Original Article

The Relationship between Hypertension and Kidney Stone Formation: Insights from a Cross-Sectional Study

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Abstract

Background: Urolithiasis and hypertension are common chronic diseases that occur in combination with some metabolic, vascular similarities. The evidence is lacking on the link between these two conditions in Middle Eastern populations where climatic and dietary factors may affect stone risk. **Objectives:** To assess the relationship between HTN and urolithiasis, and to determine metabolic and lifestyle factors that are independently associated with kidney stone formation in an adult population in the Kurdistan Region of Iraq. **Materials and Methods:** A cross-sectional analytical study was applied on 364 subjects (aged ≥30 years) visited the urology clinic at Sulaymaniyah city from February to August 2025. Urolithiasis was established with ultrasonography and/or computed tomography. Hypertension was characterized by blood pressure reading, clinical diagnosis or antihypertensive medication. Demographic, metabolic and lifestyle information was obtained. Categorical data were compared between groups using chi-square tests, and independant predictors were determined by binary logistic regression. **Results:** The prevalence of urolithiasis was significantly greater in the hypertensive group than in normotensives (65.2% vs 47.3%; $p = 0.001$). Compared to normotensive participants, hypertensive subjects had higher levels of serum UA, but lower eGFR, as well as higher serum sodium and more urine acidity (all $p < 0.01$). In multivariable analysis, hypertension was an independent predictor of urolithiasis (OR = 2.01, 95% CI 1.30–3.10). Other independent risk factors were high serum uric acid, low urine pH, high dietary salt and low daily water. **Conclusion:** Hypertension is a strong and independent risk factor for urolithiasis and coexists with metabolic and lifestyle factors that are lithogenic. The addition of stone prevention measures into the control of hypertension might diminish the burden of kidney stones and cardio-renal consequences related to them.

Keyword: Hypertension; Urolithiasis; Kidney stones; Hyperuricemia; Urine pH; Salt intake; Hydration; Cross-sectional study

Introduction

One of the commonest urological disorder is knows as urolithiasis and the prevalence of this diseases globally has been increased and affected up to 12% of adults, at the same time it has impacted healthcare system importantly with increasing patients' morbidity [1]. Nephrolithiasis is a complex disease that results from metabolic diseases, dietary problems as well as environmental factors and genetic influences. In addition, hypertension is a very common chronic disorder worldwide and has recently been regarded as an important factor in the formation of renal stone [2]. The two-way relationship between hypertension and urolithiasis keeps getting stronger. Hypertensives often exhibit

urine biochemistry that favors crystallization [3]. In contrast, frequent stone recurrence may also induce renal tubular damage, interstitial fibrosis, and subsequent loss of kidney function, which could over magnify secondary causes of hypertension [4]. The results of previous studies suggested that the two conditions are closely connected and may represent different expressions of the same underlying disease. This relationship appears to be driven by shared biological mechanisms, especially increased oxidative stress and activation of the renin–angiotensin–aldosterone system (RAAS). Together, these processes can interfere with normal blood pressure control and place strain on kidney function, ultimately leading to disturbances in electrolyte levels and acid–base balance [5,6]. Furthermore, hypertensive patients often have multiple metabolic impairments that might be risk factors of urolithiasis, such as obesity, insulin resistance, dyslipidemia, and hyperuricemia [7,8]. Adjustable lifestyles, like high salt-containing diets and low fluid intake without paying attention to excessive animal protein ingestion, also bring a propensity for increasing the potential of hypertension and kidney disease [9,10]. Despite a growing body of global literature, empirical evidence is still limited in this context from the Kurdistan Region of Iraq (KRI). Regional features such as the high temperature, chronic dehydration, salt-rich diet, and increasing rate of hypertension could have a special influence on stone formation in this population. The majority of the local studies have predominantly documented prevalence estimates but not explored hypertension in relationship with metabolic profile and lifestyle in the same analytical framework as this study. This region lacked a clear understanding of the association between blood pressure, metabolic derangements, and lifestyle practices with urolithiasis. Therefore, the aim of this study was to analyze, for the first time, the association between hypertension and urolithiasis in a large cohort of adults (n = 364) from the Kurdistan Region of Iraq, as well as the metabolic and lifestyle factors that predispose kidney stone formation. Better understanding of these relationships is important for directing selective preventative therapies, improving clinical decision-making, and identifying patients at greatest risk of recurrent or severe stone formation.

Materials and Methods

Study Design

This was a cross-sectional analytical study conducted to measure the association between urolithiasis and hypertension, as well as related metabolic and lifestyle risk factors in adult patients. This approach allowed concurrent examination of exposure and outcome at a single time point, which is appropriate for prevalence estimation and for examining associations in clinical populations [11].

Study Setting and Population

The study was performed at the Urology Clinic of Mercy Medical City Hospital, Sulaymaniyah, Kurdistan Region of Iraq. Patients who visited the urology clinic for evaluation or follow-up were consecutively recruited from February 2025 to August 2025 to reflect routine clinical practice. A total of 364 adults aged ≥30 years participated in the study. The age threshold was selected to ensure adequate representation of hypertension, which is uncommon in younger populations and central to the study objective. Consecutive recruitment was used to minimize selection bias and mirror the typical clinical profile of patients attending the clinic during the study period.

Inclusion and Exclusion Criteria

Participants were included if they:

- Were aged 30 years or older
- Had recorded blood pressure readings
- Had positive or negative imaging reports confirming the presence or absence of urolithiasis
- Had available metabolic laboratory test results

Participants were excluded if they:

- Were pregnant
- Had end-stage renal disease or were on dialysis
- Had missing data on blood pressure, imaging findings, or key metabolic variables

Diabetes mellitus, dyslipidemia, and obesity were not used as exclusion criteria, as these conditions frequently coexist with hypertension and urolithiasis and represent shared metabolic and pathophysiological pathways. Excluding these comorbidities would reduce the external validity and real-world applicability of the findings.

Sample Size Considerations

The final sample of 364 participants was considered sufficient to detect a clinically meaningful association between hypertension and urolithiasis and to allow multivariable binary logistic regression analysis.

Data Collection Procedures

Data were collected from electronic medical records, radiological reports, and laboratory databases. Confirmation of urolithiasis status was obtained using ultrasonography and/or computed tomography (CT) performed as part of routine clinical care. Although non-contrast CT is considered the gold standard, ultrasonography was also used because of its widespread clinical availability and lower radiation exposure. Blood pressure was measured during clinic visits using standardized clinical techniques, and metabolic laboratory tests were obtained during the most recent clinical assessment.

Variables and Operational Definitions

Outcome Variable

Urolithiasis was considered when the patient had one or multiple stones on ultrasound/CT. Patients were identified as stone-free only if imaging at the time of clinic visit corroborated a lack of stones [12].

Exposure Variable

Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, a documented clinical diagnosis of hypertension, or current use of antihypertensive medications. Participants were classified as hypertensive or non-hypertensive accordingly [13].

Sociodemographic Variables

Age (years), sex (male/female), body mass index (BMI, kg/m²), smoking status (current smoker or non-smoker), and family history of urolithiasis (yes/no) were recorded.

Metabolic Variables

Laboratory parameters included serum uric acid (mg/dL), serum creatinine (mg/dL), estimated glomerular filtration rate (eGFR, mL/min/1.73 m²), serum sodium (mEq/L), and urine pH. Serum uric acid was analyzed as a continuous variable.

Lifestyle Variables

Daily water intake was categorized as low (<2.0 L/day) or adequate (≥2.0 L/day) based on patient self-report. Dietary salt intake was categorized as high or normal based on habitual dietary patterns, including frequent addition of table salt and consumption of salt-rich foods, as reported during patient interviews. These variables were included due to their established associations with hypertension and urolithiasis [14].

Comorbidities

The presence of diabetes mellitus, dyslipidemia, and obesity (BMI ≥30 kg/m²) was recorded for descriptive and interpretative purposes.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27. Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. The association between hypertension and urolithiasis was assessed using the chi-square test. Differences in metabolic variables according to hypertension status were evaluated using the independent t-test. Univariable and multivariable binary logistic regression analyses were conducted to identify independent predictors of urolithiasis. The multivariable model included hypertension status, serum uric acid, urine pH, dietary salt intake, and daily water intake. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A two-sided p-value of <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the College of Medicine, University of Sulaimani. All participants provided written informed consent prior to enrollment. Data were anonymized to ensure confidentiality and privacy.

Results

Sociodemographic Characteristics

A total of 364 study subjects were included (Table 1). The average age of the study population was 56.7 ± 16.0 years, suggesting a primarily middle-aged and elderly group of individuals. The proportions of females were 52.2% compared to 47.8% for males in the study population. The average body mass index was 26.9 ± 4.8 kg/m², which indicated that the population was predominantly overweight. Forty-seven point five % of participants were smokers and 53.0% had a positive family history for urolithiasis, suggesting considerable lifestyle and hereditary risk factors.

Table 1: Sociodemographic Characteristics of Participants

Variable	Value
Total sample	364
Mean age (years)	56.7 ± 16.0
Male	47.8%
Female	52.2%
Mean BMI (kg/m²)	26.9 ± 4.8
Current smokers	47.5%
Family history of urolithiasis	53.0%

Prevalence of Urolithiasis by Hypertension Status

It was a statistically significant that association of HTN with Urolithiasis (Table 2). The frequency of urolithiasis in the forms hypertensive and non-hypertensive group were 65.2% (n = 181) and 47.3% (n = 183), p-value <0.001, respectively. This variation was extremely significant (p=< 0.001), demonstrating that patients with hypertension also had a higher chance of kidney stones. These findings clearly support the thesis of a possible hypertensive etiologic role for stone disease and/or its concomitant role as an independent factor among other metabolic factors that might also be predisposing patients to urolithiasis.

Table 2: Prevalence of Urolithiasis by Hypertension Status

Hypertension Status	Total	Stone Presence (%)	P-value
Hypertensive	181	65.2%	< 0.001
Non-hypertensive	183	47.3%	

Metabolic Differences between Hypertensive and Non-Hypertensive Patients

Table 3 indicates significant differences between hypertensive and non-hypertensive for metabolism. Hypertensive participants had a significantly higher median serum uric acid of 7.1 ± 1.4 mg/dL compared to non-hypertensive subjects (5.9 ± 1.2 mg/dL, p <0.001). Hypertensive individuals also had decreased renal function as indicated by the eGFR [64.2 ± 18.1 mL/min vs. 78.5 ± 16.7 mL/min (p < 0.001) in non-hypertensive participants]. Serum sodium was higher in hypertensives (142.4 ± 4.2 vs. 139.7 ± 3.8 mEq/L, p = 0.005), which might reflect dietary habits or changes in sodium homeostasis. Of interest hypertensives also presented a more acidic urine (urine pH 5.3 ± 0.7 vs5.8 ± 0.6 in non-hypertensives; p < 0,001). These metabolic features jointly indicate a more favorable physiologic environment for stone formation in hypertensives.

Table 3: Comparison of Metabolic Profiles between Hypertensive and Non-Hypertensive Patients

Metabolic Variable	Hypertensive (Mean ± SD)	Non-Hypertensive	P-value
Uric acid (mg/dL)	7.1 ± 1.4	5.9 ± 1.2	<0.001
eGFR (mL/min)	64.2 ± 18.1	78.5 ± 16.7	<0.001
Serum sodium (mEq/L)	142.4 ± 4.2	139.7 ± 3.8	0.005
Urine pH	5.3 ± 0.7	5.8 ± 0.6	<0.001

Predictors of Urolithiasis (Logistic Regression Analysis)

The independent predictors for urolithiasis in our patients were generated by logistic regression (Table 4). Hypertension also remained independently predictive; hypertensive patients were two times more likely to have stone disease (OR = 2.01, CI: 1.30–3.10, p = 0.002). There was also a significant risk for higher serum uric acid levels (OR:1.45, 95 % CI: 1.20–1.75, p < 0.001), which also supports the known lithogenic potential of hyperuricemia. Another well-known risk factor, a low urinary pH, was also statistically associated with an increased odds of stone formation (OR = 1.62; 95% CI: 1.18–2.22; and p value = 0.003). Contribution of lifestyle factors was also observed that high-salt diet (OR = 1.84, p = 0.02) and low drinking volume (OR = 2.50, p = 0.001) were significantly associated with the risk to urolithiasis.

Table 4: Logistic Regression Predicting Urolithiasis

Predictor	OR*	95% CI*	P-value
Hypertension	2.01	1.30–3.10	0.002
Serum uric acid	1.45	1.20–1.75	<0.001
Low urine pH	1.62	1.18–2.22	0.003
High salt intake	1.84	1.10–3.05	0.02
Low water intake	2.50	1.40–4.55	0.001

Discussion

The aim of the current study was to evaluate the association between hypertension and urolithiasis and its metabolic and lifestyle predictors. The chief observation was that the prevalence of urolithiasis (65.2%) among hypertensive patients was much higher than that detected in non-hypertensive subjects (47.3%). Hypertension was still an independent risk factor for stone disease after correction for potential confounders, and hypertensive cases were two times more likely to exhibit stones. Further, compared with the non-hypertensive patients, those who were hypertensive showed significantly higher serum uric acid, lower eGFR, high-level sodium, and low urine pH, suggesting that these metabolic disorders are also closely related to hypertension and that hypertensives may have a more lithogenic biochemical milieu. The recent findings are

consistent with the previous and recent studies which showed an increased risk of nephrolithiasis among hypertensives. Prospective cohort data found that the incidence of stones was two times higher in hypertensive than normotensive men even when confounders were considered [15]. A meta-analyses had also described another association, that having nephrolithiasis in the past makes you more likely to develop hypertension as well, suggesting it could be bidirectional [16]. A more recent study found that first symptomatic kidney stone event was related to increased hypertension risk in a subsequent follow-up period, and it may be evidence of shared pathophysiologic mechanisms including those involving endothelial function and dysregulated renal sodium transport [17]. There's also an equivalently sized effect of a grade or so of hypertension (OR ≈ 2.0) that nowadays is being matched in newer large-n and genetic studies. In a recent cross-sectional analysis in conjunction with Mendelian randomization, there were positive associations between hypertension and the risk and progression of kidney stones, suggesting that high blood pressure may causally contribute to stone risk [8,18]. It has been also reported that hypertension was a risk factor associated with kidney stones, but it had no effect on the location of the stone in the kidney [19]. In an Iraqi study, it has been found that although hypertension was more common in stone formers, the relationship ceased to be statistically significant after controlling for age and BMI, indicating that some part of this association is mediated by obesity and ageing [3].Another study dealing with cardiovascular risk factors, published at 2025 showed that a link between CVDs occur when considered as a whole and urolithiasis is fully established but if the hypertension alone being analysed there result in associations that are less well defined or even negative possibly due to various confounding effects from combined effects of treatments [20,21]. Overall, our results are at the high limit of reported risk estimates and contribute to a growing body of evidence indicating that hypertension and nephrolithiasis associate with common metabolic and vascular pathways, albeit with some degree of heterogeneity across populations as well as confounder models. The metabolic data in our study also correlate well with contemporary knowledge regarding stone pathophysiology. We found that serum uric acid concentrations were higher in hypertensive patients and that uric acid was an independent predictor of urolithiasis. Recently, reviews and observational research all point to hyperuricemia as an important systemic disorder which predisposes to kidney stones, more particularly uric acid stones and mixed types of stones [22,23]. The 2024 and 2025 contributions have reiterated that hyperuricemia is causally associated with nephrolithiasis as well as chronic kidney damage and cardiovascular events, highlighting uric acid as a pivotal metabolic point between renal stones and hypertension and cardio-renal diseases [24–26]. Furthermore, in a recent Mendelian randomize-ation study, data had revealed that gout/ hyperuricemia were causally implicated in the risk of kidney stone formation, which was consistent with our findings showing higher serum uric acid leads to increased stone risk [8]. The lower urine pH in our hypertensive partici-pants is further substantiated by recent publica-tions. Recent reviews on uric acid nephrolithiasis highlight that persistently low urinary pH—commonly less than 5.5—is the most important risk factor for uric acid stones because of a strong decrease in solubility of uric acid [22,27,28]. In a 2022 trial in gout subjects, urine pH (2–2.5 L/d) and dietary sodium restriction remain mainstays in preventing recurrent stones

[29]. Additionally, a systematic review and meta-analysis on water intake and uric acid nephrolithiasis demonstrated that low urine volume is a major risk factor for stone events, as greater water intake clearly decreases the incidence of symptomatic stones [30]. More recent reviews also state that hyperuricosuria, acidic urine, and low volume of urine are often mixed in subjects with metabolic syndrome and in those with hypertension, supporting the practical significance of hydration and diet as interventions [24,31–33]. The independent effect of high salt intake on stone risk in our cohort further exemplifies the well-established associations between dietary sodium, hypercalciuria and stones, while also reflecting the involvement of sodium in blood pressure elevation, making salt reduction especially appealing as a dual-benefit target [34,35]. In addition to the specific relationship of hypertension to stones, our findings also need to be interpreted within a larger context: kidney stone disease is related to long-term renal and cardiovascular outcomes.Recent studies and meta-analyses have reported a higher risk for CKD, CVD or mortality in stone formers [17,23,36–38]. Because hypertension is itself a strong driver of CKD and CVD, the concurrence of hypertension with nephrolithiasis may be associated with an even higher risk for cardio-renal disease; this perspective warrants active screening and intensive risk factor control. This study has several strengths. We had a relatively large sample size of individuals with and without hypertension and could observe objective laboratory results (serum uric acid, eGFR, serum sodium, urine pH) rather than depend solely on self-reported information. Application of multivariable logistic regression enabled us to determine independent predictors of urolithiasis when adjusting for relevant confounders. Furthermore, our findings contribute to a paucity of literature on the subject amongst Middle Eastern populations, where both hypertension and stone disease are common and likely influenced by regional dietary and climatic factors. Nevertheless, there are several limitations that need to be mentioned. First, the cross-sectional design of the study precludes evaluation of temporality or causality; therefore, we cannot conclude with certainty whether hypertension predisposes individuals to urolithiasis, whether recurrent stones and renal injury contribute to secondary hypertension, or whether both conditions arise from shared upstream metabolic abnormalities. Second, this was a hospital-based, single-center study using consecutive sampling of patients attending a urology clinic rather than population-based randomization, which may limit the generalizability of the findings to the wider community. However, this approach reflects real-world clinical practice, and similar methodologies have been widely used in clinical epidemiological research. Third, dietary sodium intake and daily fluid consumption were estimated based on self-reported questionnaire data and therefore are subject to recall bias and social desirability bias. Fourth, urolithiasis diagnosis was based on both ultrasonography and computed tomography (CT). Although non-contrast CT is the gold standard for stone detection, ultrasonography was also used because of its routine availability and lower radiation exposure; thus, the potential for operator dependency and under-detection of small stones cannot be excluded. Fifth, this study was conducted in a single geographic region, which may limit extrapolation of the results to populations with different ethnic, dietary, or environmental characteristics. Finally, residual confounding from unmeasured factors—such as type and duration of antihypertensive medication use, degree of blood pressure control, detailed

dietary composition, and lack of 24-hour urinary metabolic evaluation (e.g., calcium, oxalate, citrate, and uric acid) cannot be completely excluded and may have influenced the observed associations. However, despite these weaknesses, the present results have several important clinical implications. The independent and powerful association between hypertension and urolithiasis, as well as the identification of potentially modifiable metabolic and lifestyle risk factors supporting the high-risk nature of HTN patients with stones, may have great relevance for targeted stone prevention strategies. It might be advisable to routinely screen stone risk (by history of colic, imaging when suggested, and basic metabolic evaluation: uric acid, eGFR, and urine pH) in hypertensive patients, especially if overweight, with a family history of stones, or with low fluid intake and a high-salt diet. Practically, promoting stone prevention within hypertension clinics-highlighting adequate hydration, avoidance of dietary salt excess, weight control, and (where indicated) pharmacologic therapy for hyperuricosuria or urinary acidification/ alkalinization may limit the burden of stones and the longer-term cardio-renal consequences. In addition, our findings underscore the necessity of longitudinal and interventional research in this cohort to determine causation, assess differences in stone risk based on antihypertensive regimen, and evaluate the efficacy of lifestyle and pharmacological modifications aimed at decreasing incident and recurrent stones among those with hypertension.

Conclusion

The present study reveals a significant and positive association of hypertension with urolithiasis. Patients with hypertension had a significantly higher prevalence of kidney stones and metabolic profiles (higher uric acid, lower estimated glomerular filtration rate [eGFR], higher sodium level, and lower urine pH) that favor lithogenesis. Independent stone-former risk factors were demonstrated by logistic regression analysis to be hypertension, hyperuricemia, low pH of urine, and high dwarf salt, as well as less water uptake. These results suggest that metabolic and lifestyle parameters influence the increased stone risk associated with HTN. A number of recommendations are suggested, considering the above findings. Routine assessment of the risk for urolithiasis should be considered by clinicians when hypertensive patients with concomitant metabolic abnormalities are being treated. Lifestyle advice, especially regarding increasing water intake to >2.5 L/day and reducing salt consumption, should be included in hypertension management programs. Screening for hyperuricemia and abnormal urine pH in hypertensive patients may be used as an opportunity to prescribe early intervention measures to prevent stone formation. Measures targeting hydration awareness and salt reduction in public health programs could potentially reduce urolithiasis incidence at the community level. Lastly, future longitudinal and interventional studies are needed to determine the directionality of this relationship and to understand the potential effectiveness of lifestyle changes in reducing stone risk among hypertensive individuals.

Consent for Publication

Not applicable. No individual-level identifiable data or images are included in this manuscript.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by the authors. The first draft of the manuscript was written by the corresponding author, and all authors critically reviewed and approved the final version of the manuscript.

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