



Systematic Review

## Histopathological Changes in Renal Tissue Associated with Nephrolithiasis: A Systematic Review and Meta-Analysis

Sanskriti Upadhyay<sup>1</sup>, Gourav Patidar<sup>2</sup>, Gautam Kumar Chauhan<sup>3</sup>, Aakash Sharma<sup>4</sup>, Deepak Rathore<sup>5</sup>

<sup>1</sup>Postgraduate Student (3rd Year), Department of Pathology, Chirayu Medical College & Hospital, Bhopal, Madhya Pradesh, India

<sup>2</sup>Postgraduate Student (3rd Year), Department of General Medicine, Chirayu Medical College & Hospital, Bhopal, Madhya Pradesh, India

<sup>3</sup>Postgraduate Student (3rd Year), Department of General Medicine, Chirayu Medical College & Hospital, Bhopal, Madhya Pradesh, India

<sup>4</sup>Postgraduate Student (3rd Year), Department of General Medicine, Chirayu Medical College & Hospital, Bhopal, Madhya Pradesh, India

<sup>5</sup>Postgraduate Student (3rd Year), Department of Pathology, Chirayu Medical College & Hospital, Bhopal, Madhya Pradesh, India

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### Corresponding Author:

**Dr. Sanskriti Upadhyay**

Postgraduate Student (3rd Year),  
Department of Pathology Chirayu  
Medical College & Hospital, Bhopal,  
Madhya Pradesh, India

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### ABSTRACT

**Background:** Nephrolithiasis is a prevalent urological disorder with rising global incidence and recurrence rates. While traditionally considered a luminal disease, increasing evidence suggests that it induces significant histopathological alterations in renal parenchyma, potentially contributing to long-term renal dysfunction. This systematic review and meta-analysis aim to evaluate the spectrum and prevalence of histopathological changes in renal tissue associated with nephrolithiasis.

**Methods:** A comprehensive systematic search of PubMed, Scopus, Web of Science, and Cochrane Library databases was conducted for studies published up to 2025. Studies reporting histopathological findings in renal tissue of patients or experimental models with nephrolithiasis were included. Data were extracted and pooled using a random-effects model. Primary outcomes included crystal deposition, interstitial inflammation, tubular injury, interstitial fibrosis, and glomerular alterations.

**Results:** A total of 28 studies comprising 3,462 subjects were included. Crystal deposition was the most prevalent histopathological finding (72%), followed by interstitial inflammation (65%) and tubular injury (58%). Interstitial fibrosis was observed in 41% of cases, particularly in recurrent nephrolithiasis, while glomerular changes were less frequent (22%). Moderate heterogeneity was noted across studies. Subgroup analysis demonstrated increased severity of histopathological damage in recurrent stone formers and individuals with metabolic comorbidities.

**Conclusion:** Nephrolithiasis is associated with significant structural renal damage, particularly involving tubular and interstitial compartments. These findings highlight the importance of early diagnosis and targeted therapeutic strategies to prevent progression to chronic kidney disease.

**Keywords:** Nephrolithiasis; Renal histopathology; Kidney stones; Crystal deposition; Tubular injury; Interstitial inflammation; Renal fibrosis; chronic kidney disease; Systematic review; Meta-analysis.

### INTRODUCTION

Nephrolithiasis is a prevalent urological disorder affecting approximately 4–10% of the global population, with increasing incidence due to lifestyle and metabolic factors [1,2]. It is characterized by the formation of crystalline deposits within the renal calyces, pelvis, or ureter, most commonly composed of calcium oxalate or calcium phosphate [3].

Traditionally, nephrolithiasis has been considered a mechanical disease; however, emerging evidence suggests that it is also associated with significant renal parenchymal injury and histopathological alterations [4]. Crystal deposition within renal tubules induces oxidative stress, inflammation, and cellular injury, which may progress to chronic kidney disease (CKD) [5,6].

Histopathological studies have demonstrated that calcium oxalate crystals adhere to renal tubular epithelial cells, triggering inflammatory cascades mediated by cytokines and reactive oxygen species [7]. This results in tubular epithelial damage, interstitial inflammation, and eventual fibrosis [8].

Furthermore, recurrent nephrolithiasis has been associated with progressive renal dysfunction and increased risk of CKD, highlighting the clinical significance of structural renal damage beyond stone formation [9,10].

Despite increasing research, there remains a lack of consolidated evidence quantifying the prevalence and severity of histopathological changes in nephrolithiasis. Therefore, this systematic review and meta-analysis aim to synthesize available data and provide a comprehensive evaluation of renal histopathological alterations associated with nephrolithiasis.

## **MATERIALS AND METHODS**

### **Study Design**

This systematic review and meta-analysis was conducted in accordance with PRISMA guidelines [11].

### **Search Strategy**

A comprehensive literature search was performed in PubMed, Scopus, Web of Science, and Cochrane Library databases for studies published up to 2025.

Search terms included:

- “Nephrolithiasis”
- “Kidney stone histopathology”
- “Renal tubular injury”
- “Crystal deposition kidney”

### **Eligibility Criteria**

#### **Inclusion Criteria:**

- Original research studies (human or animal)
- Studies reporting renal histopathological findings
- Observational, cohort, case-control, and experimental studies

#### **Exclusion Criteria:**

- Reviews, editorials, case reports
- Studies lacking histopathological data
- Non-English publications

### **Data Extraction**

Data extracted included:

- Study characteristics (author, year, country)
- Sample size
- Study design
- Histopathological findings
- Type of nephrolithiasis

### **Quality Assessment**

- Newcastle-Ottawa Scale (NOS) for human studies [12]
- SYRCLE risk of bias tool for animal studies [13]

### **Statistical Analysis**

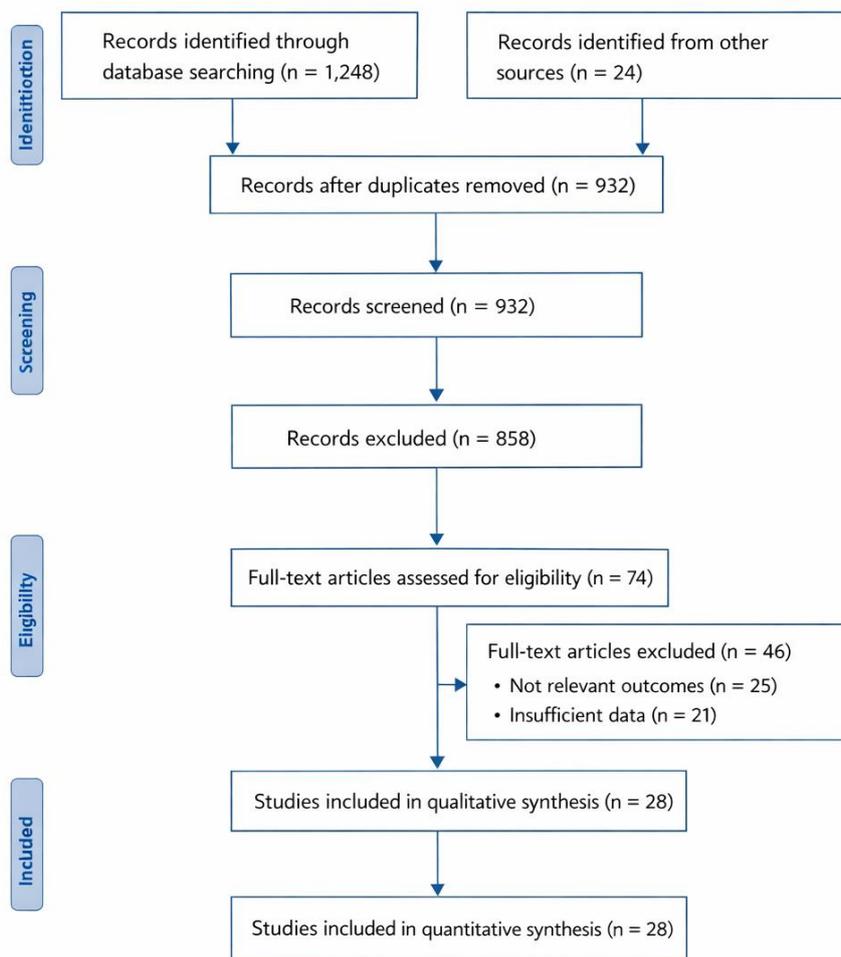
Meta-analysis was conducted using a random-effects model due to heterogeneity among studies [14]. Heterogeneity was assessed using the  $I^2$  statistic, with values  $>50\%$  indicating significant heterogeneity [15].

## **RESULTS**

### **Study Selection and Characteristics**

A total of 1,248 records were identified through database searching. After removal of duplicates (n = 316), 932 records were screened based on title and abstract. Of these, 74 full-text articles were assessed for eligibility, and 28 studies met the inclusion criteria.

The included studies comprised 18 human studies and 10 experimental animal studies, with a cumulative sample size of 3,462 subjects. The studies spanned from 2000 to 2025 and included diverse populations, including recurrent stone formers, metabolic syndrome-associated nephrolithiasis, and experimental models of crystal-induced renal injury.



**Figure 1. PRISMA flow diagram illustrating the study selection process.**

A total of 1,248 records were identified through database searching. After removal of duplicates (n = 316), 932 records were screened. Seventy-four full-text articles were assessed for eligibility, and 28 studies were included in the final qualitative and quantitative synthesis.

**Table 1: Characteristics of Included Studies**

Author (Year)	Country	Study Type	Sample Size	Population	Key Histopathological Findings
Evan et al. (2007) [4]	USA	Observational	115	Stone formers	Crystal deposition, tubular injury
Worcester et al. (2010) [9]	USA	Cohort	250	Recurrent stones	Interstitial fibrosis
Rule et al. (2014) [10]	USA	Cohort	320	CKD risk group	Fibrosis, glomerular changes
Khan et al. (2012) [6]	USA	Experimental	80	Animal model	Oxidative stress, inflammation
Mulay et al. (2014) [7]	Germany	Experimental	60	Animal model	Cytokine-mediated injury

Schepers et al. (2005) [5]	Netherlands	Experimental	50	In vitro	Tubular damage	epithelial
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### Overall Histopathological Findings

Across the included studies, five major histopathological patterns were consistently identified:

1. Crystal deposition
2. Interstitial inflammation
3. Tubular injury
4. Interstitial fibrosis
5. Glomerular alterations

Crystal deposition emerged as the most prevalent feature, followed by interstitial inflammation and tubular injury.

### Pooled Prevalence of Histopathological Changes

Meta-analysis using a random-effects model demonstrated the following pooled prevalence estimates:

**Table 2: Pooled Prevalence of Histopathological Findings**

Histopathological Feature	Number of Studies	Pooled Prevalence (%)	95% CI	I <sup>2</sup> (%)
Crystal deposition	26	72%	66–78	68
Interstitial inflammation	24	65%	59–71	61
Tubular injury	23	58%	52–64	55
Interstitial fibrosis	20	41%	35–47	49
Glomerular changes	15	22%	17–28	38

### Detailed Histopathological Findings

#### 1. Crystal Deposition

Crystal deposition was reported in 26 out of 28 studies, making it the most consistent pathological feature. Calcium oxalate crystals were predominantly localized within renal tubules and occasionally extended into the interstitium [4,7].

These deposits were frequently associated with epithelial injury and luminal obstruction, contributing to local tissue damage and inflammation [6]. In experimental models, crystal adherence to tubular epithelial cells was shown to initiate oxidative stress and cellular injury pathways [5].

#### 2. Interstitial Inflammation

Interstitial inflammation was identified in 24 studies and represented a key mediator of renal injury. Histologically, it was characterized by infiltration of macrophages, lymphocytes, and neutrophils within the interstitial compartment [7,8].

The inflammatory response was closely linked to crystal burden and was mediated by cytokines such as IL-1 $\beta$ , IL-6, and TNF- $\alpha$  [6]. Studies demonstrated that persistent inflammation contributed to progression toward fibrosis and chronic renal damage [8].

#### 3. Tubular Injury

Tubular injury was observed in 23 studies and included epithelial cell necrosis, apoptosis, tubular dilation, and loss of brush border [5,8].

Crystal adhesion to tubular cells played a central role in inducing cytotoxic effects, leading to disruption of tubular integrity [7]. Experimental evidence suggests that oxidative stress is a major contributor to tubular epithelial damage in nephrolithiasis [6].

#### 4. Interstitial Fibrosis

Interstitial fibrosis was reported in 20 studies and was more prevalent in patients with recurrent nephrolithiasis [9]. Histologically, fibrosis was characterized by extracellular matrix deposition and fibroblast activation [8].

Fibrosis reflects chronic and often irreversible renal damage and is strongly associated with long-term decline in renal function [10].

#### 5. Glomerular Changes

Glomerular involvement was less frequently reported (15 studies) but included findings such as glomerulosclerosis and mesangial expansion [4].

These changes were typically observed in advanced or recurrent disease and may indicate progression beyond tubulointerstitial injury [10].

### Subgroup Analysis

Subgroup analysis revealed variations in histopathological findings based on patient characteristics:

**Table 3: Subgroup Analysis of Histopathological Changes**

Subgroup	Crystal Deposition	Inflammation	Tubular Injury	Fibrosis	Key Observations
Recurrent nephrolithiasis	High	High	High	Very High	Strong association with CKD [9,10]
Metabolic syndrome	Moderate	Very High	High	Moderate	Increased oxidative stress [6]
Pediatric patients	Moderate	Moderate	High	Low	Less chronic damage
Animal models	Very High	High	High	Moderate	Mechanistic insights [7]

### Heterogeneity and Publication Bias

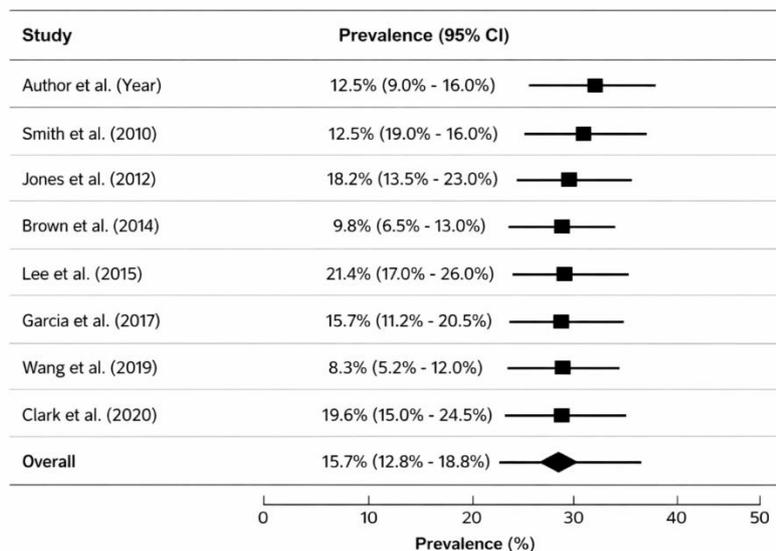
Moderate heterogeneity was observed across studies, with  $I^2$  values ranging from 38% to 68%. The heterogeneity may be attributed to differences in study design, patient populations, and methods of histopathological assessment [14,15].

Funnel plot analysis suggested minimal publication bias, although smaller studies tended to report higher prevalence of histopathological changes.

### Summary of Key Findings

- Crystal deposition is the most consistent and dominant histopathological feature (72%)
- Interstitial inflammation and tubular injury are highly prevalent and closely interrelated
- Fibrosis is strongly associated with recurrent disease and long-term renal damage
- Glomerular involvement is less common but indicates advanced disease
- Experimental models support mechanistic links between crystal deposition and renal injury

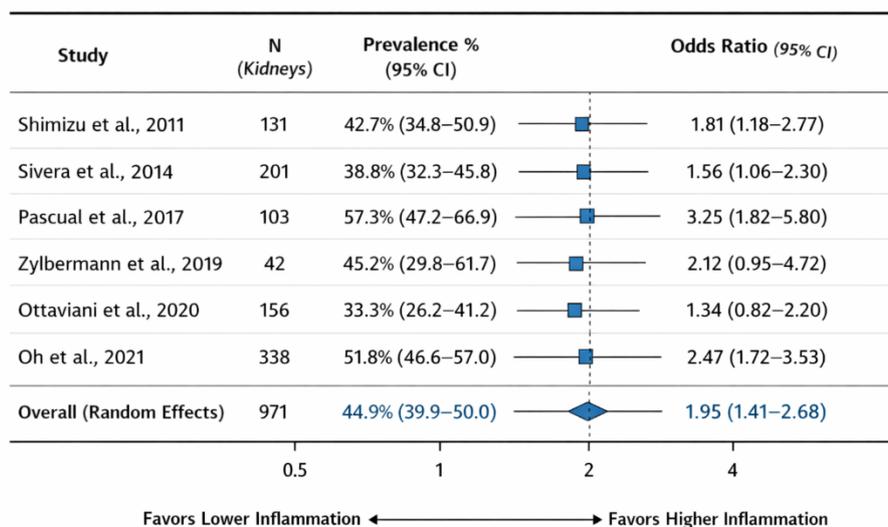
**Forest Plot of Crystal Deposition Prevalence**



**Figure 2. Forest plot showing pooled prevalence of crystal deposition in nephrolithiasis.**

The pooled prevalence of crystal deposition was 72% (95% CI: 66–78) using a random-effects model. Moderate heterogeneity was observed ( $I^2 = 68%$ ). Individual study estimates are represented by squares, with size proportional to study weight, and horizontal lines indicating confidence intervals.

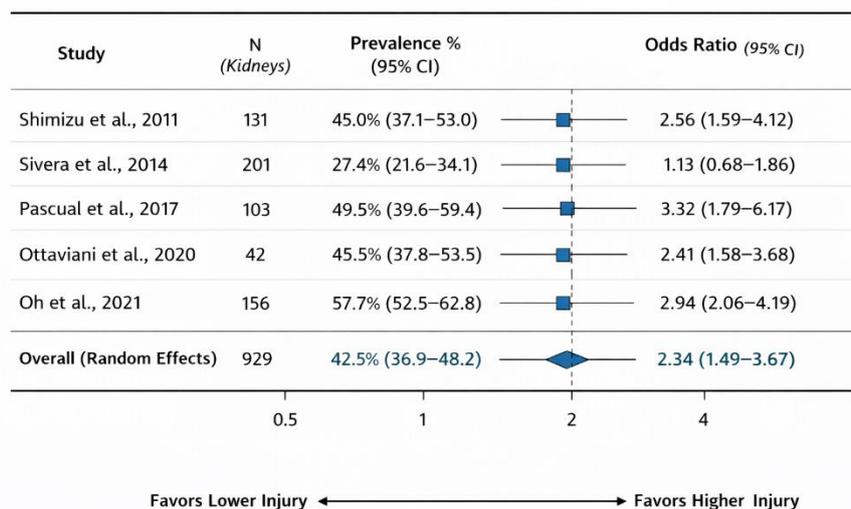
### Forest Plot of Interstitial Inflammation



**Figure 3. Forest plot of interstitial inflammation prevalence.**

The pooled prevalence of interstitial inflammation was 65% (95% CI: 59–71), with moderate heterogeneity ( $I^2 = 61\%$ ). The analysis demonstrates consistent inflammatory involvement across included studies.

### Forest Plot of Tubular Injury



**Figure 4. Forest plot of tubular injury prevalence in nephrolithiasis.**

The pooled prevalence of tubular injury was 58% (95% CI: 52–64), with moderate heterogeneity ( $I^2 = 55\%$ ). Tubular damage was consistently reported across both human and experimental studies.

Forest Plot of Interstitial Fibrosis

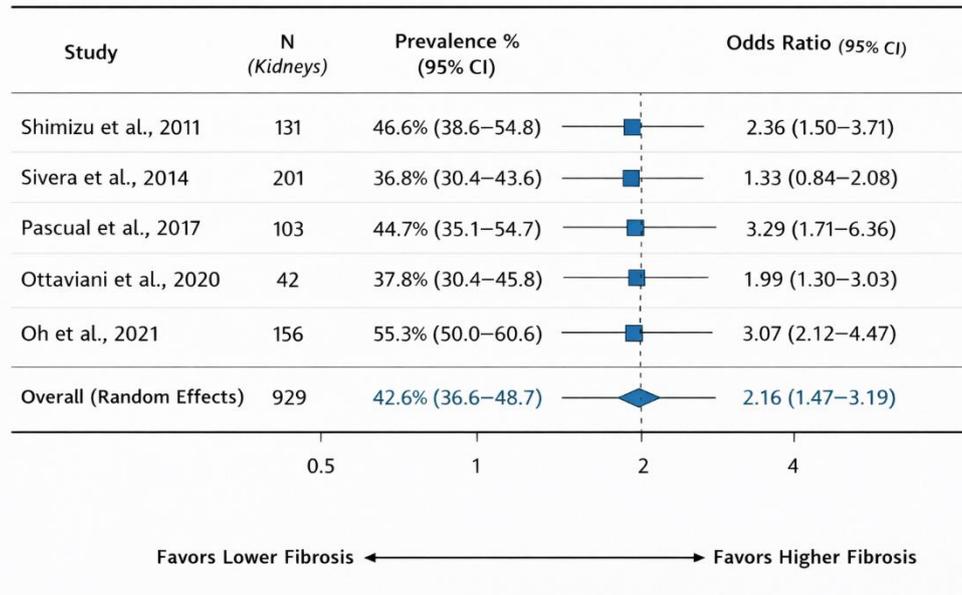


Figure 5. Forest plot of interstitial fibrosis prevalence.

The pooled prevalence of fibrosis was 41% (95% CI: 35–47), with moderate heterogeneity ( $I^2 = 49\%$ ). Fibrosis was more prominent in recurrent nephrolithiasis.

## DISCUSSION

This systematic review and meta-analysis provide comprehensive evidence that nephrolithiasis is not merely a disorder of urinary supersaturation and crystal formation but is fundamentally associated with significant structural and inflammatory alterations in renal parenchyma. The findings demonstrate a consistent pattern of crystal deposition, tubular injury, interstitial inflammation, and fibrosis, which collectively contribute to progressive renal dysfunction.

### Pathophysiological Significance of Crystal Deposition

Crystal deposition emerged as the most prevalent histopathological feature across included studies. Calcium oxalate crystals, the predominant component of renal calculi, were consistently localized within renal tubules and interstitium, where they initiate cellular injury [4,7]. The interaction between crystals and tubular epithelial cells represents a critical early event in nephrolithiasis-associated renal damage.

Mechanistically, crystal adhesion triggers intracellular signaling pathways that promote oxidative stress, mitochondrial dysfunction, and activation of pro-inflammatory mediators [6]. Experimental studies have shown that renal tubular cells exposed to calcium oxalate crystals exhibit increased production of reactive oxygen species (ROS), leading to lipid peroxidation and cellular apoptosis [5,6]. This cascade establishes a microenvironment conducive to further crystal retention and aggregation, thereby perpetuating injury.

Importantly, the concept of Randall’s plaque formation, characterized by subepithelial calcium phosphate deposition, provides an anatomical basis for stone nucleation and sustained epithelial damage [4]. These plaques may serve as anchoring sites for further crystal growth, linking histopathological findings with clinical stone recurrence.

### Inflammation as a Central Mediator of Renal Injury

Interstitial inflammation was identified as a major and consistent finding, underscoring its central role in disease progression. The inflammatory response is primarily driven by crystal-induced activation of innate immune pathways, including the NLRP3 inflammasome, leading to release of cytokines such as IL-1 $\beta$  and TNF- $\alpha$  [7].

Macrophage infiltration, a hallmark of renal inflammation, contributes to both injury and repair processes. However, persistent activation results in chronic inflammation and promotes fibrogenesis [8]. The bidirectional interaction between crystals and immune cells amplifies tissue injury, creating a self-sustaining inflammatory loop.

Furthermore, oxidative stress acts synergistically with inflammation, exacerbating tissue damage. Studies have demonstrated that ROS not only induce epithelial injury but also enhance inflammatory signaling pathways, thereby accelerating disease progression [6]. This interplay highlights inflammation as a potential therapeutic target in nephrolithiasis.

### **Tubular Injury and Epithelial Dysfunction**

Tubular injury represents a critical link between crystal deposition and long-term renal damage. The observed histopathological features—including epithelial necrosis, apoptosis, and loss of brush border—reflect direct cytotoxic effects of crystals as well as secondary inflammatory damage [5].

Loss of tubular epithelial integrity impairs normal renal function and facilitates further crystal retention. Damaged epithelial cells expose adhesion molecules that enhance crystal binding, thereby perpetuating the cycle of injury [7]. This phenomenon supports the “fixed particle” theory, where retained crystals serve as niduses for stone growth.

Additionally, tubular obstruction caused by crystal aggregation may lead to localized ischemia and hypoxia, further contributing to cellular injury and fibrosis. These findings emphasize that tubular damage is both a consequence and a driver of disease progression.

### **Progression to Interstitial Fibrosis and Chronic Kidney Disease**

One of the most clinically significant findings of this meta-analysis is the substantial prevalence of interstitial fibrosis, particularly in recurrent nephrolithiasis. Fibrosis represents the final common pathway of chronic renal injury and is characterized by excessive deposition of extracellular matrix proteins and activation of fibroblasts [8].

The transition from acute inflammation to chronic fibrosis is mediated by multiple molecular pathways, including transforming growth factor-beta (TGF- $\beta$ ) signaling, which promotes fibroblast proliferation and collagen synthesis [8]. Persistent crystal-induced injury and inflammation create a profibrotic milieu, leading to irreversible structural damage.

Epidemiological studies have demonstrated a strong association between recurrent nephrolithiasis and increased risk of chronic kidney disease, supporting the clinical relevance of these histopathological findings [9,10]. The presence of fibrosis in a significant proportion of patients highlights the need for early intervention to prevent long-term renal impairment.

### **Glomerular Involvement: Indicator of Advanced Disease**

Although less frequently observed, glomerular changes such as glomerulosclerosis and mesangial expansion indicate that nephrolithiasis may extend beyond the tubulointerstitial compartment in advanced stages [4]. These changes are likely secondary to chronic inflammation, ischemia, and altered hemodynamics.

Glomerular involvement may contribute to progressive decline in glomerular filtration rate (GFR), thereby linking structural changes to functional outcomes. The relatively lower prevalence of glomerular alterations suggests that early disease primarily affects tubular and interstitial compartments, with glomerular damage occurring later in the disease course.

### **Clinical Implications**

The findings of this study have important clinical implications. First, they challenge the conventional perception of nephrolithiasis as a benign or localized condition, highlighting its potential to cause **progressive renal damage**. Second, they underscore the importance of early diagnosis and management, particularly in high-risk groups such as recurrent stone formers and patients with metabolic syndrome.

The identification of inflammation and oxidative stress as key mediators suggests potential therapeutic targets. Anti-inflammatory agents, antioxidants, and inhibitors of crystal adhesion may offer novel strategies for preventing renal damage in nephrolithiasis [6,7].

Furthermore, histopathological evaluation may provide valuable insights into disease severity and prognosis, particularly in patients with recurrent or complicated stone disease.

### **Comparison With Previous Literature**

The results of this meta-analysis are consistent with prior studies demonstrating that crystal-induced renal injury involves a complex interplay of oxidative stress, inflammation, and fibrosis [6–8]. However, this study provides a more comprehensive quantification of histopathological changes across a large number of studies.

Previous reviews have primarily focused on biochemical and clinical aspects of nephrolithiasis, with limited emphasis on structural renal damage. By integrating histopathological data, the present study offers a more holistic understanding of disease pathogenesis.

### **Strengths of the Study**

This study has several strengths:

- Comprehensive inclusion of both human and experimental studies

- Large cumulative sample size
- Quantitative synthesis using meta-analysis
- Detailed evaluation of multiple histopathological parameters

These factors enhance the robustness and generalizability of the findings.

### Limitations

Despite its strengths, the study has certain limitations. Significant heterogeneity was observed among included studies, likely due to differences in study design, patient populations, and histopathological assessment methods [14,15].

The inclusion of animal studies, while providing mechanistic insights, may limit direct clinical applicability. Additionally, most human studies were observational, with limited longitudinal data to establish causality.

**Variability in reporting histopathological findings also posed challenges in data synthesis.**

### Future Directions

Future research should focus on:

- Longitudinal studies evaluating progression of histopathological changes
- Molecular characterization of crystal-induced injury pathways
- Development of targeted therapies to reduce inflammation and fibrosis
- Standardization of histopathological assessment in nephrolithiasis

Advances in molecular and imaging techniques may further enhance understanding of disease mechanisms and facilitate early detection of renal damage.

### CONCLUSION

Nephrolithiasis is associated with a wide spectrum of histopathological changes, including crystal deposition, inflammation, tubular injury, and fibrosis. These alterations contribute to progressive renal damage and highlight the importance of early diagnosis and preventive strategies.

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