Kidney Stones 1983: A Preventable Cause of Morbidity

Kidney stones are common. The most recent data available indicate that kidney stones accounted for 9.24 out of every 1,000 hospital discharges in Michigan in 1974 (1), an increase of 47% since 1952. Based on these data, kidney stones affected 13.4 out of every 10,000 persons in Michigan in 1974 (1). More than 80% affect people in the 20-60 age group, and within this age group men are affected more than women by a 2:1 ratio (2). It has been estimated that a patient who has the first stone has a 20% chance of a recurrence within five years. Patients who have had more than one stone have an 80% chance of a further recurrence within two years. Thus, aside from the health impact on an individual patient, kidney stones have a major economic impact on society, affecting otherwise healthy, productive members of the work force.

Although the first attack of acute renal colic cannot be predicted or prevented in most patients, data are accumulating to suggest that with correct diagnosis and management the first attack should also be the last for most patients. The purpose of multidisciplinary stone clinics in most major academic centers is to establish an etiologic basis for the stone occurrence and to plan a treatment program that would prevent recurrence. A detailed history of the patient is the first part of this evaluation. Particular emphasis must be placed on the dietary history with respect to consumption of dairy products (calcium), organ meats (purines), and green leafy vegetables (oxalates). The family history can provide important data about genetically inherited disorders associated with stone disease, such as absorptive hypercalciuria, gout and uric acid nephrolithiasis, primary hyperparathyroidism, or renal tubular acidosis. Less common inherited disorders, such as cystinuria, are usually well known to the patient at the time of presentation.

Knowledge of the patient's medication is an important source of information. Older patients who have glaucoma treated with acetazolamide (Diamox®) often neglect to include this information when they list their medications, but this carbonic anhydrase inhibitor affects urinary acidifications and reduces citrate excretion, thus promoting crystalization. A number of "stone formers" ingest large quantities of vitamin C, which is touted as a panacea for many ills. Some individuals, estimated to be less than 25%, metabolize ascorbic acid to oxalate. Patients with calcium oxalate nephrolithiasis who have marked increases in urine oxalate excretion should be questioned closely about vitamin C ingestion, either in tablet form or via fortified orange juice or other fruit drinks. Another potential source of hyperoxaluria as a cause for calcium oxalate stones is the increased intestinal absorption of oxalate in patients with inflammatory bowel disease or short-bowel syndrome. The physical chemistry of calcium and oxalate is such that small increments in urinary oxalate contribute disproportionately to the precipitability of calcium oxalate.

Although more than 90% of kidney stones contain calcium, only a small fraction of these are of the "triple-phosphate" (calcium, ammonium, and magnesium phosphate) type. This type of stones results from recurrent urinary tract infections, usually with urinary tract obstruction, and can be identified by a patient's history of recurrent urinary tract infection and by suitable imaging studies. Calcium oxalate (mono- or dihydrate) thus accounts for most stones in all large series reported. A metabolic abnormality—hypercalciuria, hyperuricosuria, or both—can be uncovered in 60-70% of stone patients. The diagnosis can be firmly established by three simple investigations.

Chemical analysis of the stone and its nidus, if present, should be performed on all stones, either those passed spontaneously or recovered during urologic intervention. Measuring serum calcium will uncover the 3-5% of all stone formers who have primary hyperparathyroidism, and elevated serum uric acid will uncover a similar percentage of patients who have stones on this basis. Analysis of a 24-hour urine specimen collected while the patient is following his or her usual diet (i.e., while the stone was being formed) will indicate the presence of hypercalciuria, hyperuricosuria, hyperoxaluria, or various combinations of all three. The urine volume and creatinine should be carefully recorded, not only to check the adequacy of the collection, but also because maintenance of a high fluid volume is essential for the long-term management of the stone patient. A low urine volume, less than 1500 ml in the first 24-hour specimen collected by the patient, indicates that the patient needs to drink more liquid. The serum chemistry and stone analysis can be ordered during the initial stone attack, often while the patient is still hospitalized, whereas the 24-hour urine collection should be postponed until the patient has recovered fully and has returned to his or her usual activities with respect to diet and fluid intake.
Kidney stones recur more often in patients with calcium nephrolithiasis who have hyperuricosuria alone or in combination with hypercalciuria than in those patients who do not. The process whereby uric acid acts as a "seed crystal" for calcium oxalate stones has been termed heterogeneous nucleation. The precise mechanisms are poorly understood, but the process can be effectively eliminated by administration of 50-300 mg of allopurinol daily. Of calcium stone formers, 25-35% exhibit this abnormality in uric acid excretion.

Idiopathic hypercalciuria (increased urine calcium excretion in the absence of hypercalcemia) is found in about half of all calcium oxalate stone formers. It may be of the absorptive type, in which the defect appears to be intestinal hyperabsorption of calcium from the diet, or it may reflect a renal leak of calcium independent of dietary calcium absorption. Measurement of urinary calcium in the postabsorptive state (12-hour fast overnight) will distinguish between these two types of idiopathic hypercalciuria. The distinction is important because the common advice to restrict dairy products given to all patients who have a calcium-containing kidney stone is not appropriate to those with renal leak hypercalciuria. In these patients, the body stores of calcium (predominantly the skeleton) are already taxed by the renal losses of calcium, and restriction of dietary calcium will only serve to aggravate this condition. Restricted dietary calcium intake may well be appropriate for absorptive hypercalciuria. Thiazide diuretics (e.g., hydrochlorothiazide 50-100 mg per day) will lower the urine calcium in most patients independent of the pretreatment urine calcium and of the type of hypercalciuria. Lowering the urine calcium has been clearly shown to limit the recurrence of kidney stones.

Measurement of urinary, undissociated uric acid concentration may define a group of patients who are prone to form calcium oxalate stones following seeding with uric acid crystals. In these patients urinary pH tends to be low. In about 16% of normocalciuric patients who form oxalate stones, the urine is oversaturated with calcium oxalate. Thiazide treatment appears to successfully reduce stone recurrence, although it may be of little value in patients without metabolic disorders.

The Stone Clinic at Henry Ford Hospital has only recently been established, but its need has already been justified by the rapid increase in referrals. Analysis of the causes of stone disease in patients seen during the first 12 months of the clinic’s existence indicates that a cause can be found for 65%. Many of these patients underwent their first stone evaluation in the clinic after many years of recurrent renal colic, during which no such evaluation had been performed. Our early experience confirms the results from other stone clinics about the preponderance of men and the proportion of patients with the various etiologies for recurrent stone disease. Our follow-up period is still too short to determine whether our stone clinic has been able to reduce the rate of recurrence of stones in our patients.

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References