Dietary Treatment of Idiopathic Hypercalciuria

L. R. I. BAKER and W. J. W. MALLINSON
Department of Nephrology, St Bartholomew's Hospital, London

Summary—Eighty-eight urinary tract stone formers (74 men) with idiopathic hypercalciuria were treated by dietary calcium restriction alone. Short-term control of hypercalciuria was achieved in only 27 patients and all but 12 eventually escaped control. Failure of control was twice as likely in patients with severe hypercalciuria.

Almost all patients lived in a hard water area. In such areas at least, attempts to control hypercalciuria by diet alone are likely to fail and early introduction of additional treatment is advisable. In most severe hypercalciurics, such treatment should be introduced from the start.

Idiopathic hypercalciuria is commonly associated with recurrent urinary tract stone formation (Flocks, 1939; Rose and Harrison, 1974), the hypercalciuria being widely believed to predispose to the development of stones. Understandably, therefore, attempts at prophylaxis have been aimed in the main at reducing urinary calcium excretion. Dietary calcium restriction and treatment with thiazides or sodium cellulose phosphate are most frequently employed.

Dietary calcium restriction has the merit of simplicity and freedom from drug-induced side effects. Since hypercalciuria is a consequence of increased gastrointestinal calcium absorption in most, if not all patients, it is also logical treatment. For these reasons, dietary restriction alone is often the first prophylactic measure to be introduced. Unfortunately, we find in clinical practice in a hard water area that dietary control of hypercalciuria is seldom achieved.

Patients and Methods
Two hundred and two urinary tract stone formers were referred to the Stone Clinic at St Bartholomew's Hospital between 1971 and 1975 for metabolic investigation. One hundred and thirty-four (66%) were found to have idiopathic hypercalciuria, defined as a mean of 2 or more urinary calcium measurements greater than 7.5 mmol/24 h in men and greater than 6.25 mmol/24 h in women. Hyperparathyroidism and other causes of hypercalciuria were excluded.

All urine measurements were made on outpatients taking a normal diet, urine being collected in vessels containing 15 ml of 6M HCl. Two consecutive collections were made and patients were given written instructions to increase the accuracy of collections. Almost all lived in East London and Essex, both of which are hard water areas (mean tap water calcium concentration in 14 patients randomly investigated 2.6 mmol/l, range 2.1 to 3.3 mmol/l). Urine calcium concentration was measured with a Perkin-Elmer 30 Atomic Absorption Spectrophotometer.

Of 134 patients with idiopathic hypercalciuria, 88 (74 male) were suitable for study. Twenty-five patients were treated with sodium cellulose phosphate or a thiazide ab initio. Seventeen patients failed to attend again after the diagnosis had been made and 4 declined treatment after discussion.

In 58 patients, daily dietary calcium intake was assessed on detailed questioning by a qualified dietician. Verbal and written instruction in a diet containing less than 10 mmol of calcium in food daily was given to all 88 patients. The exact degree of dietary calcium restriction was agreed by discussion between patient and dietician. Most patients agreed to take 7.5 to 8.75 mmol calcium daily. Patients were advised to maintain a high fluid intake. Dietary sodium and oxalate were not restricted.

After starting the diet, urinary calcium measurements were repeated within 1 to 3 months. Short-term control was defined as a fall in urinary calcium excretion (mean of two 24-h measurements) to less than 6.25 mmol/24 h in men and to less than 5.0 mmol/24 h in women. Urinary
calcium measurements were made every 2 to 6 months after this initial assessment and have been examined to determine the number of patients who later escaped control (vide infra). Duration of follow-up ranged from 2 months to 5 years.

Domestic water softeners (Permutit) were supplied to 9 patients who were not controlled by diet alone.

Statistical analysis of results was by student's t-test for paired or unpaired data as appropriate.

Results

Results are expressed throughout as mean ± standard deviation (Fig.). Of the 88 patients, 74 were men (mean age 42 years) and 14 women (mean age 50 years). Overall pre-treatment urinary calcium was 10.0 ± 2.3 mmol/24 h, being 10.2 ± 2.3 in men and 9.1 ± 1.7 in women. Overall urinary calcium on diet was 7.9 ± 2.8 mmol/24 h, being 8.2 ± 2.9 in men and 6.3 ± 2.0 in women. In both men and women, urine calcium excretion/24 h was significantly reduced on diet (P < 0.001). Twenty-three (30%) of the men and 4 (25%) of the women were controlled by diet. Pre-treatment urinary calcium in those patients who were controlled was significantly lower than in those remaining uncontrolled (9.1 ± 1.9 v 10.4 ± 2.3, P < 0.02). Of 51 men with urine calcium > 8.75 mmol/24 h, 10 were controlled on diet, in contrast to 13 of 23 men with urine calcium excretion below 8.75 mmol/24 h. This difference is significant (P < 0.005). Mean percentage fall in urine calcium was identical (20%) in patients with severe (> 8.75 mmol/24 h) and less severe (< 8.75 mmol/24 h) hypercalciuria.

Mean daily dietary calcium intake recorded in 58 patients was 36 mmol (49 men, 36.9 mmol; 9 women, 31.0 mmol). There was no significant difference in initial calcium intake between the controlled and uncontrolled groups.

On follow-up, all but 12 patients escaped from control, the mean of 2 or more measurements of 24-h calcium excretion exceeding 6.25 mmol in men and 5.0 mmol in women.

Escape from control typically occurred within 12 months of commencing the diet. The majority of "long-term controlled" patients have been followed for longer than 12 months.

In 9 patients, urinary calcium before a domestic water softener was used was 7.9 ± 1.95 mmol/24 h. Urinary calcium with the softener was 6.5 ± 1.8 mmol/24 h. This reduction did not achieve statistical significance (0.10 > P > 0.05). Three of the 9 patients were brought into the controlled range.

Mean 24-h urine volume measured on 200 occasions in 15 out-patients instructed to maintain a high fluid intake was 1860 ml. Given a mean tap water calcium of 2.6 mmol/l, water intake would account for a mean daily additional calcium intake of 4.8 mmol.

Discussion

No treatment is of proven value in reducing the increased risk of stone formation in idiopathic hypercalciuria since prospective, adequately controlled studies are lacking. Though falling short of formal proof, the results of treatment with thiazides are highly suggestive of benefit (Yendt et al., 1970); encouraging early results with sodium cellulose phosphate have also been reported (Pak et al., 1974). Both of these agents reduce urinary calcium excretion and it is reasonable to suppose (though in the case of thiazides, especially, open to question) that benefit, if any, derives from this action. It is thus reasonable current practice to attempt to correct hypercalciuria in recurrent stone formers.

Our definitions of hypercalciuria and adequate control are open to debate. We have taken the upper limit of normal urinary calcium to be that recommended by Hodgkinson and Pyrah (1958) and have attempted to reduce urinary calcium to well within their normal range. Some authors suggest that higher upper limits for normal urinary calcium excretion should be accepted (Rose and Harrison, 1974; Watson and Dale, 1966). The observations of Watson and Dale on out-patients in London are particularly convincing in this regard. It is self-evident that less stringent defi-
nitions of "hypercalciuria" and of "control" would exclude some of the patients we have studied from consideration and would also increase the percentage regarded as being satisfactorily treated. We regard our definitions as reasonable in the present state of knowledge. If this be accepted, only 12 out of 88 patients (14%) were controlled in the long term. If the 17 patients who failed to attend follow-up are considered to be treatment failures and are included in the assessment of results, only 11% of patients were controlled.

A major factor in failure of dietary treatment was non-compliance: out-patients on the diet typically admitted to breaking it or were found to be doing so on assessment by a dietician. Almost all patients complained that restriction of dairy products and normal bread was irksome. Motivation to adhere to the diet was often weak as most patients were symptom-free for most of the time.

A possible further reason for the relative failure of dietary treatment was the additional calcium load provided by drinking water in patients advised to maintain a high fluid intake. The provision of a water softener markedly reduced urine calcium in a few patients, but the majority remained uncontrolled. This additional measure seems worthy of trial in those whose main source of drinking water is domestic.

It is unlikely that even more rigid dietary calcium restriction than we have attempted would be acceptable to more than the occasional patient. Diets aiming to restrict calcium intake below 5 mmol/day—and therefore eliminating milk and normal bread entirely, as well as restricting some vegetables—are practicable only for the occasional dedicated individual such as the patient described by Dent and Watson (1965).

We conclude that mild hypercalciurics will frequently need additional treatment, such as a thiazide diuretic or sodium cellulose phosphate, and that severe hypercalciurics should receive such treatment from the start.

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References


The Authors

L. R. I. Baker, MD, FRCP, Consultant Physician.
W. J. W. Mallinson, MB, MRCP, Senior Registrar.

Requests for reprints to: W. J. W. Mallinson, Department of Nephrology, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE.