

Renal Lithiasis: A Comprehensive Review of Risk Factors and Surgical Management

Carlos Rafael Hernández Álvarez¹, Roberto López Castillo², Julio César Gutiérrez Magaña³, Ana Alicia Castillo Alejandro⁴, Daniel Arturo Prior Catalán⁵, Diana Patricia López Hernández⁶, Raúl Cristóbal de Jesús Rodríguez Arcos⁷, José Manuel Gómez Pérez⁸, Jesús Alberto Lizárraga Castro⁹

¹Hospital General de Zona No. 11, IMSS

²Hospital General "Dr. Daniel Gurría Urgell", ISSSTE

³Hospital Regional de Alta Especialidad "Dr. Juan Graham Casasús"

⁴Hospital Regional de Alta Especialidad "Dr. Gustavo A. Roviroso Pérez"

⁵Hospital de Especialidades ISSSTEP

⁶Hospital Regional de Alta Especialidad "Dr. Juan Graham Casasús"

⁷Hospital Regional de Alta Especialidad "Dr. Juan Graham Casasús"

⁸Instituto Jalisciense de Cirugía Reconstructiva "Dr. José Guerrero-Santos"

⁹Hospital Regional de Alta Especialidad "Dr. Gustavo A. Roviroso Pérez"

ABSTRACT

Renal lithiasis, or kidney stone disease, is a frequent and recurrent urological disorder characterized by the formation of mineral concretions within the urinary tract. Its pathogenesis is multifactorial, involving metabolic imbalances, dietary habits, environmental influences, and genetic predispositions. Risk factors such as dehydration, high sodium and oxalate intake, obesity, metabolic disorders, and anatomical abnormalities contribute to stone formation and recurrence. While many cases can be managed conservatively through lifestyle and dietary modifications, surgical intervention is often necessary in symptomatic or complicated stones. Modern minimally invasive techniques, including ureteroscopy, percutaneous nephrolithotomy, and extracorporeal shock wave lithotripsy, have significantly improved outcomes. This review provides a detailed analysis of the risk factors for renal lithiasis and highlights the role of surgical management in comprehensive patient care.

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INTRODUCTION

Renal lithiasis, also referred to as nephrolithiasis, is the development of stones (calculi) within the renal collecting system or anywhere along the urinary tract. It represents a major health concern globally due to its increasing prevalence, high recurrence rate, and potential complications such as obstructive uropathy, infections, and renal impairment.

Recent decades have witnessed a surge in stone disease, attributed to changes in diet, sedentary behavior, climate, and metabolic disorders. The estimated lifetime prevalence ranges between 10% and 15%, with recurrence rates as high as 50% within five years. The economic burden of renal lithiasis includes not only diagnostic and medical management costs but also surgical procedures and lost productivity.

Kidney stones may present as incidental findings or with acute symptoms such as renal colic, hematuria, or urinary tract infections. Understanding the underlying risk factors is crucial to prevent recurrence and tailor therapeutic strategies, including the decision for surgical intervention.

DEVELOPMENT OF THE TOPIC

1. Metabolic Risk Factors

Metabolic disturbances are among the most important contributors to stone formation, especially in patients with recurrent lithiasis.

- **Hypercalciuria** is the most frequent metabolic abnormality, found in up to 50% of calcium stone formers. It may be idiopathic or secondary to conditions such as hyperparathyroidism or renal tubular defects. Increased urinary calcium enhances

the risk of calcium oxalate and calcium phosphate crystallization.

- **Hyperoxaluria**, either primary (genetic) or secondary (enteric), contributes to calcium oxalate stone formation. It is frequently observed in patients with chronic diarrhea, inflammatory bowel disease, or after intestinal surgery (e.g., Roux-en-Y gastric bypass).
- **Hypocitraturia** reduces urinary citrate levels, which normally inhibit crystal formation by binding to calcium. It is common in metabolic acidosis, high animal protein intake, and potassium depletion.
- **Hyperuricosuria** is associated with uric acid stones and may also promote calcium stone formation by serving as a nidus for crystallization. It is often seen in individuals with high purine intake or gout.

Routine metabolic evaluation with 24-hour urine analysis is recommended in patients with recurrent stones to identify and manage these risk factors.

2. Dietary and Fluid Intake Factors

Diet plays a critical role in stone formation. Certain dietary habits can either increase or reduce the risk of nephrolithiasis:

- **Low fluid intake** is one of the strongest modifiable risk factors. Insufficient hydration leads to low urine volume and high concentration of lithogenic substances, promoting stone formation. A urine output of at least 2.5 liters per day is recommended.
- **High sodium intake** increases calcium excretion in urine, favoring calcium stone formation. Excessive salt consumption is particularly harmful in patients with hypercalciuria.
- **Excess animal protein** (e.g., red meat) leads to increased acid load, decreased urinary citrate, and increased uric acid and calcium levels in urine.
- **High oxalate intake**, through foods like spinach, chocolate, nuts, and tea, contributes to calcium oxalate supersaturation.
- **Low calcium diets**, paradoxically, can increase the risk of oxalate stones by allowing more oxalate absorption in the gut. Balanced calcium intake from dietary sources is protective.
- **Citrate-rich foods**, particularly fruits like lemons and oranges, can help prevent stone formation by increasing urinary citrate levels.

3. Lifestyle and Environmental Factors

Modern sedentary lifestyles and environmental exposures play a significant role in the increasing incidence of kidney stones:

- **Obesity**, especially abdominal obesity, is independently associated with increased urinary

excretion of calcium, oxalate, and uric acid. Obesity also contributes to insulin resistance, which can lead to lower urine pH and uric acid stone formation.

- **Physical inactivity** contributes to bone demineralization and hypercalciuria, increasing stone risk.
- **Hot climates** and dehydration are known to raise the incidence of kidney stones. Individuals living in tropical or arid environments have a higher likelihood of stone formation due to reduced urine output.
- **Occupational factors**: Jobs that restrict fluid intake or access to restrooms, such as long-haul drivers, military personnel, or factory workers, are associated with increased risk.

4. Genetic and Familial Factors

Family history is a well-established risk factor for nephrolithiasis. Up to 40% of patients report a first-degree relative with kidney stones. Although most cases are polygenic, some inherited disorders are strongly associated with stone formation:

- **Cystinuria** is a rare autosomal recessive disorder characterized by impaired renal reabsorption of cystine, leading to recurrent cystine stones.
- **Primary hyperoxaluria** is a genetic defect in hepatic glyoxylate metabolism, resulting in excessive oxalate production and recurrent stones from early childhood.
- Several polymorphisms in genes involved in calcium, oxalate, and uric acid handling have been associated with increased stone risk.

5. Comorbidities and Medical Conditions

Several systemic diseases contribute to lithogenesis:

- **Type 2 diabetes mellitus** is associated with uric acid stones due to insulin resistance and acidic urinary pH.
- **Hypertension and metabolic syndrome** are frequently observed in stone formers and are thought to share pathophysiological pathways involving oxidative stress and endothelial dysfunction.
- **Gastrointestinal disorders** like Crohn's disease, ulcerative colitis, chronic diarrhea, and post-bariatric surgery states cause fat malabsorption, enhancing oxalate absorption and hyperoxaluria.
- **Renal tubular acidosis**, especially distal types, impairs urinary acidification and citrate excretion, favoring calcium phosphate stones.
- **Urinary tract infections**, especially with urease-producing bacteria (e.g., *Proteus*), can lead to struvite (infection-related) stones.



Figure 1. Kidney stones after surgery



Figure 2. Left kidney with 3 hyperdense images, the largest measuring 2.5 x 1 cm in the lower middle calyx, up to 805 HU (these were on admission), without pyelocalyceal ectasia.

6. Surgical Management of Renal Lithiasis

Although many cases of nephrolithiasis can be managed conservatively, surgical intervention becomes necessary in several scenarios, including:

- Stones causing obstruction and persistent pain not responsive to medical therapy.
- Recurrent urinary tract infections or hematuria.
- Large or staghorn calculi that are unlikely to pass spontaneously.
- Failed conservative management or failed previous surgical attempts.

- Anatomic anomalies that prevent normal stone clearance.

Current surgical options include:

- Extracorporeal Shock Wave Lithotripsy (ESWL): Non-invasive option best suited for stones <2 cm located in the kidney or upper ureter. It fragments stones using high-energy shock waves.
- Ureteroscopy (URS): A minimally invasive endoscopic technique used for stones in the ureter or intrarenal pelvis. Holmium laser lithotripsy is often employed for fragmentation.

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- Percutaneous Nephrolithotomy (PCNL): The preferred method for large, complex, or staghorn stones. It involves percutaneous access to the renal collecting system for stone removal and is considered the gold standard in such cases.
- Laparoscopic or robotic-assisted stone surgery: Reserved for rare cases where endoscopic or percutaneous methods fail or are contraindicated, often due to anatomic anomalies or combined surgical needs.
- Open surgery: Rarely performed in the modern era but still indicated in complex or recurrent cases not amenable to minimally invasive approaches.

Selection of the appropriate modality depends on stone size, location, composition, patient anatomy, comorbidities, and available resources. Surgical management, when combined with metabolic evaluation and lifestyle counseling, significantly reduces recurrence rates and improves quality of life.

CONCLUSIONS

Renal lithiasis is a complex and multifactorial condition involving a combination of metabolic, dietary, environmental, genetic, and systemic risk factors. Identifying and addressing these contributors is essential to prevent recurrence and reduce long-term morbidity.

While conservative management focusing on hydration, dietary changes, and metabolic correction remains the cornerstone of prevention, surgical intervention plays a pivotal role in symptomatic or complicated cases. The advent of minimally invasive techniques has revolutionized the management of kidney stones, offering safe and effective options with rapid recovery.

A comprehensive, patient-centered approach—combining preventive strategies, individualized risk assessment, and timely surgical management—is essential to optimize outcomes and reduce the burden of renal lithiasis in both primary and recurrent stone formers.

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