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Selection of Isolated Colonies for Sensitivity in Vitek Mic Automated System: Influence of Presence of Crystals in Urine and Patient on Prior Antibiotic Medication: Study Done At Atertiary Care Laboratory in Northen India

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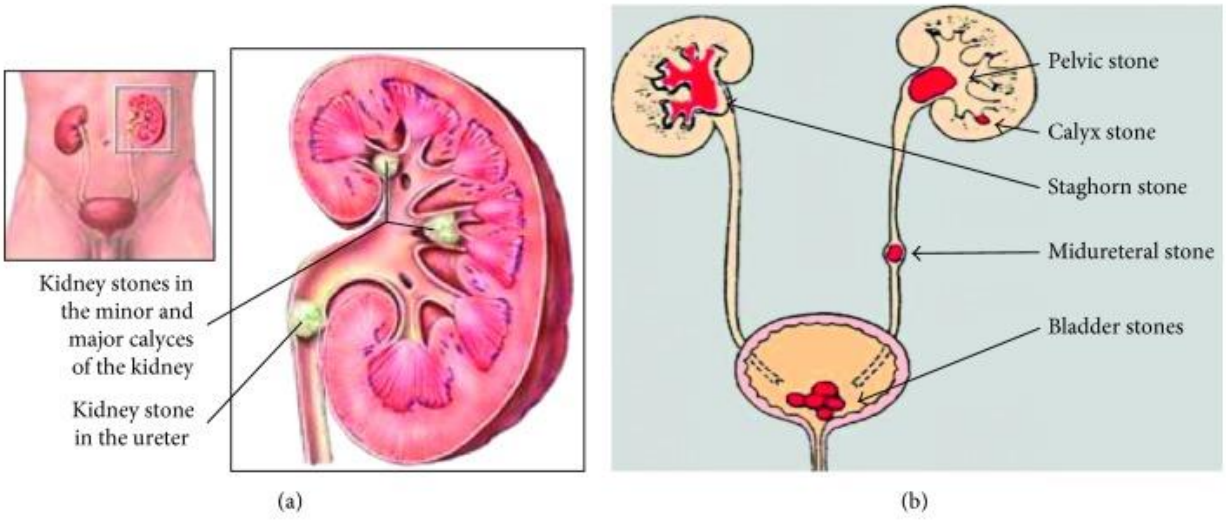
1. **AIM:** Selection of isolated colonies for sensitivity in vitek mic automated system:influence of presence of crystals in urine and patient on prior antibiotic medication: study done at tertiary care laboratory in northen india.

2. **MATERIAL** CLED AGAR PLATE, VITEK PANELS, CALIBRATED LOOP. BUNSEN BURNER

Review of literature: The Urinary System and Stones
After formation in the glomerulus, the urine filtrate travels into the tubules, where it undergoes volume and content changes due to secretions and reabsorption. The collecting ducts and distal tubules are responsible for fine-tuning the urine's composition, whereas the proximal tubules handle the majority of solute reabsorption. Urine: 95% water, 2.5% urea, and 2.5% a combination of minerals, sodium chloride, hormones, as well as enzymes is concentrated by the loop of Henle. Essential nutrients including bicarbonate, potassium, amino acids, proteins, salt, chloride, as well as water are reabsorbed as well as returned to the circulatory system stream in the proximal tubules. The regulation of blood salt as well as acid-base balance occurs in the distal tubule []. The stones' placement could change, as shown in Figure 1.



Kidney stone locations in the urinary system. (a) Adopted from [4]. (b) Adopted from [5].

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3. Types of Kidney Stones:

Kidney stones are characterised by an imbalance in the chemical make-up of urine. Size, form, and mineralogy are three ways in which stones vary from one another [6]. There are five main categories of kidney stones, distinguished by differences in mineral content and pathophysiology [7].

3.1. Calcium Stones: Calcium Oxalate and Calcium Phosphate

Calcium stones are predominant renal stones comprising about 80% of all urinary calculi [8]. The proportion of calcium stones may account for pure calcium oxalate (CaOx) (50%), calcium phosphate (CaP, termed as apatite) (5%), and a mixture of both (45%) [9] . The main constituent of calcium stones is brushite (calcium hydrogen phosphate) or hydroxyapatite . Calcium oxalate is found in the majority of kidney stones and exists in the form of CaOx monohydrate (COM, termed as mineral names: whewellite, $CaC_2O_4 \cdot H_2O$), and CaOx dihydrate (COD, weddellite, $CaC_2O_4 \cdot 2H_2O$), or as a combination of both which accounts for greater than 60%. COM is the most thermodynamically stable form of stone. COM is more frequently observed than COD in clinical stones [10].

Many factors contribute to CaOx stone formation such as hypercalciuria (resorptive, renal leak, absorptive, and metabolic diseases), hyperuricosuria, hyperoxaluria, hypocitraturia, hypomagnesuria, and hypercystinuria [11]. Mostly, urinary pH of 5.0 to 6.5 promotes CaOx stones [12], whereas calcium phosphate stones occur when pH is greater than 7.5. The recurrence of calcium stone is greater than other types of kidney stones.

3.2. Struvite or Magnesium Ammonium Phosphate Stones

Struvite stones occur to the extent of 10–15% and have also been referred to as infection stones and triple phosphate stones. It occurs among patients with chronic urinary tract infections that produce urease, the most common being *Proteus mirabilis* and less common pathogens include *Klebsiella pneumonia*, *Pseudomonas aeruginosa*, and *Enterobacter* . Urease is necessary to split/cleave urea to ammonia and CO_2, making urine more alkaline which elevates pH (typically > 7). Phosphate is less soluble at alkaline versus acidic pH, so phosphate precipitates on to the insoluble ammonium products, yielding to a large staghorn stone formation [13]. Women's are likely to develop this type of stone than the male. *Escherichia coli* is not capable of splitting urea and is not associated with struvite stones [14].

3.3. Uric Acid Stones or Urate

This accounts approximately for 3–10% of all stone types [1, 29]. Diets high in purines especially those containing animal protein diet such as meat and fish, results in hyperuricosuria, low urine volume, and low urinary pH (pH < 5.05) exacerbates uric acid stone formation [11, 28, 39]. Peoples with gouty arthritis may form stones in the kidney(s). The most prevalent cause of uric acid nephrolithiasis is idiopathic [38], and uric acid stones are more common in men than in women.

3.4. Cystine Stones

These stones comprise less than 2% of all stone types. It is a genetic disorder of the transport of an amino acid and cystine. It results in an excess of cystinuria in urinary excretions [1, 29], which is an autosomal recessive disorder caused by a defect in the rBAT gene on chromosome 2 [40], resulting in impaired renal tubular absorption of cystine or leaking cystine into urine. It does not dissolve in urine and leads to cystine stone formation [11]. People who are homozygous for cystinuria excrete more than 600 millimole insoluble cystine per day [28]. The development of urinary cystine is the only clinical manifestation of this cystine stone disease [40].

3.5. Drug-Induced Stones

This accounts for about 1% of all stone types [1]. Drugs such as guaifenesin, triamterene, atazanavir, and sulfa drugs induce these stones. For instance, people who take the protease inhibitor indinavir sulphate, a drug used to treat HIV infection, are at risk of developing kidney stones [28]. Such lithogenic drugs or its metabolites may deposit to form a nidus or on renal calculi already present. On the other hand, these drugs may induce the formation of calculi through its metabolic action by interfering with calcium oxalate or purine metabolisms [38].

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4. Kidney Stone Compositions

Crystals and noncrystalline phases, or the organic matrix, make up the chemical compositions in urinary stones. Urinary stones include lipids, glycosaminoglycans (GAGs), carbohydrates, proteins, and a variety of macromolecules in their organic matrix. These molecules are crucial because they either facilitate or impede the processes that lead to the formation of kidney stones (Table 1). Inorganic ash(10.4%), water(10%), hexosamine as glucosamine(5%), nonamino sugars(9.6%), and proteins(64%) make up the bulk of the stone matrix. When kidney stones are being assembled, the matrix is used as a template. Approximately 10.3% of stone matrix is composed of phospholipids, which account for 8.6% of total lipids in all stones. Calcium oxalate as well as calcium phosphate stones are encouraged to form by cellular membrane phospholipids, which are a component of the organic matrix [41]. The matrix of every kind of stone is mostly albumin [42].

Table 1

Modules of crystallisation in nephrolithiasis involving proteins in the urinary stone matrix **[34, 41].**

Serial Number	Name of protein	Role in crystallization			
		Nucleation	Growth	Aggregation	Cell adherence
1	Nephrocalcin (NC)	I	I	I	—
2	Tamm–Horsfall protein (THP)	P	—	I/P	—

Serial Number	Name of protein	Role in crystallization			
		Nucleation	Growth	Aggregation	Cell adherence
3	Osteopontin/uropontin (OPN)	I	I	I	I/P
4	Albumin	P	—	I	—
5	Urinary prothrombin fragment-1 (UPTF1)	I	I	I	—
6	Alpha-1-microglobulin	—	—	I	—
7	S100A	—	I	I	—
8	Inter-alpha-inhibitor	I	I	I	I
9	Bikunin	I	I	I	I
10	Renal lithostathine	—	I	—	—
11	Alpha defensin	—	P	P	—
12	Human phosphatecytidylyl transferase 1, choline, beta	—	I	—	—
13	Myeloperoxidase	—	P	P	—
14	Nucleolin	—	—	—	P
15	Histone-lysine N methyltransferase	—	I	I	—
16	Inward rectifier K channel	—	I	I	—
17	Protein Wnt-2	—	I	I	—
18	Alpha-2HS glycoprotein	P	I	—	—
19	Crystal adhesion inhibitor (CAI)	—	—	—	I
20	Hyaluronic acid (HA)	—	—	—	P
21	Chondroitin sulphate	—	I	I	—
22	Heparin sulphate (HS)	—	I	—	—
23	Human urinary trefoil factor 1(THF1)	—	I	—	—
24	Monocyte chemoattractant protein-1 (MCP 1)	—	—	—	P
25	Annexin II	—	—	—	P
26	CD44	—	—	—	P
27	Matrix Gla protein (MGP)	—	I	—	I
28	Histone H1B	—	P	—	—
29	Fibronectin	—	—	I	I

Serial Number	Name of protein	Role in crystallization			
		Nucleation	Growth	Aggregation	Cell adherence
30	Collagen	P	—	—	—
31	Glycosaminoglycans	I	I	I	I
32	Citrate	—	I	—	—
33	Pyrophosphate	—	I	—	—
34	Magnesium	—	I	—	—

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I: inhibitor; P: promoter; "—": no effect.

One quarter of individuals with calcium phosphate (CaP) develop stones that include brushite, a hard phosphate mineral that is becoming more common [43]. Hydroxapatite, carbonate apatite, or brushite (calcium monohydrogen phosphate dihydrate, CaHPO4·2H2O) are three possible forms of calcium phosphate that may be found in the urinary system. There is no way to treat brushite using shock waves or ultrasonic lithotripsy [44].

4.1. Etiology of Kidney Stones

Factors both internal (such as gender and genetics) and external (such as location, weather, food, mineral makeup, and fluid consumption) contribute to the complicated and multi-step process of kidney stone formation (calculogenesis) [15]. In Table 2 we can see a list of potential reasons why kidney stones may occur.

Table 2

Factors that increase the likelihood of developing kidney stones.

Number	Risk factors	References
1	*Lifestyle habits and dietary/nutritional factors*: such as a diet lacking in chelating substances (such as citrate, fibre, and alkaline foods) and an overabundance of salt and animal proteins	[9, 13, 19, 45]
2	*Metabolic disorders*: symptoms such as an excess of calcium in the urine, an inadequate breakdown of citrate, an increase in the amount of uric acid in the urine, and a past history of gout	[38, 46–48]
3	*Hypercalcemic disorders*: calcium metabolism abnormalities, including primary hyperparathyroidism	[49]
4	*Urine composition*: an imbalance between the excretion of inhibitory chemicals (urine lacking in inhibitory substances) and the excretion of promoters to urinary crystallisation	[1, 45, 49]
5	*Low urine volume*: lack of water consumption leading to dehydration and urine that is too saturated	[45, 49, 50]

Number	Risk factors	References
6	*Recurrent urinary tract infections*: irregularities in the urine's pH and the alkalinization of urine caused by urease, a bacterium like Proteus mirabilis	[38, 49]
7	*Genetic predisposition/inherited disorders*: hereditary renal tubular acidosis, a history of kidney stones in the family, and hereditary monogenic illnesses	[1, 9, 48, 49, 51]
8	*Anatomical abnormalities*: variables such pyeloureteral duplication, polycystic renal disease, horseshoe kidney, ureteropelvic junction stenosis, and abnormalities in the medullary sponge kidney	[1, 48, 49, 52]
9	*Hypertension*	[46]
10	*Obesity*	[46–48]
11	Factors such as climate change (the warming of the planet), employment, geographical location, and seasonal changes (summer being higher than winter)	[1, 49]
12	Conditions characterised by intestinal malabsorption, including inflammatory bowel disease	[9, 49]
13	No oxalate-degrading bacteria in the intestines	[53, 54]
14	*Lithogenic drugs*: protease inhibitors such indinavir (Crixivan), uricosuric drugs like sulfadiazine, which have limited solubility and encourage the development of calculi, and ceftriaxone, which requires a large dosage over an extended period of time.	[28, 38, 49, 50]

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5. Mechanisms of Renal Stone Formation

Kidney stone pathophysiology, also known as biomineralization, is a complicated biochemical process that is still not fully understood [41]. Supersaturation of urine and physicochemical changes constitute the biological process of renal stone development. A supersaturated solution is one in which there is more solute than the solvent would normally be able to dissolve [34]. Solutes precipitate is urine due to supersaturation, which in turn causes nucleation and the eventual formation of crystal concretions. When the level of both ions in a solution beyond their saturation point, crystallisation takes place [55]. Phosphorus and particular amounts of surplus chemicals affect the phase transition from liquid to solid. Risk factors for crystallisation include low urine volume, high levels of stone-forming components such as calcium, phosphorus, uric acid, oxalate, and cystine, and a high degree of urinary saturation relative to these substances [1, 56]. So, crystallisation is dependent on the nucleation-inducing thermodynamics and the crystal-growth-consuming kinetics of a supersaturated fluid [57]. Hence, staying away from supersaturation is the best way to avoid lithiasis.
Keep in mind that the relative abundance of urinary inhibitors and crystallisation promoters is often what determines the severity of stone formation. In terms of the mineral stage of stone production, all stones go through the same sequence of events. However, the specific chain of actions that culminates in stone

creation varies with stone type and urine chemistry. So, for example, calcium oxalate , calcium phosphate stones may form in supersaturated urine with low inhibitor concentrations. Calcium oxalate (CaOx) stones are more likely to develop when uric acid prevents the mineral from dissolving. The crystallisation process is inhibited and becomes safe in healthy controls [1]. In order for stones to develop, a series of processes must occur, including crystal nucleation, development, aggregation, and their retention by the kidneys [27, 58].

5.1. Crystal Nucleation

The first step in the formation of kidney stone begins by the formation of nucleus (termed as nidus) from supersaturated urine retained inside the kidneys [11, 42]. In a supersaturated liquid, free atoms, ions, or molecules start forming microscopic clusters that precipitate when the bulk free energy of the cluster is less than that of the liquid. For example, charged soluble molecules such as calcium and oxalate combine to form calcium oxalate crystals and become insoluble [34]. Nucleation may be formed in the kidney through free particle or fixed particle mechanism [26, 34]. In supersaturated solutions, if promoters exceed that of inhibitors, nucleation starts [34].

Once a nucleus is created (and/or if it is anchored), crystallization can occur at lower chemical pressure than required for the formation of the initial nucleus. Existing epithelial cells, urinary casts, RBCs, and other crystals in urine can act as nucleating centers in the process of nuclei formation termed as heterogeneous nucleation [41]. The organic matrix, mucopolysaccharide acts as a binding agent by increasing heterogeneous nucleation and crystal aggregation [59]. On the other hand, nanobacteria is claimed to form apatite structures serving as a crystallization center for stone formation [60]. The whole process potentiates stone formation. The role of oxalate-degrading bacteria, such as *Oxalobacterformigenes*, in CaOx stone formation is a subject of current research [61]. Thus, treatment which targets the process of nucleation intervention is one of the best approaches to control kidney stone.

5.2. Crystal Growth

Crystals in urine stick together to form a small hard mass of stone referred as crystal growth. Stone growth is accomplished through aggregation of preformed crystals or secondary nucleation of crystal on the matrix-coated surface [62]. Once a nidus has achieved, the overall free energy is decreased by adding new crystal components to its surface. The total free energy of the cluster is increased by the surface energy. The process of stone growth is slow and requires longer time to obstruct the renal tubules [34]. From organic matrix, mainly Tamm–Horsfall protein and osteopontin are promoters of CaOx stone formation [13]. Under in vitro study, crystals induced in human urine demonstrated an intimate association between calcium-containing crystals and organic matrix (lipids and proteins). Lipids of cellular membranes are basically believed to involve in nucleation of crystals [63].

5.3. Crystal Aggregation

The process whereby a small hard mass of a crystal in solution sticks together to form a larger stone is called aggregation. All models of CaOx urolithiasis concede that crystal aggregation is probably involved in crystal retention within the kidneys [41]. Crystal aggregation is considered to be the most critical step in stone formation.

5.4. Crystal-Cell Interaction

The attachment of grown crystals with the renal tubule lining of epithelial cells is termed as crystal retention or crystal-cell interaction [41, 64]. In individuals with hyperoxaluria, renal tubular epithelial cells were injured due to exposure to high oxalate concentrations or sharp calcium oxalate monohydrate (COM) crystals [10, 65, 66]. Crystal-cell interaction results in the movement of crystals from basolateral side of

cells to the basement membrane [10]. Then, crystals could be taken into cells and anchored to the basement membrane of the kidneys [66]. The interaction of COM crystals with the surface of renal epithelial cells could be a critical initiating event in nephrolithiasis. An increased retention force between the crystal and injured renal tubule epithelium cells promotes CaOx crystallization [67]. Most of the crystals attached to epithelial cells are thought to be digested by macrophages and/or lysosomes inside cells and then discharged with urine [66].

Following renal tubular cell injury, cellular degradation produces numerous membrane vesicles which are nucleators of calcium crystals as supported by in vitro and in vivo studies [41]. Agglomeration of COM crystals is induced by chemicals released by injured cells, such as renal prothrombin fragment-1 and other anionic proteins [68]. It is believed that reactive oxygen species are a component of renal cell damage [69]. This suggests that lowering renal oxidative stress may be a viable therapeutic strategy. Crystals may attach to damaged cells because their membranes are anionic to the urine environment. Compared to crystals of calcium oxalate dihydrate (COD), COM crystals show a greater attachment affinity towards the inverted anionic membrane [69]. Researchers found that COM crystals were more often deposited in Madin-Darby canine kidney epithelial cells (MDCK cells) compared to proximal tubular epithelial cells generated from pig kidney (LLC-PK1 cells) [71]. One possible explanation for this preference is that Madin-Darby canine kidney epithelial cells have a binding molecule, like hyaluronan, that allows COM crystals to connect to them [67]. While the exact manner in which crystals and cells interact are still a mystery, regulating crystal-cell retentions is a promising strategy for treating urolithiasis.

5.5. Endocytosis of CaOx Crystals

The first step in the production of kidney stones is endocytosis, which is the engulfment by crystals by cells in the renal tubules. Research into the interactions between tissue culture crystals and cells has shown that COM crystals are able to quickly bind to microvilli on cell surfaces and then be internalised. Crystals may be coated with polyanion molecules found in tubular fluid/urine, such as citrate, glycosaminoglycans, or glycoproteins, which prevent COM crystals from attaching to cell membranes [41]. In stone formation, for instance, Tamm-Horsfall glycoproteins (THP) have a dual biological purpose. Initiating the interaction of COM crystals to distal tubular cells in the nephron may be one mechanism by which THP promotes renal stone formation, according to Lieske et al. [72]. According to another research, when the pH is lowered and the ionic strength is increased, the viscosity of THP rises. This makes it more likely to polymerize, but it doesn't stop crystallisation. In addition, when more calcium ions are present, THP becomes a potent crystallisation activator [73]. Hess found that THP inhibited COM aggregation at high pH and low ionic strength, suggesting that it may protect against COM stone formation [73]. Desialylated THP enhanced COM aggregation, but normal THP prevented aggregation, according to COM aggregation tests [74]. According to similar studies, uromodulin promotes aggregation of calcium oxalate crystals [75], whereas THP inhibits this process. The production of calcium crystals for adult kidneys occurs spontaneously when the THP gene is inactivated in mouse embryonic stem cells. The results show that THP is an effective urinary inhibitor for nephrolithiasis in humans [76].

The creation of stones is a complex process that involves several cellular and extracellular activities. To prevent stones from forming, modulators might be useful in halting the process from supersaturation into crystal retention. Another possible method to avoid stone formation is to obstruct crystal binding molecules that are expressed on the membranes of epithelial cells, including osteopontin, hyaluronic acid, sialic acid, as well as monocyte chemoattractant protein-1 [41]. Research has shown that oxidative stress and reactive oxygen species (ROS) are the catalysts for stone calcification [77]. Research has shown that

CaOx crystals are harmful to renal epithelial cells, leading to damage and cell death in both laboratory and animal experiments [78, 79, 80, 81]. Similarly, hypercalciuria causes cellular damage and lipid peroxidation mediated by reactive oxygen species, which in turn promotes the deposition of calcium oxalate [82]. Urinary stone formation's pathogenesis remains poorly understood. Here is a rundown of the several processes that go into making stones (Figure 2) Treating Simple UTIs From three days to six weeks, antibiotic therapy has traditionally ranged. Even though it only takes three days of treatment with "mini-dose therapy," the cure rate is quite high. The prevalence of E. coli strains that are resistant to widely used antibiotics varies throughout the nation. If the resistance rate to an antibiotic is more than 50%, it is recommended to choose an alternative medicine.

Nitrofurantoin, sulfamethoxazole/trimethoprim, fosfomycin, as well as first-generation cephalosporins are often used as first-line treatments for simple UTIs. When it comes to first-line treatment, pivmecillinam is likewise a top choice outside of the US.

- • For simple UTIs, **nitrofurantoin** may be the best option; however, it is only bacteriostatic and has to be taken for 5 to 7 days rather than bacteriocidal. Resistance is rare since it affects germs via several methods. It is not appropriate for the treatment of suspected or confirmed pyelonephritis and works exclusively in the lower part of the bladder because of low tissue concentrations. Patients having recurrent UTIs are best served by using this medicine for long-term prevention at low doses.[5]
- Resistance rates are significant in many locations, although **sulfamethoxazole/trimethoprim** for three days is an effective mini-dose treatment. If the local bacterial resistance are more than 20% or if a patient has an allergy to sulfa, it should not be utilised.[40][41] The Long-term prophylaxis in individuals with recurrent UTIs is often best accomplished with the combination of sulfamethoxazole and trimethoprim.
- For simple UTIs, **Fosfomycin** is OK to take as a single dosage, according to the FDA.In cases when other antibiotics have developed substantial resistance, it may be useful [42].[43] When compared to other medicines, which need 7–10 days of treatment to achieve therapeutic urine concentrations, this one dosage lasts 2–4 days.[42][44] Additional symptomatic alleviation may be achieved with adjunctive treatment with phenazopyridine for many days.[45]
- To prevent resistance, **first-generation cephalosporins** should not be misused; nonetheless, they are suitable options for mini-dose (3-day) treatment.
- Despite their high resistance, **fluoroquinolones** are the preferred choice for treating pyelonephritis and prostatitis because of their high levels of tissue penetration, particularly in the prostate. Because of this, fluoroquinolones should not be used for simple UTIs, although they may be if other options are not tolerated.[46](47, 48) It is not recommended to combine fluoroquinolones with nitrofurantoin since they are hostile to each other. You should give serious thought to the recent FDA warnings on fluoroquinolone adverse effects. A course of norfloxacin may be appropriate for mild cases of cystitis. This quinolone isn't for pyelonephritis; it's just for UTIs in the lower urinary tract.
- **Pivmecillinam** is a first-line treatment for simple UTIs in countries where it is accessible, but not in the United States. Because of the risk of insufficient tissue penetration, it is not advised for use in cases of pyelonephritis nor suspected systemic infections.[49]

About 20% of women will see a spontaneous resolution of their UTIs, particularly with improved hydration, even in the absence of medication. Acute pyelonephritis is highly unlikely to occur in a healthy, nonpregnant woman.

Management of Recurrent UTIs

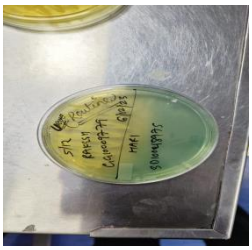
To manage recurrent UTIs, one should practise good personal cleanliness, take additional measures after sexual contact, use vitamin C to acidify the urine, and maybe use preventive antibiotics or antiseptics this nitrofurantoin.[39] (For further information, consider reading the related StatPearls reference page on "Recurrent Urinary Tract Infections.") [5]

- The usual treatment for recurring UTIs is **nitrofurantoin** low-dose long-term prophylaxis. Standard dosing is 50 milligrammes taken every 12 hours. The drug has a low risk of bacterial resistance because of its many antibacterial activities, it is seldom associated with allergies or intolerance, and it is well-tolerated since therapy is restricted to the urinary system, which reduces the likelihood of adverse effects.[5] Other options include sulfamethoxazole with trimethoprim or trimethoprim on its own. Additionally, in some instances, norfloxacin as well as fosfomycin may be administered.
- If the urine pH is less than 5.5, **methylamine** is transformed into formaldehyde in the bladder. It is common practice to utilise vitamin C to acidify the urine and reach this pH. Methenamine may help prevent recurring UTIs, while the evidence is mixed.[50][51] In certain cases, it could be a good substitute for antibiotics.[52][53]
- **Cranberry** has also been proposed, and there is some evidence that it works (in the form of juice, tablets, or extract), albeit the data is inconsistent. [51][54] The[55] The Compared to low-dose antibiotic treatment, several studies reveal a decrease in UTIs of 30–40%.[50][54][56]
- There is some indication that using **D-mannose** as a preventative agent may be beneficial.[57]in the 58th[59][60][61] But there isn't enough evidence to make a formal recommendation just yet.[5][50][51][62][63]
- Atrophic vaginitis is treatable in postmenopausal women who use **oestrogen vaginal cream** twice weekly.[50][64]
- It is beneficial for women to drink more fluids if their urine output is poor.[64][65]

  The typical course of preventative medicine is between six and twelve months. Although this may be prolonged, there is a lack of sufficient evidence and a significant number of individuals will need to resume their preventative therapy. [39] (66, 67) Some have even proposed making the preventative therapy duration two years long. [68][69]

  Both the American Urological Association Guidelines on Recurrent Urinary Tract Infections as well as our StatPearls reference page on "Recurrent Urinary Tract Infections" detail the diagnosis and treatment of recurring UTIs. [5][39]

  It is important to thoroughly inspect for a cause, such as a stone infection or a poorly emptied diverticulum, in cases of relapsing infections (in which the infecting organism is same on all cultures). [1] Refer to our supplementary StatPearls page on "Complicated Urinary Tract Infections" for more information.[1]



Figures

Figure 1. Urine sample with GNB growth with patient on prior medication forming deposits merged with isolated colonies

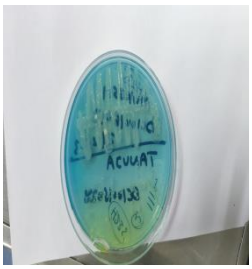


Figure 1. Urine sample with GNB growth with crystal in urine merged with colonies

IMPORTANCE: When the fluid in your urine is unable to dilute the concentration of crystal-forming chemicals including calcium, oxalate, and uric acid, kidney stones will develop. Kidney stones may develop in urine that doesn't contain chemicals that stop crystals from staying together. To test this, you can centrifuge your urine and use the sediment for culturing, and the supernatant for regular clinical pathology tests. furthepre treatment of urine sample with plasma to remove inhibitors like crsytals and antibiotic residue on patients on empirical may lead to non termination of cards in vitek.

CONCLUSION: Urine sample should be pre treated with plasma to aid in removal of crystals to put antibiotic sensitivity and prevent cards from terminating.

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