Scientific Research Publishing

Open Journal of Endocrine and Metabolic Diseases, 2025, 15(7), 111-122
https://www.scirp.org/journal/ojemd
ISSN Online: 2165-7432
ISSN Print: 2165-7424

Asymptomatic Hyperuricemia in Type 2 Diabetic Patients in Northern Cameroon: Prevalence and Determinants

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How to cite this paper: Hadja Inna, A.A., Deunga Njandeu, R., Oumarou, M., Ibrahima, A., Tchameni Sidi, C., Hassana, S., Esame Ndive, P., Lombat Ngwet, A.M. and Hamadou, B. (2025) Asymptomatic Hyperuricemia in Type 2 Diabetic Patients in Northern Cameroon: Prevalence and Determinants. *Open Journal of Endocrine and Metabolic Diseases*, **15**, 111-122.
https://doi.org/10.4236/ojemd.2025.157011

Received: June 18, 2025
Accepted: July 26, 2025
Published: July 29, 2025

Abstract

Introduction: Asymptomatic hyperuricemia (AHU) is a metabolic complication that worsens cardiovascular morbidity and mortality, particularly in type 2 diabetic patients (T2DM). Our study aimed to determine the prevalence and identify the determinants of AHU in type 2 diabetic patients in the Northern region of Cameroon. **Methodology:** This was a cross-sectional and analytic study conducted over two years, from May 1st, 2023, to April 30th, 2025, focused on type 2 diabetic patients at the diabetic outpatient consultation unit of the Garoua General Hospital (GGH). We included all T2DM patient files with uric acid measurements during the study period. We excluded incomplete files, patients with gout, and those with symptoms of acute gout flares. The logistic regression model revealed the determinants of AHU with a significance threshold of 5%. **Results:** Of the 210 patients included, with a median age of 52 (IQR: 43 - 62) years, 58.57% were men. The median duration of T2DM was 5 (IQR: 2 - 9) years, with 85.78% having poor glycemic control (A1c > 7%). The prevalence (95% CI) of AHU in our study population was 40.48 (33.82 - 47.45)%. The identified determinants [aOR (95% CI), p] to AHU were a past history of obesity [1.99 (1.03 - 3.83), p = 0.040] and a glomerular filtration rate <60 mL/mn/1.73m^2 [5.95 (1.51 - 23.38), p = 0.011].

Conclusion: The high prevalence of AHU and its determinants (obesity and GFR < 60 mL/mn/1.73m^2) require systematic screening and management to reduce cardiovascular risk.

Keywords

Asymptomatic Hyperuricemia, Type 2 Diabetes, Determinants, Northern-Cameroon

1. Introduction

Hyperuricemia is defined as a uric acid level above 60 mg/L [1]. It can be asymptomatic or cause microcrystalline arthropathy, also known as gout. Asymptomatic hyperuricemia (AHU) is hyperuricemia without gout. It has three preclinical stages: asymptomatic hyperuricemia (no clinical manifestations of gout), asymptomatic uric acid crystal deposits (imaging evidence of crystal deposits without gout symptoms), and asymptomatic hyperuricemia with uric acid crystal deposits (hyperuricemia with imaging evidence of crystal deposits without gout symptoms) [1]. Often overlooked, AHU is an independent risk factor for metabolic and cardiovascular diseases. A close relationship between hyperuricemia, diabetes mellitus, hypertension, obesity, renal failure, and cardiovascular diseases has been highlighted in recent decades [2]. Managing AHU involves a healthy lifestyle and controlling associated chronic conditions. However, initiating hypouricemic therapy may be more harmful than beneficial [3] [4].

Diabetes mellitus is a major health issue with a steady increase worldwide. According to the International Diabetes Federation, about 589 million adults will live with diabetes in 2024, and this may rise to 852 million by 2050 if nothing is done for its prevention [5]. The burden of diabetes mellitus is due to its high morbidity and mortality and the development of chronic complications. Hyperuricemia worsens this burden by increasing the prevalence of microvascular complications, namely diabetic nephropathy, diabetic retinopathy, and diabetic neuropathy. In diabetic patients, several mechanisms, including insulin resistance and renal failure, contribute to the accumulation of uric acid [6].

In our context, where the financial burden of diabetes is particularly high and resources are limited, screening, knowledge, and efficient management of asymptomatic hyperuricemia are necessary. We conducted this study to evaluate the prevalence and identify the determinants of asymptomatic hyperuricemia in patients with type 2 diabetes (T2DM) in Northern Cameroon.

2. Methodology

2.1. Study Design and Study Population

This was a cross-sectional, analytic, and retrospective study over a two-year period from May 1st, 2023, to April 30th, 2025, at the diabetic outpatient consultation unit

of the Garoua General Hospital. This reference health facility is located in the capital of the North region of Cameroon, with a population of about 2.5 million. It is a first-category hospital open to the public since September 1st, 2022. Accessible via a well-paved road, it is situated 15 minutes from the city centre. The hospital consists of ten units divided into three groups: administrative services, clinical services, and auxiliary services. The diabetic unit includes a therapeutic education unit, a nutrition unit, an outpatient consultations unit, and a conventional hospitalization unit. Outpatient endocrinology/diabetes consultations have been conducted every Tuesday and Thursday by the same endocrinologist since May 2023. Patients can also receive consultations in nutrition, cardiovascular, ophthalmological workups, and various biological investigations.

We included all type 2 diabetic patients' files who measured their uricemia during the study period. Incomplete files, patients with gout, and those presenting symptoms of acute gouty crises were excluded.

2.2. Sampling

We conducted a consecutive, non-probabilistic, and exhaustive sampling for this in-hospital study.

2.3. Data Collection

Files of patients from diabetic outpatient consultations and those hospitalized in the diabetic unit were retrieved from the archives. A pre-established and validated electronic questionnaire, created with Epi Info software version 7.2.6.0, was completed to allow direct data entry during the survey. The following data were collected:

- **Sociodemographic data:** age, sex and nationality.
- **Data concerning diabetes:** type of diabetes, duration, current treatment, and control status.
- **Past history:** hypertension, cardiovascular disease, renal disease, positive family history of hyperuricemia, gout, menopause, dyslipidemia, personal cancer.
- **Nutrition and lifestyle:** sedentary lifestyle, consumption of purine-rich or processed foods, smoking, alcohol consumption, diuretic-based treatment, tuberculosis therapy, or aspirin in preventive doses (75 to 300 mg/day).
- **General physical examination:** weight and height measurements, abdominal circumference, and blood pressure measurements following standard procedures. Body mass index (BMI) calculation: weight/(height)^2
- **Biological data:** glycated hemoglobin (A1c), current blood creatinine levels with calculation of glomerular filtration rate (eGFR), and blood uric acid levels.

2.4. Definition of Operational Terms

Case definition: hyperuricemia in the absence of gout (1st preclinical stage). Musculoskeletal ultrasound and dual-energy computed tomography were not per-

formed to detect urate crystal deposits in tissues.

Prevalence of asymptomatic hyperuricemia: number of hyperuricemia cases out of the total number of diabetic patients enrolled in the study.

Glycaemic control [5]:

- Optimal glycaemic control: A1c ≤ 7%.
- Poor glycaemic control: A1c > 7%.

Android obesity: abdominal circumference > 94 cm for men and >80 cm for women [7].

BMI interpretation [8]:

- Normal weight: BMI between 18.5 and 24.9 kg/m^2.
- Overweight: BMI between 25 and 29.9 kg/m^2.
- Grade 1 obesity: BMI between 30 and 34.9 kg/m^2.
- Grade 2 obesity: BMI between 35 and 39.9 kg/m^2.
- Grade 3 obesity: BMI ≥ 40 kg/m^2.

2.5. Statistical Analysis

Data collected from Epi Info software version 7.2.6.0 were exported to IBM SPSS software version 23 for statistical analysis. Numbers and frequencies were calculated for all qualitative variables. The mean [standard deviation (SD)] and the median [interquartile range (IQR)] were used to characterize quantitative variables, depending on whether the data distribution was normal or not, respectively. The prevalence [confidence interval at 95% (95% CI)] of asymptomatic hyperuricemia was calculated as the proportion of asymptomatic hyperuricemia cases among the total number of diabetic patients recruited. Patients were divided into two groups based on whether they had asymptomatic hyperuricemia (AHU) or not. We used Student's t-test or its non-parametric equivalent to compare continuous variables. Proportions were compared using the chi-square test. Logistic regression was used to identify factors associated with asymptomatic hyperuricemia. Explanatory variables with $p < 0.05$ in univariate analysis were included in the multinomial logistic regression model to identify independent determinants of asymptomatic hyperuricemia. The significance threshold in this multivariate analysis was set at 5%. The power of association was assessed with the adjusted odds ratio (aOR) and its 95% CI.

3. Results

We included a total of 210 patients who met our inclusion criteria at the diabetic outpatient consultation unit during the study period.

3.1. General Data of the Study Population

The study population consisted of 123 men (58.57%) and 87 women (41.43%), with a male-to-female ratio of 1.41. The median age (IQR) of patients was 52 (43-62) years. The majority of patients were under 55 years old (118; 56.19%) and were of Cameroonian nationality (200; 95.24%) (**Table 1**).

Table 1. General data of the study population.

Variable	Total N = 210	AHU		OR	95% CI	p
		Present N = 85 (%)	Absent N = 125 (%)			
Sex						
Male	123	56 (45.5)	67 (54.5)	1.67	[0.94 - 2.95]	0.076
Female	87	29 (33.3)	58 (66.7)			
Age						
≥55 years	92	45 (48.9)	47 (51.1)	1.87	[1.07 - 3.27]	**0.028**
<55 years	118	40 (33.9)	78 (66.1)			

3.2. Past History

The median (IQR) duration of diabetes in our study population was 5 (2 - 9) years, and the majority presented poor glycemic control (175; 85.78%). The main cardiovascular risk factors associated with diabetes in our study population were a sedentary lifestyle (181; 86.19%), dyslipidemia (123; 58.57%), hypertension (71; 33.81%), and obesity (70; 33.33%) (Table 2(a)).

Table 2. (a) Past history of the study population. (b) Nutrition and lifestyle of the study population.

(a)

Variable	Total N = 210	AHU		OR	95% CI	p
		Present N = 85 (%)	Absent N = 125 (%)			
A1c						
>7%	175	71 (40.6)	104 (59.4)	0.84	[0.38 - 1.85]	0.667
≤7%	29	13 (44.8)	16 (55.2)			
Hypertension						
Yes	71	37 (52.1)	34 (47.9)	2.06	[1.15 - 3.69]	**0.014**
No	139	48 (34.5)	91 (65.5)			
History of obesity						
Yes	70	35 (50.0)	35 (50.0)	1.80	[1.00 - 3.22]	**0.047**
No	140	50 (35.7)	90 (64.3)			
Android obesity (cm)						
Yes	101	46 (45.5)	55 (54.5)	1.50	[0.86 - 2.61]	0.150
No	109	39 (35.8)	70 (64.2)			
Known cardiovascular disease						
Yes	12	4 (33.3)	8 (66.7)	0.72	[0.21 - 2.48]	0.604
No	198	81 (40.9)	117 (59.1)			
Known renal disease						

Continued

Yes	7	7 (100)	0 (0)	/	/	**0.001**
No	203	78 (38.4)	125 (61.6)			
Family history of hyperuricemia/gout						
Yes	0	0 (0)	0 (0)	/	/	/
No	210	85 (40.5)	125 (59.5)			
Menopause						
Yes	42	17 (40.5)	25 (59.5)	1.87	[0.76 - 4.61]	0.172
No	45	12 (26.7)	33 (73.3)			
Dyslipidaemia						
Yes	123	52 (42.3)	71 (57.7)	1.19	[0.68 - 2.10]	0.527
No	87	33 (37.9)	54 (62.1)			
Personal cancer						
Yes	2	0 (0)	2 (100)	/	/	0.241
No	208	85 (40.9)	123 (59.1)			

(b)

Variable	**Total N = 210**	**AHU**		**OR**	**95% CI**	**p**
		Present N = 85 (%)	**Absent N = 125 (%)**			
Consumption of purine rich food						
Yes	207	83 (40.1)	124 (59.9)	0.33	[0.03 - 3.75]	0.352
No	3	2 (66.7)	1 (33.3)			
Consumption of processed food						
Yes	14	4 (28.6)	10 (71.4)	0.57	[0.17 - 1.87]	0.348
Non	196	81 (41.3)	115 (58.7)			
Thiazide diuretics						
Yes	11	6 (54.4)	5 (45.5)	1.82	[0.54 - 6.18]	0.329
No	199	79 (39.7)	120 (60.3)			
Tuberculosis therapy						
Yes	1	0 (0)	1 (100)	/	/	0.683
No	209	85 (40.7)	124 (59.3)			
Preventive dose aspirin						
Yes	11	5 (45.5)	6 (54.5)	1.24	[0.37 - 4.20]	0.730
No	199	80 (40.2)	119 (59.8)			
Smoking						
Yes	2	2 (100)	0 (0)	/	/	0.085
No	208	83 (39.9)	125 (60.1)			
Alcohol consumption						

Continued

Yes	20	9 (45.0)	11 (55.0)	1.23	[0.48 - 3.10]	0.665
No	190	76 (40.0)	114 (60.0)			
Sedentary lifestyle						
Yes	181	75 (41.4)	106 (58.6)	1.34	[0.59 - 3.05]	0.479
No	29	10 (34.5)	19 (65.5)			

Note: A1c = Glycated Hemoglobin.

Almost all participants consumed purine-rich foods (207; 98.57%), whereas few consumed processed food (14; 6.67%). Twenty-seven (12.87%) patients were on diuretics, mainly loop diuretics (16; 59.26%) (Table 2(b)).

3.3. Anthropometric Measurements of the Study Population

According to the American NCEP-ATP III classification and sex, 101 (48.10%) patients in our sample had android obesity, and 64 (30.48%) were overweight (Table 3).

Table 3. Anthropometric measurements of the study population.

Variable	Total N = 210	AHU		OR	95% CI	p
		Present N = 85 (%)	Absent N = 125 (%)			
Body mass index						
<25 kg/m²	80	28 (35.0)	52 (65.0)	1		
25 - 29.9 kg/m²	64	28 (43.7)	36 (56.3)	1.44	[0.73 - 2.84]	0.285
≥30 kg/m²	66	29 (43.9)	37 (56.1)	1.47	[0.75 - 2.84]	0.270

3.4. Biological Parameters of the Study Population

Table 4 summarizes the biological parameters of the study population and their distribution according to the glomerular filtration rate and uricemia levels.

Table 4. (a) Glomerular filtration rate of the study population; (b) Distribution of the study population according to uricemia levels.

(a)

Variable	Total (N = 190)	AHU		OR	95% CI	p
		Present N = 76 (%)	Absent N = 114 (%)			
eGFR ≤ 60 mL/mn/1.73m²	162	53 (32.7)	109 (67.2)	2.93	[5.34 - 40.77]	0.051
eGFR > 60 mL/mn/1.73m²	28	23 (82.1)	5 (17.9)	12.70	[4.35 - 37.01]	**<0.001**

(b)

Variable	Number (N = 210)	Frequency (%)
Level of uricemia		

Continued

<60 mg/L	125	59.52
60 - 100 mg/L	75	35.71
≥100 mg/L	10	4.76

Note: eGFR = Glomerular Filtration Rate.

3.5. Prevalence of Asymptomatic Hyperuricemia

The prevalence (95% CI) of asymptomatic hyperuricemia in our study population was **40.48%** (33.82% - 47.45%).

3.6. Determinants of Asymptomatic Hyperuricemia

On bivariate analysis, age ≥ 55 years (p = 0.028), the presence of hypertension (p = 0.014), a past history of obesity (p = 0.047), a past history of renal disease (p = 0.001), and eGFR <60 mL/min/1.73m^2 (p < 0.001) were significantly associated with asymptomatic hyperuricemia.

On multivariate analysis, independent factors associated [aOR (95% CI), p] with asymptomatic hyperuricemia were a past history of obesity [1.99 (1.03 - 3.83), p = 0.040] and eGFR < 60 mL/min/1.73m^2 [5.95 (1.51 - 23.38), p = 0.011] (Table 5).

Table 5. Determinants of asymptomatic hyperuricemia.

Variable	Adjusted OR	95% Confidence Interval	Adjusted p-value
Age ≥ 55 years	1.22	[0.65 - 2.29]	0.541
Hypertension	1.16	[0.58 - 2.31]	0.673
Past history of obesity	1.99	[1.03 - 3.83]	**0.040**
eGFR < 60 mL/mn/1.73m^2	5.95	[1.51 - 23.38]	**0.011**

4. Discussion

Our study aimed to evaluate the prevalence and identify the determinants of asymptomatic hyperuricemia in patients with type 2 diabetes in Northern Cameroon, involving 210 patients. The results are as follows:

- The prevalence of asymptomatic hyperuricemia in our study population was 40.48%.
- The identified determinants were a history of obesity and eGFR < 60 mL/mn/1.73m^2.

4.1. Prevalence of Asymptomatic Hyperuricemia

In our study, the prevalence (95% CI) of AHU was 40.48% [33.82 - 47.45], similar to results found by Choukem *et al.* at 38.1% in a population of 438 patients with T2DM in Douala, Cameroon [9]. Some studies in sub-Saharan Africa with smaller sample sizes revealed lower prevalence rates: 29.4% in Bouaké, Côte d'Ivoire, and

28.7% in Cotonou, Benin [10] [11]. These authors studied hyperuricemia in general, including gout and systemic hyperuricemia. The difference in prevalence could be explained by a greater proportion of patients presenting with symptomatic hyperuricemia, usually managed in rheumatology outpatient consultations rather than diabetic consultations.

The prevalence of AHU was 45.50% in men versus 33.30% in women, with no statistically significant difference in our study (p = 0.076). This aligns with the masculine predominance in our sample and the results of *Choukem et al.* in Douala (40.40% in men versus 35.20% in women) [9].

Patients aged 55 years and above had a high prevalence of AHU (48.90%) and hypertension (52.10%), with a statistically significant difference (p = 0.014). It has been shown that the loss of physiological vasodilation with advancing age positively correlates with the onset of cardiovascular diseases and their risk factors, such as hypertension, hyperuricemia, and diabetes mellitus [12].

4.2. Determinants of Asymptomatic Hyperuricemia

Our study identified a statistically significant relationship between GFR <60 mL/min/1.73m^2 and AHU [aOR 5.95 (CI at 39%: 1.51 - 23.38), p = 0.011]. This relationship was also highlighted by *Choukem et al.* in Douala, Cameroon (OR 5.6; CI at 95%: 1.4 - 5.9) [9]; *Wanvoegbe et al.* in Cotonou, Benin (p = 0.026) [11]; and *Aksas et al.* in Bab el Oued, Algeria (p < 0.001) [13]. Notably, 82.1% of T2DM patients with eGFR < 60 mL/min/1.73m^2 had AHU compared to 17.9% who did not. Insulin resistance and hyperinsulinism, the physiological bases of type 2 diabetes, restrain cellular utilization of glucose and limit the proximal tubular reabsorption of uric acid, leading to hyperuricemia [13]. The accumulation of uric acid in the kidneys can result in the formation of urate stones and renal lesions. Several authors report a high prevalence of kidney stones in patients with T2DM [14] [15]. Additionally, hyperuricemia appears to be a more detrimental factor in renal progression when diabetes mellitus is present, creating a vicious cycle in the metabolic and renal continuum.

The second determinant of AHU [aOR (CI at 95%), p] identified in our study was obesity with a BMI ≥ 30 kg/m^2 [1.99 (1.03 - 3.83), p = 0.040]. These findings are similar to those of sub-Saharan authors like *Choukem et al.* (OR 2.9; CI at 95%: 2.3 - 13.7) [9] and *Wanvoegbe et al.* (p = 0.04) [11]. Obesity, as a precursor of insulin resistance and hyperinsulinism, results in hyperuricemia by increasing renal reabsorption of uric acid. In the metabolic and renal continuum, hyperuricemia is linked to metabolic syndrome (android obesity, dyslipidemia, diabetes mellitus, and hypertension), a claim dating back to the 19th century through cellular cultures and animal models [1].

Hyperuricemia has a U-shaped relationship with mortality in both men and women. This mortality is linked to increased cardiovascular and renal mortality, as proven by epidemiological studies [1]. On the pathophysiological level, hyperuricemia stimulates the production of reactive oxygen species and inhibits nitric

oxide. This leads to endothelial dysfunction, resulting in platelet adhesion, increased LDL cholesterol oxidation, and consequently atherosclerosis, contributing to cardiovascular diseases [13].

Lifestyle changes, regular physical activity, and weight loss form the basis for managing AHU and associated comorbidities to reduce global cardiovascular risk [9]. Initiating hypouricemic therapy in AHU remains controversial due to a lack of strong epidemiological evidence, despite clear benefits in AHU associated with renal failure [16] [17].

4.3. Implications of the Findings for Public Health Policies and Everyday Practice

The high prevalence of AHU and key determinants such as obesity and eGFR < 60 mL/min/1.73m^2 highlight the need for integrating systematic screening strategies and proactive management of AHU in care programs for T2DM patients. Public health policies must encourage awareness programs addressing cardiovascular risks associated with AHU. Improving diagnostic capabilities by reducing the costs of screening tests, such as plasma uric acid measurement, musculoskeletal ultrasound, and dual-energy computed tomography, could be crucial in preventing complications associated with AHU. In daily clinical practice, the results call for an integrated approach to better control modifiable risk factors, such as obesity, and monitor renal function in diabetic patients to reduce overall cardiovascular risk in this vulnerable population.

4.4. Study Limitations

This study has several limitations that should be acknowledged. Specifically, the lack of musculoskeletal ultrasound and dual-energy computed tomography to identify tissue deposition of urate crystals limited the characterization of preclinical stages, despite their availability in our healthcare system. Additionally, purine intake is prevalent in Northern Cameroon due to the development of livestock farming in this area. However, quantifying purine intake poses a limitation due to the near-daily consumption of red meat by participants. Furthermore, a longitudinal assessment of serum creatinine levels over a three-month period would have provided a more robust evaluation of renal function impairment in this at-risk population.

5. Conclusion

Asymptomatic hyperuricemia is common in our population of patients with T2DM. The determinants were obesity and eGFR < 60 mL/min/1.73m^2. It is essential to systematically test plasma uric acid and manage global cardiovascular risk for T2DM patients to reduce morbidity and mortality.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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