Vitamin D in high dose, 600,000 UI, is necessary to achieve normal blood levels.

03/05/10

Effect of a Single ‘Megadose’ Intramuscular Vitamin D (600,000 IU) Injection on Vitamin D Concentrations and Bone Mineral Density Following Bilipancreatic Diversion Surgery.


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Background: Vitamin D (VitD) deficiency is common following biliopancreatic diversion (VitD3, Arachitol Solvay Pharmaceuticals) as an adjunct to regular oral VitD supplementation (CitraCalH-D) for a period of 12 months following VitD surgery. METHODS: Some 29 patients who had undergone BPD during 2000-2005 were recruited and received a single injection of 600,000 IU of cholecalciferol. Venous blood VitD, parathyroid hormone (PTH), alkaline phosphatase (ALP), ionised calcium and urinary N-telopeptide (NTX) were assessed at baseline and at 1.5, 3, 6, 9 and 12 months post-injection. Bone mineral density (BMD) was determined at baseline and 12 months post-injection. RESULTS: VitD concentrations (mean +/- SD) were significantly increased from baseline values (61.5 +/- 18.8 nmol/L) at 1.5 months (92.4 +/- 21.5, p < 0.001), 3 months (100.5 +/- 24.4, p < 0.001) and 6 months (79.1 +/- 20.9, p = 0.014) post-injection, with non-significant elevations at 9 months (73.3 +/- 15.1, p = 0.248) and 12 months (73.4 +/- 17.3, p = 0.278). The proportion of patients with ‘normalised’ VitD levels was significantly higher at all post-injection time points (range, 93-100%) compared with baseline (71.4%, p < 0.01). Ionised calcium and ALP remained within normal levels at baseline and all follow-up time points, although ionised calcium decreased by 3.4% (p = 0.015) and ALP increased by 14.6% (p = 0.021) at 12 months compared with baseline. No significant change in PTH, NTX or BMD was observed. CONCLUSIONS: Intramuscular cholecalciferol injection, as an adjunct to oral supplementation, appears a safe and effective method to increase and maintain VitD levels after BPD.

PMID: 19949888

Annual intramuscular injection of a megadose of cholecalciferol for treatment of vitamin D deficiency: efficacy and safety data.

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AIM: To evaluate the efficacy and safety of an annual intramuscular injection of cholecalciferol for vitamin D deficiency. DESIGN: Prospective open-label study. PARTICIPANTS: Five men and 45 women (mean age 66.3 years) with vitamin D deficiency who were given a single therapeutic intramuscular injection of 600,000 IU (15 mg) cholecalciferol (vitamin D3). OUTCOME MEASURES: Serum levels of calcium, creatinine, 25-hydroxyvitamin D3 (25OHD3) and parathyroid hormone, as well as early morning 2-hour urine calcium/creatinine excretion index. Specimens were collected at baseline and after 4 and 12 months of therapy. Data are reported as mean +/- SD. RESULTS: Vitamin D deficiency was severe (< 12.5 nmol/L) in one participant, moderate (12.5-24 nmol/L) in 14, and mild (25-49 nmol/L) in 35. Twenty-four participants (48%) had secondary hyperparathyroidism. Following intramuscular cholecalciferol injection, serum 25OHD3 levels normalised in all participants and remained above 50 nmol/L throughout the study. Serum 25OHD3 levels were significantly higher at 4 months (114 +/- 35 nmol/L), and 12 months (79.1 +/- 20.9, p = 0.014) post-injection, with non-significant elevations at 9 months (73.3 +/- 15.1, p = 0.248) and 12 months (73.4 +/- 17.3, p = 0.278). The proportion of patients with ‘normalised’ VitD levels was significantly higher at all post-injection time points (range, 93-100%) compared with baseline (71.4%, p < 0.01). Ionised calcium and ALP remained within normal levels at baseline and all follow-up time points, although ionised calcium decreased by 3.4% (p = 0.015) and ALP increased by 14.6% (p = 0.021) at 12 months compared with baseline. No significant change in PTH, NTX or BMD was observed. CONCLUSIONS: Once-yearly intramuscular cholecalciferol injection (600,000 IU) is effective therapy for vitamin D deficiency. While this therapy appears to be safe, the potential for developing hypercalcemia needs to be examined in a large randomised controlled trial.

PMID: 15992330


Calcium homeostasis in 40 adolescents with beta-thalassemia major: a case-control study of the effects of intramuscular injection of a megadose of cholecalciferol.

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The prevalence of disturbed homeostasis in adolescents with beta thalassemia major (T) varies among different populations. Moreover, the cholecalciferol uptake required to achieve or maintain any given serum 25-hydroxycholecalciferol level is not well known, particularly within ranges of the probable physiologic supply of the vitamin. OBJECTIVES: In this prospective study we measured parameters of calcium homeostasis in 40 adolescents with T and 40 matched non-thalassemic (NT) controls. An IM dose of vitamin D3 on serum 25-hydroxycholecalciferol (25-OH D) concentration and other calcium homeostasis parameters in vitamin D deficient (VDD) thalassemic adolescents, and to compare these results with those for non-thalassemic adolescents with VDD. DESIGN: In this prospective study we measured parameters of calcium homeostasis in 40 adolescents with T and 40 matched non-thalassemic (NT) controls. An IM dose of vitamin D3 on serum 25-hydroxycholecalciferol (25-OH D) concentration and other calcium homeostasis parameters in vitamin D deficient (VDD) thalassemic adolescents, and to compare these results with those for non-thalassemic adolescents with VDD. RESULTS: Of the 40 adolescents with T, 2 had hypoparathyroidism and low 25-OH D, and 2 had hypocalcemia with hypophosphatemia, high alkaline phosphatase (ALP), high PTH and serum 25-OH D below normal levels. The rest of the patients (n=36) had low circulating 25-OH D concentrations with normal serum Ca and P04 concentrations. Of the 40 non-thalassemic adolescents, 26 had 25-OH D levels below 20 ng/ml (65%). Patients with T and VDD had lower circulating PTH

and ALP concentrations compared to non-thalassemic patients with VDD. Significant improvement of symptoms related to vitamin D deficiency was reported in 18 out of 26 of symptomatic T and 12 out of 16 of NT adolescents at 1 to 3 months after the injection. Three months after injecting vitamin D the mean serum 25-OH D concentration was lower in the T group as compared to the NT group but the majority of patients had 25-OH D levels equal to or greater than 20 ng/ml. CONCLUSION: Vitamin D deficiency was detected in 100 % of our thalassemic adolescents. An IM injection of a mega dose of cholecalciferol is an effective therapy for treatment of hypovitaminosis D in thalassemic and non-thalassemic adolescents for 3 months but its effects do not persist for 6 months. PMID: 19337170


Bone mineral density in response to two different regimes in rickets.

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The aim of this study was to compare the bone mineral density (BMD) of two different treatment regimens in infants with nutritional vitamin D deficient rickets (VDR). Ten patients (Group 1) were treated with a single dose of 600,000 IU of oral vitamin D3 and another ten patients (Group 2) were treated with 20,000 IU/day of oral vitamin D3 for 30 days. BMD was measured in the lumbar spine twice in all infants before the treatment and on the 31st day after initiating the treatment. The increases of BMD after treatment compared to pretreatment levels were statistically significant in both groups (P = 0.005 in Group 1 and P = 0.047 in Group 2). The increments of BMD were statistically similar between Group 1 and 2 (P = 0.096). The present study suggests that these two different treatment regimens bring about similar healing in BMD. PMID: 16735766


Comparison of low and high dose of vitamin D treatment in nutritional vitamin D deficiency rickets.

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In this study, we compared three different therapy modes (150,000 IU, 300,000 IU, and 600,000 IU vitamin D p.o.) in infants with nutritional vitamin D deficiency rickets (VDR). Our purpose was to determine the most effective dosage of vitamin D with least side effects for treating VDR. The study included 56 infants, 3-36 months of age, with nutritional VDR and 20 age-matched control infants. In all infants, serum calcium, phosphorus, alkaline phosphatase, magnesium, serum 25-hydroxycholecalciferol, plasma intact parathormone levels and urinary Ca/creatinine ratio were determined. Of 56 patients, 52 were able to be followed long-term. These patients were reexamined on the 3rd day, 7-10th day, and 25-30th day after treatment. On the 30th day post-treatment, we did not find any difference between the doses in the improvement of rickets. However, hypercalcemia was present in eight infants who had been administered 300,000 IU (two infants) and 600,000 IU (six infants) of vitamin D. In conclusion, our findings showed that 150,000 IU or 300,000 IU of vitamin D was adequate in the treatment of VDR, but 600,000 IU of vitamin D may carry the risk of hypercalcemia. PMID: 14594170


Unique form of rickets with low serum 25-hydroxyvitamin D in two normally nourished children.

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We present an unusual type of rickets involving two children: a 2 year old boy and a 15 month old boy, who presented with marked bowing of the lower extremities and bulging of costochondral junctions. Both children had normal growth, with their height and body weight greater than the 50th and 97th percentile for age. Roentgenograms of their extremities showed the typical changes of vitamin D refractory rickets. Serum alkaline phosphatase levels were elevated and serum levels of calcium and phosphates were both within the normal range. No primary cause for the rickets, including nutritional deficiencies, was found in the two patients. Characteristic findings were persistently low serum 25-hydroxyvitamin D (25-OH-D) and normal 1,25-dihydroxyvitamin D (1,25-(OH)2-D). Improvements in clinical and X-ray findings were observed after either oral administration of 1 alpha-(OH)-D3 (9-15 micrograms per day) or massive vitamin D2 therapy (600,000 IU single injection). The low serum levels of 25-OH-D did not increase unless massive vitamin D2 therapy was also given. These two cases represent a unique form of rickets that does not meet the criteria for any type of previously known rickets.

PMID: 7793252


Effect of vitamin D3 administration on serum 25-hydroxyvitamin D3, 1,25-dihydroxyvitamin D3 and osteocalcin in vitamin D-deficient elderly people.

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In elderly institutionalized people, confined to bedroom and receiving no vitamin D supplementation, the frequency of vitamin D deficiency is found very high. Systematic administration of vitamin D has, therefore, been proposed to correct vitamin D deficiency. Within this context, we studied 40 elderly institutionalized subjects (mean age 80.5 ± 7.2 yr) with low 25(OH)D3 concentrations (4.4 ± 1.8 micrograms/l). Sixteen of them (Group I) had low serum calcium concentrations (less than 2.3 mmol/l) and 24 (Group II) had normal serum calcium concentrations (from 2.3 to 2.6 mmol/l). As hypocalcemia has been shown to regulate 1,25(OH)D3 production independent of PTH in animals and in humans, we compared their respective responses to the administration of vitamin D3. Subjects received a total dose of 15 mg (600,000 IU) of vitamin D3 divided into 3 i.m. injections at one month intervals and were explored before therapy and one and 6 months after the last dose of vitamin D3. The treatment induced a similar marked rise in 25(OH)D3 levels (from 4.1 ± 1.7 to 24.4 ± 8.7 micrograms/l for group I and from 5.1 ± 1.8 to 27.2 ± 8.0 micrograms/l for group II) in both groups but increased the 1,25(OH)2D3 concentrations only in group I (from 22.9 ± 6.9 to 32.6 ± 11.3 ng/l). Meanwhile serum calcium concentrations rose in group I (to normal range i.e. 2.31 ± 0.07 mmol/l) and were unaffected in group II. These results suggest that hypocalcemia is a potent stimulator of renal 1-hydroxylase in elderly people. Furthermore, a transient significant (P less than 0.01) increase in serum osteocalcin (from 10.6 ± 4.1 to 14.1 ± 5.9 micrograms/l) could be observed in group II which demonstrates for the first time that the osteocalcin response of osteoblasts to stimulation by 1,25(OH)2D3 is retained in very old people.

PMID: 2559250


Intermittent high-dose vitamin D prophylaxis during infancy: effect on vitamin D metabolites, calcium, and phosphorus.

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In infants receiving intermittent high dose vitamin D prophylaxis (600,000 IU ergocalciferol per dose orally) every 3-5 mo, the serum concentrations of vitamin D metabolites, calcium (Ca), and phosphorus (P) were determined before and 2 wk after each dose. The
25-hydroxyvitamin D (OHD) concentrations increased to well above normal but the values returned to the normal range before each subsequent dose. The 24,25- and 25,26-dihydroxyvitamin D ([OH]2D) levels followed a pattern similar to that of 25-OHD, and both were closely related to the latter ($r = 0.85$, $p$ less than 0.005, and $r = 0.84$, $p$ less than 0.005, respectively). The 1,25-(OH)2D concentrations did not vary in a consistent pattern and remained largely within the normal range. All infants had normal Ca levels before the first dose but 14 infants (34%) later had one or both Ca values above the upper normal limit of 2.60 mmol/L (2.81-3.32 mmol/L), indicating that the vitamin D doses were excessive despite the lack of accumulative increases in serum vitamin D concentrations.

PMID: 3499065