



Systematic Review Article

ASSOCIATION BETWEEN HYPERURICEMIA AND LONG-TERM KIDNEY FUNCTION DECLINE: A SYSTEMATIC REVIEW

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Received : 26/07/2026
Received in revised form : 23/08/2026
Accepted : 05/09/2026

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DOI: 10.70034/ijmedph.2026.3.938

Source of Support: Nil,

Conflict of Interest: None declared

Int J Med Pub Health

2026; 16 (3); 6090-6095

ABSTRACT

Background: Chronic kidney disease (CKD) is a major global health problem, and identifying modifiable risk factors is essential for preventing disease progression. Hyperuricemia has been proposed as a potential contributor to kidney function decline through mechanisms including oxidative stress and renal inflammation. However, evidence remains inconsistent. This systematic review aims to evaluate the association between hyperuricemia and long-term decline in kidney function and its potential role as a predictor of CKD progression. The objective is to systematically evaluate the association between hyperuricemia and long-term decline in kidney function, including changes in estimated glomerular filtration rate (eGFR), rapid kidney function decline, and the development of chronic kidney disease (CKD), while assessing the influence of demographic and clinical factors, variations in hyperuricemia severity, potential confounding factors.

Materials and Methods: This systematic review was conducted according to the PRISMA guidelines. Relevant studies were identified through searches of PubMed/MEDLINE, Embase, Scopus, Web of Science, and the Cochrane Library using keywords related to hyperuricemia, serum uric acid, kidney function, eGFR decline, chronic kidney disease and renal function decline. Studies were screened according to predefined inclusion and exclusion criteria. Studies published from year 2000 and onwards were included. Eligible studies underwent independent screening and standardized data extraction. Study quality and risk of bias were assessed using an appropriate assessment tool. Findings were synthesized narratively.

Results: Most studies demonstrated that elevated serum uric acid was independently associated with accelerated eGFR decline, incident chronic kidney disease, and progression to advanced kidney disease. Although a few studies reported no independent association after multivariable adjustment or observed a U-shaped relationship between serum uric acid and renal outcomes, the overall evidence supports hyperuricemia as a significant predictor of long-term kidney function decline.

Conclusion: Overall, elevated serum uric acid is an independent predictor of long-term kidney function decline and CKD progression. Further prospective studies and randomized trials are needed to confirm causality and the renoprotective effects of uric acid lowering therapy.

Keywords: hyperuricemia, serum uric acid, kidney function, eGFR decline, chronic kidney disease, renal function decline.

INTRODUCTION

Chronic kidney disease (CKD) is a major global health concern associated with increased

cardiovascular morbidity, premature mortality, reduced quality of life, and substantial healthcare burden. Identifying potentially modifiable risk factors for progressive kidney function decline is

therefore important for the prevention and early management of CKD.

Serum uric acid, the final product of purine metabolism, is primarily eliminated through the kidneys. Although hyperuricemia has traditionally been considered a consequence of reduced renal excretion, emerging evidence suggests that elevated uric acid may contribute to kidney dysfunction through oxidative stress, endothelial dysfunction, activation of the renin–angiotensin–aldosterone system, renal vasoconstriction, and tubulointerstitial inflammation.

Waheed Y et al. (2021) reviewed the evidence linking asymptomatic hyperuricemia with chronic kidney disease progression and cardiovascular disease. The authors suggested that renin–angiotensin system activation, nitric oxide inhibition, and vascular injury may underlie these associations, although the role of serum uric acid as a marker of renal vascular disease remains controversial.^[1]

Lee CL & Tsai SF, (2021) studied 1,860 non-diabetic CKD patients and found that serum uric acid (SUA) ≥ 9 mg/dL was associated with increased all-cause mortality in moderate CKD (Hazard Ratio [HR] 2.15), whereas SUA < 5 mg/dL was linked to higher all-cause mortality in severe CKD. The findings suggest a stage-dependent relationship between serum uric acid levels and mortality in CKD.^[2]

Zhang L et al. (2012) followed 1,410 adults with normal kidney function for 4 years and found that 11.9% developed renal function decline. Each 1 mg/dL increase in plasma uric acid was independently associated with a 19% higher risk of renal function decline (Odds Ratio [OR] 1.19, 95% Confidence Interval [CI]: 1.04–1.38), with a stronger association in participants without hypertension or diabetes (OR 1.50, 95% CI: 1.13–1.98).^[3]

Sedaghat S et al. (2013) found that higher serum uric acid was associated with accelerated eGFR decline, with a stronger association in individuals with hypertension. Although the cohort analysis did not show a significant association with incident CKD, the pooled meta-analysis supported hyperuricemia as an independent risk factor for CKD progression.^[4]

Longitudinal studies have reported associations between hyperuricemia, accelerated estimated glomerular filtration rate decline, incident CKD, and progression of kidney disease. However, findings remain inconsistent because of variations in study populations, definitions of hyperuricemia, follow-up duration, kidney outcomes, and adjustment for confounding factors. Therefore, this systematic review aims to evaluate the association between hyperuricemia and long-term decline in kidney function and assess its potential role as a predictor of progressive kidney dysfunction.

Objective: To systematically evaluate the association between hyperuricemia and long-term kidney function decline, including changes in estimated glomerular filtration rate (eGFR), rapid decline in kidney function, and the development of chronic kidney disease (CKD), while examining the

influence of demographic and clinical characteristics, the severity of hyperuricemia, and potential confounding factors.

MATERIALS AND METHODS

This systematic review followed the PRISMA guidelines. A comprehensive literature search was conducted in PubMed/MEDLINE, Scopus, Web of Science, Embase, the Cochrane Library, and Google Scholar using relevant keywords including hyperuricemia, serum uric acid, kidney function, estimated glomerular filtration rate (eGFR), chronic kidney disease, and renal function decline. Original research studies, including case-control studies, cohort studies, other longitudinal observational studies published from year 2000 onwards were included. Studies that did not reporting relevant renal outcomes, had non-extractable data, or lacked clearly defined outcomes were excluded, along with editorials, conference abstracts and letters.

Titles, abstracts, and full-text articles were independently screened by two reviewers according to predefined inclusion and exclusion criteria, with disagreements resolved through discussion. Data extraction and quality assessment were performed independently using standardized tools appropriate to the study design. The findings were synthesized narratively.

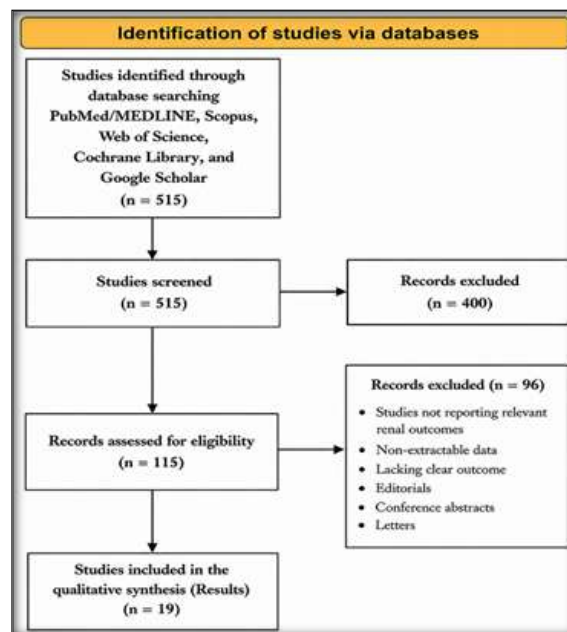


Figure 1: PRISMA flow diagram of study selection process

RESULTS & DISCUSSION

Iseki K et al. (2001) analyzed data from 6,403 Japanese adults followed for 2 years and found that 241 participants developed elevated serum creatinine. Hyperuricemia was an independent predictor of renal function decline, with individuals having serum uric acid ≥ 8.0 mg/dL showing a significantly higher risk

of developing elevated serum creatinine compared with those with <5.0 mg/dL (men: adjusted Relative Risk [RR] 2.91, 95% Confidence Interval [CI] 1.79–4.75; women: adjusted RR 10.39, 95% CI 1.91–56.62). The study concluded that elevated serum uric acid is a significant predictor of subsequent renal dysfunction.^[5]

Iseki K et al. (2004) conducted a population-based cohort study of 48,177 Japanese adults to investigate the association between hyperuricemia and the risk of end-stage renal disease (ESRD). During follow-up, 103 participants initiated dialysis. The incidence of ESRD per 1,000 participants was 4.64 vs. 1.22 in hyperuricemic versus non-hyperuricemic men and 9.03 vs. 0.87 in hyperuricemic versus non-hyperuricemic women. After adjustment for potential confounders, hyperuricemia was associated with a significantly increased risk of ESRD in women (Hazard Ratio [HR] 5.77, 95% CI 2.31–14.42; $P = 0.0002$), whereas the association in men was not statistically significant (HR 2.00, 95% CI 0.90–4.44).^[6]

Domrongkitchaiporn S et al. (2005) conducted a 12-year cohort study involving 3,499 Thai adults and observed that the prevalence of decreased kidney function (GFR <60 mL/min) increased from 1.7% in 1985 to 6.8% in 1997. After adjustment for potential confounders, hyperuricemia (>6.29 mg/dL) was independently associated with an increased risk of developing decreased kidney function (adjusted Odds Ratio [OR] 1.82, 95% CI 1.12–2.98). Systolic hypertension (adjusted OR 2.57, 95% CI 1.00–6.81) and elevated body mass index (adjusted OR 1.68, 95% CI 1.02–2.77) were also identified as significant predictors.^[7]

Chonchol M et al. (2007) evaluated the association between serum uric acid and kidney function decline and found that the prevalence of eGFR <60 mL/min/1.73 m² increased across increasing uric acid quintiles (7%, 14%, 12%, 25%, and 42%, respectively). Higher uric acid levels were associated with a modest but significant risk of kidney function decline, with adjusted odds ratios of 1.47 (95% CI 1.04–2.07) and 1.49 (95% CI 1.00–2.22) for the fourth and fifth quintiles, respectively. However, no significant association was observed between serum uric acid and incident CKD (adjusted OR 1.00, 95% CI 0.89–1.14).^[8]

Weiner DE et al. (2008) analyzed data from 13,338 adults with normal kidney function from two community-based cohorts over a mean follow-up of 8.5 years. During follow-up, 712 participants (5.6%) developed incident kidney disease based on eGFR decline, and 302 (2.3%) developed kidney disease based on increased serum creatinine. After adjustment for demographic, clinical, and laboratory variables, each 1 mg/dL increase in serum uric acid was associated with a higher risk of incident kidney disease defined by eGFR decline (adjusted OR 1.07, 95% CI 1.01–1.14) and serum creatinine increase (adjusted OR 1.11, 95% CI 1.02–1.21), indicating

that elevated serum uric acid is an independent, though modest, predictor of kidney disease.^[9]

Obermayr RP et al. (2008) conducted a prospective cohort study of 21,475 healthy adults with a median follow-up of 7 years and found that elevated serum uric acid independently predicted incident kidney disease (eGFR <60 mL/min/1.73 m²). Compared with normal uric acid levels, slightly elevated uric acid (7.0–8.9 mg/dL) was associated with 74% higher odds of incident kidney disease (adjusted OR 1.74, 95% CI 1.45–2.09), while uric acid ≥ 9.0 mg/dL was associated with a more than threefold increase in risk (adjusted OR 3.12, 95% CI 2.29–4.25). These associations remained significant after adjustment for baseline kidney function and other confounding factors, indicating that hyperuricemia is an independent predictor of new-onset kidney disease.^[10]

Bellomo G et al. (2010) conducted a prospective cohort study of 900 healthy normotensive adults with a median follow-up of 59 months. During follow-up, mean eGFR declined from 97 ± 16 to 88 ± 14 mL/min/1.73 m². Higher serum uric acid levels were independently associated with an increased risk of eGFR decline >10 mL/min/1.73 m², with an adjusted hazard ratio (HR) of 1.28 (95% CI 1.12–1.48) for each 1 mg/dL increase in serum uric acid. The association remained consistent across different eGFR estimation equations and in both men and women.^[11]

Cain L et al. (2010) analyzed data from 49,295 adults without cardiovascular disease in the C8 Health Study to examine the association between serum uric acid (SUA) and chronic kidney disease (CKD). Higher SUA levels were independently associated with greater odds of CKD. Compared with the lowest SUA quartile, the adjusted odds ratios (95% CI) for CKD were 1.53 (1.31–1.78), 2.16 (1.86–2.50), and 4.67 (4.07–5.36) across quartiles 2–4, respectively (P -trend < 0.0001). This significant association was observed in both men and women. The authors concluded that elevated SUA is strongly associated with CKD and may partly explain its link with cardiovascular disease.^[12]

A community-based screening study of 31,331 Taiwanese adults aged >40 years found that hyperuricemia was independently associated with an increased risk of chronic kidney disease (CKD). After adjustment for potential confounders, individuals with hyperuricemia had a 3.649-fold higher risk of CKD (unadjusted OR: 4.036). Serum uric acid levels were inversely correlated with estimated glomerular filtration rate (eGFR), with a significant association observed in stage 3 CKD but not in stages 4 or 5. The findings suggest that hyperuricemia is an independent risk factor for CKD and support early screening for elevated serum uric acid in middle-aged and older adults (Chang HY et al., 2010).^[13]

Yamada T et al. (2011) conducted a 5-year cohort study involving 14,399 adults without baseline chronic kidney disease (CKD) or treatment for hyperuricemia. CKD developed in 4.1% of men and

3.7% of women during follow-up. After adjusting for potential confounders, higher serum uric acid levels independently predicted incident CKD in both sexes, with an odds ratio (95% CI) of 1.42 (1.28–1.58) per 1 mg/dL increase in men and 1.32 (1.12–1.56) in women. The study concluded that elevated serum uric acid is an independent predictor of CKD in adults undergoing routine health screening.^[14]

Zoppini G et al. (2012) followed patients with type 2 diabetes and preserved kidney function for 5 years and found that 13.4% developed chronic kidney disease (CKD). CKD incidence was significantly higher in patients with hyperuricemia than in those without (29.5% vs. 11.4%, $P < 0.001$). After adjustment for potential confounders, hyperuricemia remained independently associated with incident CKD (adjusted OR 2.10, 95% CI: 1.16–3.76, $P < 0.01$). Additionally, each 1- standard deviation increase in serum uric acid was associated with a 21% higher risk of developing CKD.^[15]

Kanda E et al. (2015) conducted a prospective cohort study of 4,188 healthy Japanese adults and found that both low and high serum uric acid levels were associated with kidney function decline. In men, serum uric acid showed a significant U-shaped relationship with incident CKD and eGFR decline (linear $P = 0.0001$). Among individuals with low serum uric acid (<5 mg/dL in men; <3.6 mg/dL in women), lower levels were independently associated with progressive eGFR decline over time (men: $P = 0.0001$; women: $P = 0.045$). The authors concluded that both hyperuricemia and hypouricemia may predict renal function decline.^[16]

Toyama T et al. (2015) analyzed 41,632 Japanese men over a mean follow-up of 4 years and found that 7.6% developed kidney impairment. Compared with serum uric acid (SUA) levels of 4.0–4.9 mg/dL, participants with $SUA \geq 6.0$ mg/dL had a significantly higher risk of kidney dysfunction. The risk threshold differed by baseline renal function, with $SUA \geq 7.0$ mg/dL in those with normal kidney function and ≥ 6.0 mg/dL in those with mildly reduced kidney function. Similar associations were observed for eGFR decline. The authors concluded that hyperuricemia is an independent risk factor for kidney impairment, particularly in individuals with pre-existing mild renal dysfunction.^[17]

Son YB et al. (2021) followed 13,133 healthy adults with normal kidney function and found that higher serum uric acid (SUA) independently predicted renal function decline. Participants in the highest SUA quartile had a significantly greater risk of 30% eGFR decline (Relative Risk [RR] 3.701, 95% CI: 1.504–

9.108) and a 2.2-fold higher risk of developing chronic kidney disease (CKD) (95% CI: 1.182–4.177). The study concluded that elevated SUA is an independent risk factor for CKD development and kidney function decline.^[18]

Cao X et al. (2018) conducted a cohort study of 6,495 Chinese adults with normal baseline kidney function and found that 24.8% experienced combined renal events, 23.2% had rapid renal function decline, and 5.7% developed chronic kidney disease during a mean follow-up of 52.8 months. After adjustment for potential confounders, participants in the highest serum uric acid quartile had a significantly higher risk of rapid eGFR decline (OR 1.32, 95% CI: 1.02–2.97; P for trend < 0.001) and incident CKD compared with the lowest quartile. The findings indicate that elevated serum uric acid is an independent predictor of progressive renal dysfunction.^[19]

Oh TR et al. (2019) analyzed 2,042 patients with chronic kidney disease from the KNOW-CKD cohort and found that higher serum uric acid (SUA) levels independently predicted CKD progression. Each 1 mg/dL increase in SUA was associated with a 28% higher risk of composite renal outcomes (HR 1.277, 95% CI: 1.212–1.345), while the highest SUA quartile had a 3.59-fold greater risk of renal disease progression (HR 3.590, 95% CI: 2.546–5.063). However, treatment with allopurinol or febuxostat did not significantly reduce the risk of adverse renal outcomes.^[20]

Madero M et al. (2009) analyzed 838 patients with stage 3–4 CKD and found that each 1 mg/dL increase in serum uric acid was associated with a 17% higher risk of all-cause mortality (HR 1.17, 95% CI: 1.05–1.30) and a 16% higher risk of cardiovascular mortality (HR 1.16, 95% CI: 1.01–1.33), but not with progression to kidney failure.^[21]

Kuriyama S et al. (2015) followed 8,223 adults for 4 years and found that higher serum uric acid levels independently predicted new-onset hypertension (HR 1.112, 95% CI: 1.024–1.207) but were not independently associated with incident CKD after multivariable adjustment.^[22]

Mwasongwe SE et al., (2018) analyzed 3,702 African American adults from the Jackson Heart Study and found that participants in the highest serum uric acid quartile had significantly greater odds of rapid kidney function decline (OR 1.80, 95% CI: 1.25–2.49) and incident CKD (OR 2.00, 95% CI: 1.31–3.06) compared with those in the lowest quartile, indicating that elevated serum uric acid is an independent predictor of renal function decline.^[23]

Table 1: Summary of Studies Evaluating the Association Between Hyperuricemia and Long-Term Kidney Function Decline

Author (Year)	Key Findings
Iseki K et al. (2001)	Hyperuricemia independently predicted subsequent renal dysfunction.
Iseki K et al. (2004)	Hyperuricemia increased the risk of ESRD, particularly in women.
Domrongkitchaiporn S et al. (2005)	Hyperuricemia independently predicted developing decreased kidney function.
Chonchol M et al. (2007)	Higher serum uric acid was associated with risk of kidney function decline but not incident CKD.
Weiner DE et al. (2008)	Elevated serum uric acid modestly predicted kidney disease.

Obermayr RP et al. (2008)	Hyperuricemia independently predicted new-onset kidney disease.
Bellomo G et al. (2010)	Elevated serum uric acid independently associated with eGFR decline.
Cain L et al. (2010)	Higher serum uric acid was strongly associated with CKD.
Chang HY et al. (2010)	Hyperuricemia was an independent risk factor for CKD.
Yamada T et al. (2011)	Elevated serum uric acid independently predicted incident CKD.
Zoppini G et al. (2012)	Hyperuricemia increased CKD risk in patients with type 2 diabetes.
Kanda E et al. (2015)	Both low and high serum uric acid levels were associated with kidney function decline.
Toyama T et al. (2015)	Hyperuricemia is an independent risk factor for kidney function impairment and eGFR decline.
Son YB et al. (2021)	Elevated serum uric acid is an independent risk factor for CKD development and kidney function decline.
Cao X et al. (2018)	Elevated serum uric acid is an independent predictor of progressive renal dysfunction.
Oh TR et al. (2019)	Elevated serum uric acid independently predicted CKD progression; urate-lowering therapy showed no clear renal benefit.
Madero M et al. (2009)	Increase in serum uric acid was associated with a 17% higher risk of all-cause mortality but not with progression to kidney failure
Kuriyama S et al. (2015)	Hyperuricemia predicted hypertension but not independently associated with incident CKD.
Mwasongwe SE et al. (2018)	Higher serum uric acid predicted rapid kidney function decline and incident CKD.

CONCLUSION

Iseki K et al. (2001) concluded that elevated serum uric acid is a significant predictor of subsequent renal dysfunction.^[5]

Chonchol M et al. (2007) observed that uric acid was strongly associated with prevalent CKD but showed only a modest association with kidney disease progression.^[8]

Hyperuricemia is an independent risk factor for CKD and support early screening for elevated serum uric acid in middle-aged and older adults (Chang HY et al., 2010).^[13]

Yamada T et al. (2011) concluded that elevated serum uric acid is an independent predictor of CKD in adults undergoing routine health screening.^[14]

Kanda E et al. (2015) concluded that both reduced and elevated serum uric acid levels are linked to declining kidney function, with hypouricemia emerging as a potential independent predictor of renal function deterioration in otherwise healthy individuals.^[16]

Toyama T et al. (2015) concluded that hyperuricemia is an independent risk factor for kidney impairment, particularly in individuals with pre-existing mild renal dysfunction.^[17]

Son YB et al. (2021) concluded that elevated SUA is an independent risk factor for CKD development and kidney function decline.^[18]

Cao X et al. (2018) findings indicate that elevated serum uric acid is an independent predictor of progressive renal dysfunction.^[19]

Kuriyama S et al. (2015) concluded that elevated serum uric acid independently predicted new-onset hypertension but not incident chronic kidney disease.^[22]

Tsai CW et al. (2017) retrospectively analyzed 739 patients and found that higher baseline serum uric acid was independently associated with faster eGFR decline and an increased risk of kidney failure (HR 1.07 per 1 mg/dL increase; 95% CI: 1.00–1.14). The association was strongest in patients without proteinuria, suggesting hyperuricemia is a potentially modifiable risk factor for CKD progression.^[24]

Overall, Current evidence suggests that elevated serum uric acid is an independent predictor of long-term kidney function decline and chronic kidney disease progression. Routine assessment of serum uric acid may help identify individuals at increased risk of renal impairment and facilitate early preventive strategies. Further well-designed, large-scale prospective studies and randomized controlled trials are needed to clarify the causal role of hyperuricemia in CKD development, evaluate the long-term renoprotective effects of uric acid-lowering interventions, identify high-risk populations most likely to benefit from treatment, and establish evidence-based clinical management strategies.

Acknowledgment

The authors sincerely thank all researchers whose published work contributed to this systematic review. They also express their gratitude to their mentors, and colleagues for their valuable guidance, encouragement, and support throughout the preparation of this manuscript. No funding was received for this study, and the authors declare that they have no conflicts of interest related to this work.

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