**Eurasia Journal of Science and Technology**
**Print ISSN: 2663-1024**
**Online ISSN: 2663-1016**
**DOI: https://doi.org/10.61784/ejst3160**

# CONSTRUCTION OF SIRI NOMOGRAM FOR PREDICTING UROLITHIASIS RISK IN PATIENTS WITH METABOLIC SYNDROME AND TEMPORAL EXTERNAL VALIDATION OF ITS DEVELOPMENT

FangMing Xu[1*], QiMing Xu[2], ShuBin Cao[2], Li Yao[1], Min Xiong[1], Rajesh Kumar Yadav[1], Li Gao[1*]
*[1]The Second Affiliated Hospital of Guilin Medical University, Guilin 541100, Guangxi, China.*
*[2]The First Affiliated Hospital of Guilin Medical University, Guilin 541001, Guangxi, China.*
*[*]FangMing Xu and Li Gao are both the corresponding authors.*
*[*]Corresponding Author: FangMing Xu, Li Gao*

**Abstract:** Objective: To explore the relationship between systemic inflammatory markers and urolithiasis, and to build a reliable nomogram to predict the risk of patients. Methods: A total of 878 patients were included in this study, and all of them were diagnosed with MetS and were admitted to the hospital between October 2022 and October 2025. All patients were divided into two groups, one with stones (n=246) and the other without stones (n=632). Logistic regression and LASSO regression were used to identify independent risk factors, and ROC curves, calibration plots, DCA and other methods were used to verify the effectiveness of the nomogram. In order to achieve temporal external validation, 111 patients were selected from other hospitals for analysis, and the study followed the TRIPOD statement. Results: Seven variables were identified as independent predictors: gender, age, urinary uric acid, urinary citrate, hs-CRP, IL-6, and the systemic inflammatory response index (SIRI). The nomogram demonstrated good discriminative capacity with an AUC of 0.751 (95% CI: 0.716–0.787) in the derivation cohort and 0.725 (95% CI: 0.617–0.833) in the validation cohort. The calibration intercept was 0.048, indicating excellent agreement between the predicted and observed risks. Decision curve analysis (DCA) confirmed a significant clinical net benefit. Conclusion: In the process of managing urolithiasis patients, the tool based on SIRI can play an important role, which is conducive to the implementation of personalized management and risk stratification.
**Keywords:** Metabolic syndrome; Urolithiasis; Systemic inflammation; Predictive model

## 1 INTRODUCTION

In the field of urology, urolithiasis is a common disease, and in recent years, the incidence of this disease has been increasing worldwide [1]. The recurrence rate of this disease is relatively high, which not only threatens the health of patients, but also causes heavy economic burden on patients. MetS is a kind of metabolic disorder, which can lead to cardiovascular disease and urolithiasis [2].

At present, more and more scholars have confirmed that the two diseases have a certain relationship in epidemiology, and they are related to obesity, hypertension and other components, which are the key factors leading to the occurrence of the disease [3]. More and more scholars have recognized that the occurrence of metabolic syndrome is related to chronic inflammation and oxidative stress, and have a unified understanding of its comorbidities [4].

The use of hematological inflammatory indicators for prediction has achieved good results. The SII index can accurately predict the risk of nephrolithiasis in young people [5], while the SIRI index can accurately grasp the immune-inflammatory equilibrium [6]. Although some scholars have found that there is a certain relationship between SIRI and the prevalence of kidney stones [7], the predictive effect of this index on the incidence of urolithiasis in Chinese people with metabolic syndrome has not been fully explored.

In order to make up for this deficiency, the model is constructed with the help of SII and SIRI, and combined with clinical metabolic parameters to predict the risk. In this study, the risk factors of metabolic syndrome combined with urolithiasis were analyzed, and a risk stratification tool was provided for the development of the model. The purpose of constructing the model is to achieve early intervention, so that high-risk patients can be identified as soon as possible, and the incidence of urolithiasis can be reduced.

## 2 MATERIALS AND METHODS

### 2.1 Study Design and Study Population

We conducted a retrospective evaluation of 878 MetS patients at the Second Affiliated Hospital of Guilin Medical University (October 2022–October 2025). Depending on radiographic and clinical findings, we categorized subjects into those with concomitant urolithiasis (n=246) or a stone-free control cohort (n=632). Our methodology prioritized ethical standards; the study followed the Helsinki Declaration and received formal clearance from the hospital's Ethics

Committee. Furthermore, we ensured total data anonymity and benchmarked our reporting against the TRIPOD statement. Comprehensive baseline data for both groups are presented in Table 1.

**Table 1** Baseline Characteristics of Participants

| Variables | MetS urolithiasis Group | MetS Group | P |
|---|---|---|---|
| Gender<br>Male<br>Female | <br>161<br>85 | <br>326<br>306 | P<0.05 |
| Age(years) | 59.74±11.22 | 53.39±11.41 | P<0.05 |
| BMI(kg/m2) | 29.8±23.45 | 23.58±2.72 | P<0.001 |
| Triglyceride(mmol/L) | 2.14±0.86 | 1.25±0.42 | P<0.001 |
| Total Cholesterol (mmol/L) | 5.36±1.12 | 4.82±0.95 | P<0.01 |
| High-Density Lipoprotein Cholesterol (mmol/L) | 0.98±0.21 | 1.36±0.32 | P<0.001 |
| Low-Density Lipoprotein Cholesterol (mmol/L) | 3.42±0.88 | 2.95±0.74 | P<0.001 |
| White Blood Cell Count(×109/L) | 7.51±1.62 | 7.3±1.51 | P>0.05 |
| Neutrophil Count(×109/L) | 4.74±1.48 | 4.48±1.41 | P<0.05 |
| Monocyte Count(×109/L) | 0.57±0.25 | 0.52±0.22 | P<0.05 |
| Lymphocyte Count(×109/L) | 1.92±0.58 | 2.03±0.58 | P<0.05 |
| High-Sensitivity C-Reactive Protein | 2.83±2.33 | 2.34±1.77 | P<0.05 |
| IL-6 | 6.96±5.66 | 5.82±4.88 | P<0.05 |
| Platelet Count(×109/L) | 219.18±59.34 | 220.05±58.81 | P>0.05 |
| SII | 605.21±346.44 | 537.41±316.31 | P<0.05 |
| SIRI | 1.51±0.87 | 1.18±0.8 | P<0.05 |
| Albumin | 42.08±4 | 42.26±4.1 | P>0.05 |
| NLR | 2.74±1.31 | 2.43±1.19 | P<0.05 |
| PLR | 128.73±64.38 | 119.95±58.21 | P<0.05 |
| Urinary Uric Acid (mg/g Cr) | 457.55 ± 147.92 | 419.36 ± 146.72 | P<0.01 |
| Urinary Oxalate (mg/g Cr) | 22.83 ± 7.39 | 22.92 ± 6.55 | P>0.05 |
| Urinary Citrate (mg/g Cr) | 339.54 ± 113.18 | 353.52 ± 114.58 | P<0.01 |
| Urinary Creatinine (mmol/L) | 11.86±2.85 | 12.16±3.01 | P>0.05 |

Note: Values are expressed as mean ± SD or median [IQR]. P-values were determined using the independent samples t-test for normally distributed continuous variables, Mann-Whitney U test for non-normally distributed continuous variables, and Pearson $\chi^2$ test for categorical variables.

### 2.2 Data Extraction and Variable Selection

We retrieved comprehensive patient profiles, including medical history and age/gender data, directly from the Electronic Medical Record (EMR) system. Beyond calculating BMI from physical measurements, we strictly mandated radiographic confirmation (CT or ultrasound) to verify stone status. Our diagnostic laboratory panel integrated hematological indices (WBC and lymphocyte subsets) with metabolic markers like fasting glucose and lipid fractions. Furthermore, we quantified serum hs-CRP and IL-6 levels via standardized immunoassays, strictly adhering to established laboratory reference protocols.

### 2.3 Diagnostic Criteria for Metabolic Syndrome

The diagnosis of MetS is based on the standards issued by the Chinese Diabetes Society in 2017. If the patient has any three of the five components, it can be diagnosed as MetS: (1) abdominal obesity (male waist circumference ≥90 cm, female waist circumference ≥85 cm); (2) Hyperglycemia (fasting blood glucose ≥6.1 mmol/L or diagnosed with type 2 diabetes); (3) Hypertension (≥130/85 mmHg or taking antihypertensive drugs); (4) Fasting triglycerides ≥1.7 mmol/L; (5) Fasting high density lipoprotein cholesterol <1.04 mmol/L.

### 2.4 Radiographic Assessment and Urolithiasis Diagnosis

We confirmed stone presence using non-contrast CT or color Doppler, identifying hyperattenuating or hyperechoic foci with clear posterior shadowing across the urinary tract, irrespective of hydronephrosis. Quality control involved independent double-blinded reviews by two senior urologists (each with >5 years of experience). To validate our grouping accuracy, we performed a random internal consistency check on 50 patients; the resulting 0.82 concordance rate (Cohen's kappa) demonstrated the high reliability of our radiographic data.

### 2.5 Exclusion Criteria

We maintained strict recruitment standards to isolate the inflammatory impact of MetS. Participants were ineligible if they presented with acute systemic inflammation (fever >38.0°C or marked leukocytosis) or had undergone major surgery in the preceding month. We also ruled out individuals with autoimmune pathologies, active cancers, or severe organ failure (ESRD/dialysis) to prevent confounding. Given the sensitivity of the SIRI index, patients using steroids, biologics, or hormonal agents within 90 days were omitted. Finally, we excluded pregnant women and those with anatomical-based secondary stones or large staghorn calculi (>30 mm).

### 2.6 Sample Collection and Laboratory Analysis

In the morning, all subjects were fasting for more than 8 hours and collected midstream urine and venous blood samples. The Clinical Laboratory of the Second Affiliated Hospital of Guilin Medical University analyzed the samples according to the standard operating procedures.

In order to avoid the impact of urine concentration and dilution on the results, all urine samples were analyzed to determine the concentration of creatinine. The results are expressed as mg/g Cr. The formula for calculating the corrected value is as follows:

$$\text{Corrected Value}=\frac{[\text{Measured Analyte Concentration}]}{[\text{Urinary Creatinine Concentration}]} \quad (1)$$

The implementation of this strategy can make a reasonable comparison of the metabolic parameters of individuals with different hydration, and can also reflect the true excretion of urinary solute.

### 2.7 Statistical Analysis

We processed all analytical tasks using R software (version 4.4.1). After assessing data distribution, we applied parametric or non-parametric tests as appropriate, alongside $\chi^2$ tests for proportions. Our modeling strategy utilized a two-stage selection process: initially, we introduced LASSO regression to distill candidate markers while suppressing multicollinearity, fine-tuning the penalty parameter ($\lambda$) through 10-fold cross-validation. To maintain statistical integrity, we strategically omitted primary MetS components from this regression to prevent redundancy. Variables retained from LASSO were then integrated into a multivariable logistic framework to derive Odds Ratios. We benchmarked the resulting nomogram's discriminative power via AUC and mapped its practical net benefit using Decision Curve Analysis (DCA).

## 3 RESULTS

### 3.1 Comparison of Patient Profiles and Biomarkers

Significant disparities emerged between the comorbid MetS-urolithiasis (n = 246) and isolated MetS (n = 632) cohorts. Patients in the urolithiasis group were notably older (54.5 ± 11.2 vs. 49.8 ± 10.8 years) and showed a distinct male predominance (65.4% vs. 54.1%, $P < 0.05$; Table 1). Regarding urinary chemistry, the comorbid group exhibited higher uric acid (457.55 ± 147.92 vs. 419.36 ± 146.72 mg/g Cr) and lower citrate levels (339.54 ± 113.18 vs. 353.52 ± 114.58 mg/g Cr, $P<0.01$).

Importantly, systemic inflammatory burden was markedly higher in the stone-forming group. Beyond conventional markers (hs-CRP and IL-6), all composite indices (specifically NLR, PLR, SII, and SIRI) were significantly elevated (all $P < 0.001$). The SIRI index showed the most substantial variance (1.51 ± 0.87 vs. 1.18 ± 0.80), suggesting a potent link between integrated myeloid inflammation and lithogenesis.

### 3.2 Selection of Predictors and Regression Analysis

**Table 2** Independent Risk Factors for Urolithiasis in Patients with MetS

| Variables | β | OR | 95%CI | | P |
|---|---|---|---|---|---|
| Gender | 0.71 | 2.03 | 1.45 | 2.85 | P<0.001 |
| Age | 0.06 | 1.06 | 1.05 | 1.08 | P<0.001 |
| Urinary Uric Acid | 0.19 | 1.21 | 1.06 | 1.39 | P<0.01 |
| Urinary Citrate | -0.39 | 0.675 | 0.56 | 0.83 | P<0.001 |
| High-Sensitivity C-Reactive Protein (mg/L) | 0.16 | 1.18 | 1.09 | 1.28 | P<0.001 |
| IL-6(pg/mL) | 0.05 | 1.06 | 1.024 | 1.09 | P<0.001 |
| SIRI | 1.81 | 1.80 | 1.49 | 2.18 | P<0.001 |

To refine our predictive framework and resolve multicollinearity, we applied LASSO regression to 18 candidate variables. Utilizing 10-fold cross-validation and the 1-SE criterion (λ1se = 0.0315), we distilled these into seven robust predictors: SIRI, IL-6, hs-CRP, age, gender, urinary uric acid, and urinary citrate (Figure 1a & 1b).
Subsequent multivariable logistic regression confirmed SIRI as a dominant independent risk factor (adjusted OR = 1.80, 95% CI: 1.49–2.18, P < 0.001). Other critical drivers included male (OR = 2.03) and advanced age (OR = 1.06), while urinary citrate remained a protective factor (OR = 0.68) (Figure 1c). These findings highlight that a SIRI-quantified immune-inflammatory imbalance significantly escalates urolithiasis risk within the MetS population (Table 2).

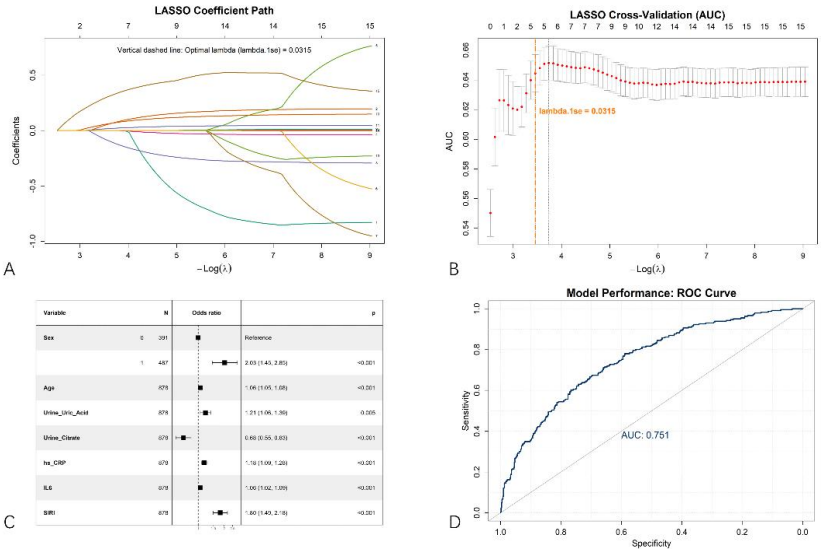


**Figure 1** (a) LASSO Regression Coefficient Path Plot; (b) 10-Fold Cross-Validation Curve for LASSO Regression; (c) Forest Plot of Multivariate Logistic Regression for Independent Predictors of MetS-Associated Urolithiasis; (d) Receiver Operating Characteristic (ROC) Curve for Discriminative Performance of the Final Predictive Nomogram

### 3.3 Nomogram Construction and Validation

In the primary cohort, this model demonstrated superior discriminative power with an AUC of 0.751 (95% CI: 0.716–0.787) (Figure 1d). The distribution of predicted probabilities further verified the good discrimination ability of the prediction model (Figure 2a). The predicted probability of urolithiasis was mainly concentrated in the low-risk range (0-0.3) in patients with MetS alone, while it was mostly concentrated in the medium-to-high risk range (0.2-0.7) in patients with MetS combined with urolithiasis.

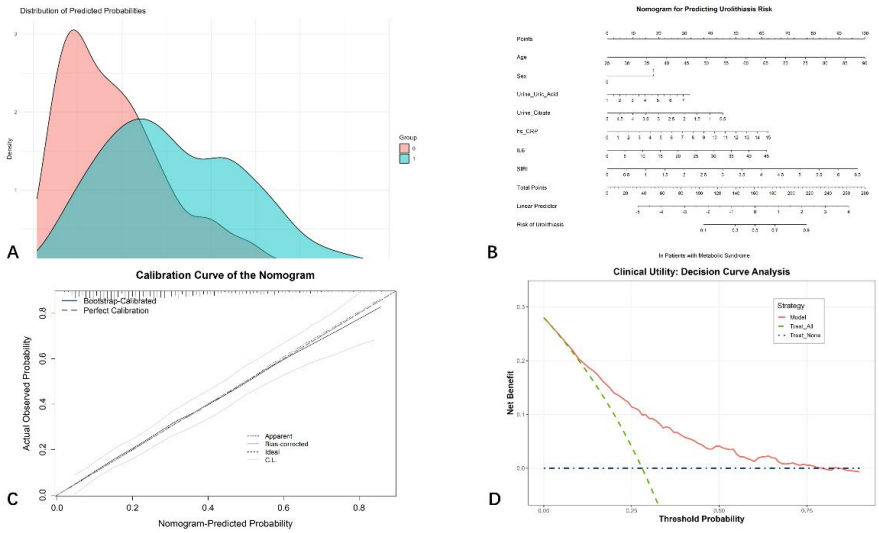


**Figure 2** (a) Predicted Probability Density Distribution Plot; (b) Predictive Nomogram for MetS-Associated Urolithiasis; (c) Calibration Plot of the Final Predictive Nomogram; (d) Decision Curve Analysis (DCA) for Clinical Utility of the Predictive Nomogram

We integrated these independent predictors into a clinical nomogram, a clinical nomogram was developed to provide a visual and individualized tool for urolithiasis risk estimation (Figure 2b). Calibration plots, utilizing 1,000 bootstrap

resamples, indicated high consistency between predicted and observed probabilities (Mean Absolute Error = 0.006) (Figure 2c).
Decision Curve Analysis (DCA) confirmed substantial clinical net benefit across a threshold probability range of 15% to 75% (Figure 2d).

### 3.4 Temporal External Validation

Temporal external validation (n = 111) further substantiated the model's transportability. Despite the independent nature of the validation set, the AUC remained robust at 0.725 (95% CI: 0.617–0.833; Figure 3a). Calibration and DCA in this cohort mirrored the primary results, with a calibration intercept of 0.048, indicating no significant risk overestimation. This robust discrimination was further evidenced by the distribution of predicted probabilities (Figure 3b).

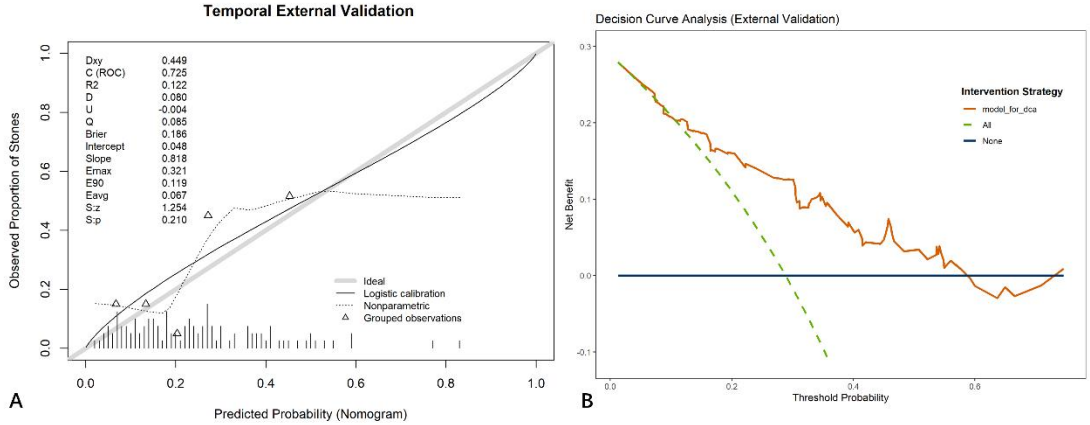


**Figure 3** (a) Calibration Plot of the External Validation Nomogram; (b) Decision Curve Analysis of the Predictive Nomogram in the Independent External Validation Cohort

## 4 DISCUSSION AND CONCLUSION

Urolithiasis is a serious problem in the field of global health, and the incidence in southern China, especially Guangdong and Guangxi, is relatively high [8]. In the process of lifestyle change, the incidence of urolithiasis and metabolic syndrome is increasing, which requires an in-depth analysis of the "inflammation-metabolism" axis [9]. In this study, multivariate modeling and LASSO-based feature selection are used to analyze that SIRI is the most critical risk factor for urolithiasis in patients with metabolic syndrome. A clinical nomogram was constructed, which had strong discrimination ability (AUC=0.751) and good calibration (MAE=0.006).
In this study, it is further proved that chronic low-grade inflammation is an important bridge between stone formation and metabolic disorders, and the same conclusion is also drawn in the previous study [7, 10]. SIRI, can accurately grasp the changes in lymphocytes, monocytes and neutrophils, and its predictive power is obviously higher than that of traditional markers, which is enough to show its superiority, because it can grasp the changes in the whole immune system.
Monocytes and M1 macrophages are key mediators of calcium oxalate formation [11]. In the MetS environment, the above two types of cells can seep into the renal interstitium and secrete a variety of pro-inflammatory cytokines, including IL-6 and TNF-α [12], which can promote the occurrence of EMT and form Randall plaques, which are the basis for crystal aggregation [13]. In addition, activated neutrophils can also release a large number of ROS, causing oxidative stress damage to renal tubular cells [14]. The occurrence of this kind of cell injury can increase the expression of adhesion molecules, including osteopontin and hyaluronic acid, and promote the nucleation of calcium salt and uric acid [15]. It is worth noting that the lymphocyte depletion in the comorbid group can lead to the decline of anti-inflammatory adaptive response, and the deposition of crystals can be promoted [16].
We found that patients with metabolic syndrome have a common feature, that is, the uric acid content in urine is relatively high and citrate is relatively low. When the uric acid content in the urine increases, the pH value will decrease, and the urate will be in a supersaturated state, which will also provide a basis for the formation of calcium oxalate crystals [3, 17]. Citrate in urine can effectively inhibit the formation of crystals [17]. The relationship between SIRI and citrate is negative, which indicates that the patient's body is in a state of systemic inflammation, which will lead to a series of changes in acid-base transporters in the kidneys, and the citrate reabsorption capacity will increase significantly, which will weaken the inhibitory capacity of urine [18]. It should be noted that there is no significant difference in the excretion of oxalate between the two groups, which is different from the conclusions of Western scholars [19]. The reason for this phenomenon may be related to the geographical environment and diet of the patients, and the metabolism of oxalate alone may not be the main factor [20-21].
In the process of building a model, we strictly abide by the TRIPOD statement, and select seven variables, including age, gender, hs-CRP, SIRI and so on. Through LASSO regression, the model has strong robustness, and the relationship

between the model and clinical practice is analyzed, which is more reasonable. The results show that the incidence of the disease in male patients is higher than that in female patients, which is related to the low citrate level and heavy metabolic burden [22]. With the increase of age, the incidence of the disease increases, which is related to the decline of kidney function and the increase of inflammatory factors [3, 23].

The most prominent advantage of this model is that it has strong accessibility. Some models need to pay a higher price for molecular experiments, while this model can get standard experimental results in the laboratory, which is conducive to its promotion and application in primary and tertiary hospitals in China. Decision Curve Analysis (DCA) is used to analyze the data, and the threshold range is 15-75%, which is significantly better than the traditional strategy.

## 5 COMPARISON WITH PRIOR WORK AND LIMITATIONS

In this study, the results are consistent with those of scholars at home and abroad, which are enough to show that systemic inflammation has a significant impact on the occurrence and development of stones [5, 7]. However, in the previous research, NLR and PLR are the focus of attention [24-25], but this paper focuses on SIRI and believes that it can more objectively reflect the chronic inflammatory state of MetS.

Although the research has strong advantages, it also has some shortcomings, only a single center retrospective study can not draw a certain causal relationship, and the research sample is only in a certain region, which is not conducive to the promotion of the research results to other groups. Also, the baseline value can not reflect the changes of inflammatory markers dynamically. In the process of research, we have carried out external validation, but we need to carry out multi-center research to improve the accuracy of the model and analyze the biological mechanism.

## COMPETING INTERESTS

The authors have no relevant financial or non-financial interests to disclose.

## FUNDING

This research was funded by the Self-Funded Scientific Research Projects of the Guangxi Regional Health Commission, Z-C20240938 funding, and the Open Project of Guangxi Key Laboratory of Tumor Immunity and Microenvironment Regulation, 2024KF009.

## AUTHOR CONTRIBUTIONS

Conceptualization, FM.X. and L.G.; methodology, FM.X.; software, FM.X.; validation, QM.X. and R.K.Y.; formal analysis, FM.X.; investigation, S.B.C.; resources, L.G.; data curation, M.X., L.Y.; writing—original draft preparation, FM.X.; writing—review and editing, QM.X., L.G.; visualization, FM.X., SB.C.; supervision, SB.C.; project administration, L.G.; funding acquisition, FM.X. All authors have read and agreed to the published version of the manuscript.

Fangming Xu and Qiming Xu contributed equally to this work.

## ETHICAL APPROVAL

In the process of this study, the Ethics Committee approved the research activities, and all regulations were implemented in accordance with the requirements of the institution and the relevant laws and regulations of the country. In order to make a written commitment, all participants were required to sign before enrollment. The Ethics Committee of the Second Hospital of Guilin Medical University, No. QWJW-2024018.

## INFORMED CONSENT

Informed consent was obtained from all subjects involved in the study.

## REFERENCES

[1] Skolarikos A, Somani B, Neisius A, et al. Metabolic Evaluation and Recurrence Prevention for Urinary Stone Patients: An EAU Guidelines Update. Eur Urol, 2024, 86(4): 343-363. DOI: 10.1016/j.eururo.2024.05.029.

[2] Samson S L, Garber A J. Metabolic syndrome. Endocrinol Metab Clin North Am, 2014, 43(1): 1-23. DOI: 10.1016/j.ecl.2013.09.009.

[3] Bargagli M, Scoglio M, Howles S A, et al. Kidney stone disease: risk factors, pathophysiology and management. Nat Rev Nephrol, 2025, 21(11): 794-808. DOI: 10.1038/s41581-025-00990-x.

[4] Khan S R. Reactive oxygen species, inflammation and calcium oxalate nephrolithiasis. Transl Androl Urol, 2014, 3(3): 256-276. DOI: 10.3978/j.issn.2223-4683.2014.06.04.

[5] Di X, Liu S, Xiang L, et al. Association between the systemic immune-inflammation index and kidney stone: A cross-sectional study of NHANES 2007-2018. Front Immunol, 2023, 14: 1116224. DOI: 10.3389/fimmu.2023.1116224.

[6] Qi Q, Zhuang L, Shen Y, et al. A novel systemic inflammation response index (SIRI) for predicting the survival of patients with pancreatic cancer after chemotherapy. Cancer, 2016, 122(14): 2158-2167. DOI: 10.1002/cncr.30057.
[7] Zhang Z, Wang G, Dai X, et al. Association between the systemic inflammation response index and kidney stones in US adults: a cross-sectional study based on NHANES 2007-2018. Urolithiasis, 2024, 52(1): 165. DOI: 10.1007/s00240-024-01668-y.
[8] Tan S, Yuan D, Su H, et al. Prevalence of urolithiasis in China: a systematic review and meta-analysis. BJU Int, 2024, 133(1): 34-43. DOI: 10.1111/bju.16179.
[9] Ye Z, Wu C, Xiong Y, et al. Obesity, metabolic dysfunction, and risk of kidney stone disease: a national cross-sectional study. Aging Male Off J Int Soc Study Aging Male, 2023, 26(1): 2195932. DOI: 10.1080/13685538.2023.2195932.
[10] Weinberg A E, Patel C J, Chertow G M, et al. Diabetic severity and risk of kidney stone disease. Eur Urol, 2014, 65(1): 242-247. DOI: 10.1016/j.eururo.2013.03.026.
[11] Taguchi K, Okada A, Hamamoto S, et al. M1/M2-macrophage phenotypes regulate renal calcium oxalate crystal development. Sci Rep, 2016, 6: 35167. DOI: 10.1038/srep35167.
[12] Dominguez-Gutierrez P R, Kusmartsev S, Canales B K, et al. Calcium Oxalate Differentiates Human Monocytes Into Inflammatory M1 Macrophages. Front Immunol, 2018, 9: 1863. DOI: 10.3389/fimmu.2018.01863.
[13] Taguchi K, Hamamoto S, Okada A, et al. Genome-Wide Gene Expression Profiling of Randall's Plaques in Calcium Oxalate Stone Formers. J Am Soc Nephrol JASN, 2017, 28(1): 333-347. DOI: 10.1681/ASN.2015111271.
[14] Verma S, Singh P, Khurana S, et al. Implications of oxidative stress in chronic kidney disease: a review on current concepts and therapies. Kidney Res Clin Pract, 2021, 40(2): 183-193. DOI: 10.23876/j.krcp.20.163.
[15] Asselman M, Verhulst A, De Broe M E, et al. Calcium oxalate crystal adherence to hyaluronan-, osteopontin-, and CD44-expressing injured/regenerating tubular epithelial cells in rat kidneys. J Am Soc Nephrol JASN, 2003, 14(12): 3155-3166. DOI: 10.1097/01.asn.0000099380.18995.f7.
[16] Dejban P, Wilson E M, Jayachandran M, et al. Inflammatory Cells in Nephrectomy Tissue from Patients without and with a History of Urinary Stone Disease. Clin J Am Soc Nephrol CJASN, 2022, 17(3): 414-422. DOI: 10.2215/CJN.11730921.
[17] Moe O W, Preisig P A. Dual role of citrate in mammalian urine. Curr Opin Nephrol Hypertens, 2006, 15(4): 419-424. DOI: 10.1097/01.mnh.0000232882.35469.72.
[18] Leslie S W, Bashir K. Hypocitraturia and Renal Calculi. In: StatPearls. StatPearls Publishing, 2026. http://www.ncbi.nlm.nih.gov/books/NBK564392/ (accessed March 18, 2026).
[19] Siener R, Glatz S, Nicolay C, et al. Prospective study on the efficacy of a selective treatment and risk factors for relapse in recurrent calcium oxalate stone patients. Eur Urol, 2003, 44(4): 467-474. DOI: 10.1016/s0302-2838(03)00317-8.
[20] Siener R, Hesse A. The effect of different diets on urine composition and the risk of calcium oxalate crystallisation in healthy subjects. Eur Urol, 2002, 42(3): 289-296. DOI: 10.1016/s0302-2838(02)00316-0.
[21] Siener R. Nutrition and Kidney Stone Disease. Nutrients, 2021, 13(6): 1917. DOI: 10.3390/nu13061917.
[22] Xu J Z, Li C, Xia Q D, et al. Sex disparities and the risk of urolithiasis: a large cross-sectional study. Ann Med, 2022, 54(1): 1627-1635. DOI: 10.1080/07853890.2022.2085882.
[23] Grundy S M. Metabolic syndrome pandemic. Arterioscler Thromb Vasc Biol, 2008, 28(4): 629-636. DOI: 10.1161/ATVBAHA.107.151092.
[24] Tang Y, Zhang C, Mo C, et al. Predictive Model for Systemic Infection After Percutaneous Nephrolithotomy and Related Factors Analysis. Front Surg, 2021, 8: 696463. DOI: 10.3389/fsurg.2021.696463.
[25] Villanueva-Congote J, Hinojosa-Gonzalez D, Segall M, et al. The relationship between neutrophil/lymphocyte ratio, platelet/neutrophil ratio, and risk of urosepsis in patients who present with ureteral stones and suspected urinary tract infection. World J Urol, 2024, 42(1): 596. DOI: 10.1007/s00345-024-05229-1.