Review Article

Interactions between Gout and Chronic Kidney Disease: A Systematic Review

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ABSTRACT

Despite the recognized clinical overlap between gout and chronic kidney disease (CKD), a comprehensive synthesis of the evidence regarding their intricate relationship, encompassing disease prevalence, risk of progression, and associated outcomes, remains crucial. This systematic review aims to synthesize existing evidence on the association between gout and CKD, focusing on these key aspects. A comprehensive search of four databases led to the discovery of 816 relevant publications. After eliminating duplicates and assessing each article for relevance, 51 full-text articles were examined, and ultimately, 9 studies were selected based on the inclusion criteria. Nine studies were included, with a total of 190,760 gout patients and 133,015 (69.7%) were males. The prevalence of CKD among gout patients ranged from 16.2% to 84.1%. Adjusted hazard ratios for incident or progressive CKD ranged from 1.28 to 3.05, confirming a significantly increased risk in gout patients. Elevated serum uric acid levels were associated with worse renal function, while achieving target urate levels (<6 mg/dL) was linked to a reduced risk of end-stage kidney disease. Patients with both gout and CKD had higher healthcare utilization and increased mortality. Gout appears to be a clinically relevant risk factor for the development and progression of CKD. Routine renal screening and effective urate-lowering therapy may play a critical role in mitigating renal outcomes and improving the overall prognosis of this high-risk patient population.

Keywords: Gout; Chronic kidney disease; Renal disease; Serum uric acid; systematic review.

Introduction

High blood uric acid levels, or hyperuricemia, are the main cause of gout, a type of inflammatory arthritis marked by the buildup of monosodium urate crystals in soft tissues and joints [1]. Conversely, chronic kidney disease (CKD) is characterised by a gradual decline in kidney function over time, impacting the kidneys' capacity to filter waste, control electrolytes, and preserve acid-base balance. [2]. About 10% of people worldwide suffer from chronic kidney disease (CKD), which is defined by a progressive decrease of renal function and is linked to an elevated risk of cardiovascular disease and death [2]. Recent epidemiological studies suggest that there is a bidirectional relationship between gout and CKD. The relationship between gout and CKD is bidirectional: hyperuricemia and gout contribute to kidney function decline, while CKD exacerbates hyperuricemia by

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reducing uric acid excretion [3]. Individuals with gout are at increased risk of developing CKD, primarily due to longstanding hyperuricemia that may promote renal interstitial inflammation and fibrosis, leading to nephron damage [4]. Conversely, patients suffering from CKD are more likely to experience flares of gout due to impaired renal clearance of uric acid, thereby setting up a vicious cycle where each condition exacerbates the other [5]. A more profound understanding of this bidirectional relationship is therefore imperative for informing public health strategies, guiding clinical management protocols, and ultimately improving patient outcomes in this complex comorbidity. In clinical practice, the management of patients suffering from both gout and CKD poses a unique set of challenges. Traditional urate-lowering therapies, such as allopurinol, have established efficacy for treating gout; however, their use must be carefully monitored in patients with compromised kidney function due to the risk of adverse effects and the potential for drug accumulation. As current treatment paradigms evolve, there is a pressing need for a comprehensive understanding of how best to manage these concurrent conditions safely and effectively [6]. Furthermore, the socio-economic implications of gout and CKD extend beyond individual patient management. A more comprehensive approach to research that looks at public health policies, educational programs, and preventive measures is required due to the strain on healthcare systems, which includes higher healthcare expenses and a lower quality of life for people impacted. There is also a need for clinical trials specifically targeting populations with both gout and CKD; such investigations could provide insights into optimal therapeutic strategies and potential novel treatment options [7, 8]. Gout and CKD are two prevalent conditions that frequently coexist, leading to a complex interplay that can significantly impact patient health outcomes. Uric acid, a key player in gout pathophysiology, can exacerbate renal dysfunction, while CKD may impair uric acid excretion, creating a vicious cycle that complicates management strategies. The aim of this study is to assess and summarize current evidence on association between gout and CKD.

Methods

To maintain scientific rigour and openness, this systematic review complied with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria [9]. The study aimed to synthesize existing evidence on the bidirectional relationship between gout and CKD, evaluating pathophysiological mechanisms, clinical associations, and therapeutic challenges.

Search Strategy: To find pertinent English-language research on the connections between gout and chronic kidney disease (CKD), a thorough electronic search was carried out across several databases, including PubMed, Web of Science, Scopus, and Embase. The search approach made use of keywords and Medical Subject Headings (MeSH) terminology associated with:

• Gout (e.g., "hyperuricemia," "monosodium urate crystals," "gouty arthritis")

• CKD (e.g., "renal impairment," "CKD progression," "nephropathy")

• Associated outcomes (e.g., "urate-lowering therapy," "renal function decline," "inflammation," "comorbidity")

The search was narrowed down using boolean operators (AND, OR), and the reference lists of the included research were manually examined for other pertinent articles.

Study Selection and Eligibility Criteria: Two independent reviewers screened the search results, assessed study eligibility, extracted data, and evaluated study quality using standardized tools. Discrepancies were resolved through discussion or consultation with a third reviewer.

Inclusion Criteria:

• Studies investigating adult populations (≥18 years) with gout and/or CKD

• Assessment of hyperuricemia, gout flares, or CKD progression using validated measures (e.g., serum urate levels, eGFR, CKD staging)

• Outcomes including disease progression, renal function decline, treatment efficacy, or biomarker correlations

• Study designs: Observational studies (cohort, case-control, cross-sectional) and clinical trials published in English

Exclusion Criteria:

• Studies not reporting on gout-CKD interactions.

• Animal studies, reviews, editorials, case reports (<10 subjects), or conference abstracts.

• Studies lacking appropriate control groups or those combining gout/CKD with other metabolic disorders without separate analysis.

Data Extraction: The screening process was managed using Rayyan (QCRI) [10] to enhance transparency and minimize bias. After the initial title/abstract screening, full-text articles of eligible studies were reviewed. A standardized extraction form collected study characteristics (author, year, country, design), population details (sample size, gout/CKD severity, urate-lowering therapy use), key outcomes (renal

function decline, gout flare frequency, treatment responses), and statistical findings (adjusted risk estimates, hazard ratios, p-values).

Risk of Bias Assessment: The listed studies' methodological quality was carefully assessed. The Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) tool was used to evaluate non-randomized studies [11].

Results

The search process initially identified 816 publications (Figure 1). After removing 452 duplicates, 364 trials were screened based on their titles and abstracts. Of these, 311 did not meet the eligibility criteria, leaving 51 full-text articles for in-depth evaluation. In the end, 9 studies met the inclusion criteria and were selected for evidence synthesis and analysis. Sociodemographic and clinical outcomes: Nine studies were included, with a total of 190,760 gout patients and 133,015 (69.7%) were males. The included studies comprised eight retrospective cohort studies [12, 13, 15-20], and one cross-sectional study [14]. Three studies were conducted in the UK [13, 18, 19], two in Nigeria [14, 16], one in the USA [12], one in China [15], one in Israel [17], and one in Turkey [20] (Table 1). The studies collectively in (Table 2) demonstrate a strong association between gout and CKD, with varying prevalence rates and clinical outcomes across different cohorts. In one large retrospective cohort, gout was associated with a threefold increased risk of developing CKD, even after adjusting for various comorbidities. CKD was present in 23.5% of the study population, supporting the role of gout as an independent risk factor for renal impairment [12]. Another cohort study found that individuals with gout had a significantly higher incidence of CKD stage ≥3 compared to those without gout (28.6 vs. 15.8 per 10,000 person-years), with an adjusted hazard ratio of 1.78 and CKD prevalence of 16.2% [13]. In a cross-sectional analysis, 27.4% of gout patients were identified with CKD. These patients were significantly older and more likely to have proteinuria, along with lower BMI and packed cell volume, suggesting more advanced disease compared to those without CKD [14]. Another retrospective cohort reported a CKD prevalence of 41.4% among gout patients and showed that achieving a target serum urate level (<6 mg/dL) led to a modest but statistically significant reduction in the 5-year risk of severe or end-stage kidney disease (10.32% vs. 12.73%) [15]. One study reported a particularly high CKD prevalence of 84.1% among gout patients. Higher serum uric acid levels were associated with worse renal function, showing a positive correlation with serum creatinine and a negative correlation with eGFR [16]. Similarly, another cohort revealed a 35.9% CKD prevalence in newly diagnosed gout patients, where the presence of CKD was linked to higher hospital admissions, increased medical visits, greater urate-lowering therapy use, and higher 5-year all-cause mortality [17]. Furthermore, another study showed an incidence rate of advanced CKD of 8.54 per 1000 patient-years in gout patients, nearly double that of controls. The adjusted hazard ratio for advanced CKD was 1.29, with CKD prevalence at 16.3%[18]. Consistent findings in another cohort showed a 78% increased risk of incident CKD in gout patients (adjusted HR 1.78), though the effect of urate-lowering therapy was attenuated after adjustment, except in a subgroup of women over a 3-year period [19]. Lastly, in another study, 39.5% of patients had CKD stages 3–5, and 33.3% were in stage 2, indicating a substantial renal burden among individuals with gout [20].

Discussion

The collective findings from this review affirm a robust and consistent association between gout and CKD. Across a range of retrospective cohort and cross-sectional studies, the prevalence of CKD among patients with gout varied widely, from approximately 16% to over 80%, depending on population characteristics and study settings. This substantial burden supports the notion that gout is not only a coexisting condition but also a potential contributor to CKD development and progression. Several studies reported significantly elevated hazard ratios for incident CKD in gout patients, even after adjusting for common comorbidities such as hypertension, diabetes mellitus, and cardiovascular disease. These results point toward an independent pathogenic link, likely mediated by sustained hyperuricemia, chronic inflammation, and urate crystal deposition affecting renal structures. In a systematic review and meta-analysis of observational studies by Roughley et al., found that individuals with gout should undergo routine screening for CKD, and healthcare providers should be informed about the established links between gout, CKD, and nephrolithiasis. CKD often advances silently, without noticeable symptoms, and may go undetected unless actively investigated—typically only becoming apparent in its later stages [21]. Earlier epidemiological studies have demonstrated that CKD is an independent risk factor for gout [22]. However, there are several plausible biological pathways through which gout itself may

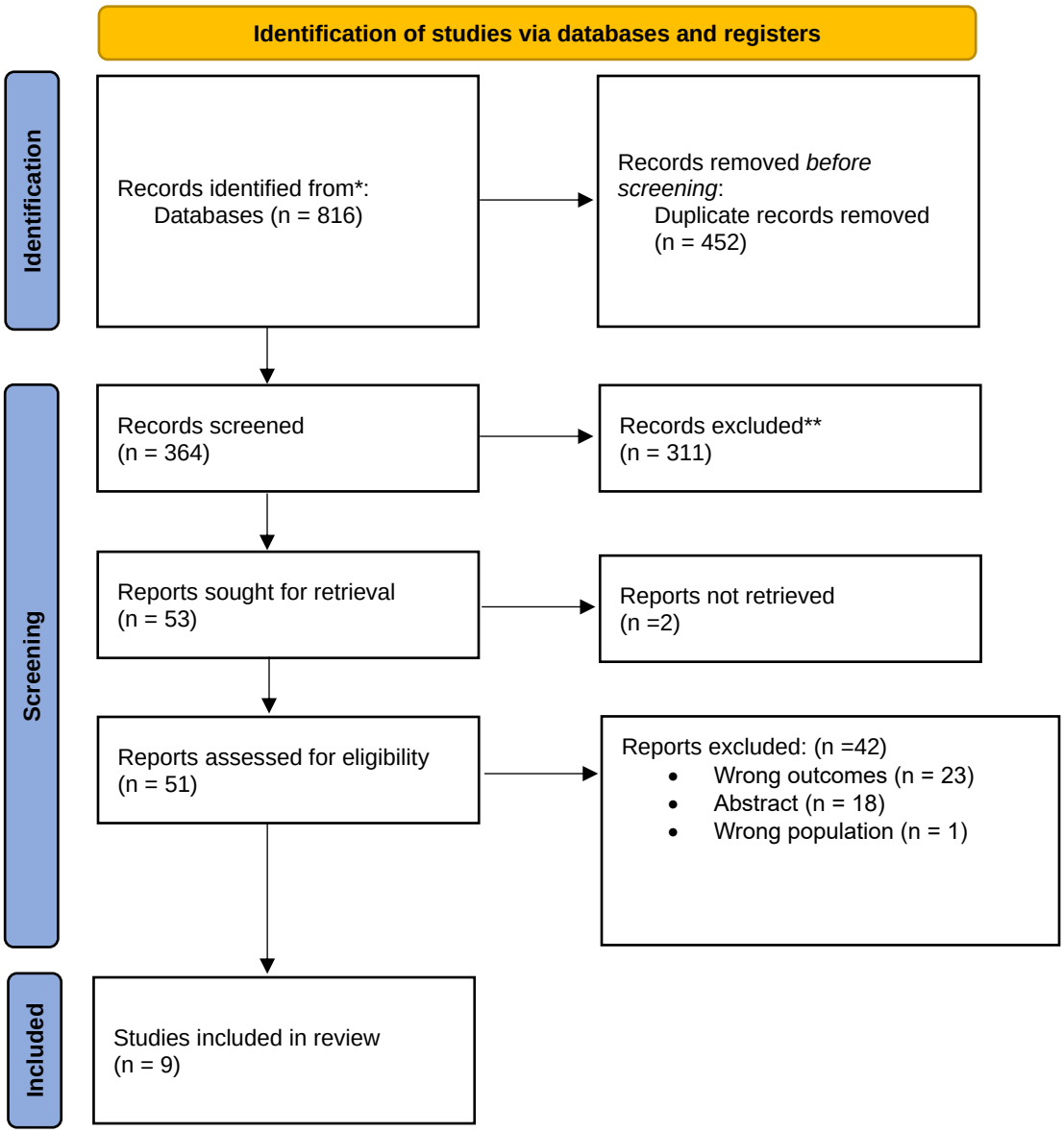

Figure (1): Search summary illustrated in PRISMA flowchart.

Table (1): Summary of demographic from the included studies.

Study ID	Country	Study design	Sociodemographic	CKD (%)	Main outcomes
Singh et al., 2019 [12]	USA	Retrospective cohort	Cases: 76,309 Age: >65 Males: 44,740 (58%)	17,903 (23.5%)	Multivariable-adjusted analyses showed that gout was associated with a threefold increased risk of developing CKD (HR ~3.0), even after accounting for various comorbidities and performing sensitivity analyses. This consistent association highlights gout as an independent risk factor for CKD.
Roughley et al., 2018 [13]	UK	Retrospective cohort	Cases: 41,446 Mean age: 57.2 Males: 33,574 (81%)	6694 (16.2%)	Individuals with gout had a significantly higher risk of developing CKD stage ≥3 compared to those without gout (28.6 vs. 15.8 per 10,000 person-years), with an adjusted hazard ratio of 1.78 (95% CI 1.70–1.85).
Akpabio et al., 2018 [14]	Nigeria	Cross-sectional	Cases: 106 Mean age: 56.5 Males: 94 (88.7%)	29 (27.4%)	Gout patients with CKD were significantly older, more likely to have proteinuria, and had lower BMI and packed cell volume (PCV) compared to those without CKD.
Wang et al., 2025 [15]	China	Retrospective cohort	Cases: 14,792 Age range: 40-89 Males: 9215 (62.3%)	6132 (41.4%)	The target serum urate level (<6 mg/dL) was associated with a lower 5-year risk of progressing to severe or end-stage kidney disease (10.32% vs. 12.73%). Patients who reached this target had a modest but statistically significant risk reduction
Yerima et al., 2023 [16]	Nigeria	Retrospective cohort	Cases: 107 Mean age: 53.5 Males: 61 (57%)	90 (84.1%)	Higher serum uric acid (SUA) levels were associated with worse kidney function, showing a positive correlation with serum creatinine and a negative correlation with eGFR.
Jaffe et al., 2019 [17]	Israel	Retrospective cohort	Cases: 12,940 Mean age: 63.8 Males: 9819 (52.2%)	4645 (35.9%)	CKD was present in 36% of the gout patients at diagnosis and was linked to increased hospital admissions, more frequent medical visits, and greater use of ULT. Furthermore, 5-year all-cause mortality was notably higher in gout patients with CKD, across all age groups. These findings confirm that gout and CKD frequently coexist and negatively influence each other

Stack et al., 2019 [18]	UK	Retrospective cohort	Cases: 3452 Males: 1803 (52.2%)	563 (16.3%)	Patients with gout face a significantly higher risk of developing advanced CKD, with an incidence rate of 8.54 per 1000 patient-years compared to 4.08 in non-gout individuals. Gout was linked to increased risk of advanced CKD in both unadjusted and adjusted analyses (adjusted HR: 1.29)
Roughley et al., 2018 [19]	UK	Retrospective cohort	Cases: 41,446 Mean age: 57.2 Males: 33,574 (81%)	6694 (16.2%)	Gout was associated with a 78% increased risk of incident CKD (adjusted HR 1.78). Although ULT was initially linked to higher CKD risk, this association disappeared after adjustment, except in women over a 3-year period.
Hüzmeli et al., 2019 [20]	Turkey	Retrospective cohort	Cases: 162 Mean age: 59.6 Males: 135 (83.3%)	64 (39.5%)	Gout patients had a high prevalence of CKD, with 39.5% in stages 3–5 and 33.3% in stage 2.

Table (2): Risk of bias assessment using ROBINS-I.

Study ID	Bias due to confounding	Bias in the selection of participants into	Bias in the classification of interventions	Bias due to deviations from the intended interval	Bias due to missing data	Bias in the measurement of outcomes	Bias in the selection of reported result	Overall bias
Singh et al., 2019 [12]	Low	Low	Mod	Low	Low	Low	Mod	Low
Roughley et al., 2018 [13]	Mod	Low	Low	Low	Low	Mod	Low	Low
Akpabio et al., 2018 [14]	Low	Low	Mod	Low	Low	Low	Mod	Low
Wang et al., 2025 [15]	Low	Low	Mod	Low	Mod			Moderate

						Mod	Mod	
Yerima et al., 2023 [16]	Mod	Mod	Mod	Low	Low	Low	Mod	Moderate
Jaffe et al., 2019 [17]	Mod	Mod	Low	Low	Low	Mod	Low	Moderate
Stack et al., 2019 [18]	Mod	Mod	Low	Low	Low	Mod	Mod	Moderate
Roughley et al., 2018 [19]	Mod	Mod	Low	Mod	Low	Mod	Mod	Moderate
Hüzmeli et al., 2019 [20]	Mod	Mod	High	Low	Low	Mod	Low	High

Mod: Moderate

contribute to the development of CKD. Renal damage may arise from coexisting hypertension and diabetes, uric acid–induced endothelial dysfunction and renovascular disease [23], as well as from the use of nonsteroidal anti-inflammatory drugs. Although allopurinol has historically been considered potentially harmful to kidney function based on early findings [24], a more recent systematic review suggests it may actually offer protection against CKD progression [25]. Moreover, inflammation in gout is now increasingly recognized to persist during the inter-critical periods between flares [26], supporting the idea that chronic inflammation could play a role in increasing vascular risk, as has been proposed in other inflammatory arthritides [27]. Notably, studies in this review that examined serum uric acid (SUA) levels observed a strong correlation between elevated SUA and worsening kidney function. This was consistently evidenced by increases in serum creatinine and decreases in eGFR. Conversely, individuals who achieved target urate levels below 6 mg/dL, typically through ULT, experienced a modest but statistically significant reduction in the risk of progression to severe or end-stage kidney disease. This finding is of clinical relevance, as it underscores the potential benefit of adequate gout control not only for joint health but also for preserving renal function. Kannuthurai & Gaffo reported that ULT is advised for managing recurrent gout flares, tophaceous deposits, and in individuals with moderate to severe CKD, with a target serum urate level of less than 6 mg/dL to help prevent future flares. Although concerns persist regarding the safety of ULT in patients with kidney impairment, emerging evidence suggests that these treatments can be administered safely in this population. However, uncertainty remains about the appropriateness of gout management in patients undergoing renal replacement therapy (RRT). Additionally, studies offer mixed findings on whether ULT has any direct impact on kidney function or cardiovascular outcomes in affected individuals [28]. Premachandra et al., also found that in patients with CKD, urate-lowering therapy (ULT) should be initiated at a low dose and gradually increased, guided by serum urate levels rather than creatinine clearance. If first-line treatment is ineffective, alternative medications may be considered, with the choice depending on factors such as drug availability, severity of the disease, and the presence of other comorbid conditions [29]. The clinical burden of combined gout and CKD was further reflected in healthcare resource utilization and survival data. Patients with both conditions had higher rates of hospital admissions, outpatient visits, and medication use, along with notably increased all-cause mortality over five years. These outcomes emphasize the need for integrated

care approaches and timely interventions to mitigate the risks associated with this dual pathology. Strengths: A key strength of this review is the inclusion of large-scale, population-based cohort studies with longitudinal data, which enhances the reliability and generalizability of the findings. Most studies employed standardized definitions for CKD staging and validated laboratory measures for SUA and eGFR, enabling meaningful comparisons. The consistent use of multivariable-adjusted analyses also lends robustness to the observed associations, as it minimizes the confounding effects of comorbid conditions and demographic variables. The presence of data from diverse geographic regions and healthcare systems further supports the external validity of the results.

Limitations: Despite these strengths, several limitations must be acknowledged. All included studies were observational in nature, precluding causal inference. Although many adjusted for key confounders, residual confounding cannot be entirely ruled out. Variability in the definitions of gout and CKD across studies, particularly in diagnostic coding and laboratory thresholds, may introduce heterogeneity in outcome interpretation. In some studies, reliance on administrative databases without clinical validation could have led to misclassification of diagnoses or underreporting of important variables such as medication adherence, flare frequency, or dietary factors. Moreover, most studies lacked granular data on gout severity, duration, and the presence of tophi or polyarticular involvement, which may influence renal outcomes. Lastly, there was limited exploration of sex-based differences, except in a few subgroup analyses, despite evidence suggesting potential variation in disease expression and treatment response.

Conclusion

Elevated serum uric acid levels, common in gout, appear to play a pivotal role in worsening renal function, while achievement of urate targets through effective therapy may reduce the risk of kidney damage. The co-occurrence of these two conditions results in a higher clinical and economic burden, underscoring the need for early identification and aggressive management. Integrating renal monitoring into the standard of care for gout patients, along with individualized urate-lowering strategies, has the potential to improve both renal and overall patient outcomes.

Conflict of Interest

None

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None

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