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ORIGINAL PAPER

Effects of calcium supplementation on body weight reduction in overweight calcium stone formers

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Abstract A randomized, placebo-controlled trial was conducted in overweight calcium stone-forming (CSF) patients, to evaluate the effect of calcium supplementation associated with a calorie-restricted diet on body weight (BW) and fat reduction and its potential changes upon serum and urinary parameters. Fifteen patients were placed on a hypocaloric diet for 3 months, supplemented with either calcium carbonate ($CaCO_3$, $n = 8$) or placebo ($n = 7$), 500 mg bid. Blood and 24-h urine samples were collected and body composition was assessed at baseline and after the intervention. At the end of the study, final BW was significantly lower vs baseline in both $CaCO_3$ (74 ± 14 vs. 80 ± 14 kg, $P = 0.01$) and placebo groups (80 ± 10 vs. 87 ± 9 kg, $P = 0.02$) but the mean percentage of loss of body weight and body fat did not differ between $CaCO_3$ and placebo (7.0 ± 2.0 vs. $8.0 \pm 3.0\%$, $P = 0.40$ and 13.0 ± 7.0 vs. $13.0 \pm 10.0\%$; $P = 0.81$, respectively). After $CaCO_3$ or placebo, no significant differences versus baseline were observed for urinary parameters in both $CaCO_3$ and placebo, except for a higher mean urinary citrate in placebo group. These data suggest that increasing calcium intake by calcium carbonate supplementation did not contribute to a further reduction of BW and fat in overweight CSF patients submitted to a hypocaloric diet nor altered urinary lithogenic parameters.

Keywords Calcium intake · Weight loss · Kidney stones · Vitamin D

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Introduction

The lifetime prevalence of symptomatic nephrolithiasis is approximately 12% in men and 5% in women [1, 2]. The formation of urinary tract stones is a result of increases in urinary supersaturation, crystal nucleation, aggregation, the retention of crystals by the urothelium, and the continued growth of the stone on the retained crystals. The main determinants of urinary supersaturation are excessive excretion of promoters of urinary crystallization: calcium, oxalate, sodium, uric acid, reduced excretion of inhibitors of urinary crystallization such as citrate or glycosaminoglycans, abnormalities of urinary pH and low urine volume.

Recent epidemiological data have shown that stone disease is associated with a higher BW and Body Mass Index, BMI [3–5]. In one study, BMI was found to be positively associated with urinary excretion of uric acid, sodium, ammonium and phosphate and inversely with urinary pH in both men and women, increasing the risk of stone formation [4]. Urinary calcium was directly correlated with BMI only among men [4]. In a large population of kidney stone formers, the higher the BW, the lower was the urinary pH [6]. Taylor et al. [5] observed that not only obesity but also weight gain increases the risk of symptomatic nephrolithiasis.

Several studies have reported an anti-obesity effect of dietary calcium and dairy products [7–9]. The first underlying mechanism proposed by Zemel [10, 11], is

the effect of dietary calcium on depressing the levels of parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D_3 [$1,25(OH)_2D_3$], leading to a potential reduction in the levels of intracellular calcium, thereby inhibiting lipogenesis and stimulating lipolysis, culminating in accelerated weight loss. The second proposed mechanism by which calcium could impact on BW is that increased dietary calcium might bind more fatty acids in the intestinal colon, hence inhibiting fat absorption [12, 13]. On the other hand, many other investigators did not observe additional effects of calcium supplementation on weight reduction in patients submitted to a calorie-restricted diet [14–19].

In the past, calcium restriction was recommended to kidney stone formers to reduce urinary calcium levels. However, a large prospective epidemiological study conducted in healthy men with different levels of calcium intake showed that the lower the calcium intake the higher was the risk for stone formation probably due to an increase in intestinal absorption of free oxalate because of less calcium left in the lumen to bind with oxalate [20], resulting in secondary hyperoxaluria. In a 5-year randomized controlled trial in hypercalciuric male patients, Borghi et al. [21] observed that a restricted intake of animal protein and salt combined with a normal calcium intake was more effective in preventing stone recurrence than the traditional low calcium diet, once again suggesting that calcium restriction should not be recommended.

Considering that overweight and obesity increase the risk of stone formation, and that calcium supplementation could possibly help in reducing weight and chelating oxalate, as long as taken with meals [22], the present study aimed to evaluate the effect of calcium supplementation on BW reduction in overweight calcium stone-forming (CSF) patients submitted to a calorie-restricted diet and its potential changes upon serum and urinary parameters related to the risk of stone formation.

Patients and methods

Twenty-one (21) overweight CSF female patients (BMI $\geq$ 25 kg/m^2) referred to the Renal Lithiasis Unit of the Nephrology Division, Universidade Federal de São Paulo were asked to complete a 3-day dietary record to assess their food intake. Only 15 patients whose usual calcium intake was $\leq$650 mg/day were selected to be included in the present study. Nine (9) patients have voided calculi in the past, but their stones were not available for analysis. On enrollment, all 15 patients presented uni or bilateral radio-opaque stones (1–5 per patient) consistent with calcium composition. During the period of preceding follow up in the Unit (6 months to 10 years) none of them had formed new stones. Data concerning the definition of distinct metabolic abnormalities related to stone formation were obtained from the results of 24-h urine samples, collected in two non-consecutive occasions, and available in their medical records. Exclusion criteria were abnormal renal function, diabetes, cystinuria, renal tubular acidosis, inflammatory bowel disease, diseases affecting calcium metabolism (hyperthyroidism, primary hyperparathyroidism, acromegaly, sarcoidosis, or neoplasia), use of drugs such as corticosteroids, anticonvulsants, vitamin D, or others. A written consent was obtained from all subjects and the local Ethics Committee of the Universidade Federal de São Paulo approved the study.

Protocol

All patients were submitted to an anthropometric evaluation, body fat measurement through skinfold thickness at four sites (biceps, triceps, subscapular and suprailiac (Lange® Skinfold Caliper, Cambridge, USA) and fat and fat-free mass determination by bioelectrical impedance (BIA 101 Quantum, RJL Systems, Detroit, USA) at baseline and at the end of the study. A fasting blood sample and a 24-h urine sample were collected on both occasions. On completion of the baseline 24-h urine collection and upon its delivery to the laboratory, patients were randomized in a double-blind manner to a calorie-restricted diet (energy deficit of 500 calories/day based on their previous dietary record) during 3 months, supplemented with either calcium carbonate ($CaCO_3$, $n = 8$), 500 mg bid, taken after lunch and dinner (400 mg of elemental calcium per day) or placebo ($n = 7$). During the whole period of study they were instructed to maintain the same calcium intake as assessed by the dietary record and to avoid consuming oxalate-rich foods and vitamin C supplements not to interfere with oxalate excretion. They received weekly diet counselling by telephone and BW was measured biweekly until the completion of the study. Adherence was assessed by pill count at the end of the study, whereas compliance to diet was estimated from a 3-day dietary record obtained at the second and third month. In the fasting blood samples, glucose levels, ionized calcium, phosphorus, lipids (total-cholesterol [TC], low-density lipoprotein [LDL], high-density lipoprotein [HDL], very low-density lipoprotein [VLDL], triglycerides [TG]), uric acid, creatinine, urea, insulin, insulin resistance (estimated by homeostatic model assessment, HOMA-IR), adiponectin, 25-hydroxyvitamin D_3 [$25(OH)D_3$], $1,25(OH)_2D_3$ and PTH were determined. In the 24-h urine samples, calcium, oxalate, sodium, potassium, urea, creatinine, magnesium, uric acid, phosphorus, citrate, urinary pH and a calcium crystallization index were determined.

Nutritional assessment

Daily total energy, lipids, carbohydrate and calcium intakes were assessed through the 3-day dietary record. Sodium chloride (NaCl) intake and protein equivalent of nitrogen appearance (PNA), as a marker of protein intake, were calculated based on 24-h sodium and urea excretion, respectively. Nutrient intakes were calculated using computer software developed in our Department, which contains the US Department of Agriculture (USDA) tables as the nutrient database. The PNA was calculated according to the NKF-KDOQI [23]. Waist circumference was measured with an anthropometric tape to an accuracy of 0.5 cm in the standing position, midway between the lateral lower rib margin and the iliac crest.

Biochemical parameters

Creatinine was determined according to a modified Jaffé reaction through the method of isotope dilution mass spectrometry (IDMS) traceable; urea, citrate, uric acid, serum TC, HDL and TG by an enzymatic method and VLDL/LDL calculated according to the Lipid Research Clinics Program. Phosphorus, magnesium and glucose were measured by a colorimetric method (Olympus AV 400, Tokyo, Japan). Oxalate was determined using a commercial kit (Sigma Diagnostics, St. Louis, USA). Ionized calcium, sodium and potassium were determined by an ion-selective electrode. Hypercalciuria had been defined by serum calcium within normal limits and 24-h urinary excretion of calcium ≥250 mg/day or 300 mg/24-h, for female and male, respectively, hyperuricosuria was considered as urinary uric acid >750 or 800 mg/24-h, for female and male, respectively, hypocitraturia as urinary citrate <320 mg/24-h, and hyperoxaluria as urinary oxalate >45 mg/24-h, as described elsewhere [1].

Urinary pH was measured with an electrode pH meter. Insulin, T4 and TSH levels were determined by immunofluorometric assay in Axsym (Abbott, Chicago, USA). Serum concentrations of $25(OH)D_3$ (normal range: 40–100 ng/mL) and $1,25(OH)_2D_3$ were measured by radioimmunoassay (DiaSorin, Stillwater, USA) and PTH (normal range: 10–65 pg/mL) by chemiluminescence (Immulite, Los Angeles, USA). Plasma concentrations of adiponectin were measured by a sandwich enzyme-linked immunosorbent assay, ELISA (Linco Research, St. Charles, USA). HOMA-IR was calculated according to Matthews et al. [24]. The risk of calcium oxalate crystallization was calculated by Tiselius Index [25].

Statistical analysis

Results were reported as mean ± standard deviation (SD). The Wilcoxon test was used for intra-groups comparisons and Mann–Whitney for inter-groups comparisons and the level of significance was set as $P \leq 0.05$.

Results

The whole sample consisted of 15 stone-forming females, and the BMI categories were distributed as follows: 25.0–29.9 kg/m^2 (26.7%), 30.0–34.9 kg/m^2 (33.3%), 35.0–39.9 kg/m^2 (33.3%), and greater than 40.0 kg/m^2 (6.7%). Hypercalciuria and hypocitraturia were present in 23 and 46% of subjects, respectively, whereas hyperuricosuria was seen in 8% of them. These disturbances appeared isolated or in association. Hyperoxaluria was absent. Table 1 shows the mean values of anthropometric and body composition parameters at baseline and after $CaCO_3$ or placebo supplementation. Mean age and baseline parameters were not statistically different between groups. After supplementation, we did not observe statistical differences between groups except for a lower mean fat-free mass in $CaCO_3$ versus placebo (44 ± 4 vs. 48 ± 3, $P \leq 0.05$). Delta changes in BW and body fat were not statistically different between placebo and $CaCO_3$ groups (7.0 ± 3.0 vs. 5.5 ± 2.0 kg) and (5.0 ± 4.0 vs. 4.0 ± 2.0 kg), respectively. At the end of the study, the intra-group comparisons showed that mean values of BW, BMI, waist and hip circumference and body fat, assessed by skinfold thickness or BIA were significantly lower versus baseline. Mean abdominal circumference decrease was significant only in placebo group, and fat-free mass did not change in any of the groups. As seen in Fig. 1, the values of percentage of loss in $CaCO_3$ and placebo groups, were not significantly different for BW (7.0 ± 2.0 vs. 8.0 ± 3.0%, $P = 0.40$), body fat (13.0 ± 7.0 vs. 13.0 ± 10.0%, $P = 0.81$) and waist circumference (4.0 ± 3.0 vs. 5.0 ± 3.0%, $P = 0.40$).

Dietary intakes at baseline and after 3 months are presented in Table 2. Nutrient intakes at baseline were not significantly different between groups. At the end of the study, the mean reported intakes of energy, lipids and carbohydrate were significantly lower in both groups. Protein intake assessed by PNA, did not differ from baseline in any group. It is noteworthy that the mean value of daily energy achieved at the end of the study was very low for both groups, revealing that patients exceeded the recommendation of a deficit of 500 calories during the intervention. Despite of instruction to maintain the level of calcium intake, patients from both groups were consuming less calcium at the end of the study. The higher value observed in the $CaCO_3$ group at completion of study was ascribed to the calcium supplementation.

Table 3 shows the mean values of serum parameters at baseline and after $CaCO_3$ or placebo supplementation. Significant differences compared to baseline were not

Table 1 Anthropometrics and body composition parameters at baseline (pre) and after 3 months (post) of calcium supplementation ($CaCO_3$) or placebo

	Placebo group ($n = 7$)		$CaCO_3$ group ($n = 8$)	
	Pre	Post	Pre	Post
Weight (kg)	87 ± 9	80 ± 10[a]	80 ± 14	74 ± 14[b]
BMI (kg/m^2)	34 ± 4	32 ± 4[a]	34 ± 5	31 ± 5[b]
Waist circumference (cm)	97 ± 4	92 ± 6[a]	93 ± 11	90 ± 11[a]
Hip circumference (cm)	115 ± 10	111 ± 9[a]	114 ± 12	110 ± 13[a]
Abdominal circumference (cm)	105 ± 3	100 ± 4[a]	102 ± 12	98 ± 12
Body fat (skinfold thickness), %	42 ± 2	39 ± 3[a]	40 ± 5	38 ± 5[a]
Body fat (BIA), kg	38 ± 8	33 ± 8[a]	32 ± 9	28 ± 10[a]
Fat-free mass (BIA), kg	49 ± 4	48 ± 3	45 ± 4	44 ± 4[c]
Δ Weight loss, kg	–	7.0 ± 3.0	–	5.5 ± 2.0
Δ Fat loss, kg	–	5.0 ± 4.0	–	4.0 ± 2.0

[a] $P \leq 0.05$ versus pre;
[b] $P \leq 0.01$ versus pre;
[c] $P \leq 0.05$ versus placebo in the same period; Δ (delta changes)

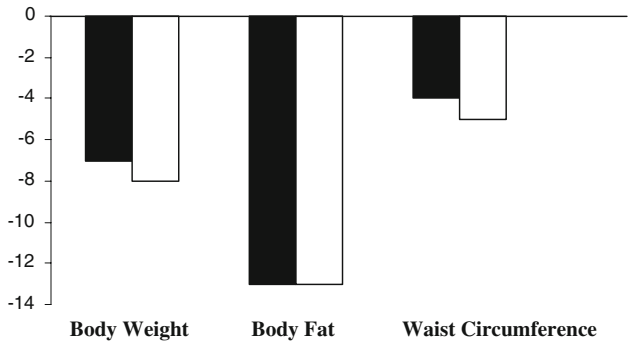

Fig. 1 Effects of different treatments on the percentage of reduction of body weight, body fat and waist circumference. *Filled boxes* refer to $CaCO_3$ and open to placebo groups

observed, except for fasting insulin, HOMA-IR and adiponectin in $CaCO_3$ group and serum urea and VLDL-cholesterol in placebo group.

As shown in Table 4, mean urinary parameters after $CaCO_3$ or placebo supplementation did not differ from baseline, except for a higher mean urinary citrate in placebo group.

Discussion

Previous studies have demonstrated that overweight and obesity are associated with an elevated risk of kidney stone formation [3–5]. Evidence from clinical and experimental research and epidemiological investigations support an inverse relationship between calcium intake and kidney stone risk [20, 21] as well as between calcium intake and BW [7–9, 26].

In the present study, we evaluated the effect of a double-blind randomization of overweight CSF patients to a hypocaloric diet with or without calcium supplementation for 3 months and its potential changes upon serum and urinary parameters related to the risk of stone formation.

The compliance to the weight loss program was very good in the present series, since at the end of the study the mean decrease of BW was around 6–7 kg (7–8% of BW) in $CaCO_3$ and placebo groups with body fat being reduced accordingly. Actually, reported intakes of energy and macronutrients at completion of the study were significantly lower than baseline, and beyond the recommendation of a deficit of 500 calories suggesting that patients were motivated by the intervention. However, calcium supplementation (1 g/day) neither accelerate weight or fat loss as expected, nor was able to change serum levels of $1,25(OH)_2D_3$. In agreement with our results, Ricci et al. [14] and Shapses et al. [15] also observed similar amounts of weight loss over 6 months of a weight reduction program in premenopausal and postmenopausal women supplemented or not with 1 g/day of calcium carbonate. The lack of effect of calcium supplementation on acceleration of

Table 2 Nutrient intake at baseline (pre) and after 3 months (post) of calcium supplementation ($CaCO_3$) or placebo

	Placebo group ($n = 7$)		$CaCO_3$ group ($n = 8$)	
	Pre	Post	Pre	Post
Energy (Kcal/day)	2,378 ± 804	1,168 ± 311[a]	2,111 ± 337	1,308 ± 300[b]
Fat (g/day)	87 ± 29	34 ± 15[a]	74 ± 17	44 ± 20[a]
Carbohydrates (g/day)	305 ± 120	161 ± 60[a]	278 ± 58	167 ± 34[b]
Calcium (mg/day)	575 ± 59	358 ± 123[a]	495 ± 99	364 ± 206[b,c]
PNA (g protein/day)	60 ± 24	49 ± 17	53 ± 15	52 ± 14
NaCl (g/day)	16 ± 6	13 ± 7	11 ± 4	10 ± 5

[a] $P \leq 0.05$ versus pre;
[b] $P \leq 0.01$ versus pre;
[c] plus $CaCO_3$ (1,000 mg/day)

Table 3 Serum parameters at baseline (pre) and after 3 months (post) of calcium supplementation (CaCO3) or placebo

	Placebo group ($n = 7$)		$CaCO_3$ group ($n = 8$)	
	Pre	Post	Pre	Post
Ionized calcium (mmol/L)	1.31 ± 0.02	1.31 ± 0.03	1.3 ± 0.1	1.3 ± 0.1
PTH (pg/mL)	61 ± 28	50 ± 18	67 ± 31	45 ± 15
25 $(OH)D_3$ (ng/mL)	25 ± 8	27 ± 7	26 ± 8	24 ± 11
1.25 $(OH)_2D_3$ (pg/mL)	67 ± 30	80 ± 42	60 ± 14	75 ± 30
T4 (ng/dL)	1.1 ± 0.1	1.1 ± 0.1	1.0 ± 0.1	1.1 ± 0.1
TSH (μUI/mL)	2.0 ± 0.8	2.0 ± 1.0	2.0 ± 1.0	2.0 ± 1.1
Fasting Glucose (mg/dL)	90 ± 5	92 ± 10	87 ± 7	89 ± 4
Fasting Insulin (μUI/mL)	11 ± 5	10 ± 4	10 ± 3	7 ± 2[a]
HOMA-IR	2.4 ± 1.2	2.1 ± 1.0	2.1 ± 0.7	2.0 ± 0.4[a]
Adiponectin (μg/mL)	9 ± 4	9 ± 3	11 ± 11	9 ± 8[a]
Total-cholesterol (mg/dL)	190 ± 48	181 ± 42	178 ± 39	172 ± 35
HDL-cholesterol (mg/dL)	57 ± 17	55 ± 13	53 ± 14	51 ± 13
LDL-cholesterol (mg/dL)	102 ± 34	101 ± 29	102 ± 38	101 ± 33
VLDL-cholesterol (mg/dL)	31 ± 8	24 ± 10[a]	24 ± 12	20 ± 6
Triglycerides (mg/dL)	154 ± 38	122 ± 50[a]	122 ± 60	99 ± 32
Creatinine (mg/dL)	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.2
Urea (mg/dL)	29 ± 9	25 ± 6[a]	28 ± 7	26 ± 6
Uric Acid (mg/dL)	5.4 ± 1.0	5.0 ± 1.0	6.0 ± 1.0	5.3 ± 0.5
Magnesium (mg/dL)	2.0 ± 0.2	2.0 ± 0.3	2.1 ± 0.2	2.0 ± 0.1
Phosphorus (mg/dL)	3.3 ± 0.4	3.5 ± 0.5	3.4 ± 1.0	3.4 ± 0.4

[a] $P \leq 0.05$ versus pre

Table 4 Urinary parameters at baseline (pre) and after 3 months (post) of calcium supplementation ($CaCO_3$) or placebo

	Placebo group ($n = 7$)		$CaCO_3$ group ($n = 8$)	
	Pre	Post	Pre	Post
Calcium (mg/day)	174 ± 80	211 ± 185	140 ± 61	158 ± 69
Oxalate (mg/day)	28 ± 6	28 ± 8	25 ± 8	26 ± 5
Uric Acid (mg/day)	607 ± 118	491 ± 133	519 ± 118	485 ± 114
Magnesium (mg/day)	76 ± 27	79 ± 29	65 ± 33	60 ± 10
Citrate (mg/day)	290 ± 141	446 ± 206[a]	450 ± 392	481 ± 331
Sodium (mEq/day)	267 ± 96	214 ± 114	179 ± 68	170 ± 76
Potassium (mEq/day)	49 ± 28	54 ± 23	65 ± 68	44 ± 13
Phosphorus (mg/day)	806 ± 277	644 ± 292	681 ± 292	620 ± 199
Urea (g/day)	21 ± 8	17 ± 6	18 ± 5	18 ± 5
Creatinine (mg/day)	1,194 ± 262	1,298 ± 371	1,197 ± 353	1,217 ± 352
pH	6.2 ± 0.8	6.2 ± 0.5	6.1 ± 0.3	6.2 ± 0.4
Tiselius index	0.47 ± 0.70	0.66 ± 0.60	1.20 ± 1.10	1.10 ± 1.05

[a] $P \leq 0.05$ versus pre

weight loss in the present series could have been ascribed to the smaller amount of elemental calcium or due to its shorter duration. Nevertheless, the aforementioned studies that utilized 1,000 mg of elemental calcium [14, 15, 17] rather than 400 mg as we did, and lasted 6–30 months [14, 15, 17, 18], also did not show positive results. The rationale of not using an even higher amount of calcium was because this series consisted of stone-formers where increases of dietary calcium up to 1,500 mg/day were believed to be adequate for the protocol hence producing no harm. Strong experimental evidences in the literature [27, 28] had suggested that increases on calcium intake could lead to a potential inhibition of $1,25(OH)_2D_3$ and PTH thereby reducing intracellular calcium uptake on adipocytes stimulating lipolysis and decreasing lipogenesis, favoring weight and fat loss. Nevertheless, clinical data regarding the effect of calcium supplementation on calciotropic hormones during weight loss are heterogeneous with respect to type,

amount and duration of supplementation and the majority of them did not report important reductions on serum 1,25 vitamin D levels. In the current study, we observed a trend, albeit not significant ($P = 0.06$), for suppression of PTH of 33% in the $CaCO_3$ group, not observed in placebo. This finding agrees well with the report of Ricci et al. [14] who found 13.5% reduction of PTH levels with calcium supplementation.

In our series, $1,25(OH)_2D_3$ levels were not significantly reduced by calcium supplementation. Gunther et al. [16], also did not report changes in $1,25(OH)_2D_3$ levels in a study whose effects of dairy products upon weight and fat mass reduction were also negative.

In the present study, a slight by significant decrease in insulin levels and HOMA-IR had been detected in the $CaCO_3$ supplemented group but not in placebo, after weight loss intervention. These results are in agreement with the hypothesis that dairy products may decrease intracellular Ca^{++} within pancreatic islet cells hence inhibiting insulin and lipogenesis and enhancing lipolysis [9, 29]. The effect on HOMA-IR presently found might have been related to calcium supplementation and not attributable to the weight loss itself since both groups lost weight. Nonetheless, Melanson et al. [30] demonstrated that fasting insulin levels were not significantly reduced by calcium supplementation.

Some studies reported that obese subjects have low levels of adiponectin [31–33], a protein hormone secreted by adipocytes with insulin-sensitizing effects and that a weight loss intervention could increase adiponectin plasma levels [32, 34]. However, in the present series, we observed a small but significant decrease of adiponectin levels on $CaCO_3$ group instead. Most of the studies dealing with calcium supplementation have not determined plasma levels of adiponectin except for Ryan et al. [34] who did not find changes on adiponectin levels on a 6 months weight reduction program associated with physical activity.

Since, taking calcium supplement with a meal can avoid the increase on the risk of calcium stone formation due to an oxalate chelating effect without compromising intestinal calcium absorption [22], we had chosen to prescribe the supplements according to this schedule. In the present series, neither urinary calcium increased nor urinary oxalate decreased. Riedt et al. [19] also did not observe increases in calcium excretion after 1.8 g/day of calcium supplementation. Our finding might be explained by the lower amount of calcium we had employed compared to the dose of 3 g given in the study of Domrongkitchaiporn et al. [22]. It is also possible that the lack of decrease in oxalate excretion had been due to the low oxalate intake recommended during the whole period of the study. As observed by Heller et al. [35], dietary oxalate content in the diet influences the ability of dietary calcium to alter urinary oxalate, as small amounts may be insufficient to be bound by calcium.

Due to its alkaline content, calcium carbonate supplementation was expected to increase urinary citrate and pH. However, despite of small increments in both, the difference did not reach statistical significance in the present study. It is again possible that calcium amount was not enough to produce such an effect. On the other hand, in agreement with our results, other authors [36, 37] also did not observe significant changes in these urinary parameters following calcium carbonate.

Although both clinical [7–9] and experimental data [27, 28] reveal that high calcium intakes are associated with significant decreases on BW and body fat, data in humans remain controversial [14–19]. As shown by Zemel et al. [7] dairy products exert a substantially greater effect on both weight and fat loss compared to an equivalent amount of supplemental calcium, possibly due to bioactive compounds in dairy products such as conjugated linoleic acid (CLA) and branched-chain amino acids (BCAA) which could act independently or synergistically with the calcium to inhibit lipogenesis and stimulate lipolysis. Since the mechanism of this additional effect exerted by dairy product are not yet clear, further series that employ dairy supplementation aimed to evaluate weight loss as well as potential changes upon serum and urinary parameters related to the risk of stone formation in overweight CSF patients are necessary.

In conclusion, overweight stone-forming patients submitted to a calorie restriction lost weight but did not benefit from a calcium carbonate supplementation with respect to further weight and fat mass reductions. Present data also suggested that increasing calcium intake up to approximately 1,500 mg/day did not alter urinary lithogenic parameters in CSF patients whose previous calcium intake was below 650 mg/day.

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