

RISK FACTORS FOR URINARY STONE FORMATION*

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Urinary tract stone remains a medical enigma. Stones may be considered as infective or metabolic and the main causes are shown in Table I. The reason for the enigma is that for calculi to form, several factors must operate and the process is multifactorial. In Western Europe and North America metabolic stones are frequent. They may afflict up to 3.8 % of the population in Scotland¹. The most common stones in the United Kingdom are metabolic stones and it is these that will be considered in this abstract. The major constituents are calcium and oxalate. The role of calcium has long been realised and 5% of patients with recurrent renal stones have been found to have hypercalciuria in association with an excess secretion of parathormone from a parathyroid adenoma. Other causes of hypercalciuria such as excess absorption from the intestine or renal leak account for a proportion of cases.

There remain a substantial proportion where there is no overt metabolic abnormality to account for calcium oxalate stone formation. Recent work from the Medical Research Council Mineral Metabolism Unit at Leeds has thrown some light on this problem². If the urinary concentration of calcium, oxalate, uric acid and glycoaminoglycosans is considered along with the urinary pH, a risk equation for stone formation can be calculated. Although each of these factors may be within normal limits the risk for stone formation may nevertheless be high because of interaction between these factors. Stone formation is more likely if there is a high concentration of calcium, oxalate or urate. Stone formation is less likely if there is a high concentration of glycoaminoglycosans (eg. compounds with chemical consistency similar to heparin which when present in the urine inhibit crystal nucleation and crystal aggregation). The urinary pH may be raised by ammonia and this will facilitate formation of magnesium ammonium phosphate stones found typically with urine infection. If the urine pH is low (acidic), uric acid and calcium oxalate stone formation are favored.

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The role of matrix in the formation of renal stones is not fully understood. All urinary tract stones have a matrix component which is composed of a complex of amino acids, carbohydrates and amino sugars. It is thought that disturbances in the renal tubular metabolism caused by hypercalcemia may lead to proximal tubular damage with a leak of these substances and that mineralization is a secondary event. A rarer variant is the pure matrix stone which is usually associated with urinary tract infection. Whether matrix formation is a primary or secondary event remains to be established.

The major contributory factor to stone formation in Western Europe would appear to be excess of nutrients in the diet and in particular protein². Prevention of renal stone may require a change in dietary habits as well as adequate hydration. Patients with recurrent calcium oxalate stones associated with increased risk because of calcium or oxalate may benefit from thiazide or cellulose phosphate therapy, and those with increased risk because of urate may benefit from allopurinol (i.e. in some cases allopurinol may reduce the risk of calcium oxalate stones). Adequate hydration is necessary to reduce concentrations of calcium oxalate and urate and indeed out of all the treatments for renal stone, despite our increasing understanding of metabolism, a high fluid intake remains the most effective treatment.

TABLE I
Causes of Stone Formation

Physiochemical	1. Excess crystalloids 2. Deficiency of inhibitors 3. Matrix
Anatomical	1. Impaired drainage 2. Calcification in renal papillae

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