Relationship between Obesity, Serum Uric Acid, Serum Potassium and Glomerular Filtration Rate with Electric Left Ventricular Hypertrophy in Blacks Central Africans with High Blood Pressure

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Abstract

The authors conducted a retrospective study over a period of 6 months in a hypertensive population in order to determine the correlation between serum uric acid on glomerular filtration rate (GFR) and estimated serum potassium with left ventricular hypertrophy (LVH) and obesity. This study enrolled 122 patients including 63 women (51.6%). The mean age was 55.9 ± 10.6 years (range 30 to 74 years). Obesity weight was found in 38 cases (31.1%) of which 20 were men (33.9%) and 18 women (28.6%). Abdominal obesity was found in 104 cases (85.2%). The average serum uric acid in patients with obesity was 63.3 ± 18.9 mg/L vs 62.4 ± 14.2 mg/L for no-obese patients (p = 0.63). The average serum potassium in obese patients was 4.06 ± 0.42 mEq/L vs 4.02 ± 0.46 mEq/L for no-obese (p = 0.65). The average GFR was 73.4 ± 21.4 ml/L in obese patients vs 66.6 ± 22.6 ml/min in no-obese (p = 0.03). The LVH was found in 81 cases (66.4%). The LVH was found in 65 (62.5%) obese patients vs 16 (88%) non-obese patients (OR = 4.8, 95% 1.04 - 22, p = 0.02). Only abdominal obesity has been correlated with left ventricular hypertrophy after multivariate analysis. Emphasis must be focused on public health actions for effective and appropriate measures against obesity and hypertension, whose prevalence is increasing in our region.
Keywords

Obesity, High Blood Pressure, Glomerular Filtration Rate, Uric Acid, Serum Potassium, Black African

1. Introduction

The management of high blood pressure (HBP) and other cardiovascular risk factors remains problematic in sub-Saharan Africa. Indeed, because of poverty, achieving minimum balance required to identify factors associated with HBP remains uncertain because of low socio-economic development. The correlation of obesity with dyslipidemia or diabetes has been established by several authors [1] [2] [3] [4]. The serum uric acid, independent cardiovascular risk factor, remains little tested in our environment [5]. The prevalence of kidney disease evaluated by estimated glomerular filtration rate (GFR) is increasing in rural Africa [6]. The left ventricular hypertrophy (LVH) represents the anomaly found in most of the electrocardiogram in HBP patients and is often correlated with other cardiovascular risk factors [7]. Hitherto, no study has been conducted in Brazzaville to assess the relationship of various cardiovascular risk factors with LVH in patients with HBP.

This study was conducted to establish the correlation between serum uric acid, serum potassium and GFR with electrical LVH and obesity in Congolese patients with HBP.

2. Patients and Method

This cross sectional study was conducted from April to September 2014 (6 months) in the outpatient unit of University Hospital of Brazzaville.

We included the patients with HBP in initial consultation, with no current antihypertensive treatment, followed as an outpatient and whose records included the following variables: age, sex, weight (cm), height (m), waist circumference (cm), medical history of cardiovascular risk factor and disease, The diet blood biochemistry analysis (uric acid, creatinine, serum potassium) are effectuate in the laboratory of biochemistry of Teaching Hospital of Brazzaville. The automaton HORIBA® has been used. The electrocardiogram (EKG) have been realised with SCHILLER® device.

Patients treated with gout or allopurinol were excluded from the study as well as those who had in the history impaired renal function or aged 75 and older. Ethical issues were taken into account according to the Helsinki Declaration.

The following variables were analyzed: age, sex, obesity, underweight and overweight across the body mass index (BMI), abdominal obesity, uric acid in mg/L, serum creatinine in mg/L, GFR in ml/min, the serum potassium in mEq/L and LVH.

BMI was calculated manually by the formula weight (in kilograms) on the square of height (in meters), weight obesity was defined as BMI greater than 30,
overweight was defined as BMI between 25 and 29.9. Abdominal obesity was defined as waist circumference (PA) measured with a tape—m ≥ 94 cm for men and 80 cm for women based on the International Diabetes Foundation criteria (IDF) [8]. The GFR was calculated manually by the formula Crockcroft-Gault (mL/min) is \((140 - \text{age in years}) \times \text{weight in kilograms}/7.2 \times \text{blood levels of creatinine in mg/l (for women the result is multiplied by 0.85)} \) [9]; positivity criteria of LVH were the Sokolow-Lyon (S amplitude in V1 plus R amplitude in V5 or V6) and Cornell (R amplitude in aVL plus S amplitude in V3) [7]. We found that LVH was present if the Sokolow was ≥3.5 mV or Cornell index ≥ 2 mV in men and ≥2.8 mV in women.

Serum creatinine, uric acid and potassium were performed at the biochemistry laboratory of the CHU of Brazzaville with an automaton PLC Lysa® 500. ECGs were conducted with a Schiller AT-1® device.

The variables have been analyzed by the software Epi Info 3.3.2 of CDC (Atlanta, USA). The qualitative variables were expressed as number and percentage, quantitative variables as mean ± standard deviation and extremes. The univariate analysis had use, the calculation of Odds ratio (OR) with confidence interval (CI) of 95%. A logistic regression model of LVH was used for multivariate analysis adjusting for independent variables. The significance level of the comparisons was less than 5%.

3. Results

This study included 122 patients, 63 women (51.6%). The population characteristics are reported in Table 1.

<table>
<thead>
<tr>
<th>Parameters of obesity</th>
<th>All ((n = 122))</th>
<th>Men ((n = 59))</th>
<th>Women ((n = 63))</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean age</strong> ((\text{years}))</td>
<td>55.9 ± 10.6</td>
<td>56.2 ± 10.6</td>
<td>55.7 ± 10.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Medical history</strong></th>
<th>All ((n = 122))</th>
<th>Men ((n = 59))</th>
<th>Women ((n = 63))</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No medical history</td>
<td>42 (34.4%)</td>
<td>22 (37.3%)</td>
<td>20 (31.8%)</td>
<td>NS</td>
</tr>
<tr>
<td>No CVD**</td>
<td>61 (50%)</td>
<td>32 (50.8%)</td>
<td>29 (49.2%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Blood biochemia</strong></th>
<th>All ((n = 122))</th>
<th>Men ((n = 59))</th>
<th>Women ((n = 63))</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum creatinine* ((\text{mg/l}))</td>
<td>13.21 ± 3.23</td>
<td>14.1 ± 3.5</td>
<td>12.4 ± 2.7</td>
<td>0.01</td>
</tr>
<tr>
<td>Kalemia* ((\text{mEq/l}))</td>
<td>4 ± 0.45</td>
<td>4 ± 0.52</td>
<td>4 ± 0.37</td>
<td>NS</td>
</tr>
<tr>
<td>Serum uric acid* ((\text{mg/l}))</td>
<td>63.35 ± 15.7</td>
<td>63.35 ± 15.7</td>
<td>63.35 ± 15.7</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*variables range: Age: 30 and 74; serum creatinine 6 and 26; BMI (Body mass index) 22 and 36; waist circumference 75 and 122; kalemia 3.1 and 5.7; serum uric acid 40 and 128; GFR (Glomerular filtration rate) 23 and 122. **CVD: cardiovascular disease, NS: no significant.
3.1. Obesity Weight

Obesity weight was found in 38 cases (31.1%), divided into 20 men (33.9%) and 18 women (28.6%) \([OR = 0.78, 95\% CI 0.36 - 1.68, p = 0.33]\). The average serum uric acid was 63.3 ± 18.9 mg/L for patients with obesity underweight, and 62.4 ± 14.2 mg/L for non-obese patients \((p = 0.63)\). The uric acid was normal in 19 obese patients (37.3%) and high in 19 obese patients (26.8%) whereas in non-obese, it was normal in 32 cases (62.7%) and high in 52 cases (73.2%) \([OR = 1.6, 95\% CI 0.74 - 3.52, p = 0.15]\). Among the obese patients, the average serum potassium was 4.06 ± 0.42 mEq/L, and for non-obese 4.02 ± 0.46 mEq/L \((p = 0.65)\).

The average serum creatinine was 13.91 ± 2.9 ml/L for obese, and 12.9 ± 3.3 ml/L for non-obese \((p = 0.13)\). The average GFR was 73.4 ± 21.4 ml/min for obese and 66.6 ± 22.6 ml/min for non-obese patients \((p = 0.03)\). The LVH was determinate in 24 obese patients (63.2%) and in 54 non-obese patients (67.9%) \([OR = 0.81, 95\% CI 0.36 - 1.81, p = 0.37]\).

3.2. Abdominal Obesity

Abdominal obesity was found in 104 cases (85.2%), 44 men (74.6%) and 60 women (95.2%) \([OR = 7.14, 95\% CI 1.8 - 25, p = 0.001]\). The average serum uric acid was 63.8 ± 16.1 mg/L in non-obese, and 62.8 ± 15.7 mg/L in the obese \((p = 0.80)\). The serum potassium was on average 4 ± 0.64 mEq/L for non-obese patients and 4.04 ± 0.41 mEq/L for obese patients \((p = 0.20)\). The average serum creatinine was 13 ± 2.95 mg/L for obese patients and 14.57 ± 4.56 for non-obese patients \((p = 0.28)\). The average GFR was 55 ± 15.7 ml/min for non-obese patients, and 71.1 ± 22.5 ml/min for the obese \((p = 0.007)\).

The LVH was found in 65 obese patients (62.5%) and in 16 non-obese patients (88%). The LVH was absent in two non-obese patients (11.1%) and in 39 obese patients (37.5%) \([OR = 4.8, 95\% CI 1.04 to 22, p = 0.02]\).

3.3. LVH

The LVH was found in 81 cases (66.4%). There were 42 men (51.9%) and 39 women (48.1%). The average serum creatinine was 12.92 ± 3.44 mg/L for patients with LVH and 2.72 ± 13.78 mg/L for patients without LVH \((p = 0.11)\). The average serum potassium was 4 ± 0.46 mEq/L for patients with LVH, and 4 ± 0.41 mEq/L for patients without LVH \((p = 0.60)\). The average serum uric acid was 64.6 ± 16.3 mg/L for patients with LVH, and 60.8 ± 14.1 mg/L for patients without LVH \((p = 0.21)\). The DFG has averaged 70.8 ± 23.1 ml/min for patients with LVH, and 64.6 ± 20.5 ml/min for patients without LVH \((p = 0.15)\).

The multivariate analysis between LVH and different parameters is given in Table 2.

4. Discussion

The change in lifestyle and eating habits has led to an increase in the prevalence of obesity and hypertension in sub-Saharan Africa \([1] [2]\). The prevalence of
Table 2. Multivariate analysis between left ventricular hypertrophy and other parameters.

<table>
<thead>
<tr>
<th></th>
<th>Odds Ratio</th>
<th>IC 95%</th>
<th>Z-Statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GFR*</td>
<td>1.0199</td>
<td>1.000 - 1.0402</td>
<td>1.9577</td>
<td>0.0503</td>
</tr>
<tr>
<td>Kalemia</td>
<td>0.9904</td>
<td>0.9014 - 1.0881</td>
<td>−0.2008</td>
<td>0.8409</td>
</tr>
<tr>
<td>Obesity</td>
<td>0.9528</td>
<td>0.4058 - 2.2375</td>
<td>−0.1109</td>
<td>0.9117</td>
</tr>
<tr>
<td>Waist obesity</td>
<td>0.1561</td>
<td>0.0318 - 0.7665</td>
<td>−2.2876</td>
<td>0.0222</td>
</tr>
<tr>
<td>Serum uric acid</td>
<td>1.0146</td>
<td>0.9877 - 1.0421</td>
<td>1.0577</td>
<td>0.2902</td>
</tr>
<tr>
<td>Constant</td>
<td>-</td>
<td>-</td>
<td>0.222</td>
<td>0.8243</td>
</tr>
</tbody>
</table>

*GFR: glomerular filtration rate.

abdominal obesity in this study (85.2%) is higher than the weight of obesity (31.1%), the first is predominant among women and second among men.

In sub-Saharan Africa, the prevalence of obesity and hypertension weight varies by region [3], and appear lower than that found in populations of the black diaspora. In fact, they remain subject to the same constraints as other populations of Western society.

Abdominal obesity, which represents a risk factor in the metabolic syndrome [9] [10], is very high in our study population. The metabolic syndrome is increasing substantially in different cities in sub-Saharan Africa with a corollary to increased cardiovascular risk [11].

Different studies have not established a clear correlation between obesity and hypertension except in South Africa [3], but obesity is a predictor of hypertension especially in female subjects [12].

The increasing prevalence of obesity being related to the Westernization of lifestyle in Africa, it also denotes a larger increase in disadvantaged populations, this seems related to low education and job insecurity [2] [13].

The GFR was higher in obese patients than in non-obese patients in our series. This is linked to glomerular hyperfiltration physiological linked to overweight [9]. Obesity does not appear to be correlated with serum uric acid, or potassium, or the GFR in our series. According to the literature, uric acid seems rather to be correlated with increased blood glucose and the installation of type 2 diabetes [14] [15]. Through this indirect pathway it could intervene on the emergence/appearance of obesity.

Hyperuricemia is considered an independent risk factor, it plays a pathological role in the progression of hypertension and impaired renal function [16]. Drug treatment of hyperuricemia allows better control of hypertension and renal function, but also would reduce the cardiovascular risk [17] [18]. These observations are unreliable, and large-scale studies are needed to confirm these observations [15] [16] [19].

The change in lifestyle of people has increased the prevalence of renal failure [4] [6] [20] [21], obesity [2] [3] in addition to hypertension and diabetes [3].

Among the factors analyzed in study notes, only abdominal obesity seems positively correlated with LVH. Indeed, among the factors induced by obesity in
hypertensive patients, LVH is revealed most strongly correlated according to Norton, in a series in South Africa [22]. Among the factors analyzed, only the GFR would be implicated in the occurrence of LVH [23] [24] [25]. It is also recognized that the decrease in GFR increase cardiovascular mortality [26]. LVH is negatively correlated with abdominal obesity, but not with obesity. In his series, Son et al found positive correlation with abdominal obesity and obesity. The difference may be methodological, because of the high number of patients included in his study [27].

5. Conclusion

It appears that among the various parameters analyzed, only abdominal obesity is correlated with LVH. Given the change in lifestyle now westernized in African cities, the emphasis must be placed on public health actions for public awareness for effective preventive and control measures against obesity and HBP.

References


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