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A. van Faassen · E. M. C. van der Ploeg
H. M. L. Habets · R. van der Meer
R. J. J. Hermus · R. A. Janknegt

The effects of the calcium-restricted diet of urolithiasis patients with absorptive hypercalciuria type II on risk factors for kidney stones and osteopenia

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Abstract The calcium (Ca)-restricted diet of urolithiasis patients with absorptive hypercalciuria type II may decrease Ca excretion but increase biochemical markers of risk for osteopenia. We randomly allocated 25 patients from six hospitals into an experimental group (Ca restriction to 500 mg/day, oxalate-rich products discouraged and normalization of animal protein and sodium) and a control group (no restrictions) for one month. The urinary Ca excretion did not decrease significantly, but the oxalate excretion decreased, although not significantly. The hydroxyproline:creatinine ratio in fasting urine seemed to increase and the calcium:creatinine ratio to decrease. The deoxypyridinoline:creatinine ratio in fasting urine did not change. We conclude that our Ca-restricted diet, which is lower in Ca, animal protein and table salt due to the omission of dairy products, may be of benefit for absorptive hypercalciuria type II patients without enhancing the risk for osteopenia. However, a long-term clinical trial is required.

Key words Diet · Risk factors · Calcium · Urolithiasis · Osteoporosis prevention

A. van Faassen (✉) · R. A. Janknegt
Department of Urology, University Hospital,
P.O. Box 5800, NL-6202 AZ
Maastricht, The Netherlands; fax: (+31) 0433614349

E. M. C. van der Ploeg
Department of Dietetics, University Hospital, Maastricht,
The Netherlands

H. M. L. Habets
Department of Clinical Chemistry, University Hospital,
Maastricht, The Netherlands

R. van der Meer
Department of Nutrition, Netherlands Institute
for Dairy Research, Ede, The Netherlands

R. J. J. Hermus
Department of Nutrition and Food Research,
TNO, Zeist, The Netherlands

Introduction

Urolithiasis patients diagnosed as absorptive hypercalciuria type II are those who respond to a calcium (Ca)- and sodium-deprived diet during the metabolic screening of recurrent stone formers [17]. Our therapeutic diet for these patients consists of a diet moderately restricted in Ca (500 mg/day), oxalate (< 400 mg/day) and table salt (100–150 mmol/day). Dietary excesses of animal protein (> 50 g/day) are eliminated [16].

A lowering effect on Ca excretion of 4 days of severe Ca restriction (250 mg/day) has been detected in male, hypercalciuric renal stone forming subjects [14]. Ca restriction to 400 mg/day and moderate sodium restriction over 4 days lowered the Ca excretion in hypercalciuria type II patients [2].

Patients with hypercalciuria are shown to have decreased vertebral bone density. In particular patients on a low-Ca diet (*c*. 400 mg/day) showed a reduction in bone mineral content [9]. Therefore a Ca-restricted diet prescribed to absorptive hypercalciuria type II patients may further enhance their risk for osteopenia.

This paper describes the effect of the Ca-restricted diet of our absorptive hypercalciuria type II patients on risk factors for Ca oxalate stones (urinary excretion of Ca, oxalate, citrate, magnesium, phosphate and uric acid) and markers for bone metabolism, i.e. hydroxyproline and deoxypyridinoline in fasting urine, alkaline phosphatase in fasting blood and Ca in fasting urine [8].

Subjects and methods

The study protocol was approved by the Medical Ethics Committee of the University Hospital Maastricht (PF8). Patients with recurrent urolithiasis were metabolically screened as described by Pak [17] using a computerized program. Before the screening, the patients filled in a structured dietary questionnaire [10], improved by measures for portion size and by more detailed questions about intake of Ca, salt and oxalate. Urologists in six Dutch hospitals participated in the study, using the same version (2.0.1) of the computer program. Patients with a urinary Ca excretion on their

regular diet > 5 mmol/day and an excretion below this value after 1 week on a diet restricted in Ca (400 mg/day) and sodium (100 mmol/day), were diagnosed as absorptive hypercalciuria type II. These patients were invited to participate in the study by their urologist, after which informed consent was obtained. Exclusion criteria were use of laxatives, cholesterol-lowering medicines, gallbladder disease and bone disorders. After deciding to participate, the patients received detailed information and materials to collect 24-h urine and 48-h feces. The project manager explained the trial extensively to the patients by telephone and a data-plan was drawn up together. Randomization between the experimental (Ca-restriction) group and the control (regular diet) group was performed within strata for Ca intake (less and more than 1000 mg/day) and sex.

Before and after the study period of 4 weeks, fasting urine and blood were sampled at the patient's hospital. At both sampling times a questionnaire about defecation frequencies and problems during sampling was filled in by the patients. Moreover, at the end of the 4 weeks the patients filled in the dietary questionnaire mentioned before, to measure dietary practices during the trial.

The dietary questionnaire of the respondents was controlled by the dietitian at the patient's hospital and calculated for Ca and other nutrients by a dietitian at the Department of Dietetics of the University Hospital in Maastricht using the standardized Dutch Uniform Encoding System and the 1990 release of the National Computerized Dutch Nutrient Databank. The experimental subjects visited their dietitian to discuss the Ca-restricted diet. The minimum amount of fluid was prescribed as 2.5 l per day. The Ca intake was limited to 500 mg/day. In The Netherlands this is mainly achieved by omitting dairy products such as cheese and other milk products. No intake lower than 500 mg/day was allowed, because at low intakes of Ca, oxalate absorption might be increased. Oxalate-rich vegetables such as spinach, red beet and rhubarb were discouraged. Lightly-brewed black tea was allowed to a maximum of 1 l, because its high content of oxalic acid may result in a high excretion of oxalate in urine. The diet was normalized in relation to animal protein (50 g/day) and table salt (4500 mg = 200 mmol/day), because these dietary factors increase the Ca excretion in the urine [17]. Alcohol was restricted to 4 drinks per day because alcohol also may lead to a high excretion of Ca in urine [24].

Fasting urine was sampled after discarding the first urine of the morning at the patient's hospital. It was poured directly into a glass tube, covered with a drop of toluene and frozen at −20°C. Fasting blood was sampled in lithium heparin, immediately centrifuged for 10 min at 1500 *g*, the plasma portioned into plastic tubes and the tubes frozen at −20°C.

Urine during 2 × 24 h was sampled by the patients on weekdays using the U-mate P of Sumitomo Bakelite (Tokyo, Japan) with the lower part filled with 1.7 g boric acid as a preservative. It is based on the principle described by Tochikubo et al. [22], taking 1/50 of every urine voidance. With this small apparatus, 24-h urine can be sampled very easily without the need to carry a large 2.5 l jerrycan. The sampler can be cleaned and disinfected by soap and hypochlorite. After they had been given a clear description of how to use it, about 30 men and women between 20 and 65 years of age used this apparatus without reporting any difficulties. We controlled the validity of urine sampling by analyzing the creatinine excretion [17].

Feces during 48 h were collected over a weekend using a plastic bucket that fits into the toilet (Emergo, Landsmeer, The Netherlands) and coded plastic bags that fit into the bucket. Immediately after defecation, the bags containing the stools were placed between four frozen elements of a freezing pack surrounded by Styrofoam plates in a cardboard box. In this way sampling materials and fecal samples can easily be mailed and the samples stayed below 5°C for 4 days. For 7 days before collection the patients swallowed 10 radio-opaque pellets (Resprecare Medical, The Hague, The Netherlands) at three times during the day to correct excretion values for fecal flow [4]. Each collected stool was weighed, flattened for radiography and stored in a freezer at −20°C. Radiographs were made using AGFA-Gevaert Curix RPI films of 24 × 30 cm in Kodak X-omatic cassettes in a Philips Practix camera. The samples (four per plate) were illuminated at 60 cm distance by 50 kV for 0.2 s. The pellets were counted on a viewing box.

Urinary creatinine, urea, phosphate and uric acid were measured using the Dimension clinical chemistry system (Dupont de Nemours, Wilmington, DE). Sodium was analysed by flame spectroscopy on a photometer (model 943, Instrumentation Laboratory, Milan, Italy); Ca and magnesium on an atomic absorption spectrophotometer (model AA/AE 551, Instrumentation Laboratory) with a long-path air acetylene burner (IL 43005-02). Sulfate was analyzed by a turbidimetric method using polyethylene glycol 6000 [13]. Oxalate and citrate were measured by enzymatic kits from Sigma and Boehringer, respectively. Hydroxyproline in fasting urine was analysed using the Organon Technika kit. Plasma total and bone alkaline phosphatase were measured in fasting serum with the Boehringer kit based on precipitation of the bone isomer with wheat-germ lectin. The concentration of deoxypyridinoline in fasting urine was measured by immunoassay (Pyrilinks-D, Metra Biosystems, Palo Alto, Calif.) following the manufacturer's instructions.

Ca, magnesium and phosphorus in feces were analysed as described above after incubating wet feces in 4 N hydrogen chloride and 7.5% hydrogen peroxide at 100°C for 3 h, centifuging for 10 min at 3000 *g* and filtering through a Filter Sampler (a standard model blood serum filters; Porex Medical, Fairburn, Ga.).

We evaluated urinary calcium oxalate oversaturation by calculating the most recent Tiselius' index [21]. Absolute values and differences in changes of dietary constituents, variables of blood, urine and feces and the Tiselius' index between experimental and control subjects were analysed by Student's *t*-test, or the Wilcoxon test if the variables were not normally distributed. Correlations between (shifts in) parameters were calculated by the Pearson correlation coefficient [1].

Results

Twenty-nine subjects from six hospitals volunteered into the study. Six male and two female patients were invited by their urologist but did not wish to participate. From three patients (one male and two females) we did not receive the dietary questionnaire, sent by mail. The fasting urine and blood of two patients were not collected properly by their hospital. The 24-h urine of four patients was not collected correctly as shown by creatinine excretion values. Two female patients refused to collect feces and the fecal analyses of two patients were unreliable; one because she had had a gall-bladder operation and the other because the photograph of the pellets in his feces was not valid. Therefore the dietary data of 26 subjects, the bone metabolism data of 27 subjects and the urinary and fecal excretion values of 25 subjects were statistically analysed.

The values at the beginning of the trial and the changes in dietary intakes of the patients in the control and Ca-restricted groups are shown in Table 1. The changes (value after 4 weeks minus value at the beginning) in intake of Ca, animal protein, mono- and disaccharides, polysaccharides, cholesterol, saturated fatty acids, energy, phosphorus and sodium were significantly greater for the experimental group than the control group. The initial intakes of animal protein, total fat and saturated fat were significantly higher in the Ca-restricted group. The Ca intake appeared higher initially in the experimental group, although not significantly.

The combined urinary and fecal excretion values (Table 2) showed that the experimental group excreted

Table 1 Mean (SD) of baseline values and changes in nutrients

Dietary constituent	Control diet ($n = 13$)		Ca-restricted diet ($n = 13$)	
	Baseline	Change	Baseline	Change
Energy (kJ/day)	9690 (2830)	−400 (1270)	12130 (3650)	−3420 (3070)*
Total protein (g/day)	75 (20)	−4 (14)	98 (31)	−37 (24)*
Animal protein (g/day)	48 (17)	−5 (11)	67 (27)*	−33 (29)**
Total fat (g/day)	100 (31)	−9 (19)	133 (45)*	−46 (39)
Saturated fat (g/day)	40 (13)	−5 (8)	54 (17)*	−26 (13)
Cholesterol (mg/day)	237 (86)	−24 (43)	320 (115)*	−144 (108)*
Carbohydrates (g/day)	251 (98)	−3 (48)	317 (108)	−76 (75)**
Mono- + disaccharides (g/day)	111 (64)	0 (48)	155 (68)	−47 (64)*
Polysaccharides (g/day)	140 (50)	−3 (23)	162 (56)	−29 (35)*
Fibre (g/day)	28.5 (9.4)	1.1 (7.9)	27.9 (8.7)	−2.7 (6.9)
Calcium (mmol/day)	23.2 (9.2)	−1.5 (6.9)	31.2 (16.8)	−17.8 (18.5)*
Phosphorus (mmol/day)	47.5 (13.6)	−1.1 (9.5)	57.9 (19.3)	−23.5 (17.1)*
Sodium (mmol/day)	182 (47)	−11 (23)	203 (39)	−43 (23)**
Fluid (l/day)	2.9 (0.8)	−0.1 (0.7)	2.9 (1.0)	−0.3 (1.1)

*$P < 0.05$; **$P < 0.01$

Table 2 Mean (SD) baseline values and changes in urinary plus fecal excretion of minerals

Balance parameters (mmol/day)	Control diet ($n = 11$)		Ca-restricted diet ($n = 9$)	
	Baseline	Change	Baseline	Change
Calcium	29.1 (14.9)	2.8 (28.5)	32.3 (22.6)	−17.0 (25.4)*
Magnesium	21.1 (14.0)	−3.1 (17.8)	18.8 (10.8)	−6.7 (12.1)
Phosphorus	53.4 (19.3)	−6.3 (24.7)	50.1 (22.1)	−11.8 (22.1)

*$P < 0.05$

Table 3 Mean (SD) of baseline values and changes in urinary parameters

Urinary parameters	Control diet ($n = 13$)		Ca-restricted diet ($n = 12$)	
	Baseline	Change	Baseline	Change
Urine (l/day)	1.6 (0.4)	0.06 (0.6)	1.7 (0.8)	0.05 (1.1)
Urea (mmol/day)	334 (102)	−9.2 (75.5)	342 (154)	−31 (132)
Sodium (mmol/day)	153 (53)	−0.5 (46.5)	169 (66)	−25 (88)
Creatinine (mmol/day)	13 (4)	0 (3)	13 (4)	−1 (6)
Calcium (mmol/day)	6.1 (1.4)	−0.2 (2.5)	6.3 (2.3)	−0.7 (2.9)
Oxalate (mmol/day)	0.38 (0.17)	0.05 (0.15)	0.43 (0.25)	−0.12 (0.27)#
Citrate (mmol/day)	2.4 (1.3)	−0.3 (1.0)	3.1 (1.4)	−0.3 (1.6)
Uric acid (mmol/day)	2.6 (0.9)	0.2 (0.8)	3.0 (1.5)	−0.1 (1.7)
Phosphate (mmol/day)	31.2 (9.6)	−1.9 (6.5)	33.0 (13.9)	−5.3 (14.8)
Sulfate (mmol/day)	17.7 (8.2)	−0.3 (8.1)	19.1 (6.5)	−3.8 (9.0)
Magnesium (mmol/day)	4.7 (1.4)	−0.4 (1.4)	5.0 (2.7)	0.1 (2.4)
Tiselius' index	1.41 (0.45)	0.30 (0.76)	1.45 (0.50)	−0.14 (1.0)

#$P < 0.1$

about 20 mmol (=800 mg) less Ca per day during the trial than the control group. Moreover the initial data for Ca were similar for both groups.

The urinary excretion of urea and sodium decreased in the experimental group (Table 3), although the changes were not statistically significant. These urinary markers of dietary intake of protein and sodium respectively [3], show that there was no difference in initial intake of these dietary constituents between the two groups.

The excretion of Ca in the urine decreased, but there was no significant difference between the Ca-restricted and the control group. Moreover the variation in shifts was extremely high. The Tiselius' index did not change significantly.

Table 4 shows that the hydroxyproline:creatinine ratio increased more in the experimental group than in the control group ($P = 0.05$). The data on this parameter are in the range of normal values [9]. The Ca:creatinine ratio decreased more in the Ca-restricted group ($P = 0.06$). The deoxypyridinoline:creatinine ratio and the activities of total alkaline phosphatase and its bone isomer did not change.

Discussion

The excretion data on Ca, magnesium and phosphorus and biomarkers of dietary intake such as urea indicated no difference in dietary intake of the two groups. The difference in dietary intake data may be caused by an underreporting of the intake of, for example, milk products by the control group, which happens to contain 3 times more subjects with a body mass index higher

Table 4 Mean (SD) baseline values and changes in parameters of bone metabolism

Parameters for bone metabolism	Control diet ($n = 12$)		Ca-restricted diet ($n = 15$)	
	Baseline	Change	Baseline	Change
Hydroxyproline:creatinine ratio in fasting urine (μmol/mmol)	11.7 (3.7)	−2.4 (4.5)	12.1 (5.4)	0.7 (2.8)[#]
Deoxypyridinoline:creatinine ratio in fasting urine (nmol/mmol)	4.4 (0.7)	−0.1 (0.7)	4.7 (1.4)	0.0 (1.2)
Calcium:creatinine ratio in fasting urine (nmol/mmol)	0.2 (0.1)	0.1 (0.1)	0.3 (0.2)	−0.1 (0.2)[#]
Total alkaline phosphatase in fasting plasma (U/l)	63.8 (23.7)	2.5 (2.5)	59.0 (16.1)	2.3 (7.8)
Bone alkaline phosphatase in fasting plasma (U/l)	15.3 (10.5)	4.3 (8.5)	12.4 (14.5)	5.3 (16.5)

[#] $P < 0.1$

than 26, including two with an index higher than 30 (H Boeing, personal communication).

The Ca-restricted diet did not significantly lower Ca in urine. Moreover the variability in the responses was extreme. When males and females were considered separately, the calcium excretion decreased in 16 men by 1 mmol/day, while the intake of Ca, protein and salt decreased. This small effect might be caused by an increase in serum calcitriol when subjects are maintained on a low Ca-diet for at least 10 days [6].

The lower excretion of oxalate will have been caused by the lack of protein from dairy products and the lower intake of fat and carbohydrates on the Ca-restricted diet. The expected higher excretion of oxalate due to an enhanced solubility of oxalate in the intestine and therefore an increased absorption of oxalate, may play a minor role when the subjects are already consuming a diet moderately restricted in oxalate [15]. Moreover, the effect of a lower intake of protein, fat and carbohydrates may be stronger than the lowered intake of Ca [15, 20].

The Ca-restricted diet did not influence the urinary excretion of citrate. It has been suggested [11] that dairy protein does not lower the citrate excretion, although dairy protein is a form of animal protein. Dairy products may contain constituents that lead to a lower acidification in the nephron and therefore to reduced lowering of citrate excretion. This hypothesis is supported by our data on sulfate in the 24-h urine (Table 3), which was not influenced significantly by the experimental diet.

The hydroxyproline:creatinine ratio in the experimental group seemed to increase compared with the control group. However, the other parameters of bone resorption (Ca:creatinine ratio and deoxypyridinoline:creatinine ratio in fasting urine) showed respectively a decrease and no change in bone metabolism. Deoxypyridinoline excretion is a more specific marker of bone metabolism than hydroxyproline, because the deoxypyridinoline is derived mainly from bone tissue while hydroxyproline occurs also in soft tissues and complement [8].

Reduced bone density has been found in kidney stone patients on a low Ca-diet [9]. However, there is evidence for a role of sodium (and animal protein) in the pathology of bone loss in absorptive hypercalciuria [18]. Renal stone formers seem more susceptible to sodium than healthy subjects [7]. This may explain the low bone mineral density seen in the study by Fuss et al. [9] and in the recently presented case of a woman with kidney stones and low bone density [23]. On the other hand, subjects may differ in their susceptibility to a Ca-restricted diet; a new test, the osteocalcin response, may differentiate between subjects who are and are not susceptible to increased bone turnover due to a Ca-restricted diet [19].

A high sodium intake increases the urinary excretion of Ca [7] and therefore may increase bone dissolution. In our Ca-restricted diet, the consumption of sodium was also lowered. This may have counteracted an enhancing effect of the Ca restriction on bone dissolution as measured by deoxypyridinoline excretion.

The decrease in intake of animal protein and saturated fat will have been caused by the omission of dairy products such as milk and cheese due to the restriction of Ca. The subjects most probably did not substitute their milk products. This may be due to their high dietary intake: the intake of protein, carbohydrates and energy were respectively 8 g, 50 g and 1350 kJ higher than the age- and sex-controlled values of healthy subjects in The Netherlands [12]. This is reflected in the high body mass indices of about half of the patients. The lower intake of carbohydrates during the diet may be due to a lower intake of sweetened milk products such as drinking chocolate and puddings.

The therapeutic regimen of drinking at least 2.5 l water per day was not followed by all patients, as can be seen from their volume of daily urine. The difference between intake and excretion might be about 0.5–1 l [17]. On the Ca-restricted diet three patients drank much less than on their normal diet. Most probably they did not compensate for their lower intake of milk and milk products.

Instead of omitting dairy products, which are valuable sources of nutrients such as vitamin B_2, potassium animal protein and fluid, the diet may be restricted to 0.5 g/kg ideal body weight and 3100 mg sodium (a prudent diet). This would lower the Ca excretion in urine by about 2 mmol/day [5, 7] would not lower the intake of fluid, would favor bone resorption due to its high Ca:protein ratio and could be checked easily by measuring urea and sodium in 24-h urine.

In conclusion, our diet for absorptive hypercalciuria type II patients may benefit these patients without enhancing their risk for osteopenia, because it lowers the excretion of oxalate in urine and does not enhance

biochemical markers of bone turnover. However, a long-term clinical trial on the effects of the Ca-restricted diet on (markers of) bone metabolism is required before using this diet as a metaphylaxis strategy. Moreover, a diet restricted in animal protein and table salt may be more effective on risk factors for calcium oxalate stones.

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