Kidney Stones

Nutritional considerations

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The incidence of kidney stones (also referred to as nephrolithiasis) is increasing in the UK and in other western countries. The prevalence increases with age and 16% of men and 7% women will have at least one stone by the age of 70 years old.[1] People of all ages suffer from kidney stones, but the peak age for the first stone is around 45 years old.[1] Risk factors for stone formation include genetic predisposition, some medications, certain medical conditions, infections, dietary and fluid intake, and environmental factors. Fluid intake is one of the most important modifiable risk factors. Nephrolithiasis is associated with a two-fold increase in risk of chronic kidney disease (CKD).[2]

This article aims to provide a general introduction to kidney stones and their classification, including nutritional considerations in the dietetic assessment of the individual affected by kidney stones. Furthermore, there is a focus on the dietary management of patients affected by secondary hyperoxaluria and uric acid kidney stones with gastrointestinal (GI) disorders. The dietary management of idiopathic hyperoxaluria and cystine stones are outside the scope of this article.

Definition & classification

Kidney and ureteric stones are hard pieces of material that form in the kidney when high levels of certain minerals are present in the urine. Kidney stones are painful and people who have had them want to prevent them from reoccurring.[3] The main pathway for kidney stone formation is urinary supersaturation of the stone component, allowing crystals to form and grow in the urinary tract and become stones. Low excretion of inhibitors of crystallization (such as citrate) may also contribuite to stone formation. Kidney stone formation falls under several categories:

- Idiopathic kidney stones, which represent the majority of kidney stones. These do not have a specific disease linked to their formation. They are referred to as non-infection related kidney stones, such as calcium oxalate, calcium phosphate and uric acid kidney stones
- Genetic causes, such as cystinuria, primary hyperoxaluria
- Acquired diseases, such as granulomatous disease, primary hyperparathyroidism, distal renal tubular acidosis
- Infection-related kidney stones, such as magnesium ammonium phosphate, ammonium urate kidney stones
- Kidney stones secondary to inflammatory bowel disease, short bowel syndrome, chronic diarrhoea/ileostomy formation and/or linked to enteric hyperoxaluria (mostly calcium oxalate or uric acid kidney stones)
- Other causes, e.g. drug-related, unknown cause.

There are over 25 chemical substances that can form kidney stones. However, the types of kidney stones are often summarised in 5 main categories – see **Table 1**.

General nutritional principles

Dietary and fluid intake have a clear role in the management of patients with kidney stones.[1] Calcium homeostasis is influenced by dietary sodium and animal protein, which may contribute to the formation of calcium stones. Timing and dosage of dietary calcium in patients at risk of calcium stones are important to reduce calcium stone formation.[4] Therefore, dietary advice plays a significant role in calcium stones. On the other hand, struvite kidney stones are associated with urinary tract infections, especially in women, and the formation of this kidney stone is caused by the action of bacteria. As a result, these types of kidney stones need medical or surgical treatment rather than direct dietetic intervention.[3, 4] Cysteine stones can occur in patients with cystinuria. These patients do respond well to a high fluid intake and a reduction of animal protein, which may favour a lower pH in the urine and may amend kidney stone formation.[4]

From a nutritional point of view, knowing the type of kidney stone, the likely cause of its origin, along with medical, drug and diet histories are of extreme importance before dietary advice can be tailored to the individual.[1, 3, 4]

Nutritional factors may play a role in the development of kidney stones. High salt intake, high protein intake and high sucrose intake are considered promoters of calcium containing kidney stones. On the other hand, high potassium, high magnesium, high fibre and high vitamin B6 intakes are considered as dietary inhibitors of calcium containing kidney stones.[3]

Increasing fluid intake is a strong important modifiable factor to reduce the risk of kidney stone formation, especially their reformation.[1, 3] According to the European Association of Urology (EAU)[1] and the National Institute for Health and Care Excellence (NICE) guidelines,[3] in addition to high fluid intake, a diet more consistent with Dietary Approaches to Stop Hypertension (DASH) – high intake of fruit and vegetables, nuts, legumes, low fat dairy, wholegrain, and a low intake of sodium, sweetened beverages, red and processed meat – decreases the incidence of kidney stones.[1, 3, 4] General dietary guidelines have been proposed and summarised in **Table 2**.

Drug history

Some medications may induce kidney stones. Kidney stones may form by the crystallised compound from the drug or due to unfavourable changes in the urine composition linked to a particular drug therapy. **Table 3** summarises drugs associated with kidney stones formation. Over the counter preparations, especially if used in excess, can contribute to kidney stones formation. For example, vitamin C taken at more than 1000 mg/day can increase the risk of calcium oxalate kidney stones due to the metabolism of ascorbic acid into oxalate.[4, 5]

Table 1: Types of kidney stones

Type of stones	Incidence	Comments
Calcium stones	Most common 70-85%	Calcium oxalate are by far the most common compared to calcium phosphate (65-70% and 15-20%, respectively). Calcium oxalate stones may present with a small component of calcium phosphate. Pure calcium phosphate stones are less common (less than 17% and more prevalent in female above 50 years old).
Uric acid stones	8-10%	More common after the age of 50 years old.
Struvite stones	Rare	Stones which occur as a result of an infection.
Cystine stone	Rare	Stones which are linked to an inborn error of cystine metabolism.
Stone of other causes	5%	Stones may be formed due to specific drug therapies, surgical procedures (some bariatric surgery) and those with unknown origin.

Table 2: Dietary and fluid principles to prevent kidney stone in adults[1, 3, 4]

Diet and fluid intake principle to prevent kidney stone
• Drink 2.5-3 litres of water per day (avoid sugary drinks)
• Adding fresh lemon juice to drinking water
• Avoiding carbonated drinks
• Limit daily salt intake to no more than 5-6 g/day
• Not restricting dietary calcium intake, but maintaining a normal dietary calcium intake of 700-1,200 mg per day
• Moderate protein intake 0.8-1.0 g/kg/body weight, with a preference for vegetable protein
• Avoid high doses of ascorbic acid (over 1000 mg a day) from over-the-counter supplements

Table 3: Medications associated with the formation of kidney stones (list not exhaustive)[1, 4, 5]

Drugs with active compounds that may crystallise in the urine	Drugs that may affect urine pH and/or its composition
Allopurinol	Allopurinol
Amoxicillin	Ascorbic acid (excessive doses)
HIV protease inhibitor	Vitamin D (excessive doses)
Quinolones	Calcium supplements (if they are not taken with meals)
Triamterene	Furosemide
Magnesium trisilicate	Laxative
Sulfonamide	Orlistat
	Losartan
	Topiramate
	Sodium-glucose cotransporter 2 inhibitor

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Diet history

When conducting a diet history with patients with kidney stones this should, at the very minimum, cover the following key nutritional points:

Fluid intake

A diet history should focus on fluid intake both in the form of volume (quantity over 24 hours) and quality (type of fluid drunk). Increasing fluid intake is the simple most efficient, well-tolerated, and safe advice that patient with kidney stones should follow.[1, 3] Fluid intake of 2 litres or more has been shown to reduce the 5-year rate of stone recurrence by more than half.[1] The EAU suggests drinking 2.5-3.0 litres a day of neutral pH beverages (aiming for a urine output of more than 2.0 litre per day). People who drink more not only have a lower super concentration, but a stone is more likely to be washed away. The ingestion of caffeine containing drinks (coffee, tea) has some controversial views. While a moderate consumption (4 cups of coffee a day) has been associated with a lower incidence of kidney stones,[6] the use of tea is more controversial because the content of oxalate in black tea and green tea can vary significantly.[7] The use of soft drinks, especially those acidified by phosphoric acid and sweetened soda has been linked to increased risk of the development of kidney stones and should be avoided.[7]

Citrus food & drinks

Foods that are rich in citrate are citrus. There is a wide variety of citrus fruits and derivative products available, such as lemonade, and citrus-based juices and preserves. A systematic review meta-analysis, in 2017, showed that citrus-based products can increase urinary citrate and urine pH significantly compared to control treatments.[8] On the other hand, a narrative review, in 2021, demonstrated that the regular consumption of grapefruit juice was associated with an increased risk of stone formation.[9] The use of fruit juice is also contradictory because of their high content of sugar, ascorbic acid and oxalate.[7]

Dietary calcium

Low dietary calcium has been associated with increasing the risk of calcium stones.[7] Calcium absorption, calcium protein binding and renal handling affect calcium in the urine. Calcium reabsorption from the nephron is affected by sodium (where sodium excess will increase calcium secretion). Limiting dietary calcium will reduce urinary calcium but will not reduce the risk of recurrent kidney stones.[10] Even patients with calcium oxalate kidney stones are encouraged to achieve a dietary calcium intake of 1000-1200 mg/day.[10, 11] Increasing calcium intake leads to an increase in the binding of oxalate in the gastrointestinal tract, limiting absorption of oxalate. The supplementation of calcium (using calcium supplements rather than high calcium diet) is controversial as an increased incidence of kidney stones has been observed in patients using regular calcium supplements,[12] together with an increased risk of cardiovascular events.[13] The current EAU guidelines support the dietary supplementation with vitamin D, together with dietary calcium from food (up to 1200 mg/day) rather than using calcium supplements (such as calcium carbonate), alongside a fluid intake of 2-3 litres a day. If calcium supplements are needed, the EAU suggests the use of calcium citrate rather than calcium carbonate and that they should be taken with, or shortly after, a meal to bind oxalate in the GI tract and reduce its absorption (not on an empty stomach).

Salt intake

A high salt diet promotes kidney stone formation because it increases calcium excretion in the urine. Furthermore, evidence from a randomised controlled trial suggests that a low salt diet is effective in reducing calcium excretion in patients with idiopathic kidney stones, and its effectiveness should not be underestimated.[7, 14]

Protein intake & fruit and vegetables

A diet high in animal protein can lead to an increase in overall dietary acid load, which may increase urinary calcium and reduce urine pH and citrate excretion. However, the evidence supporting this statement is limited and controversial.[7] While the evidence *per se* is limited, large observational studies found that a higher dietary net acid load was associated with a higher risk of stone formation.[7] This suggests that the amount of fruit and vegetables consumed, rather than total protein, could be an indicator for the risk of kidney stone formation.[7] Fruit and vegetables have an alkalizing potential and can *'neutralize the proton load metabolically generated from ingested protein'*.[7] Current guidelines suggest aiming for a protein intake of 0.8-1.0 g/kg of body weight (in people with a healthy body mass index [BMI]), with an emphasis on vegetable protein, limiting animal protein and encouraging a high intake of fruit and vegetables.[1, 3, 7]

Phytate intake

Phytates are mostly found in beans, grains, nuts and seeds. It is thought that phytates inhibit kidney stone formation by complexing with calcium and preventing crystallisation; and their intake should be increased in the diet, when possible.[7]

Gastrointestinal disorders & kidney stones

Patients with inflammatory bowel disease (IBD), short bowel syndrome, ileostomy or chronic diarrhoea can be at higher risk of developing kidney stones, and it is estimated that between 9-18% of patients with some GI disorder may develop kidney stones.[4, 15] Some have an increased absorption of oxalate and can develop calcium oxalate kidney stones (such as patients with short bowel syndrome), others have an increased risk of uric acid stones formation (such as patients with ileostomy).

Patients can also develop hypocitraturia because of the bicarbonate losses via the GI tract and/or hypomagnesuria due to the GI losses of magnesium, often combined with the chronic use of proton pump inhibitors. The decreased absorption of water, potassium, magnesium, citrate and bicarbonate may result in urinary supersaturation. Patients may present with low urinary pH, low urinary calcium, and low urinary volume.[4, 15]

Calcium oxalate stones

Enteric hyperoxaluria occurs in patients suffering from fat malabsorption, such as short bowel disease, Crohn's disease, chronic pancreatitis, orlistat treatment and disorders associated with bile acid malabsorption.[16] Calcium normally binds in the gut with oxalate and prevents its absorption. Unabsorbed fatty acids bind to calcium in the gut, which consequently reduce the calcium available to bind to oxalate. As a result, an increased concentration of free oxalate occurs in the colon, where more oxalate is absorbed, leading to an increased risk of calcium oxalate formation.[15] Increased dietary calcium has been shown to decrease hyperoxaluria after ileal resection.[1, 4, 15] The dietary management of secondary hyperoxaluria may also include a reduction of high oxalate food, increase in dietary calcium intake (and/or calcium supplements) with the use of cholestyramine, if appropriate.[1, 15]

Although the EAU guidelines[1] recommend limiting high oxalate food in secondary hyperoxaluria, limiting dietary oxalate is difficult in dietetic practice. The first step should be to provide adequate dietary calcium at every meal (300-400 mg).[4] If urinary oxalate is still elevated despite the increased provision of calcium in the diet, some patients may benefit from reducing oxalate content. However, it is the opinion of the author, and others,[4] that limiting dietary oxalate is not easy. This is because oxalate concentration in food is influenced by many factors. These include different lab techniques for food analysis, how ripe the food was when it was picked, the soil composition, cooking method and food processing. **Table 4** shows a short list of some of the main foods considered to have an elevated oxalate content.

Uric acid stone

Low urine pH is the most common cause of uric acid stones. Measuring urine pH is probably more important than the actual concentration of uric acid in the urine.[4, 15, 17] Patients at risk of uric acid stones are those with a high ileostomy output with significant losses of bicarbonate via their stoma. In addition, chronic loss of water and salt via the ileostomy can lead to a reduction of urinary volume.[7] The main treatment for uric acid kidney stones is to make the urine less acidic. Dietary management focuses on replacing alkali – for example, the use of sodium bicarbonate with the aim of urine alkalization by reaching a urinary pH of 6.5-7, together with appropriate fluid intake, by slowing down ileostomy output by maximising pharmacological management and consideration of rehydration solution.[4, 15]

Conclusion

The recent publication of the EAU guidelines,[1] as well as recently updated evidence,[4, 15] provide detailed information on the dietary management of people affected by kidneys stones, which should equip health professionals with enough information to tailor advice to individuals affected by kidney stones. Diet and fluid intake clearly play a key role in people affected by kidney stones and effective dietary advice has been shown to reduce their reformation, which can contribute to reduced hospitalisation and improvement in patients' quality of life.

Table 4: Food with moderate and very high content of oxalate*[4, 16]

Very high oxalate food	Moderate oxalate food
Nuts, peanut butter	Sweet potato
Rhubarb, currants, lemon and orange peel, starfruit	Chocolate/Ovaltine®/cocoa
Beetroot	Bran
Chard, spinach, kale	Marmalade
Tofu	Okra
Sesame seed	Tea

*List not exhaustive

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