Normocalcaemic Secondary Hyperparathyroidism due to Vitamin D Deficiency with higher offset point

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Introduction

Vitamin D deficiency occurs more frequently in patients with primary hyperparathyroidism compared to general population, and is usually associated with an aggravated form of the disease. Current guidelines recommend measurement of vitamin D level in all patients with primary hyperparathyroidism, and their repletion if the levels are less than 50nmol/L.

Case Presentation

56 years old Caucasian lady who underwent right parathyroidectomy & thyroid lobectomy for right parathyroid carcinoma in 1998. Her initial serum PTH level was 48.2pmol/L (1.1-6.4). Postoperatively it fell to 7.5pmol/L, but never returned to normal. She was followed up in endocrine clinic for persistent normocalcaemic hyperparathyroidism without evidence of recurrent parathyroid cancer. In January 2010 her PTH level was 7.8pmol/L with concomitant 25OH vitamin D level being 80nmol/L. She was started on Ergocalciferol 800iu and Calcium 1g daily and, after six months, PTH level had fallen to 4.3pmol/L, suggesting secondary hyperparathyroidism, but she was unable to tolerate Calcium supplements long-term. In March 2012, PTH level was again elevated at 6.6pmol/L despite 25OHD 86nmol/L. She was advised to continue with same dose of vitamin D supplement. In March 2014, PTH had increased further to 8.6nmol/L despite 25OHD 107nmol/L. This time it was decided to increase the dose of vitamin D to 2000 IU per day.
After six months of treatment with increased dose of vitamin D, her parathyroid level normalized to 4.6pmol/L with 25OHD level 152nmol/L. In this patient with history of parathyroid cancer, attainment of a serum 25OHD significantly above recommended “healthy” level was required to normalize serum PTH level.

Results

Parathyroid levels normalized after achieving a serum 25OH D level significantly higher than “healthy” recommended.

Conclusions

Contrary to previous assertions, there is no consistent population 25OHD threshold required to maintain normal-range PTH levels. Some patients require much higher levels to normalize serum PTH.
Instead, there is huge inter-individual variation that likely reflects both dietary calcium intake and genetic predisposicion. We speculate that individuals with abnormally-high 25OHD thresholds for suppression of PTH secretion may be predisposed to chronic PTH hypersecretion, leading to parathyroid gland hyperplasia and, ultimately, autonomous secretion in relation to adenomatous change, or very rarely, carcinoma.

References

J Clin Endocrinol Metabolism 2014 Oct;99(10):3561-9. doi: 10.1210/jc.2014-1413.
Ann Endocrinol (Paris). 2015 Apr 24. pii: S0003-4266(15)00043-8. doi: 10.1016/j.ando.2015.03.022.
Lancet Diabetes Endocrinol. 2013 Dec;1(4):329-40. doi: 10.1016/S2213-8587(13)70083-4.
J Clin Endocrinol Metab. 2012 Nov;97(11):3989-95. doi: 10.1210/jc.2012-2276.

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