

## Editorial

### DIETARY FACTORS IN UROLITHIASIS

Complicated metabolic factors play a major role in the formation of urinary calculi (Smith et al., 1978). Fluid intake is a well established factor in both the treatment and aetiology of urinary stones. Adequate urinary flow has been known to benefit the patients with urolithiasis. Recurrent stone formation has been controlled by adequate diet adjustment and adequate fluid intake to maintain 2500 mls of urine output per 24 hours (Nordin et al., 1973). Such volume of urinary out-put can be maintained if the patient is able to take 250 to 300 mls of fluid per hour and at least 50 per cent of this fluid should be in the form of water.

Calcium as oxalate and phosphate is the major cation of urinary stone. 10 to 25% of calcium in diet is utilised under the influence of Vit. D and parathormone and the excess is excreted by the kidneys. In case of excessive intake of calcium intestines adjust and the calcium absorption is reduced. In idiopathic urinary calculi 40 to 60% patients show hypercalciuria (more than 300 mgm per 24 hours) which is related to increased calcium absorption by intestine (Peacock et al., 1968). Recent work indicates that there is increased production of dihydroxy vitamin D the active form of vitamin D (Kaplan et al., 1977) but the reason for this change is not known. Reduction in dietary calcium in these patients to 600 mgm per 24 hours seems to reduce the calcium excretion in urine (Smith et al., 1978).

Oxalate is an important anion of idiopathic urolithiasis. The normal excretion of oxalate in urine is less than 40 mgm per 24 hours. An increase in oxalate concentration may increase the calcium oxalate activity product resulting in crystallisation. Thus the food with high oxalate contents such as spinach, tea, cold drinks, citrus fruits and juices may produce oxaluria with increased tendency to stone formation. Calcium in diet combines with oxalate and makes it unavailable for absorption. Thus a diet low in calcium and high in oxalate will tend to cause oxaluria. In certain disorders of gastro-intestinal tract absorption of dietary oxalate is increased from the large intestine (Earnest et al., 1974) and in such situations dietary oxalate may be responsible for urolithiasis.

Phosphates are also commonly present as anions in urinary calculi. The excretion of phosphates directly depends upon its dietary intake. Low phosphate diet has been used to prevent urolithiasis with adverse effects (Thomas 1976). Phosphorus supplements have been found effective

in prevention of recurrent calcium urolithiasis when used in adequate dosage (Smith 1977).

Uric Acid lithiasis can be satisfactorily managed with increased fluid intake and uricosuric agents.

Reduction of urinary concentration of calcium oxalate and urate seems rational regardless of the cause of stone formation and thus high fluid intake is highly recommended. Dietary restriction on calcium intake is unnecessary except where more than one litre of milk is consumed. However, a number of patients with hypercalciuria might be highly sensitive to calcium and a restriction in dietary calcium may be considered. Hypercalciuria can be reduced by the use of thiazides and urate excretion can be minimized by allopurinol. The long term effects of these agents have been reported recently (Coe 1977). In hypercalciuric patients with recurrent stones the results were excellent with recurrences in only four patients out of 78 after thiazide administration. Similar results were obtained for urate stones and the use of allopurinol (Coe 1977).

The encouraging results however, will have to be studied critically and prospective controlled studies will be required to determine the efficacy of such regimes.

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