Increasing prevalence of kidney stones in the United States

The recent trend to not restrict calcium intake in people afflicted with calcium kidney stones represents the culmination of several lines of investigation, including studies of urine chemistry, bone density and physiology, intestinal transport, and, lately, a well-performed randomized controlled trial [1]. A strong impetus to this development was the analyses of large epidemiologic databases that were too powerful to ignore [2]. In this issue of Kidney International, Stamatelou et al [3] have exploited another large database to yield information regarding stone disease of which nephrologists should be aware [3].

Using data from the National Health and Nutrition Examination Surveys (NHANES), Stamatelou et al demonstrated a 37% increase in the prevalence of kidney stones between two recent periods studied. The first period was 1976 to 1980 and the data were obtained from NHANES II, while the second period was 1988 to 1994, using data obtained from NHANES III. The lifetime prevalence of kidney stones increased from 3.2% ± 0.2% in the first period to 5.2% ± 0.3% in the second ($P < 0.001$). The increase occurred in both the total male and female populations. Although the absolute increases in prevalence were similar in men and women, so that the well-known male predominance in stone prevalence was preserved in all age groups, the relative increase in women was larger than in men. Prevalence increased for Caucasians, but not for African Americans. The power of these findings derives from the large number of participants surveyed: more than 15,000 adults in the first period, and more than 16,000 in the second, all between 20 and 74 years of age. The importance of the findings derives from the lack of previous data showing this trend in the United States, although comparable epidemiologic data showed a similar trend in Italy.

The limitations of the study are candidly described by the authors and include the self-reporting of the diagnosis of stone disease by the participants, without confirmation by a physician or medical record. It seems fair to assume that the accuracy of self-reporting of stones is more accurate than reporting of, for example, "heart attacks." A more serious and unquantifiable problem is the surveillance bias introduced by the two study periods, corresponding to a time when enormous technologic advances led to more widespread utilization of radiologic imaging in the United States. There can be little doubt that more stones, and more asymptomatic stones, would be detected by the more liberal and more accurate application of ultrasound and computed tomographic (CT) scanning, a problem that the NHANES databases cannot resolve. Perhaps this bias is reflected in the fact that when the two genders were divided into age groups by decades, instead of broader ranges, two of the only three groups demonstrating statistically significant increases in prevalence over time were the 70- to 74-year-old males and females. Arguably, these were the patients undergoing the most ultrasound and CT examinations in the early 1990s. Despite this limitation, the external validity of the study is enhanced by the confirmation of previous findings demonstrating a relatively low prevalence of stones in African Americans and Hispanics compared to Caucasians, and the relatively higher prevalence rate in the Southern United States.

Assuming that the authors are correct, and the prevalence of stones is truly increasing, this study constitutes evidence of a change in powerful environmental, rather than genetic, factors promoting stone disease, although the study cannot conclude what these factors might be. The multivariate logistic regression analysis, which included intake of various dietary constituents as possible independent variables, does not appear to have been used to test whether the increase in prevalence with time was associated with increased (or decreased) intake of any of these putative risk factors for stones, and the authors suggest that the data available would not have had sufficient explanatory power. Stone composition is not known; one would assume that the increase is attributable to calcium oxalate stones. The lack of concomitant urinary analytes in some subset of surveyed patients also means that specific causes for the observed increase cannot be pinned down. Several possible explanations are suggested by trends noted lately in the habits of our society.

A recent study, using American databases containing data obtained in 1977 from the National Food Consumption Survey and in 1989 and 1996 from the Continuing Survey of Food Intake for Individuals, sponsored by the United States Department of Agriculture, concluded that food portion sizes and energy intake for a number of food types have increased markedly over a nearly 20-year period, similar to that investigated by Nielsen and Pookin.
Portion sizes increased both in and out of the home; the increase occurred for salty snacks, hamburgers and, in fact, all of the key foods surveyed except pizza. Salt intake is associated with increases in urinary calcium excretion and increased protein intake has myriad effects on urinary chemistry that promote lithogenicity as well. Portion size is conceivably a correlate of stone risk.

If it were not for the calories, it might be good news for stone formers that soft drink serving sizes have increased in these surveys by 52%. Increases in salt intake have recently been shown, as one would expect, to be associated with increases in urine volume (about 350 mL of urine more per additional 100 mmol of sodium excreted) that might mitigate the effects of sodium-induced calciuria on urinary supersaturation [5]. On the other hand, although epidemiologic studies have not demonstrated an association of more soda intake with more stones, one randomized trial of soft drink restriction showed a significant decrease in stone incidence [6]. The trend to “supersize” American soft drink portion sizes will, therefore, not necessarily overcome the “supersized” food servings when it comes to stone risk.

The increased prevalence of kidney stones also parallels the well-publicized increase in the nation’s prevalence of obesity from 14.5% in 1971 to 30.9% in 1999 [7]. Besides perhaps representing an increased intake of protein and salt, obesity itself may be a risk factor for stone disease, particularly in women, as suggested by a correlation demonstrated between stone prevalence and both weight and body mass index [8]. The reason for this association might be that oxalate excretion too correlates with body size [9]. One recent response to increasing obesity has been the popularity of the high protein, high fat Atkins diet, which promotes weight loss and stone formation. Notwithstanding its current faddish status, the more widespread utilization of this diet is relatively recent and not responsible for the trend demonstrated by Stamatelou et al over the period they studied.

Decreases, rather than increases, in intake of calcium could also contribute to an increased prevalence of stones. Higher dietary calcium intake is associated with a lower prevalence of kidney stones in both men and women [2]. This may occur as calcium in the intestinal lumen binds oxalate and diminishes its absorption [9]. It is, therefore, of some interest that dietary calcium intake over the period examined has declined [10].

Nephrologists have become used to hearing of the coming obesity-related epidemics of diabetes and diabetic nephropathy and the expected ensuing influx of patients with end-stage renal disease (ESRD). Possibly these spates of E SRD and stones are related, with some etiologies in common. Stone disease is not associated today with the morbidity and mortality of ESRD, of course, but as a disease that often strikes a younger and healthier population its cost to the economy is substantial. The prevention of stones can be successful while being cost-effective and satisfying. Nephrologists have been called to halt the progression of chronic kidney disease and now should increase their commitment to preventing stone disease as well.

David S. Goldfarb
New York, New York

Correspondence to David S. Goldfarb, M.D., Director, Kidney Stone Prevention and Treatment Programs, New York VA Medical Center, and St. Vincent’s Hospital, and Associate Professor of Medicine, New York University School of Medicine, Nephrology Section/111G New York DVAMC, 423 E. 23 St., New York, N.Y. 10010.
E-mail: david.goldfarb@med.va.gov

REFERENCES