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Association Between Clinical-biological Factors and Hypertension in the Urban-rural Population of Boma in the Democratic Republic of the Congo

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Abstract: Hypertension is the main preventable risk factor for cardiovascular disease. Knowledge and control of multiple risk factors for hypertension will reduce its prevalence, better management and therefore a reduction in the burden of disease. This study targeted other risk factors for high blood pressure in Boma, Democratic Republic of Congo. Methods: This was an analytical cross-sectional study. The study population consisted of individuals of the inhabitants of Boma aged at least 18 years living in rural and urban areas for at least one year. This study is a continuation of two previous studies. The sampling was randomized to several degrees which made it possible to select the neighborhoods, avenues, households and then adults to be examined. A total of 1,781 households were listed and 3,800 people were expected, but only 3,510 people were examined and whose blood samples were kept and biological analyzes are being carried out gradually. Data were analyzed using SPSS version 21 software for Windows. They were expressed in mean standard deviations (SD) for continuous variables and in frequencies (n) and percentages (%) for categorical variables. The percentages were compared using the chi-square test. Logistic regression was used to identify independent factors associated with hypertension. The threshold of statistical significance was set at a value of $p < 0.05$. Results: Information from 252 first blood collection samples from Congolese adults living in Boma and over 18 years of age was included in this analysis. The mean age of the study participants was 40.9 ± 15.4 years, of which 71.4% were female. Tobacco, alcohol, diabetes mellitus, and physical inactivity were the most common histories. BMI ($p = 0.001$), TT ($p = <0.001$), TH ($p = <0.001$), TT/Height ($p = 0.001$) and Uric acid ($p = <0.001$) values were significantly higher in urban areas than rural. In multivariate analysis, the probability of being associated with hypertension was almost times higher, respectively, AIP≥0.24 ($p = 0.357$) and Smoking ($p = 0.693$). It was almost 2 times higher, respectively, for Age≥50 years ($p = 0.007$), Physical inactivity ($p = 0.015$), Obesity ($p = 0.103$). It was nearly 4 times higher, respectively, for Uric Acid> 420 mg/dl ($p = 0.001$) and CRP> 3 mg/dl ($p = 0.015$). In the end, it was 10 times higher for Subclinical atherosclerosis ($p = 0.001$). Conclusion: The results revealed that high blood pressure has several modifiable risk factors such as CRP, uric acid and non-modifiable.

Keywords: Hypertension, Associated Factors, Boma

1. Introduction

Cardiovascular disease (CVD) is one of the leading causes of death and morbidity, and a major global health problem [1].

Hypertension is recognized as the most important modifiable risk factor for cardiovascular disease [2, 3]. Globally, it affects about 22% of the population aged 18 and over and is responsible for about 9.4 million deaths per year [4, 5].

About 75% of the world's hypertensive population lives in low- and middle-income countries (LMICs) [6].

Hypertension remains an important concern for public health systems especially in low and middle income countries because of its high prevalence and the multiple associated risk factors (ARF), some of which can be modifiable and others not modifiable [7].

In addition to several environmental factors related to lifestyle (salt consumption, physical inactivity, obesity and alcohol consumption, alcohol consumption, smoking, poor eating habits of glucose intolerance, atherosclerotic dyslipidemia and markers of Inflammation and increased arterial stiffness have been identified as independent predictors of high blood pressure [8].

Taking into account the WHO global action plan for the prevention and control of NCDs, 25% of global mortality from NCDs, Control and control of modifiable ARFs would inevitably reduce, the prevalence of hypertension and the number of imputable deaths from CVD and hypertension [8].

Understanding modifiable risk factors for hypertension can help prevent and reduce the burden of hypertension and CVD [9].

In the Democratic Republic of Congo (DRC), hypertension, considered rare or even non-existent in the general population in 1960 [10], has currently reached a prevalence of around 40% in both urban and rural areas [11-21].

To our knowledge, only the study by Lepira et al has investigated the relationship between hypertension and lipid, uric acid and the marker of inflammation in the hospital setting [22].

It was necessary to conduct this study in the general population in order to fill this quickly and resolve the limitations of our previous publications [19, 20].

2. Methods

2.1. Study Population and Study Design

We used the baseline data from the study on the determinants of hypertension in the city of Boma.

All details, including the rationale, framework, design and methodology of the study, have already been published [40, 41].

Both studies included all residents of Boma aged 18 or over living in rural and urban areas for at least one year. A multi-stage random sampling procedure made it possible to select the neighborhoods, avenues, households and then adults to be examined. A total of 1781 households were listed and 3800 people were expected, but only 3510 people were examined and whose blood samples have been kept and biological analyzes are being carried out gradually. This study, the first of its kind in the DRC, presents the results of 252 first samples examined.

2.2. Data Collection

Data collection was standardized using standard operating procedures at all study sites. Information on socio-demographic data, medical history and treatment was obtained using a questionnaire through interviews conducted by trained investigators.

2.3. Measures

Anthropometric measurements (such as body weight, waist circumference, height), blood pressure, and heart rate were collected by well-trained medical students. Blood pressure was measured using digital blood pressure monitors (OMRON MIT5 Connect, Kyoto, Japan). The average of the two measurements was used in the final analysis. Height was measured, while standing, in a participant without shoes, using a flexible tape measure (Hemostyl, Sulzbach, Germany). Body weight was also measured with individuals wearing light clothing or standing without shoes using a digital scale (Deluxe GBS-721; Seca Deutschland, Hamburg, Germany). Body mass index (BMI) was calculated as weight in kilograms divided by height in square meters (Kg/m^2). A flexible tape measure was used to measure the waist at the level connecting the two iliac crests. Blood Pressure was measured after a minimum of 5 minutes of rest and was recorded three times using a semi-automated device with the use of an appropriately sized cuff on the participant's left arm, the participant being seated.

The average of the last two readings was used in the analysis. HTN was defined as an SBP of at least 140 mmHg, or a DBP of at least 90 mmHg, or antihypertensive therapy, based on the list of medications prescribed to the participant [23]. The level of education and the socioeconomic level (NSE) was developed according to the 2013-2014 Demographic and Health Survey (EDS) in the DRC and was divided into categories: none or elementary education, lower secondary education, upper secondary education and higher and in low, medium and high level respectively for the education and the socioeconomic level (NSE) [24].

Physical activity was measured using the WHO Global Physical Activity Questionnaire (GPAQ) version 2 [25]. Then the participants were classified into three categories: low, moderate or high level of physical activity.

Body Mass Index (BMI): was obtained by the ratio of the weight in Kg on the height in square meters (Kg/m^2). BMI was then classified into four categories; underweight (BMI

<18.5 Kg/m$^{2)}$, normal (BMI 18.5-24.99 Kg/m^2), overweight (BMI 25-29.99 Kg/m^2) and obese (BMI 30 Kg/m^2) [26]. Waist circumference (WC) was used as a surrogate for abdominal obesity, defined as a WC value> 94 cm in men and> 80 cm in women [27].

Alcoholism was defined as consuming at least 20 g of alcohol per day or> 2 standard glasses of beer per day for men and> 1 standard glass of beer per day for women for at least one year [28].

Smoking has been defined as the common use of smoked or non-smoked tobacco [29].

Low fruit/vegetable intake was defined as taking less than 5 servings of fresh and/or cooked fruits/vegetables per day [30]. Two fasting venous blood samples (5 ml) were collected by investigators according to standard operating procedure.

Cholesterol, high density lipoprotein (HDL) and TG levels were determined using colorimetric test kits and analyzed using the ABX Pentra 400 chemical analyzer (HORBIA ABX, Montpellier, France).

High sensitivity CRP (hs-CRP) levels were determined in heparin plasma by particle enhanced immuno turbidimetry assay. Agglutinates of human CRP with latex particles were coated with anti-CRP monoclonal antibodies. The aggregates were determined by turbidimetry using the ABX Pentra 400 chemistry analyzer (HORIBA ABX).

Dyslipidemia was defined as total cholesterol (CT) ≥ 6.22 mmol/L and/or triglycerides (TG) ≥ 2.26 mmol/L and/or high density lipoprotein cholesterol (HDL-C) <1, 04 mmol/L and/or low density lipoprotein cholesterol (LDL-C) ≥ 4.14 mmol/L [31].

We calculated the atherogenic risk using the indices.

Castelli Risk Index (CIR):

1. Castelli Risk Index is based on three important parameters of lipid profile i.e. TC, LDLc and HDLc and it is classified into two; CIR-1 and CIR-11 [32].

CRI-1: CRI-I is based on the relationship of two important parameters of the lipid profile, TC and HDLc. Mathematically, it is estimated as;

$$CRI\text{-}I = TC/HDLc$$

CRI-II is calculated as the ratio of LDLc to HDLc. Mathematically, it is estimated as; CRI-II = LDLc/HDLc ratio.

2. Plasma atherogenic index (AIP) = atherogenic

Index of Plasma was proposed by Dobiasova and Frohlich in 2001. AIP is a logarithmic transformation ratio TG/HDLc [33].

$$AIP = Log10\ (TG/HDLc)\ ratio$$

3. Atherogenic Coefficient (AC) = Atherogenic is an indirect measure of cholesterol in the VLDLc lipoprotein fractions, IDLc and LDLc are referenced with the HDLc fraction. Mathematically, it is expressed as;

$$AC = (TC- HDLc)/HDLc\ [32\text{-}35].$$

Data were stratified by CRP concentrations divided into two levels, normal levels (hs-CRP <3 mg/l) and high levels (CRP 3 mg/l) [32, 36]. Uric acid was measured by enzymatic methods (ROCHE Cobas8000C701 chemistry analyzer, USA) Hyperuricemia was defined as a serum uric acid concentration> 6.0 mg/dL (> 360 µmol/L) in women [37, 38].

2.4. Data Analysis

Data were analyzed using SPSS version 21 software for Windows. They were expressed in mean standard deviations (SD) for continuous variables and in frequencies (n) and percentages (%) for categorical variables.

The percentages were compared using the chi-square test. Logistic regression was used to identify independent factors associated with hypertension. The threshold of statistical significance was set at a value of $p < 0.05$.

3. Results

A total of 252 respondents were included in this study..

Table 1 presents the general characteristics of the study population.

A female predominance with a sex ratio of 2.5F/1H. The majority were recruited in rural areas, ie 83.7%. 33.3% of the respondents were over 50 years old, As a background: 19.8%; 42.9%; 2%; 52.8% respectively for tobacco, Alcohol Known diabetes and physical inactivity but without any significant difference between urban and rural areas. The majority were NSE Low 63.5% and 65.1 respondents reported consuming a diet very high in cholesterol. Clinically and paraclinically, Obesity 63 (25.0%), Abdominal obesity 169 (67.1%), RCM 183 (72.6%), AIP High risk 155 (61.5%), AC High risk 148 (58.7%), Ac Uric Pathologique 57 (22.6%) had a statistically significant difference between rural and urban.

This table shows BMI (p = 0.001), TT (p = <0.001), TH (p = <0.001), TT/Height (p = 0.001) and Uric Acid (p = <0.001) values were significantly higher in urban than rural. AIP = Atherogenic index of plasma, CRI = Castelli's risk index, AC = Atherogenic coefficient.

The atherogenic risk was 61.5%; 58.7%; 41.3%; 39.3% and 21.8% respectively for AIP, AC, CRI-II, CT/TG and CRI-I (Figure 1). By comparing the CRP value and uric acid of non-hypertensive people with hypertensive ones, it emerges that hypertensive people had significantly (p = 0.001) higher CRP values than non-hypertensive people.

The probability of being associated with hypertension was almost times higher, respectively, AIP≥0.24 (p = 0.357) and Smoking (p = 0.693). It was almost 2 times higher, respectively, for Age≥50 years (p = 0.007), Physical inactivity (p = 0.015), Obesity (p = 0.103). It was almost 4 times higher, respectively, for Uric Acid> 420 mg/dl (p = 0.001) and CRP> 3 mg/dl (p = 0.015). In the end, it was 10 times higher for Subclinical atherosclerosis (p = 0.001) (Table 3).

4. Discussion

The results of this study show that there are several determinants of high blood pressure.

Older age was associated with hypertension in the present study. Our observation is in agreement with the results of previous studies carried out in the DRC which found that advanced age as one of the main determinants of hypertension in Congolese environment [10-22].

This same observation has also been made in several other studies carried out in Africa [39-41]. Subclinical atherosclerosis was also associated with hypertension in the present study. The association between subclinical atherosclerosis and hypertension is bidirectional. In fact, hypertension, through pressure overload (Laplace's law) and the deleterious effect of the activation of the sympathetic nervous systems and renin angiotensin on the vascular wall, will lead to oxidative stress, inflammation and endothelial dysfunction responsible for atherosclerosis. Abdominal overweight/obesity was associated with hypertension in the present study. This observation is in agreement with that made by Katchunga et al. [14] and Bayauli et al. [13] in the Congolese environment. Overweight/obesity can induce, through the release of adipokines (TNFα...), insulin resistance and subsequent hyperinsulinemia, the rise in BP is explained by several including stimulation of the sympathetic nervous systems and renin angiotensin, sodium hydroxide retention through stimulation of the Na +/H + antiport of the proximal convoluted tube of the kidneys [42].

Our study established a positive association between hypertension and elevated CRP [43].

While many studies have reported a higher CRP level in patients with prehypertension and hypertension compared to those with normal BP [44, 45], in many other studies elevated CRP appears to be a marker of risk for other CVD risk factors rather than an independent risk factor for hypertension [46]. Still others have found no association between CRP and HTN, despite a very high level of CRP. The absence of the association between CRP and hypertension in these patients could be explained in particular by the existence of infectious pathologies [47].

A large cohort study followed for a median of 7.8 years found that hs-CRP levels played an essential role in the subsequent development of hypertension, suggesting that hypertension was an inflammatory disorder [48] In addition, it was shown in another research followed for a median of 11 years that the incidence of hypertension could be predicted by several independent factors, not only CRP but also abdominal obesity and smoking behavior [49].

In this study, high uric acid levels were associated with high blood pressure. The result of this study in the literature corroborates those reports is consistent with previous studies [50, 51]. Several pathogenic mechanisms have been implicated in the association between uric acid and hypertension [52-54].

Although uric acid is an antioxidant in the extracellular environment, it causes oxidative stress in cells with inhibition of the endothelial nitric oxide pathway and activation of the renin-angiotensin system. These effects result in systemic and renal vasoconstriction and the development of salt-resistant hypertension. Over time, uric acid induces vascular smooth muscle cell proliferation and inflammation and can lead to irreversible damage to small kidney vessels, leading to kidney microvascular damage and subsequent elevation in blood pressure [54, 55]. Our study found that lipids, atherogenicity indices and hypertension. This finding is reported by several studies which have indicated a significant relationship between lipids, atherosclerosis and hypertension [56, 57].

In this study, the Castelli I (TC/HDL-C) and II (LDL-C/HDL-C) risk indices were assessed. These are very powerful risk indicators, which have a higher predictive value than data isolated from the lipid profile of CVD, because they reflect the alteration of a very important component of vascular risk, the decrease in the levels of the protective fraction of cholesterol, HDL -C, to the detriment of the increase in TC and more precisely of the risk fraction, LDL-C [58].

The results obtained in this study did not show a difference depending on the environment. This could be justified firstly, the lipid was not considered by age and secondarily by sex.

In multivariate analysis only AIP≥0.24 which emerged as a determinant of hypertension. Indeed, several studies to date have evaluated the relationship between unique serum lipid parameters and arterial stiffness, an increase in peripheral resistance and consequently an increase in blood pressure [59-61].

AIP (log 10 [triglyceride/HDL-C]) has recently been proposed as a higher index which better accounts for the interactions between the different lipid fractions, reflecting both the composition of plasma lipoproteins [62].

Compared to more traditional simple lipid parameters, AIP exhibits a normal distribution and is therefore better suited for mathematical modeling of key cardiovascular variables [63]. Thus, the addition of AIP in routine blood lipid reports will have a broad prospect of application in the assessment of arteriosclerosis in patients with hypertension.

The potential mechanisms regarding the relationship of PIA with arterial stiffness are not entirely clear. The value of AIP in assessing cardiovascular disease risk is probably related to its positive relationship with cholesterol levels, lipoprotein particle size, and lipoproteinemia [64]. AIP reflects the distribution of small dense low density lipoproteins (LDL) [65] and LDL levels are closely related to oxidative stress and inflammation [66].

Numerous studies have confirmed that oxidative stress and inflammation contribute to arterial stiffness via worsening endothelial dysfunction [67], promoting up regulation of elastin-degrading enzymes [68], resulting in the passage of smooth muscle cells from contractile phenotypes to synthetic phenotypes [69] and enhancing the expression of fibroblast-derived extracellular matrix metallopeptidase [69]. These above studies may explain why AIP is positively correlated with arterial stiffness.

5. Conclusion

In summary, we found that age> 50 years, physical inactivity, CRP, uric acid, AIP were positively correlated with hypertension. These results indicate that AIP may represent a valuable surrogate predictor of arterial stiffness in hypertensive patients with Boma.

Author's Contribution

All the authors contributed to the realization of this study.

Conflict of Interest

The authors declare no conflict of interest.

Acknowledgements

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Appendix

Table 1. *Sociodemographic parameters by place of residence.*

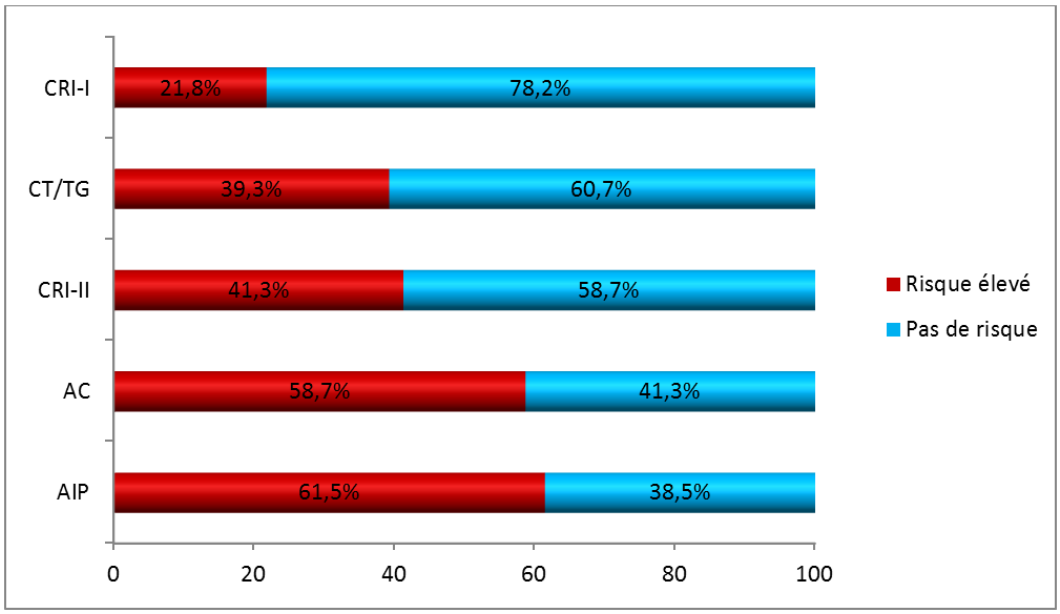
Variables	All (n = 252)	Urban setting (n=41)	Rural (n = 211)	p
Gender, n (%)				0,329
Female	180 (71,4)	31 (75,6)	149 (70,6)	
Male	72 (28,6)	10 (24,4)	62 (29,4)	
Age≥50 years	84 (33,3)	17 (41,5)	67 (31,8)	0,153
Tobacco	50 (19,8)	7 (17,1)	43 (20,4)	0,404
Alcohol intake,	108 (42,9)	17 (41,5)	91 (43,1)	0,492
Known diabetes	5 (2,0)	1 (2,4)	4 (1,9)	0,592
Physical inactivity,n	133 (52,8)	24 (58,5)	109 (51,7)	0,263
SES				0,005
Low	160 (63,5)	25 (61,0)	135 (64,0)	
Middle	66 (26,2)	6 (14,6)	60 (28,4)	
High	26 (10,3)	10 (24,4)	16 (7,6)	
Power type				0,900
Improved cholesterol	2 (0,8)	0 (0,0)	2 (0,9)	
No optimal in cholesterol	86 (34,1)	15 (36,6)	71 (33,6)	
Severe in cholesterol	164 (65,1)	26 (63,4)	138 (65,4)	
Vegetable & fruit consumption				0,125
Poor	188 (74,6)	34 (82,9)	154 (73,0)	
Raised	64 (25,4)	7 (17,1)	57 (27,0)	
ATS	35 (13,9)	5 (12,2)	30 (14,2)	0,478
Overweight	69 (27,4)	11 (26,8)	58 (27,5)	0,549
Obesity	63 (25,0)	20 (48,8)	43 (20,4)	<0,001
Abdominal obesity	169 (67,1)	34 (82,9)	135 (64,0)	0,012
RCM	183 (72,6)	35 (85,4)	148 (70,1)	0,031
RAH	142 (56,3)	27 (65,9)	115 (54,5)	0,121
HTN t	131 (52,0)	25 (61,0)	106 (50,2)	0,138
CRI-I High risk	55 (21,8)	8 (19,5)	47 (22,3)	0,437
CRI-II High risk	104 (41,3)	16 (39,0)	88 (41,7)	0,445
AIP High risk	155 (61,5)	20 (48,8)	135 (64,0)	0,027
AC High risk	148 (58,7)	21 (51,2)	127 (60,2)	0,018
CT/TG High risk	99 (39,3)	12 (29,3)	87 (41,2)	0,051
TC High	59 (23,4)	7 (17,1)	52 (24,6)	0,201
TG High	90 (35,7)	11 (26,8)	79 (37,4)	0,131
LDL High	91 (36,1)	13 (31,7)	78 (37,0)	0,325
HDL-c low	164 (65,1)	23 (56,1)	141 (66,8)	0,059
Ac Uric Pathological	57 (22,6)	16 (39,0)	41 (19,4)	0,007
Pathological CRP	86 (34,1)	13 (31,7)	73 (34,6)	0,435

Table 2. *Clinical features.*

Variables	Tous (n=252)	Urban (n=41)	Rural (n=211)	p
AGE	40,9±15,4	42,8±13,7	40,5±15,7	0,378
SBP	132,5±26,2	138,4±21,4	131,4±26,9	0,118
DBP	88,3±17,2	92,7±15,2	87,5±17,5	0,075
PAM	103,1±19,1	107,9±16,5	102,1±19,4	0,073
PP	44,2±16,9	45,8±12,8	43,9±17,6	0,542
HR	81,9±11,4	84,4±11,1	81,5±11,4	0,133
IMC	27,1±8,4	31,2±9,9	26,3±7,8	0,001
TT	91,5±14,5	99,2±12,7	90,1±14,3	<0,001
TH	103,0±16,7	111,6±16,2	101,4±16,3	<0,001
TT/TH	0,90±0,15	0,90±0,13	0,90±0,15	0,998
TT/Size	0,57±0,10	0,61±0,09	0,56±0,10	0,001

Variables	Tous (n=252)	Urban (n=41)	Rural (n=211)	p
Uric acid	317,8±113,7	381,7±114,9	305,36±109,5	<0,001
CT	6,53±2,7	4,4±1,0	6,95±2,9	0,570
Triglyceride	3,50±2,30	1,39±0,55	3,91±2,52	0,523
LDL-	4,72±2,61	2,96±0,92	5,06±2,8	0,638
HDL-c	1,12±0,49	1,13±0,43	1,11±0,50	0,786
CRP	4,54±1,34	4,57±2,26	4,53±1,07	0,857
CRI_I	6,29±2,3	4,37±1,88	6,66±2,52	0,561
CRI_II	4,57±2,25	3,1±1,8	4,85±2,46	0,650
AIP	0,17±0,28	0,09±0,19	0,18±0,29	0,058
AC	5,23±2,3	3,37±1,9	5,66±52,5	0,561

Data are expressed as mean ± standard deviation, absolute (n) and relative (in percent) frequency. Abbreviations: BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Blood Pressure; PP: Pulse Pressure; HR: Heart Rate; bpm: Beat Per Minute. AIP=Atherogenic index of plasma, CRI=Castelli's risk index, AC=Atherogenic coefficient.



AIP=Atherogenic index of plasma, CRI=Castelli's risk index, AC=Atherogenic coefficient.

Figure 1. *Comparison of risk of atherogenicity according to the indicess.*

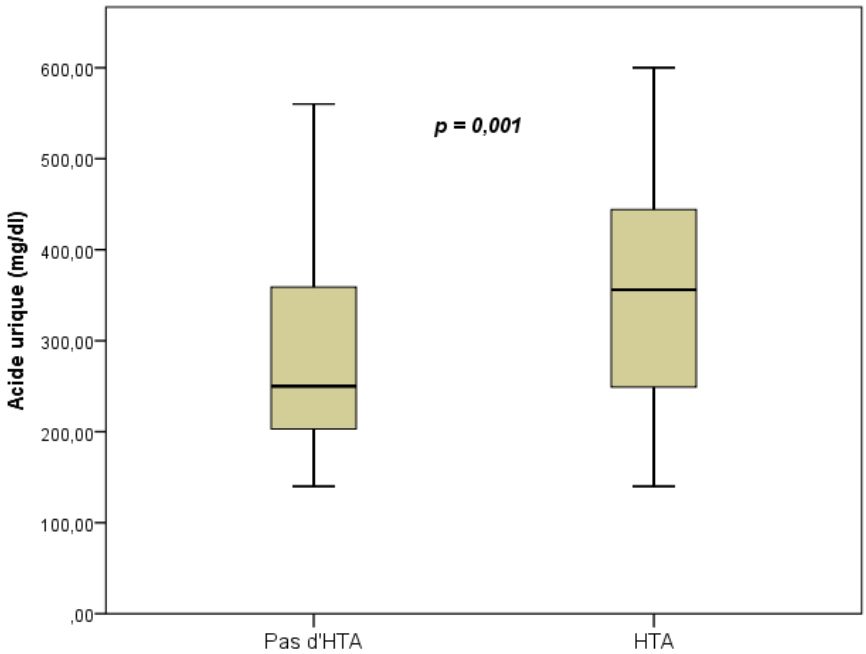


Figure 2. *Relation of hypertension and uric acid.*

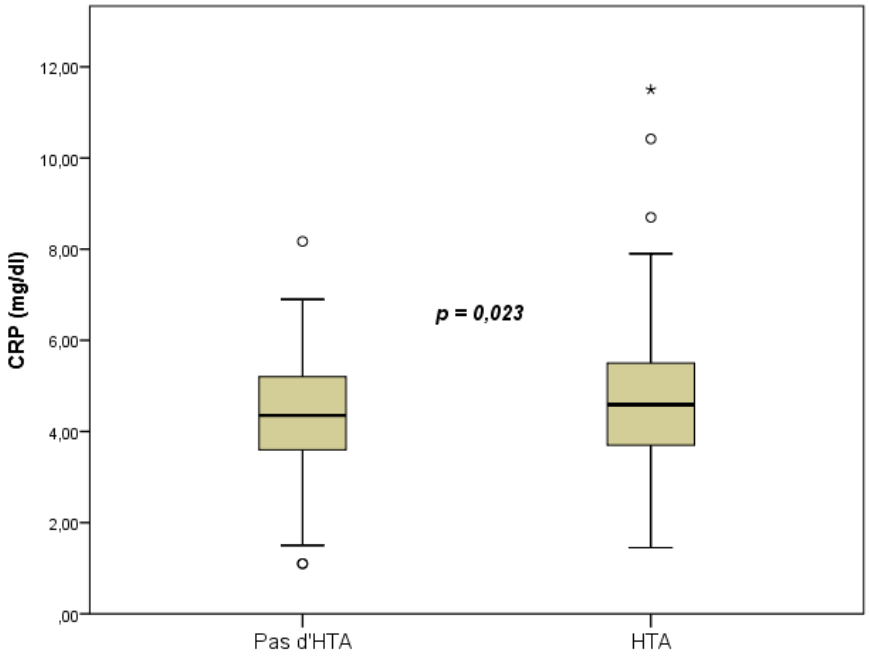


Figure 3. *HTN and CRP relationship.*

Table 3. *Clinico-biological determinants of hypertension in the study population.*

Variables	Univariate analysis		Multivariate Analysis	
	p	OR (IC95%)	p	ORa (IC95%)
Age≥50 ans				
No		1		1
Yes	0,000	3,7 (2,1-6,6)	0,007	2,4 (1,3-4,6)
Tobacco				
No		1		1
Yes	0,046	1,9 (1,1-3,5)	0,693	1,2 (0,6-2,4)
Known diabetes				
No		1		1
Yes	0,024	3,9 (1,4-7,3)	0,027	2,3 (1,1-2,9)
Physical inactivity				
No		1		1
Yes	0,013	1,9 (1,1-3,1)	0,015	1,9 (1,1-2,7)
ATS				
No		1		1
Yes	0,000	12,7 (3,8-42,8)	0,001	10,0 (2,7-16,7)
Obesity				
No		1		1
Yes	0,036	1,9 (1,04-3,4)	0,103	1,7 (0,9-3,3)
AIP≥0,24				
No		1		1
Yes	0,025	3,3 (1,8-5,2)	0,357	1,3 (0,7-2,3)
Uric Ac >420 mg/dl				
No		1		1
Yes	0,000	4,2 (2,1-8,3)	0,001	3,7 (1,7-8,3)
CRP >3 mg/dl				
No		1		1
Yes	0,042	2,2 (1,1-2,9)	0,015	3,6 (1,3-7,2)

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