



## Biological Profile of Gout in Obese and Non-Obese Subjects in Yaoundé, Cameroon

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### Abstract:

**Background:** Gout, the leading cause of arthritis in adults, is frequently associated with obesity which promotes chronic hyperuricemia. Biological signs seem more severe in obese subjects. The aim of this work was to study the biological characteristics of gout in obese subjects in the city of Yaoundé.

**Methods:** We carried out a cross-sectional study. The study population consisted of obese and non-obese subjects, suffering from gout, who were compared from socio-demographic and para-clinical characteristics. The Mann Whitney test was used to compare the medians between the two groups. The significance threshold was set at 0.05.

**Results:** Among the 101 recruited, 44 were obese and 57 were not obese. The median ages of the subjects were 61 [51 - 68] years and 58.50 [53 - 64.75] years respectively for the non-obese and the obese. There was no significant difference between uric acid levels, serum creatinine, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides between the groups of non-obese and obese. However, we observed that C-reactive protein levels were significantly higher in the non-obese group compared to obese.

**Keywords:** Gout, Obesity, Hyperuricemia, Metabolic Syndrome.

**Abbreviation:** HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein; BMI: Body Mass Index; CRP: C-Reactive Protein

### 1. INTRODUCTION

Gout is the leading cause of arthritides in adults [1]. It is characterized by an ever increasing frequency and high morbidity [2]. Its global prevalence varies between 1 and 4%, as long as in Cameroon a study carried out in 2007 found a prevalence of 5.9% [3].

Gout is thus a frequent pathology in our context, responsible for joint pain but also for premature death [2]. This significant morbidity is explained by a strong association between hyperuricemia and cardiovascular and renal diseases, whose relationship increases with the duration of hyperuricemia. Hyperuricemia is responsible for activation of the renin angiotensin aldosterone system, decrease in nitric oxide activity, proliferation of vascular myocytes and insulin resistance. Through these mechanisms, hyperuricemia and gout promotes the occurrence of cardiometabolic pathologies thus worsening the prognosis of these patients [4,5].

Obesity is the most important comorbidity of gout compared to hypertension, diabetes and dyslipidemia[6,7]. In Cameroon, it is present in 78 to 84% of subjects suffering from gout [8,9]. Obesity is associated with gout according to a dose dependent effect as it is responsible for an exponential increase in uricemia in gouty patients, in particular by the secretion of pro-inflammatory cytokines[10,11]. These two mechanisms suggest that the biological profile of gout could vary depending on the body mass index of patients, which explains the higher morbidity of obese gouty patients. The metabolic alterations of gout depend on hyperuricemia and could be amplified by the

accentuation of the inflammatory process caused by obesity [12]. Therefore, this study aimed to compare the biological profile of gout in obese and non-obese patients.

## 2. MATERIAL AND METHODS

### 2.1. Study Framework

We conducted a cross-sectional study from January to April 2019 in 06 hospitals in the city of Yaoundé: Yaoundé Central Hospital, *Hôpital de la Caisse Nationale de Prévoyances Sociales de Yaoundé* (HCNPS), Biyem-Assi district hospital, Yaoundé University Hospital and the Efoulan district hospital.

### 2.2. Participants

Were included all patients over the age of 18 who met the diagnostic criteria for gout according to ACR / EULAR 2015 or followed for documented chronic gout[13]. Patients with intensive physical activity, on corticosteroid, hormonal contraceptives, or with muscle wasting, ascites and edematous syndrome were not included. All patients with another etiology of arthritis associated with gout were excluded.

The participants were then divided into two groups:

Group 1: Obese patients: BMI  $\geq 30$  Kg / m<sup>2</sup> or waistline > 102 cm for men or waistline > 88 cm for women [14].

Group 2: Non-obese patients: BMI < 30 Kg / m<sup>2</sup> or waistline  $\leq$  102 cm for men or waistline  $\leq$  88 cm for women.

### 2.3. Ethical Considerations

The study was approved by the institutional research ethics committee of the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé 1. It was conducted in strict accordance with the principles of the Declaration of Helsinki.

### 2.4. Data Collection

Participants were recruited from internal medicine consultation and hospitalization. Data were collected by the principal investigator using a data collection sheet comprising the following information: socio-demographic and anthropometric characteristics and the history of gout. Then a venous sample was taken to carry out dosages of: C-reactive protein, uricemia, creatinemia, total cholesterol, HDL cholesterol, triglycerides, and LDL cholesterol calculated by the Friedwald equation when the concentration of triglycerides was less than 3.4 g / l.

### 2.5. Statistical Analysis

We built a database using Excel 2013 software, realized analyzes using SPSS software version 22. The data were reported in median and interquartile range for continuous variables and in proportions for categorical variables. The Mann Whitney test was used to compare the medians between the groups. The significance threshold was set at 0.05.

## 3. RESULTS AND DISCUSSION

### 3.1. Results

#### 3.1.1. General Characteristics of the Study Population

We included 101 subjects, including 57 non-obese with a BMI of 26.80 [24.14 - 27.91] Kg / m<sup>2</sup> and 44 obese BMI 32.53 [30.89 - 35.38] Kg / m<sup>2</sup>. The median gout durations were 4.00 [1.00 - 7.75] years for group 1 and 4.00 [2.00 - 7.00] years for group 2. The median ages were similar in the two groups (p = 0.369). The socio-demographic characteristics of the study population are presented in Table 1.

**Table 1.** Characteristics of the study population

	Non obese (n=57)	Obese (n=44)	p
Age	61.00 [51.00 – 68.00]	58,50 [53.00 – 64.75]	>0.05
Sex ratio (H/F)	10.4	10	>0.05
Sedentary lifestyle, n (%)	22 (38.60)	27 (61.36)	>0.05
Hypertension, n (%)	21 (36.84)	20 (45.45)	>0.05
Diabetes, n (%)	10 (17.54)	8 (18.18)	>0.05
Metabolic syndrome, n (%)	13 (22.8)	24 (54.5)	<0.05

3.1.2. Biological Profile

The non-obese group's uricemia was 87.00 [66.53 - 98.10] mg / l compared to 86.13 [74.00 - 100.86] mg / l. There was no significant difference between the two groups (p = 0.885). The effect of obesity on inflammation in gouty patients was evaluated using C-reactive protein. The CRP of non-obese patients (24.80 [12.00 - 96.00] mg / l) was significantly higher than that of obese patients (5.50 [2.50 - 30.75] mg / l); p = 0.002. Regarding renal function, we did not observe any significant difference between the two groups (p = 0.213). The serum creatinine level of non-obese patients was 12.82 [9.49 - 17.75] mg / l compared to 11.09 [9.20 - 14.07] mg / l for obese patients.

Table2. Biological profile

	Non obese (n=57)	Obese (n=44)	p
Uricemia (mg/l)	87.00 [66.53 – 98.10]	86.13 [74.00 – 100.86]	>0.05
CRP (mg/l)	24.80 [12.00 – 96.00]	5.50 [2.50 – 30.75]	<0.01
Créatininemia (mg/l)	12.82 [9.49 – 17.75]	11.09 [9.20 – 14.07]	>0.05
Total cholestérol (mg/l)	1.56 [1.36 – 2.32]	1.98 [1.30 – 2.45]	>0.05
HDL cholestérol (mg/l)	0.46 [0.35 – 0.55]	0.37 [0.34 – 0.55]	>0.05
LDL cholestérol (mg/l)	1.05 [0.76 – 1.42]	1.45 [0.93 – 1.72]	>0.05
Triglycérides (mg/l)	1.17 [0.92 – 1.50]	1.00 [0.93 – 1.72]	>0.05

Regarding the lipid profile, total cholesterol was higher in obese patients (1.98 [1.30 - 2.45] mg / l) compared to non-obese patients (1.56 [1.36 - 2.32] mg/l). Likewise, LDL cholesterol levels were higher in the obese group 1.45 [0.93 - 1.72] mg/l compared to 1.05 [0.76 - 1.42] mg/l. HDL cholesterol was higher in non-obese patients (0.46 [0.35 - 0.55] mg / l compared to 0.37 [0.34 - 0.55] mg / l). However, these differences were not statistically significant.

4. DISCUSSION

We conducted a cross-sectional study to compare the biological profiles of gout in obese and non-obese patients. It appears that the rates of serum uricemia and serum creatinine are similar in the two groups. In contrast, obese patients had lower CRP and HDL cholesterol, higher total cholesterol and LDL cholesterol levels than non-obese patients.

The main biological marker for gout is uricemia. Its serum levels are similar in our study between the obese and the non-obese. These results are in contrast to those found in the literature showing a strong and positive correlation between the body mass index and the uricemia, making the latter one of the main risk factors for obesity [15,16]. Our observation suggests that obesity plays a less important role than other factors such as diet and genetics in our population. However, the size of our sample does not allow us to confirm this hypothesis. In addition, CRP, the inflammatory marker evaluated in this study, is higher in non-obese patients; obesity therefore seems to be a factor in controlling inflammation in gouty patients. This observation, similar to that made with uricemia, diverges from numerous studies which report that obesity, which itself causes systemic inflammation, aggravates the inflammation caused by deposits of uric acid crystals [17, 18]. However, a study from Taiwan on recent changes in clinical manifestations and risk factors associated with gout shows that, despite an increase in body mass index after 1992, the frequency of episodes and the severity of inflammatory signs have significantly decreased [19]. In addition, obesity is associated with hyperuricemia and the latter can constitute a protective factor against the oxidative stress found during gout. In fact, obesity is associated with a predominant production of superoxide dismutase (SOD), an antioxidant enzyme which seems to have a negative correlation with IL-6 and TNF- $\alpha$ , thus reducing the chronic inflammatory process which leads to formation of tophi during gout [20,21]. Obesity could therefore currently have a protective effect on the severity of gout, contrary to what has been observed in the past.

The lipid profile assessment found that obese patients had higher levels of total cholesterol and LDL cholesterol and lower levels of triglycerides and HDL cholesterol. These results, although not significant, agree with those found in the literature and are due to the presence of other cardiovascular risk factors such as physical inactivity and an unbalanced diet in obese patients. We found a predominance of the metabolic syndrome in obese subjects. This predominance of metabolic syndrome in obese subjects was also noted by Moulin et al in Angola in 2016 who had an average of 24.5% of subjects with metabolic syndrome [22]. In Cameroon, Doualla et al found a higher prevalence, 54.6% of the metabolic syndrome, which can be explained by the size of their sample, which was larger than ours [9]. The predominance of metabolic syndrome in obese subjects that we obtained can be explained by the fact that obesity is a major

determinant of metabolic syndrome as described by many studies, including that of Sherling et al in 2017 in the United States. United [22].

## **5. CONCLUSION**

Our results suggest that obesity appears to reduce the inflammatory response during gout, and do not have any effect on creatinemia and uricemia.

## **RESULTS**

- [1] Our results suggest that obesity appears to reduce the inflammatory response during gout, and do not have any effect on creatinemia and uricemia.
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