

DO VITAMIN D SUPPLEMENTS IMPROVE THE PHYSICAL CAPABILITIES OF ELDERLY HOSPITAL PATIENTS?

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Summary

A randomized double-blind controlled trial of the effect of vitamin D supplementation on the abilities of elderly hospital patients to carry out basic activities of daily life is described. Those patients included in the trial had plasma 25-hydroxyvitamin D concentrations which were low or low normal as judged by the normal range in young adults. After 2 to 9 months on the trial there was no significant difference in the performance of the control and treatment groups.

INTRODUCTION

Many patients in long-stay geriatric wards have very low plasma 25-hydroxyvitamin D [25(OH)D] concentrations (1), due to a combination of poor sunlight exposure, inadequate diet, malabsorption of vitamin D and subsequent defects in hydroxylation (2-6). The low plasma 25(OH)D can be corrected by dietary changes, administration of vitamin D and by providing a low intensity ultraviolet source in the patient's day room (7). However, before advocating such policies it was felt important to determine whether raising the plasma 25(OH)D actually resulted in an improvement in the patient's physical capabilities. This paper describes a randomized double-blind controlled trial of the effect of oral vitamin D supplements on the ability of elderly hospital patients with low or low normal plasma 25(OH)D to perform basic activities of daily living.

Patients and Methods

Three hundred and two patients in the geriatric wards of four hospitals who had been in-patients for a period exceeding four weeks and were judged likely to stay for at least a further eight weeks were suggested by the participating clinicians for inclusion in the trial. In addition, 18 patients attending a geriatric day hospital were also considered. The following were exclusion criteria: (a) overt clinical osteomalacia, either plasma calcium less than 1.95 mmol/l or Looser's zones, or on calciferol therapy (9 patients); (b) a judgement that he or she was unlikely to be able to co-operate in the trial (144 patients); (c) plasma creatinine more than 150 µmol/l, potassium less than 3.3 mmol/l (14 patients);

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(d) plasma 25(OH)D more than 40 nmol/l (16 ng/ml) (50 excluded); (e) refused consent or unable to give informed consent (21 patients). Those 82 patients remaining (which included six from the day-hospital) were then further characterized according to the presence or absence of (i) stroke, (ii) painful arthropathy, (iii) Parkinsonism, (iv) a chairbound state, (v) low or high score in the mental assessment test (see below) and (vi) low or low normal plasma 25(OH)D (0–20, 21.1–40 nmol/l). The patients were then randomized into two groups (control and treatment) by a computer program which aimed to provide approximately equal numbers of patients with the above disorders in the two groups (see below). A base-line activity of daily living (ADL) assessment (see below) was then made by an occupational therapist, a further blood sample taken for measurement of plasma 25(OH)D, urea and electrolytes, albumin, calcium, phosphorus and alkaline phosphatase, and administration then started of either 9000 units vitamin D₂ (treatment group) or a similar number of tablets of identical appearance containing lactose as a placebo (control group). Further ADL and mental assessments were made at successive intervals of approximately 2½, 6, 10, 16, 24, 30 and 40 weeks after the start of treatment.

The following were pre-established criteria for withdrawal from the trial: (i) failure to complete eight weeks of the trial (17 patients), (ii) development of overt osteomalacia, (iii) rise of plasma calcium to more than 2.75 mmol/l or fall to 2.1 mmol/l, (iv) a period of unwillingness to co-operate lasting more than six weeks, (v) inability to take tablets for a period greater than four weeks because of intercurrent illness. Neither the patients nor those involved in their assessment or care were aware of the nature of the tablets administered. Although the occupational therapists were aware of the plasma 25(OH)D values before the start of the trial, the subsequent values were unknown to them, and to all other staff involved in their care.

The ADL assessment comprised three groups of tasks, designed respectively to assess mobility, self-care and domestic capabilities. These tasks were: (a) ability to sit on edge of bed from the supine position, to transfer from bed to chair, and to rise from chair to standing position (using three different chair heights), (b) putting on a shirt or cardigan, putting on sock or stocking, combing hair, (c) pouring from a standard teapot containing 0.57 kg water, (d) filling and lifting a standard kettle containing 1.71 kg water, (e) walking 5 m indoors, (f) climbing up and down standard stairs (Rank Stanley Cox) twice.

The assessment and scoring was based on the Northwick Park ADL system (8, 9). Scoring was done in two ways; in the first, a score of 1, 2 or 3 was given according to whether the patient demonstrated total independence, partial independence or total dependence, respectively, for the task concerned. A score of 3 was also given when a patient judged able to perform a task refused to do so. The second method of scoring was by timing the performance of the task in those who were partially or totally independent. Because the different tasks were judged of varying importance in determining the overall ability of the patient, each task was given a weighting factor related to the

Table 1. Weightings applied to task scores to derive the 'independence' and 'muscle function' indices

	<i>Independence</i>	<i>Muscle function</i>
Transfer: sit on edge of bed from supine	10	9
Transfer: from bed (50 cm high) to chair (47 cm high)	10	8
Transfer: chair to standing, chair height 53 cm	10	4
47 cm	0	6
36 cm	0	10
Dressing: shirt/cardigan	7	0
Grooming: combing hair (including back of head)	7	3
Cooking: fill and move standard kettle	3	0
pouring tea	3	0
Mobility: walking 5 m, with aids if necessary	10	0
stairs	0	10

degree of importance as perceived by the participating clinicians; this weighting factor, which was decided before any data analysis was undertaken, was applied to each task score in order to arrive at two overall scores for each assessment, one principally reflecting *muscle function* and the other mainly indicating the patient's degree of *independence*. The tasks contributing to each of the two overall scores and the weights attributed to them are shown in Table I.

The mental assessment was performed to help determine whether any changes in mental state occurred which may have influenced ability to perform the tasks, and consisted of a short questionnaire (10) followed by a simple practical test of cognitive function designed by Silver (11). The maximum score in the two parts of the test was 22 marks (10+12), scores of 12 and above being regarded as 'high' and those under 12 as 'low'.

Blood samples were obtained at 6-week intervals for measurement of plasma urea, creatinine, electrolytes, albumin, calcium, alkaline phosphatase and 25(OH)D. The last of these was estimated by the method of Haddad and Chyu (12) (lower limit of winter reference range in non-elderly adults—20 nmol/l) and the remainder by standard automated techniques. Plasma calcium (Ca_p) values were corrected (Ca_{corr}) for deviation from the mean laboratory normal plasma albumin value (46 g/l) by the following formula:

$$Ca_{corr} \text{ (mmol/l)} = Ca_p + 0.02 (46 - A)$$

where A is the measured plasma albumin (g/l).

Ethics Committee approval was given for the study and written informed consent was obtained.

Statistical and allocation methods

The procedure for allocation of patients to control and treatment groups was a standard method of minimization (25) and used a FORTRAN program written by one of the authors (S.E.). Comparison between the treatment groups was by chi-squared tests for categorical variables. Continuous variables were analysed using *t* tests for independent samples. As there were no significant changes in the ADL index scores, it was justifiable to use the most powerful parametric statistical tests for these scores as well as for continuous variables. Non-parametric regression (26) was used to estimate average changes in plasma 25(OH)D, 'muscle strength', and 'independence' scores. The slopes derived by this method were analysed using Spearman correlation.

RESULTS

Table II shows the mean age, sex and major diagnostic categories in the control and treatment groups and their initial mean mental assessment and ADL scores. Close clinical matching was achieved. The number completing all seven assessments was just significantly less in the treatment group than in the control group. This was principally due to the greater number of withdrawals in the treatment group (see Table III), each for one of a variety of reasons apparently unrelated to the treatment except in one case. Seventeen patients failed to complete 8 weeks of the trial (8 from the control group, 9 from the treatment group) and were thus excluded from analysis and from Table II. Table III shows the reasons for withdrawal from or termination of the trial both before 8 weeks (thus excluded from analysis) and after 8 weeks (included). Table IV gives the mean initial biochemical measurements in the patients who completed at least 8 weeks of the trial.

The Figure shows the mean plasma 25(OH)D values during the trial. Almost all patients in the treatment group sustained a substantial rise, but no important change occurred in the control group, indicating that extraneous sources of vitamin D, such as diet or sunlight were not confusing the groupings. A small number of patients in

Table II. Characteristics of patients completing at least 8 weeks of trial

	Control	Treatment
Number		
Total	33	32
Male	7	7
Female	26	25
Mean age (s.e.m.)	82.3 (6.0)	82.6 (6.9)
Stroke	12	10
Painful arthropathy	15	13
Parkinsonism	1	3
Chairbound	19	20
Mean mental assessment score (s.e.m.)	13.0 (5.4)	12.3 (6.4)
Low mental assessment score (<12)	14	13
Plasma 25(OH)D <20 nmol/l (8 ng/ml)	23	21
Mean time in trial (s.e.m.)	33.0 (13.6)	25.9 (13.9)
Completed 7 assessments	17	8 ($P < 0.05$)

the treatment group, mainly from those attending the day hospital, showed no significant rise in plasma 25(OH)D, suggesting poor compliance.

Table V shows the base-line mean 'muscle power' and 'independence' scores and changes from these values at intervals throughout the trial. No significant differences were found between the two groups before or at any time during the trial. The unweighted total ADL score results are also shown, and again show no

Table III. Reasons for failure to complete trial

	Failing at 8 weeks	Failing at 8 weeks but before 7th assessment†
Died	5 (4/1)*	11 (4/7)
Discharged	3 (2/1)	7 (3/4)
Patient request to leave trial	4 (1/3)	0
Inadvertently prescribed vitamin D	2 (1/1)	0
25(OH)D subsequently found too high at start	1 (0/1)	0
Hypercalcaemia	1 (0/1)‡	0
Failed to take tablets	0	1 (0/1)
Became confused	0	2 (0/2)
Apparently unrelated intercurrent illness	1 (0/1)	1 (0/1)
Impossible to obtain blood samples	0	1 (0/1)
Total	17 (8/9)	23 (7/16)

*Numbers in parentheses indicate (control/treatment).

†17 patients (9 control, 8 treatment) completed more than 8 weeks, but failed to reach the 7th assessment because of overall termination of the trial, and are not included.

‡This patient was subsequently found to have hyperparathyroidism, initially masked by vitamin D deficiency.

Table IV. Initial plasma concentrations in the control and treatment groups (mean \pm s.e.m.)

	Control group	Treatment group
Calcium (mmol/l)	2.32 \pm 0.02	2.34 \pm 0.02
Phosphorus (mmol/l)	1.03 \pm 0.03	1.07 \pm 0.02
Alkaline phosphatase (iu/l)	98.7 \pm 13.2	98.1 \pm 9.5
Albumin (g/l)	36.4 \pm 0.6	37.1 \pm 0.7
Creatinine (μ mol/l)	87.9 \pm 3.8	94.7 \pm 4.8
25-Hydroxyvitamin D (nmol/l)	17.63 \pm 2.05	16.60 \pm 2.10

Conversion: SI to traditional units—calcium: 1 mmol/l=4 mg/100 ml, phosphorus: 1 mmol/l=3.1 mg/100 ml, creatinine 100 μ mol/l=1.17 mg/100 ml, 25-hydroxyvitamin D: 1 nmol/l=0.4 ng/ml.

significant change. No significant differences were found in the timings, though there were far fewer measurements available, because of the inability of many of the patients to perform certain of the tests even with assistance. Also shown are the mental assessment scores which again did not differ between groups. When the individual components of the ADL test were analysed, rather than the overall score, no significant differences were found except in the case of the change in ability to rise from the lowest chair over the first 8 weeks of the trial. In this case, the mean change in score for the control group was 0.03 ± 0.05 , compared with $+0.26 \pm 0.09$ for the treatment group ($P=0.009$). However, this difference might be expected purely by chance in this substantial number of analyses and this impression is reinforced by a tendency in the opposite direction in the case of rising from the highest chair. All the analyses were repeated with each group divided on the basis of their initial plasma 25(OH)D into subgroups with plasma 25(OH)D more than or less than 20 nmol/l (8 ng/ml). Again, no significant differences were found. Because of some overlap in plasma 25(OH)D concentrations between the two groups, overall correlations were examined between

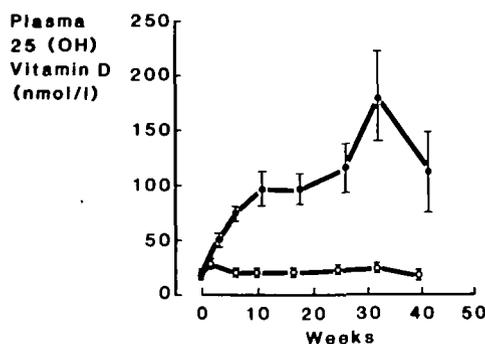


Figure. Time course of plasma 25(OH) vitamin D in the control (○) and the treatment groups (●). Conversion: 25-hydroxyvitamin D, 1 nmol/l=0.4 ng/ml.

Table V. Base-line and changes from base-line in ADL and mental assessment tests

		Base-line	Changes at		
			10 weeks	24 weeks	40 weeks
*Number in trial	C	33	33	23	17
	T	32	31	15	8
'Independence' score	C	1.89±0.10	+0.04±0.07	-0.08±0.10	-0.06±0.11
	T	1.89±0.10	+0.02±0.09	-0.05±0.11	-0.15±0.26
'Muscle strength' score	C	2.20±0.10	+0.06±0.06	-0.04±0.09	+0.01±0.09
	T	2.26±0.09	+0.09±0.07	+0.08±0.13	-0.05±0.28
Unweighted total ADL	C	23.6±1.0	+0.6±0.7	-0.3±0.9	+0.1±1.2
	T	23.8±0.9	+0.7±0.8	+0.1±1.3	-1.0±3.0
Mean time taken (s)/test	C	37.2±3.2	+2.0±3.9	+4.7±3.4	+0.6±4.7
	T	37.6±4.0	-1.3±4.3	-4.3±10.2	-4.7±7.5
Mean number of tests unable to do	C	4.9±0.6	-0.2±0.4	0.0±0.05	0.0±0.6
	T	5.0±0.5	-0.5±0.5	-0.6±0.06	-0.3±1.5
Mental assessment score	C	13.0±0.9	+1.2±0.8	+0.2±0.9	+1.2±1.2
	T	12.3±0.6	+1.5±0.7	-0.6±0.7	-1.4±1.3

A negative sign of the score change indicates worsening performance (except in the case of mean time/test). None of the differences between the two groups at any time point reached significance nor were any of the changes themselves significantly different from zero. The mean time taken for test was calculated from the tests that the patient was able to carry out. C=control; T=treatment. Results are expressed as mean (\pm s.e.m.).

*Patients who completed more than 8 weeks.

changes in plasma 25(OH)D and changes in the 'muscle strength' and 'independence' indices. No significant correlations were found ($r=0.26$, $P>0.1$, for independence score; $r=0.12$, $P>0.3$ for muscle strength score).

The clinical condition of the participants during the trial was reviewed by the geriatricians (without knowledge of the treatment group allocation), with special reference to changes in neurological, locomotor, cardiorespiratory and psychiatric status which could have affected performance of the ADL tasks. No gross trends were found in either group.

Mean plasma creatinine, albumin and alkaline phosphatase were comparable in the two groups before the start of the trial and there were no significant trends in any of these variables during the trial. Although mean plasma calciums were similar at the outset, there was after the 11th week a slight but consistent tendency for plasma calcium in the treated group to exceed that in the placebo group—by almost 0.06 mmol/l.

DISCUSSION

In this study we attempted to assess vitamin D therapy in elderly patients with low or low normal plasma 25-hydroxy vitamin D in terms of benefits which might be perceived by the patients themselves. For this reason capabilities related to their personal independence were used as the prime outcome variables, rather

than intermediate measures of a biochemical or histological nature (23); furthermore, formal direct tests of muscle strength are not generally practicable in the type of patient studied. It seemed possible, in particular, that the myopathy associated with vitamin D deficiency might have been a substantial factor in these patients' dependence on long-term care and that its correction would have reduced dependency despite their numerous other disabilities. Though myopathy (mainly proximal) due to vitamin D deficiency is a well established entity, its quantitative relationship with plasma vitamin D metabolite concentrations has not been clearly established and evidence concerning the precise metabolites responsible for integrity of muscle function is conflicting, arguments having been advanced for both 25(OH)D (13) and 1,25-dihydroxyvitamin D [$1,25(\text{OH})_2\text{D}$] (14).

In addition it was possible that pain due to osteomalacia might have contributed to dependence. Again, the relationship of osteomalacia to vitamin D metabolite levels is unclear. It is well established that subjects with low or very low plasma concentration of 25(OH)D may have no clinical (15) or histological (17, 24) evidence of osteomalacia, perhaps because production of $1,25(\text{OH})_2\text{D}$ from its precursor is enhanced (16). On the other hand, advanced nutritional osteomalacia has been reported in the presence of normal plasma $1,25(\text{OH})_2\text{D}$ but subnormal concentrations of 25(OH)D and $24,25(\text{OH})_2\text{D}$ (18). In contrast, in elderly subjects with 'bone loss of ageing' increased osteoid tissue has been found despite normal plasma 25(OH)D (14, 19, 20); the osteoid excess disappeared on treatment with $1\alpha(\text{OH})\text{D}$ (14). This uncertainty about the relationship of plasma 25(OH)D to bone disease was the reason for including in the present study some subjects with plasma 25(OH)D in the lower part of the young adult normal range.

Because of evidence in the elderly of malabsorption of vitamin D (2), and poor 25- and 1-hydroxylation (2–5) we chose to use a somewhat higher dose (9000 units daily) of calciferol than is customarily used for treating vitamin D deficiency in younger subjects. The duration of the trial and timing of assessments were determined by the time course of the clinical response to treatment of vitamin D deficiency in younger subjects. A minor fraction of muscular strength is rapidly regained in the first few weeks—but at least 6–12 months is required for complete recovery, probably because of the need for regrowth of atrophic type II muscle fibres (15); nevertheless the initial rapid phase is sufficient to result in substantial improvement in the physical capabilities. For this reason eight weeks was regarded as the minimum period in the trial.

The main finding of the present study is that no difference was observed between the treated and control groups in the capacity to perform essential activities of daily living. Particular care was taken to ensure as far as possible that the incidence of other disabilities was similar in the two groups. The power of the study to detect a difference if it had indeed occurred was quite high. It may be estimated (from the actual data) that at three months the chance of missing a 20% difference in ADL weighted indices was less than 5%. At later times fewer subjects remained in the trial, but at six months the chance of missing a 20% difference was still less than 25%. The measurements of plasma 25(OH)D showed a clear rise in the mean level to the upper part of the 'young adult' normal

range in the treated group, but no change in the control group. It is unlikely that the small degree of overlap in plasma 25(OH)D between the groups during the trial was responsible for the negative result, since there were no significant correlations between the changes in plasma 25(OH)D and those in the ADL weighted scores.

It may be concluded from the present trial either (1) that elderly hospital patients (mainly from geriatric wards) with low or low normal plasma 25(OH)D have no significant vitamin D related pathology serious enough to affect their capacity to perform essential activities of daily living, or (2) that, if indeed they do have such pathology which is reversible by vitamin D treatment, their other disabilities are such as to prevent expression of improvement in a useful form, or (3) that the pathology is irreversible (21). The study therefore offers no support for any general recommendation that geriatric patients who remain in hospital for long periods should receive routine vitamin D supplementation in order to maintain or improve their independence. There are reasons, however, for further study of the effects of vitamin D supplementation in the elderly. For instance, it is common to find evidence of osteomalacia in elderly patients with fractures, particularly of the femoral neck (22), and it has been argued (6) that vitamin D depletion also contributes to osteoporosis in the elderly. A trial of the effect of vitamin D supplementation on the incidence of femoral neck fractures has not been published and would inherently require very much larger numbers of patients than participated in the present study. Finally, nothing in the present findings removes the need for vitamin D deficiency to be considered in individual elderly patients with bone or muscle symptoms.

ACKNOWLEDGEMENTS

This work was supported by the Department of Health and Social Security. We are very grateful to the many members of the staff of New Cross, Bethnal Green, Mile End and St Matthew's Hospitals who co-operated in the trial and to Drs A. T. Brain and M. R. Bliss who gave permission for their patients to be included.

REFERENCES

1. Corless D, Beer M, Boucher BJ, Gupta SP, Cohen RD. Vitamin D status in long-stay geriatric patients. *Lancet* 1975; **i**:1404-6.
2. Barragry JM, France MW, Corless D, et al. Intestinal cholecalciferol absorption in the elderly and in younger adults. *Clin Sci Mol Med* 1978; **55**:213-20.
3. Rushton C. Vitamin D hydroxylation in youth and old age. *Age Ageing* 1978; **7**:91-5.
4. Gallagher JC, Riggs BL, Eisman J, Hamstra A, Arnaud SB, DeLuca HF. Intestinal calcium absorption and serum vitamin D metabolites in normal subjects and osteoporotic patients. *J. Clin Invest* 1979; **64**:729-36.
5. Slovick DM, Adams JS, Neer RM, Holick MF, Potts JT. Production of 1,25 dihydroxyvitamin D in elderly osteoporotic patients. *N Engl J Med* 1981; **305**:372-4.
6. Baker MR, Peacock M, Nordin BEC. The decline in vitamin D status with age. *Age Ageing* 1980; **9**:249-52.
7. Corless D, Gupta SP, Switala S, et al. Response of plasma 25-hydroxy vitamin D to ultra-violet irradiation in long-stay geriatric patients. *Lancet* 1978; **ii**:649-51.

8. Benjamin J. The Northwick Park ADL Index. *Occup Ther* 1976;301-6.
9. Sheikh K, Smith DS, Meade TW, Goldenberg E, Brennan P, Kinsella G. Repeatability and validity of modified activities of daily living (ADL) index in studies of chronic disability. *Int Rehabil Med* 1979;1:51-8.
10. Hodkinson HM. Evaluation of a mental test score for assessment of mental impairment in the elderly. *Age Ageing* 1972;1:233-8.
11. Silver CP. Simple methods of testing ability in geriatric patients. *Gerontol Clin* 1972;14:110-22.
12. Haddad JG, Chyu KJ. Competitive protein-binding radioassay for 25-hydroxycholecalciferol. *J Clin Endocrinol* 1971;33:992-5.
13. Birge SG, Haddad JG. 25-Hydroxycholecalciferol stimulation of muscle metabolism. *J Clin Invest* 1975;56:1100-7.
14. Sørensen OH, Land B, Sattin B, et al. Myopathy in bone loss of ageing: improvement by treatment with 1-hydroxycholecalciferol and calcium. *Clin Sci* 1979;56:157-61.
15. Young A, Brenton DP, Edwards RHT. Analysis of muscle weakness in osteomalacia. *Clin Sci Mol Med* 1978;54:31p.
16. Stanbury SW. Vitamin D and hyperparathyroidism. *J R Coll Physicians* 1981;15:205-17.
17. Davie M, Lawson DEM, Jung JT. Low plasma 25-hydroxyvitamin D without osteomalacia. *Lancet* 1978;i:820.
18. Eastwood JB, de Wardener HE, Fray RW, Lemann JL. Normal plasma 1,25(OH)₂ vitamin D concentrations in nutritional osteomalacia. *Lancet* 1979;ii:1377-8.
19. Sørensen OH, Andersen RB, Christensen MS, et al. Treatment of senile osteoporosis with 1-hydroxyvitamin D. *Clin Endocrinol* 1977;7:1695-1755.
20. Kafetz K, Hodkinson HM. Osteomalacia in the presence of 'normal' serum 25-hydroxycholecalciferol concentration. *Br Med J* 1981;283:1437.
21. Isenberg DA, Newham D, Edwards RHT, Wiles CM, Young A. Muscle strength and pre-osteomalacia in vegetarian Asian women. *Lancet* 1982;i:55.
22. Aaron JE, Gallagher JC, Nordin BEC. Seasonal variation of histological osteomalacia in femoral neck fractures. *Lancet* 1974;ii:84-5.
23. Hosking DJ, Campbell GA, Kemm JR, et al. Screening for subclinical osteomalacia in the elderly: normal ranges or pragmatism? *Lancet* 1983;ii:1290-92.
24. Hodkinson HM, Hodkinson I. Range for 25-hydroxyvitamin D in elderly subjects in whom osteomalacia has been excluded on histological and biochemical criteria. *J Clin Exp Gerontol* 1980;2:133-9.
25. Pocock SJ. *Clinical trials*. Chichester: Wiley, 1983.
26. Conover WJ. *Practical non-parametric statistics*, 2nd ed. New York: Wiley, 1980.

Date accepted 5 July 1984.

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