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Hyperuricemia and associated factors among hypertensive patients attending an academic hospital of Ethiopia: A cross-sectional study

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ABSTRACT

Background: Hypertension is a major public health problem in developing countries. Globally, nearly 1.13 billion adults had hypertension in 2015 and this is estimated to increase to 1.56 billion by 2025. Hyperuricemia is an important predictor of the progression of hypertension and is common in hypertensive patients. Hypertensive patients with hyperuricemia are at higher risk of cardiovascular disease.

Objective: To assess the prevalence of hyperuricemia and its associated factors among hypertensive patients attending the University of Gondar Comprehensive Specialized Hospital (UGCSH).

Method: An institutional-based cross-sectional study was conducted on 248 hypertensive patients attending the University of Gondar Comprehensive Specialized Hospital from January 2020 to February 2021. A convenient sampling technique was employed to select study participants. Socio-demographic and clinical characteristics were collected using a structured questionnaire via face-to-face interviews and reviewing medical records respectively. The biochemical parameters were measured by using a Mindray BS-200E chemistry analyzer. Data was entered using EpiData version 4.6.0.0 and analyzed using STATA vs. 14.0. Bivariable and multivariable binary logistic regression were fitted to identify factors associated with hyperuricemia. The odds ratio and 95 % CI were calculated to assess the strength of the association and a P-value <0.05 in the multivariable was considered statistically significant.

Results: A total of 248 patients were enrolled; 140 (56.5 %) were female. The mean age of patients was 57.9 ± 10.5 years. The overall prevalence of hyperuricemia was 42.3 %; males had a prevalence of 36.1 % and females of 47.1 %. High waist circumference, high body mass index, dyslipidemia, low estimated Glomerular Filtration Rate, elevated fasting blood glucose, elevated total cholesterol, elevated triglycerides, elevated Low-Density Lipoprotein cholesterol, and Low High-Density Lipoprotein cholesterol were found to be significantly associated with hyperuricemia.

Conclusion: This study demonstrated the predominant existence of hyperuricemia in hypertensive patients. Therefore, early diagnosis and monitoring of hyperuricemia are required before further complications occur.

1. Introduction

Hypertension or high blood pressure is defined as a systolic blood pressure (SBP) at or above 140mmHg and/or a diastolic blood pressure (DBP) at or above 90 mmHg [1]. Hypertension makes the heart work tougher to push the blood throughout the whole body [1,2]. There are two types of hypertension; primary or essential hypertension which is a form of hypertension that has no identifiable cause. It approximately affecting about 95 % of hypertensive cases, and it is likely to be hereditary and tends to be the result of an interaction between familial, environmental, or ecological factors, while secondary hypertension accounts for 5–10 % of cases [3]. A new classification recommended that the blood pressure criteria define normal blood pressure pre-hypertension, hypertension, and isolated systolic hypertension which is a usual occasion among the aged [4].

High renin levels predispose to high blood pressure by causing sodium retention through the subsequent mechanism as Increased renin→ Increased angiotensin II → Increased vasoconstriction, thirst/ADH, and aldosterone → Increased sodium reabsorption in the kidneys (DCT and CD) → Increased blood pressure [5]. Results from animal models

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propose a mechanism in two phases for the development of hyperuricemic hypertension in which uric acid (UA) induces acute vasoconstriction by activating the renin-angiotensin system followed by enhanced UA uptake into vascular smooth muscle cells leading to the cellular proliferation and secondary arteriolosclerosis that damages pressure natriuresis [6].

UA is the end result of purine catabolism and is produced from its precursor's xanthines and hypoxanthines primarily in the liver and the intestine. While this catabolic pathway has been well conserved during evolution in most of the living existing species, man, as well as apes, dogs, and some kinds of birds, have lost the functionality of the catabolic pathway of UA, consequently tending to accumulate in serum [7]. The rise in levels of serum UA (SUA) greater than 4.5 mg/dl in women and 5.5 mg/dl in men needs an attentive evaluation, while plasma concentrations greater than 7 mg/dl in men and 6 mg/dl in women classify a condition of overt hyperuricemia. From the biochemical point of view, a sparse solubility is the main reason for the accumulation of UA in tissue which leads to the formation of deposits of monosodium urate as crystalline [8] which leads to the onset of an inflammatory state.

This is particularly manifest in kidneys and joints leading to nephrolithiasis and gout in some predisposed patients. In terms of pathophysiology, the levels of SUA are influenced by many mechanisms for instance, both endogenous and exogenous. Hyperuricemia can result due to either high UA production or most frequently, a low renal excretion with impaired renal function [9]. Since the kidney is the main and most important route of UA clearance, renal function integrity is important for the maintenance of normal or standard UA levels while excretion of the small bowel contributes to only a small percentage. In the past few years, the increased interest in the molecular mechanisms of renal responsible for the clearance of UA has led to the discovery of various transport systems of urate located in the proximal tubule of the kidney. These proteins or transport systems are involved in the processes of secretion and reabsorption of UA, and their loss-of-function mutations are linked with some specific polymorphisms that may contribute to the development of hyperuricemia [10].

Lifestyle habits and dietary intake also have a large influence on the levels of serum urate. For instance, consumption of alcohol >15 g/day can lead to a high risk of hyperuricemia, particularly for beer drinkers [11] with a 93 % relative risk of experiencing a gout attack compared to that of non-alcohol consumers [12]. The concept that UA may be involved in hypertension is not a new one. In fact, in a paper published in 1879 that originally described essential hypertension, Frederick Akbar Mohamed noted that many of his subjects came from families with a history of gout. He hypothesized that the UA might be integral to the development of essential hypertension [13].

Effective control of hypertension depends on important advances in our knowledge and understanding of its risk factors [2]. Animal models and epidemiologic studies have verified that raised serum uric acid is associated with blood pressure progression and hypertension [14–16]. Recently, the prevalence of HUA (Hyperuricemia) has potentially endorsed recent shifts in diet and lifestyle, better medical care, and increased long life [17]. Even though knowing the level of serum uric acid and its associated factors in hypertensive patients is crucial, the problems were not well studied in Ethiopia, and flimsy with conflicting data are available. For this reason, conducting the study is important to improve early diagnosis, prevention of complications, and ensuring the quality of life of hypertensive patients.

2. Methods and materials

2.1. Study design, area, and period

The study was conducted at the University of Gondar Comprehensive Specialized Hospital from January 1, 2020 to February 15, 2021 on hypertensive patients in the chronic ward attending their follow-up. Facility-based cross-sectional study design was conducted to carry out the project.

2.2. Sample size determination and sampling technique

A single population proportion formula, [$n = (Z\ \alpha/2)^2\ p\ (1\text{-}p)/d^2$], was used to calculate the required sample size by considering the following assumptions: The prevalence of hyperuricemic among hypertensive patients = 46.9 % [18], 95 % confidence level, and 5 % degree of precision which gives 383. Finally, the population correction formula was employed since the population is less than 10,000 (i.e. 700) and the final sample size for the study was 248. All consecutively identified hypertensive patients who fulfilled the inclusion criteria were enrolled in the study. The source population of this study was all patients attending chronic wards of UGCSH. The study population was all sampled hypertensive patients attending follow-up at the chronic ward and fulfilled the inclusion criteria. Hypertensive patients with pregnancy, those who are critically ill, and those on chemotherapy were excluded from the study.

2.3. Data collection procedures

Socio-demographic and clinical data were collected by trained nurses using an interviewer-administered structured questionnaire. In addition, the blood samples were collected and analyzed by trained laboratory technologists. Two days of training were given to the data collectors before the data collection period to familiarize them with the objective of the study.

2.4. Sample collection and processing

After obtaining written informed consent from the study participant, about 5 ml venous blood was collected by venipuncture from superficial veins of the antecubital fossa after strictly following the standard operating procedure (SOPs), under aseptic conditions and after cleaning the venipuncture site with 70 % alcohol. Smaller-gauge (20 or 25-gauge) needles were employed to collect the sample. Butterfly needles were used for some patients with small or invisible veins. Experienced laboratory technologists collected the blood samples by the following procedure: Firstly, 5 mL of venous blood were collected from each study subject after overnight fasting. Secondly, the blood specimens were allowed to stay for 20–30 min for clot formation. Then, the specimens were centrifuged at 3000 rpm and the serum was separated from the whole blood, stored under an ice bag, and transferred to the University of Gondar Hospital, clinical chemistry section for analysis.

2.5. Laboratory test principles

The blood collected from each hypertensive patient was processed for biochemical tests. The biochemical tests were determined at the University of Gondar Hospital, clinical chemistry section. The serum was analyzed for serum uric acid, serum creatinine, serum glucose, and lipid profiles and these were measured by using a Mindray BS-200E chemistry analyzer (Shenzhen Mindray Bio-Medical Electronics Co. Ltd, China).

Triglyceride (TG) was measured enzymatically in serum using a series of coupled reactions in which TG is hydrolyzed to produce glycerol. Glycerol is then oxidized to produce 4-(benzoquinone-monoimino)-phenazone that is used to absorb light. The amount of absorbed light that is directly proportional to the concentration of TAG in the sample was measured spectrophotometrically at 500 nm. The High-Density Lipoprotein Cholesterol (HDL-C) is measured directly in the serum in which apo-B containing lipoproteins such as chylomicrons (CM), Low-Density Lipoprotein (LDL), and Very Low-Density Lipoprotein (VLDL) in the specimen are reacted with a blocking reagent that makes them non-reactive with the enzymatic cholesterol reagent under conditions of the assay. Consequently, the apo-B-containing lipoproteins are effectively and efficiently excluded from the assay and only the HDL-C is

detected under the assay condition.

Uric acid was also measured enzymatically in which uric acid is oxidized by uricase enzyme to produce allantoin and hydrogen peroxide. The generated hydrogen peroxide (H_2O_2) reacts with 4- amino-antipyrine and 3,5-dichloro-2-hydroxybenzene sulfonate in a reaction catalyzed by the enzyme peroxidase to produce a colored product. The formed colored product was measured spectrophotometrically at 520 nm. The amount of light absorbed by the colored product is directly proportional to the concentration of serum uric acid in the sample.

Fasting blood sugar was measured enzymatically based on the test principle of glucose oxidase in which the enzyme glucose oxidase oxidizes beta D-glucose into D-gluconic acid and hydrogen peroxide. The hydrogen peroxide (H_2O_2) enters into the second reaction involving p-hydroxybenzoic acid and 4-aminoantipyrine in the presence of peroxidase with the formation of a quinoneimine dye complex. This complex whose concentration is directly proportional to the concentration of glucose in the sample was measured spectrophotometrically at 510 nm.

Serum creatinine was measured enzymatically in which cups were systematically set on a rack that goes onto a Mindray BS-200E chemistry analyzer (Shenzhen Mindray Bio-Medical Electronics Co. Ltd, China). This is an auto-analyzer that uses the Jaffe reaction to quantify creatinine; creatinine reacts with picric acid in the presence of an alkaline pH to produce a yellow-red complex that has a maximum absorbance at 512 nm. The rate of dye formation is proportional to the level of creatinine in the sample. The analyzer reads out this absorbance and based on its software, it calculates the concentration of serum creatinine.

The estimated glomerular filtrate (eGFR) was calculated using the CKD-EPI (Chronic Kidney Disease-Epidemiology Consortium) equation, expressed as a single equation:

GFR = 141 X min (Scr/κ, 1) α X max (Scr/κ, 1)-1.209 X 0.993Age X 1.018 [if female] X

1.159 [if black] where Scr is serum creatinine (mg/dL), κ is 0.7 for females and 0.9 for males, α is −0.329 for females and −0.411 for males, min indicates the minimum of Scr/κ or 1, and max indicates the maximum of Scr/κ or 1 [19]. This formula was chosen based on the recent local study which indicated that CKD-EPI was superior to other equations like Cockcroft and Gault (CG), Modification of Diet in Renal Disease (MDRD) for serum creatinine and estimated glomerular filtration rates in HIV positive and negative adults in Ethiopia [20].

2.6. Measurement of blood pressure

Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using the instrument mercury-based sphygmomanometer after the participants were rested for more than or equal to 10 min. For those study subjects with a SBP ≥140 mm of mercury (mmHg) and a DBP ≥90 mmHg, blood pressure was measured again after 1 min and finally, the average values were taken. The blood pressure of study participants was measured by experienced nurses.

2.7. Anthropometric measurements

Anthropometric measurements (weight, height, and waist circumference) were measured according to the WHO stepwise approach guideline. Height was measured to the nearest 0.5 cm using a standiometer and weight was recorded to the nearest 0.1 kg with the patient wearing light clothes using a balance. BMI was calculated as weight divided by height squared (kg/m2) [21].

Waist circumference (WC) was measured at the midpoint between the lower margin of the least palpable rib and the top of the iliac crest, using a stretch-resistant tape. WC was measured after instructing each study subject to stand with feet close together, arms at the side, and body weight evenly distributed, wearing light clothes.

2.8. Operational definitions

Hypertension (HBP) was defined as Systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg or current use of blood pressure-lowering medication was used to define hypertension [22].

Hyperuricemia was defined as serum uric acid ≥7 mg/dl in males or ≥6 mg/dl in females [23].

Hypercholesterolemia was defined as a total cholesterol level ≥200 mg/dl;

Hypertriglyceridemia was defined as a total TG ≥ 150 mg/dl;

A High LDL level was defined as a total LDL level ≥130 mg/dl;

Low HDL level was defined as HDL level <40 mg/dl in males and <50 mg/dl in females [24].

Elevated fasting blood sugar (FBS) was defined as FBS/FBG >115 mg/dl [22].

Study participants were classified as:

- Underweight (BMI<18.5 kg/m2),
- Normal weight (18.5–24.9 kg/m2),
- Overweight (BMI = 25-29.9Kg/m2) and
- Obese (BMI ≥30Kg/m2) [21].

Waist circumference (WC) > 88 cm for females and WC > 101 cm for males was taken as high WC [21]. Estimated Glomerular Filtration Rate (eGFR) was calculated by using the Chronic Kidney Disease Epidemiology Collaboration/Creatinine (CKDEPI) formula which was chosen based on a study conducted in our country [20].

2.9. Data quality assurance

To ensure data quality, the questionnaire had been prepared in English and translated to the Amharic language and then re-translated back to English to see its consistency. The questionnaire was pre-tested on 5 % of the sample size at Gondar Poly Health Center to ensure its validity. During data collection, close supervision was conducted throughout the whole study period. There was continuous monitoring of test quality and checking of all steps. The pre-analytical, analytical, and post-analytical factors that can interfere with the results of biochemical measurements were controlled and maintained by the medical laboratory technologist who was conducting the biochemical analysis. The proper functioning of instruments, laboratory reagents, and technical performance were checked daily by using quality control samples before running patient samples. The Experiments were repeated for the results found outside established values.

2.10. Statistical analysis

Data were checked, coded, and cleaned for inconsistencies and missing values, and then entered into EpiData version 4.6.0.0 statistical software. Then after, data is exported to STATA version 14.0 for analysis. Descriptive statistics (mean, median, frequency, percentage) were used to summarize the characteristics of the study population through tables and charts. To assess the distribution of data, the Shapiro-Wilk test was conducted. A p-value >0.05 in the Shapiro-Wilk test is considered as data are normally distributed. To identify factors associated with hyperuricemia, a binary logistic regression model was fitted. The Hosmer-Lemeshow goodness-of-fit test was used to assess the fitness of the model. Independent variables having a p-value less than or equal to 0.2 in bi-variable analyses were included in the multi-variable analysis to control confounders in the binary logistic regression model. An odds ratio (OR) at 95 % confidence interval (CI) was determined to see the strength of the association between the independent variables and outcome variables. P-value <0.05 in the multi-variable regression model was considered statistically significant.

3. Results

3.1. Socio-demographic characteristics of the study participants

A total of 248 hypertensive patients participated in the study; of the participants, 140 (56.45 %) were females. The patient's age ranged from 29 to 85 years with a mean age of 57.92 years (SD 10.5 years) and the majority being in the age group between 41 and 70 years (83.5 %);

Regarding their marital status, one hundred forty-four (58.1 %) of the respondents were married. Concerning their educational status, one hundred thirty-one (52.8 %) of the patients had no formal education. More than two-thirds, 176 (70.9 %) were urban dwellers. The majority, 90 (36.3 %) of patients were housewives (Table 1).

As shown in Table 1 above the association of UA level with different variables was studied using bivariable logistic regression; from the result, there was no significant association between hyperuricemia and socio-demographic characteristics. Gender was not associated with the presence of abnormal serum uric acid concentration, p = 0.082. Age was categorized as ≥45 and < 45; it was not associated with hyperuricemia, p = 0.503. Marital status, educational level, residence, and occupational status were 0.434, 0.102, 0.674, and 0.451 respectively.

3.2. Clinical and anthropometric characteristics of the patients

Out of the 248 patients, 84 (33.9 %) had a family history of hypertension. As it has been described in Table 2 below 49 (19.8 %) patients had high waist circumference with a mean WC of 68.4 (SD: 16.4) cm. Median BMI of 26.7 (IQR: 24.43, 30.115) kg/m^2. More than half of the patients 134 (54.03 %) had SBP >140 mm Hg, and also more than half (51.6 %) had DBP >90 mm Hg. The Mean systolic blood pressure (SBP) was 136.5 ± 13.98 mm Hg, and the median diastolic blood pressure (DBP) was 90.0 (IQR: 80.0, 90.0) mm Hg. The median duration of hypertension was 5 years (IQR: 3, 8). Among 248 patients, only 38 (15.3 %) were physically active, 27 (10.9 %) had a history of alcohol use, 90 (36.3 %) had coffee drinking habit, and 8 (3.2 %) had a history of smoking. Other clinical characteristics are summarized in Table 2.

According to the above table, in the bivariable logistic regression, there was an association between the presence of hyperuricemia and waist circumference, body mass index, and systolic blood pressure. Family history of hypertension, diastolic blood pressure, duration of hypertension, physical activity, alcohol use, coffee consumption, and smoking habit were not associated with the presence of hyperuricemia, p = 0.509, 0.081, 0.516, 0.457, 0.162, 0.574, and 0.618, respectively. The study participants who had hyperuricemia were more likely to be obese (≥30) compared to those without the presence of hyperuricemia. Grouped BMI showed that higher BMI levels were associated with the presence of hyperuricemia, p = 0.001 (Table 2).

3.3. Association between hyperuricemia and laboratory findings

In bivariable logistic regression, elevated total cholesterol was associated with the presence of hyperuricemia, p = 0.013. Elevated triglycerides was also associated with the presence of hyperuricemia, 79 (75.2 %) vs. 74(51.7 %), p = 0.001. The study participants with low level of HDL were more likely to have hyperuricemia, p = 0.004. The study participants who had high level of LDL was significantly associated with the presence of hyperuricemia, p = 0.001.

The presence of any dyslipidemia was associated with hyperuricemia, hyperuricemic group-102(97.1 %) vs. normouricaemia group-120 (83.9 %), p < 0.0001. Low levels of eGFR were associated with hyperuricemia, p < 0.0001. Chronic kidney disease (CKD) stage 3 or worse (IV and V) was associated with the presence of hyperuricemia, 22 (20.9 %) vs. 5(3.5 %), p = 0.003; (Table 3).

3.4. Correlation of hyperuricemia with clinical predictor variables and laboratory findings

Pearson's correlation coefficient indicated that, a significantly positive correlation between elevated serum uric acid and biochemical parameters like tCho (r = 0.3, p-value = 0.001), TG (r = 0.4, p-value<0.0001), LDL (r = 0.4, p-value<0.0001), FBG (r = 0.2, p-value = 0.003), and with HDL, there is a significantly negative correlation (r = −0.3, p-value = 0.001). In addition to that, some clinical and anthropometric characteristics including SBP (r = 0.2), WC (r = 0.3), and BMI (r = 0.3) have a significantly positive correlation with elevated serum uric acid (Table 4).

Table 1
Socio-demographic characteristics of hypertensive patients attending UOGCSH, Northwest Ethiopia.

Variables	Category	N (%) N = 248	Uric acid level, N (%)		P-value
			N = 248		
			Hyperuricemia	Normouricaemia	
			N = 105	N = 143	
Gender	Male	108(43.5)	39(37.1)	69(48.3)	0.082
	Female	140(56.5)	66(62.9)	74(51.7)	
Age	≥45	213(85.9)	92(87.6)	121(84.6)	0.503
	<45	35(14.1)	13(12.4)	22(15.4)	
Marital status	Single	11(4.4)	4(3.8)	7(4.9)	0.434
	Married	144(58.1)	61(58.1)	83(58.0)	
	Divorced	12(4.8)	1(1)	11(7.7)	
	widowed	81(32.7)	39(37.1)	42(29.4)	
Educational level	Illiterate	92(37.1)	47(44.8)	45(31.4)	0.102
	Able to read & write	39(15.7)	15(14.3)	24(16.8)	
	Primary education	40(16.1)	12(11.4)	28(19.6)	
	Secondary & above	77(31.1)	31(29.5)	46(32.2)	
Residence	Urban	176(71.0)	76(72.4)	100(69.9)	0.674
	Rural	72(29.0)	29(27.6)	43(30.1)	
Occupation	Housewife	90(36.3)	43(41.0)	47(32.8)	0.451
	Self-employed	60(24.2)	25(23.8)	35(24.5)	
	Gov't employed	45(18.1)	17(16.2)	28(19.6)	
	Unemployed	23(9.3)	9(8.6)	14(9.8)	
	Farmer	17(6.9)	6(5.7)	11(7.7)	
	Others	13(5.2)	5(4.7)	8(5.6)	

Table 2
Clinical and anthropometric characteristics of hypertensive patients attending the UOGCSH, Northwest Ethiopia.

Variables	Category	N (%) N = 248	Uric acid level, N (%)		P-value
			N = 248		
			Hyperuricemia	Normouricaemia	
			N = 105	N = 143	
FHHTN	Yes	84(33.9)	38(36.2)	46(32.2)	0.509
	No	164(66.1)	67(63.8)	97(67.8)	
WC	High	49(19.8)	35(33.3)	14(9.8)	**<0.0001**[a]
	Normal	199(80.2)	70(66.7)	129(90.2)	
BMI	Underweight (<18.5)	0(0 %)	0(0 %)	0(0 %)	**0.001**[a]
	Normal(18.5–24.9)	80(32.3)	19(18.1)	61(42.6)	
	Overweight(25–29.9)	103(41.5)	50(47.6)	53(37.1)	
	0bese(≥30)	65(26.2)	36(34.3)	29(20.3)	
SBP	>140 mmHg	134(54.0)	65(61.9)	69(48.2)	**0.034**[a]
	<140 mmHg	114(46.0)	40(38.1)	74(51.8)	
DBP	>90 mmHg	128(51.6)120(48.4)	61(58.1)	67(46.9)	0.081
	<90 mmHg		44(41.9)	76(53.1)	
Duration of HTN	<5 year	132(53.2)	55(52.4)	77(53.8)	0.516
	6–10 year	100(40.3)	41(39.0)	59(41.3)	
	>10 year	16(6.5)	9(8.6)	7(4.9)	
Physical activity	No	210(84.7)	91(86.7)	119(83.2)	0.457
	Yes	38(15.3)	14(13.3)	24(16.8)	
Alcohol	Yes	27(10.9)	8(7.6)	19(13.3)	0.162
	No	221(89.1)	97(92.4)	124(86.7)	
Coffee	Yes	90(36.3)	36(34.3)	54(37.8)	0.574
	No	158(63.7)	69(65.7)	89(62.2)	
Smoking	Current smoker	1(0.4)	0(0 %)	1(0.7)	0.618
	Ex-smoker	7(2.8)	3(2.9)	4(2.8)	
	Never smoker	240(96.8)	102(97.1)	138(96.5)	

FHHTN: Family History of Hypertension; WC: Waist Circumference; BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.
[a] P-value <0.05, statistically significant association.

Table 3
Association between hyperuricemia and laboratory findings among hypertensive patients attending at UOGCSH, Northwest Ethiopia.

Variables	Category	N (%) N = 248	Uric acid level, N (%)		P-value
			N = 248		
			Hyperuricemia	Normouricaemia	
			N = 105	N = 143	
Total	>200	105(42.3)	54(51.4)	51(35.7)	**0.013***
cholesterol	<200	143(57.7)	51(48.6)	92(64.3)	
Triglycerides	>150	153(61.7)	79(75.2)	74(51.7)	**0.001***
	<150	95(38.3)	26(24.8)	69(48.3)	
HDL	Low	128(51.6)	68(64.8)	60(42.0)	**0.004***
	High	120(48.4)	37(35.2)	83(58.0)	
LDL	>130	59(23.8)	36(34.3)	23(16.1)	**0.001***
	<130	189 (76.2)	69(65.7)	120(83.9)	
Dyslipidemia			102(97.1)	120(83.9)	**<0.0001***
FBG	High	40(16.1)	25(23.8)	15(10.5)	**0.006***
	Normal	208(83.9)	80(76.2)	128(89.5)	
eGFR	above 60	221(89.1)	83(79.1)	138(96.5)	**<0.0001***
	30–60	13(5.3)	10(9.5)	3(2.1)	
	15–30	10 (4.0)	8(7.6)	2(1.4)	
	<15	4(1.6)	4(3.8)	0(0 %)	
Stage 3 or worse CKD			22(20.9)	5(3.5)	**0.003***

HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein; FBG: Fasting Blood Glucose; eGFR: estimated Glomerular Filtration Rate; CKD: Chronic Kidney Disease; mg/dl: milligram per deciliter; *P-value <0.05, statistically significant association.
Dyslipidemia- Defined as elevated total cholesterol, low-density lipoprotein (LDL), triglycerides or low levels of high-density lipoprotein (HDL).

3.5. Bivariable and multivariable analyses of socio-demographic, clinical, and anthropometric characteristics

A binary logistic regression analysis model was executed to assess the association of each variable with hyperuricemia among hypertensive patients. Based on this, clinical variables elevated waist circumference, being overweight (25–29.9) and obese (≥30) in body mass index (BMI), and elevated systolic blood pressure (SBP) exhibited statistically significant association with hyperuricemia in the binary logistic regression model. However, in the multivariable logistic regression analysis model, only elevated waist circumference (AOR = 4.97; 95%CI: 1.80–13.72), being BMI between 25 and 29.9 kg per m^2 (AOR = 2.94; 95%CI: 1.50–5.76), have remained associated factors with hyperuricemia prevalence in hypertensive patients (Table 5).

4. Discussion

4.1. Prevalence of hyperuricemia among hypertensive patients

Various literature displays that hyperuricemia prevalence varies in

Table 4
Pearson's correlation of biochemical parameters and some clinical characteristics with serum uric acid level at UOGCSH, Northwest Ethiopia.

Parameters	Mean ± SD	Correlation coefficients	P-value
tCho (mg/dl)	200.1 ± 69.2	0.3	0.001[a]
eGFR	88.1 ± 29.0	−0.3	<0.0001[a]
TG (mg/dl)	177.7 ± 68.3	0.4	<0.0001[a]
HDL (mg/dl)	42.9 ± 12.3	−0.3	0.001[a]
LDL (mg/dl)	109.7 ± 33.6	0.4	<0.0001[a]
FBG (mg/dl)	106.5 ± 23.2	0.2	0.003[a]
SBP (mmHg)	136.5 ± 13.9	0.2	0.020[a]
DBP (mmHg)	82.7 ± 9.5	0.1	0.066
WC (cm)	72.4 ± 16.4	0.3	<0.0001[a]
BMI (kg/m2)	27.2 ± 3.5	0.3	<0.0001[a]

tCho: Total Cholesterol; eGFR: estimated Glomerular Filtration Rate; TG: Triglyceride; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; FBG: Fasting Blood Glucose; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; WC: Waist Circumference; BMI: Body Mass Index; mmHg: millimeter mercury; mg/dl: milligram per deciliter; kg/m2: Kilogram per meter square.
[a] P-value <0.05 is statistically significant.

different population groups and settings. In the current study, hyperuricemia prevalent is (42.3 %) in hypertensive patients. The prevalence of hyperuricemia was 36.1 % among male and 47.1 % among female participants which is lower than a retrospective case-control study conducted in Nigeria to assess serum uric acid levels among hypertensive patients. They found a prevalence of 59 % in the male and 62 % in the female participants [25]. This could be due to lower cutoff serum uric acid level which was >5.5 mg/dl for both female and male hypertensive patients. Our study's cutoff was >6 mg/dl for females and >7 mg/dl for male hypertensive patients.

A cross-sectional study carried out by Nguedia et al. in Cameroon, found a prevalence of 49.5 % of hyperuricemia among hypertensive patients from both females and males [26]. Another study in Kenya found a prevalence of 44 % among hypertensive patients [27]. A study conducted by Lin et al. in Taiwan on hypertensive patients on treatment found a prevalence of 35 % among male patients and 45 % among female patients [18] and a study carried out among US adult hypertensive patients obtained a prevalence of 41.7 % [28]. These findings are consistent with our study since the overall prevalence was 42.3 %. According to a cross-sectional study carried out by Oumar et al. in Mali, they obtained a higher prevalence of 66.7 % (75.9 % in female and 35.3 % in male patients) among 51 hypertensive patients on follow-up [29]. This could be due to the smaller sample size (51) in their study compared to ours which was 248 patients.

The result obtained from our study was higher than a longitudinal analytical and comparative study conducted by Ikama et al. in Brazzaville, Congo. They found the prevalence of 30.2 % hyperuricemia among hypertensive patients [30]. A community-based cross-sectional study carried out by Kamdem et al. in Douala, Cameroon found the prevalence of hyperuricemia 31.8 % (95 % CI: 28.7–34.9) [31]. A hospital-based cross-sectional study conducted in Nepal by Poudel et al. found a prevalence of 28.8 % among hypertensive patients [32]. These findings are inconsistent with our study. The difference could be explained by various reasons such as newly diagnosed patients, variations in study design, and sample size.

4.2. Socio-demographic characteristics with hyperuricemia

There was a female dominance (56.5 %) in the current study. This could be due to health-seeking behavior among the sexes rather than

Table 5
Bivariable and multivariable analyses of socio-demographic, clinical, and anthropometric characteristics as predictors of hyperuricemia among hypertensive patients at UOGCSH, Northwest Ethiopia.

Variables	Category	Hyperuricemia		P-value	COR (95 % CI)	AOR (95 % CI)
		Yes N (%)	No N (%)			
Gender	Male	39(37.1)	69(48.3)	0.082	0.63(0.38–1.06)	0.87(0.45–1.66)
	Female	66(62.9)	74(51.7)		1[a]	1[a]
Age	≥45	92(87.6)	121(84.6)	0.503	1.29(0.62–2.69)	1.55(0.66–3.62)
	<45	13(12.4)	22(15.4)		1[a]	1[a]
Educational level	Illiterate	47(44.8)	45(31.4)	0.161	1.55(0.84–2.86)	1.12(0.54–2.32)
	Able to read & write	15(14.3)	24(16.8)	0.852	0.93(0.42–2.04)	0.71(0.29–1.72)
	Primary education	12(11.4)	28(19.6)	0.277	0.64(0.28–1.44)	0.50(0.20–1.24)
	Secondary & above	31(29.5)	46(32.2)		1[a]	1[a]
FHHTN	Yes	38(36.2)	46(32.2)	0.509	1.20(0.70–2.03)	1.13(0.62–2.05)
	No	67(63.8)	97(67.8)		1[a]	1[a]
WC	High	35(33.3)	14(9.8)	<0.0001	4.61(2.32–9.14)	**4.97(1.80–13.72)**[b]
	Normal	70(66.7)	129(90.2)		1[a]	1[a]
BMI	Normal (18.5–24.9)	19(18.1)	61(42.6)		1[a]	1[a]
	Overweight (25–29.9)	50(47.6)	53(37.1)	0.001	3.03(1.59–5.77)	**2.94(1.50–5.76)**[b]
	0bese (≥30)	36(34.3)	29(20.3)	<0.0001	3.98(1.96–8.11)	1.30(0.47–3.61)
eGFR	above 60	83(79.1)	138(96.5)		1[a]	1[a]
	30–60	10(9.5)	3(2.1)			
	15–30	8(7.6)	2(1.4)			
	<15	4(3.8)	0(0 %)	**<0.0001**[b]		**0.30 (0.15–0.59)**[b]
Diastolic BP	>90 mmHg	61(58.1)	67(46.9)	0.081	1.57(0.95–2.61)	0.55(0.10–3.64)
	<90 mmHg	44(41.9)	76(53.1)		1[a]	1[a]
Duration of HTN	<5 year	55(52.4)	77(53.8)		1[a]	1[a]
	6–10 year	41(39.0)	59(41.3)	0.919	0.97(0.57–1.65)	0.80(0.44–1.46)
	>10 year	9(8.6)	7(4.9)	0.271	1.8(0.63–5.13)	1.62(0.48–5.44)
Physical activity	No	91(86.7)	119(83.2)	0.457	1.31(0.64–2.68)	1.17(0.51–2.69)
	Yes	14(13.3)	24(16.8)		1[a]	1[a]
Alcohol	Yes	8(7.6)	19(13.3)	0.162	0.54(0.23–1.28)	0.52(0.20–1.33)
Use	No	97(92.4)	124(86.7)		1[a]	1[a]
Coffee	Yes	36(34.3)	54(37.8)	0.574	0.86(0.51–1.46)	0.81(0.44–1.49)
consumption	No	69(65.7)	89(62.2)		1[a]	1[a]

FHHTN: Family History of Hypertension; WC: Waist Circumference; BMI: Body Mass Index; eGFR: estimated Glomerular Filtration Rate; HTN: Hypertension; BP: Blood Pressure; COR: Crude Odds Ratio; AOR: Adjusted Odds Ratio.
[a] Reference category.
[b] Indicates statistically significant association.

Table 6
The final logistic model (adjusted one) with all variables.

Logistic regression			Number of obs		= 248
			LR chi2(13)		= 45.19
			Prob > chi2		= 0.0000
Log likelihood = −146.38196			Pseudo R2		= 0.1337
Uricemia	Coef.	Std. Err.	z	P > z	[95 % Conf. Interval]
Gender	−0.0295261	0.3152698	−0.09	0.925	−0.6474436 0.5883914
Educational level	−0.076815	0.1224408	−0.63	0.530	−0.3167947 0.1631646
Physical activity	0.078404	0.4199579	0.19	0.852	−0.7446984 0.9015063
Alcohol consumption	0.4302947	0.4874067	0.88	0.377	−0.5250048 1.385594
Coffee consumption	0.0785688	0.3049451	0.26	0.797	−0.5191127 0.6762502
Smoking habit	0.022461	0.7528943	0.03	0.976	−1.453185 1.498107
SBP	1.052351	0.9682973	1.09	0.277	−0.8454768 2.950179
BMI	0.2834663	0.2320213	1.22	0.022	1.501287 5.7682196
Duration of HTN	0.0464271	0.2401544	0.19	0.847	−0.4242669 0.5171212
Waist circumference	1.068052	0.4554702	2.34	0.019	0.1753469 1.960757
eGFR	−1.275791	0.3665632	−3.48	0.001	−1.994241 -.5573401
Age	−0.2136068	0.4256928	−0.50	0.616	−1.047949 0.6207357
DBP	−0.8310847	0.9527032	−0.87	0.383	−2.698349 1.036179
_cons	0.2211591	2.563716	0.09	0.931	−4.803632 5.24595

Table 6 refers to the adjusted logistic model with all variables.
Coef = Coefficient.
Std. Err = Standard Error.

higher prevalence among females. The study by Nguedia et al. in Cameroon revealed that female patients were 169 (56.9 %) and 128 (43.1 %) male patients, which also shows female dominance [26]. The patient's age ranged from 29 to 85 years old with a mean age of 57.9 years old (SD: 10.5 years) and the repeated age group was between 41 and 70 years 207 (83.5 %) which is similar to the Cameroonian study where the mean age of patients was 41.95 ± 14.83 years (range: 20–76 years) [26]. The Nigerian study revealed that male patients ranged from 27 to 75 years old with a mean age of 51.2 years (SD: 12.1 years) while the female patient's age ranged from 18 to 84 years old with a mean age of 51.8 years (SD: 16.4 years) [25]. In Mali, study participants were nearly similar to our study, aged between 46 and 60 and those above 60 years were the majority of their patients with 39.2 % and 37.3 % respectively [29].

In the current study, age was not associated with hyperuricemia, p = 0.115. Grouping patients by age groups revealed that a higher percentage of participants aged at least 40 years and above had elevated serum uric acid but still, there was no significant association. This was not in line with studies carried out on uric acid and age. Consequently, we analyzed data further by adjusting for the covariates (AOR). However, there was no significant association between age and hyperuricemia, AOR: 1.19 (95 % CI: 0.93–1.53). This is in line with a study conducted by Oumar et al. in Bamako, Mali, and by Poudel et al. in Nepal. They found that there was no significant association between age and hyperuricemia, (p = 0.87, p = 0.251) [29,32]. However, Ofori et al. and Teng et al. found a contrary result where serum uric acid was associated with the presence of hypertension in the elderly group and mean age respectively [33,34]. The variances in these findings could be attributed to the difference in the characteristics of participants and age is not directly associated with higher uric acid levels because uric acid production and excretion are regulated by various hormones and enzymes in the body.

In the present study, married individuals are predominant and this finding is in line with the study conducted in Douala, Cameroon by Kamdem et al. and both are not significantly associated with the presence of hyperuricemia [31]. The same study done in Douala, Cameroon found that a secondary education and above takes the highest number; this is consistent with our study. Oumar et al. found that urban dwellers take health service advantage over rural dwellers (41, 80.4 %). This is in agreement with our study, (176, 71 %) of patients were urban dwellers [29]. A study conducted in Mali revealed that housewives are predominant (21, 41.2 %, p = 0.98). This finding is in agreement with our result since housewives were high among other occupations (90, 36.3 %, p = 0.459) [29].

4.3. Clinical and laboratory characteristics with hyperuricemia

In the current study, the family history of hypertension was not significantly associated with the presence of hyperuricemia, AOR: 1.13 (95%CI: 0.62–2.05), p = 0.689. This is in contrast with a cross-sectional study conducted in Cameroon by Kamdem et al. They found that a family history of hypertension was significantly associated with the elevated SUA, p = 0.047 [31]. This could be due to the large sample size (839) of the Cameroonian study compared to our sample size (248). In our study, elevated waist circumference was significantly associated with the presence of hyperuricemia (AOR: 4.97 (95 % CI: 1.80–13.72) or (r = 0.3; p < 0.0001). This study is in line with a study carried out in River State, Nigeria by Ofori and Odia. They found waist circumference (r = 0.489; P < 0.001) [33]. This may be explained by the fact that leptin could be a pathogenic factor responsible for hyperuricemia in obese patients [35].

In the current study, there was a significant association between hyperuricemia and BMI (1.63 (1.10–2.42) or r = 0.305, p < 0.001). A descriptive cross-sectional study conducted in River state, Nigeria by Ofori and Odia is in agreement with the present study since r = 0.476, p = 0.001. However, in another study [29] researchers did not get an association between hyperuricemia and body mass index, as their study was limited only to a small sample size of 51 cases.

Blood pressure was noted to be inadequately controlled in our patients with more than half (54 %) having SBP> 140 mm Hg and more than half (51.6 %) with DBP>90 mm Hg. This was almost similar to the Taiwan study in Taipei where the mean blood pressure of their patients was SBP of 137 and a DBP of 81 [18]. In our study, we found SBP of 136.5 mmHg and a DBP of 82.7 mmHg. Poor adherence and health-seeking behavior might be responsible for the poor control of BP. However, the study conducted in Cameroon by Nguedia et al. had a lower percentage of 32.7 % with a blood pressure of ≥140/90 mmHg [26]. In their study, they included both hypertensive patients and normotensive individuals who were considered as controls, therefore this could explain the lower blood pressures in the Cameroonian study.

In this study significant association between hyperuricemia and blood pressure was not noted which is similar to the study conducted in Mali, and the United Kingdom [29,36]. However, in a study conducted in Cameroon by Nguedia et al., they found a significant independent

association between SUA with both SBP and DBP; an increase in both SBP and DBP was also marked by a subsequent rise in SUA level [26]. In Nigerian study, there was a significant positive correlation between serum uric acid with SBP ($r = 0.192$; $p < 0.001$) and DBP ($r = 0.216$; $p < 0.001$) [25]. These two Cameroonian and Nigerian studies compared hypertension patients and controls whose BP was <120/80 mmHg. In the Mali study, all participants were known hypertensive patients; hence there was an association but no significant association.

In our study, the median duration of hypertension was 5 years (IQR: 3, 8). A study conducted in Mali on 51 hypertensive patients had a mean duration of hypertension of 10.73 months [29]. The difference could be due to the upper limit of our study which is 16 years and in the Mali study, it was 40 months. In both studies, this variable was not statistically significant. A study conducted in Taiwan confirms that SUA level increases with increases in the duration of hypertension. They grouped the duration of hypertension into 4 groups (≤1 year, >1 to ≤ 3 years, >3 to ≤ 5 years, >5 years) and increases from 9.8 %, 19.7 %, 17.0 %, and 50.8 % respectively [18]. Also, our study showed similar trends since SUA increased with the duration of hypertension, 41 % (<5 years), 41 % (6–10 years), and 56 % (>10 years).

In our study, 38 (15.3 %) of study participants were physically active and not significantly associated with hyperuricemia, AOR: 1.17(95 % CI: 0.51–2.69), $p = 0.703$. This is in contrast with a study conducted in Cameroon by Nguedia et al. They found that physical exercise and hyperuricemia were significantly associated ($r = 0.274$; $P < 0.0001$) [26]. Coffee consumption was not significantly associated with the presence of hyperuricemia, AOR: 0.81 (95 % CI: 0.44–1.49), $p = 0.499$. This is in contrast with a study carried out in Japan by Pham et al. They showed evident inverse associations of coffee intake with SUA levels and hyperuricemia [37]. This could be explained due to the use of a large sample size (12,957) in the Japanese study compared to ours (248). In the Japanese study, participants are controlled for coffee consumption. Coffee may also contain substances that inhibit xanthine oxidase, an enzyme converting xanthine to uric acid [37].

The Cameroonian study [26] revealed a significant association between hyperuricemia with a history of smoking ($r = 0.377$; $P < 0.0001$) and a history of alcohol use ($r = 0.391$; $P < 0.0001$). In the current study, there was no significant association between hyperuricemia with a history of smoking ($p = 0.618$) and a history of alcohol use ($p = 0.162$). The disparity between the two studies might be under-reporting by the hypertensive patients in the current study and the behavioral variations of study participants in Cameroon.

The result of this study indicated that dyslipidemia a known risk factor for high blood pressure and elevated SUA was widespread among these patients (89.5 %). This was revealed by a high prevalence of high total cholesterol (42.3 %), high triglycerides (61.7 %), low HDL (51.6 %), and high LDL (23.8 %). Dyslipidemia, total cholesterol, triglycerides, low HDL, and LDL were significantly associated with hyperuricemia. The study conducted in Nigerian by Emokpae et al. found that although total cholesterol, triglyceride low HDL, and LDL were laid between normal limits of reference ranges, statistically significant differences were observed [25]. Similarly, the Cameroon study [26] observed a significant association between serum uric acid and triglycerides.

A study was conducted to examine the independent relationship between SUA and lipid profiles using The Third National Health and Nutrition Examination Survey (NHANES III) which represents a well-designed population-based study with a large sample size of United States adults. They concluded that serum LDL, triglycerides, and total cholesterol are strongly associated with serum uric acid concentrations, whereas serum HDL cholesterol levels are significantly inversely associated [38]. Triglycerides have been linked to insulin resistance which promotes the incidence of hypertension via renal tubular sodium reabsorption, augmentation of the systematic nervous system reactivity, and activation of the renin-angiotensin system [14].

In the current study, the mean fasting blood glucose (FBG) was 106.5 ± 23.2 and it was significantly correlated with the presence of hyperuricemia ($r = 0.2$, $p = 0.003$). This was in agreement with the study carried out in Korea by Lee et al. They found that the mean fasting blood sugar was 91.3 ± 17.7 and significantly associated with the presence of hyperuricemia, $p < 0.001$ [39]. Hyperuricemia is strongly associated with insulin resistance and abnormal glucose metabolism [32,39,40].

The median eGFR of the current study was 90.5 (IQR: 70.5, 109.0) ml/min per 1.73 m^2. Low levels of eGFR were significantly associated with the presence of hyperuricemia, 0.30 (0.15–0.59), $p < 0.0001$. A similar study conducted in Nigeria by Emokpae et al. obtained a statistically significant difference when creatinine and urea of the patients with elevated serum uric acid were compared with the patients whose serum uric acid was within the normal reference range, $p < 0.005$ and $p < 0.001$ respectively [25]. A study conducted in Cameroon by Nguedia et al. found that there was a higher level of creatinine in the patients with elevated uric acid but not statistically significant [26]. In the UK a study conducted on treated hypertensive found a linear decrease in estimated GFR with the increasing quartiles of SUA in both women and men. Participants in the highest quartile of serum uric acid showed a 10.7 (95 % CI: 13.6–7.9) ml/min/1.73 m^2 and 12.2 (95 % CI: 15.2–9.2) ml/min/1.73 m^2 decrease in estimated GFR in men and women, respectively, compared with the lowest quartile of serum uric acid [36]. A study carried out in Taiwan by Lin et al. found that SUA values were significantly correlated with 4 quintiles of serum creatinine level ($p < 0.0001$) independent of diuretic usage [18]. SUA increases as the eGFR falls, with around half of the individuals becoming hyperuricemic by the time dialysis is initiated [41].

4.3.1. Limitation of the study

The main limitation of this study was the cross-sectional nature of its design, which is difficult to establish a cause-effect relationship between hyperuricemia and associated factors as it is a temporal association. In addition, the sample size in the current study was relatively small, therefore our findings may not represent for the entire population. Finally, the information on some associated variables such as drinking habit and smoking was prone to underreporting or lacking. Given those limitations, further longitudinal cohort studies are needed to observe and analyze the causative relationship of hyperuricemia and associated factors among hypertensive patients.

4.3.2. Practical implications of the study

The study titled "Hyperuricemia and Associated Factors among Hypertensive Patients Attending the University of Gondar Comprehensive Specialized Hospital, Northwest Ethiopia" has several practical implications:

Clinical practice: The study's findings can help healthcare professionals better understand the factors associated with hyperuricemia in hypertensive patients. This knowledge can inform clinical decision-making and treatment strategies for managing hyperuricemia and related conditions, such as gout and kidney disease.

Public health: The study's findings can help public health officials and policymakers develop targeted interventions to reduce the prevalence of hyperuricemia and related conditions in hypertensive patients. This may involve promoting healthy lifestyles, such as regular physical activity and a balanced diet, as well as improving access to healthcare services and medications for managing hyperuricemia.

Research: The study's findings can provide valuable insights for future research on the relationship between hyperuricemia, hypertension, and other health conditions. This knowledge can help researchers identify potential targets for drug development and other interventions to improve patient outcomes.

Patient education: The study's findings can help healthcare professionals educate hypertensive patients about the importance of managing hyperuricemia and related conditions. This may involve providing patients with information about lifestyle changes and medications that

can help reduce uric acid levels and prevent complications.

Healthcare system: The study's findings can help healthcare systems identify areas where they may need to improve their management of hyperuricemia and related conditions in hypertensive patients. This may involve investing in additional resources, such as specialized clinics or support services, to help patients better manage their hyperuricemia and related conditions.

In summary, the study's findings have important practical implications for clinical practice, public health, research, patient education, and healthcare systems. By understanding the factors associated with hyperuricemia in hypertensive patients, we can work towards improving patient outcomes and reducing the burden of hyperuricemia and related conditions on individuals and society as a whole.

5. Conclusion

In this study, there was a high prevalence (42.3 %) of hyperuricemia among hypertensive patients attending the University of Gondar Comprehensive and Specialized Hospital. The major independent risk factors after adjusting for covariates were high WC, high BMI, dyslipidemia, and low eGFR (high serum creatinine level). There was a significant positive correlation between hyperuricemia with high total cholesterol, high triglycerides, high LDL cholesterol, fasting blood glucose, increased BMI, and high WC; and there was a significant negative correlation between hyperuricemia and HDL cholesterol. Promoting the determination of serum uric acid levels in hypertensive patients was recommended to minimize the emergence of hyperuricemia.

Ethical consideration

Formal ethical approval was obtained from the School of Medicine Ethical Review Committee, College of Medicine and Health Sciences, University of Gondar. Permission letter to conduct the study was obtained from the University of Gondar Specialized Referral Hospital Chief Executive Officer. Written informed consent was obtained from the hypertensive patients/participants. To ensure the confidentiality of data, study subjects were identified using codes, and only authorized persons accessed the collected data. An abnormal result of the study participants was communicated to the physicians or nurses who are working at the chronic center for proper patient care.

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CRediT authorship contribution statement

Oman Philmon Daka: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Tesfahun Bekele Jember:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Methodology, Investigation. **Kibur Hunie Tesfa:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare no conflict of interest.

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