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Hyperuricemia and associated factors: The case of outpatients at the Bafoussam Regional Hospital- Cameroon, an analytical cross-sectional study

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Abstract

Background and Aims: Hyperuricemia constitutes a major public health issue due to its implication in many chronic diseases and metabolic syndromes. We propose to study the prevalence and associated factors of hyperuricemia to diagnose asymptomatic patients and make prognoses on the state of health of the patients. **Methods:** An analytic cross-sectional study has been carried out at the Bafoussam Regional Hospital and the Biochemistry laboratory of the Université des Montagnes over 2 months. Sociodemographic and anthropometric characteristic was obtained; a blood sample was collected from the chosen patients and a biochemical test (uric acid, creatinine, urea, total cholesterol, high density lipoproteins cholesterol, triglyceride) was analyzed by spectrophotometric method. Statistical tests were carried out using SPSS statistical software. Logistic regression analyses identified factors associated with variables of interest. The significance was measured by a *p* < 0.05 with a confidential level of 95%.

Results: The patient population was made up of 100 patients. The sex ratio was 1.22 in favor of men. The prevalence of hyperuricemia in our study was 28.0% with 31.1% in women and 27.3% in men. The mean average of uric acid in the hyperuricemia population was $7.50 \pm 1.24 \text{ mg/L}$ and the normal uricemia population was $4.69 \pm 1.49 \text{ mg/L}$ (p < 0.0001). The mean average triglyceride in the hyperuricemia population was 143 ± 14 and $117.55 \pm 55.52 \text{ mg/dL}$ in normal uricemia with p = 0.046. Age range [35–45] and hypertriglyceridemia have been associated with hyperuricemia with respectively (odds ratio [OR] = 4.07, p < 0.015) confidence interval, CI: [0.89: 97.0]) and ([OR = 2.50, p < 0.046] CI: [1.01: 6.09]).

Conclusion: The prevalence of hyperuricemia was relatively high and has been associated with metabolic disorders in the population. It is necessary to focus on early diagnoses, treatment, and early intervention in view to prevent chronic diseases associated with hyperuricemia.

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1 | INTRODUCTION

Uric acid is the end product of metabolic degradation of purine nucleotide.¹ It is in the soluble form of urate in the plasma and is excreted largely by the kidneys. Its concentration may be increased in certain situations. Hyperuricemia can therefore be defined as serum uric acid (SUA) concentrations greater than 7.0 mg/dL in men, and greater than 5.6 mg/dL in women.² The causes of hyperuricemia listed can be classified into two functional types: on the one hand increased production of uric acid and on the other hand the reduction of its excretion. Common causes of increased production include a diet rich in purine and increased purine metabolism. However, the causes of the decrease in its excretion include renal disorders, certain drugs, and competition for excretion between uric acid and other molecules.^{3,4} Other causes include high levels of alcohol and starvation.⁵ Prevalence of hyperuricemia has been reported by several studies including the study of Liu et al. in China which report a prevalence of 34.05%.⁶ O et al. in Nigeria report a prevalence of 35.8% in the male population studied.⁷ Kamdem et al. in Douala-Cameroon reported 31.8% hyperuricemia in their study population.⁸ However, hyperuricemia remains asymptomatic in several situations. Asymptomatic hyperuricemia indicates individuals with high SUA who have not been diagnosed with gout or nephrolithiasis in the past; It is then considered a nonpathological state while its prevalence is estimated between 10% and 20% worldwide.⁹ Hyperuricemia has gradually become an important worldwide issue because, in this condition, there is a high risk of crystallization of urate into monosodium urate and the crystals form usually deposits on cells, joints causing silent tissue damage and inflammations leading to gout and kidney stones if not treated.¹⁰ It has been reported that high SUA would increase the risk of onset and progression of chronic renal failure, end-stage renal disease,¹¹ the occurrence of cardiovascular events, and in extreme cases death.^{2,12} People with SUA elevation are found to have an increased risk of readmission for heart failure causing extended hospital stays.⁵ Hyperuricemia is associated with obesity, hypertension, diabetes, dyslipidemia, metabolic syndrome, liver dysfunction, cardiovascular disease, and chronic kidney disease as reported by several authors even if the mechanisms remain to be elucidated.¹³⁻¹⁹ Given the appearance of cardiovascular and renal comorbidities after long exposure to hyperuricemia, it is difficult to know whether hyperuricemia is a risk mark for nongouty disease or factors associated with its.²

Hyperuricemia constitutes a major public health issue due to its implication in many chronic diseases and metabolic syndromes. Several authors have reported his involvement in the development of several metabolic diseases as raised above.^{5,7,20} In Africa in general and Cameroon in particular, patients are victims of cardiovascular and renal diseases caused by hyperuricemia due to its asymptomatic presence in some cases and the negligence of the population in other

cases; thus creating silent tissue damage leading to gout and kidney stones etc and the consequence of which in extreme cases is death if adequate and timely management is not done. Given this problem, we propose to study the prevalence and associated factors of hyperuricemia in outpatients at the Bafoussam Regional Hospital (BRH) to diagnose asymptomatic patients and make prognoses on the state of health of the patients.

2 | MATERIALS AND METHODS

2.1 | Study design

We carried out an analytical cross-sectional study for 2 months from August 29 to November 1, 2022, at the BRH. The study population consisted of patients coming to the general consultation service of the regional hospital of Bafoussam. Sampling was consecutive and participants whose consent was obtained were included in the study. All patients who have for at least 8 h, and of age ranging from 25 to 45 were included. Patients presenting symptoms of hyperuricemia or any known disease in association with hyperuricemia, those under medication treatment, and pregnant women were not included. Pathologies such as cancers and others that do not affect uricemia were determined and/or identified by the interviewees, and these pathologies have been confirmed with their doctors. A questionnaire was developed to collect the sociodemographic, anthropometric (height, weight, body mass index), and bio-clinical information of participants.

2.2 | Biological analyzes

A volume of 3-4 mL of blood was collected in dry tubes in participants enrolled in the study. After blood collection, the samples were transported in the cooler containing ice packs directly to the Biochemistry laboratory of the Université des Montagnes accompanied by the completed technical questionnaire.

2.3 | Biochemical analyzes

The determination of SUA concentration was done according to the spectrophotometric method using the Beer–Lambert principle which measures the concentration of Uric acid in colored solution; it was made according to the "Human laboratory kit." The evaluation of renal function was achieved by determination of urea and determination of creatininemia by spectrophotometric method according to SGM Italia laboratory kit. The glomerular filtration flow rate was deduced from the modification of the diet in renal disease (MDRD)

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equation.²¹ Moreover, uremia has been determined; and lipid profile was evaluated by total cholesterol, triglyceride, high density lipoproteins (HDL) cholesterol, and low density lipoproteins (LDL) cholesterol by spectrophotometric method following the assay methods and protocol of the following manufacturers SGM Italia laboratory kit, Biolabo kit, and Inmesco laboratory kit.

2.4 Data management and statistical analysis

Hyperuricemia was defined for each participant after analysis on two different blood samples, and for threshold uric acid concentrations of 3.4–7 mg/dL in men and 2.4–5.6 mg/dL in women as reported by the human laboratory kit and as highlighted in the literature. The interpretation of the uremia was made using the reference values of 10–50 mg/dL defined by the manufacturer's kit used. Creatininemia was interpreted using the following reference values: 9–13 mg/dL in men and 6–11 mg/dL in women. The glomerular filtration flow rate was deduced from the MDRD equation. Total cholesterol was interpreted from the cut-off value \leq 200 mg/dL; for triglycerides 60–165 mg/dL in men and 40–140 mg/dL in women. HDL and LDL cholesterols are also interpreted according to the values defined by the manufacturer.

The data were collected and saved in an Excel 2016 spreadsheet and statistical analyses were performed using SPSS software Version 26. The variables studied were sociodemographic, clinical, and anthropometric characteristics: (height, weight, body mass index), age, sex, habits (alcohol, tobacco, protein intake), blood pressure; biological variables: uricemia, creatininemia, total cholesterol, HDL cholesterol, triglyceride, urea. Each of the biochemical parameters studied has been dichotomized and grouped into "normal, high, or low" according to the reference values and cutoff defined by the World Health Organization, the manufacturer of each reagent used, and the scientific literature. The variables were presented in frequency for qualitative and mean ± standard deviation (SD) for quantitative variables. The χ^2 test and/or the Fisher exact test made it possible to compare the proportions between different groups. The comparison of the mean ± SD between the groups was made from the student test. Univariate logistic regression analyses were used to identify factors associated with metabolic complications in our study population. Significance was measured by a p < 0.05 and a 95% confidence level for all tests.

2.5 | Ethical considerations

This study has been validated and approved by the Institutional Committee of Ethics and Research of the *Université des Montagnes* (AUTHORIZATION N°. 2022/149/UdM/PR/CEAQ). The Regional Hospital of Bafoussam has issued the authorization for the collection and analysis of samples for the eligible patients of the studies

TABLE 1	Sociodemographic,	lifestyle, and	bio-clinical
characteristic	s of the population.		

.	Female N = 45,	Male	Total
Parameters	n (%)	N = 55, n (%)	N = 100, n (%)
Region	0 (0 00)	1 (1 00)	1 (10.00)
Center	0 (0.00)	1 (1.89)	1 (10.00)
Northwest	4 (8.89)	0 (0.0)	4 (4.00)
West	41 (91.11)	54 (98.18)	95 (95.00)
Family history backgrou		= (10 = 20)	
Gout	3 (6.67)	7 (12.73)	10 (10)
Hypertension	10 (22.22)	18 (32.23)	28 (28.00)
Kidney diseases	2 (4.44)	3 (5.45)	5 (5.00)
Cancer	3 (6.67)	10 (18.18)	13 (13)
None	27 (60)	17 (30.9)	44 (44)
Body mass index			
Normal corpulence	9 (20)	19 (34.55)	28 (28.00)
Overweight	13 (28.89)	17 (30.91)	30 (30.00)
Obese ≥ 30	23 (51.11)	19 (34.55)	42 (42.00)
BP			
Normal BP	25 (55.56)	18 (32.73)	43 (43)
Prehypertension	15 (33.33)	28 (50.91)	43 (43)
Hypertension	5 (11.11)	9 (16.36)	14 (14)
Eating habits			
Food rich in fat	45 (100)	55 (100)	100 (100.00)
Food rich in glucose	45 (100)	55 (100)	100 (100.00)
Food rich in protein	45 (100)	55 (100)	100 (100.00)
Meat			
Meat	44 (97.78)	54 (98.18)	98 (98.00)
1–2 meat per week	21 (46.67)	19 (34.55)	40 (40.00)
2-3 meat per week	6 (13.33)	8 (14.55)	14 (14.00)
3-4 meat per week	11 (24.44)	11 (20)	22 (22.00)
Everyday	5 (11.11)	14 (25.45)	19 (19.00)
Drinks: alcohol and wat	er		
Alcohol	31 (68.89)	51 (92.73)	82 (82.00)
Sufficient water	21 (46.67)	27 (49.09)	48 (48.00)
Others			
Cigarette	4 (8.89)	7 (12.73)	11 (11.00)
Notion on uric acid	18 (40.00)	32 (58.18)	50 (50.00)
Notion on gout	40 (88.89)	47 (85.45)	87 (87.00)

Note: Sufficient water (1.5-3 L daily).

Abbreviation: BP, blood pressure.

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(N° 840/L/MINSANTE/SG/DRSPO/HRB/D). Each of the participants received an information leaflet providing information on the objectives, benefits, and risks related to the study. For eligible participants, one signature and consent were obtained per signature. The results obtained after the research were kept confidential.

3 | RESULTS

3.1 | Sociodemographic, lifestyle, and bioclinical characteristics of the population

Demographic data collected from n = 100 participants indicated that the majority 55 (55%) were male and a minority for female 45 (45%) for a sex ratio of 1.22. The mean ± SD age was 35.17 ± 6.93 with extrema of 25 and 45 years. Table 1 describes the sociodemographic, bioclinical, and lifestyle characteristics of the population.

From the table, the majority of individuals were from the western region (95%) followed by the northwest region (4%). 28% of our population had a medical family background of hypertension, and 13% for cancer. The population had a diversified diet characterized mainly by a diet rich in fat, glucose, and protein. 98% of the population regularly consumed meat, 82% alcohol, and 48% water. 42% of the total population are obese and 25% are overweight. 49.5% of our population have prehypertension over 19.72% have hypertension.

3.2 | Prevalence of hyperuricemia and study population by biological parameters

3.2.1 | Distribution of hyperuricemia according to sex

The prevalence of hyperuricemia was 28%. Table 2 presents the distribution of hyperuricemia between different sexes.

From the table, the frequency of hyperuricemia in women is 31.1% and in men 27.3%.

3.2.2 | Biological parameters in the population

In Table 3, for renal profiles, 42.86% of patients with hyperuricemia presented low clearance creatinine against 41.67% for patients with normal uricemia. For lipid check, 37.71% of patients with hyperuricemia presented hypertriglyceridemia against 13.89% for patients with normal uricemia (p = 0.046).

In Table 4, the mean ± SD of uric acid in the hyperuricemia population was $7.50 \pm 1.24 \text{ mg/L}$ and normal uricemia population was $4.69 \pm 1.49 \text{ mg/L}$ (p < 0.0001); the means ± SD of urea in the hyperuricemia population was $130.15 \pm 109.50 \text{ mg/L}$ and normal uricemia was $88.91 \pm 49 \text{ mg/L}$ (p = 0.089). The means ± SD of triglyceride in the hyperuricemia population was 143 ± 14 and $117.55 \pm 55.52 \text{ mg/dL}$ in normal uricemia with p = 0.046.

3.2.3 | Anthropometric and clinical parameters according to uricemia

Table 5 shows the distribution of anthropometric and clinical parameters according to the uricemia population; in clinical parameters, 60.71% of patients with hyperuricemia were obese over 34.72% with normal uricemia, finally, 57.27% of patients with hyperuricemia had hypertension over 42.73%.

3.3 | Factors associated with hyperuricemia

Table 6 shows the factors associated with hyperuricemia. Age range [35–45] and hypertriglyceridemia have been associated with hyperuricemia with, respectively ([odds ratio [OR] = 4.07, p = 0.015; confidence interval [CI] [0.89: 9.70]) and ([OR = 2.50, p = 0.046] CI: [1.01: 6.09]).

4 | DISCUSSION

This study aimed to determine the prevalence of hyperuricemia in outpatients at the BRH and to identify the factors associated with it because as reported by authors, it implies many chronic diseases and metabolic syndromes.

The sex ratio was 1.22 in favor of men. The explanation could be that during the period of study, more men came for a health check routine. The study population was relatively young with a mean age of 35.17 ± 6.93 years. The population had a diversified diet characterized mainly by a diet rich in fat, carbohydrate, and protein. 98% of the population regularly consumed meat, 82% alcohol, and 48% water. This diversity can be due to the mechanization of agriculture in rural areas and the increase which would be the main source of energy expenditure in rural areas.²² Anthropometric parameter reveals that 42% of the population was obese. Among other things, adiposity is a risk factor for cardiovascular diseases, cancer, and type 2 diabetes mellitus.^{17,22} The frequencies of

Parameter	Female, <i>n</i> (%)	Male, n (%)	Total, <i>n</i> (%)	p Value
Normal uricemia	33 (68.9)	39 (72.7)	72 (72.00)	0.17
Hyperuricemia	12 (31.1)	16 (27.3)	28 (28.00)	

TABLE 2 Distribution of hyperuricemia according to sex.

Note: χ^2 test.

Parameters	Normal uricemia, N = 72	Hyperuricemia, N = 28	Total, <i>N</i> = 100	p Value
Uric acid				
Low	7 (8.33)	0 (0.00)	6 (6.00)	<0.0001*
High	0 (0.00)	28 (100)	28 (28.00)	
Normal	65 (90.28)	0 (0.00)	65 (65.00)	
Creatinine clearance	e			
Low	30 (41.67)	12 (42.86)	42 (42.00)	0.581
High	13 (18.06)	4 (14.29)	17 (17.00)	
Normal	29 (40.28)	12 (42.86)	41 (41.00)	
Urea				
Low	34 (47.22)	8 (28.57)	58 (58.00)	0.089
High	38 (47.22)	20 (71.43)	42 (42.00)	
Cholesterol total				
High	17 (23.61)	9 (32.61)	26 (26.00)	0.382
Low	53 (76.39)	19 (67.86)	74 (74.00)	
Cholesterol total				
High	19 (26.39)	3 (10.71)	22 (78.00)	0.089
Low	53 (73.61)	25 (89.29)	78 (78.00)	
Triglyceride				
Low	5 (6.94)	2 (7.14)	7 (7.00)	0.046*
High	10 (13.89)	10 (37.71)	20 (20.00)	
Normal	57 (79.17)	16 (57.14)	73 (73.00)	

TABLE 3 Distribution of biological test according to normal uricemia and hyperuricemia.

Note: Uric acid (mg/L); creatinine clearance (mL/min); urea (mg/dL); cholesterol total (g/L); HdL cholesterol (mg/dL); triglyceride (mg/dL); χ^2 test. *Significant difference at p < 0.05.

TABLE 4	mean average o	f biological pa	arameters in	population.
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Parameters	Normal uricemia, N = 72	Hyperuricemia, N = 28	Total, <i>N</i> = 100	p Value
Uric acid	4.69 ± 1.49	7.50 ± 1.24	5.47 ± 1.91	<0.0001*
Clearance creatinine	120.91 ± 81.88	147.30 ± 85.4	128.30 ± 119.69	0.581
Urea	88.91±49	130.15 ± 109.50	100.46 ± 73.13	0.089
Total cholesterol	1.73 ± 0.35	1.79 ± 0.41	1.75 ± 0.36	0.382
Hdl cholesterol	49.78 ± 36.28	44.15 ± 23.18	48.21 ± 33.12	0.089
Triglyceride	117.55 ± 55.52	143 ± 14	124.71 ± 67.19	0.046*

Note: Mean ± standard deviation; uric acid (mg/L); creatinine clearance (mL/min); urea (mg/dL); cholesterol total (g/L); HDL cholesterol (mg/dL); triglyceride (mg/dL); student test.

*Significant difference at p < 0.05.

overweight and obesity reported in this study are also higher than those of Adebayo et al.²³ reported in the adult Nigerian rural population (20.9% and 8.4%, respectively) and those reported recently in the Ethiopian study (19.9% and 8.6%, respectively).²⁴ However, the prevalence of overweight and obesity in our study is lower than those reported in previous studies of urban adult

populations in Cameroon and South Africa.^{25,26} In addition, 30% of the population was underweight; this could be due to an increase in food expenditure and the social value of being overweight, which is perceived by "popular imagery" as a sign of well-being; not to mention the traditional energy found in the West, region of Cameroon as indicated above.²²

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TABLE 5 Distribution of anthropometric and clinical parameters according to uricemia in the population.

Parameters	Normal uricemia, N = 72, n (%)	Hyperuricemia, N = 28, n (%)	Total, N = 100, n (%)	p Value
BMI				
Normal corpulence	22 (30.56)	6 (30.56)	28 (28.0)	0.760
Underweight	25 (34.72)	5 (17.86)	30 (25.0)	0.982
Obesity	25 (34.72)	17 (60.71)	42 (42.0)	0.055
BP				
Normal BP	30 (27.75)	19 (22.15)	49 (49.5)	0.697
Prehypertension	8 (20.58)	21 (79.42)	39 (30.58)	0.059
Hypertension	3 (42.73)	8 (57.27)	11 (19.72)	0.956

Note: Normal BP (<120/85 mmHg); prehypertension (N \ge 120/85 mmHg); hypertension (\ge 140/85 mmHg); normal corpulence (\ge 18.5, <25); underweight (\ge 25, <30); obesity (\ge 30); χ^2 test.

Abbreviations: BMI, body mass index; BP, blood pressure.

TABLE 6 Fa	actors associated	with h	nyperuricemia.
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	Hyperuricemia			
Parameters	Yes, n = 28 (%)	No, <i>n</i> = 72 (%)	OR (CI 95%)	p Value
Age (35-45)	9 (31.03)	12 (16.9)	4.07 (0.89; 9.7)	0.015*
Hypertension	8 (57.27)	3 (42.73)	1.81 (0.1; 5.41)	0.288
Alcohol	24 (85.71)	58 (80.56)	5.01 (23; 10.40)	0.305
Meat	13 (46.43)	63 (87.5)	9.48 (0.9; 10.0)	0.982
0.5 L water	6 (21.43)	30 (46.67)	1.03 (0.5; 4.80)	0.982
Obesity	17 (60.71)	25 (34.72)	2.49 (0.84; 7.44)	0.101
Hypertriglyceridemia	10 (37.71)	10 (13.89)	2.50 (1.01; 6.09)	0.046*

Note: Univariate analysis.

Abbreviations: CI, confidence interval; OR, odds ratio.

*Significant difference at p < 0.05.

The determination of uricemia was done by colorimetric measure with a spectrophotometer. At the end of this, the prevalence of hyperuricemia was 28%. This reflects the high frequency of hyperuricemia in the Cameroonian population in general and the West, in particular, came in routine consultation and lack of any treatment. This high prevalence would reflect hypermetabolism or a renal excretion deficit; this can be explained by the fact that this study population was cosmopolitan and had a diversified diet rich in purine and lipids obtained from the questionnaire or maybe because a patient coming to the hospital may have an unknown metabolic disorder or pathologies that could increase their SUAs count.²⁷ As described in the literature, the causes of hyperuricemia include a large intake of purine through diet and increased purine metabolism.^{3,4} Studies have estimated the prevalence of hyperuricemia obtained by Liu et al. in China (34.05%),⁶ Suraamornkul et al. in the Bangkok population (24.4%),²⁸ O et al. (35.8%),⁷ Kamdem et al. in Douala-Cameroon (31.8%).⁸ These reported prevalences are close to ours with some differences. These differences could be explained by the diversity and differences that may exist between the different study populations.

Clinical and biochemical parameters of uricemia were evaluated in population, blood pressure, lipid profile, and renal function. This evaluation permitted to identifier that, 42.86% of patients with hyperuricemia presented low clearance creatinine against 41.67% for patients with normal uricemia, and 71.43% of patients with hyperuricemia presented high serum urea against 47.22% for patients with normal uricemia (p = 0.089), this result demonstrates that renal dysfunction can lead to accumulation of uric acid.^{29,30} Indeed, hyperuricemia is common in patients with chronic renal failure. Serum UA level could therefore be considered as a marker of kidney disease whose increase is mainly observed due to its altered excretion. These observations were also described by Kaze et al. who report hyperuricemia as a predictor of renal disease ([3.12 (1.58–6.16), p = 0.002]).³¹

After evaluation of the lipid profile, it appears that dyslipidemia was present in the population with hyperuricemia. Indeed, 32.61% of patients with hyperuricemia presented total hypercholesterolemia against 23.61% for patients with normal uricemia, 37.71% of patients with hyperuricemia presented hypertriglyceridemia against 13.89% for patients with normal uricemia (p = 0.046), 84.29% of patients with hyperuricemia presented low HDL cholesterol against 73.61% for patients with normal uricemia. Indeed, several authors report a significant association between hyperuricemia and lipid profile. They suggest that hyperuricemia may promote metabolic alterations, resulting in postprandial increases in triglyceride levels, a buildup of triglycerides in the liver tissue, as well as disturbances in the insulin response in the liver, adipose tissue, and muscular. This justifies the association between asymptomatic hyperuricemia and lipid profile indicators found in our study. They also suggest that hyperuricemia results in disruptions of lipid homeostasis; in this case, uric acid levels can be used as a biomarker for predicting the future incidence of dyslipidemia in a healthy person.⁹

Regarding clinical parameters, 60.71% of patients with hyperuricemia were obese over 34.72% with normal uricemia, finally, 79.42% of patients with hyperuricemia had prehypertension over 20.58% (p = 0.059). Authors have also reported an association of hyperuricemia with arterial hypertension; In particular, they report the role of hyperuricemia in the pathogenesis of hypertension. This is notably the case of Yu et al. in China who reported 67.6% of hypertensive in its patient population with hyperuricemia.³² SUA has been reported to be correlated with an increased risk of hypertension, diabetes, kidney failure, obesity, and metabolic syndrome. The potential mechanisms include nitric oxide inhibition, vascular smooth muscle cell proliferation, activation of the angiotensin-renin system, endothelial cell dysfunction, inflammation, and induction of oxidative stress.^{10,27,29,32}

The secondary aim of this study was to identify factors associated with hyperuricemia in this population. Results for associated factors show a significant association between Uric acid and age ranging from 35 to 45 ([OR = 4.07, p = 0.015] CI: [0.89: 9.70]), from our result, we may say that as age increases uric acid levels also increased; As reported by previous studies, the age was confirmed as an independent risk factor for hyperuricemia¹²; also reported by Hague et al.³³ This can be explained by an increase in cells and nutrition metabolism.⁴ The increase in SUA levels with age may be the result of normal purine metabolism, but a reduction in the excretion of its byproduct; kidney function therefore deteriorates with age.²⁷ Our finding is in agreement with some studies showing age as a risk factor for high blood uric acid levels.^{12,34} Hypertriglyceridemia was also positively and significantly associated with hyperuricemia in the study ([OR = 2.50, p = 0.046] CI: [1.01: 6.09]). Indeed, as described, the increase in free fatty acids (obesity) results from the increase in triglycerides resulting in the production of adenosine triphosphate which leads to an increase in uric acid, being the end product of metabolism.²⁷ This result is consistent with the results of Liu et al. in Guangdong province of China.⁶ Alcohol

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([OR = 5,01, p < 0.304] CI: [0.23: 19.41]) was also associated with hyperuricemia, this can be explained by the fact that beer is highly rich in purines especially guanosine which favors uric acid production. It has been reported that alcohol is the most important cause of hyperuricemia.³⁵ This observation was also reported by O et al. in Abakaliki in Nigeria.⁷ Finally, hypertension ([OR = 1.87, p < 0.288] [0.34: 37.55]) was positively associated with hyperuricemia. This observation was also reported by O et al. in Abakaliki Nigeria, and Liu et al. in Guangdong province of China.⁶

5 | CONCLUSION

At the end of our study, the prevalence of hyperuricemia was relatively high (28%) between outpatients at the regional hospital of Bafoussam. Hyperuricemia has been associated with metabolic disorders in the population. Age range [35–45] and hypertriglyceridemia have been associated with hyperuricemia with respectively ([OR = 4.07, p < 0.015] CI: [0.89: 97.0]) and ([OR = 2.50, p < 0.046] CI: [1.01: 6.09]). It is necessary to focus on early diagnoses, treatment, and early intervention in view to prevent chronic diseases associated with hyperuricemia.

AUTHOR CONTRIBUTIONS

Romaric De Manfouo Tuono: Conceptualization; investigation; methodology; supervision; validation; writing-review and editing. Aude Bernie Famessi Samou: Methodology; writing-original draft. Marius Tchoumke Mbiandjeu: Writing-original draft; writing-review and editing. Pascal Blaise A Koul Well A Well: Writing-review and editing. Pascal Dieudonne Djamen Chuisseu: Methodology; supervision; writing-review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data used to support the findings of this study are available from the corresponding author upon request. The authors confirm that the data supporting the findings of this study are available within the article [and/or] its supplementary materials.

TRANSPARENCY STATEMENT

The lead author Romaric De Manfouo Tuono affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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