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Hyperparathyreoidism and lithium therapy

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Abstract

Long-term lithium therapy can lead to hyperparathyroidism-HPT with hypercalcemia and hypocalciuria [1,2]. The mechanisms that lead to parathyroid hyperplasia are multiple (increased calcium sensitivity treshold at the parathyroid glands-CasR, direct hyperproduction of parathyroid hormon-PTH, inhibition of transmembranous calcium transport) [3]. We presented a patient with a rare complication of lithium therapy.

Introduction

Lithium has proven effectiveness in the treatment of bipolar disorder and has been associated with a variety of renal impairments including urinary acidic disorder, chronic interstitial nephropathy, nephrotic syndrome, nephrogenic diabetes insipidus- NDI [4].

The most reported side effect is NDI (down-regulation of aquaporin 2 -AQP2 with direct antidiuretic hormone-ADH resistance) [5] with an incidence of about 12% [6].

Literature data suggest that in a smaller number of cases it may also cause hypercalcemia with or without HPT [1,7,8], as has been shown in our case.

A possible therapeutic approach to this disorder is the administration of cinacalcet [1,8].

Case report

Female patient, 55 years old, has been receiving psychiatric treatment for almost two decades for bipolar disorder. She has been taking lithium over the entire period combined with other psychiatric medications.

She is now hospitalized with acute renal impairment and physical finding of a vasculitis rash on the extremities (at admission: urea 7.8mmol / L, creatinine 158 umol / L, K 3.5 mmol / L, urine sediment: E +++, proteins +++, BIURET 2.87 g / dU).

A kidney biopsy was diagnosed with IgA-vasculitis (M1, E1, S0, T0, C0) and corticosteroid treatment was started (Pozzi's scheme) with good clinical and laboratory response.

During monitoring, elevated serum calcium (2.74mmol / L) as well as PTH (8.74pmol / L) were verified. There were no signs of nephrolithiasis on the kidney ultrasound.

Further investigation eliminated primary HPT. In the absence of other causes of HPT and as high PTH as well as serum Ca are probably due to the effect of lithium on CaSR on the parathyroid glands, while increased urinary calcium excretion is a result of currently normal renal function (ClCr 68.1 ml / min), we concluded that this disorder is most likely due to lithium therapy.

Discussion

The patient was taking lithium as a basic therapy for psychiatric disorder with adequate remission over a long period of time. She was taking 2-3 pills of Li-CO3 per day and blood concentration was within the normal range (0.6-1.09) and developed rare complication. We consider that in order to achieve early detection of this type of complications patients on chronic lithium therapy should be monitored for serum electrolytes and iPTH. This report can and should serve as starting point for grand scale prospective study which should determine probability and frequency of developing lithium induced hyperparathyroidism.

Conclusion

We presented a patient initially treated for acute renal impairment on the basis of histologically confirmed IgA-vasculitis. Previously, she was in good remission of bipolar disorder on chronic lithium therapy with the incidental finding of a rare complication at the parathyroid level and excluding other causes of HPT.

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