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ORIGINAL COMMUNICATION

Influence of a mineral water rich in calcium, magnesium and bicarbonate on urine composition and the risk of calcium oxalate crystallization

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Objective: To evaluate the effect of a mineral water rich in magnesium (337 mg/l), calcium (232 mg/l) and bicarbonate (3388 mg/l) on urine composition and the risk of calcium oxalate crystallization.
Design: A total of 12 healthy male volunteers participated in the study. During the baseline phase, subjects collected two 24-h urine samples while on their usual diet. Throughout the control and test phases, lasting 5 days each, the subjects received a standardized diet calculated according to the recommendations. During the control phase, subjects consumed 1.4 l/day of a neutral fruit tea, which was replaced by an equal volume of a mineral water during the test phase. On the follow-up phase, subjects continued to drink 1.4 l/day of the mineral water on their usual diet and collected 24-h urine samples weekly.
Results: During the intake of mineral water, urinary pH, magnesium and citrate excretion increased significantly on both standardized and normal dietary conditions. The mineral water led to a significant increase in urinary calcium excretion only on the standardized diet, and to a significantly higher urinary volume and decreased supersaturation with calcium oxalate only on the usual diet.
Conclusions: The magnesium and bicarbonate content of the mineral water resulted in favorable changes in urinary pH, magnesium and citrate excretion, inhibitors of calcium oxalate stone formation, counterbalancing increased calcium excretion. Since urinary oxalate excretion did not diminish, further studies are necessary to evaluate whether the ingestion of calcium-rich mineral water with, rather than between, meals may complex oxalate in the gut thus limiting intestinal absorption and urinary excretion of calcium and oxalate.
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Introduction

An adequate urine volume achieved by a sufficient fluid intake is the most important therapeutic measure for the prevention of recurrent urinary stone disease, irrespective of stone composition. In a prospective, randomized trial Borghi *et al* (1996) demonstrated that 88% of calcium oxalate stone formers treated with a high water intake remained stone-free during the 5-y follow-up period *vs* 73% of patients without any treatment. Furthermore, the interval for stone recurrence was significantly longer in patients with a high water intake than in the untreated control group.

In calcium oxalate stone formation, rehydration of daily water losses from lungs, skin, urine and stool is essential and can be obtained by the consumption of mineral waters. Several constituents of mineral water such as magnesium, calcium and bicarbonate can additionally influence urine composition and therefore the risk of crystal and stone formation. Therefore, mineral water might represent a natural alternative to drugs, especially in calcium oxalate stone patients with hypomagnesuria, hypocitraturia, hyperoxaluria and acidic urinary pH, the most frequent pathological urine findings.

The application of supplemental magnesium is recommended in calcium oxalate stone patients with hyperoxaluria. The intake of magnesium is suggested to reduce oxalate absorption and urinary excretion nearly as effective as calcium by binding oxalate in the gut (Liebman & Costa,

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2000). Another potential benefit of magnesium supplementation could be an increase in urinary citrate excretion in subjects with magnesium deficiency (Reungjui *et al*, 2002). Furthermore, a high urinary excretion and concentration of magnesium has been shown to decrease both nucleation and growth rates of calcium oxalate crystals, due to the higher solubility of magnesium oxalate compared with calcium oxalate (Li *et al*, 1985; Kohri *et al*, 1988).

Various epidemiological studies revealed a significant negative correlation between dietary calcium intake and the risk of calcium oxalate stone formation, suggesting an apparent protective effect of dietary calcium (Curhan *et al*, 1993, 1997). Liebman and Costa (2000) demonstrated that a treatment with calcium carbonate can decrease urinary oxalate by complexing oxalate in the gut, thereby limiting intestinal oxalate absorption.

The bicarbonate content of mineral water can replace alkalization therapy with potassium citrate and contribute to urine inhibitory power by increasing citric acid levels (Keßler & Hesse, 2000). Bicarbonate induces metabolic alkalosis, leading to increased urinary pH value and citrate excretion (Simpson, 1983). An alkaline urine at the upper physiological limit of pH 6.8 decreases the saturation level with calcium oxalate, whereas citrate acts as an inhibitor of calcium oxalate crystallization by forming a highly soluble complex with citrate, thereby lowering the saturation of calcium salts in urine (Nicar *et al*, 1987).

The energy-free supply of high amounts of the potential beneficial agents magnesium, calcium and bicarbonate with mineral water could achieve the mentioned effects on urine composition and the risk of calcium oxalate crystallization, avoiding the use of additional drugs and drug-related intolerance or low compliance. The objectives of the present study were to evaluate the physiological effect of a simultaneous increase in the dietary intake of magnesium, calcium and bicarbonate with mineral water on urinary risk profiles in healthy subjects without disturbances in oxalate and calcium metabolism or acid–base status and to assess the long-term effect of mineral water administration.

Subjects and methods

Subjects

A total of 12 healthy male subjects with a mean age of 26.5 y (range 23–30 y) participated in the study. All participating subjects had normal urine findings from multiparameter test strips (Combur^9-Test, Boehringer, Mannheim, Germany) measuring pH, nitrite, protein, glucose, ketones, urobilinogen, bilirubin, leukocytes and blood, and had no prior history of stone formation or other renal diseases. Exercise and the use of medication were not allowed during the study.

Study design

The study design is summarized in Table 1. Throughout the baseline phase, subjects collected two 24-h urine samples

Table 1 Study protocol

Baseline phase		*Control phase*					*Test phase*					*Follow-up phase*				
Week 1	Week 2 Usual diet Usual beverages	Day 1	Day 2	Day 3 Standardized diet Fruit tea	Day 4	Day 5	Day 1	Day 2	Day 3 Standardized diet Mineral water	Day 4	Day 5	Week 1	Week 2	Week 3 Usual diet Usual beverages+mineral water	Week 4
24-h urine	24-h urine	24-h urine	24-h urine	24-h urine	24-h urine	Urine fractions	24-h urine	24-h urine	24-h urine	24-h urine	Urine fractions	24-h urine	24-h urine	24-h urine	24-h Urine
Control	Control	Adaptation			Control	Control	Adaptation			Control	Control	Control	Control	Control	Control

Table 2 Energy and nutrient composition of the standardized diets including fruit tea (control phase) and mineral water (test phase)

	Control phase	*Test phase*
Energy (kcal)	2533	2533
Energy (MJ)	10612	10612
Protein (g)	96	96
Fat (g)	107	107
Carbohydrates (g)	290	290
Calcium (mg)	823	1088
Magnesium (mg)	404	862
Potassium (mg)	2500	2556
Sodium (mg)	4100	4587

once weekly while on their usual diet. During the subsequent control and test phases, lasting 5 days each, the subjects consumed a standardized diet, calculated according to the dietary recommendations (German, Austrian and Swiss Societies of Nutrition, 2000). The standardized diet corresponded to a balanced mixed diet and provided a constant fluid intake with beverages of 2.5 l per day. After a few days of adaptation, the standardized diet, which means the daily constant intake of prescribed foods and fluids, leads to a steady state of metabolism, so that urinary values reach constant levels (Massey & Kynast-Gales, 1998; Keßler & Hesse, 2002). During the test phase, 1.4 l of a neutral fruit tea was replaced by an equal volume of a mineral water. The volume of 1.4 l/day corresponded to two bottles of mineral water. The fruit tea was prepared with tap water and did not influence urine composition. The oxalate content of the fruit tea, analyzed by ion chromatography, was 0 mg/l. In contrast to the small amounts of magnesium (9.9 mg/l), calcium (42.7 mg/l), potassium (3.5 mg/l) and sodium (17.1 mg/l) of the fruit tea, reflecting the composition of the tap water used for preparation, the mineral water contained 337 mg/l magnesium, 232 mg/l calcium, 365 mg/l sodium, 43.5 mg/l potassium, 22.8 mg/l sulfate and 3388 mg/l bicarbonate. The energy and nutrient composition of the standardized diet including beverages is presented in Table 2.

During ingestion of the standardized diet, daily 24-h urine samples were collected from each subject and analyzed for urinary parameters to ensure the adaptation to the diet. On day 5 of the control and test phase, respectively, fractional urine collection was performed to determine the circadian rhythm of urinary parameters. Subjects were instructed to drink five glasses (1.4 l/day) of the neutral fruit tea (control phase) and the mineral water (test phase), respectively, at fixed times (7.00, 10.00, 13.00, 16.00 and 19.00 h). Urine was collected every 3 h during daytime and for 9 h during the night.

On the follow-up phase, lasting 4 weeks, subjects returned to their usual diet but continued to drink 1.4 l/day of the mineral water. During the follow-up phase, subjects collected 24-h urine samples weekly.

Sample preparation and analyses

Urine collections were preserved with thymol in isopropanol and kept refrigerated at 4°C during collection. For the determination of calcium, magnesium and oxalic acid, urine samples were acidified with hydrochloric acid. The samples were stored below −20°C until analysis. Analysis of urine included urine volume, pH and the concentrations of creatinine (Jaffé reaction; CV 2.0%), calcium and magnesium (atomic absorption spectrophotometry; CV 0.3%), chloride (coulomb metric titration; CV 2.0%), sodium and potassium (flame photometry; CV 1.3%), inorganic sulfate (nephelometry; CV <5%), inorganic phosphate (phosphate molybdate reaction; CV <5%), ammonium (ion selective electrode; CV 1.5%), citrate (enzymatically, citrate lyase; CV 1.6%), uric acid (enzymatically, uricase; CV <5%), and oxalic acid (ion chromatography; CV <2%).

The risk of calcium oxalate crystallization computed as relative supersaturation for calcium oxalate was calculated with the EQUIL2 computer program based on urine composition (Werness *et al*, 1985).

Statistical analysis

Differences between urinary parameters of the corresponding days of each phase were assessed by the two-tailed Wilcoxon matched pairs signed rank test. *P*-values lower than 0.05 were considered to indicate significant differences. Days 4 and 5 of the control and test phases were considered as control and test days, since then conditions of steady state were reached. Moreover, the 24-h urine collections during the follow-up phase were compared with the baseline 24-h urine collections.

Results

The 24-h urine compositions on the corresponding days of the standardized control and test phases are summarized in Table 3. The high magnesium and calcium content of the mineral water led to a significant increase in urinary calcium and magnesium excretion. Moreover, urinary pH and citrate excretion increased significantly, whereas urinary ammonium and phosphate excretion decreased significantly during ingestion of the mineral water. The administration of mineral water had no influence on urinary oxalic acid excretion. The calculation of the relative supersaturation for calcium oxalate, based on urine components, revealed no change in the risk of calcium oxalate crystallization on the ingestion of the mineral water during the standardized test phase.

The circadian rhythm of urinary parameters is demonstrated in Figures 1–4. On the consumption of the mineral water, urinary pH values were significantly higher in each urine fraction compared to the control fractions (Figure 1). Fractional urinary magnesium excretion was higher on the magnesium-rich mineral water, with significant differences in the third, fourth and fifth fractions (Figure 2). The high

Table 3 The 24-h urine parameters and the relative supersaturation for calcium oxalate (RS CaOx) of 12 healthy male subjects on the standardized diet receiving neutral fruit tea (control phase) or mineral water (test phase); mean values (s.e.)

	Control phase				*Test phase*			
	Day 1	*Day 2*	*Day 3*	*Day 4*	*Day 1*	*Day 2*	*Day 3*	*Day 4*
Volume (l/24 h)	2.32 (0.11)	2.55 (0.11)	2.46 (0.11)	2.50 (0.12)	2.55 (0.09)	2.36 (0.11)	2.25 (0.12)	2.29 (0.17)
pH value	6.15 (0.08)	6.26 (0.09)	6.10 (0.07)	6.10 (0.07)	6.52 (0.06)*	6.64 (0.07)*	6.54 (0.06)*	6.59 (0.06)*
Sodium (mmol/day)	173 (14)	180 (11)	181 (11)	166 (9)	184 (11)	191 (7)	186 (8)	180 (7)
Potassium (mmol/day)	80 (4)	71 (3)	73 (3)	71 (3)	62 (3)	64 (3)	67 (2)	61 (3)
Calcium (mmol/day)	5.11 (0.34)	5.21 (0.35)	5.77 (0.48)	5.04 (0.51)	5.89 (0.42)*	5.73 (0.38)*	6.06 (0.51)	5.74 (0.44)*
Magnesium (mmol/day)	5.05 (0.54)	4.92 (0.38)	5.54 (0.45)	4.63 (0.20)	5.80 (0.37)	6.26 (0.42)*	7.41 (0.44)*	7.41 (0.40)*
Ammonium (mmol/day)	43.2 (2.8)	37.7 (3.3)	42.1 (3.2)	37.2 (2.8)	28.4 (1.5)*	27.0 (2.4)*	28.4 (2.1)*	23.8 (1.9)*
Chloride (mmol/day)	188 (14)	180 (14)	191 (10)	188 (12)	185 (9)	176 (8)	169 (8)	160 (7)
Phosphate (mmol/day)	37.3 (2.0)	35.9 (1.9)	38.7 (2.1)	34.6 (1.7)	31.7 (1.8)	30.9 (1.8)	33.1 (1.8)*	29.4 (1.5)*
Sulfate (mmol/day)	28.2 (1.7)	28.1 (1.7)	31.5 (1.9)	27.9 (1.5)	23.3 (1.4)	23.7 (1.1)	27.5 (1.9)	25.0 (1.3)
Creatinine (mmol/day)	17.22 (1.17)	17.25 (0.91)	18.95 (1.28)	16.31 (1.07)	17.27 (0.85)	17.56 (0.80)	18.44 (1.27)	17.60 (0.92)
Uric acid (mmol/day)	3.76 (0.79)	4.09 (2.10)	3.95 (0.61)	3.39 (0.46)	3.37 (0.69)	3.11 (0.80)	3.52 (0.58)	3.25 (0.64)
Oxalic acid (mmol/day)	0.423 (0.097)	0.444 (0.085)	0.418 (0.083)	0.358 (0.065)	0.358 (0.073)*	0.338 (0.060)*	0.373 (0.068)*	0.359 (0.069)
Citrate (mmol/day)	2.626 (0.936)	3.121 (0.639)	3.218 (0.738)	3.045 (0.696)	3.833 (0.869)*	4.188 (1.032)*	4.449 (1.001)*	4.554 (1.094)*
RS CaOx	4.47 (0.60)	3.80 (0.36)	4.55 (0.59)	3.41 (0.36)	3.65 (0.28)	3.48 (0.39)	4.09 (0.43)	4.14 (0.65)

*$P<0.05$.

Figure 1 Circadian rhythm of urinary pH value in 3-h urine collections (the value of the 9-h night interval was divided by 3) of 12 healthy male subjects on the standardized diet receiving neutral fruit tea (control phase, ---) or mineral water (test phase, ——) (*$P<0.05$).

Figure 2 Circadian rhythm of urinary magnesium excretion in 3-h urine collections (the value of the 9-h night interval was divided by 3) of 12 healthy male subjects on the standardized diet receiving neutral fruit tea (control phase, ---), or mineral water (test phase, ——) (*$P<0.05$).

bicarbonate content of the mineral water resulted in an increase in urinary citrate excretion throughout 24 h, with significant differences in the third and fifth fractions (Figure 3). In spite of a significantly higher 24-h urinary calcium excretion on the intake of mineral water, fractional calcium excretion differed strongly from the control only in the third fraction. Although no significant difference was established, relative supersaturation for calcium oxalate was lower during the ingestion of the mineral water, with the exception of the third fraction (Figure 4).

The consumption of the mineral water during the follow-up phase under usual dietary conditions significantly altered urinary volume, pH value and urinary excretion of magnesium, citrate and ammonium compared to baseline phase (Table 4). The favorable changes in urinary parameters resulted in a significant decrease in the relative super-

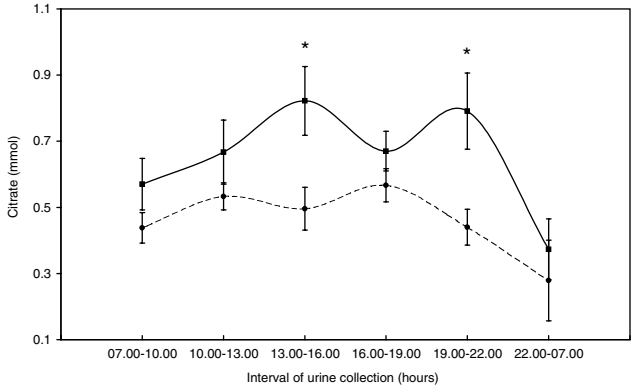

Figure 3 Circadian rhythm of urinary citrate excretion in 3-h urine collections (the value of the 9-h night interval was divided by 3) of 12 healthy male subjects on the standardized diet receiving neutral fruit tea (control phase, ---), or mineral water (test phase, ——) (*$P<0.05$).

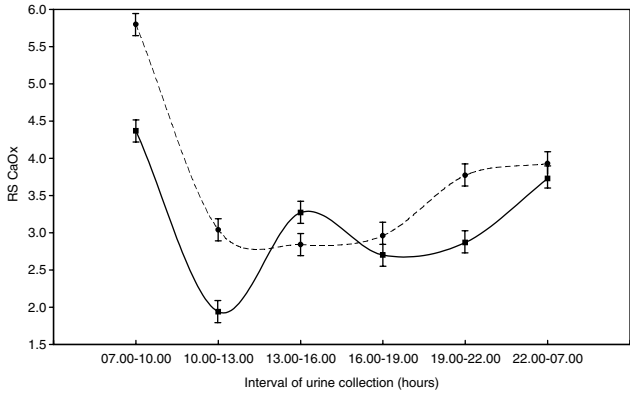

Figure 4 Circadian rhythm of the relative supersaturation for calcium oxalate (RS CaOx) in 3-h urine collections (the value of the 9-h night interval was divided by 3) of 12 healthy male subjects on the standardized diet receiving neutral fruit tea (control phase, ---), or mineral water (test phase, ——).

saturation for calcium oxalate throughout the entire follow-up interval of 4 weeks.

Discussion

The changes in urine composition on standardized conditions corresponded to the high mineral and bicarbonate content of the mineral water. The most evident changes attributable to the consumption of the mineral water were significant changes in urinary pH value and magnesium, calcium, citrate, phosphate and ammonium excretion.

The magnesium supplementation with mineral water led to a significant increase in urinary excretion of magnesium on both standardized and normal dietary conditions. On a dietary magnesium intake of 16.63 mmol/day during the control phase, 4.63 mmol/day magnesium was excreted on average, corresponding to 28% of the supply. On a total magnesium intake of 35.47 mmol/day during the test phase, including that from diet and mineral water, mean magnesium excretion was 7.41 mmol/day, which is 21% of the intake. The results indicate that the increase in magnesium intake by more than 100% diminishes clearly urinary magnesium excretion as a percentage of estimated dietary intake. For a healthy individual in magnesium balance, urinary magnesium excretion reflects approximately intestinal absorption rate (Quamme, 1993). Magnesium absorption in healthy individuals is dose dependent, but may also be influenced by inhibiting and enhancing dietary components. Since diet composition did not vary throughout the standardized phase, the main factor of influence on urinary magnesium excretion in the present study is the ingested amount of magnesium. The level of intestinal magnesium absorption is suggested to be inversely related to the intake. Graham *et al* (1960) demonstrated that 76% of the ingested magnesium was absorbed with a very low dietary magnesium intake (0.95 mmol), while a high intake (23.5 mmol) resulted in a decrease in the absorption to 24%. A recent study in healthy subjects using a stable-isotope technique showed that magnesium from a magnesium-rich mineral water was highly bioavailable and that magnesium absorption was further enhanced when the mineral water was consumed with a meal (Sabatier *et al*, 2002).

Similar changes in the urinary calcium excretion rate resulted from the increase in calcium intake with mineral water under standardized conditions. Although total urinary calcium excretion increased significantly on the consumption of mineral water by 14% on average, urinary calcium excretion as a percentage of dietary intake declined from 25% on the control phase to 21% on the test phase with the application of the mineral water. Our results are consistent with findings of Couzy *et al* (1995). Mean calcium absorption measured with a dual-label stable-isotope technique in healthy female subjects was 23.8%. No relation was established between urinary sodium and calcium excretion, neither on standardized nor on normal dietary conditions. The majority of the studies evaluating the effect of the calcium content of water on urine composition revealed significant increases in urinary calcium excretion both in normal subjects (Rodgers, 1997; Coen *et al*, 2001) and stone formers (Ackermann *et al*, 1988; Marangella *et al*, 1996; Rodgers, 1997; Caudarella *et al*, 1998) on a high as opposed to a low calcium load with water.

In the present study, oxalic acid excretion was not affected by the calcium content of the mineral water, which may be explained by the timing of mineral water ingestion. Individuals were instructed to consume the mineral water between the meals to avoid an enhancement of intestinal calcium absorption by coingested dietary components. Calcium was therefore obviously not available for complexation with oxalate in the gut. In accordance with the present results, Bellizzi *et al* (1999) found no change in urinary oxalate excretion in patients with idiopathic nephrolithiasis during ingestion of hard water as compared with soft water.

Table 4 The 24-h urine parameters and the relative supersaturation for calcium oxalate (RS CaOx) of 12 healthy male subjects on their individual diets receiving mineral water (follow-up phase); mean values (s.e.)

	Control week 1	*Control week 2*	*Week 1*	*Week 2*	*Week 3*	*Week 4*
Volume (l/24 h)	1.87 (0.15)	1.93 (0.17)	2.63 (0.26)*	2.87 (0.32)*	2.56 (0.18)*	2.88 (0.20)*
pH value	6.23 (0.13)	6.34 (0.12)	6.56 (0.11)*	6.69 (0.09)*	6.51 (0.09)*	6.66 (0.07)*
Sodium (mmol/day)	185 (22)	193 (18)	234 (33)*	236 (30)*	239 (19)*	235 (27)*
Potassium (mmol/day)	78 (4)	76 (5)	77 (7)	94 (8)	81 (7)	92 (10)
Calcium (mmol/day)	5.84 (0.56)	6.01 (0.05)	6.16 (0.50)	6.34 (0.54)	6.22 (0.55)	6.17 (0.73)
Magnesium (mmol/day)	4.49 (0.38)	5.18 (0.47)	6.92 (0.76)*	6.93 (0.63)*	7.19 (0.71)*	6.98 (0.72*)
Ammonium (mmol/day)	36.6 (2.1)	35.7 (2.5)	26.9 (2.5)*	24.0 (2.4)*	30.5 (2.4)*	28.4 (2.2)*
Chloride (mmol/day)	188 (19)	190 (18)	213 (30)	213 (21)	210 (19)	211 (27)
Phosphate (mmol/day)	34.6 (1.8)	37.7 (3.0)	32.5 (4.1)	36.7 (3.8)	38.2 (3.0)	34.7 (2.3)
Sulfate (mmol/day)	25.6 (1.5)	25.3 (1.0)	27.4 (1.9)	28.0 (1.9)	29.5 (2.0)	27.6 (2.1)
Creatinine (mmol/day)	16.08 (1.02)	16.23 (1.02)	17.19 (0.94)	18.23 (1.16)	18.56 (1.31)	17.71 (1.09)
Uric acid (mmol/day)	3.23 (1.26)	3.46 (1.01)	3.90 (0.57)	4.18 (1.06)	4.47 (1.16)	4.01 (1.06)
Oxalic acid (mmol/day)	0.446 (0.115)	0.493 (0.224)	0.362 (0.123)	0.514 (0.208)	0.373 (0.118)	0.401 (0.101)
Citrate (mmol/day)	3.191 (0.911)	3.196 (0.611)	4.555 (1.083)*	4.202 (0.731)*	3.863 (0.569)*	4.173 (0.999)*
RS CaOx	6.84 (1.00)	6.54 (0.70)	3.37 (0.46)*	4.05 (0.53)*	3.74 (0.74)*	3.17 (0.47)*

*$P<0.05$.

Even the supply of magnesium with mineral water did not alter urinary oxalate excretion. Although daily doses of 200–400 mg (8.23–16.46 mmol) magnesium, corresponding to the additional magnesium intake with mineral water, are recommended for the treatment of hyperoxaluric calcium oxalate stone formers (Hesse *et al*, 2002), no change in oxalate excretion was observed after the administration of mineral water.

The application of a mineral water rich in bicarbonate (1715 mg/l) to healthy subjects has previously been demonstrated to provide an alkali load equivalent to that of a sodium potassium citrate supplement (Keßler & Hesse, 2000). In the present study, the high bicarbonate content of the mineral water resulted in metabolically induced significant increases in urinary pH and citrate excretion under both standardized and normal dietary conditions. The alkalization of the urine and the increase in citrate excretion are rapidly achieved after water intake, as apparent from circadian rhythm. Moreover, the increases in urinary pH and citric acid excretion are attained over the entire day (Figures 1 and 3). The potassium content of mineral water promoted urinary alkalization and led to a further increase in urinary citrate excretion. During administration of the mineral water, mean urinary pH increased to values between 6.5 and 6.7 (upper physiological limit: 6.8), whereas urinary citrate excretion exceeded 3.9 mmol/day with mineral water already on the subjects' usual diet. These effects of mineral water administration are beneficial for the treatment of recurrent calcium oxalate stone disease. Owing to a decrease in the excretion of hydrogen ions as a result of urine alkalization, urinary ammonium excretion decreased significantly in the present study.

In spite of favorable changes in urinary inhibitory parameters, i.e., the increase in pH value, citric acid and magnesium excretion, the risk of calcium oxalate crystallization remained unchanged during the standardized test phase due to the significant increase in urinary calcium, counterbalancing increased citric acid and magnesium excretion. Whereas fluid intake was kept equal throughout the standardized phases, the results of the follow-up phase showed the diluting effect of an increased water intake under usual dietary conditions. The prescription of an intake of 1.4 l/day of the mineral water accounted for the significant decline in the supersaturation with calcium oxalate due to the significant increase in urinary volume and consequently the decrease in the concentration of lithogenic substances. Moreover, the higher urinary pH value, magnesium and citrate excretion contributed to the decrease in the risk of calcium oxalate crystallization during long-term follow-up.

In conclusion, the administration of the mineral water resulted in favorable changes in urinary constituents. The long-term administration demonstrated that mineral water can contribute to a sufficiently high urine volume and consequently to a decline in the concentration of lithogenic substances and the risk of calcium oxalate crystallization. Furthermore, the high magnesium and bicarbonate content resulted in significant increases in urinary pH value, citrate and magnesium excretion, inhibitors of calcium oxalate stone formation, counterbalancing increased calcium excretion. The results suggest that the mineral water could represent an alternative to pharmacological supplements, especially in calcium oxalate stone patients with hypomagnesuria, hypocitraturia and acidic urinary pH. As a result of the significant increase in urinary calcium excretion on standardized conditions already in healthy subjects without disturbances in calcium metabolism, a high dietary calcium intake, as suggested by Curhan *et al* (1993), seems to be not suitable for calcium oxalate stone formers with absorptive hypercalciuria. Although a sufficiently high fluid intake is recommended in calcium oxalate stone disease, the type of beverage should be carefully selected, since ingestion of a

large amount of any fluid that contains electrolytes or other solutes could have complex effects on urine composition. Since mineral water consumption did not reduce urinary oxalate excretion, further studies are necessary to evaluate whether the ingestion of calcium-rich mineral water with, rather than between, meals is more likely to bind oxalate in the gut thereby limiting intestinal absorption and urinary excretion of oxalate and calcium.

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