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TRANSIENT HYPEROXALURIA AFTER INGESTION OF FOOD RICH IN OXALIC ACID AS A HIGH-RISK FACTOR FOR CALCIUM OXALATE CALCULI

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Summary

In six male volunteers the variation of diurnal oxalic acid excretion after ingestion of food rich in oxalic acid was studied. The ingestion of chocolate caused a transient hyperoxaluria reaching the values found in cases with primary hyperoxaluria. The peak urinary oxalic acid excretion rate occurred within two to four hours after ingestion of the chocolate. These peak values were 289 per cent of the initial rate when 100g of chocolate was eaten and 235 per cent after the intake of 50g of chocolate. Ranitidine, a potent H_2 receptor antagonist, had no influence on the behaviour of the oxalic acid excretion. Our results emphasize the importance of the restriction of alimentary oxalic acid in cases at risk for stone disorders.

Introduction

Dietary factors are involved in the pathogenesis of idiopathic calcium oxalate stones. Alimentary oxalic acid is believed to be of minor importance since only a negligible increase of total urinary oxalic acid excretion was noted in 24-hour samples after ingestion of food rich in oxalic acid [1]. We have supposed that important variations of oxalic acid excretion are not reflected in 24-hour samples. Therefore we studied the diurnal variation of urinary oxalic acid excretion after ingestion of food rich in oxalic acid. Moreover, the influence of ranitidine, a potent H_2 receptor antagonist, on the behaviour of oxalic acid excretion was investigated.

Material and methods

Six male volunteers with normal kidney function were investigated. The mean age was 31 (22–52) years and the mean body surface area was $1.98 \pm 0.16 m^2$. After an overnight fast and a standard breakfast consisting of bread, jam and

a cup of tea or coffee (oxalic acid content about 10mg, calcium content about 40mg) the volunteers ingested 100g chocolate or 50g chocolate respectively at 9.00am. Fluid intake was unrestricted. For lunch (1.00pm) and dinner (6.00pm) food rich in oxalic acid was avoided. Urine samples were collected in two hour periods from 7.00am until 1.00am and in a night period lasting from 1.00am to 7.00am. Moreover the trial with 100g chocolate was repeated in four of the six volunteers after administration of ranitidine (150mg orally twice a day, two days prior to and on the day of the study).

Oxalic acid was determined using the oxalate decarboxylase – formate dehydrogenase – method (UV-method for the determination of oxalic acid in foodstuffs and other materials by Boehringer Mannheim). Prior to the determination of the urine samples the pH was adjusted to pH1 by addition of concentrated hydrochloric acid in order to dissolve calcium oxalate crystals. This procedure is necessary since crystalline oxalic acid can escape the enzymatic determination [2]. While stirring the urinary samples the urinary pH was finally adjusted to 5.0, the value required for the enzymatic method.

Urinary calcium excretion and the calcium concentration of the chocolate were measured by a flame photometer (Eppendorf).

Results

Trial with 100g of chocolate

The mean fasting urinary oxalic acid excretion of the volunteers was 1.00± 0.26mg/hr. After ingestion of 100g chocolate the oxalic acid excretion increased to 2.23±0.36mg/hr within two hours ($t=7.61$; $p<0.001$) and to 2.89± 0.60mg/hr within two to four hours ($t=8.64$; $p<0.001$). This peak value was 289 per cent of the initial excretion. Thereafter the oxalic acid excretion decreased rapidly. It was 2.05±0.52mg/hr within a further two hours and fell approximately to the initial value in the following two hours (1.56±0.28mg/hr) (Figure 1). Total urinary oxalic acid excretion was 34.83±4.43mg/day (normal <45mg/day).

Urinary calcium excretion was as follows: pre-value 12.70±5.60mg/hr; within two hours 10.78±3.60mg/hr; within two to four hours 8.60±5.20mg/hr; within four to six hours 8.00±4.00mg/hr; within six to eight hours 10.4± 3.48mg/hr. The differences were not statistically significant. Total urinary calcium excretion was 244.8±77.8mg/day.

Trial with 50g of chocolate

Similar to the trial with 100g of chocolate a striking increase of oxalic acid excretion was found after ingestion of 50g chocolate. The fasting oxalic acid excretion rose from 1.12±0.37mg/hr to 2.63±0.83mg/hr within two hours ($t=4.4$; $p<0.001$, 235 per cent of the initial value) and to 2.48±0.84 within two to four hours ($t=5.69$, $p<0.001$, 222 per cent of the initial value). Thereafter the oxalic acid excretion fell rapidly, approximately to the initial value

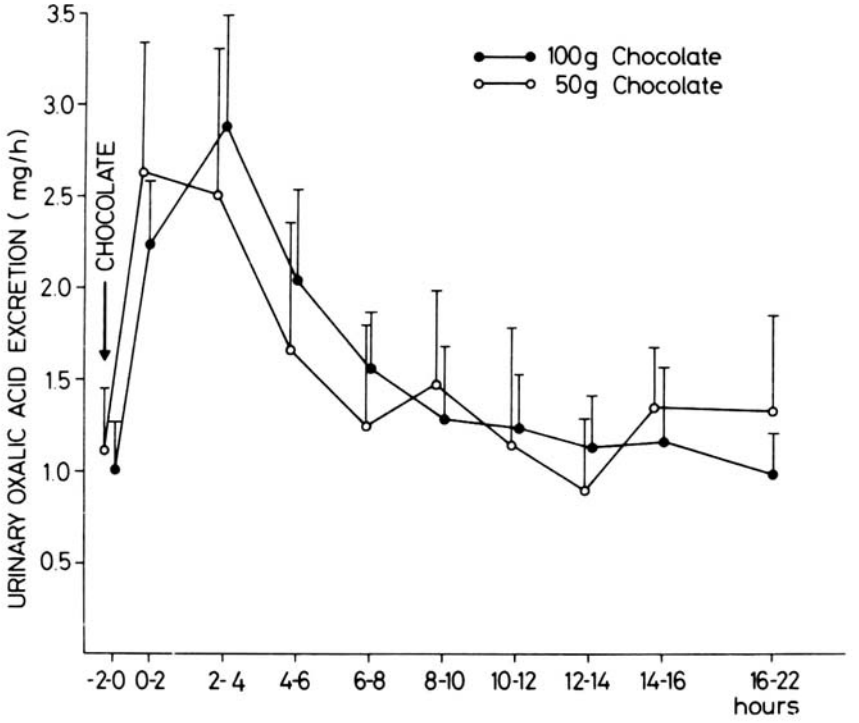

Figure 1. Behaviour of the urinary oxalic acid excretion in six volunteers before and after ingestion of 100g and 50g of chocolate respectively

within four further hours (Figure 1). Total urinary oxalic acid excretion was 36.58±7.82mg/day. The behaviour of the oxalic acid excretion did not differ statistically significantly from the values of the trial with 100g of chocolate.

Trial with 100g of chocolate after ranitidine administration

In four out of six volunteers the trial was repeated while they were on ranitidine administration. In these the fasting oxalic acid excretion increased from 1.01± 0.59mg/hr to 2.05±1.06mg/hr within two hours ($t=2.70$; $p<0.05$) and to 2.68± 0.86mg/hr within two to four hours ($t=8.24$; $p<0.005$). Total oxalic acid excretion was 39.08±11.26mg. The behaviour of the oxalic acid excretion did not differ significantly from the trial with 100g and 50g chocolate.

Estimation of the oxalate and calcium content of the chocolate

Oxalic acid content of the chocolate was 111.16mg/100g of chocolate. Calcium content of the chocolate was 60.8mg/100g of chocolate.

Discussion

The pathogenesis of idiopathic calcium oxalate stones is believed to be multifactorial [1]. Our study shows that dietary oxalic acid intake could play a more important role than commonly assumed. It can be concluded from our data that oxalic acid ingested causes a striking transient hyperoxaluria due to absorption in the upper gastrointestinal tract. The peak oxalic acid excretion occurs within two to four hours of intake. The peak excretion rates were 289 per cent of the initial value in the trial with 100g of chocolate and 235 per cent of the initial value in the trial with 50g of chocolate.

Experimental studies had shown that oxalic acid and calcium form calcium oxalate crystals at a pH value of more than 2.0 [2]. These crystals cannot pass the mucosal barrier. Therefore a further investigation was performed in order to study whether the behaviour of oxalic acid absorption could be influenced by administration of ranitidine which increases the gastric pH value. However our trial failed to show any effect of ranitidine administration on oxalic acid absorption.

The peak oxalic acid excretion rates in our investigations reaches the values found in cases with primary hyperoxaluria, who have a two to threefold increase of urinary oxalic acid excretion [3]. It is therefore likely that the ingestion of food rich in oxalic acid such as chocolate could be an important factor for the formation of calcium oxalate calculi in patients with stone disorders. This risk factor is not reflected in 24-hour samples which are commonly performed, since total oxalic acid excretion in our subjects was within the normal range.

Acknowledgment

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