

Possible Complications Associated to Renal Litiasis, A Brief Review

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ABSTRACT

Renal lithiasis, commonly known as kidney stones, is a prevalent condition characterized by the formation of crystalline mineral concretions within the urinary tract. The pathogenesis involves supersaturation of urine with stone-forming elements, crystal nucleation, growth, and aggregation, often influenced by metabolic, dietary, and genetic factors. While some stones remain asymptomatic, others can cause severe complications such as obstructive uropathy, urinary tract infections, and progressive renal impairment. Untreated stones increase the risk of acute kidney injury, chronic kidney disease, and systemic issues including cardiovascular morbidity. Diagnosis relies on imaging modalities, and management encompasses pain control, stone removal, and preventative strategies targeting metabolic abnormalities. Recognizing the potential complications and implementing timely, individualized treatment are critical to preserving renal function and improving patient outcomes.

KEYWORDS: Renal lithiasis, Kidney stones, Nephrolithiasis, Urolithiasis, Kidney stone disease, Stone formation, Obstructive uropathy, Renal impairment, Kidney function loss, Urinary tract infection, Metabolic factors, Diagnosis, Management

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INTRODUCTION

Renal lithiasis, also known as nephrolithiasis or kidney stone disease, refers to the formation of crystalline mineral concretions (calculi) within the renal parenchyma or collecting system. These stones are typically composed of calcium oxalate, calcium phosphate, uric acid, struvite, or cystine, with calcium-based stones being the most prevalent. Stone formation is driven by supersaturation of urine with stone-forming constituents such as calcium, oxalate, phosphate, or uric acid, often in the context of reduced urinary inhibitors like citrate or magnesium, and can be influenced by dietary, metabolic, and genetic factors.[1-3]

The pathogenesis involves nucleation, growth, aggregation, and retention of crystals within the renal tubules or collecting system. Crystals may adhere to renal tubular epithelial surfaces, particularly at sites of interstitial apatite deposits

(Randall's plaques), serving as a nidus for further stone growth.[4] Clinical manifestations range from asymptomatic stones detected incidentally to acute renal colic, hematuria, urinary tract infection, or, in severe cases, obstruction and renal impairment.[4] In the context of renal transplantation, lithiasis is an uncommon but important complication, with similar pathophysiology and management principles as in the general population.[5-7]

Diagnosis relies on imaging modalities such as ultrasonography, plain radiography, or computed tomography, and management includes acute pain control, facilitation of stone passage, and prevention of recurrence through dietary modification, correction of metabolic abnormalities, and, when indicated, pharmacologic therapy.[2-3]



Figure 1 complete coralliform Stone



Figure 2 Simple abdominopelvic tomography: Left renal unit with regular borders, homogeneous parenchyma, heterogeneous collecting system at the expense of a hyperdense image that partially occupies it with a branched morphology of up to 590 HU compatible with coralliform lithiasis, with moderate ectasia.

Possible complications

Untreated renal lithiasis can result in a spectrum of complications, primarily due to mechanical obstruction, recurrent infection, and direct renal parenchymal injury. The most common and clinically significant complications include:

1. Obstructive uropathy and hydronephrosis: Stones can cause partial or complete obstruction of urinary flow, leading to hydronephrosis and, if persistent, progressive renal parenchymal atrophy and loss of function. Acute obstruction may present as renal colic, while chronic obstruction can be insidious and result in irreversible renal damage.[1-2]
2. Acute kidney injury (AKI) and chronic kidney disease (CKD): Obstructive stones, especially if bilateral or in a solitary kidney, can precipitate AKI. Epidemiologic data demonstrate that stone formers have an increased risk of

developing CKD and end-stage renal disease (ESRD), with the risk approximately doubled compared to non-stone formers. The risk is higher in patients with recurrent stones, large stone burden, or associated comorbidities such as recurrent urinary tract infections, structural urinary tract abnormalities, or monogenic disorders (e.g., cystinuria, primary hyperoxaluria).[3-6]

3. Recurrent urinary tract infections (UTIs) and pyelonephritis: Stones, particularly those causing obstruction, predispose to recurrent UTIs and pyelonephritis. Struvite stones are especially associated with infection and can lead to the formation of staghorn calculi, which further increase the risk of chronic infection and renal scarring.[4-6]

4. Renal parenchymal injury and scarring: Direct injury from crystal deposition and the associated inflammatory response can result in interstitial fibrosis and tubular atrophy,

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contributing to progressive renal dysfunction even in the absence of overt obstruction.[2][4]

5. Urosepsis: Obstructed, infected systems can rapidly progress to urosepsis, a life-threatening complication requiring urgent intervention.[1][4]

6. Other systemic complications: Epidemiological studies indicate that patients with nephrolithiasis have an increased risk of hypertension, coronary heart disease, and cerebrovascular events, independent of traditional risk

factors. The mechanisms are not fully elucidated but may involve shared metabolic risk factors and chronic inflammation.[7]

In summary, untreated renal lithiasis can lead to acute and chronic renal dysfunction, recurrent infections, urosepsis, and increased cardiovascular and cerebrovascular morbidity. The risk and severity of complications are influenced by stone size, location, composition, frequency of recurrence, and the presence of comorbid conditions.[1][7]

Aspect	Details
Definition	Formation of crystalline mineral stones within the kidney or urinary tract.
Common Components	Calcium oxalate, calcium phosphate, uric acid, struvite, cystine.
Pathogenesis	Urine supersaturation → crystal nucleation, growth, and aggregation → stone formation.
Risk Factors	Dietary, metabolic, genetic factors, reduced urinary inhibitors, dehydration, infections.
Clinical Presentation	Asymptomatic, renal colic, hematuria, urinary tract infections, obstruction.
Diagnosis	Ultrasonography, plain radiography, computed tomography (CT).
Complications	Obstructive uropathy, hydronephrosis, acute and chronic kidney injury, infections, systemic morbidity.
Management	Pain control, stone removal (ureteroscopy, lithotripsy, surgery), prevention (diet, medication).
Prevention	Dietary modification, hydration, metabolic correction, pharmacotherapy for recurrent stones.
Systemic Impact	Increased risk of CKD, cardiovascular disease, and cerebrovascular events.

CONCLUSION

In conclusion, renal lithiasis is a common condition characterized by the formation of kidney stones that can lead to significant renal and systemic complications if left untreated. The pathogenesis involves complex interactions between mineral supersaturation, crystal formation, and site-specific adherence within the urinary tract. Clinically, untreated stones pose risks including obstructive uropathy, infections, progressive renal impairment, and potential systemic consequences such as hypertension and cardiovascular disease. Effective diagnosis, timely management—including pain control, removal strategies, and preventative measures—are essential to mitigate these risks and preserve renal function. Addressing underlying metabolic abnormalities and reducing recurrence are pivotal in improving patient outcomes and preventing long-term morbidity associated with renal lithiasis.

REFERENCES

I. ACR Appropriateness Criteria® Acute Onset Flank Pain—Suspicion of Stone Disease (Urolithiasis). Gupta RT, Kalisz K, Khatri G, et al. *Journal of the American College of Radiology*. 2023;20(11S):S315–S328. doi:10.1016/j.jacr.2023.08.020.

II. Mulay SR, Anders HJ. Crystallopathies. *The New England Journal of Medicine*. 2016;374(25):2465–76. doi:10.1056/NEJMra1601611.

III. Alexander RT, Hemmelgarn BR, Wiebe N, et al. Kidney Stones and Kidney Function Loss: A Cohort Study. *BMJ (Clinical Research Ed.)*. 2012;345:e5287. doi:10.1136/bmj.e5287.

IV. Zisman AL, Evan AP, Coe FL, Worcester EM. Do Kidney Stone Formers Have a Kidney Disease? *Kidney International*. 2015;88(6):1240–1249. doi:10.1038/ki.2015.254.

V. Gambaro G, Croppi E, Bushinsky D, et al. The Risk of Chronic Kidney Disease Associated With Urolithiasis and Its Urological Treatments: A Review. *The Journal of Urology*. 2017;198(2):268–273. doi:10.1016/j.juro.2016.12.135.

VI. Keddis MT, Rule AD. Nephrolithiasis and Loss of Kidney Function. *Current Opinion in Nephrology and Hypertension*. 2013;22(4):390–396. doi:10.1097/MNH.0b013e32836214b9.

VII. Sáenz-Medina J, Gómez Dos Santos V, Rodríguez-Monsalve M, et al. Cardiovascular and Cerebrovascular Morbidity in Patients With Urolithiasis: An Epidemiological Approach Based on Hospitalization Burden Data From 1997 to 2021. *Journal of Clinical Medicine*. 2024;13(12):3564. doi:10.3390/jcm13123564.