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Prevalence and associated factors of hyperuricemia in chronic kidney disease: evidence from a single-center hospital-based study in Ethiopia, 2024

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Hyperuricemia has been implicated in accelerating the progression of chronic kidney disease (CKD). Identifying associated factors may help guide future interventions aimed at delaying CKD progression. To determine the prevalence of hyperuricemia and its associated factors among CKD patients. An institution-based cross-sectional study was conducted among adult CKD patients at the University of Gondar Comprehensive Specialized Hospital from September 2023 to January 2024. A total of 218 patients were enrolled using a consecutive sampling technique. Pretested, structured, interviewer-administered questionnaires were used to collect sociodemographic and clinical data. Data were entered using Epi Data Manager version 4.6 and analyzed with SPSS version 27.0. Multivariate logistic regression analysis was performed to identify factors associated with hyperuricemia, and variables with a p-value of less than 0.05 were considered statistically significant. The mean serum uric acid (SUA) level over the past 3 months was 7.76 mg/dL (SD ± 2.93). Among non-dialysis CKD patients ($n = 189$), 65.6% (95% CI: 58.7–72.0%) had hyperuricemia. Among all CKD patients ($n = 218$), the prevalence was 66.1%, and among dialysis patients ($n = 29$), it was 69.0%. Factors significantly associated with hyperuricemia among non-dialysis CKD patients were: male sex (AOR = 2.01; 95% CI: 1.01–4.28), serum triglyceride > 150 mg/dL (AOR = 4.05; 95% CI: 1.85–8.80), eGFR stage 4 (AOR = 6.31; 95% CI: 2.43–16.40), eGFR stage 5 (AOR = 4.62; 95% CI: 1.59–13.40), BMI 25–29.9 kg/m^2 (AOR = 4.12; 95% CI: 1.77–9.60), BMI ≥ 30 kg/m^2 (AOR = 6.24; 95% CI: 2.09–18.60), and 24-hour total urine protein > 3.5 g (AOR = 2.68; 95% CI: 1.10–6.52). This study found a high prevalence of hyperuricemia among non-dialysis CKD patients. Significant associated factors included male sex, high triglyceride levels, advanced CKD stages, elevated BMI, and heavy proteinuria. It highlights the importance of identifying individuals at higher risk of hyperuricemia for potential early interventions that may help delay CKD progression, and routine hyperuricemia screening may be considered in CKD patients with identified risk factors.

Keywords Chronic kidney disease, Ethiopia, Gondar, Hyperuricemia, Non-dialysis

Abbreviations

AOR	Adjusted odds ratio
ADPKD	Autosomal dominant polycystic kidney disease
CAD	Coronary artery disease
CGN	Chronic glomerulonephritis
CI	Confidence interval
CKD	Chronic kidney disease
CVD	Cardiovascular disease
DM	Diabetes mellitus

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FBS	Fasting blood sugar
NAFLD	Nonalcoholic fatty liver disease
HTN	Hypertension
HUA	Hyperuricemia
HbA1c	Hemoglobin A1c
IQR	Interquartile range
OR	Odds ratio
Spot Urine PCR	spot urine protein to creatinine ratio
SSA	Sub-Saharan Africa
SUA	Serum uric acid
WHO	World health organization

Chronic Kidney Disease (CKD) is a long-term disorder characterized by either renal impairment or reduction in kidney function for a minimum of three months, regardless of etiology[1]. The disorder is usually diagnosed by signs such as albuminuria, abnormal urinary sediments, imaging, or histopathological results, and by a history of kidney transplantation; it is classified by estimated glomerular filtration rate (eGFR), the degree of albuminuria, and etiology[1].

Globally, CKD is an emerging public health problem. In 2010, its age-standardized prevalence was 10.4% in men and 11.8% in women[2], with recent estimates indicating that 8–16% of the global population, around 500 million people, are affected, 78% of whom reside in low- and middle-income countries[3].

In Ethiopia, CKD is present in approximately 21.7% of adults with chronic diseases, with more than 70% presenting at advanced stages due to a lack of screening and diagnostic facilities[4,5]. Access to healthcare is limited, with just 13 nephrologists for more than 120 million individuals and dialysis facilities restricted to large urban centers[6,7]. Environmental factors like repeated infections, nephrotoxic traditional medications, and excessive salt consumption can exacerbate CKD, whereas genetic predisposition (e.g., APOL1 variants) is largely uninvestigated[8,9]. These realities underscore the necessity for setting-specific research to guide national CKD policy and control in low-income environments[10]. Early hyperuricemia screening could be an affordable approach for detecting high-risk CKD patients, particularly where nephrology services and diagnostics are restricted.

Several factors influence the development and progression of CKD, including genetics, sociodemographic variables, and comorbidities such as hypertension, diabetes, and hyperuricemia[11]. Among these, hyperuricemia defined as serum urate ≥ 7.0 mg/dl for male and ≥ 6.0 for female, is particularly noteworthy due to its potential role as both a marker and cause of CKD[12].

Recent advances in nephrology have transformed the understanding of hyperuricemia's role in CKD, from a mere byproduct of reduced renal clearance to a potential contributor to disease progression and modifiable therapeutic target. Updated guidelines and expert opinion, e.g., KDIGO and EULAR, now underscore the value of monitoring and perhaps treating raised serum uric acid levels, particularly in individuals at higher risk of progression or cardiovascular events[1,13]. Novel agents such as febuxostat and SGLT2 inhibitors have shown renoprotective effects through urate lowering, further implicating uric acid's role in the management of CKD[14,15]. Large-scale cohort studies, e.g., the J-CKD-DB from Japan, and systematic reviews such as Zhang et al.'s (2022) provide updated prevalence estimates and confirm the complex, bidirectional relationship between CKD stage and hyperuricemia[16,17].

The prevalence of hyperuricemia in CKD is geographically heterogeneous, depending on disease severity, comorbidities, and diagnostic thresholds. A recent meta-analysis and systematic review by reported a pooled worldwide prevalence of 52.6%, with considerable regional heterogeneity[17]. In the United States, the estimate was 76.7%, while Italian data reported 72.0%[18,19]. A Japanese large cohort study (J-CKD-DB) revealed that 64.7% of stage 5 CKD patients were affected by hyperuricemia[16]. Corresponding prevalence estimates in low and middle-income countries include 67.0% in Cameroon[20], 57.3% in Iranian CKD patients with NAFLD[21], and 47.5% in Nigerian pre-dialysis patients[22]. By contrast, a population-based study from Chad reported a much lower prevalence of 15.2%, plausibly reflecting CKD severity and metabolic burden differences[23].

The primary cause of hyperuricemia in CKD is impaired renal clearance, but other causes include obesity, alcohol use, and lack of physical activity[1,13,16,17]. Shared risk factors include advanced age, low eGFR, proteinuria, anemia, diuretic use, and comorbid conditions such as diabetes, cardiovascular disease, and dyslipidemia[1,14–19]. In light of the above, this study aims to identify the prevalence and associated factors of hyperuricemia in Ethiopian CKD patients since local evidence is scarce. This may inform future intervention strategies and contribute to understanding factors associated with CKD progression.

Methods and materials

The research was carried out in the University of Gondar Comprehensive Specialized Hospital (UoG CSH), a teaching and referral hospital located in the town of Gondar, Northwest Ethiopia. Gondar belongs to the Amhara Region, about 748 km northwest of the capital city of the country, Addis Ababa. The UoG Hospital is a tertiary teaching and referral hospital and serves as the central referral site for over 12 district hospitals with a catchment population of approximately 7 million individuals.

The nephrology service, under the department of internal medicine, has within its care a renal referral clinic, hemodialysis unit, renal function test laboratory (inclusive of blood urea nitrogen and serum creatinine), serum electrolyte determination, serum uric acid determination, dipstick and microscopy urinalysis, and abdominopelvic ultrasound imaging. The renal referral clinic and the dialysis center operated by a single nephrologist and serve an average of 30 patients and 20 patients weekly, respectively.

Study design and participants

This study was an institution-based cross-sectional study. The source populations were all adult patients with CKD attending the University of Gondar Comprehensive Specialized Hospital, and all CKD patients who were admitted to the hospital during the study period were used as study populations. Patients aged ≥ 18 years with CKD stage 3a and above were included in the study. Participants with gout or history of gout, on medications affecting uric acid levels (diuretics, allopurinol, etc.), active malignancy or recent chemotherapy, severe liver disease, and those who were critically ill or uncooperative were excluded from the study. Patients on dialysis ($n = 29$, 13.3%) were described separately but excluded from inferential analyses to ensure homogeneity.

Sample size and sampling design

The sample size was initially estimated using a single population proportion formula with assumed hyperuricemia in CKD prevalence of 15.2% from the Chad study[23]. At a 95% confidence level and 5% error margin, the sample size was approximated at 198. Adding a 10% non-response rate, the final sample size becomes 218. The participants were enrolled by a consecutive sampling process during the study period. Of the 218 participants, 169 (77.5%) were recruited from the renal clinic, 29 (13.3%) from the hemodialysis unit, and 20 (9.2%) from the general medical ward.

We also recalculated the sample size using the observed prevalence of hyperuricemia in our study (66.1%) and confirmed that a sample of 218 was adequate for prevalence estimation. Since the primary aim of this study was to identify factors associated with hyperuricemia, sample size for analytical objectives was also considered. Using standard formulas for comparing two proportions at a 5% significance level and 80% power, the minimum sample sizes required for key predictors were: sex (male vs. female, OR = 1.8, p0 = 0.60): 94 participants per group, total 188, BMI ≥ 25 vs. < 25 (OR = 2.0, p0 = 0.55): 66 participants per group, total 132, and hypertension (yes vs. no, OR = 2.2, p0 = 0.50): 57 participants per group, total 114 participants.

With 189 non-dialysis participants and 124 hyperuricemia cases included in the analytical cohort, the study had sufficient power to detect these moderate associations. Applying the "10 events per variable" rule for logistic regression, up to 12 predictors could be reliably included in multivariable models.

Study variables

The dependent variable was the presence of hyperuricemia among CKD patients. The independent variable included sociodemographic (area of residence, sex, age, income, marital status, occupational level, educational level), clinical parameters (weight, height, BMI, compliance with drugs, exercise, alcohol use, smoking, diet), comorbidities (diabetes mellitus, hypertension, stroke, heart failure, bronchial asthma, HIV, BPH), causes of CKD (diabetic nephropathy, hypertensive nephropathy, chronic glomerulonephritis, ADPKD, obstructive nephropathy, others (e.g., HIVAN, renovascular disease)), stage of CKD, and laboratory tests (24-hour urine protein, hemoglobin, BUN, creatinine, ionized calcium, lipid panel, serum protein, and albumin).

Data collection procedure

Both primary and secondary data were obtained using a systematic checklist to capture sociodemographic, clinical, laboratory, and comorbidity-related characteristics. Any missing laboratory results, including serum uric acid levels, were obtained by the principal researcher as necessary. The checklist was formulated based on the study objectives and pretested for feasibility assessment. Items found infeasible were removed. The checklist was written in English, suitable for trained data collectors. Three trained Bachelor of Science nurses and the principal researcher collected data. Initial screening was done for 5% of patient records. Data collectors were trained for a day before actual data collection. Daily data verification and feedback were provided by the principal researcher.

Two knowledge domains (i.e., awareness of optimal serum uric acid levels and health risks of hyperuricemia) were assessed using structured items adapted from WHO CKD patient education materials and modified for local context after expert review. A pilot pretest ($n = 20$) was conducted and internal consistency was assessed using Cronbach's alpha ($\alpha = 0.78$). Responses were binary (Yes/No); knowledge was coded as adequate if both items were answered correctly.

Serum uric acid and creatinine were measured enzymatically on the Beckman Coulter DxC 700 AU analyzer. The laboratory participates in external quality assurance (EQA) schemes and conducts daily internal quality control using manufacturer-provided control materials. Body weight was measured to the nearest 0.5 kg on a calibrated adult scale with the participants in light clothing and without shoes. Height was measured to the nearest centimeter using a standard stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared.

Data processing and analysis

The data were entered into EPI data version 4.6 and then transferred to SPSS 27.0 statistical packages for analysis. Data was cleaned before performing the descriptive analysis. The baseline characteristics are presented as numbers and percentages. The findings were summarized in tables. Continuous variables like BMI and eGFR were categorized based on WHO and KDIGO classification schemes, respectively, to facilitate clinical interpretation.

Variables with p values < 0.25 in the bivariate analysis were transferred to multivariate analysis and entered hierarchically to fit the logistic regression model. Statistically significant associations were determined based on the adjusted odds ratio (AOR) with its 95% CI and the P-value < 0.05. Hosner-Lemeshow test ($p = 0.71$) was used to assess model fitness, discrimination was assessed using Area Under the ROC Curve (AUC = 0.83), and multicollinearity test (all variables IVF value was < 2.0) was conducted to check the absence of correlation between independent variables. Normality of continuous variables was checked using the Shapiro–Wilk test. Group comparisons between hyperuricemic and normouricemic participants were conducted using Chi-square

or Fisher's exact test for categorical variables, and t-tests or Mann–Whitney U tests for continuous variables. All variables included in the analysis had complete data with no missing values; thus, no imputation was performed.

Operational definitions

CKD: It is defined based on the documented diagnosis of CKD in a patient's file labeled by the physician. For this study, participants with eGFR of < 60 ml/min/1.73m2 were included.

Staging of CKD: defined as G1 – GFR > 90 mL/min per 1.73 m^2, G2 – GFR 60 to 89 mL/min per 1.73 m^2, G3a – GFR 45 to 59 mL/min per 1.73 m^2, G3b – GFR 30 to 44 mL/min per 1.73 m^2, G4 – GFR 15 to 29 mL/min per 1.73m^2, and G5 – GFR < 15 mL/min per 1.73 m^2 or treatment by dialysis[1].

Hyperuricemia: if serum uric acid is ≥ 7.0 mg/dl for male and ≥ 6.0 for female, determined using an automated enzyme (uricase) analyzer[12].

Anemia: if hemoglobin < 12 g/dL for women, < 13 g/dL for men[24].

Ionized calcium: normal if between 4.65 and 5.25 mg/dL, hypocalcemia if < 4.65 mg/dl, and hypercalcemia if > 5.25 mg/dl[25].

Proteinuria: determined using 24 h urine total protein. Normal if < 0.5 g/day, nephrotic range if 0.5 g/day – 3.5 g/day, and massive proteinuria if > 3.5 g/day[26].

Adherence to medication: considered as adherent if the patient took all his/ her medication in the last seven days[27].

Alcohol consumption: considered as positive if the patient reported consumption of any amount of alcohol twelve months before the survey[28].

Adherence to diet: Adherent if the patient followed dietary recommendations on ≥ 4 days in the past week[29].

Adherence to exercise: Active if engaged in moderate activity ≥ 3 times/week as per WHO CKD self-care guidelines[30].

Lipid panel: hypertriglyceridemia is defined by serum triglyceride level 150 mg/dl and above, high cholesterol level is defined by TC 220 mg/dl and above, high LDL is defined by 160 mg/dl and above, low HDL is defined by HDL level below 35 mg/dl[31].

Results

Sociodemographic characteristics of study participants

As depicted in Table 1, a total of 218 CKD patients were included in the study. The mean age of participants was 53.11 years (SD ± 14.76). The majority were male (122, 56.0%), married (132, 60.6%), unemployed (48, 22.0%),

Variable	Category	Frequency (%)
Age (years)	Mean ± SD	53.11 ± 14.76
	18–34	59 (27.1%)
	35–49	54 (24.8%)
	50–64	57 (26.1%)
	≥ 65	48 (22.0%)
Sex	Male	122 (56.0%)
	Female	96 (44.0%)
Marital status	Single	25 (11.5%)
	Married	132 (60.6%)
	Divorced	22 (10.1%)
	Widowed	39 (17.9%)
Educational level	No formal education	66 (30.3%)
	Primary education	56 (25.7%)
	Secondary education	49 (22.5%)
	College and above	47 (21.6%)
Area of residency	Urban	161 (73.9%)
	Rural	57 (26.1%)
Occupation	Unemployed	48 (22.0%)
	Government/private employee	39 (17.9%)
	Self-employed	28 (12.8%)
	Housewife	44 (20.2%)
	Retired	36 (16.5%)
	Farmer	23 (10.6%)
Monthly income (ETB)	< 1500	30 (13.8%)
	1500–5000	133 (61.0%)
	> 5000	55 (25.2%)

Table 1. Sociodemographic characteristics of study participants at the university of Gondar comprehensive specialized hospital, Ethiopia, 2024 ($n = 218$). *ETB* Ethiopian Birr.

and resided in urban areas (161, 73.9%). Most had no formal education (66, 30.3%). The majority of participants reported a monthly income ranging from 1500 to 5,000 Ethiopian Birr (ETB).

Hyperuricemia and CKD self-care activity characteristics

As presented in Tables 2 and 176 participants (80.7%) were unaware of the optimal SUA level, and 180 (82.6%) did not know the associated health risks of elevated SUA. More than three-quarters (156, 71.6%) were non-adherent to their prescribed medications, and 128 (58.7%) did not follow dietary recommendations. A majority (129, 59.2%) were physically inactive, engaging in physical activity fewer than three times per week. Only 16 participants (7.3%) were current smokers, while 18 (8.3%) were ex-smokers. Regarding alcohol use, 120 (55%) were non-drinkers, and 66 (30.3%) had ceased alcohol consumption.

Clinical and disease-related factors

The mean eGFR was 30.53 ml/min/1.73m^2 (SD ± 18.43). Among the participants, 80 (36.7%) were in stage 4 CKD, 50 (22.9%) in stage 3a, 47 (21.6%) in stage 5, and 41 (18.8%) in stage 3b. The leading causes of CKD were

Variable	Category	Frequency (%)
Knowledge of optimal serum uric acid (UA) level	Yes	42 (19.3%)
	No	176 (80.7%)
Knowledge of hazards of high UA	Yes	38 (17.4%)
	No	180 (82.6%)
Drug adherence	Less than 7 days/week	156 (71.6%)
	7 days/week	62 (28.4%)
Diet adherence	Adherent	128(58.7%)
	Non-adherent	90(41.3%)
Physical activity	Active	128 (58.7%)
	Inactive	90 (41.3%)
Smoking status	Current smoker	16 (7.3%)
	Ex-smoker (> 1 year)	18 (8.3%)
	Non-smoker	184 (84.4%)
Alcohol use	Regular user	5 (2.3%)
	Occasional user	27 (12.4%)
	Stopped	66 (30.3%)
	Non-user	120 (55.0%)
Body mass index (BMI)	Mean ± SD	22.67 ± 2.95
BMI category (kg/m^2)	< 18.5	9 (4.1%)
	18.5–24.9	105 (48.2%)
	25–29.9	71 (32.6%)
	≥ 30	33 (15.1%)
Comorbidity	Yes	158 (72.5%)
	No	60 (27.5%)
Type of comorbidity	Diabetes mellitus (DM)	8 (5.1%)
	Hypertension (HTN)	61 (38.6%)
	Stroke	4 (2.5%)
	Heart failure	1 (0.6%)
	Two or more	77 (48.7%)
	Others*	7 (4.4%)
Cause of CKD	DM	51 (23.4%)
	HTN	99 (45.4%)
	Chronic glomerulonephritis (CGN)	40 (18.3%)
	ADPKD	11 (5.0%)
	Obstructive causes	2 (0.9%)
	Others**	15 (6.9%)
Dialysis started	Yes	29 (13.3%)
	No	189 (86.7%)

Table 2. Clinical and disease-related factors of study participants at the university of Gondar comprehensive specialized hospital, Ethiopia, 2024 (n = 218). Others*: HIV, bronchial asthma, hypothyroidism, Others**: HIV-associated (6), solitary kidney (3), renovascular disease (3), urate nephropathy (3), ADPKD – Autosomal Dominant Polycystic Kidney Disease; CGN – Chronic Glomerulonephritis; DM – Diabetes Mellitus; HTN – Hypertension; UA – Uric Acid.

hypertension (99, 45.4%), diabetes mellitus (51, 23.4%), and chronic glomerulonephritis (40, 18.3%). Other causes included HIV-associated nephropathy, solitary kidney, renovascular disease, and urate nephropathy (15, 6.9%), autosomal dominant polycystic kidney disease (ADPKD) (11, 5%), and obstructive nephropathy (2, 0.9%).

Comorbidities were present in 158 participants (72.5%). Among them, 77 (48.7%) had two or more comorbidities, 61 (38.6%) had hypertension alone, 8 (5.1%) had diabetes, 6 (4.4%) had other conditions (e.g., HIV, bronchial asthma, hypothyroidism), 4 (2.5%) had stroke, and 1 (0.6%) had heart failure. Only 29 participants (13.3%) had started dialysis.

The mean body mass index (BMI) was 22.67 kg/m^2 (SD ± 2.95). Nearly half (105, 48.2%) had a normal BMI (18.5–24.9 kg/m^2), 71 (32.6%) were overweight (25.0–29.9 kg/m^2), 33 (15.1%) were obese (≥ 30.0 kg/m^2), and 9 (4.1%) were underweight (BMI < 18.5 kg/m^2).

Laboratory values of study participants

As depicted in Table 3, the mean hemoglobin level was 11.24 mg/dl (SD ± 3.15), with 125 participants (57.3%) having anemia. The mean blood urea nitrogen (BUN) was 76.45 mg/dl (SD ± 71.03), and 196 (89.9%) had BUN levels above 20 mg/dl.

Regarding ionized calcium, 91 participants (41.7%) had low levels (< 4.65 mg/dl), 84 (38.5%) were within the normal range (4.65–5.25 mg/dl), and 43 (19.7%) had elevated levels (> 5.25 mg/dl). A total of 118 (54.1%) had serum albumin levels below 4 mg/dl, while 171 (78.4%) had normal serum total protein (6–8.3 mg/dl).

The mean 24-hour urine protein was 889.45 mg (SD ± 1155.31). Of the participants, 89 (40.8%) had protein levels < 500 mg, 66 (30.3%) had levels between 500 and 3500 mg, and 63 (28.9%) had proteinuria > 3500 mg. Total cholesterol levels were < 220 mg/dl in 147 (67.4%) participants. Triglyceride levels were > 150 mg/dl in 112 (58.4%), and HDL levels ≥ 35 mg/dl in 127 (58.3%). LDL levels were < 160 mg/dl in 147 (67.4%) of participants.

Descriptive summary of Dialysis patients

The mean age was 55.2 years (SD ± 13.8), 65.5% were male, and 82.8% had advanced CKD (stage 5D). The prevalence of hyperuricemia among this subgroup was 69.0%. Due to small numbers and distinct treatment-related mechanisms of uric acid clearance along with methodological considerations, dialysis patients were excluded from regression analyses ($n = 189$).

Prevalence of hyperuricemia in CKD patients

Among non-dialysis CKD patients ($n = 189$), 124 (65.6%, 95% CI: 58.7–72.0%) had hyperuricemia. Among all CKD patients ($n = 218$), the prevalence was 66.1%, and among dialysis patients ($n = 29$), it was 69.0%.

Factors associated with hyperuricemia in CKD patients

The relationship between SUA levels in non-dialysis CKD patients ($n = 189$) and various independent variables was examined using bivariate and multivariate logistic regression analyses. In the bivariate analysis, sex, smoking, comorbidity, hemoglobin, triglyceride level, total cholesterol, BMI, eGFR, and 24-hour urine protein were significantly associated with hyperuricemia (as depicted in Table 4).

In the multivariate logistic regression analysis, the following factors were independently associated with hyperuricemia: male, triglycerides > 150 mg/dl, eGFR Stage 4, eGFR Stage 5, BMI 25–29.9 kg/m^2, BMI ≥ 30 kg/m^2, and 24-hour urine protein > 3.5 gm.

Discussion

In this study, the prevalence of hyperuricemia among non-dialysis CKD patients was 65.6%, which falls at the upper range of values reported globally but is slightly lower than figures observed in some settings. For instance, a meta-analysis by Zhang et al. (2022) reported prevalence rates as high as 76.7% in the United States and 72% in Italy among CKD patients, particularly in more metabolically compromised populations or where lifestyle-related risk factors (e.g., obesity, diabetes) are more prevalent[17]. Similarly, in a large Japanese cohort (J-CKD-DB), the prevalence reached 64.7% among stage 5 CKD patients, increasing with disease severity[16]. The slightly lower prevalence in our Ethiopian cohort may be partly explained by differences in dietary purine intake, genetic background, or underdiagnosis due to limited healthcare access. Furthermore, although a substantial proportion of our population was at advanced CKD stages, the lower prevalence of obesity, hyperlipidemia, and western dietary patterns may have moderated uric acid levels.

On the other hand, our research also revealed a greater prevalence of hyperuricemia compared to other research. For instance, a large CKD cohort from china documented an overall prevalence of 52%, rising from 14.9% in early CKD to 64.7% in stage 4–5[32]. In Iran, among patients with both CKD and NAFLD, prevalence was 57.3%[21], while a Nigerian predialysis study found only 47.5% prevalence[22]. A Chinese urban survey of general participants, mostly without CKD, reported just 11.5% overall prevalence, and only 15.2% among those with reduced eGFR[33]. The lower rates likely reflect differences in CKD severity, with milder stages in some cohorts. Lifestyle and dietary habits, including purine intake and alcohol consumption, which differ across geographic regions, might also play a role. Lastly, disparities in laboratory methods and assay calibration could affect measurement accuracy and prevalence estimates across settings.

When considering all CKD patients, the prevalence was slightly higher (66.1%), and dialysis patients had an even higher prevalence (69.0%), although inferential analyses were not performed for this subgroup. The higher prevalence in dialysis patients likely reflects more advanced CKD with decreased uric acid excretion and altered purine metabolism, as reported in similar cohorts. These findings highlight the high burden of hyperuricemia among CKD patients and underscore the importance of monitoring uric acid levels, particularly in non-dialysis populations where interventions may be most feasible.

Variable	Category	Frequency (%)/mean ± SD
Blood Urea Nitrogen (BUN)	Mean ± SD	76.44 ± 71.02
BUN (mg/dL)	< 20	22 (10.1%)
	≥ 20	196 (89.9%)
Estimated Glomerular Filtration Rate (eGFR)	Mean ± SD	30.53 ± 18.42
eGFR (mL/min/1.73 m^2)	≥ 45	50 (22.9%)
	30–44	41 (18.8%)
	15–29	80 (36.7%)
	< 15	47 (21.6%)
Uric acid	Mean ± SD (mg/dL)	7.76 ± 2.93
Uric acid level (mg/dL)	< 7.0 for male, < 6.0 for female	74 (33.9%)
	≥ 7.0 for male, ≥ 6.0 for female	144 (66.1%)
24-hour urine protein (24 h UP)	Mean ± SD (mg/24hr)	889.45 ± 1155.30
24 h urine protein (g/day)	< 0.5	89 (40.8%)
	0.5–3.5	66 (30.3%)
	> 3.5	63 (28.9%)
Hemoglobin	Mean ± SD (g/dL)	11.23 ± 3.15
Hemoglobin level (g/dL)	< 12 for female, < 13 for male	125 (57.3%)
	≥ 12 for female, ≥ 13 for male	93 (42.7%)
Ionized Ca (mg/dL)	< 4.65	91 (41.7%)
	4.65–5.25	84 (38.5%)
	> 5.25	43 (19.7%)
HDL (mg/dL)	< 35	91 (41.7%)
	≥ 35	127 (58.3%)
Triglycerides (TGA)	< 150 mg/dL	106 (41.6%)
	≥ 150 mg/dL	112 (58.4%)
Low-density lipoprotein (LDL)	< 160 mg/dL	147 (67.4%)
	≥ 160 mg/dL	71 (32.6%)
Total cholesterol	< 220 mg/dL	147 (67.4%)
	≥ 220 mg/dL	71 (32.6%)
Serum total protein (TP)	< 6 g/dL	39 (17.9%)
	6–8.3 g/dL	171 (78.4%)
	> 8.3 g/dL	8 (3.7%)
Serum albumin (ALB)	< 4.0 g/dL	118 (54.1%)
	≥ 4.0 g/dL	100 (45.9%)

Table 3. Laboratory results of study participants at the university of Gondar comprehensive specialized hospital, Ethiopia, 2024 ($n = 218$). 24 hrUP – 24-hour Urine Protein, ALB – albumin, BMI – Body Mass Index, BUN – Blood Urea Nitrogen, Ca – Calcium, eGFR – estimated Glomerular Filtration Rate, HDL – High Density Lipoprotein, LDL – Low Density Lipoprotein, SD – Standard Deviation, TGA – triglyceride, TP – Total Protein.

As depicted in Table 5, male sex, eGFR, serum triglyceride levels, BMI, and urinary protein excretion were significant factors associated with hyperuricemia in non-dialysis CKD patients.

In this study, male participants were two times more likely to have hyperuricemia in CKD compared to female participants (AOR = 2.01, 95% CI: 1.03–4.28, $p = 0.04$). This is consistent with studies in Italy and Cameroon[34,35]. The higher prevalence of hyperuricemia among males may be partly attributed to the uricosuric effect of estrogen, which lowers uric acid levels in premenopausal women; this sex difference tends to narrow with aging and hormonal decline[36].

Participants with eGFR of 15–29 mL/min/1.73m^2 were 6.3 times more likely (AOR = 6.31, 95% CI: 2.4–16.4, $p < 0.001$), and those with eGFR < 15 mL/min/1.73m^2 were 4.6 times more likely (AOR = 4.62, 95% CI: 1.59–13.40, $p = 0.005$) to develop hyperuricemia compared to those with eGFR > 30 mL/min/1.73m^2. These findings are consistent with research in Cameroon, China, and Iran[21,33,35]. The positive relationship between declining eGFR and hyperuricemia is due to impaired renal excretion of uric acid, as the kidneys are the primary route of uric acid elimination. As kidney function worsens, uric acid filtration decreases while tubular reabsorption increases along with upregulation of urate transporters (URAT1 and GLUT9) and increased uric acid production exacerbate hyperuricemia[37,38].

Participants with a 24-hour urine protein of ≥ 3.5 g were 2.6 times more likely to have (AOR = 2.68, 95% CI: 1.10–6.52, $p = 0.03$) hyperuricemia than participants with proteinuria of < 0.5 g. The observation agrees with reports from studies conducted in Cameroon, Italy, and the United State[18,19,35]. The possible explanation

Variable	Category	Normal UA (*n*)	Hyperuricemia (*n*)	COR (95% CI)	*p*-value
Sex	Female	38	51	1.00	–
	Male	27	73	2.05 (1.10–3.82)	0.023
BMI (kg/m^2)	18.5–24.9	44	50	1.00	–
	< 18.5	5	3	0.50 (0.12–2.20)	0.360
	25–29.9	12	53	3.90 (1.85–8.20)	< 0.001
	≥ 30	4	18	3.30 (1.25–8.65)	0.015
eGFR (mL/min/1.73 m^2)	≥ 45	27	18	1.00	–
	30–44	17	22	1.95 (0.80–4.65)	0.130
	15–29	15	58	5.20 (2.20–12.3)	< 0.001
	< 15 (non-dialysis)	6	26	6.00 (2.35–15.4)	< 0.001
Proteinuria (g/day)	< 0.5	33	47	1.00	–
	0.5–3.5	21	41	1.25 (0.65–2.45)	0.510
	≥ 3.5	11	36	1.95 (0.95–4.05)	0.070
Triglyceride (mg/dL)	< 150	43	57	1.00	–
	≥ 150	22	67	2.60 (1.40–4.90)	0.002
Total cholesterol (mg/dL)	< 220	49	87	1.00	–
	≥ 220	16	37	1.50 (0.75–2.90)	0.210
Smoking	Non-smoker	59	109	1.00	–
	Ex-smoker	4	9	1.40 (0.45–4.25)	0.510
	Current smoker	2	6	2.40 (0.60–9.50)	0.190
Comorbidity	No	21	34	1.00	–
	Yes	44	90	0.70 (0.35–1.35)	0.240

Table 4. Bivariate logistic regression analysis of factors associated with hyperuricemia among non-dialysis CKD patients, Gondar, Ethiopia, 2024 ($n = 189$). *BMI* body mass index, *COR* crude odds ratio, *CI* confidence interval, *eGFR* estimated glomerular filtration rate, *mg/dL* milligrams per deciliter, *TG* triglyceride.

Variable	Category	Normal UA (*n*)	Hyperuricemia (*n*)	AOR (95% CI)	*p*-value
Sex	Female	38	51	1.00	–
	Male	27	73	2.05 (1.02–4.12)	0.041*
BMI (kg/m^2)	18.5–24.9	44	50	1.00	–
	< 18.5	5	3	0.65 (0.12–3.55)	0.620
	25.0–29.9	12	53	4.05 (1.70–9.65)	0.001*
	≥ 30	4	18	6.10 (2.05–18.1)	0.001*
eGFR (mL/min/1.73 m^2)	≥ 45	27	18	1.00	–
	30–44	17	22	1.40 (0.50–3.90)	0.540
	15–29	15	58	6.10 (2.30–16.2)	< 0.001*
	< 15 (non-dialysis only)	6	26	4.50 (1.55–13.1)	0.006*
Proteinuria (g/day)	< 0.5	33	47	1.00	–
	0.5–3.5	21	41	–	–
	≥ 3.5	11	36	2.55 (1.05–6.35)	0.038*
Triglyceride (mg/dL)	< 150	43	57	1.00	–
	≥ 150	22	67	3.95 (1.75–8.65)	< 0.001*
Total cholesterol (mg/dL)	< 220	49	87	1.00	–
	≥ 220	16	37	1.75 (0.80–3.80)	0.140
Smoking	Non-smoker	59	109	1.00	–
	Ex-smoker	4	9	1.40 (0.45–4.25)	0.510
	Current smoker	2	6	1.80 (0.50–6.50)	0.370
Comorbidity	No	21	34	1.00	–
	Yes	44	90	0.75 (0.35–1.60)	0.260

Table 5. Multivariate logistic regression analysis of factors associated with hyperuricemia among non-dialysis CKD patients, Gondar, Ethiopia, 2024 ($n = 189$). *Statistically significant with $p < 0.05$; All VIF value < 2.0, AUC = 0.83, and H-L $p = 0.71$. *AOR* adjusted odds ratio, *BMI* body mass index, COR: *CI* confidence interval, *eGFR* estimated glomerular filtration rate, *g/dL* gram per deciliter, *mg/dL* milligrams per deciliter, *TG* triglyceride.

is that heavy proteinuria, as seen in nephrotic-range proteinuria, is associated with reduced renal function, proximal tubular damage, and an alteration in the expression of urate transporters such as URAT1 and GLUT9, leading to increased reabsorption of uric acid which impairs uric acid handling. Furthermore, proteinuria is often accompanied by systemic inflammation and oxidative stress, which may stimulate uric acid production and retention[39].

Participants with serum triglyceride levels above 150 mg/dL were four time more likely to have hyperuricemia (AOR = 4.05, 95% CI: 1.86–8.80, $p < 0.001$) compared to participants with lower levels of triglycerides (i.e., it has wide 95% CI indicating potential imprecision in the effect estimate). This finding is consistent with other studies conducted in different nations such as China, Iran, Italy, and Japan[16,18,21,33]. The observed correlation can be explained by the common effect of abnormalities in lipid metabolism and uric acid metabolism The possible mechanism triglyceride causes hyperuricemia may be due to insulin resistance, hepatic overproduction of uric acid, and reduced renal excretion[40].

Participants who had BMI 25–29.9 kg/m^2 were 4.1 times (AOR = 4.12, 95% CI: 1.77–9.60, $p = 0.001$), and those with BMI ≥ 30 kg/m^2 were 6.2 times (AOR = 6.24, 95% CI: 2.09–18.60, $p = 0.001$) more likely to have hyperuricemia compared to participants with normal BMI (i.e., it has wide 95% CI indicating potential imprecision in the effect estimate). These findings are in line with earlier reports in Cameroon, Iran, and Italy[18,21,36]. Obesity and elevated BMI contribute to hyperuricemia through chronic low-grade inflammation, oxidative stress, and impaired urate transporter expression in renal tubular cells[41].

The observed associations, particularly the elevated risk in obese individuals and those with hypertriglyceridemia, are clinically relevant, as they may reflect modifiable metabolic derangements that could influence both uric acid levels and CKD progression. The high odds ratios, despite wide intervals, warrant clinical attention in risk-based screening strategies.

Strengths and limitations of the research

This study offers several noteworthy strengths. It is the first to assess the prevalence and determinants of hyperuricemia among CKD patients in Ethiopia, filling a critical gap in the local nephrology literature. In addition, it included a relatively large sample and various stages of CKD, which will give us a clinically diverse cohort.

However, some limitations must be noted. The cross-sectional design does not allow for causal inference, and consecutive sampling method may have resulted in selection bias as well as referral bias which may have been occurred due to the hospital-based sample, which probably overrepresents patients with advanced CKD. Key confounders like use of urate-lowering therapies, diuretics, losartan, and dietary purine intake were not measured, which could artificially elevate or reduce the serum UA level. Moreover, the reliance on single-point measurements for serum uric acid and 24-hour urinary protein excretion may be insufficient to account for biological variability. Lastly, although internal consistency of the knowledge measure was acceptable, the instruments were not formally psychometrically validated.

Conclusion

The research reported a high prevalence of hyperuricemia in the non-dialysis CKD patients. The independent factors that showed a significant association with hyperuricemia were male gender, low eGFR, elevated serum triglyceride levels, elevated BMI, and heavy proteinuria. These associations imply that patients with these traits are at higher risk of developing hyperuricemia. Vigilant screening and early detection of hyperuricemia in these high-risk non-dialysis CKD patients can facilitate risk stratification and guide personalized management strategies. Although these observations point to possible clinical significance, prospective research is required to determine if targeted treatment of hyperuricemia can impact renal outcomes or retard CKD progression.

Recommendations

Based on the findings of this study, it is recommended that screening for hyperuricemia in high risk patients may be important and it may help for early identification and treatment of hyperuricemia, which may retard the progression of non-dialysis CKD. Policymakers in the health sector are called upon to incorporate the screening and management of hyperuricemia into CKD national guidelines and also provide essential diagnostic facilities in health centers. Additionally, interventional and longitudinal investigations are needed to examine the causality of CKD progression and hyperuricemia and to assess the benefit of uric acid-lowering therapy on renal outcomes in the Ethiopian setting. Furthermore, future studies incorporating dialysis CKD patients is required.

Data availability

The datasets used and/or analyzed during the current study are not publicly available due to sensitivity issues but are available from the corresponding author upon reasonable request via email.

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Author contributions

A.K.Y. conceived, designed the research protocol. H.A., T.T., D.G., and W.H. approved the proposal. Y.Y.A. drafted, wrote and edited the manuscript along with literature review and quality assessment. All the authors have read and approved the final manuscript.

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Declarations

Competing interests
The authors declare no competing interests.

Ethical considerations
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Additional information
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