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Kidney stones epidemiology

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GUIDELINES

No recommendations possible based on Level I or II evidence

SUGGESTIONS FOR CLINICAL CARE

(Suggestions are based on Level III and IV evidence)
Not applicable for this topic.

BACKGROUND

Kidney stone disease varies in frequency and stone type between different climates and racial groups. Understanding the epidemiology of stones disease is important to determine the significance of the disease at a community level, the associations and risk factors for individuals and the likelihood of stone recurrence. This section attempts to describe the epidemiology of kidney stone disease including its association and risk factors. The majority of evidence is from overseas studies however, Australian data is included for comparison where this exists.

No levels of evidence exist for epidemiology making this section largely descriptive. This section also does not contain guidelines or suggestions for clinical care. As with the remainder of this guideline, only the epidemiology of stone disease in adults is covered.

SEARCH STRATEGY

Databases searched: Medline (1966 to July Week 3, 2004). MeSH terms and text words for kidney stones were combined with MeSH terms and text words for Australia. The Australian Medical Index, Aboriginal and Torres Strait Islander Health and rural databases were also searched.
Websites searched: Australian Institute of Health and Welfare, Australian Kidney Foundation and AusStats (Bureau of Statistics).
Date of search: 23 July 2004.

WHAT IS THE EVIDENCE?

No randomized controlled trials (RCTs) are available which address this issue.

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Incidence and prevalence

There are no population-based data on the incidence or prevalence of kidney stones in Australia. Overseas studies from other Western countries show that kidney stones are uncommon before the age of 20 years, their incidence rises between the ages of 20 and 30 years and then remains relatively constant until the age of 70 years, after which the incidence falls again. Estimates of the incidence of first kidney stones between the ages of 30 and 70 years vary between approximately 100–300/100 000/year in men and 50–100/100 000/year in women.[1–4] Overall, the prevalence of kidney stones is approximately 6–9% in men and 3–4% in women and this appears to be increasing.[2–7]

Age and sex differences

Men are at greatest risk of developing kidney stones with incidence and prevalence rates between two and four times that of women.[2,3]

A 15 year retrospective study by Baker *et al.*[8] was performed at the Institute of Medical and Veterinary Science in Adelaide, which performs the majority of stone analysis in South Australia. This study found that 70% of all stones analysed were from men. Men were at greater risk of producing calcium oxalate stones (73% were in men) and uric acid stones (79% were in men). Women were at greater risk of infection stones (58% occur in women). In a separate study by Gault and Chafe in Canada, women were also found to be more likely to produce calcium phosphate stones than men.[9]

Baker *et al.*[8] found that the peak age for the development of calcium oxalate stones was between 50 and 60 years. Uric acid stones tended to occur in an older population with an average age of 60–65 years. Infection stones, however, occurred in younger people, most commonly in women between the ages of 20 and 55 years. A second peak is seen, particularly in men, between 55 and 70 years of age.

A study performed by Lavan *et al.*[10] reported the characteristics of 619 consecutive patients attending a stone clinic at a Sydney hospital. In this study, 64% of patients were male and 69% of patients first developed symptoms of kidney stones between the ages of 20 and 49 years.

Racial differences

Several groups have reported racial differences in the risk of developing kidney stones. Soucie *et al.*[6] in a large cross-sectional survey in the United States found that the prevalence of kidney stones was highest among White people and lowest in Black people. Hispanic and Asian people had an intermediate prevalence. Others have reported similar findings.[2] One study of the metabolic risk factors for stone formation in different ethnic groups in the United States demonstrated that white stone formers had a higher incidence of hypercalcuria.[11] Other urinary metabolic abnormalities were, however, evenly distributed between the ethnic groups studied.

A report of a small study performed in the radiology department of Alice Springs Hospital indicated that while kidney stones appeared to be less common in Aboriginal adults than in the White population, the majority of the stones (6/11) that did occur were staghorn calculi.[12] These stones were all in women, usually related to urinary tract infection and were presumably mostly infection or triple phosphate stones.

Several reports, however, indicate that the rate of urinary stones is increased in Aboriginal children living in remote and arid areas. These are most commonly urate stones, usually in the upper urinary tract and often associated with urinary infection but are not covered further in this section.[12–15]

Stone type

The relative frequency of various stone types is similar in Australia to other industrialized countries. Baker *et al.*[8] found that calcium oxalate stones (with or without phosphate) were the most frequent (68% of stones). The remainders include uric acid 17%, infection stones 12% and pure calcium phosphate stones 3%.

By comparison, in a large prospective cohort study performed in the United States, the frequencies of different stone types in men were calcium 71.5%, uric acid 23.1%, struvite 5% and cystine 0.5% (44% of stones were available for analysis).[16] In women, the composition of stones (45% were available for analysis) were calcium 86.2%, uric acid 11.3%, struvite 1.3% and cystine 1.3%.[1]

Seasonal variation

Baker *et al.*[8] found that uric acid stones increased significantly during summer and autumn (by 69% ($P < 0.001$) and 62% ($P < 0.01$), respectively) and that infection stones decreased during spring and summer (by 27% ($P < 0.05$) and 37% ($P < 0.01$), respectively). The frequency of other stone types did not vary significantly throughout the year. It was postulated that the increase in uric acid stones was due to the fall in urine volume during summer combined with a reduction in urinary pH. In support of this, a recent review of over 28 000 24 h urine test results in the United States demonstrated that urinary volumes fall significantly in men, but not in women, during summer. This was associated with a fall in urinary pH and an increase in the supersaturation of both uric acid and calcium oxalate.[17]

A 4 year retrospective review of stones seen during intravenous pyelography (IVP) in a district hospital in Perth was published in 1973.[18] In 1204 IVP studies, 188 small calculi were identified in the kidneys or ureters. Of these 132 were 'free' stones (in the renal pelvis or ureter) while 56 were 'fixed' stones (overlying the papillae or calyces). The indication for IVP was renal colic in almost all patients found to have free stones. Free stones were found most frequently between December and March, which were the hottest 4 months of the year. Overall 46% of stones were found on examinations performed during these months indicating that stone formation was more frequent during this time.

A prospective study of 45 289 men in the United States failed to find an increased incidence of kidney stones over a 6 year period in the hotter south-eastern states; however, the prevalence of stones at baseline was higher in these states.[19] This indicates that there was a modest increase in the risk of kidney stones in the hotter regions. Other studies performed in the United States and Israel have indicated that the incidence of stone disease increases both in the hotter areas of a country and during the hottest months of the year.[20,21]

Underlying causes

The frequency of the identification of the cause of kidney stones varies widely between studies and is often heavily influenced by the patient population being studied. For instance, a population-based study might find a lower incidence of hyperparathyroidism than that reported by a specialist stone clinic. The extent of investigations performed and the definitions used will also affect the reported incidences.

Medical conditions that are associated with an increased risk of kidney stone formation include hyperparathyroidism,[22] hyperthyroidism, sarcoidosis, gout,[23] malabsorption (inflammatory bowel disease, ileal resection or bypass),[24] cystic fibrosis[25] and immobilization.

Renal abnormalities such as medullary sponge kidney, distal renal tubular acidosis and anatomical abnormalities that predispose to urinary stasis (including polycystic kidney disease) also increase the risk of stone disease.

People with spinal cord injuries have increased urinary catheterization and infection, combined with urinary stasis and immobilization and are at high risk of stones. These occur most frequently in the first months after the injury and are usually infection stones.[26,27]

In the large US population-based cohort study, an underlying contributing factor was found in 18.4% of men presenting with their first kidney stone (inflammatory bowel disease 6.7%, hyperthyroidism 1.2%, hyperparathyroidism 0.6%, urinary tract infection 8.5% and prolonged immobilization 1.4%).[16] In a study of women, 25.8% had a potentially contributing medical condition including urinary tract infection at the time of stone formation or stone due to urinary infection (17.3%), hyperthyroidism (3.4%), inflam-

matory bowel disease (3%) and hyperparathyroidism (2.1%).[1]

Lavan *et al.*[10] found histologically proven primary hyperparathyroidism in 5% of women and 2% of men attending a Sydney stone clinic, with several more having the diagnosis suspected.

Metabolic abnormalities

People who form kidney stones often have metabolic or other abnormalities detectable on urinary testing. The common abnormalities include low urinary volume, hypercalcuria (25–40%), hyperoxaluria (10–50%), hyperuricosuria (8–30%) and hypocitraturia (5–30%).[28–32] There is, however, significant overlap with healthy controls who also often have biochemical 'abnormalities', albeit less frequently.[29,32]

OTHER ASSOCIATIONS AND RISK FACTORS

Diet

The association between diet and kidney stones has long been suspected. Prospective studies show that high dietary calcium intake reduces the risk of kidney stones, possibly by reducing gut absorption of oxalate.[1,16,33] Calcium supplements, however, might be associated with an increased risk of stone disease in older women although this has not been found in younger women.[1,34]

High fluid intake is associated with a lower risk of developing kidney stones in men and women.[35,36] Certain beverages also appear to provide additional protection with coffee, tea, beer and wine consumption associated with reduced risk of kidney stones while grapefruit juice consumption was associated with an increased risk.[35–37] A high dietary intake of magnesium and potassium has also been associated with a lower risk of stone development in men in a large observational study.[38]

Obesity

Lavan *et al.* found that male patients with idiopathic hypercalcuria or uric acid stones were significantly heavier than other male stone formers (weights 78.5 kg, 83.2 kg and 73.5 kg, respectively).[10]

A recent prospective study combining three large cohorts in the United States found that weight, body mass index and waist circumference were positively associated with the risk of kidney stones.[39] The relative risk of developing kidney stones was greater for obese women than it was for men.

Insulin resistance has also recently been reported to be associated with low urinary ammonium and pH and increased risk of uric acid nephrolithiasis.[40–42]

Hypertension

A modest association has been reported between hypertension and nephrolithiasis in both sexes.[5,43–45] In prospective studies, people with a history of nephrolithiasis are more likely to develop hypertension[5,44] and those with hypertension are more likely to develop kidney stones, especially when they are overweight.[46,47]

Family history

A large US cohort study found that men who had developed a kidney stone were three times more likely to have a family history of kidney stones than other men.[48] A family history also increased the likelihood of developing a stone in men who had never had one previously.

A family history of kidney stones was also obtained more frequently from patients with kidney stones than from controls in the study by Lavan *et al.* (16.8% of men and 22.7% of women with kidney stones *vs* 6% of controls, $P < 0.01$).[10]

Medications

Indinavir, a protease inhibitor used in the treatment of HIV-1 infection, can cause kidney stones composed mainly of the drug itself.[49] Other medications associated with stones include allopurinol, triamterene and trisilicate (silica stones).[50]

Vitamin C is metabolized to oxalate and has been shown in a small metabolic study to increase the urinary excretion of oxalate.[51] No association between vitamin C intake and the incidence of kidney stones has been demonstrated in women.[52] However, in an observational study, high vitamin C intake (>1000 mg/day) has been shown to be associated with an increased risk of stones in men, when compared with an intake of 90 mg/day.[38]

Other factors

Other factors reported to be associated with an increased risk of developing kidney stones include the absence of intestinal oxalate degrading bacteria.[53,54]

STONE RECURRENCE

Prospective studies have shown stone recurrence rates of between 26% and 53% after 10 years.[31,55,56] This estimate is lower than previous retrospective studies that reported stone recurrence rates of 40–75%.[3,57–61]

Different groups have reported various factors to be associated with an increased risk of stone recurrence. These have included presenting with a first stone at a younger age, family history, stone number and gouty arthritis; however, the results of many of these studies are conflicting.[31,62–64]

Previous extracorporeal shock wave lithotripsy has also been found to be a risk factor in some studies[63,65] but not in others.[66]

It has been found by several groups that metabolic abnormalities found on standard laboratory tests in calcium stone formers (e.g. hypercalcuria, hyperoxaluria) are usually not helpful in predicting stone recurrence.[31,67]

SUMMARY OF THE EVIDENCE

There are no RCTs on this topic.

Although data on kidney stone disease in Australia are limited, the available studies suggest that the epidemiology is similar to other Western countries. Of particular interest in Australia is the likelihood that climate and racial background play a role in the risk of kidney stone disease and the identification of subgroups within the community at high risk of the disease.

WHAT DO THE OTHER GUIDELINES SAY?

Kidney Disease Outcomes Quality Initiative: No recommendation.
UK Renal Association: No recommendation.
Canadian Society of Nephrology: No recommendation.
European Best Practice Guidelines: No recommendation.

INTERNATIONAL GUIDELINES

No recommendation.

IMPLEMENTATION AND AUDIT

No recommendation.

SUGGESTIONS FOR FUTURE RESEARCH

Further study of the incidence and risk factors for stone formation in Australia, particularly in indigenous Australians.

CONFLICT OF INTEREST

Peter Hughes has no relevant financial affiliations that would cause a conflict of interest according to the conflict of interest statement set down by CARI.

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