Stone Diseases

Ureteric Stones

Presentation

Ureteric stones usually present with sudden onset of severe flank pain which is colicky (waves of increasing severity are followed by a reduction in severity, but it seldom goes away completely). It may radiate to the groin as the stone passes into the lower ureter. May be presented with only scrotal pain. Site and side of stone determine the differential diagnosis.

Examination

Spend a few seconds looking at the patient. Ureteric stone pain is colicky—the patient moves around, trying to find a comfortable position. They may be doubled-up with pain. Patients with conditions causing peritonitis (e.g. appendicitis, a ruptured ectopic pregnancy) lie very still: movement and abdominal palpation are very painful.

- **Pregnancy test**

Arrange a pregnancy test in pre-menopausal women (this is mandatory in any pre-menopausal woman who is going to undergo imaging using ionizing radiation).

- **Dipstick or microscopic hematuria**

Many patients with ureteric stones have dipstick or microscopic hematuria (and, more rarely, macroscopic hematuria), but 10-30% have no blood in their urine. And may be a coincidental finding because of non-stone urological disease (e.g. neoplasm, infection) or a false +ve test

- **Temperature**

The most important aspect of examination in a patient with a ureteric stone confirmed on imaging is to measure their temperature. If the patient has a stone and a fever, they may have infection proximal to the stone. A fever in the presence of an obstructing stone is an indication for urine and blood culture, intravenous fluids and antibiotics, and nephrostomy drainage if the fever does not resolve within a matter of hours.

- **diagnostic radiological imaging**

The intravenous urogram (IVU), for many years the mainstay of imaging in patients with flank pain, has been replaced by CT urography (CTU) Unenhanced CT_ scan.

Compared with IVU, CTU:

1. • Has greater specificity (95%) and sensitivity (97%) for diagnosing ureteric stones. it can identify other, non-stone causes of flank pain.
2. • Requires no contrast administration so avoiding the chance of a contrast reaction.
3. • Is faster, taking just a few minutes to image the kidneys and ureters.
4. • Is equivalent in cost to IVU

MR urography

This a very accurate way of determining whether a stone is present in the ureter or not.[4] However, at the present time, cost and restricted availability limit its usefulness as a routine diagnostic method of imaging in cases of acute flank pain. This may change as MR scanners become more widely available.

Managment

- **Acute management**

While appropriate imaging studies are being organized, pain relief should be given.

- ❖ A non-steroidal anti-inflammatory (e.g. diclofenac) by intramuscular or intravenous injection, by mouth or per rectum. Provides rapid and effective pain control. Analgesic effect—partly anti-inflammatory, partly by reducing ureteric peristalsis.
- ❖ Where NSAIDS are inadequate, *opiate* analgesics such as pethidine or morphine are added
- ❖ Calcium channel antagonists (e.g. nifedipine) may reduce the pain of ureteric colic by reducing the frequency of ureteric contractions.'
- ❖ Desmopresine (Antidiuretic hormone) recently used to relief pain.

There is no need to encourage the patient neither to drink copious amounts of fluids nor to give them large volumes of fluids intravenously in the hope that this will 'flush' the stone out. Renal blood flow and urine output from the affected kidney falls during an episode of acute, partial obstruction due to a stone. Excess urine output will tend to cause a greater degree of hydronephrosis in the affected kidney which will make ureteric peristalsis even less efficient than it already is.

- **Watchful waiting**

Small ureteric stones will pass spontaneously within days or a few weeks, with analgesic supplements for exacerbations of pain. Chances of spontaneous stone passage depend principally on stone size. Between 90-98% of stones measuring <4mm will pass spontaneously. Average time for spontaneous stone passage for stones 4—6mm in diameter is 3 weeks. Stones that have not passed in 2 months are unlikely to do so. Therefore, accurate determination of stone size (on plain abdominal X-ray or by CTU) helps predict chances of spontaneous stone passage. Nifedipine and Tamsulosin (an alpha adrenergic adrenoceptor blocking drug) may assist spontaneous stone passage and reduce frequency of ureteric colic.

Indications for early intervention to relieve obstruction and/or remove the stone:

1. Pain which fails to respond to analgesics or recurs and cannot be controlled with additional pain relief.
2. Fever in obstructed kidney is one of Urological emergencies.
3. Impaired renal function (solitary kidney obstructed by a stone, bilateral ureteric stones, or pre-existing renal impairment which gets worse as a consequence of a ureteric stone).
4. Prolonged unrelieved obstruction. This can result in long-term loss of renal function. How long it takes for this loss of renal function to occur is uncertain, but generally speaking the period of watchful waiting for spontaneous stone passage tends to be limited to 4-6 weeks., though large stones do sometimes drop out of the ureter at the last moment
5. Social reasons. Young, active patients, Airline pilots and some other professions are unable to work until they are stone free.

- **Emergency treatment of the stone**

Temporary relief of the obstruction can be obtained by insertion of a double J stent or percutaneous nephrostomy tube (PCN) (Percutaneous nephrostomy tube can restore efficient peristalsis by restoring the ability of the ureteric wall to coapt.). JJ stent insertion or percutaneous nephrostomy tube can be done quickly, but the stone is still present. It may pass down and out of the ureter with a stent or nephrostomy *in situ*, but in many instances it simply sits where it is and subsequent definitive treatment is still required. While JJ stents can relieve stone pain, they can cause bothersome irritative bladder symptoms (pain in the bladder, frequency, and urgency). JJ stents do make subsequent stone treatment in the form of ureteroscopy technically easier by causing passive dilatation of the ureter.

- **Definitive treatment (Removing the stone)**

Treatment options for ureteric stones

• ESWL: *in situ;* after 'push-back' into the kidney (i.e. into the renal pelvis or calyces); or after JJ stent insertion
• Ureteroscopy and Intracorporeal lithotripsy
• PCNL
• Open ureterolithotomy
• Laparoscopic ureterolithotomy

Basketing of stones (blind or under radiographic 'control') are historical treatments (the potential for serious ureteric injury is significant).

The ureter can be divided into two halves (proximal and distal to the iliac vessels) or in thirds (upper third from the PUJ to the upper edge of the sacrum; middle third from the upper to the lower edge of the sacrum; lower third from the lower edge of the sacrum to the VUJ).

Recommendations

Proximal ureteric stones

• <1cm diameter: ESWL *(in situ,* push-back)
• >1cm diameter: ESWL, ureteroscopy, PCNL

JJ stent insertion does not increase stone free rates and is therefore not required in 'routine' cases. It is indicated for pain relief, relief of obstruction, and in those with solitary kidneys.

Distal ureteric stones

• Both ESWL and ureteroscopy are acceptable options.

• Stone free rate <1cm: 80-90% for both ESWL and ureteroscopy; >1cm: 75% for both ESWL and ureteroscopy.

Bladder stones

Composition Struvite (i.e. they are infection stones) or uric acid (in non-infected urine).

Adults Bladder calculi are predominantly a disease of men aged >50 and with bladder outlet obstruction due to BPH. They also occur in the chronically catheterized patient (e.g. spinal cord injury patients), where the chance of developing a bladder stone is 25% over 5 years.

Children Bladder stones are still common in Thailand, Indonesia, North Africa, the Middle East, and Burma. In these endemic areas they are usually composed of a combination of ammonium urate and calcium oxalate. A low-phosphate diet in these areas (a diet of breast milk and polished rice or millet) results in high peaks of ammonia excretion in the urine.

Symptoms May be symptomless (incidental finding on KUB X-ray or bladder ultrasound or on cystoscopy)—the common presentation in patient with spinal injury who have limited or no bladder sensation). In the neurologically intact patient—suprapubic or perineal pain, haematuria, urgency and/or urge incontinence, recurrent UTI, LUTS (hesitancy, poor flow).

Diagnosis If you suspect a bladder stone, they will be visible on KUB X-ray or renal ultrasound .

Treatment Most stones are small enough to be removed cystoscopically (endo-scopic cystolitholapaxy), using stone-fragmenting forceps for stones that can be engaged by the jaws of the forceps and EHL or pneumatic lith-otripsy for those that cannot. Large stones can be removed by open surgery (open cystolitholapaxy).

Evaluation *of the* stone former

Determination of stone type and a metabolic evaluation allows identification of the factors that led to stone formation, so advice can be given to prevent future stone formation.

Metabolic evaluation depends, to an extent, on the stone type. In many cases a stone is retrieved. Stone type is analysed by polarising microscopy, X-ray diffraction, and infrared spectroscopy, rather than by chemical analysis. Where no stone is retrieved, its nature must be inferred from its radiological appearance (e.g. a completely radiolucent stone is likely to be composed of uric acid) or from more detailed metabolic evaluation.

In most patients, multiple factors are involved in the genesis of kidney stones and, as a general guide, the following evaluation is appropriate in most patients.

- **Risk factors for stone disease**

• *Diet.* Enquire about volume of fluid intake, meat consumption (causes hypercalciuria, high uric acid levels, low urine pH, low urinary citrate), multivitamins (vitamin D increases intestinal calcium absorption), high doses of vitamin C (ascorbic acid causes hyperoxaluria).

•*Drugs.* Corticosteroids (increase enteric absorption of calcium, leading to hypercalciuria); chemotherapeutic agents (breakdown products of malignant cells leads to hyperuricaemia). Triamitren and Anti HIV druge.

• Urinary *tract infection.* Urease-producing bacteria (Proteus, Klebsiella, Serratia, Enterobacter) predispose to struvite stones.

• *Mobility.* Low activity levels predispose to bone demineralization and hypercalciuria.

• Systemic *disease.* Gout, primary hyperparathyroidism, sarcoidosis.

• *Family history.* Cystinuria, RTA.

• *Renal anatomy.* PUJO, horseshoe kidney, medullary sponge kidney.

• *Previous bowel resection or inflammatory bowel disease:* Causes intestinal hyperoxaluria.

Metabolic evaluation of the stone former

Patients can be categorized as low risk and high risk for subsequent stone formation.

High risk: previous history of a stone, family history of stones, GlT disease, gout, chronic UTI, nephrocalcinosis.

Low-risk patient evaluation:

Urea and electrolytes, CBP (to detect undiagnosed haematological malignancy), serum calcium (corrected for serum albumin), and uric acid, urine culture, urine dipstick for pH.

High-risk patient evaluation:

As for low-risk patients plus 24-h urine for calcium, oxalate, uric acid, cystine; evaluation for RTA.

Urine *pH*: Urine pH in normal individuals shows variation, from pH 5-7. After a meal, pH is initially acid because of acid production from metabolism of purines (nucleic acids in, for example, meat). This is followed by an 'alkaline tide', pH rising to >6.5. Urine pH can help establish what type of stone the patient may have (if a stone is not available for analysis), and can help the urologist and patient in determining whether preventative measures are likely to be effective or not.

• PH <6 in a patient with radiolucent stones suggests the presence of uric acid stones.

• PH consistently >5.5 suggests type 1 (distal) RTA (70% of such patients will form calcium phosphate stones).

Evaluation for RTA Evaluate for RTA if: calcium phosphate stones, bilateral stones, nephro-calcinosis, MSK, hypocitraturia.

• If fasting morning urine pH (i.e. first urine of the day) is >5.5, the patient has complete distal RTA.

• First and second morning urine pH is a useful screening test for detection of incomplete distal RTA, over 90% of cases of RTA having a pH >6 on both specimens. The ammonium chloride loading test involves an oral dose of ammonium chloride (0.1g per kg; an acid load). If serum pH falls <7.3 or serum bicarbonate falls <16mmol/l, but urine pH remains >5.5, the patient has incomplete distal RTA.

Prevention of calcium oxalate stone formation

- **Fluid intake**

Low fluid intake may be the single most important risk factor for recurrent stone formation. High fluid intake is protective, by reducing urinary saturation of calcium, oxalate, and urate. Time to recurrent stone formation is prolonged from 2 to 3 years in previous stone formers randomized to high fluid vs. low fluid intake (averaging about 2.5 vs. 1L/day) and over 5 years, risk of recurrent stones was 27% in low-volume controls compared with 12% in high-volume patients.

- **Dietary calcium**

Conventional teaching was that high calcium intake increases the risk of calcium oxalate stone disease. The Harvard Medical School studies have shown that low calcium intake is, paradoxically, associated with an increased risk of forming kidney stones, in both men and women.

- **Calcium supplements**

It is possible that consuming calcium supplements with a meal or with oxalate-containing foods could reduce the risk of inducing kidney stones.

- **Other dietary risk factors related to stone formation**

Increased risk of stone formation

• Sucrose

• Sodium : high sodium intake (leading to natriuresis) causes hypercalciuria

• Potassium I

- **Animal proteins**

High intake of animal proteins causes increased urinary excretion of calcium, reduced pH, high urinary uric acid, and reduced urinary citrate,}all of which predispose to stone formation.

- **Vegetarian Diet :** protect against the risk of stone formation
- **Dietary Oxalate:** A small increase in urinary oxalate concentration increases calcium oxalate supersaturation much more than does an increase in urinary calcium concentration.