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Effects of cardiometabolic risk factors on blood pressure in outpatients at Sominé DOLO hospital, Mopti, Mali

Modibo Coulibaly[1,*], Lamine Sidibé[1], Djibril Traoré[2], Mamoudou Barry[3], Valentin Sagara[1], Bakary Maiga[4]

[1]*Dept. of Biomedical Laboratory, University of Science, Techniques and Technologies of Bamako/Sominé DOLO Hospital, Mopti, Mali*
[2]*Dept. of General Surgery, University of Science, Techniques and Technologies of Bamako/Sominé DOLO Hospital, Mopti, Mali*
[3]*Dept. of Biomedical Laboratory, Sominé DOLO Hospital, Mopti, Mali*
[4]*Dept. of Immunology, Faculty of Medicine and Odonto-Stomatology of the USTTB, Bamako, Mali*

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ABSTRACT

Background: Blood cardiometabolic impairments are associated with high blood pressure which is a pivot cardiovascular risk factor. The objective of this study was to assess cardiometabolic risk factors rates in subjects with high blood pressure in the steady state.

Materials and Methods: A total of 292 subjects, 107 cases and 185 controls were enrolled in cross-sectional study. Clinical and biological data were assessed during visits and after overnight fasting. Data were analyzed on R. A p-value < 0.05 was considered for statistical significance.

Results: Univariate analysis showed that age > 50 years, visceral obesity, metabolic syndrome and *hs*-CRP ≥ 3 mg/L were significant predictors of high blood pressure: OR = 2.1, 95% CI [1.3-3.5], p = 0.003; OR = 1.6, 95% CI [1.0-2.6], p = 0.05; OR = 3.3; 95% CI [2.0-5.4], p < 0.001; OR = 16.8; 95% CI [9.4-31.4], p < 0.001, respectively. Multivariate analysis showed a positive association between obesity, metabolic syndrome, *hs*-CRP and high blood pressure: aOR = 2.29; 95% CI [1.14-4.69], p = 0.02; aOR = 3.47; 95% CI [1.64-7.61], p = 0.001; aOR = 18.10; CI, 2.5% to 95% [9.40-36.99], p < 0.001, respectively. In contrast, female sex was negatively associated with high blood pressure aOR = 0.31; CI, 95% CI [0.13-0.72], p < 0.008.

Conclusion: Prevention policies should take into account blood cardiometabolic level for subjects with high blood pressure even though in the steady-state.

1. Introduction

According to the World Health Organization (WHO), cardiovascular diseases (CVDs) are the leading cause of death globally in 2021. An estimated 17.9 million peoples died from CVDs in 2019, representing 32% of all global deaths. The most important behavioral risk factors of heart disease and stroke are unhealthy diet which may show up in individuals as raised blood pressure.[1] Alongside this, there are underlying cardiometabolic risk factors (CMRF) which play a pivotal role in the development of high blood pressure (HBP) that also in turn plays a central role in the pathogenesis of CVDs. Among these CMRF, there are traditional risk factor and modern risk factors. One of the modern risk factor is the high sensitive C reactive protein (*hs*-CRP), which constitutes an independent CVDs risk factor.[2] According to the harmonized criteria from the International Diabetes Federation (IDF) and

* *Corresponding author*.
E-mail address: modibocoulibaly@yahoo.com (M. Coulibaly).

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the American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI), metabolic syndrome (MetS) is defined as abdominal obesity, insulin resistance, atherogenic dyslipidemia and hyperglycemia.[3] MetS is a cluster of comorbid states which could lead to a serious health condition that puts people at higher risk of heart disease, diabetes, stroke and diseases related to fatty buildups in artery walls atherosclerosis. Based on the AHA/NHLBI 2009 criteria, we reported in a previous study a prevalence of 23.6% MetS.[4] In the meantime, a new definition has been proposed.[5] In the new definition, HBP is considered a key feature of MetS.[6] Also, MetS is accompanied by a pro-inflammatory state which explains the elevation of various inflammatory markers such as *hs*-CRP, insulin resistance which results in increased obesity-induced systemic oxidative stress and activates downstream inflammatory cascades, leading to tissue fibrosis, atherogenesis, and, subsequently CVD.[7] Low density lipoprotein cholesterol (LDL-C) level < 70 mg/dL and *hs*-CRP level < 2.0 mg/L with a statin in monotherapy or in combination with ezetimibe has been associated with significant CVD risk reduction.[8,9] However, the impact of CMRF in subjects with HBP in the steady-state is not well-known, The objective of this study was to assess CMRF rates on subjects with HBP in the steady-state.

2. Materials and Methods

2.1. Sample size and subjects inclusion criteria

The sample size was computed according to the proportion of outpatients consultation in cardiology in the department of medicine data not published (3.96%). By using the Daniel Schwartz formula N = $\frac{Z^2 * pq}{i^2}$ with a margin error of 5% and a confidence level of 95%. The number of steady-state outpatient required for the study was 292. So two hundred ninety-two (292) outpatient in steady state aged from 25 to 70 years, who attend our department of medicine were enrolled in a cross-sectional study by consecutive aleatory recruitment from April to November 2017. The inclusion criteria were as follow: steady state outpatients aged from 25 to 70 years, and having HBP or not. Pregnant women and patients with apparent pathologies were not enrolled.

2.2. Metabolic syndrome criteria and high blood pressure definition

MetS was defined according to Dobrowolski P et al newly proposed criteria for the MetS.[5]

According to this new point, a person has MetS when:

1. **There is abdominal obesity** (waist circumference greater than 88 cm in women and 102 in men) plus two of the following additional diagnostic criteria.
   (a) **Prediabetes or diabetes**: fasting glucose ≥ 100 mg/dL or ≥ 140 mg/dl after 120. min in oral glucose tolerance test or HbA1C ≥ 5.7% or on glucose-lowering drug treatment,
   (b) **Elevated non-HDL cholesterol level**: non-HDL cholesterol level ≥ 130 mg/dl or on lipid-lowering drug treatment,
   (c) **High normal blood pressure or hypertension**: systolic blood pressure (SBP) ≥ 130 and/or diastolic blood pressure (DBP) ≥ 85 mm Hg (in-office measurement) or SBP ≥ 130 and/or DBP ≥ 80 mm Hg (home measurement) or on anti-hypertensive treatment.
2. **There is a b ody mass index (BMI):** ≥ 30 kg/m2 plus two of the following additional diagnostic criteria
   (a) **Prediabetes or diabetes**: Fasting glucose ≥ 100 mg/dL or ≥ 140 mg/dl after 120. min in oral glucose tolerance test or HbA1C ≥ 5.7% or on glucose-lowering drug treatment,
   (b) **Elevated non-HDL cholesterol level**: non-HDL cholesterol level ≥ 130 mg/dl or on lipid-lowering drug treatment,
   (c) **High normal blood pressure or hypertension**: SBP ≥ 130 and/or DBP ≥ 85 mm Hg (in-office measurement) or SBP ≥ 130 and/or DBP ≥ 80 mm Hg (home measurement) or on anti-hypertensive treatment.
3. Other definitions
   (a) **High blood pressure:** SBP >140 mm Hg and/or DBP ≥ 90 mm Hg or on anti-hypertensive treatment.
   (b) **High triglycerides**: The triglyceride level is equal to or greater than 1.7 mmol/L, the equivalent of 150 mg/dL.
   (c) **Low HDL cholesterol level**: the HDL cholesterol level is less than 1.03 mmol/L (40 mg/dL) in a man and 1.29 mmol/L (50 mg/dL) in a woman.
   (d) **High fasting glucose**: fasting venous blood glucose is equal to or greater than 5.6 mmol/L (100 mg/L).

2.3. Study population and design

The study population was divided into two groups based on their blood pressures which were accurately measured manually by two different cardiologists using the device Vaquez-Laubry Nano^{TM}, Spengler (Lyon, France). Subjects with SBP < 140 mmHg and or DBP < 90 mmHg were considered as having normal blood pressure. The first group was the high blood pressure (HBP) group where the SBP is ≥ 140 and or the DBP > 90 mmHg and/or on anti-hypertensive treatment and the second was the group with normal or hypo blood pressure called non-high blood pressure (NHBP). Subjects who were pregnant, underlying severe diseases, or had physical

impairments, and did not like to participate in the study were not enrolled. Demographic and clinical data: age, sex, smoking statute, alcohol consumption, height, weight, body mass index (BMI), waist size, hip circumference, waist circumference, SBP, and use of anti-hypertension or anti-diabetes treatments, were measured. Blood samples were also collected to perform the following laboratory tests: total cholesterol (TC), triglyceride (TG), HDL cholesterol (HDL-C), direct LDL-C, *hs*-CRP, creatinine, fasting uric acid, and fasting glucose.

2.4. Laboratory measurements

Fasting plasma samples were stored at -80 °C and never thawed until use. The study tests were performed on the cobas c111analyzer^{TM}, Roche Diagnostics (Rotkreuz, Switzerland). This device uses photometric analysis and includes an optional ion-selective electrode (ISE) module. It is a continuous and random access analyzer designed for the in vitro determination of clinical chemistry and electrolyte parameters in fluid samples (serum, plasma, urine and whole blood). It uses the same reagent formulations as leading cobas clinical chemistry analyzers. This allows for standardization of results, an important necessary recommendation to enable comparisons.

2.4.1. Accuracy, intermediate fidelity and specifications of laboratory tests assays

For the validation of laboratory tests, we used two ranges of PreciPath HDL-C/ LDL-C Control (4 x 3 mL) Roche Diagnostics (Rotkreuz, Switzerland) Ref #11778552122 and PreciControl ClinChem Multi 2 (4 × 5 mL) Roche Diagnostics (Rotkreuz, Switzerland) Ref #05947774 190. These two control levels were included at the start and end of each series of assay. The assay methods were only declared in the state of the art after the two levels of control were included in the reference intervals of the manufacturer and or respecting sigma metrics rules. If not, a corrective action or calibration with a calibrator for automated systems (C. F.A.S.) Lipids (3 × 1 mL) or C. F.A.S. Protein for hs-CRP had been undertaken in order to have precision and a correct intermediate fidelity. Plasma level of TC was performed by using Cholesterol Gen.2, 4 × 100 tests cobas c111 ^{TM} Roche Diagnostics (Rotkreuz, Switzerland) (CHOD/PAP Gen. 2, liquid / liquid) and TG were determined by standard enzymatic methods using assay kits from Roche Diagnostics Triglyceride 4 x 5 mL cobas c111^{TM} (GPO, liquid / liquid) Roche Diagnostics (Rotkreuz, Switzerland) Ref #04657584190. Direct HDL-C level was measured with a homogeneous, automated assay provided by HDL-C Gen.4 (350 tests) cobas c111^{TM} (CHER, CHOD/POD Gen. 4, liquid/liquid) Roche Diagnostics (Rotkreuz, Switzerland) Ref # 07 7589 4. Direct LDL-C concentration was measured with LDL-C Gen.3 (200 tests) cobas c111^{TM} (Detergent, CHOS/POD Gen. 3, liquid/liquid) Roche Diagnostics (Rotkreuz, Switzerland) Ref # 07 75657. *hs*-CRP was measured with a high-sensitivity nephelometric immunoassay Cardiac C-Reactive Protein (Latex) High Sensitive (2 × 50 tests) Roche Diagnostics (Rotkreuz, Switzerland). Fasting blood glucose levels were measured by Glucose HK 4x17.2, (HK/G-6-PDH**,** liquid / liquid) Roche Diagnostics (Rotkreuz, Switzerland) Ref # 04657527 190.

2.5. Data analysis

Data were captured in Excel and analyzed by Ri386 version 4.0.3. Quantitative data normality were assessed by using the Cholmogorov-Smirnov test. Descriptive statistics were determined for all variables and expressed as Mean ± standard deviation (SD) for variables with normal distribution or median inter quartile range (IQR) for none normally distributed variables. For normally distributed variables, Student's t-test was applied to compare the two groups. The Kruskal- Wallis test was used for the comparison of continuous non-normal variables means. For categorical parameters, one-proportion Z-tests were used to test whether the proportions of clinical or biochemistry variables were greater according to the blood pressure level (one-tailed test). The generalized linear model (GLM) analyses were used to assess the association of clinical and biochemistry variables (independent variables) with blood pressure (dependent variable). The univariate analyses analysiswere performed to identify simple associations between independent variables and blood pressure whereas multivariate analysis were used to identify which independent variables were significantly associated with dependent variables after adjustment for confounding variables. A p-value < 0.05 was considered as being statistically significant.

2.6. Ethics statement

Participant informed consent was obtained during the sampling period of the previous study. The ethical clearance was obtained from the hospital institution's ethical committee which also granted the permission to use the laboratory's patient records. All patient records were handled according to the declaration of Helsinki and confidentiality was maintained by removing all patient identifiers.

3. Results

A total of 292 outpatients were enrolled, of which 114/292 (39.7%) were women. The medians of age and *hs*-CRP were 43 years, interquartile range (IQR) = 20.3 years and 2.7 mg/L, IQR = 35.2 mg/L, respectively. The proportions of Age > 50 years, smokers, BMI was ≥ 30 kg/m^2, LDL-C ≥ 3.5 mmol/L, MetS and *hs*-CRP ≥ 3 mg/dL were 114/292 (39.0%), 41/292

(14.0%),86/292(29.4%), 24/292(8.2%), 135/292(46.2%), 125/292(42.8%), respectively (Table 1). The proportion of age over 50 years (86.0% versus 5.1%; $p < 0.0001$); visceral obesity (57.8% versus 10.7%; $p < 0.0001$); smokers (84.4% versus 28.7%; $p < 0.0001$); BMI ≥ 30 kg/m^2 (66.3% versus 24.3%; $p < 0.0001$); LDL-cholesterol was ≥ 3.5 mmol/L (79.2% versus 32.8%; $p < 0.0002$); Fasting blood glucose (FBG) levels ≥ 5.6 mmol/L (57.1% versus 31.0%; $p = 0.0002$); MetS (68.1% versus 9.6%; $p < 0.0001$) and *hs*-CRP concentration ≥ 3 mg/L (74.4% versus 8.4%; $p < 0.0001$) were significantly higher in the group of HBP compared to the control group. In contrast, the proportion of normal or elevated HLD-C (19.8% versus 48.5%) was significantly lower in the groups of HBP ; $p < 0.0001$ (Table 2). Univariate analysis shows positives associations between age > 50 years, odds ratio = 2.1, CI 95% [1.3-3.5], $p = 0.003$; visceral obesity odds ratio = 1.6, CI 95% [1, 0-2.6], $p = 0.05$; MetS odds ratio = 3.3; CI 95% [2.0-5.4], $p < 0.0001$; *hs*-CRP concentration ≥ 3 mg/L odds ratio = 16.8; CI 95% [9.4-31.4], $p < 0.0001$ and HBP. There was no significant difference between the proportions of sex, smoker, alcohol consumption, BMI, elevated creatinine, TC, TG, HDL-C, LDL-C, FBG and uric acid in the HBP group compared to the control group (Table 3). The multivariate analysis summarized in Figure 1 shows a negative association between the female sex and HBP odds ratio = 0.31; CI 95% [0.13-0.72], $p < 0.008$. On the overhand, positive associations were noted between BMI ≥ 30 kg/m^2 odds ratio = 2.29; CI 95% [1.14-4.69], $p = 0.02$; Met odds ratio = 3.47;CI 95% [1.64-7.61], $p = 0.001$, *hs*-CRP odds ratio = 18.10; CI 95% [9.40-36.99], $p < 0.001$and the HBP.

4. Discussion

According to WHO data, HBP is a leading heritable and modifiable risk factor for CVDs like stroke and coronary artery disease. It is estimated to be 1.28 billion adults aged 30-70 years worldwide in 2021.[10] BP is determined by complex interactions between life-course exposures which are known as modifiable factors and genetic background.[11] We conducted a cross-sectional study on outpatients consulting in the medical department of the Sominé DOLO hospital of Mopti, Mali to assess the proportions of modifiable factors between the HBP group and the control. The sex-ratio in our series was 1.5. Ekou A et al also found a male predominance with a sex-ratio of 1.4 in patients practicing blood pressure self-measurement in a population of hypertensives in sub-Saharan Africa[12] whereas Mipinda JB et al reported a female predominance in patients seen as an outpatient at the Cardiology Department of the Libreville University Hospital (Gabon) with a sex ratio of 0.82.[13]

The median age in our series was 43 years with interquartile range (IQR) = 20.3 years which was higher than those reported by Mipinda et al who found a median age of 41 years. This difference results in the different age range in the two series: 25-70 years in our series and 17-87 years in the Mipinda series. In our series, the overweight (BMI ≥ 30 kg/m^2) proportion of patients was 29.4% with an average body mass index (BMI) of 26.10 kg/m^2. These results are below those obtained during the screening for CVD risk factors in a cohort of 270 Cameroonians who reported that 37% of the patient was obese.[14]

The proportions of the smoker in our patients 14.0% was higher than that of the survey on the prevalence of CVDs risk factors in the general population of Saint-Louis (Senegal) with a prevalence of 5.8%.[15] Our study reported a high rate of alcohol consumption up to 13.0% which was highest than those reported by Berthé et al.[16] 1.1%. The high prevalence of alcohol consumption in our series could be explained by the fact that Mopti has been plagued by insecurity and the war since 2012 which may have adverse effects on behavior like alcohol consumption by stress and exposure to terrorists according to Massey Z et al.[17]

HBP proportion was 36.6% in our series, Diop TM et al reported that the HBP was the most common cardiovascular risk factor with 54% in the department of emergency at the teaching hospital of Gabriel Touré, Mali.[18] The HBP was more prevalent in our series than that of Bâ HO et al who reported 21.1 and 24.7% in a rural and urban setting, respectively.[19] The high prevalence of HBP in our series could be explained by the different study design, our series was made up with patients who consulted in the department of medicine while the study of Bâ HO was a population based study. However, Baldé MD et al reported a high prevalence of 43.6% hypertension at 43.6% in an outpatients study at Foutah-Djallon in Guinea.[20]

Elevated FBG (> 5.6 mmol/L) was 19.9% in our series. Lower prevalences of FBG were reported by Zaoui S et al, 14.2% in an Algerian series.[21] Pessinaba S et al, 10.4% in Senegalese series.,[15] Al-Mawali A et al, 16% in Sultanate of Oman.[17] In contrast, Solomon S et al reported a higher prevalence 32.6% of FBG in a study of disease burden and associated risk factors for MetS among adults in Ethiopia.[22]

The prevalence of the MetS was 46.2%, this result was higher than that of Touré K et al who reported 27.7% of MetS with a female predominance of 30.8% compared to all employees in the workplace during the annual medical visit.[23] Also, our MetS proportion was higher than those reported by Daouas A et al.[24] who found that the prevalence adjusted for age and sex of the MetS was 36.5% CI 95% [33.0%-38.9%] according to the IDF definition and 23.0% CI 95% [20.4%-25.6%] according to that of NCEP-ATP III. These differences could be explained by the use of different criteria for the definition of the MetS, one according to the NCEP-ATP III criteria and the other according to the FID criteria and the intrinsic characteristics of the samples. In contrast, a higher proportion of MetS than

Variable		N	Odds ratio	p
IYEARS	≥ 50 Years	114	Reference	
	< 50 Years	178	0.64 (0.33, 1.24)	0.186
SEX	Male	116	Reference	
	Female	176	0.31 (0.13, 0.72)	0.008
SMOKER	Non-smoker	251	Reference	
	Smoker	41	1.03 (0.30, 3.51)	0.962
ALC	Non-alcoholic	254	Reference	
	Alcohol consumption	38	0.77 (0.21, 2.71)	0.693
WS	Normal WC	131	Reference	
	Visceral obesity	161	1.88 (0.87, 4.16)	0.111
IBMI	Hypo or normo BMI	206	Reference	
	Obesity	86	2.29 (1.14, 4.69)	0.021
ICREA	Hypo or normo CREAT	258	Reference	
	High CREAT	34	1.29 (0.43, 3.80)	0.648
ICHOL	Hypo or normo CHOL	280	Reference	
	High CHOL	12	1.05 (0.09, 11.14)	0.968
ITRIG	Hypo or normo TRIG	257	Reference	
	High TRIG	35	1.25 (0.45, 3.41)	0.663
IHDL	Hypo or normo HDL	171	Reference	
	High HDL	121	1.18 (0.59, 2.36)	0.646
ILDL	Hypo or normo LDL	268	Reference	
	High LDL	24	1.53 (0.29, 7.81)	0.608
IGLU	Hypo or normo GLU	229	Reference	
	High GLU	63	0.96 (0.42, 2.16)	0.926
IUA	High UA	44	Reference	
	Hypo or normo UA	248	0.96 (0.36, 2.58)	0.930
MetSyn	Non-MetS	157	Reference	
	MetS	135	3.47 (1.64, 7.61)	0.001
ICRP	Hypo or normo hs-CRP	167	Reference	
	High hs-CRP	125	18.10 (9.40, 36.99)	<0.001

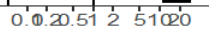

Fig. 1: Multivariate analysis by using blood pressure as dependent variable and clinical and biochemistry variables as independent variables. ALC: Alcohol; WS: Waist Size, WC: Waist Circumference, BMI: Body Mass Index, CREAT: Creatinine, CHOL: CHOL: Cholesterol, TRIG: Triglycerides, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, GLU: Glucose, UA: Uric Acid, MetS:Metabolic Syndrome, *hs*-CRP: High-Sensitivity C Reactive Protein

ours was reported by Sudip DB et al in a cross-sectional study who reported 48% of MetS in their series.[25]

The *hs*-CRP is a protein of the acute phase of the inflammation process and has been reported to be involved in the pathogenesis of CVDs.[26,27] In our series, the median of *hs*-CRP was 2.7 mg/L with IQR = 35.2. Moreover, subjects with high *hs*--CRP level (> 3mg/L) accounted for 42.8% in our series. *hs*-CRP level increases during the pathogenesis of atherosclerosis by focal inflammation, stimulated by oxidized LDL, hyperlipidemia, smoking and diabetes. Furthermore, Xue Q et al reported that both higher baseline *hs*-CRP and longitudinal *hs*-CRP increases were associated with higher risks of incident MetS. Thus, patients with high *hs*-CRP levels may need to be closely monitored for future risk of MetS.[28]

The proportions of the following subjects were significantly higher in HBP group than the control: age > 50 (86.0% versus 5.1%; p < 0.001); visceral obesity (57.8% versus 10.7%; p < 0.001); smokers (84.4% versus 28.7%; p < 0.001); obese (66.3% versus 24.3%; p < 0.001); LDL-C ≥ 3.5 mmol/L (79.2% versus 32.8%; p < 0.001); FBG levels ≥ 5.6 mmol/L (57.1% versus 31.0%; p = 0.001). These results are in line with Tofano RJ et al who reported that hypertensive individuals had significantly higher glycaemia (124.14 ± 45.33 mg/dL) or diabetes miletus, higher values of triglycerides (195.27 ± 74.52 mg/dL), waist circumference (98.52 ± 12.52 cm), body mass index (29.99 ± 1.41 kg/m^2).[29] Moreover, Tang N et al reported that people with dyslipidemia had a higher risk of hypertension than those with normal lipids OR = 3.05, 95% CI [2.36–3.90] and add that there was a significant potentiating interaction effect between overweight or obesity and dyslipidemia.[29]

However, the elevated HLD-C proportion was lower in HBP group compared to the control group (19.8% versus 48.5%; p < 0.001). This result was confirmed by Hwang YC et al. who reported in a longitudinal study that among the HDL-C subclass, HDL2 cholesterol was inversely associated with hypertension incidence, but both total and HDL3 cholesterol were not. In addition,

Table 1: Proportion of clinical and cardiometabolic risk factors among study subjects

Clinical and Cardiometabolic risk factors	All cases (n = 292)
Median Age (years)	43 [IQR = 20.3]
Age > 50 years	114(39.0%)
Male patients, %	116 (39.7%)
Visceral obesity WC ≥ 102 cm men, ≥ 88 cm women, %	161 (55.1%)
Smoking, yes, %	41 (14.0%)
Alcohol consumption, %	38 (13.0%)
BMI > 30 Kg/m2	86(29.4%)
HBP ≥ 140 mm/Hg and or ≥ 90 mm/Hg%	107 (36.6%)
CREAT > 110 men, > 90 μmol/L women	34(11.6%)
TC ≥ 5.0 mmol/L, %	59 (20.2%)
TG ≥ 1.7 mmol/L, %	50 (17.1%)
LDL-C ≥ 3.5 mmol/L, %	24 (8.2%)
HDL-C > 1.03 mmol/L in men, > 1.29 mmol/L in women, %	122 (41.8%)
Glucose > 5.6 mmol/L, %	58 (19.9%)
Uric Acid ≥ 360 mmol/L, ≥ 420 mmol/L, %	44 (15.1%)
MetS, %	135 (46.2%)
Median *hs*-CRP (mg/L)	2.7 [IQR = 35.2]
hs-CRP, ≥ 3 mg/L, %	125(42.8%)

Data are mean ± SD, percentage, or median value [interquartile range].
WC: waist circumference, HBP: High Blood Pressure, TG, triglycerides; HDL-C, high-density lipoprotein-cholesterol; LDL-C, Low-density lipoprotein-cholesterol; MetS, metabolic syndrome; *hs*-CRP, C-Reactive Protein, HBP, high blood pressure.

HDL2/HDL cholesterol was inversely associated with future hypertension risk.[30]

Nevertheless, according to Cho KH et al, it should be kept in the mind that apart from factors such as age and sex, HDL-C quality depends on the features of protein and lipid content, extent of oxidation, and the extent of glycation. Also, the functionality of HDL-C represents several performance metrics of HDL, such as antioxidant, anti-inflammatory, and cholesterol efflux activities. Finally, the quantity and quality of HDL can change during one's lifetime, depending on infection, disease, and lifestyle, such as dietary habits, exercise, and smoking.[31]

The MetS (68.1% versus 9.6%; p < 0.001) and *hs*-CRP ≥ 3 mg/L (74.4% versus 8.4%; p < 0.001) proportions were significantly higher in subjects with HBP compared to the control group. Hu J et al in a multistage sampling reported that the MetS prevalence and the number of metabolic components ≥ 3 were significantly higher in the hypertensive than in normotensives.[32] According to Tofano et al., when compared with patients with low CRP (< 0.1 mg/dL), those with a high CRP (≥ 0.1 mg/dL) had a significantly higher prevalence of visceral obesity, elevated triglyceride, lower HDL-C, hypertension, impaired FBG, and a higher prevalence of MetS. Moreover, most of the hypertensive patients (93.33%) presented with MetS and were related to the presence of more severe lesions in the arteries and had passed through more invasive procedures like angioplasty and surgery.[33]

In contrast, we did not find significant differences in the proportions of gender, alcohol consumption, elevated TC, TG, uric acid, creatinine and blood pressure when the HBP group was compared to the control group.

The univariate analyses showed that age > 50 years OR = 2.1, CI 95% [1.3-3.5], p = 0.003; visceral obesity OR = 1.6, CI 95% [1, 0-2.6], p = 0.05; MetS OR = 3.3; CI 95% [2.0-5.4], p < 0.001; *hs*-CRP ≥ 3 mg/L OR = 16.8; CI 95% [9.4-31.4], p < 0.001 were significantly HBP predictors. Hwang et al also reported during 10 years of follow-up, that age, diabetes, waist circumference, SBP, DBP, FBG, insulin resistance index, TC LDL-C, and visceral adipose tissue were significant predictors for incident hypertension in an univariate analysis.[30]

The multivariate analyses that are summarized in Figure 1 retained only four (4) factors which were predictors of HBP and were as fellow: female sex, which was negatively associated with HBP OR = 0.31; CI 95% [0.13-0.72], p < 0.00.; but obesity OR = 2.29; CI 95% [1.14-4.69], p = 0.02; MetS OR = 3.47; CI 95% [1.64-7.61], p = 0.001; and *hs*-CRP OR = 18.10; CI 95% [9.40-36.99], p < 0.001were positively associated to HBP. Data from Hwang YC et al study showed that, age: OR = 1.71; CI 95% [1.26 to 2.31]; p = 0.001, SBP OR = 1.83; CI 95% [1.31 to 2.56]; p < 0.001, and HDL2/HDL-C OR = 0.71; CI 95% [0.52 to 0.98]; p = 0.035, were associated with the future development of hypertension in a multivariate analysis.[30] According to Gu H et al., the risk of MetS in overweight, pre-hypertension, hypertension subjects were OR = 4.610, CI 95% [2.415 to 8.800], OR = 2.759, CI 95% [1.519 to 5.011] and OR = 3.589, CI 95% [1.672 to 7.706] times higher than that in controls, respectively.[34]

Other multivariate logistic analyses, confirm that the visceral obesity OR = 6.54; CI 95% [2.99-14.3], low HDL-C

Table 2: Comparison of cardiometabolic risk factors according to blood pressure statute

Clinical and Cardiometabolic risk factors	N	SBP > 140 or DBP > 90 mmHg (N=107)	SBP ≤ 140 or DBP ≤ 90 mmHg (185)	p-value
> 50 years	114	98 (86.0%)	16 (14.0%)	< 0.001
≤ 50 years	178	9(5.1%)	169(94.9%)	
Male	116	45(38.8%)	71 (61.2%)	0.62
Female	176	62 (35.2%)	114 (64.8)	
VO > 102 cm men, > 88 cm women, %	161	93(57.8%)	68(42.2%)	< 0.001
VO ≤ 102 cm men, ≤ 88 cm women, %	131	14(10.7%)	117(89.3%)	
Smoker, %	41	35(85.4%)	6(%14.6)	< 0.001
Non-smoker, %	251	72(28.7%)	179(71.3%)	
Alcohol consumption, %	38	18(47.4%)	20(52.6%)	0.20
Non-alcohol consumption, %	254	89(35.0%)	165(65.0%)	
BMI ≥ 30 Kg/m2, %	86	57(66.3%)	29(33.7%)	< 0.001
BMI < 30 Kg/m2, %	206	50(24.3%)	156(75.7%)	
CREAT > 110 men, > 90 μmol/L women	34	13(38.2%)	21(61.8%)	0.99
CREAT ≤ 110 men, ≤ 90 μmol/L women	258	94(36.4%)	164(63.6%)	
TC ≥ 5.0 mmol/L, %	12	7(58.3%)	5(41.7%)	0.20
TC < 5.0 mmol/L, %	280	100(35.7%)	180(64.3%)	
TG ≥ 1.7 mmol/L, %	35	16(45.7%)	19(54.3%)	0.33
TG < 1.7 mmol /L%	257	91(35.4%)	166(64.6%)	
HDL-C > 1.03 men, > 1.29 women mmol/L, %	121	24(19.8%)	97(80.2%)	< 0.001
HDL-C ≤ 1.03 men, ≤ 1.29 women mmol/L, %	171	83(48.5%)	88(51.5%)	
LDL-C, % ≥ 3.5 mmol/L	24	19(79.2%)	5(20.8%)	0.001
LDL-C, % < 3.5 mmol/L	268	88(32.8%)	180(67.2%)	
Glucose ≥ 5.6 mmol/L, %	63	36(57.1%)	27(42.9%)	0.001
Glucose < 5.6 mmol/L, %	229	71(31.0%)	158(69.0%)	
Uric acid ≥ 360 men, ≥ 420 women mmol/L, %	44	21(25.0%)	23(75.0%)	0.14
Uric acid < 360 men, women < 420 mmol/L, %	248	86(34.7%)	162(75.3%)	
MetS, %	135	92(68.1%)	43(31.9%)	0.001
Non-MetS, %	157	15(9.6%)	142(%)	
hs-CRP ≥ 3 mg/L,%	125	93(74.4%)	32(25.6%)	< 0.001
hs-CRP < 3 mg/L, %	167	14(8.4%)	153(91.6%)	

VO: visceral obesity, BMI: body mass index, TC: total cholesterol, TG: triglycerides, HDL-C: high density lipoprotein cholesterol, LDL-C: low density lipoprotein cholesterol, Meth: metabolic syndrome, *hs*-CRP: high sensitive C Reactive Proteins

OR = 2.78; CI 95% [1.09-7.12], impaired FBG OR = 6.72; CI 95% [3.30-13.7], and MetS OR = 10.4; CI 95% [5.18-20.7] were associated with higher CRP.[35]

Some limits of our study are that our data did not include information on the treatment status of hypertension. Also, the hygienic and dietetic behaviors were not evaluated that could be confounding factors in our series. Moreover, our study was an intra-hospital one. It will be interesting to perform this study at a population level as recommended by the WHO STEP wise approach to NCDs risk factors surveillance. Given the role of inflammation in CVDs pathogenesis, it could be necessary to assess the link of others inflammatory markers to the HBP.[27] Finally, during our sampling process, subjects were not stratified into the two different groups which could be the subject to selection bias. Overall, longitudinal studies designed at the population level are needed for more detailed analyzes in order to determine the share of cardiometabolic risk factors on the rise of blood pressure. Nevertheless, our data show that in the monitoring of cardiometabolic risk factors in outpatients, special attention should be given to patients with HBP even though they are in steady-state.

Table 3: Univariate analysis byusing blood pressure as dependent variable and clinical and biochemistry variables as independent variables

Independent variables (clinical and Cardiometabolic risk factors)	Dependent variable (blood pressure) Odds Ratio	[CI, 2.5-97.5%]	p-value
> 50 years	2.1	[1.3 – 3.5]	0.003
Female	0.9	[0.6 – 1.5]	0.71
Smoker	0.9	[0.4 – 1.7]	0.72
Alcohol consomption	0.9	[0.4 – 1.8]	0.74
Visceral obesity: WC > 102 cm men, WC > 88 cm women	1.6	[1.0 – 2.6]	0.05
BMI > 30 kg.m-2	1.3	[0.8 – 2.3]	0.32
Creatinine > 110 μmol/L men, > 90 μmol/L women	1.1	[0.5 – 2.2]	0.83
CHOL ≥ 5 mmol/L	0.9	[0.2 – 2.8]	0.81
TG ≥ 1.7 mmol/L	1.2	[0.7 – 2.4]	0.66
HDL-C ≤ 1.03 men, ≤ 1.29 mmol/L women	1.2	[0.7 – 1.9]	0.51
LDL-C > 3.5 mmol/L	1.0	[0.4 - 2.4]	0.93
Glucose ≥ 5.6 mmol/L	1.0	[0.6 – 1.8]	0.98
Uric acid ≥ 360 men, ≥ 420 mmol/L women	1.0	[0.5 - 1.9]	0.97
MetS	3.3	[2.0 – 5.4]	< 0.001
CRP ≥ 3 mg/L	16.8	[9.4 – 31.4]	< 0.001

5. Conclusion

Our data showed a strong association between MetS, *hs*-CRP and HBP. Therefore, given the context of worldwide epidemic of HBP and its comorbidity on cardiovascular diseases, early detection and monitoring of cardiometabolic components could be an effective prevention policies even though in steady state HBP subjects.

6. Statement of Informed Consent

Each participant gave fully informed written consent prior to enrollment. The protocol was reviewed and approved by the Mopti hospital ethical committee.

7. Source of Funding

None.

8. Conflict of Interest

The authors certify that there is no actual or potential conflict of interest in relation to this article.

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Author biography

Modibo Coulibaly, Associate Professor in Clinical Biochemistry https://orcid.org/0000-0002-3816-978X

Lamine Sidibé, Research Fellow in Cardiology

Djibril Traoré, Research Fellow. General Surgery

Mamoudou Barry, Pharmacist

Valentin Sagara, Research Assistant in Biomedical

Bakary Maiga, Professor of Immunology

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