MICHELLE JO SEMINS: Thank you for joining. Today, we'll get started by talking about the epidemiology of kidney stones. We'll then move on to the acute stone episode, followed by management of that acute stone episode. And we'll wrap up with followup and prevention.

So there's been a precipitous rise in kidney stone disease over the last several decades. The National Health and Nutrition Examination Survey estimated the prevalence of self-reported symptomatic kidney stones in US adults rose significantly, from 3.2% in the late '70s to 5.2% in the late '80s, the early '90s, to 8.8% by 2010. Now, in addition to symptomatic self-reported kidney stones, there are also incidental asymptomatic kidney stones that are identified in approximately 5% of patients that undergo imaging for a different indication.

And when these patients are followed with serial imaging, up to 32% of them will develop symptoms, or undergo procedure, within three to four years. Now, after a symptomatic stone event, the five year recurrence rate is up to 53% for patients that do not undergo medical therapy, specifically. And this comes at a direct cost, not only to the patient, but to the health care system with an annual direct cost in the US of about $10 billion.

Now, historically, the classic stone patient is in their fourth to sixth decade of life, is male, Caucasian, lives in a warm area, sweats for a living. And often, the stones happen at higher temperature seasons, like summer. But this is historically. The gap has closed in gender, age, race, region, and now, just about everybody can get a stone.

Women are almost as frequently getting them as men. And there's an increase of five-fold over the last decade in pediatric patients with about a 10% annual increase. These increases are attributed to change in diet-- higher animal protein, higher salt, more sedentary lifestyles, increases in obesity, and climate changes.

In fact, temperature rise worldwide from global warming could lead to an increase of up to 2.2 million lifetime cases of kidney stones by 2050. Now, it's important when we're talking about kidney stones that we're on the same page with regards to communication. So when we refer to calyceal stone, it's tucked away in the kidney. It's not obstructing. It doesn't tend to have hydronephrosis associated with it unless it's focal, and it's not typically the reason for people's pain.

This is a proximal ureteral stone, a mid ureteral stone, and a distal ureteral stone. This is a mid ureteral stone with severe hydronephrosis. This is a renal pelvic stone. It's in the kidney like the calyceal stone, but it's centrally located. It's definitely responsible for symptoms, and it's also worrisome for impending obstructing-- obstruction if there's not yet hydronephrosis.

Here we have a staghorn calyceal. It takes up the entire kidney, filling all the calyces. We also may refer to partial staghorn calculus, and that's where it's still a large stone and it's branching into calyces, but typically only one or two calyces.

Now, the classic signs and symptoms of somebody presenting with a kidney stone are flank pain, lower abdominal pain, blood in the urine, and nausea and vomiting. There are patients that present with atypical signs and symptoms, and there should always be a high alert. But these are the classic signs and symptoms.

Patients can have non-colicky pain from stretching of the renal capsule. But that classic colicky pain comes from stretching of the collecting system and the ureter, not from the stone itself, but from obstruction from the stone with backflow of urine behind it. And this pain tends to wax and wane.

It waxes and wanes as it crosses three physiological narrowings-- the ureteropelvic junction, the crossing of the iliac vessels, and the ureterovesical junction. These are areas where the stone can also get hung up and become impassable. And each of these physiologic narrowings have a distinct set of symptoms.

Ureteropelvic stones have costovertebral angle pain. It tends to radiate around the flank and toward the abdomen. Mid ureteral stones where they cross the iliacs tend to have more discomfort in the lower quadrant with pain radiating to the suprapubic or scrotal region.

And distal ureteral stones have more pain associated with bladder, vulva, and scrotal discomfort. And patients will often complain of lower urinary tract symptoms that actually mimic infection frequency-- with frequency and urgency, and sometimes even dysuria. Now, these distinct set of symptoms help us to clinically detect stone location as the stone continues to travel through the system.

So it's important when we're gathering history of present illness to get information regarding pain, location, quality, onset, duration, any associated symptoms that can help potentially confirm the diagnosis of kidney stones. It's also important that we gather information about whether the patient's had fevers, if they're able to tolerate food or liquid, or even oral medications. And all of this information helps factor into the disposition plan for the patient.

It's important at this point to still keep in mind the differential diagnosis is quite wide. Gastrointestinal could be cause for these symptoms, as well, gynecologic, musculoskeletal, or even other urologic causes, such as torsion or GU infections. I personally like to get information about patient's own history, if they've ever had one before, if they've passed them on their own, if they've been required to undergo surgery for them, if they've failed the trial's passage.

I also like to know comorbidities that not only predispose people to stones, but also may change their management approach, as well. And a good GU history is important, again, because it not only increases risk for stones, but it can change the management approach, as well. Patients will have a family history of kidney stones 25% of the time.

And anybody who has a positive of the above comorbidities that predispose to stones, GU history, past stones, or family history are at a high risk for recurrence and definitely require some form of urologic followup. If there's time, just in case the patient's not going to come back, I try to know what they do because there are occupations that have higher risk for stone formation. Activity level, spinal cord patients, sedentary patients, they're going to have higher risk for stones, as well.

And I try to get just a quick dietary history, because I may be able to provide some counseling on the spot. I always look at the medications. Again, the patient may not come back, and there are a lot of culprits for increased risk of stones. And if they're not going to come back for urologic followup, I at least encourage them to discuss this with the prescribing physician.

In terms of physical exam for these patients, vital signs are the number one important thing. I want to know if they've had a fever, tachycardia, tachypnea. These-- in the setting of a presumed infection, I'm worried for sepsis. And if the blood pressure is also lower, if the patient's unresponsive, they could be in septic shock.

Obstructive pyelonephritis is infection involving the kidney when a stone is blocking the outflow of urine. And this is extremely important that an obstructing stone in the setting of infection is a urologic emergency and potentially lethal if not addressed emergently. In addition to those vital signs, it's important to do a full abdominal exam and a complete GU exam to rule out that broad differential diagnosis.

Previously, I have seen patients with torsion who were thought to have potentially a stone, and they went on to needing an orchiectomy. It's also important to get urinalysis. Urinalysis gives a ton of information.

It can tell you if there's suspicion for infection. Red blood cells can confirm that something neurologic is going on. There may be crystals, like uric acid or calcium oxalate crystals present, which helps guide management therapy, as well.

And if pH is low in the setting of uric acid crystals, then we may medically manage those patients. Specific gravity can tell me about patient's hydration status. I always get a urine culture, even if the urinalysis is not suspicious for infection.

So I never send a reflex. I always send a urine culture by itself, because of those patients who may want to be managed conservatively failed that management and come back a day or two later, I like to have even a negative culture in the system so that I can proceed for definitive management if I want to, and not just have to temporarily drain them until a culture is back.

I also like basic metabolic panel, not just to check the creatinine, but to check electrolytes, to check for acidosis. CBC can tell me if there's a leukocytosis and if there's higher suspicion for inflammation or infection. I always like to get a diff on that, too, because it'll tell me about a left shift.

Non-contrast CT scan is the gold standard for diagnosing for kidney stones. It visualizes the stone nearly 100% of the time, greater than 99%, including radiolucent stones. Indinavir is the only stone that is consistently not visible, which isn't very common on CT or X-ray.

The CT scan can also provide a ton of other information-- whether there's hydronephrosis present, if there's hydroureter present, if there's any stranding or inflammation around the kidney. If the kidney's enlarged, it tells me about hounsfield units of the stone itself, which can help guide therapy because it tells me about fragility. And all of this information allows for good surgical planning.

Here we have an intrarenal stone. It's a large stone, but this is a calyceal stone without any associated hydronephrosis. Given its size, we would probably recommend elective management of that.

This is a proximal ureteral stone on CT scan. This is the inferior pull of the kidney. So it's right as the stone dropped out of the kidney. And you can see that there's some hydroureter associated with it around that stone, and likely more proximal, as well.

This is the distal ureteral stone. This is the bladder here. The stone often is confused by the radiologist as probably a bladder stone. But I see this a lot of the time.

If a patient's having persistent symptoms, the stones lateralize into the side the symptoms are on. And there's an area of lucency around the stone. It tells me that it's actually in the tunnel, the ureterovesical junction, and not quite in the bladder yet.

Now, when we're talking about CT as the gold standard, it's important to keep ALARA in mind as low as reasonably achievable. The trend in CT use has increased exponentially over the past 30 to 40 year period. International Commission on Radiologic Protection has occupational exposure recommendations of less than 50 millisieverts per year, or 100 millisieverts over a five year period. And this is for workers.

There's no recommended limit for patient medical exposure, but it is agreed upon that unnecessary exposure should be avoided and necessary exposure should be justifiable and optimized. So how much radiation is our stone population getting? Is it worrisome?

Evaluation and management for a single stone event has been shown to be a wide range-- one to 38 millisieverts. There was another study that looked at an acute stone episode and one year of followup. They found immediate effective radiation dose in these patients of about 30 millisieverts.

20% of those patients underwent 3 and 1/2 CT scans in that one year period, which totaled over 50 millisieverts, which is more than that recommended guideline. And then there was a longer term study specifically looking at CT scans done within a five year period of an initial stone visit, and they found a good proportion of patients underwent enough CT scans to also total more than that recommended allowance-- all very concerning.

So when we're thinking about diagnosis, of course, there's still going to be times when we have to get a CT scan for surgical planning, specifically. And when we do have to get a CT scan, there are alternative methods that include low dose options. So there's a low dose CT and an ultra low dose CT.

Both have high sensitivity and specificity for stone diagnosis, and both are low effective dose compared to standard CT scans in terms of radiation doses. A standard CT can be even as high as 20. Often is around 10. But this is significantly lower.

Now, while this technology exists, utilization is very poor. There was a study looking at utilization about 10 years ago. 2% of all CT scans that are done for kidney stones. This improved about five years later to 7.6%. Obviously, still a very small number and definitely a target for change in the future.

Now, while we're talking about the low dose technology, it's important to recognize there are limitations that include decreased sensitivity and specificity for smaller stones less than 3 millimeters. They tend to be less accurate for overweight and obese patients, and less diagnostic for non-urologic pathology. So if the diagnosis is not highly suspicious, then it may not be the appropriate test to order.

Now, the typical practice when the patient presents to the emergency room with renal colic is to order a CT scan. But there was a group a few years ago that published in the *New England Journal of Medicine* challenging this typical practice. And they randomized patients to ultrasound versus CTs groups-- CT group as the initial presentation of renal colic as the initial study.

And they found that even though 40% of the patients in the ultrasound group went on to need a CT scan in that same ER visit, the ultrasound group still had lower cumulative radiation exposure than the CT group with no difference in complications. And so they now recommend ultrasound as that initial test of choice in the emergency room when patients present with renal colic. And European guidelines also echo that.

Ultrasound can identify hydronephrosis. It can also identify kidney stones within the kidney with a shadow behind it, a hyperechoic area and a shadow behind it. Hyperechoic area, shadow behind it, but it tends to be poor for diagnosis of ureteral stones.

With that said, ultrasound is still first line for pregnant patients presenting with renal colic and pediatric patients, as well. We also have plane radiography. KUB are films of the kidney, ureter, bladder.

While it's a good potential study to follow patients with a known stone to see if that's growing, and if there's more stone formation, it's challenging in the primary diagnosis of the stone because quality can be affected by phleboliths that are around, overlying stool and bowel gas. And only radiopaque stones are identified, so there's a wide variety of stones that may be missed if KUB is used alone.

When a patient's in the emergency room, acute management consists of IV fluids, as these patients often present dehydrated-- also, pain control. And nonsteroidal's our first line if they're not contraindicated. Second line or other pain medications that are not narcotics. And then third line, essentially, are narcotics.

Anti-emetics are often needed because patients do have nausea and sometimes vomiting with the renal colic. And antibiotics should be used if an infection is suspected after the culture has been sent off. There are indications for admission and/or acute intervention.

Any signs of infection, the patient needs probable intervention. Anybody is not making any urine. This may be because they have an obstructed solitary kidney, or bilateral obstruction. Anybody with renal insufficiency will likely need intervention.

And then patients who have uncontrolled pain, unable to tolerate food, liquid, or oral medications, or those that are more potentially fragile immunosuppression, multiple comorbidities will need admission with observation and possible intervention. When we're talking about acute intervention, we're most commonly talking about temporary drainage with a stent or nephrostomy tube. Definitive management is typically scheduled electively.

There are some times where we will electively intervene and treat the stone definitively in the acute setting, if it's a small stone and they don't have any signs of infection or renal insufficiency. Then we may proceed for definitive management. But most typically, we're draining them and scheduling them at a later date to manage that stone.

Urgent decompression is required for anybody with fever or signs of systemic infection, anybody with hemodynamic instability, anybody who's not making any urine, or anybody in renal failure. And most importantly, an obstructing stone, as mentioned before, with associated infection, can lead to life-threatening sepsis. Not only if the kidney is not emergently drained, but also if surgery on the stone is attempted rather than just emergently draining them.

So any manipulation with high pressure irrigation in the system will increase the risk for life-threatening sepsis, as well. And I have to explain that to patients often as to why I'm not actually treating the stone in the setting. So there is literature on this showing that surgical decompression is associated with decreased mortality in patients with sepsis and ureteral calculi.

It took a while for something to come out, but finally, somebody did put it out. And lack of decompression essentially doubled mortality compared to those who were decompressed to almost 20% in these patients. Along the same lines, delayed decompression of obstructing stones with urinary tract infection is also associated with an increased odds of death by nearly 30%. So not only is it important to drain these patients, but it's important to drain them quickly.

Obstructive pyelonephritis treatment consists of prompt decompression of the kidney via either a stent or a nephrostomy tube. Broad spectrum antibiotics, it's important to obtain a culture hopefully beforehand of the urine. Not only of bladder [INAUDIBLE] but also of kidney urine at the time of decompression, and also blood cultures. And many of these patients may require intensive care.

In terms of decompression options, we can place a ureteral stent done typically by the urologists using fluoroscopic guidance in the operating room, or the interventional radiologist can place a nephrostomy tube percutaneously via ultrasound, typically, in the interventional radiology suite. How do we decide what type of drainage to use? Stone location and size plays a role.

Any patient with anatomical abnormalities might guide you one way or another. What resources are available? Surgeon and patient preference. Some patients may have had a terrible experience with a stent and elect for a nephrostomy tube instead. And there was a prospective randomized trial done years ago showing no differences in outcome. What's important is drainage.

Trial of passage is appropriate for patients with a stone less than 10 millimeters in size who has unilateral obstruction when they have two functional kidneys if their pain and nausea is manageable with oral medications, if they have no evidence for renal insufficiency and no signs and symptoms of infection. Trial of passage consists of aggressive oral hydration. There's limited data whether this actually helps to pass the stone, but it's still important because these patients otherwise can become dehydrated.

And it's also a risk for kidney stone formation and growth, but not an acute period. Analgesia is also important. Nonsteroidals, again, are first line. Non-narcotics are also-- non-narcotic pain medications, like Tylenol, are important. And last line would be narcotics, but we try to limit these.

I will also alkalinize the patient if uric acid is suspected. So if I'm looking at that urinalysis and they have crystals in their urine, low pH, if it's radiolucent on that scout image, or if they even have a history of uric acid stones, then I'll give them medication to try to dissolve that stone medically. And it's important to strain the urine.

I tell patients to go home with a strainer, not only to be able to send that stone for analysis because it helps play a role for prevention, but also because many patients have resolution of their symptoms without passage and that stone will then represent at some point in time to the emergency room and potentially grow and maybe even cause stricture in the ureter. Trial of passage is often used with medical expulsive therapy. Medical expulsive therapy can be done with alpha blockers. And there's also literature with calcium channel blockers.

There are numerous small studies and meta analysis that show medical expulsive therapy increases likelihood of passage and decreases time to passage. It's a bit controversial in the urology literature, but there's enough evidence that most urologists would elect to take this medication as it has a low side effect profile. Results have been strongest for alpha-blockers, stones in the 5 to 10 millimeter range, and stones in the distal ureter.

Elective stone removal is scheduled for patients who fail trial of passage, and we'll typically give them four to six weeks to pass their stones. We'll reimage, either with KUB or pelvic CT, at four weeks. And if they have significant progression of the stone, then we'll give them an additional couple of weeks, as long as everything's under control to try to pass it on their own.

If a stone is unlikely to pass, so if it's greater than 10 millimeters at the time of presentation, we'll schedule them electively. Some patients may decide to not do trial of passage, either because they had past experience that was not great, they've never passed a stone on their own, or for social reasons, if they have a wedding coming up, or a vacation coming up, or for their occupation, like they're a pilot.

The incidental asymptomatic non-obstructing stone is another time that we will sometimes do elective stone removal. To treat or not to treat those stones has the multiple layers of shared decision-making and is beyond the scope of this presentation. But I have lots of talks on how to make those decisions, if anybody is interested at any point in time in discussing that further.

Surgical options, once elective stone removal is decided upon, are shockwave lithotripsy, ureteroscopy with laser lithotripsy, percutaneous nephrolithotomy. Those are the classic surgical options and the most commonly used. Laparoscopic robotic management may be appropriate at times.

For example, if a patient is undergoing a pyeloplasty for a UPJ obstruction and they have a stone at that time, it can be removed using that approach. Open surgery is really a thing of the past in this country. It's still used in other countries where the technology is not as sophisticated as ours. But I've never seen an anatrophic nephrolithotomy. Pyelolithotomy, again, would be an indication if you're there for another reason.

Shockwave lithotripsy was developed in the 1970s and introduced in 1980. This is the original machine where a patient is put to sleep under general anesthesia onto this pulley system, moved along and lowered into a bathtub. Shockwaves are then generated by this treatment head outside of the body, focused onto a point in the body, the stone, using X-ray guidance. And then those shockwaves are propagated through the body onto the stone, causing fragmentation, and thus, passage of the stone through the urinary tract on its own.

The newer model machines that are less cumbersome, more mobile, and a bit easier to use. And shockwave lithotripsy essentially revolutionized the management of upper urinary tract calculi because it was non-invasive, low morbidity, and outpatient, when historically any upper urinary tract calculus had to be treated with an open surgical procedure requiring a week of hospitalization and recovery afterwards.

Success depends on patient and stone specific features. Obese patients with a long skin to stone distance greater than 10 centimeters have a higher failure rate because the shockwave energy will dissipate as it travels through the adipose tissue. Solitary stones are preferred, less than two centimeters. Fragile stone, so Hounsfield units less than 1,000 break more easily.

And stones within the renal pelvis, upper pole, or proximal ureter where upon fragmentation they're more likely to fall gravity wise out of the urinary tract, rather than if they're sitting in the lower pole where they would have to move up and out. Complications of this procedure are low. Blood in the urine, flank pain, infection are the most common.

It's unusual to get a hematoma or renal injury. Patients may have obstruction. This tends to occur more when larger stone burden is treated. It's called Steinstrasse, or street of stones. Injury to surrounding organs is very rare but can happen.

Ureteroscopy entails the use of a flexible ureteroscope, as seen here, or a rigid scope, as seen in the upper right panel. This allows for direct visualization and manipulation of the stone. So either a basket is used to extract the stone if it's small enough, or a laser may be required to break up the stone and subsequently pull out those pieces.

A ureteral stent is placed after this procedure, which is bothersome to some patients. And for that reason, and because of entrance into the body, it's considered more invasive than shockwave lithotripsy. Ureteroscopy's typically reserved for stones less than two centimeters, although our technology is becoming excellent and we really are able to push the limit sometimes on stones 2 to 3 centimeters.

And it's a viable option for patients that can't tolerate a more invasive procedure with larger stone burden. It has greater than 90% success rate because you're visualizing the stone directly in terms of successful fragmentation and extraction. Complications are also low-- blood in the urine and infection.

Patients will have stent discomfort, potentially, some more than others. And there's a variety of medications you can use to control that stent discomfort. And it's temporary. Typically, the stent will be removed within 3 to 10 days after surgery.

5% of patients have access failure, meaning you cannot get the scope into the patient's system because of their anatomy, and then require pre-stent placement for about a week to passively dilate the system and a second stage of the procedure, and then subsequently, are successful. Ureteral injury and stricture are very rare-- 1% or less.

Percutaneous nephrolithotomy entails passage of the scope through the back directly, as seen here. It also allows for direct visualization and manipulation of the stone. And it's the only modality to employ suction, because you're able to use a larger channel going directly through the kidney.

The ureter is small so the scopes are small. The caliber of the channel in the ureteroscope clogs, and there's no suction at this point in time that we can use to evacuate stone fragments. So you can evacuate larger burden in a quicker amount of time. So this is reserved for stones greater than 2 centimeters and staghorn calculi.

It's more invasive than shockwave lithotripsy and ureteroscopy because it involves an incision and going through the kidney directly. Contraindications would be anybody with a coagulopathy, or somebody who can't go off blood thinners. And anybody with a poor respiratory status, because typically, this procedure is done in the prone position. There are some urologists that do it in the supine position, but it's more common in the prone position.

Complications are higher for this procedure, upwards of 25%, which is why it's not first line. But major complications are rare. More common complications would be bleeding transfusion risk around 1%, urinary tract infection.

If you enter high on the kidney, as seen in the top left panel there, you're going to be closer to the lung and there's going to be a higher chance of things like pneumothorax or hydrothorax. Organ injury does happen, perforation of the kidney does happen, but those are extremely rare. Pseudoaneurysm occurs in approximately 0.5% of patients, and it requires embolisation to treat that.

There are some special circumstances. Patients who have a non-functional kidneys who have a stone that needs to be treated, nephrectomy should be considered, rather than treatment of stone in most circumstances. Pregnant patients can undergo ureteroscopy safely, but shockwave lithotripsy is contraindicated in pregnancy and percutaneous nephrolithotomy is generally discouraged during pregnancy.

There are other anatomical abnormalities that may guide you to different management options, as well. For these patients who undergo elective stone management, short-term I follow them with imaging at four to six weeks. I typically will get a KUB X-ray to get a baseline to then follow them thereafter. Because there may be a parenchymal calcification, or plaque, that's not a treatable stone that I want to get that baseline to see.

And I'll get an ultrasound to make sure that there's resolution of the hydronephrosis that was present, or that there's no new hydronephrosis from ureteral stricture. Some patients who have silent hydronephrosis, and if it's not detected on this imaging because it's not done, they can present later with loss of kidney function. Ultrasound itself is not a great size estimate for stones. So I typically am getting it simply to see if there's hydronephrosis or not.

Long-term, the goal is prevention of growth and new stone formation. And this is done with medical treatment. So you can provide generic recommendations to the patient. You can do a basic assessment. And then I'll typically offer somebody a complete metabolic evaluation, as well.

And serial imaging is important. I'll typically do serial imaging every six months in the first year. And if that patient has lowered their risk significantly, then I'll back off to annually.

Now, some surgeons may exclusively offer surgical management when a kidney stone becomes symptomatic. But that's akin to me of treating heart disease after a patient has a heart attack. 95% of patients will have a predisposing abnormality present. And we have literature that shows identifying and correcting those abnormalities results in decreased stone formation. So I think it's definitely prudent.

Six large retrospective studies have shown with no specific medical therapy there's a recurrence rate of 14% at one year, 35% at five years, and 52% at 10 years. Prospective data also shows that untreated first time stone formers will have a recurrence rate of 53%.

Now, risk assessment starts with figuring out if the patient's low risk or high risk. Low risk patients are first time stone formers without any evidence for GI or bone disease, no evidence for gout, recurrent infections, no nephrocalcinosis, no diabetes, no obesity, and no family history. And those patients can undergo a simple screening evaluation.

High risk patients are anybody who's had to stone before, a child or adolescent, anybody less than 40 years old, somebody who's higher risk because they're a solitary kidney, first time stone formers with renal GI or bone disease, gout, the non-calcium oxalate stones, as calcium oxalate are the most common stones, indicating there may be some metabolic disturbance going on, recurrent urinary tract infections, patients who have nephrocalcinosis, more than one stone or large stone burden at the time of diagnosis, anybody with diabetes, obesity, fragility, multiple comorbidities and family history. And they get a full metabolic evaluation.

The screening evaluation essentially consists of the history which we already did, including medications, which are important, basic set of labs, including parathyroid hormone, if primary hypoparathyroidism is suspected. And in addition to the urinalysis, the culture is also specifically looking for urea-splitting organisms which are stone forming. And lastly, a stone should be sent for analysis because there are specific stones that you may get that may indicate a metabolic process underlying the reason for their formation. And those metabolic processes can be targeted for therapy and prevention.

Generic diet recommendations consist of fluid intake, typically around 100 ounces a day. Sodium less than two grams, animal protein less than 8 ounces daily, and recommended daily allowance of calcium. Excessive calcium can cause stones, but also restriction of calcium can increase the risk for stones, as well.

The recommended daily allowance for men and premenopausal women is 1,000 milligrams per day. And then older men and postmenopausal women is 1,200 milligrams per day. Best obtained via diet and not supplements.

Some patients will ask me about oxalate, and I'll answer them. But I don't bring this up in my generic diet recommendations if not asked. I typically will tell patients that they can get oxalate in moderation. There's a lot of good healthy foods that are higher in oxalates, and most people don't eat excessive amounts to have a high oxalate in their urine as the primary cause for their calcium oxalate stones. It's another cause.

So if asked, I tell them in moderation. I talk to them about portion control. And if there are substitutions, such as kale instead of spinach, or other spices instead of black pepper, or green tea instead of black tea, then I'll discuss those with them if they tell me that they consume those in-- at a high quantity. A metabolic evaluation, a complete one, involves what we just talked about with the screening, but also with 24 hour urine, which looks at a whole host of different things that can play a role and could be stone risk.

There are definite benefits to 24 hour urine since guidelines support it. It provides objective quantifiable data, allowing you to individualize medicine for that specific patient, which patients tend to like. It allows you to check compliance with fluid and dietary recommendations, and also medication therapy. It allows you to limit any recommendations of diet and additions of medications to very specific issues, again, individualized in that medicine, and gives patients help to a lifelong disease.

The challenges are that it can be complex to interpret. It's limited in its ability to actually predict recurrence. So it can tell you that your risk is lower, but it can't guarantee that that person's not going to form another stone.

One of the reasons why that may be is because it could fail to capture true life. Some patients are on their best behavior when they're doing their 24 hour urines. They're at home, sequestered, rather than out and about or working.

It can also be difficult for patients to carve out time to do this, and sometimes to even understand the directions on how to do it. And it may require repeat testing, which again, is sometimes a nuisance for patients. It also is not free. Either insurance or patients have to pay for it. Typically, it is covered by insurance.

Now, the American Urological Association has a guideline on the medical management of kidney stones, and they talk about what you should do for evaluation, what diet and pharmacological therapies you consider-- should consider, and what followup is required. And an update to this guideline is coming in 2021.

The most common finding is low urine volume. The causes may be decreased intake, but also, potentially increased output. Some patients have bowel disease that cause chronic diarrhea, and some patients excessively sweat, leading to a low volume.

Clinician should recommend to all stone formers-- and this is a guideline-- a fluid intake that will achieve a urine volume of at least 2.5 liters daily minimum. So whatever a patient needs to take in to make 2.5 liters is what's recommended. Other findings when we do this full metabolic workup for calcium-based stones are high calcium, high oxalate, and low citrate.

High calcium, there's a variety of causes. The most common is dietary. So high salt diets can actually lead to more calcium in the urine, and animal protein can also push calcium into the urine. So we recommend a low salt diet and moderate animal protein.

As mentioned before, and I'll get more into this in just a moment, but normal recommended daily allowance of calcium is what's recommended. Not too much, not too little. Some medications and supplements are responsible for increasing calcium in the urine. And if a patient is taking one of those, then we ask them to seek alternatives and limit their use.

If we're suspicious for primary hypoparathyroidism, we'll refer to endocrine surgery. There may be other causes of patients to have hypercalcemia, such as sarcoidosis, and we'll refer to endocrinology to help tease that out. And lastly, often we'll find idiopathic causes for high calcium in the urine.

And there is actually a gene of idiopathic hypercalceuria that runs in families. And this is management-- managed with thiazides and potassium. Thiazide diuretics augment distal tubule reabsorption of calcium and stimulate proximal tubule reabsorption of calcium, leading to lower calcium levels in the urine. And then it gets put into the bone for use.

Some thiazide options are hydrochlorothiazide, chlorthalidone and indapamide. And it's important to note that hydrochlorothiazide once a day is not sufficient to get that hypocalceuric effect. So if I have a patient who's already on hydrochlorothiazide, I may bump them to twice a day. But I prefer to use either chlorthalidone or indapamide because I find that compliance is higher with a daily medication.

Lasix of note induces a hypercalciuria, and I do not use it in the setting as it can increase stone risk. And if a patient's on Lasix, I explore the option of potentially changing them to a thiazide. I do monitor their basic metabolic panel and uric acid, as well.

Hyperoxaluria, or high oxalate in the urine is found, but not as common from dietary reasons as from enteric reasons. So bowel pathology, like Crohn's, can increase oxalate in the urine and surgeries like gastric bypass or other small bowel surgeries. The physiologic mechanism in terms of bowel pathology of getting a hyperoxaluria has to do with calcium deficiency.

So calcium typically binds oxalate in the gut, forming a complex, and then passing through the intestine into the stool. When a patient's calcium deficient, either because they're restricting their calcium without that pathology, because they think that that's what they're supposed to do, or because they have bowel pathology, whether from surgery or from a condition, then the complex goes down of calcium oxalate and intestine that free oxalate is absorbed into the system and then gets filtered through the kidney increase and the risk for stones.

Often with these enteric pathologies, you can get diarrhea, as well, leading to dehydration and bicarbonate loss, which results in low urine volume and low citrate, both of which increase the risk for kidney stones, as well. So for enteric hyperoxaluria, the treatment is liberal fluid intake. Again, the goal of 2 and 1/2 liters output.

Calcium supplementation, it's the one time that I actually will give patients calcium supplements, typically, calcium citrate. And I have them take it with meals to bind to that oxalate. Potassium citrate, we will often do because they do get a hyper-- or a hypocitraturia potentially from that bicarbonate lost from diarrhea. And I use liquid form because it tends to have increased absorption.

Sometimes we can use vitamin B6 or cholestyramine to lower oxalate levels. I don't typically use those in my practice. That would be definitely second line or less options.

Dietary hyperoxaluria, again, isn't super common, but there are very healthy eaters that sometimes end up with high oxalate in the urine that's specifically dietary related. Liberal fluid intake, again, with a goal of 2.5 because it does dilute the system. Portion control and substitutions, as mentioned before, kale instead of spinach, other spices instead of black pepper, green tea instead of black tea, things like that.

Vitamin C and cranberry also metabolize into oxalate, so I tell people to limit the use of those, as well. And again, for the physiologic mechanism just mentioned, I counsel them of normal calcium intake. Because if somebody is restricting or deficient in calcium, it can increase oxalate in the urine, as well.

Primary hyperoxaluria is a rare cause of high oxalate in the urine. It's an autosomal recessive inborn error of metabolism that results in overproduction of endogenous oxalate. These patients are treated with kidney and liver transplant, and they're typically diagnosed long before they hit a urologist's office.

The last abnormality we'll discuss for calcium based stones that we find is low citrate. And they have-- there's a variety of reasons for that, as well, including idiopathic. But thiazides can induce a hypocitraturia if their potassium gets deficient. Diarrhea, we already mentioned. And then distal renal tubular acidosis.

Distal renal tubular acidosis occurs in young women. Patients often present and they have nephrocalcinosis. They tend to form calcium phosphate stones because calcium phosphate stones form at a higher pH. And the treatment for this is potassium citrate.

They're diagnosed because of a profoundly low urine citrate, and in conjunction with a higher urinary pH. And when looking at their blood tests, they have a hyperchloremic, hypokalemic metabolic acidosis. On a side note, carbonic anhydrase inhibitors, like topiramate, induce a renal tubular acidosis and is the reason why patients on these medications sometimes will form stones, and they tend to get calcium phosphate stones, if that is the cause of their stones.

The treatment essentially is stopping the medication, and it typically reverses those changes. Uric acid based stones are completely pH dependent. They may or may not have higher gas levels in their blood or urine. They are 100% pH dependent and will be in solution form because of their pKa, if over pH of 6.

There are a variety of causes of uric acid stones-- dietary, metabolic syndrome, among other more rare conditions. Treatment is first line alkalinization, getting that pH up to over 6. And sometimes this needs to be done in conjunction with moderating their animal protein less than 8 ounces per day.

If above fails, in conjunction with still trying to get their pH up, if their uric acid level is high in their blood, then I will potentially add allopurinol. Two other stones to just mention briefly-- cystine stones and infection stones. Cystine accounts for approximately 1% of all stones that present.

It's caused by cystinuria, which is an autosomal recessive condition. And these patients typically have large burdens throughout their life, and they present at a young age, typically, as a child, but not always. Treatment consists of aggressive fluid intake.

And for these patients, their goal is 4 liters of the output per day. They also are asked to have a low salt diet, and also moderate animal protein. And then there's medications to alkalinize their urine because it'll be more likely to be in solution at higher pH levels. And there are also special orphan medications that lower those levels, and also increase the solution and decreasing their super saturation.

And the classic infection stone is a struvite stone, and it often will have a staghorn configuration. These stones can grow very quickly when infected with a certain organism. These patients tend to have a very high urine pH and high ammonia in the urine that's a result of the bacteria that's present.

It's urea-splitting organisms that cause struvite stones. The classic ones are proteus, pseudomonas, klebsiella, and staph, but there are other ones, as well. Important to note, E. Coli, which is a common urinary pathogen is not typically urea-splitting.

Treatment entails complete elimination of the stone through surgery, aggressive UTI prevention that involves improving bladder health, adequate urinary drainage, and hydration. You may have heard of a medication called acetohydroxamic acid. It's a urease inhibitor. And it's actually not used by most urologists because it tends to have a high complication rate and is poorly tolerated by patients.

I also, for these patients, after I clear their stone [INAUDIBLE], we'll use three months of culture specific suppressive antibiotics just once a day. And I'll do screening cultures, as well, to check for those urea-splitting bacteria. In terms of followup, success is based on changes in urine parameters of the changes that you made.

The guidelines state that a single 24 hour urine should be done within six months of any recommendation to assess the response to therapy, whether it be dietary or medical changes. It should then be done annually, or with greater frequency if the risk is still high, to assess adherence to the therapy. This is an example of what a 24 hour urine looks like.

So the patient has-- this patient, the two initial collections are on the bottom, and then the more recent collection after therapy is instituted is on the top. This patient had low urine volume, was able to get it to the 2.1 range. Not quite at goal, but still significantly improved.

This is super saturation of calcium oxalate. And this is a very high risk, and they were able to get this down from a variety of reasons. This patient's calcium is also high, and it's often related to high salt diet. But they had a high salt on one day and a normal salt another day, and the calcium was high on both days.

So a thiazide medication was started, and it reduced their calcium into the normal range reabsorbing it into the system. Sometimes, we will use potassium citrate with a thiazide to prevent hypocitraturia from forming. This patient already had a low citrate. They were instituted potassium citrate with the thiazide, and it brought their level up. So in general across their board, the risk is down.

This patient's oxalate is normal, even though they probably are calcium oxalate stone formers, which is very typical. It also allows you to assess for compliance. So here's potassium down here. I typically am adding citrate therapy as potassium citrate, and I can see that their potassium appropriately rose.

And lastly for followup via the guidelines, periodic blood testing is required if starting on medication to assess any adverse effects. The guidelines also recommend a repeat stone analysis when available, especially when not responding to treatment, as patients' stone compositions can change. And for struvite stones, as already mentioned, it's important according to the guidelines, as well, to monitor for reinfection and to utilize aggressive strategies to prevent recurrence.

I also-- and the guidelines recommend-- periodically obtaining followup imaging. And again, my practice is to do every six months for the first year after events, whether it be surgery or passage. And if they've successfully reduced the risk, I'll back off to annually.

So in conclusion, that covers a lot of information. But stone disease is a major public health concern, including for cost purposes. Meticulous history and physical exam is essential to gather information, not only for acute management, but also for long-term prevention.

CT scan is the gold standard for diagnosing kidney stones. But ALARA, as low as reasonably achievable, should be remembered. An alternative imaging like low dose technologies or ultrasound should be considered when appropriate.

Observation of an obstructing stone with associated infection can lead to a life-threatening sepsis. Drainage is required, and it should be done emergently. Trial of passage and medical expulsive therapy should be done in the appropriate patient, less than 10 millimeters in size, controlled symptoms.

Definitive management with surgery should be carried out when necessary. All stone formers should get a simple screening evaluation that consists of history, and basic lab work, and urine work. High risk stone formers should occur-- should get a complete metabolic evaluation that includes a 24 hour urine. And I typically will offer this to all of my stone formation-- stone formers, even if they are low risk.

And in terms of followup, it's important to not only assess compliance of any recommendations that you make, whether they be dietary or medical, but it's also important to monitor for recurrences. Stone formation can be silent until it causes symptoms.

And that's it. I will be happy to take any questions.

FEMALE SPEAKER 1: Thank you so much, Dr. Semins.