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Research Article

EFFECT OF PYRIDOXINE SUPPLEMENTATION ON URINARY CALCIUM AND OXALATE LEVELS IN HEALTHY INDIVIDUALS AND RENAL STONE PATIENTS: A COMPARATIVE STUDY

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ABSTRACT

A study was conducted to examine the effects of pyridoxine (Vitamin-B6) supplementation on calciuria and oxaluria in 40 healthy subjects and 34 subjects with renal stones. Calcium and oxalate levels in the urine were determined in presupplementation periods and after each 20 day interval during supplementation in both healthy individuals and stone patients. Depending on their pre-supplementation (baseline) oxaluria levels, stone patients were divided into mild and moderate hyperoxaluriac groups. Patients with urinary stone formed decreased their mean 24-hour urinary oxalate levels significantly when supplemented with 10 mg pyridoxine for 60 days at a rate of 10 mg/day. Controls, however, showed only a mild decrease in oxaluria levels. In both controls and stone patients, pyridoxine supplementation resulted in only mild and not significant decreases in mean calciuria levels. In light of results, pyridoxine therapy was discussed in relation to oxalate urolithiasis.

Keywords:- Urolithiasis, urinary stones, calciuria, oxaluria, and pyridoxine supplementation.

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INTRODUCTION

A number of risk factors contribute to the formation of urinary stones. Risk factors for hypercalciuria are hypercalciuria and hyperoxaluria (1). Calcium oxalate precipitates in the urinary tract as a result of these factors, causing urolithiasis. Due to its high insoluble content, calcium forms the most stubborn urinary stones. Metals such as calcium are important macronutrients. In the body, it performs a number of vital functions. It is essential for the body to maintain calcium homeostasis. In contrast, oxalic acid is not a nutrient. Insoluble calcium oxalate is formed, impairing calcium absorption. Endogenous synthesis is the main source of oxalic acid in the body. Diet also plays a role. Urine excretes it since it has no useful role to play. Therefore, calcium oxalate urolithiasis prevention should target hypercalciuria rather than hyperoxaluria. In urolithiasis, hyperoxaluria is a more potent risk factor than hypercalciuria (2).

A hyperoxaluria occurs when the body produces too much oxalates. Ascorbate degradation and glyoxylate metabolism are two sources of oxalates. As a result of increased glyoxylate formation and metabolism, humans are believed to be affected by this condition. Oxalate is excessively formed in the presence of carboligase or D-glycerate dehydrogenase deficiencies. (3)It occurs in families as an autosomal recessive trait. In recent years, efforts have been made to control oxaluria by regulating oxalate biosynthesis enzymes. The oxalate synthesis in the body is also influenced by vitamins, as is currently being investigated.

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METHODS AND MATERIALS

A total of 40 healthy normal individuals (30 males and 10 females) and 34 patients with urinary stones (28 males and 6 females). The subjects were all between the ages of 31-55. Healthy individuals engaged in diverse occupations and had no recent medical reports. A random selection of stone patients was made from the local clinics and confirmed with ultrasound or x-ray that they had urolithiasis. However, there was no recent history of familial urolithiasis among the patients.

During the experiment, no medication was administered to the stone patients. Nonvegetarian food was consumed by all subjects (healthy people and stone patients). According to their dietary recall, they have a lower risk of vitamin-B 6 deficiency. Their incomes ranged from middle to low.

Each subject's urine output was collected and stored in sterile plastic containers over the course of 24 hours. Each sample was preservative-treated with 1% thymol. Calcium and oxalate content of the samples was determined using standardized methods. Approximately half of the urine samples were treated with concentrated HCI in order to estimate calcium levels. After the samples were collected, they were used as soon as possible after they were collected. For each subject, urine samples were collected and analyzed for ten days straight. The average 24-hour urinary calcium and oxalate content was calculated.

The urinarystone patients were divided into two groups based on the mean urinary oxalate level. A group of patients with mild hyperoxaluria was grouped together for the purpose of study. Oxaluria levels in another group of stone patients were relatively high (oxalate levels > 110 mg/24 hr). As a result, all subjects were divided into three groups:

Group-I: It consisted of 40 healthy individuals.
Group-Il: These patients were diagnosed with urinary stones and had mild hyperoxalateuria.
Group-III: included 16 urinary stone patients with relatively high (moderate) urine oxalate levels (oxalate over 110 mg/24 hr).

It is worth noting that in all three groups, the same amount of pyridoxine hydrochloride (vitamin-B6) tablets (Benadon tablets) were administered orally at a dose of 10 mg per day to the subjects in all three groups.

A 60-day supplementation was administered. During this period, all subjects continued their usual dietary pattern. Urine samples were collected every 20 days for each subject. A calcium and oxalate analysis was performed on the samples.

Each N group's urinary calcium and oxalate levels for the past 24 hours were determined. The mean change in urinary calcium and oxalate levels over 60 days was then calculated. A mean percentage change was also calculated. A t-test was conducted to determine the difference between the initial and final urinary calcium-oxalate levels.

Trinder's method was used to estimate the calcium content of urine samples. During the precipitation of calcium present in a sample, napthylhydroxamic acid is used (calcium reagent). An coloured complex produced by the precipitate solution is then measured cotorimetrically (7) by combining it with ferric nitrate (colour reagent).

Oxalate in urine estimation

Using calcium chloride solution, calcium oxalate precipitates from the sample. To determine the first permanent pink coloring, the precipitate is dissolved in 1.0N-H2SO4 and titrated against a potassium permanganate standard solution. (8)

RESULTS AND DISCUSSIONS

Urinary calculogenesis is associated with hyperoxaluria. A stone was formed when urinary calcium precipitates out on a suitable surface (matrix). Oxalate stones are the most stubborn. Endogenous synthesis and dietary sources contribute to urinary oxalate. Most of the oxalate comes from endogenous synthesis (86%). Obesity is associated with increased oxalic acid synthesis.

Oxatate synthesis is also increased in the presence of vitamins, such as vitamin B 6. Additionally, pyridoxine deficiency has been associated with hyperoxaluria (5). Due to carrier-mediated oxalate uptake across intestinal brush border membranes, oxalate absorption in pyridoxine deficiency is enhanced (5).

In addition, endogenous oxalate biosynthesis increased by several fold when pyridoxine deficiency was present as glycolic acid oxidase, glycolic acid dehydrogenase, and lactate dehydrogenase convert glycolate into oxalate (5). It is unclear how pyridoxine affects carboligase and glyoxylate dehydrogenase.

There is a low daily requirement for pyridoxine (2 rag/day), which can be met by whole grain cereals, vegetables, and meat. The antituberculosis drug ioniazid is a recognized antagonist of the pyridoxine enzyme in the body.

In addition to dietary protein intake, pyridoxine is required for many metabolic reactions involving amino acids. In general, pyridoxine therapy may control hyperoxaluria since pyridoxine deficiency increases oxalate synthesis and absorption.

Table 1: Calcium levels in the urine after pyridoxine supplementation

Group	Mean urinary calcium (mg/24 hr)				Net decrease in calcium level (Initial-Final)	% decrease in calcium level	Level of significance
	Pre-supplementation (Initial)	Post supplementation Period (days)					
		20	40	60			
Group-I	123.3±13.5	119.2±15.3	121.2±16.8	118.6±227	5.7±3.4	4.8±2.7	NS·
Group-II	182.7±24.8	180.6±19.1	176.8±13.3	173.9±19.3	9.8±3.8	5.8±2.9	NS
Group-III	193.3±33.7	195.6±22.7	189.4±25.6	184.7±29.4	9.6±4.2	5.5±4.1	NS

Table 2: Supplementation with pyridoxine affects urinary oxalate levels

Group	Mean urinary calcium (mg/24 hr)				Net decrease in calcium level (Initial-Final)	% decrease in calcium level	Level of significance
	Pre-supplementation (Initial)	Post supplementation Period (days)					
		20	40	60			
Group-I	47.4± 9.2	46.2± 10.3	45.6± 6.7	43.6± 10.3	4.8±2.7	9.2±2.9	NS
Group-II	94.6±13.3	91.2±14.4	85.3± 10.3	78.4± 7.9	17.2±4.2	18.3±3.4	P < 0.02
Group-III	139.5±17.1	134.4±14.5	119.2±13.3	83.7±15.5	56.8±7.4	41.8±5.6	P < 0.002

Dietary calcium levels and gastrointestinal calcium absorption determine calcium excretion in the urine. Intestinal contents play a role in calcium absorption. Alkalinity of the intestinal contents precipitates phosphate, which is lost in the feces. Acidity of the intestinal contents increases calcium absorption. Calcitonin and parathromone regulate calcium excretion in the urine. Urine calcium excretion varies between 100 and 300 mg/day in response to a calcium diet of 800 mg/day (9). Low calcium diets can, however, range from 50-150 mg per day (9). As most of the subjects studied were middle- to low-income, 182.7±24.8 mg/day and 193.3 ±33.7 mg/day could be considered mild hypercalciuria.

According to Table 1, pyridoxine supplementation does not significantly affect urinary calcium levels. After 60 days of supplementation, neither the healthy nor stone patients' mean 24-hour urinary calcium levels have changed appreciably. Group-I normocalciudacs showed a modest decline of 5.8± 2.9 mg/24 hr only. The hypercalciurics recorded only a small decline of 9.6±4.2 mg/24 hr. There is only a 5.5 percent decrease over the initial value. Therefore, vitamin B6 does not directly affect urinary calcium output.

According to Table 2, vitamin-B 6 affects urinary oxalate levels. Urinary oxalate reference intervals are 20-60 mg/day for men and 20-55 mg/day for women (9). In the Group-I subjects, there was a decrease in the level of 4.8±2.7 mg/24 hr of oxalate after 60 days of supplementation in the Group-I subjects. There was a corresponding increase in percentage of 9.2±2.9. In patients with mild hypercalciuria (Group-II), there was an increase of 17.2±4.2 rag/24 hr for oxalate after 60 days of vitamin-B 6 treatment. The result of this was that there was a 18.3±2:2.4% decrease. For 60 days, vitamin-B supplementation significantly reduced urinary oxalate levels among stone patients with elevated oxaluria (Group-III). Compared to the previous day, there has been a significant reduction of 56.8±7.4 mg/24 hr. It is interesting to note that when compared to the mean initial value, the decrease was 41.8±5.6%. Oxalate levels decreased quite slowly at first in all cases. There was no difference in urinary oxalate levels after 20 days of supplementation in any of the groups. After 40 days, only Group-Ill showed some significant lowering. A long-term supplementation with vitamin-B 6 can significantly reduce urinary oxalate levels. An increase in vitamin-B6 load may lead to a slow decrease in endogenous oxalate synthesis.

CONCLUSION

Oral supplementation of pyridoxine (vitamin-B6) can significantly reduce urinary oxalate levels in patients with hyperoxaluriac urolithiasis. Calcium levels in the urine, however, are not significantly affected. In fact, hypercalciuria does not directly correlate with

urolithiasis risk. In urinary calculogenesis, urinary anions precipitate calcium out. Hence, oxalate level is more important when it comes to stone formation in urine. The prevention of urinary stones requires controlling endogenous oxalate synthesis. Overall, our present study indicates that long term pyridoxine therapy can benefit urolithiasis patients with moderate/severe hyperoxaluda.

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