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Observational study

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Association between a high total cholesterol-to-high-density lipoprotein cholesterol ratio and hyperuricemia among patients with hypertension: A cross-sectional study from central Uganda

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Abstract

Background: Hyperuricemia is a cardiometabolic risk factor among patients with hypertension. A high total cholesterol-to-high-density lipoprotein cholesterol ratio reflects atherogenic lipid imbalance and insulin resistance. This study investigated the association between a high total cholesterol-to-high-density lipoprotein cholesterol ratio and hyperuricemia among patients with hypertension at Uganda Heart Institute.

Methods: We conducted a cross-sectional study among 277 consecutively recruited patients with hypertension aged ≥18 years from 4 August 2025 to 20 October 2025 at Uganda Heart Institute. Hyperuricemia was defined as a serum uric acid level of ≥7 mg/dL in men and ≥6 mg/dL in women, whereas a high total cholesterol-to-high-density lipoprotein cholesterol ratio was defined as ≥5.

Results: Among 277 participants, 89 had hyperuricemia, giving a proportion of 32.1% (95% confidence interval: 26.9–37.9). Hyperuricemia was significantly more common in participants with a total cholesterol-to-high-density lipoprotein cholesterol ratio of ≥5 (53.8%) than in those with a ratio of <5 (25.5%) ($p < 0.001$). After adjustment for confounders, a high total cholesterol-to-high-density lipoprotein cholesterol ratio remained independently associated with hyperuricemia (adjusted odds ratio = 2.83, 95% confidence interval: 1.31–6.09, $p = 0.008$). The total cholesterol-to-high-density lipoprotein cholesterol ratio demonstrated moderate predictive ability for hyperuricemia, with an optimal cutoff value of 4.33 (sensitivity, 58%; specificity, 71%) and an area under the curve of 0.68 (95% confidence interval: 0.61–0.75).

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Conclusion: Hyperuricemia is prevalent among patients with hypertension. A high total cholesterol-to-high-density lipoprotein cholesterol ratio is a potential predictor of hyperuricemia in this population.

Keywords
Hyperuricemia, hypertension, uric acid, total cholesterol-to-high-density lipoprotein cholesterol ratio, cardiometabolic risk

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Introduction

Hyperuricemia, defined as serum uric acid levels ≥7.0 mg/dL in men and ≥6.0 mg/dL in women,[1–3] is increasingly recognized as a metabolic disorder with multisystem consequences. It often accompanies insulin resistance and other components of metabolic syndrome, including hypertension.[2] In fact, hyperuricemia is common among patients with hypertension and predicts worsening disease; it has been shown to be an important predictor of hypertension progression, and hypertensive patients with hyperuricemia are at higher risk of cardiovascular complications.[4] Biologically, uric acid metabolism and blood pressure regulation are intertwined. Because two-thirds of urate is excreted by the kidneys, hypertension-related renal changes favor its accumulation, whereas elevated urate can activate the renin–angiotensin system and impair endothelial function, thereby increasing blood pressure.[5] At high levels, urate shifts from an antioxidant to a pro-oxidant, stimulating inflammation through innate immune signaling and oxidative stress.[1] Thus, hypertension predisposes individuals to hyperuricemia, which in turn may amplify vascular injury and sustain hypertension.[1,5]

Epidemiologically, hyperuricemia is an emerging global public health concern,[2] with prevalence rising alongside urbanization and westernized dietary patterns. In the United States, approximately 11% of the population is affected,[2] and similarly increasing trends have been reported worldwide. Among hypertensive populations, the burden is substantial. Studies from China report hyperuricemia prevalence of 18.2% among older hypertensive adults,[6] whereas even higher rates have been observed in sub-Saharan Africa. For instance, hyperuricemia has been reported in 31.8% of newly diagnosed patients with hypertension in Cameroon,[7] whereas 59.8% of treated patients with hypertension in Tanzania had hyperuricemia.[8] Data from Uganda remain limited, but available evidence suggests a considerable burden of elevated serum uric acid. Importantly, untreated hyperuricemia in patients with hypertension can have serious sequelae: it predisposes individuals to gout and nephrolithiasis and contributes to chronic kidney disease and cardiovascular events.[2,5] Thus, failure to diagnose or manage hyperuricemia may worsen renal and vascular outcomes in hypertension.

Dyslipidemia frequently coexists with hypertension as part of metabolic syndrome,[9] largely driven by underlying insulin resistance. It is estimated that up to 50% of patients with hypertension have insulin-resistant dyslipidemia,[9] and population-based studies indicate that more than 80% of adults with hypertension have at least one atherogenic lipid abnormality.[9] Contemporary studies confirm the high prevalence of dyslipidemia in hypertension; for example, 86.4% of patients with hypertension in Iran had dyslipidemia, most commonly low high-density lipoprotein cholesterol (HDL-C).[10] The coexistence of dyslipidemia and hypertension markedly amplifies cardiovascular risk, likely through shared mechanisms including endothelial dysfunction, chronic inflammation, oxidative stress, and arterial stiffness.

Despite the recognized coexistence of hypertension, dyslipidemia, and hyperuricemia, and their contribution to increased cardiovascular and renal disease risk, there is a paucity of evidence from sub-Saharan Africa examining the relationship between lipid abnormalities and hyperuricemia among patients with hypertension. Most available studies among hypertensive populations have primarily focused on the prevalence of hyperuricemia or isolated lipid abnormalities, with limited evaluation of the association between lipid abnormalities and hyperuricemia in patients with hypertension. In Uganda, evidence on the burden of hyperuricemia among patients with hypertension is scarce, and no published study has specifically examined the association between atherogenic lipoprotein ratios and hyperuricemia in this high-risk population. This represents an important research gap because patients with hypertension in Uganda are increasingly exposed to urbanization-related lifestyle changes, including unhealthy diets, obesity, and physical inactivity, all of which predispose them to both dyslipidemia and elevated serum uric acid levels. Furthermore, resource-limited settings require simple, affordable, and clinically useful markers for the early identification of patients at increased risk of adverse cardiovascular and renal outcomes. The total cholesterol-to-high-density lipoprotein cholesterol (TC/HDL-C) ratio is an inexpensive and readily available indicator of atherogenic dyslipidemia and cardiometabolic risk; however, its relationship with hyperuricemia has not been investigated among patients with hypertension in Uganda. Generating local evidence on this

association may improve risk stratification and support the early identification of patients with hypertension who could benefit from targeted metabolic and cardiovascular risk-reduction interventions. Therefore, this study aimed to investigate the association between the TC/HDL-C ratio and hyperuricemia among patients with hypertension attending the Uganda Heart Institute (UHI).

Material and methods

The reporting of this study conforms to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational studies.[11]

Study design, population, and eligibility criteria

We conducted a hospital-based descriptive and analytical cross-sectional study among patients with hypertension at the UHI from 4 August 2025 to 20 October 2025. As a national referral facility for cardiology services, the UHI provides an ideal setting for accessing a large and diverse population of patients with hypertension from various regions of Uganda. All adult patients aged 18 years and above with a known diagnosis of hypertension who provided written informed consent were consecutively enrolled in the study. Patients receiving treatment for hyperuricemia, individuals with severe physical or communication impairments preventing reliable participation, patients presenting with acute cardiac emergencies, and pregnant or recently postpartum women were excluded due to potential physiological alterations affecting serum uric acid levels and anthropometric measurements.

Sample size determination and sampling technique

The sample size was calculated using the Kish and Leslie formula for estimating a single population proportion.[12] Because no published Ugandan study had previously reported the prevalence of hyperuricemia among patients with hypertension at the time of study design, a prevalence of hyperuricemia of 23.48% was assumed, based on findings from a previous study conducted among people living with human immunodeficiency virus (PLWH) in Southwestern Uganda.[13] This population was considered an appropriate reference because PLWH in Uganda have a high burden of hypertension and other cardiometabolic abnormalities, including metabolic syndrome and renal dysfunction, which are also closely associated with hyperuricemia. Furthermore, the use of locally generated prevalence estimates from a population with similar cardiometabolic risk profiles was considered more appropriate than using estimates from geographically or clinically different populations. A 95% confidence level was applied, corresponding to a Z value of 1.96, with a margin of error (d) of 5%.

The sample size was calculated using the following formula:

$$\frac{n = Z^2 P\,(1-P)}{d^2}$$

$$\frac{n = 1.96^2 * 0.2348(1-0.2348)}{0.052}$$

$$n = 276.1$$

Thus, a minimum sample size of 276 participants was required for the study.

A consecutive sampling technique was used to recruit study participants. In this approach, all eligible patients with hypertension attending the outpatient clinic at the UHI during the study period were approached consecutively and enrolled until the required sample size was achieved. Participants were screened for eligibility based on the predefined inclusion and exclusion criteria before enrollment.

Study variables and data collection tools

The primary outcome variable of this study was hyperuricemia, defined as serum uric acid serum uric acid ≥7 mg/dL in men and ≥6 mg/dL in women.[14] The main independent variable was the TC/HDL-C ratio, defined as a ratio ≥5.[15] Total cholesterol was considered high when TC levels were ≥200 mg/dL, whereas HDL-C was considered low when the levels were <40 mg/dL for men and <50 mg/dL for women.[15]

The other independent variables were categorized as sociodemographic factors (age, sex, marital status, religion, education, and residence), comorbidities (diabetes mellitus, human immunodeficiency virus (HIV), thyroid disease, heart

disease, and kidney disease), lifestyle factors (smoking, alcohol consumption, red meat consumption, physical activity, sleep duration, and obstructive sleep apnea), family history of chronic conditions (hypertension, diabetes mellitus, cardiovascular disease(CVD), and kidney disease), hypertension-related factors (adherence to antihypertensive medication, type of antihypertensive medication, duration of hypertension, duration on antihypertensive medication, hypertension stage, and systolic and diastolic blood pressure), and anthropometric factors (body mass index (BMI), waist circumference, and waist-to-hip ratio).

Data on sociodemographic factors, comorbidities, lifestyle factors, family history of chronic conditions, and hypertension-related factors were collected from consenting participants using a structured questionnaire composed of standard tools and review of patients' medical records. Physical activity was assessed using the International Physical Activity Questionnaire and categorized as <600, 600–3000, and >3000 metabolic equivalent of task (MET)-min/week,[16] indicating low, moderate, and high physical activity, respectively. Adherence to antihypertensive medication was assessed using the Morisky Medication Adherence Scale (MMAS-8) and categorized as low adherence (score <6), moderate adherence (score 6–7), and high adherence (score 8).[17] Sleep duration was categorized as <5, 5–7, and >7 h.[18] Obstructive sleep apnea (OSA) was assessed using the STOP-Bang scoring model, and high risk of OSA was defined as answering Yes to three or more items in the tool.[19]

Systolic and diastolic blood pressure as well as anthropometric measurements including height (cm), weight (kg), waist circumference, and hip circumference were obtained by trained investigators following standardized procedures. Blood pressure was measured using a calibrated digital sphygmomanometer with participants in a seated position after a minimum rest period of 5 min. Two blood pressure readings were taken at 5-min intervals, and the mean of the two measurements was used for analysis. High blood pressure was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg.[20] Body weight and height were measured using a stadiometer with participants wearing light clothing and no shoes. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). BMI was categorized as <25 kg/m^2, 25–29.9 kg/m^2, and ≥30 kg/m^2.[21] Waist and hip circumferences were measured using a non-stretchable Seca® 201 ergonomic measuring tape. Two measurements were obtained for each circumference; if the difference between the two measurements exceeded 3 cm, a third measurement was taken. The final waist and hip circumference values were recorded as the mean of the two closest measurements. The waist-to-hip ratio was calculated by dividing waist circumference by hip circumference. Waist circumference was considered high if the measurement was >80 cm for women and >94 cm for men.[22] Waist-to-hip ratio was considered high if the ratio was ≥0.85 for women and ≥0.9 for men.[22]

Approximately 5 ml of venous blood sample was collected from each participant into red-top vacutainer tube by the principal investigators. Within 30 min of collection, samples were centrifuged at 3000 r/min for 8 min to separate serum from cellular components. Serum uric acid, total cholesterol, and HDL-C were then measured using a well-calibrated and quality-controlled automated clinical chemistry analyzer (Accent M320: Cormay; Poland) at the UHI Laboratory.

Management of missing data

Eligible patients with hypertension were consecutively recruited throughout the study period until the minimum required sample size of 276 was attained. To minimize nonresponse and incomplete data, participants were guided through the data collection procedures by trained research staff, and questionnaires and laboratory request forms were checked for completeness at the point of data collection.

Participants with missing or incomplete data for key study variables were excluded from the final analysis and were consecutively replaced with other eligible participants who provided complete data. Consequently, only participants with complete information for all variables included in the analysis were considered in this study. This approach ensured completeness of the dataset and maintained the required statistical power for the study analyses.

Statistical analysis

Data were analyzed using Stata version 17. Categorical variables were summarized using frequencies and proportions. The prevalence of hyperuricemia was calculated as the proportion of study participants with hyperuricemia and expressed as a percentage. The distribution of hyperuricemia across different levels of categorical lipid profile parameters was compared using the chi-squared test. A p-value <0.05 was considered statistically significant. Age was tested for normality using the Shapiro–Wilk normality test and was normally distributed ($p = 0.83141$); therefore, it was summarized using mean ± SD.

Logistic regression was used to assess the association between the main independent variable (high TC/HDL-C ratio) and the binary outcome variable (hyperuricemia: 1 = present, 0 = absent). At the bivariate level, crude associations between hyperuricemia and each independent variable, including the TC/HDL-C ratio, were evaluated. Associations were quantified using crude odds ratios (cORs) with corresponding 95% confidence intervals (CIs), and statistical significance was set

at a p <0.2. The primary independent variable and other independent variables that were statistically and/or clinically significant at this level were included in the multivariable model to adjust for potential confounding. Adjusted ORs (aORs) with 95% CIs were reported and p-values were presented. The model's goodness of fit was assessed using the Hosmer–Lemeshow test, with p >0.05 indicating a good fit (p = 0 0.5603 in the final model). The final model was also tested for multicollinearity, with a mean variance inflation factor (VIF) of 1.71, which was below the acceptable threshold of 5. In the final multivariable model, associations were considered statistically significant at p <0.05.

To evaluate the predictive performance of the TC/HDL-C ratio for hyperuricemia, receiver operating characteristic (ROC) curve analysis was performed. The area under the ROC curve (AUC) was calculated to quantify the overall discriminatory ability of the TC/HDL-C ratio in distinguishing participants with and without hyperuricemia. An AUC value close to 1.0 indicates excellent discriminatory performance, whereas an AUC of 0.5 reflects performance no better than chance. Predictive performance was considered statistically significant when the 95% CI of the AUC did not include 0.5. Using the Youden index (J) method, we also determined the optimal cutoff for serum uric acid, with corresponding sensitivity and specificity, above which hyperuricemia is likely to be present in a patient with hypertension.

Ethical approval and written informed consent

Ethical approval for this study was granted by Mbarara University of Science and Technology Faculty Research Committee (FRC) (Approval Number: MUST/MLS/025–01U). Prior to conducting the study, administrative clearance was also obtained from the Research Committee of the UHI (Ref: ADM 100/110/01). Written informed consent was obtained from each participant before enrollment. The study was conducted in accordance with the Declaration of Helsinki 1975, as revised in 2024. Each study participant was assigned a unique code that could not be linked to their identity to ensure confidentiality. Participants were informed that their participation was entirely voluntary and that they could withdraw from the study at any time without penalty.

Results

Study flow chart

The study flow chart is presented in Figure 1.

Narrative of the study flow chart. A total of 302 patients were assessed for eligibility at the UHI. Twenty-five patients did not meet the inclusion criteria or declined participation, leaving 277 participants who were consecutively enrolled and analyzed. Of these, 212 (76.5%) had a TC/HDL-C ratio <5 and 65 (23.5%) had a TC/HDL-C ratio ≥5. Hyperuricemia was identified in 54 (25.5%) participants with a low TC/HDL-C ratio and in 35 (53.8%) participants with a high TC/HDL-C ratio.

Sociodemographic characteristics of the study participants

A total of 277 patients with hypertension participated in this study with a mean age of 54.5 ± 13.5 years (Table 1). The majority were women (75.5%), whereas men accounted for 24.5%. More than half of the participants (55.6%) were married and living together, whereas 17.7% were widowed, 12.3% divorced/separated, 9.7% married but not living together, and 4.7% had never been married. Regarding education, 34.3% had attained primary education, 31.8% secondary education, and 23.5% tertiary education, whereas 10.5% had no formal education. In terms of religion, 37.9% were Protestant, 32.9% Catholic, 14.8% Muslim, and 14.4% belonged to other denominations. The main source of livelihood was peasantry (35.4%), followed by self-employment (30.0%), unemployment (18.8%), and professional occupations (15.9%). Slightly more participants resided in urban areas (54.2%) than in rural settings (45.8%).

Prevalence of hyperuricemia among patients with hypertension

Out of 277 study participants, 89 had hyperuricemia, giving an overall proportion of 32.1% with a 95% CI of 26.9%–37.9% (Figure 2). Participants with low HDL-C levels showed a significantly higher proportion of hyperuricemia (38.8%) compared with those with normal HDL-C levels (24.6%) (p = 0.012). Similarly, a markedly higher proportion of hyperuricemia was observed among participants with a TC/HDL-C ratio ≥5 (53.8%) compared with those with a ratio <5 (25.5%) (p < 0.001). However, no significant difference was found in the proportion of hyperuricemia across total cholesterol categories (<200 mg/dL vs. ≥ 200 mg/dL, p = 0.260), as indicated in Table 2.

Table 1. Sociodemographic characteristics of the study participants.

Variable	Total n = 277
Age (years): mean ± SD	54.46 ± 13.50
Sex	
Male	68 (24.5%)
Female	209 (75.5%)
Marital status	
Married together	154 (55.6%)
Married not together	27 (9.7%)
Divorced/Separated	34 (12.3%)
Widowed	49 (17.7%)
Never married	13 (4.7%)
Education	
None	29 (10.5%)
Primary	95 (34.3%)
Secondary	88 (31.8%)
Tertiary	65 (23.5%)
Religion	
Catholic	91 (32.9%)
Protestant	105 (37.9%)
Muslim	41 (14.8%)
Others	40 (14.4%)
Employment	
Peasant	98 (35.4%)
Self-employed	83 (30.0%)
Professional	44 (15.9%)
Unemployed	52 (18.8%)
Residence	
Rural	127 (45.8%)
Urban	150 (54.2%)

Distribution of hyperuricemia prevalence in the different levels of lipid parameters

Total cholesterol levels were not significantly related to hyperuricemia prevalence. Although a higher proportion of participants with elevated total cholesterol (≥200 mg/dL) had hyperuricemia compared with those with normal levels (<200 mg/dL) (36.5% vs. 29.8%), this difference did not reach statistical significance ($p = 0.260$).

In contrast, significant differences were observed with respect to HDL-C levels and the TC/HDL-C ratio. Participants with low HDL-C had a significantly higher prevalence of hyperuricemia compared with those with normal HDL-C (38.8% vs. 24.6%, $p = 0.012$). Similarly, participants with an elevated TC/HDL-C ratio (≥5) exhibited a markedly higher prevalence of hyperuricemia compared with those with a ratio <5 (53.8% vs. 25.5%, $p < 0.001$), as indicated in Table 2.

Association between TC/HDL-C ratio and hyperuricemia among patients with hypertension

At bivariate analysis, there was a significant association between a high TC/HDL-C ratio and hyperuricemia (cOR = 3.41, 95% CI: 1.92–6.08, $p < 0.001$). Even after adjusting for potential confounders (Table 3), a high TC/HDL-C ratio remained significantly and independently associated with hyperuricemia. Participants with an elevated TC/HDL-C ratio had nearly threefold higher odds of hyperuricemia compared with those with a lower ratio (aOR = 2.83, 95% CI: 1.31–6.09, $p = 0.008$). At the optimal cutoff point of 4.33, with a sensitivity of 58% and specificity of 71%, the TC/HDL-C ratio demonstrated significant predictive ability, with an AUC of 0.68 (95%CI: 0.61–0.75), indicating moderate discrimination between participants with and without hyperuricemia, as shown in Figure 3. Similarly, individuals with heart disease had more than twice the odds of developing hyperuricemia compared with those without such a history (aOR = 2.38, 95% CI: 1.23–4.59, $p = 0.010$). Moreover, participants who had ever smoked had a markedly increased likelihood of hyperuricemia, with over fourfold higher odds compared with never-smokers (aOR = 4.20, 95% CI: 1.47–12.02, $p = 0.007$).

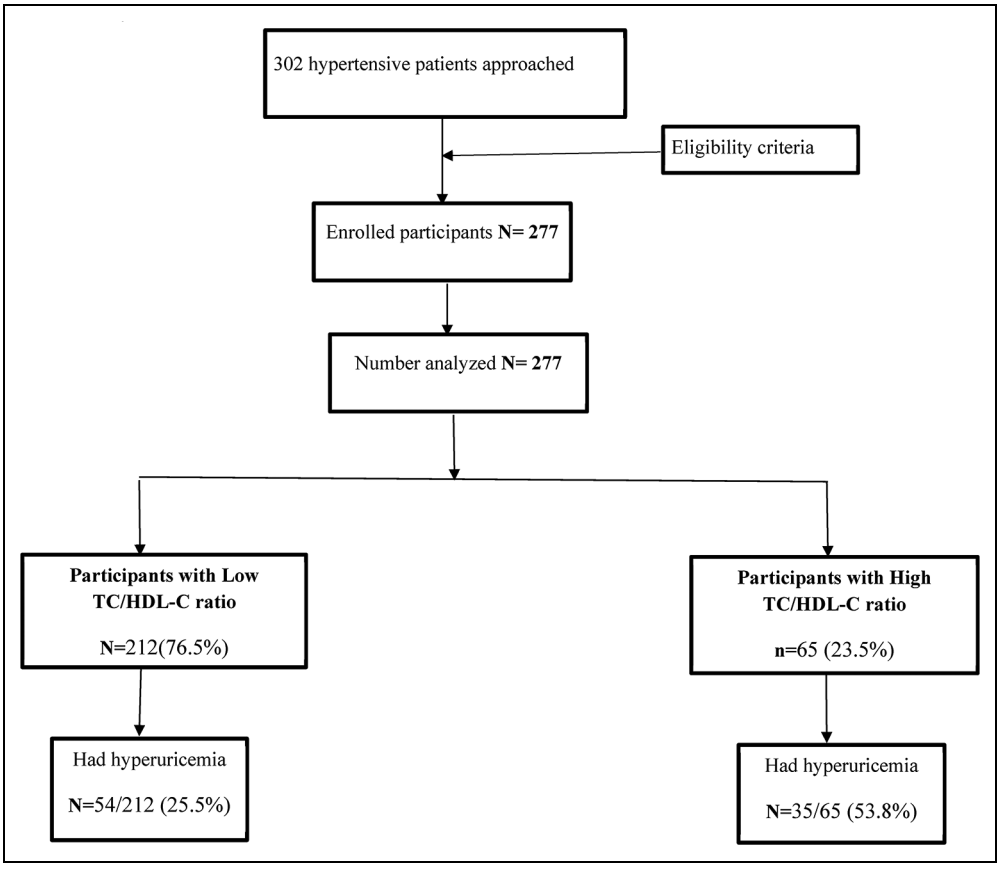


Figure 1. Study flow chart.

Discussion

In our cross-sectional study of 277 patients with hypertension attending outpatient services in Uganda, 89 (32.1%) had hyperuricemia, defined as serum uric acid ≥7.0 mg/dL in men and ≥6.0 mg/dL in women. This proportion indicates that approximately one-third of patients with hypertension in this setting had elevated serum uric acid levels, highlighting hyperuricemia as a common metabolic abnormality among individuals with hypertension. Hyperuricemia is known to induce endothelial dysfunction and oxidative stress and often coexists with metabolic syndrome and chronic kidney disease.[23] It also increases the risk of gout and contributes to renal injury, thereby compounding cardiovascular risk. Notably, many patients with hypertension receive diuretics or other medications that elevate uric acid, further increasing this burden. Thus, our findings, showing that approximately one-third of patients had hyperuricemia, suggest that routine screening for hyperuricemia and appropriate interventions in hypertension clinics may be warranted.

Our study prevalence is higher than that documented in general or HIV-infected Ugandan populations and reflects the broader trend that patients with hypertension bear an increased risk of elevated serum uric acid. For comparison, a recent Ugandan study of adults with HIV reported a hyperuricemia prevalence of 21.3%.[24] Similarly, United States National Health and Nutrition Examination Survey (US NHANES) data indicate that approximately 21% of US adults have hyperuricemia,[25] and an Angolan community survey found an overall prevalence of about 25%.[26] Even among untreated patients with hypertension worldwide, only about 25% are hyperuricemic.[3] By contrast, our hypertensive cohort's prevalence of 32.1% is markedly higher than these benchmarks, implying an increased risk of gout, chronic kidney disease, and cardiovascular events. In practical terms, this elevation underscores the potential value of uric acid monitoring in hypertensive care and tailored management to mitigate complications in this population.

Compared with other African hypertension cohorts, our finding falls within the mid-range of reported prevalences. For instance, a study of newly diagnosed patients with hypertension in Cameroon found a hyperuricemia prevalence of 31.8%[7] and a clinic-based study from Wolkite, Ethiopia reported 27.4%,[27] both of which are similar to our estimate of 32.1%. In contrast, much higher rates have been reported elsewhere. A Tanzanian outpatient study recorded a prevalence of 59.8%,[8] whereas Nigerian surveys have reported approximately 47%–59% among patients with hypertension.[28,29] These

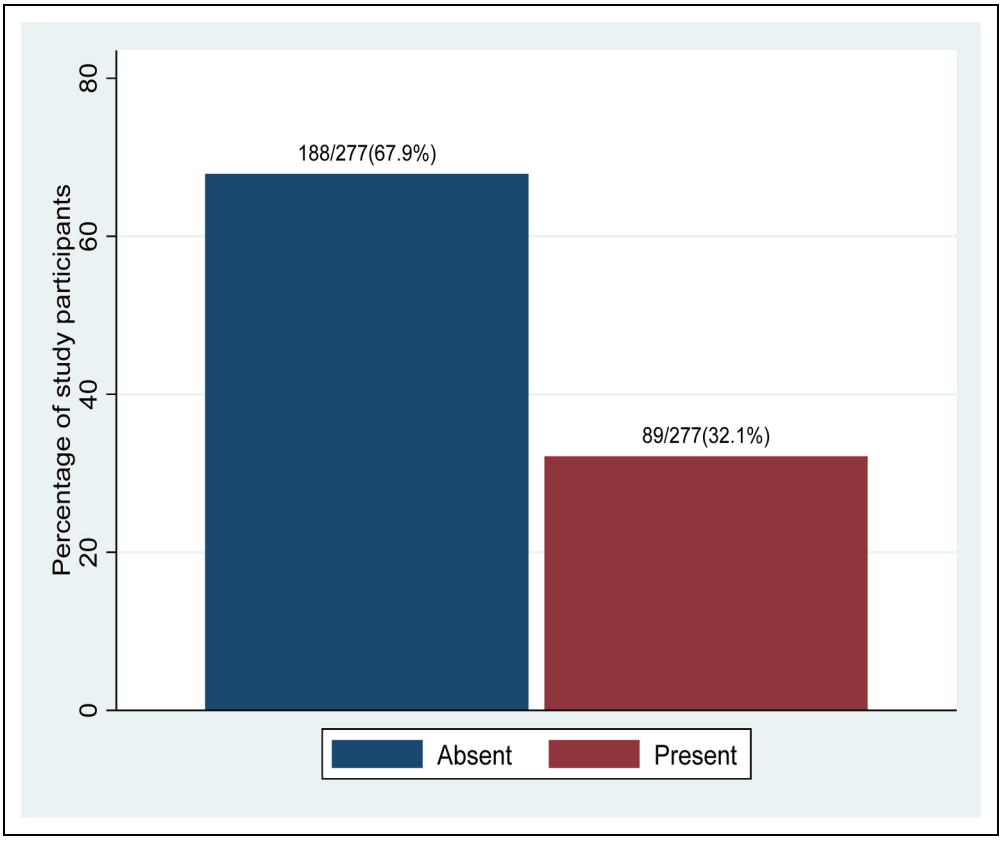


Figure 2. A bar graph showing the proportion of study participants with hyperuricemia.

Table 2. Distribution of hyperuricemia prevalence across lipid parameter levels.

Variable	Total n = 277	Hyperuricemia		p-value
		Absent n = 188	Present n = 89	
Total cholesterol (mg/dL)				0.260
<200	181 (65.3%)	127 (70.2%)	54 (29.8%)	
≥200	96 (34.7%)	61 (63.5%)	35 (36.5%)	
HDL-C (mg/dL)				**0.012**
High	130 (46.9%)	98 (75.4%)	32 (24.6%)	
Low	147 (53.1%)	90 (61.2%)	57 (38.8%)	
TC/HDL-C ratio				**<0.001**
<5	212 (76.5%)	158 (74.5%)	54 (25.5%)	
≥5	65 (23.5%)	30 (46.2%)	35 (53.8%)	

Bolded values indicate statistically significant results ($p < 0.05$).
HDL-C: high-density lipoprotein cholesterol; TC/HDL-C: total cholesterol-to-high-density lipoprotein cholesterol ratio.

discrepancies likely reflect differences in study populations and methods. For example, the Tanzanian cohort was older, had a high prevalence of obesity and diabetes, and most participants were on diuretic therapy,[8] whereas our participants, similar to those in the Cameroonian and Ethiopian studies, were relatively younger and largely untreated. Variations in serum uric acid cutoff values across studies may also contribute to differences in reported prevalence.[26] Overall, our prevalence of 32.1% is higher than estimates from the general Ugandan population and within the mid-range reported among African hypertensive populations. It underscores the clustering of hyperuricemia with other cardiometabolic and renal risk factors and suggests that integrating uric acid screening into hypertension management could help identify patients at elevated risk.

Table 3. Factors associated with hyperuricemia among patients with hypertension.

	Bivariate		Multivariate	
Variable	cOR:(95% CI)	p-value	aOR:(95% CI)	p-value
Age (years)	1.02(1.00–1.04)	0.076	1.01(0.98–1.03)	0.639
Education				
None	1.00		1.00	
Primary	0.83(0.35–1.97)	0.674	0.65(0.24–1.74)	0.392
Secondary	0.85(0.35–2.02)	0.707	0.78(0.28–2.18)	0.639
Tertiary	0.53(0.21–1.37)	0.191	0.37(0.12–1.13)	0.080
Diabetes				
No	1.00			
Yes	1.07(0.54–2.10)	0.850		
HIV status				
Negative	1.00			
Positive	0.89(0.39–2.04)	0.791		
Heart disease				
No	1.00		1.00	
Yes	**1.79(1.02–3.14)**	**0.044**	**2.38(1.23–4.59)**	**0.010**
Thyroid disease				
No	1.00			
Yes				
Kidney disease				
No	1.00		1.00	
Yes	2.74(0.72–10.46)	0.141	1.96(0.40–9.57)	0.405
Alcohol consumption				
Never	1.00			
Former (>6 months)	1.09(0.62–1.89)	0.768		
Current (within 6 months)	1.00(0.47–2.16)	0.993		
Smoking				
Never smoked	1.00		1.00	
Ever smoked	**2.51(1.02–6.15)**	**0.044**	**4.20(1.47–12.02)**	**0.007**
Red meat intake				
Never	1.00			
<5 servings/day	1.02(0.48–2.20)	0.06		
≥5servings/day	1.64(0.31–8.59)	0.58		
Red meat consumption per week				
≤4 days	0.75(0.24–2.35)	0.618		
>4 days	1.00			
Physical activity (MET-min per week)				
<600	1.63(0.83–3.21)	0.154	1.86(0.86–4.05)	0.116
600–3000	1.18(0.67–2.11)	0.565	1.04(0.52–2.05)	0.915
>3000	1.00		1.00	
Sleep duration				
<5 h	0.813(0.36–1.86)	0.624		
5–7 h	1.13(0.58–2.22)	0.715		
>7 h	1.00			
Obstructive Sleep Apnea (OSA)				
Low risk of OSA	1.00		1.00	
High of OSA	**2.04(1.08–3.86)**	**0.028**	1.56(0.71–3.43)	0.264
Family history of hypertension				
No	1.00			
Yes	0.88(0.50–1.54)	0.649		
Family history of kidney disease				
No	1.00			
Yes				
Family history of CVD				
No	1.00			
Yes	1.04(0.57–1.90)	0.901		
Family history of diabetes mellitus				

(continued)

Table 3. Continued.

Variable	Bivariate		Multivariate	
	cOR:(95% CI)	p-value	aOR:(95% CI)	p-value
No	1.00		1.00	
Yes	1.54(0.92–2.58)	0.098	1.82(1.00–3.32)	0.052
Adherence to antihypertensive medication				
Lifestyle modification and diet	1.00			
Low adherence	1.61(0.56–4.65)	0.374		
Moderate adherence	1.63(0.54–4.87)	0.384		
High adherence	0.64(0.10–3.95)	0.631		
Duration with hypertension				
<5 years	1.00		1.00	
≥5 years	1.70(1.02–2.84)	0.041	3.67(1.00–13.43)	0.050
Duration on antihypertensive medication				
<5 years	1.00		1.00	
≥5 years	1.41(0.85–2.34)	0.183	0.31(0.08–1.15)	0.081
Antihypertensive medication type				
Non-diuretic	1.30(0.44–3.84)	0.639		
Diuretic	1.76(0.61–5.10)	0.296		
Lifestyle modification and diet	1.00			
Hypertension stage				
Stage one	1.00			
Stage two	1.20(0.61–2.35)	0.591		
Blood pressure (mmHg)				
Normal	1.00		1.00	
High	0.72(0.41–1.27)	0.258	0.87(0.45–1.67)	0.671
Body mass index (kg/m^2)				
<25	1.00		1.00	
25–29.9	1.36(0.65–2.84)	0.418	1.02(0.41–2.53)	0.962
≥30	1.59(0.87–2.92)	0.134	1.61(0.66–3.91)	0.290
Waist circumference				
Normal	1.00		1.00	
High	1.39(0.75–2.56)	0.290	0.72(0.29–1.80)	0.483
Waist-to-hip ratio				
Normal	1.00			
High	1.42(0.84–2.39)	0.192		
Total cholesterol (mg/dL)				
<200	1.00		1.00	
≥200	1.35(0.80–2.28)	0.262	1.17(0.60–2.28)	0.648
HDL-C				
High	1.00		1.00	
Low	**1.94(1.15–3.25)**	**0.012**	1.66(0.82–3.37)	0.162
Total cholesterol to HDL-C ratio				
<5	1.00		1.00	
≥5	**3.41(1.92–6.08)**	**<0.001**	**2.83(1.31–6.09)**	**0.008**

Bolded values indicate statistically significant results ($p < 0.05$).
cOR: crude odds ratio; aOR: adjusted odds ratio; CI: confidence interval; HDL-C: high-density lipoprotein cholesterol; TC/HDL-C: total cholesterol-to-high-density lipoprotein cholesterol ratio; MET: metabolic equivalent of task; CVD: cardiovascular disease; HIV: human immunodeficiency virus.

Additionally, in our hypertensive study population, a high TC/HDL-C ratio was strongly associated with hyperuricemia. Patients with a TC/HDL-C ≥5 had a hyperuricemia prevalence of 53.8% compared with 25.5% among those below this threshold ($p < 0.001$), and multivariate analysis still demonstrated a significantly increased risk of hyperuricemia (aOR = 2.8, 95% CI 1.31–6.09). This finding is consistent with previous observations in Chinese populations; for example, Yu et al. reported that patients with hypertension in the highest TC/HDL-C tertile had 1.8-fold higher odds of hyperuricemia compared with those in the lowest tertile.[30] In our study, total cholesterol alone was not significantly associated with hyperuricemia ($p = 0.26$), underscoring that the ratio—reflecting low HDL-C and/or elevated TC—is the more informative marker. Our ROC analysis showed modest discriminative performance (AUC = 0.68). A cutoff value of

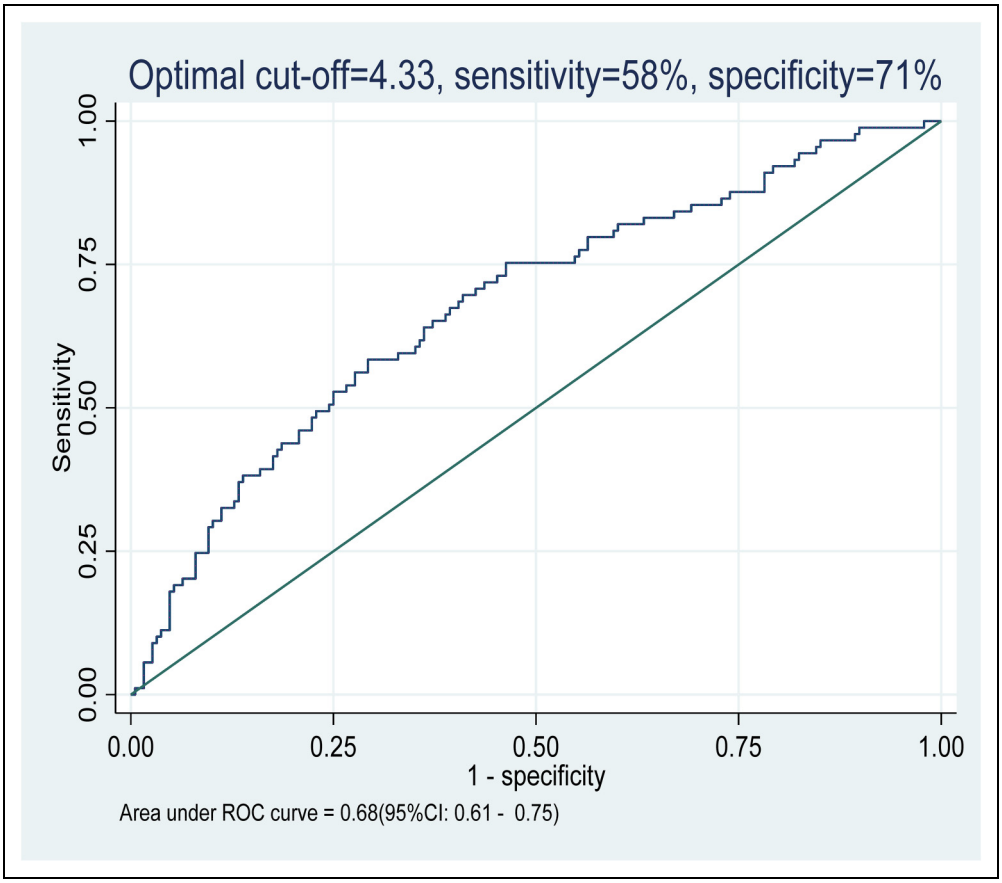


Figure 3. Receiver operating characteristic curve showing the predictive performance of the TC/HDL-C ratio for hyperuricemia. TC/HDL-C: total cholesterol-to-high-density lipoprotein cholesterol ratio.

4.33 yielded a sensitivity of 58% and specificity of 71%. Clinically, an elevated TC/HDL-C ratio, as a simple and inexpensive marker of atherogenic dyslipidemia, could be used to identify patients with hypertension who may benefit from serum uric acid testing and more aggressive management of dyslipidemia.

Our results are consistent with a growing body of evidence. In Chinese patients with hypertension, Lan et al. and Yu et al. similarly reported that higher TC/HDL-C levels were associated with significantly higher odds of hyperuricemia (aOR = 1.79 for highest vs. lowest tertiles).[30] Likewise, a recent Chinese study of older adults found that composite lipid indices, including Castelli's Risk Index I (TC/HDL-C) were significantly higher in patients with hyperuricemia (aOR per SD increase =1.50). Large-scale Chinese data also demonstrate a graded relationship: in Wuhu (n = 298,000), individuals in the highest triglyceride quartile had 3.77-fold higher odds of hyperuricemia compared with the lowest quartile, whereas those in the highest total cholesterol quartile had 1.52-fold higher odds.[31] In Korean national survey data, each 10 mg/dL increase in total cholesterol increased the odds of hyperuricemia (OR = 1.053), whereas each 10 mg/dL increase in HDL-C reduced the odds (OR = 0.804).[32] Even in sub-Saharan Africa, a study of patients with hypertension in Cameroon reported a hyperuricemia prevalence of 31.8%, with high low-density lipoprotein (LDL)-C (>100 mg/dL) and high triglycerides (>150 mg/dL) identified as independent predictors of hyperuricemia.[7] Overall, these consistent findings across diverse populations reinforce the strong association between atherogenic lipid profiles (high total cholesterol and triglycerides and low HDL-C) and elevated serum uric acid levels.

Some studies have reported weaker associations. For example, Li et al. found no independent association between serum uric acid and lipid components in overweight and obese Chinese adults.[33] This discrepancy likely reflects differences in study populations and analytical approaches, as Li et al. examined a predominantly overweight cohort (mean BMI >24 kg/m^2) and used stepwise regression methods. Notably, several studies emphasize triglyceride-related indices as even stronger predictors of hyperuricemia. In the Wuhu dataset, for instance, elevated triglycerides conferred a much greater risk than total cholesterol.[31] This suggests that although the TC/HDL-C is an important marker, hypertriglyceridemia may play a more dominant role in driving hyperuricemia in some populations. Differences in covariate adjustment and the specific lipid parameters assessed may also account for variations in reported findings.

Overall, our findings extend international evidence linking dyslipidemia to hyperuricemia[30–32] and suggest that routine lipid ratios could serve as inexpensive indicators of hyperuricemia risk. However, Ugandan research in this area remains limited. Therefore, our findings are hypothesis-generating and provide novel, context-specific evidence to guide future longitudinal studies with larger sample sizes and greater statistical power to further evaluate the association between lipoprotein ratios and hyperuricemia among patients with hypertension in Uganda. Future studies should validate the optimal TC/HDL-C threshold in diverse populations and assess whether targeting dyslipidemia can reduce incident hyperuricemia and related cardiovascular sequelae.

Limitations and recommendations

This study was cross-sectional in design and therefore could only provide a snapshot of the association between the TC/HDL-C ratio and hyperuricemia. As such, it cannot establish causal relationships or the temporal sequence of events. Additionally, some of the variables, including comorbidities and family history of chronic conditions, were self-reported, introducing the possibility of recall or reporting bias. We also acknowledge the potential influence of unmeasured confounding factors on the observed association. Despite adjustment for known confounders, residual confounding may still be present. Finally, the generalizability of the findings is limited to patients with hypertension and may not extend to other populations.

In light of these limitations, we recommend several directions for future research. Longitudinal studies are needed to establish temporal and causal relationships between a high TC/HDL-C ratio and the development of hyperuricemia, addressing the limitations of the cross-sectional design. Future studies should also incorporate objective clinical and biochemical measurements rather than relying on self-reported data for variables such as family history of chronic diseases, to reduce information bias. In addition, it is important to account for a wider range of potential confounders, including detailed lifestyle factors, comorbid conditions, and medication use, through more comprehensive data collection and robust multivariable analyses.

Conclusion

The results of this study demonstrate that hyperuricemia is prevalent among patients with hypertension at the UHI, and that a high TC/HDL-C ratio is potentially associated with hyperuricemia in this population.

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Consent for publication

All authors have consented to the publication of this work.

Authors' contributions

B.A, CNB, J.E, S.E, J.O, and S.N participated in the conceptualization of the study and data collection. C.N.B performed the data analysis and interpretation of results. B.A, C.N.B, J.E, S.E, J.O, S.N, M.J.M, A.B, J.K, F.K, J.T, and C.N contributed to drafting the initial manuscript, whereas F.S, D.N, E.M, R.N, S.P.R, and R.G provided critical revisions. All authors read and approved the final manuscript.

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Availability of data and materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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