Introduction

Vitamin D deficiency is common in ethnic minority living in the UK. It is a relatively easily treatable condition. However, if left untreated can lead to tertiary hyperparathyroidism.

CASES

Case 1

46 yrs. old man of Somali origin was referred to endocrine clinic with severe hypercalcaemia level were 2.90. Parathyroid hormones (PTH) 625 pg/ml. Vitamin D level were less than 7.

His PTH level has improved to 249 post Vitamin D replacement, however his calcium level was 2.96.

An ultrasound (Fig 4) of his parathyroid gland identified 2 adenomas, MIBI scan (Fig 1,3) consistently 2 parathyroid adenoma’s removed.

Histopathology Parathyroid -One weighs 2.3 grams and other 0.8 grams. The appearances suggest a parathyroid gland adenoma (Fig 3).

Case 2

48 yrs. old lady of Indian origin initially presented with hypercalcaemia. Calcium 3.7and PTH of 800 noticed to have severe vitamin D deficiency < 10. Both USS and MIBI scan showed Right parathyroid adenoma. Right inferior parathyroid gland was removed weighing 0.324 grams Histopathology keeping with parathyroid adenoma.

Case 3

60 year old lady of Indian origin presented with severe vitamin deficiency, noted to have high calcium 3.2 PTH of 639.Both USS and MIBI scan showed left inferior parathyroid adenoma.

Had vitamin D replacement and left inferior parathyroid adenoma removed weighing 0.525 grams. Histopathology consistent with adenoma.

Vitamin D replacement:

All 3 patients were treated with high dose colecalciferol replacement, 40,000 units for 10 days pre-operatively. None of these resulted in any significant rise in calcium in these patients.

Biochemical results pre and post- op

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Aetiology

The aetiology is unknown but may be due to monoclonal expansion of parathyroid cells (nodule formation within hyperplastic glands). A change may occur in the set point of the calcium-sensing mechanism to hypercalcemic levels. Four-gland involvement occurs in most patients. Tertiary hyperparathyroidism is observed most commonly in patients with chronic secondary hyperparathyroidism and often after renal transplant. The hypertrophied parathyroid glands fail to return to normal and continue to over secrete parathyroid hormone, despite serum calcium levels that are within the reference range or even elevated.

Here we report 3 cases of severe Vitamin D deficiency who were untreated for a prolonged period, which led to progression from secondary to autonomous parathyroid over secretion, tertiary hyperparathyroidism.

Discussion

Vitamin D is known to have a suppressive effect on parathyroid cell proliferation and parathyroid hormone synthesis. Vitamin D deficiency may result in a compensatory increase in the secretion of parathyroid hormone (secondary hyperparathyroidism) which involves hyperplasia of all four parathyroid glands. Secondary hyperparathyroidism can become autonomous and this has been termed tertiary hyperparathyroidism, the underlying pathology of which has been variably described in the literature as adenoma formation or four gland hyperplasia. The pathogenesis of parathyroid adenoma formation in vitamin D deficiency remains unclear. It is possible that a proportion of cases represent the coincidence of primary hyperparathyroidism in patients with vitamin D deficiency. Alternatively, we hypothesise that autonomous four gland hyperplasia or tertiary hyperparathyroidism. Tertiary disease is characterized by the development of autonomous hypersecretion of parathyroid hormone causing hypercalcemia.

Figures

Figure 1

Figure 2

Figure 3

Figure 4

References

1-What is Tertiary Hyperparathyroidism/ Australian and New Zealand Journal of Medicine

P. J. Somerville‡, D. J. Tiller‡, R. A. Evans§

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2-Vitamin D ADVICE ON SUPPLEMENT FOR AT RISKS

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Contact Information

Mahmud Ahmad STR Diabetes/ Endocrinology
Royal Bolton Hospital
mahmudala@yahoo.com