Research article

Relationship between the level of daily alcohol intake and serum uric acid concentrations in a sample of young men from Cameroon, sub-Saharan Africa

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Abstract

Background: Despite the fact that alcohol is predominantly consumed by young men, there are few studies among this population in Sub-Saharan Africa (SSA) about the relationship of the level of alcohol consumed and Serum Uric Acid values (SUA).

Objective: The study aimed at investigating the relationship between regular alcohol consumption and uricemia in Cameroonian men aged under 45 years.

Methods: This was a cross-sectional study done in Yaoundé (Cameroon), on healthy males aged 18 to 45 years with regular alcohol consumption within the last three months. We assessed their Ethylic Index (EI), purine-rich diet, SUA and hepatic and haematological biomarkers of chronic alcohol consumption. We compared their SUA according to the level of alcohol consumption (low <20, moderate 20-39.9 and elevated ≥40g/L) using the ANOVA test, and search for other associated factors through multivariate linear regression. The threshold of significance was 0.05.

Results: One hundred and fifty seven participants were included: mean age 30.6 (5.8) years, median duration of alcohol consumption 10 [7; 15] years and median EI 24.1 [14.4; 37.1] g/day. Beer consumption was predominant (94.9%); moderate and elevated consumption found in 36.3 and 23.3% of participants respectively. Hyperuricemia was found in 38 (24.2%) participants, associated with diet (p=0.04) and diastolic blood pressure (p=0.04). No difference in SUA between alcohol consumption groups (p=0.26). However, SAU were higher in participants with elevated Gamma Glutamyl-Transferase (GGT) (0.018), and GGT were found to be biomarkers of alcohol consumption (p<0.001).

Conclusions: EI does not seem to reflect the link between alcohol and SUA in young men. However, the association of SUA with GGT represents a relevant biological finding, linking chronic alcohol consumption to uricemia in this population.

Key words: Alcohol, Cameroonians, Uric acid, Young men

Introduction

Alcoholic beverages are among the most popular drinks in the world and chronic alcohol use disorders affect more than 237 million men worldwide, with more than 3 million deaths, particularly among those aged between 15 and 49 years[1,2]. Increasingly, Africa is confronted with the growing burden of harmful alcohol consumption and its disastrous effects[3]. Cameroon is no exception, with high consumption among young people, as found by Ntone et *al*.[4] in 2017, in a student population with a prevalence of 87.93%. Harmful alcohol consumption can be the cause of numerous hepatic, digestive and neurological complications, as well as a number of biological disturbances, including uric acid metabolism disorders, of which hyperuricemia is the main cause of morbidity[5,6].

Hyperuricemia is defined by uric acid level in the blood exceeding 70mg/l in men. It is implicated in the pathogenesis of numerous chronic conditions, including gout (the most common), hypertension and cardiovascular disease[7]. Hyperuricemia results from a constellation of factors, among which genetics and lifestyle are the most important determinants of causality. Diet in general, and alcohol consumption in particular, are risk factors for the onset of hyperuricemia in men, who generally experience their first gout attack from the age of 40-45 years, and cardiovascular complications later in life[8-10]. To prevent these metabolic and vascular consequences, it is essential to start early in life, and therefore to control the impact of lifestyle, particularly dietary factors.

Despite the fact that alcohol is predominantly consumed by young men, there remain few studies among this population, particularly in sub-Saharan Africa (SSA), reporting the impact of the level of alcohol consumed on Serum Uric Acid values (SUA). In order to improve primary prevention strategies for hyperuricemia and gout, as well as awareness of the metabolic consequences of chronic alcohol consumption, we proposed to carry out this study, aimed at investigating the relationship between regular alcohol consumption and uricemia in Cameroonian men aged under 45 years.

Materials and methods

Study design and setting: This was a cross-sectional study conducted from October 2023 to February 2024 at the National Obesity Center located at the Yaoundé Central Hospital and at the Biochemistry Laboratory of the Yaoundé University Hospital Centre (Cameroon).

Participants: We included Cameroonian males aged 18 to 45 years with regular and stable alcohol consumption who had consented to participate in the study. Regular drinking was defined as drinking at least once a week for at least one month. Stable drinking, assessed qualitatively with the participant, was defined as drinking the same beverage, at the same frequency and in the same quantity, over the past three months. We excluded any participant receiving hyper- or hypouricemic medication, as well as any participant with any chronic disease, such as impaired kidney function or inflammatory disease.

Sample size estimation: The sample size was estimated using the formula adapted to our type of study contained in the manual by Whitley and Ball[11]. Based on the prevalence of hyperuricemia of 19.3% in a group of men aged 25 to 44 years with an average alcohol consumption of 59 ml/day in a population of predominantly African origin in the Seychelles[12]. We obtained a sample size of 121 participants, considering a power of 95% and an error rate of 0.07.

Data collection

After obtaining administrative authorizations and ethical clearance, each potential participant was invited to the study by announcements in the general population residing in Yaoundé, the country's capital. For all included participants, we collected sociodemographic, clinical and biological data. Sociodemographic data included age, profession and level of education. Clinical data included:

Alcohol consumption: Duration (since first consumption), type of alcohol consumed (beer, wine, whisky) and Ethylic Index (EI). EI was calculated in ethanol equivalence using the following formula: EI (g/day) = (average daily amount of alcoholic beverage consumed (ml) * degree of alcohol of beverage consumed * 0.8)/100. 0.8 representing the density of ethanol. Participants were stratified according to EI into: low consumption (<20g/day), moderate consumption (between 20 and 39.9g/day) and elevated consumption (at least 40g/day).

Tobacco consumption: Active or discontinued smoking, with duration since first use and smoking index specified. The smoking index (in pack-years) was evaluated by multiplying the average number of cigarette packs consumed per day by the duration of smoking in years.

Lifestyle: Sedentary lifestyle and physical activity. Physical activity was stratified by duration per week into: less than 30 minutes, 30 to 60 minutes, 60 to 90 minutes, 90 to 120 minutes, 120 to 150 minutes, and more than 150 minutes. Sedentary lifestyle was defined as physical activity of less than 30 minutes three times a week.

Dietary survey: Focusing on the purine-rich diet. In the absence of validated tools in the Cameroonian population, we carried out an indirect, semi-quantitative assessment of the purine richness of the diet for each participant using a frequency-based dietary questionnaire, which we developed following the recommendations of Cade *et al*[13]. Purine-rich foods were selected with the help of a nutritionist, based on data from the African literature, with preference given to foods consumed in Cameroon[14]. Consumption frequencies were assessed on a daily, weekly and monthly basis, and weighted from 0 to 8, in order to perform a comparative analysis of participants' consumption. The total food score obtained was rated out of 64. The food questionnaire is contained in supplementary material 1. As this was a semi-quantitative questionnaire, we considered only the frequency of consumption, not the quantity consumed. The standard quantity was taken to be the amount consumed during a normal meal.

*Other clinical data***:** Family history of gout, systolic and diastolic blood pressure (mmHg), weight, height to calculate Body Mass Index (BMI), and abdominal circumference. BMI (kg/m²) was calculated as the ratio of weight (kg) to the square of height (m). It was classified according to WHO as normal BMI (18 to 24.9), overweight (25 to 29.9), obese grade 1 (30 to 34.9), 2 (35 to 39.9), and 3 (at least 40). Abdominal obesity was defined as an abdominal circumference of at least 94 cm according to the recommendations of the International Diabetes Foundation[15].

Table 1: Food frequency questionnaire for purine rich diet

Foods	Frequency (quotation)								
	Never or less than one time/ month (0)	1-3 times / month (1)	One time/ week (2)	2-4 times/ week (3)	5-6 times/ week (4)	One time a day (5)	2-3 times / day (6)	4-5 times/ day (7)	6 times per day (8)
Meat									
Giblets: liver, kidneys, tripe, heart									
Beef, bushmeat, game meat									
Charcuterie, dried, smoked or cured meats									
Meat or chicken broth									
Fish									
Sardines, fish roe									
Maquereau (Scomber), carp, fish pike, smoked or dried fish									
Shellfish and seafood									
Fish broth									
Others									
Beans									
Very fermented cheese									
Fructose rich drinks									
Total score /64									

Biological data

Biological data were evaluated on a fasting venous blood sample collected without any tourniquet. They were:

(i) Fasting blood glucose (8h fasting, method of Trinder) and serum creatinine (modified Jaffé kinetic and colorimetric method), to exclude subjects with probable diabetes (glycemia ≥1.26g/L), and those with a reduced Glomerular Filtration Rate (GFR) of less than 60ml/min. GFR was assessed according to the 4-parameter MDRD formula[16].

(ii) Biological markers of chronic alcohol consumption at the level of the liver (Alanine Aminotransferase (ALAT), Aspartate Aminotransferase (ASAT), and Gamma Glutamyltransferase (GGT)), and at the haematological level (haemoglobin level, Mean Corpuscular Volume (MCV)). Liver enzyme activities were measured according to the recommendations of the International Federation of Clinical Chemistry and Laboratory Medicine[17-19]. We considered a rise in ALAT or ASAT values to be from 40 IU/L, and GGT from 49 IU/L. The haemogram was performed using the Cyanhemato® automated system. MCV was considered normal between 80 and 100fL. Macrocytosis and microcytosis were defined as values below 80fL and above 100fL respectively. Anaemia was defined as a haemoglobin level below 13g/dL.

(iii) Serum level of uric acid (SUA) assessed by the uricase method. Hyperuricemia was defined as a serum uric acid value ≥70mg/L[7]. For all biochemical tests, we used reagents supplied by BIOLABO®.

Statistical analysis: The data collected were analyzed using SPSS software version 23.0. Graphics were designed using the same software. In the results, we present categorical variables with their count and percentages, continuous quantitative variables with their means and standard deviation, and quantitative variables not following a normal distribution with median and interquartile range [q25; q75]. Comparison of means between two or more (>2) groups was performed using Student's t-test and one-way ANOVA, respectively. The association between continuous variables was investigated using multivariate linear regression, assorted

with its regression coefficient (β). For all tests used, the significance threshold was 0.05.

Results

We assessed 210 people and finally included 157 in the study. Their ages ranged from 19 to 45 years, with a mean age of 30.6 (5.8) years. They had a median duration of alcohol consumption of 10 [7; 15] years, and a median EI of 24.1 [14.4; 37.1] g/day. Beer was the most consumed alcohol beverage (149; 94.9%). Daily consumption was moderate and high in 57 (36.3%) and 35 (23.3%) participants respectively. Relevant clinical findings included a sedentary lifestyle in 91 (58%), overweight in 53 (35.1%), and obesity in 35 (22.3%) participants. The clinical and paraclinical characteristics of the participants are presented in Table 2.

Table 2: Characteristics of the participants

Variables		Values
Age, mean (SD), years		30.6 (5.8)
EI, median [IQ range], g/day		24.1 [14.4; 37.1]
Type of alcohol beverages, n (%)		
	Beer	149 (94.9)
	Whisky	5 (3.2)
	Wine	3 (1.9)
EI level, n (%)		
	Low (<20 g/day)	65 (41.4)
	Moderate (20 to 39.9 g/day)	57 (36.3)
	Elevated (≥40 g/day)	35 (22.3)
Tobacco consumption, n (%)		21 (13.4%)
Sedentary lifestyle, n (%)		91 (58%)
Physical activity per week, n (%)		
	<30 min	42 (26.8)
	30 to 60 min	25 (15.9)
	60 to 90 min	24 (15.3)
	90 to 120 min	23 (14.6)
	120 to 150 min	12 (7.6)
	≥ 150 min	31 (19.7)
Personal history of gout, n (%)		4 (2.5)
Familial history of gout, n (%)		22 (14)
Purine-rich diet score, mean (SD)		15.2 (5.4)
SBP, mean (SD), mmHg		126 (12)
DBP, mean (SD), mmHg		83 (10)
BMI, mean (SD), kg/m²		27 (4.7)
BMI class, n (%)		
	Normal	63 (41.7)
	Overweight	53 (35.1)
	Grade 1 obesity	24 (15.9)
	Grade 2 obesity	10 (6.6)
	Grade 3 obesity	1 (0.7)

Abdominal circumference, mean (SD), cm		90 (13)
Abdominal obesity, n (%)		73 (48)
SUA, mean (SD), mg/L		57.9 (23.5)
ASAT, mean (SD), UI/L		25.5 (8.4)
ALAT, mean (SD), UI/L		24.5 (9.2)
GGT, mean (SD), UI/L		39.5 (26.8)
Haemoglobin level, mean (SD), g/dL		13.7 (5.6)
MCV, mean (SD), fL		81 (4.3)
Biological abnormalities, n (%)		
	Hyperuricemia	38 (24.2)
	Elevated ASAT	7 (4.5)
	Elevated ALAT	12 (7.6)
	Elevated GGT	37 (23.6)
	Anaemia	23 (16)
	Microcytosis	55 (38.5)
	Macrocytosis	0 (0)

ALAT: Alanine aminotransferase; ASAT: Aspartate aminotransferase; DBP: Diastolic blood pressure; EI: Ethylic index; GGT: Gamma glutamyl-transferase; IQ: interquartile range [Q25; Q75]; MCV: Mean corpuscular volume; SD: Standard deviation; SBP: systolic blood pressure; SUA: Serum uric acid level.

In multivariate linear regression, we found a significant association between serum GGT levels and participants' EI ($p<0.0001$), which was not the case for transaminases (ASAT and ALAT), mean corpuscular volume and haemoglobin levels (Table 3). Hyperuricemia was found in 38 (24.2%) participants. It was significantly associated with high-purine diet ($p=0.04$), and diastolic blood pressure ($p=0.04$) as shown in Table 4. There was no association between the purine-rich diet and alcohol consumption in linear regression ($\beta= -0.01$, $p= 0.8$).

Table 3: Association between ethylic index and biomarkers on linear regression

Variables	Ethylic index	
	β	P value
ASAT	0.12	0.2
ALAT	-0.24	0.06
GGT	0.37	**<0.0001**
Haemoglobin level	0.11	0.2
MCV	0.01	0.9

ALAT: Alanine aminotransferase; ASAT: Aspartate aminotransferase; EI: Ethylic index; GGT: Gamma glutamyl-transferase; MCV: Mean corpuscular volume

Table 4: Factors associated with hyperuricemia

Variables	Hyperuricemia		P-value
	Yes	No	
Age, years	32.3 (6)	30.1 (5.7)	0.06
Purine-rich diet score	16.8 (5.6)	14.7 (5.3)	0.04
Abdominal circumference, cm	93.2 (13.7)	89.5 (13)	0.1
BMI, kg/m²	28.2 (4.9)	26.5 (4.5)	0.05
Systolic blood pressure, mmHg	126 (12)	127 (12)	0.6
Diastolic blood pressure, mmHg	85 (8)	82 (10)	0.04

BMI: Body mass index; DBP: Diastolic blood pressure; SBP: Systolic blood pressure

We found no significant difference comparing serum uric acid levels in the different daily alcohol consumption groups (low, moderate and high) as shown in Figure 1 ($p=0.26$). However, being identified as a biomarker linked to daily alcohol consumption, we found that participants with high GGT also had higher serum uric acid levels ($p=0.018$) as presented in Figure 2.

Figure 1: Comparison of serum uric acid levels between different groups of daily alcohol consumption (p=0.26)

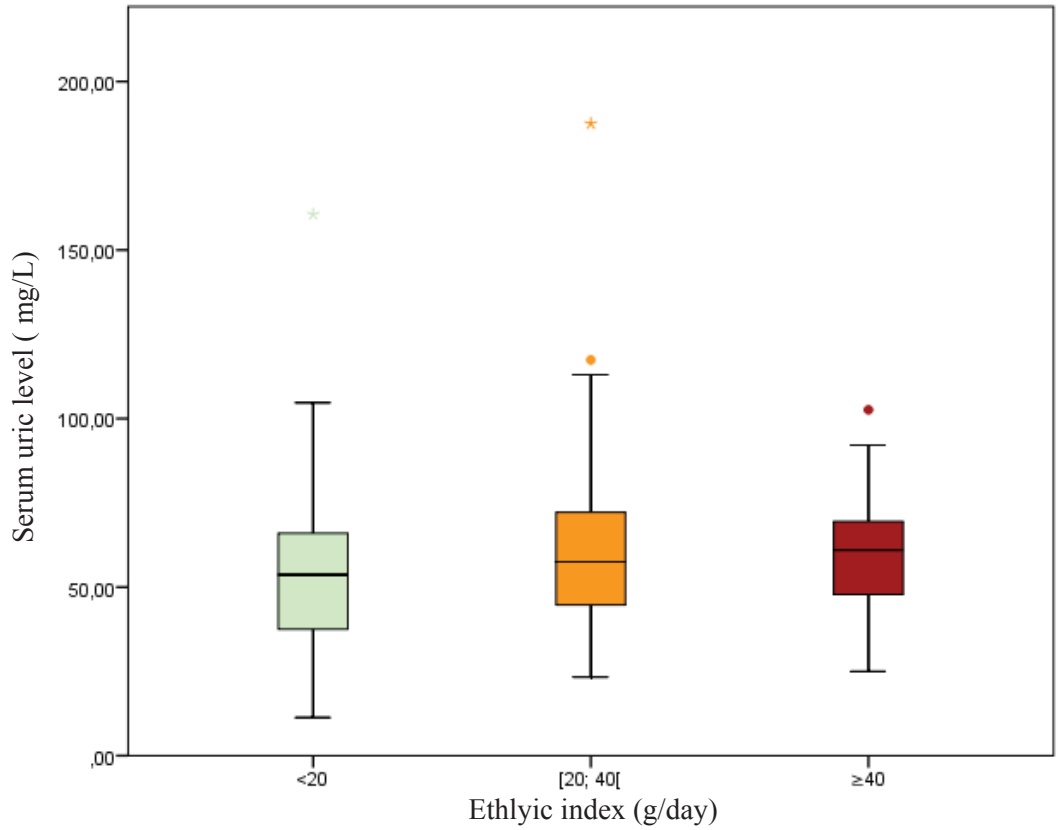
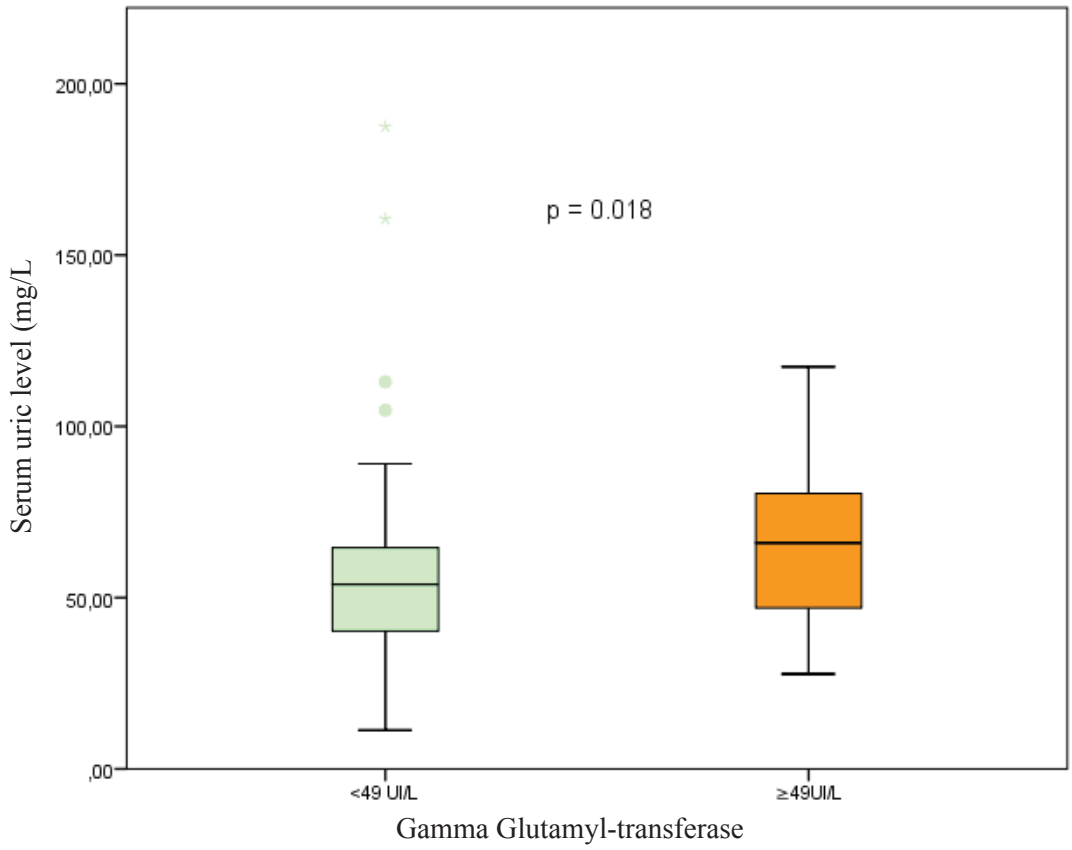


Figure 2: Comparison between serum uric acid level between participants with normal and elevated gamma glutamyl-transferase



Discussion

The contribution of alcohol to the onset of hyperuricemia and gout in adults over 40-45 years of age is undeniable. However, in young people under 45 years of age, even if they are subject to hyperuricemic factors, they also have hypouricemic factors to balance the serum equilibrium. The present study, carried out in a sub-Saharan African context, raised the question of the relevance of limiting alcohol consumption by raising the argument of hyperuricemia at this age, with a view to preventing its consequences later on. Our results contrast sharply with the literature, in that there was no overall difference in uricemia between low, moderate and heavy drinkers; however, it was clear that GGT activity, a simple marker of chronic ethylism, was related to uric acid concentrations. It is essential to contextualize our results in order to make a cautious interpretation of the data in the literature. Our discussion will focus on three key points: hyperuricemia in young male drinkers, uricemia in relation to GGT activity, and strategies to prevent hyperuricemia in young male drinkers.

Serum uric acid level and alcohol

The study found that hyperuricemia accounts for just under a quarter of young people in our population. These findings corroborate those of several authors in the literature who report a higher risk of hyperuricemia with alcohol consumption, with prevalences varying according to population in the order of 12-15% and a risk 1.2 to 2.1 times higher than those not consuming alcohol[20-23]. Compared with the general population, hyperuricemia does not appear to be more frequent in young people, but these studies were carried out on populations with a rather different epidemiological profile from our own[24-26]. Alcohol consumption is itself a source of purine, particularly beer, which is not always included in the purine-rich diet score, but needs to be evaluated[23,27]. It is frequently consumed with a hyperuricemic meal, in individuals with other hyperuricemia risk factors such as overweight/obesity found in our sample, arterial hypertension and medication[28]. In younger subjects without primary gout, metabolic factors play a lesser role, but comorbidities (cancers, inflammatory diseases) and hyperuricemic treatments (which we excluded from our population) make a greater contribution, as do better regulation of renal elimination and relatively greater physical activity/condition[29]. It is therefore vital to reinforce hyperuricemia prevention strategies in this population, as their uric acid values will continue to rise with age, as will their risk of related metabolic and cardiovascular complications.

Serum uric acid level and GGT

The study found that higher GGT levels also resulted in higher uricemia, which was not the case for the Ethylic Index (EI) when comparing participants' uricemia between groups stratified on the basis of EI. In fact, the assessment of alcohol consumption by calculating the EI has a number of limitations, notably the diversity of beverages consumed, which do not always have the same degree of alcohol, and the variability of daily quantities, which biases its interpretation[30]. Although this is an important tool in clinical practice, its limitations suggest that the use of biomarkers would be more reliable. As far as GGTs are concerned, as reported in the literature, they follow the rise in the EI. GGT activity is a marker of chronic alcohol consumption, reflecting the level of alcohol-related hepatic steatosis[31,32]. Its association with serum uric acid levels would therefore reflect an association between uricemia and alcohol consumption. However, it should be borne in mind that this is not necessarily a case of cause and effect, but a sharing of several metabolic factors, in particular the metabolic syndrome, the possibility of non-alcohol-related fatty liver disease or chronic low grade inflammatory disease and oxidative stress[33-35].

Strategies for preventing hyperuricemia in young people

We believe that prevention of hyperuricemia and its consequences should start earlier in life. If we wait until adulthood, then complications will arise. Strategies to prevent this metabolic abnormality in young people must tie in with existing strategies to combat metabolic diseases, and must include reducing alcohol consumption, for which it has been shown that no amount is safe[36]. The hypouricemic diet can easily be combined with the low-sugar, low-fat diet, rich in vegetables and fruit advocated in current practice[37]. Regular physical activity and the management of overweight and obesity, which are closely linked to hyperuricemia, can also be easily integrated. Screening for hyperuricemia should also be incorporated into the assessment of people who drink alcohol regularly, although the threshold for consumption remains to be defined in our context. In the meantime, it would be wise to consider that no consumption of alcohol is without danger, and that they can all contribute, depending on the individual, to raising uric acid levels and exposing him or her to hyperuricemia.

Study limitations

It is crucial to bear in mind that the interpretation of our findings must take into account a number of limitations.

The chosen cross-sectional study design, which cannot be used to assert a cause-and-effect relationship, does lay the foundations for a cohort study that we will implement, and also enables us to better define the key variables for this future work. The sample size was therefore small for this preliminary work. A better assessment of daily consumption is also necessary, and the use of better biomarkers of chronic alcohol consumption such as Carboxy-Deficient Transferrin (CDT) will enable a more precise understanding[30]. A concomitant evaluation of certain confounding biomarkers of GGT activity, notably alkaline phosphatases, markers of lipid metabolism and a morphological study of the liver parenchyma by means of ultrasound, in order above all to detect steatohepatitis, which represents a real public health problem today, and is very frequent in populations that frequently consume alcohol[38]. Finally, the urgent need for validation of a purine-rich diet in our population. In sum, our study gives an idea of the prerequisites for conducting a more detailed, long-term study of the effects of alcohol on uricemia in our population.

Conclusion

Hyperuricemia affects one in five young men who consume alcohol regularly in Cameroon. The study found that serum uric acid level is not related to their ethylic index. However, uricemia rises in the event of an increase in GGT, which is a biological indicator of chronic alcohol consumption. More prospective data are needed to better understand this relationship in our population, and to use these data for public health purposes.

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Declaration

Ethical approval and consent to participate: The study was approved by the Centre Regional Ethics Committee for Human Health Research in Cameroon (N°0721/CRERSHC/2023). All the participants read and signed a written informed consent before their inclusion in the study regarding the Helsinki declaration.

Consent for publication: Not applicable.

Availability of data and materials: All the dataset generated from this study are available from the corresponding author on request.

Competing interest: The authors declare that they have no competing interest.

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