CASE REPORT

Vitamin D deficiency of unknown etiology

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Abstract

We report on a 68-year-old Caucasian male who was referred to our Metabolic Clinic with a very low 25 hydroxy-vitamin D (25(OH)D) level of 4.0 ng/ml. History did not reveal any medications know to interfere with 25(OH)D metabolism or history of bone pains or weakness and clinical examination did not reveal myopathy. Relevant laboratory testing confirmed the low 25(OH)D with hypocalcinuria. All other laboratory tests were essentially normal. He was started on oral Vitamin D supplementation and had an excellent response with his most recent level at 35.1 ng/ml. Since we could not find an obvious cause for his Vitamin D deficiency we speculate that it is possibly due to his obesity and suggest that obese persons be screened for Vitamin D deficiency.

Key words

Vitamin D, Parathyroid hormone, Malabsorption, Obesity, Calcium

1 Case presentation

A 68-year-old Caucasian male was referred to the Metabolic Clinic at the Veterans Affairs Medical Center for management of Vitamin D deficiency. When he was referred he had a 25 hydroxyvitamin D (25(OH)D) level of 4.0 ng/ml. Vitamin D deficiency is defined as a level < 20 ng/ml [1]. History did not reveal any muscle weakness and he was not taking any medications know to interfere with Vitamin D metabolism such as anticonvulsants, glucocorticoids, medications for AIDS *etc*. Also he did not admit to any symptoms of malabsorption and was eating a normal diet and obtaining adequate sun exposure living in Northern California. His past history was positive for hypertension and osteoarthritis. The medications he was taking included amlodipine 10 mg/d and hydrochlorthiazide 25 mg, triamaterene 37.5 mg/d. Physical examination revealed a normal healthy male with a weight of 181 lbs a body mass index of 30.2 kg/m^2 a BP of 130/70, with normal cardiac and chest examinations. Also he had no evidence of proximal myopathy. As shown in Table 1 he has calcium, phosphate and alkaline phosphatase levels in the reference ranges. His 24 hour urine calcium was decreased. He had a normal urinalysis, serum creatinine and liver function tests. Both Parathyroid hormone (PTH) and 1.25 dihydroxy Vitamin D levels were normal. His DEXA scan revealed normal bone mineral density. Furthermore he had a normal hemoglobin of 17.1 (14 g/dl -18 g/dl), percent iron saturation of 43% (11%-46%) and a ferritin of 289 (22 ng/ml – 415 ng/ml). Also Vitamin A levels were normal 51 (18 µg/dl – 77 µg/dl). Screening for celiac disease revealed normal titers of tissue transglutaminase and endomysial antibodies. His repeat 25(OH)D level was 14 ng/ml (Supplement use could not be ruled out at this stage). Based on his low 25(OH)D levels and decreased urinary calcium excretion a diagnosis

of Vitamin D deficiency was made and he was started on treatment. Initially he received 50,000 units weekly for 6 weeks then 2,000 units/d and is presently on 1,000 units/d. He had an excellent response with levels increasing serially to 28.1, 32 and most recently 35.1 ng/ml. Also his 24 hour urinary calcium is normal (125.8 mg/d).

Table 1. Baseline laboratory tests (Reference ranges in parentheses)

25(OH)D (30-100 ng/ml)	4, 12.1
1,25DihydroxyD (10-75 pg/ml)	29.5, 35.4
Calcium (8.7-10.2 mg/dl)	9.4, 9.2
24 Hour Urinary Calcium (100-300 mg/d)	22.6
Phosphate (2.4-4.5 mg/dl)	3.3
Magnesium (1.8-2.5 mg/dl)	2.1
PTH (12-88 pg/ml)	72.5, 50
Creatinine (0.5-1.1 mg/dl)	0.87, 0.60
BUN (8-26 mg/dl)	8.0, 9.0
Albumin (3.3-4.8 g/dl)	4.0, 3.9
Bilirubin (0.3-1.2 mg/dl)	0.7, 1.0
AST (8-42 IU/L)	35
ALP (37-107 IU/L)	60, 54

2 Discussion

The major interest in the present case study is the fact that this patient has Vitamin D deficiency with no obvious cause. He was not on any medications known to cause Vitamin D deficiency, had normal kidney and hepatic function and had an excellent response to oral supplementation arguing against malabsorption. Furthermore other tests done to rule out other deficiencies revealed normal hemoglobin, normal iron studies, albumin and normal Vitamin A levels *etc*. The only possible explanation for his Vitamin D deficiency could be his obesity [1, 2]. It is argued that increase fat sequesters the Vitamin D [2, 3]. Usually with this degree of Vitamin D deficiency one with expect secondary hyperparathyroidism. Although his PTH levels was not elevated above the reference range they were in the higher end of the reference range (50 pg/ml and 72.5 pg/ml) and it could be argued that prior to his Vitamin D deficiency his PTH level was much lower *i.e*. functional hyperparathyroidism. In fact following supplementation his most recent PTH level is now 34 pg/ml. Also we have previously reported low Vitamin D levels with values below 20 ng/ml in 30% of patients with Metabolic Syndrome and 8% of controls [4]. In that study we had no obvious explanation since there was no drug causes and all patients had normal renal and hepatic function however like in the present patient the average BMI was consistent with obesity (30.1 in controls and 35.1 in patients with Metabolic Syndrome).

3 Conclusion

We report on a patient with obvious Vitamin D deficiency who had an excellent response to oral supplementation and suggest that in obese persons, Vitamin D status should be assessed and deficiency treated for optimum bone health and its evolving but unproven role in the pathogenesis of diabetes and cardiovascular disease [5].

References

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