Normal Saline Solution and Vitamin D 800 U, daily, were initiated. Consideration of surgical evaluation was made; however, neck ultrasound and Parathyroid Scan with sestamibi by nuclear medicine for location of a parathyroid adenoma were obtained and resulted unremarkable (**Image 1**). The patient was transferred to a medical unit and the medication was stopped. Nonetheless, the patient denied further medical management and decided to have out-patient follow up endocrinology after being cleared from psychiatric unit.

Discussion

Primary hyperparathyroidism is the third most common endocrinopathy and is seen in 0.5% of the total general population [3]. Hypercalcemia alone has been seen in 4 to 50% of patients using Lithium, and up to 38% of them have hyperparathyroidism as demonstrated by elevated Parathyroid Hormone (PTH) levels [4]. The mechanisms by which this occurs have not been clearly elucidated. Some theories also are based on the fact that lithium might just unmask certain people's predisposition to develop hyperparathyroidism by unknown ways [3, 4]. It has been noted that lithium changes the set point at which the calcium sensing receptors (CaSR) suppress the release of PTH [3]. It has also been proposed that Lithium inhibits glycogen synthase kinase 3 (GSK-3), which is imperative for apoptotic purposes, and that this is the mechanism by which adenoma formation can be best explained [3].

Management of hypercalcemia secondary to lithium use varies greatly based on the presentation. A simple and reasonable way to start is by stopping the medication and closely monitor for improvement of calcium levels and hyperparathyroidism. Nevertheless, many times this is not possible given the severity of the rebound psychiatric symptoms that are well controlled by lithium [3]. If hypercalcemia is refractory to drug cessation and adenomatous disease is suspected, surgical resection by focused or bilateral neck exploration with single or total parathyroidectomy [5]. Use of calcimodulators for normalization of calcium levels, as cinacalcet, has also been explored with satisfactory results as some case reports [2].

Conclusion

We had an interesting and rare case of a young male that presented to our hospital with psychiatric complaints and was noted to have elevated calcium, and parathyroid hormone suggestive of hyperparathyroidism. Primary hyperparathyroidism is an uncommon

and not so well known side effect of lithium. Hyperparathyroidism with hypercalcemia that can lead a myriad of pathologies involving multiple organ systems for which we suggest routine monitoring of said levels if initiation of Lithium is required. When possible, cessation of lithium use, as well as surgical intervention with adenoma resection if so indicated, are the mainstays of treatment of these patients.

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