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# Metabolic Abnormalities, Genetic and Behavioral Risk Factors for Hypertension among Patients Attending Bafoussam Regional Hospital, Cameroon

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**Abstract** **Background:** Hypertension is one of the most public health problems worldwide. More than 60% of the risk factors for hypertension are associated with metabolic disturbances. Literature perusal showed lack of information regarding the risk factors for hypertension in people from the West Region of Cameroon. Therefore, the present study was intended to evaluate the metabolic abnormalities and risk factors for hypertension in patients attending the Bafoussam Regional Hospital in Cameroon. **Methods:** A cross-sectional and descriptive study was conducted within the Bafoussam Regional Hospital on 343 individuals (99 normotensives and 244 hypertensives). Genetic and behavioral risk factors for hypertension were determined through a questionnaire. Hypertension was diagnosed by measuring blood pressure. Cardiac, renal and hepatic biochemical parameters were analysed in blood and urine by methods resulting from commercial kits. **Results:** Smoking (24.2%), advanced age (90.1%), obesity (33.6%), sedentary lifestyle (56.1%), family history of hypertension (63.9%), hypertriglyceridemia (20.5%), hyperglycemia (36.9%), hypernatremia (30.3%), hyperchloremia (10.7%), glycosuria (11.50%), hypercreatininemia (40.2%) and hypoglycemia (15.6%) were frequent and significantly ($p < 0.05$) higher among hypertensive individuals as compared to normotensive participants. Hypertensive patients showed low levels of hepatic metabolic abnormalities. Of the 244 hypertensive patients, 40% are complicated by various forms of metabolic abnormalities. **Conclusion:** Overall, the results of the present study indicate that genetic and behavioral parameters as well as cardiac and renal metabolic disturbances are the main risk factors of hypertension in the study population. Hence, strategies targeting the metabolic pathways may be therapeutic options for improving biochemical abnormalities and reducing blood pressure and risk of complications in metabolic hypertension.

***Keywords:*** *hypertension, prevalence, risk factors, metabolic abnormalities*

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## 1. Introduction

Hypertension or High Blood Pressure (HBP) is a serious medical condition that significantly increases the risks of heart, brain, kidney and other diseases. It's a major cause of premature death worldwide. The number of adults with hypertension worldwide increased from 594 million in 1975 to 1.13 billion in 2015. [1] Moreover, 30 to 50% of deaths are attributed to high blood pressure in developing countries. [2] In sub-Saharan Africa, an estimated 74.7 million individuals are hypertensive. [3] In Cameroon, surveys on hypertension report a prevalence varying from 12 to 22% in those above 25 years. [4] Hypertension is defined as a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg measured on two different days. [2] Traditionally, hypertension has been classified into primary and secondary hypertension, which represent 90% and 10% of total hypertension, respectively. [5] There are numerous factors that put people at risk of developing hypertension and may be either modifiable or non-modifiable. Modifiable risk factors include unhealthy diets (excessive salt consumption, a diet high in saturated fat and trans fats, low intake of fruits and vegetables), physical inactivity, consumption of tobacco and alcohol, and being overweight or obese. Non-modifiable risk factors include a family history of hypertension, age over 65 years and co-existing diseases such as diabetes or kidney disease. According to the World Health Organization [6], a significant percentage of noncommunicable diseases are preventable by addressing four main behavioral risk factors namely physical inactivity, unhealthy diet, tobacco use, and alcohol consumption.

The partial failure of antihypentensive management in general has been attributed to the metabolic adverse side

effects of many drugs (body weight gain, glucose and lipid metabolism disturbances), particularly thiazide diuretics and beta-blockers. [7,8,9] This explanation is certainly based on good evidence, but probably focuses on only one aspect of a longer story. Hypertension is frequently associated with several metabolic abnormalities among which obesity, glucose intolerance, and dyslipidemia are the most common. [5] Obesity, diabetes, dyslipidemia, high salt intake, hyperuricemia and hyperhomocysteinemia are major metabolic risk factors for hypertension. However, several studies carried out in the last 10 years have shown that metabolic abnormalities alone may contribute to the pathogenesis of hypertension. [5] According to reports from Shanghai Hypertension Institute and Chongqing Hypertension Institute, more than 80% of hypertensive patients are complicated by various forms of metabolic abnormalities, while only 20% of hypertensive patients are not complicated with metabolic disturbances. [10,11] Clinical studies have shown that metabolic disorders dramatically increase the risk of vascular disease as an independent risk factor. Hyperuricemia is linked to decreased renal blood flow and increased renal vascular resistance in hypertension. [12] Treatments for hypertension should control both blood pressure and metabolic disorders.

Overall, hypertension with versus without metabolic abnormalities is associated with higher risk for cardiovascular events. Hence, the risk stratification of hypertension is based on the number and severity of metabolic risk factors. [13] For hypertensive subjects with several metabolic disturbances, rapid initiation and intensification of blood pressure lowering therapy are strongly recommended. [14] Furthermore, literature perusal showed lack of information regarding the risk factors for hypertension in people from the West Region of Cameroon. Therefore, in order to contribute to fight against hypertension, the present study was intended to determine the metabolic abnormalities and risk factors for hypertension in patients attending the Bafoussam Regional Hospital in Cameroon.

# 2. Materials and Methods

## 2.1. Study Setting

It is a cross-sectional and descriptive study conducted within the Bafoussam Regional Hospital (West Cameroon) during the period from October 2017 to March 2018. Based on the inclusion criteria (being hypertensive or not, being on antihypertensive medication or not, being at least 21 years old and giving a favorable opinion for free participation in the study) and the non inclusion criteria (suffering from mental disorders, being pregnant and having refused to sign informed consent), 364 participants were eligible for the study. Among them, subjects with hemolyzed blood serum and who did not provide the full amount of information required (n = 21) were excluded from the analysis. Finally, 343 participants were included with approximately 2288 hypertensive patients attending cardiology departments for an annual medical checkup. These participants include 99 normotensive participants and 244 hypertensive patients. The study protocol was approved by the Regional Ethics Committee research for human health of center N° 0683 and was conducted in strict compliance with the physical, moral and psychological integrity of all participants; following the principles outlined in the Helsinki Declaration.

## 2.2. Definitions

Hypertension was defined as a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg. Overweight or obesity was defined as: 25.0 ≤ BMI < 30.0 kg/m$^2$ or BMI ≥ 30.0 kg/m$^2$. Hyperglycemia was defined as fasting plasma glucose levels ≥ 105 mg/dL. Dyslipidemia was defined as: hypertriglyceridemia (TG ≥ 1.5 g/L), hypo HDL-c (HDL-c < 0.4 g/L), hyperLDL-c (LDL-c ≥ 1.6 g/L), hypercholesterolemia (CT ≥ 2 g/L). Ionic homeostasis disorders have been established for values excluded from the following ranges: Hepatotoxicity was established for values excluded from the following ranges: ASAT (10 - 40 UI/ml), ALAT (20 - 60 UI/ml) and nephrotoxicity for values excluded from the following ranges: kaliemia (3.6 - 50 mmol/L), natremia (135 -1450 mmol/L), chloremia (98 - 105 mmol/L), creatininemia (6 ≤ creatinine ≥ 13 mg/L), hypoglycemia < 0.75 g/L, hematuria > 0 red blood cell/µL, proteinuria > 0 g/L, glycosuria > 0 mmol/L. Alcoholism was defined for alcohol consumption more than 3 glasses per day. Smoking has been defined for people who are constantly exposed to cigarette smoke or for unrestricted smokers.

## 2.3. Data Collection

### 2.3.1. Questionnaire

Data were collected using a questionnaire adapted from WHO STEPwise approach for chronic disease risk factor surveillance - Instrument v2.1. This questionnaire included informations on demographic characteristics, smoking and alcohol consumptions, physical activity, family history of hypertension and diabetes. After explaining the purpose of the study to the participants, their writing informed consent was obtained. Baseline socio-demographic data such as age, gender, place of residence, occupation, marital status, monthly income and level of education were recorded. History of cardiovascular risk factors and co-morbidities including diabetes mellitus, smoking, hyperlipidemia, past history of stroke, heart failure, chronic kidney disease, and alcohol consumption were obtained through interviews, complemented by data from the medical records. Height was measured with a calibrated stadiometer to the nearest 0.5 cm, weight in light clothes with a scale balanced to the nearest 0.5 kg. The height and the weight were used to obtain the body mass index expressed in kg/m$^2$. Blood pressures were measured by a trained interviewer three times on each participant in a sitting position with a standard mercury sphygmomanometer. The mean of the second and third systolic and diastolic blood pressure measurements were used. Hypertension was defined by systolic blood pressure ≥ 140 mm Hg, or diastolic blood pressure ≥ 90 mm Hg, or use of medication for hypertension. [2]

#### 2.3.2. Biochemical Analyzis

The urine analysis was performed using the test strips from the Cormay kit. The parameters analysed were glycosuria, proteinuria and hematuria. In the blood, glucose was quantified using the kinetic method described by Trinder (1969) with the commercial kit Liquick Cor-GLUCOSE from Cormay Laboratories. Triglycerides were quantified using the Liquick Cor-TG commercial kit from Cormay Laboratories. Total cholesterol and HDL-cholesterol were quantified using the Chronolab commercial kit, while LDL-cholesterol was determined from Friedewald's equation. [15] Creatinine was determined by Jaffé's (1886) modified method. The activities of alanine aminotransferase (ALAT) and aspartate aminotransferase (ASAT) were measured according to the protocol of Liquick Cor- ALAT and Liquick Cor-ASAT commercial kits from Cormay Laboratories. Serum concentrations of K+, Na+ and Cl- ions were measured using the Labcare Diagnostic Kits.

### 2.4. Statistical Analysis

Data compile from the questionnaire were analyzed using SPSS 16.0 for Windows. Descriptive analysis results were presented as means ± standard deviations for continuous variables and as frequencies for categorial variables. Chi square and student t- tests were performed to compare categorial and continuous variables, respectively, with statistical significance at $p < 0.05$. Means of each biochemical parameter in different groups were compared using Waller-Duncan test at 5% when differences were detected by ANOVA. Cut-off values provided by the kits were used to classify the participant as abnormal (value out of the reference range) and normal (value within the reference range). The bivariate correlation of Pearson was used to determine the strength of relationship between biochemical parameters, while the Receiver Operating Characteristic (ROC) curve analysis was performed to measure the association between variations of the biochemical parameters and the hypertension status of the patients.

## 3. Results

### 3.1. General Characteristics

The study population was mainly composed of hypertensive patients (71.1%), while normotensive patients (28.9%) were in the minority. (Table 1) No significant difference was observed between women and men in the study population. Hypertensive patients (24.2%) were six time more smoking than normotensive participants ($p = 0.000$). However, alcohol consumption of at least 3 glasses per day is more represented in normotensive participants (66.7%) than in hypertensive patients (50.8%) ($p = 0.005$). The frequency of physical activity was also higher among normotensive patients (52.52%) than among hypertensive patients (43.9%) ($p = 0.090$). According to the employment, workers of the informal sector were significantly ($p = 0.000$) more represented and was higher in hypertensive patients (59.7%) than in normotensive participants (44.4%). Married participants were most represented in all both groups while single and widower participants were most represented ($p = 0.000$) in normotensive and hypertensive participants, respectively. Participants who stopped their study at secondary school were significantly more represented ($p = 0.000$) among normotensive (34.3%) and hypertensive patients (32.8%), followed by those of primary school. Illiterate people were the least represented among normotensive people, while among hypertensive people, it was the people of highest level of education.

**Table 1. Distribution of socio-demographic characteristics and environmental factors according to normotensive and hypertensive status**

| Characteristics | Normotensive participants n (%) = 99 (28.9%) | Hypertensive patients n (%) = 244 (71.1%) | P- value |
|---|---|---|---|
| **Sex** | | | |
| Female | 62 (62.6%) | 144 (59.0%) | 0.311 |
| Male | 37 (37.4%) | 100 (41.0%) | |
| **Family history** | | | |
| Family history of hypertension | 37 (37.4%) | 156 (63.9%) | 0.000 |
| **Environmental factors** | | | |
| Physical activity practice (at least 1 time per day) | 52 (52.5%) | 107 (43.9%) | 0.090 |
| Smoking | 4 (4.0%) | 59 (24.2%) | 0.000 |
| Alcohol consumption (at least 3glass per day) | 66 (66.7%) | 124 (50.8%) | 0.005 |
| **Employment** | | | |
| Civil servants | 23 (23.2%) | 41 (16.9%) | 0.000 |
| Formal sector | 26 (26.3%) | 18 (7.4%) | |
| Informal sector | 44 (44.4%) | 145 (59.7%) | |
| Retirees | 6 (6.1%) | 39 (16.0%) | |
| **Marital status** | | | |
| Married | 55 (55.6%) | 169 (69.3%) | 0.000 |
| Single | 34 (34.3%) | 6 (2.5%) | |
| Widowed | 10 (10.1%) | 69 (28.3%) | |
| **Level of education** | | | |
| Illiteracy | 13 (13.1%) | 67 (27.5%) | 0.000 |
| Primary school | 21 (21.2%) | 68 (27.9%) | |
| Secondary school | 34 (34.3%) | 80 (32.8%) | |
| Higher | 31 (31.3%) | 29 (11.9%) | |

**Table 2. Distribution of anthropometric and hemodynamic characteristics according to normotensive and hypertensive status**

| Characteristics | Normotensive participants n (%) = 99 (28.9%) | Hypertensive patients n (%) = 244 (71.1%) | *p-value* |
|---|---|---|---|
| **Age (years)** | | | |
| Mean | 47.45 ± 15.980[a] | 63.11 ± 11.848[b] | |
| < 40 | 40 (40.4%) | 6 (2.5%) | 0.000 |
| [40 - 50[ | 13 (13.1%) | 18 (7.4%) | |
| [50 - 60[ | 18 (18.2%) | 75 (30.7%) | |
| [60 - 70[ | 20 (20.2%) | 70 (28.7%) | |
| [70 - 80[ | 6 (6.1%) | 58 (23.8%) | |
| ≥ 80 | 2 (2.0%) | 17 (7.0%) | |
| **BMI** | | | |
| Mean (kg/m$^2$) | 27.28 ± 3.75[a] | 28.15 ± 5.29[b] | |
| Normal | 48 (48.5%) | 82 (33.6%) | 0.000 |
| Overweight (25 < BMI < 30) | 40 (40.4%) | 80 (32.8%) | |
| Obesy (BMI ≥ 30) | 11 (11.1%) | 82 (33.6%) | |
| **Heart rate** | | | |
| Mean (pulse/min) | 63.64 ± 7.14[a] | 78.37 ± 14.11[b] | |
| Normal (60 - 100) | 96 (97.0%) | 212 (86.9%) | 0.012 |
| Low (< 60) | 3 (3.0%) | 16 (6.6%) | |
| High (> 100) | 0 (0.0%) | 16 (6.6%) | |
| **Blood pressure** | | | |
| SBP (mmHg) | 123.30 ± 12.43[a] | 169.62 ± 19.27[b] | |
| DBP (mmHg) | 75.44 ± 8.52[a] | 95.43 ± 13.07[b] | |

On the same line, the values with different letters are significantly different at $p < 0.05$ with student t-test. BMI: Body Mass Index, SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.

The mean age of hypertensive patients was significantly ($p < 0.05$) higher than that of normotensive patients. (Table 2) Regarding the age group, hypertensive patients were significantly most represented between 50 - 80 years while the majority of normotensive participants have less than 50 years. The mean value of BMIs in normotensive participants was lower than that of hypertensive patients. Even if the overweight percentage was highest in normotensive participants, the frequency of obesity was three times highest in hypertensive patients. The mean values of Systolic Blood Pressures (SBPs) and Diastolic Blood Pressures (DBPs) in hypertensive patients were significantly ($p < 0.05$) higher than those of normotensive participants. The mean value of heart rate in hypertensive patients was significantly ($p < 0.05$) higher than that of normotensive patients. Even if these mean values were within the normal range, few patients in both groups had abnormal values of heart rate (6.6% of bradycardia / tachycardia in hypertensive patients versus 3.0% of tachycardia in normotensive participants).

## 3.2. Biochemical Characteristics of the Study Population

In hypertensive patients, the mean values of glycemia, LDL-cholesterolemia, total-cholesterolemia, creatininemia, plasma ALAT and plasma ASAT were significantly ($p < 0.05$) higher compared to those of normotensive subjects. (Table 3) The mean values of HDL-cholesterolemia (in normotensive participants), glycemia and creatininemia (in hypertensive patients) are out of the normal ranges. However, the mean values of other biochemical parameters were within the normal ranges.

**Table 3. Mean values of selected biochemical parameters according to normotensive and hypertensive participants.**

| Parameters (reference values) | Normotensive participants (n = 99) | Hypertensive patients (n = 244) | p - value |
|---|---|---|---|
| Glycemia (0 .75 - 1.05 g/l) | 0.99 ± 0.19 | 1.12 ± 0.51 | 0.001 |
| Triglyceridemia (< 1.5 g/l) | 1.04 ± 0.37 | 1.10 ± 0.47 | 0.211 |
| Total Cholesterolemia (< 2 g/l) | 1.40 ± 0.35 | 1.70 ± 0.40 | 0.000 |
| LDL-cholesterolemia (< 1.6 g/l) | 0.93 ± 0.26 | 1.19 ± 0.33 | 0.000 |
| HDL-cholesterolemia (> 0.4 g/l) | 0.32 ± 0.14 | 0.69 ± 3.50 | 0.103 |
| ALAT activity (10 - 40 UI/ml) | 16.06 ± 2.59 | 19.84 ± 9.47 | 0.000 |
| ASAT activity (20 - 60 UI/ml) | 24.22 ± 4.89 | 26.45 ± 9.17 | 0.004 |
| Creatininemia (6 - 13 mg/dl) | 10.80 ± 1.60 | 13.23 ± 5.04 | 0.000 |
| Kaliemia (3.6 - 5 mmol/L) | 4.44 ± 0.60 | 4.53 ± 0.93 | 0.348 |
| Chloremia (98 - 105 mmol/L) | 100.96 ± 5.27 | 101.74 ± 3.77 | 0.175 |
| Natremia (135 - 145 mmol/L) | 143.10 ± 4.06 | 143.46 ± 4.76 | 0.478 |

ASAT : aspartate aminotransferase, ALAT : alanine aminotransferase.

### 3.3. Prevalence of Cardiometabolic Risk Factors in the Study Population

The most important cardiometabolic risk factors in hypertensive patients were hyperglycemia and hypertriglyceridemia. (Table 4) Of the 244 hypertensive patients, 38% are complicated by different forms of cardiometabolic abnormalities. No significant differences were found between the frequencies of total hypercholesterolemia and LDL-hypercholesterolemia. However, the frequencies of HDL-hypocholesterolemia were significantly (p = 0.000) higher in normotensive participants compared with hypertensive patients.

### 3.4. Prevalence of Renal Metabolic Disorders in the Study Population

Of the 244 hypertensive patients, 40% are complicated by various forms of renal metabolic disturbances. (Table 5) The frequencies of hypoglycemia, creatininemia, hypernatremia, hyperchloremia and glycosuria, were significantly ($p < 0.05$) highest in hypertensive patients compared with those of normotensive participants. However, no significant ($p \geq 0.05$) differences were found between the frequencies of hyperkaliemia, hyponatremia, hypochloremia, hematuria and proteinuria in the two groups of the study population even if these frequencies were higher in hypertensive patients than in normotensive participants.

### 3.5. Prevalence of Hepatic Metabolic Disorders in the Study Population

No significant different were found between the two group of the study population according to transaminases, even if the frequencies of hyper ASAT and hyper ALAT were higher in hypertensives than in normotensive participants. (Table 6) Of the 244 hypertensive patients, only 7% are complicated by different forms of liver metabolic abnormalities.

### 3.6. Prevalence of Biochemical Abnormalities According to Some Sociodemographic Characteristics of the Study Population

The prevalences of biochemical abnormalities in hypertensive patients have in some cases been influenced by socio-demographic characteristics such as level of education, marital status and occupation. (Table 7) Hence, the prevalences of hypercreatininemia were significantly ($p = 0.000$) higher among single participants than married and widowers, among retirees than civil servants and private sector workers ($p = 0.001$) and among high level of education than in other levels of study ($p = 0.001$). The prevalences of hematuria, proteinuria, glycosuria, natremia, chloremia, triglyceridemia, ASAT and ALT abnormalities were not affected ($p \geq 0.05$) by the studied socio-demographic features. The same is true for kaliemia except with a poor higher prevalence of hyperkaliemia among widowed participants than among married and celibates ($p = 0.004$). The prevalence of total hypercholesterolemia was higher among civil servants than in other type of occupation ($p = 0.000$); and not associated by the level of education and marital status. Such as hypercholesterolemia, HDL-hypocholesterolemia is only influenced by employment with a higher number of workers in the formal sector than in civil servants, retirees and workers in the formal sector ($p = 0.000$). Hyperglycemia was more observed in the illiterate ($p = 0.0018$), widowers ($p = 0.0001$), retirees and patients working in the informal sector (p = 0.0018). Hypoglycemia was most noted in celibates as well as in patients at the primary level of education and those working in the informal sector.

**Table 4. Distribution of cardiometabolic risk factors according to normotensive and hypertensive participants**

| Cardiometabolic risk factors | Normotensive participants (n = 99) | Hypertensive patients (n = 244) | p - value |
|---|---|---|---|
| Hyperglycemia | 29 (29.3%) | 90 (36.9%) | 0.001 |
| Hypertriglyceridemia | 6 (6.1%) | 50 (20.5%) | 0.001 |
| Total Hypercholesterolemia | 2 (2.0%) | 11 (4.5%) | 0.362 |
| LDL-Hypercholesterolemia | 3 (3.0%) | 19 (7.8%) | 1.144 |
| HDL-Hypocholesterolemia | 83 (83.8%) | 93 (38.1%) | 0.000 |

**Table 5. Distribution of renal metabolic disorders according to normotensive and hypertensive participants**

| Renal metabolic disorders | Normotensive participants (n = 99) | Hypertensive patients (n = 244) | p - value |
|---|---|---|---|
| Hypoglycemia | 4 (4.0%) | 38 (15.6%) | 0.001 |
| Creatininemia | 10.1% (10) | 98 (40.2%) | 0.000 |
| Hyper Kaliemia | 8 (8.1%) | 41 (16.8%) | 0.112 |
| Hypo Natremia | 2 (2.0%) | 6 (2.5%) | 0.099 |
| Hyper Natremia | 19 (19.2%) | 74 (30.3%) | 0.043 |
| Hypo Chloremia | 10 (10.1%) | 28 (11.5%) | 0.090 |
| Hyper Chloremia | 3 (3.0%) | 26 (10.7%) | 0.023 |
| Hematuria | 2 (2.0%) | 18 (7.4%) | 0.073 |
| Proteinuria | 29 (29.3%) | 93 (38.1%) | 0.136 |
| Glycosuria | 4 (4.0%) | 28 (11.5%) | 0.039 |

**Table 6. Distribution of hepatic disorders according to normotensive and hypertensive participants**

| Hepatic metabolic disorders | Normotensive participants (n = 99) | Hypertensive patients (n = 244) | p - value |
|---|---|---|---|
| Hyper aspartate aminotransferase activity | 2 (2.0%) | 17 (7.0%) | 0.114 |
| Hyper alanine aminotransferase activity | 0 (0%) | 3 (1.2%) | 0.560 |

**Table 7. Prevalences of biochemical abnormalities in hypertensive patients according to some sociodemographic characteristics of the study population**

| **Parameters** | | **Level of education** | | | | **Marital status** | | |
|---|---|---|---|---|---|---|---|---|
| | | Illiterate | Primary school | Secondary school | Higher | Married | Single | Widowed |
| Creatininemia | Normal creatininemia | 43(53.8%) | 43(53.8%) | 43(53.8%) | 43(53.8%) | 88(52.1%) | 2(33.3%) | 56(81.2%) |
| | Hyper creatininemia | 31(46.3%) | 14(20.6%) | 37(46.3%) | 16(52.2%) | 81(47.9%) | 4(66.7%) | 13(18/8%) |
| | *p*-value | 0.001 | | | | 0.000 | | |
| Hematuria | Hematuria (-) | 64(95.5%) | 67(98.5%) | 70(87.5%) | 25(86.2%) | 153(90.5%) | 5(83.3%) | 68(98.6%) |
| | Hematuria (+) | 3(4.5%) | 1(1.5%) | 10(12.5%) | 4(13.8%) | 16(9.5%) | 1(16.7%) | 1(1.4%) |
| | *p*-value | 0.028 | | | | 0.068 | | |
| Proteinuria | Proteinuria (-) | 32(47.8%) | 49 (72.1%) | 51(63.8%) | 19(65.5%) | 96(56.8%) | 4(66.7%) | 51(73.9%) |
| | Proteinuria (+) | 35(52.2%) | 19(27.9%) | 29(36.3%) | 10(34.5%) | 73(43 .2%) | 2(33.3%) | 18(26.1%) |
| | *p*-value | 0.030 | | | | 0.046 | | |
| Glycosuria | Glycosuria (-) | 61(91.0%) | 57(83.8%) | 75(93.8%) | 23(79.3%) | 156(92.3%) | 5(83.3%) | 55(79.7%) |
| | Glycosuria (+) | 6(9.0%) | 11(16.2%) | 5(6.3%) | 6(20.7%) | 13(7.7%) | 1(16.7%) | 14(20.3%) |
| | *p*-value | 0.091 | | | | 0.020 | | |
| Kalemia | Normal kaliemia | 52(77.6%) | 47(69.1%) | 63(78.8%) | 25(86.2%) | 137(81.1%) | 3(50.0%) | 47(68.1%) |
| | Hypokaliemia | 0(0.0%) | 5(7.4%) | 9(11.3%) | 2(6.9%) | 11(6.5%) | 2(33.3%) | 3(4.3%) |
| | Hyperkaliemia | 15(22.4%) | 16(23.5%) | 8(10.0%) | 2(6.9%) | 21(12.4%) | 1(16.7%) | 19(27.5%) |
| | *p*-value | 0.020 | | | | 0.004 | | |
| Natremia | Normal natremia | 48(71.6%) | 47(69.1%) | 47(58.8%) | 22(75.9%) | 111(65.7%) | 3(50.0%) | 50(72.5%) |
| | Hyponatremia | 3(4.5%) | 0(0.0%) | 1(1.3%) | 2(6.9%) | 2(1.2%) | 1(16.7%) | 3(4.3%) |
| | Hypernatremia | 16(23.9%) | 21(30.9%) | 32(40.0%) | 5(17.2%) | 56(33.1%) | 2(33.3%) | 16(23.2%) |
| | *p*-value | 0.062 | | | | 0.055 | | |
| Chloremia | Normal chloremia | 53(79.1%) | 49(72.1%) | 66(82.5%) | 22(75.9%) | 137(81.1%) | 3(50.0%) | 50(72.5%) |
| | Hypochloremia | 6(9.0%) | 4(5.9%) | 12(15.0%) | 4(13.8%) | 15(8.9%) | 2(33.3%) | 9(13.0%) |
| | Hyperchloremia | 8(11.9%) | 15(22.1%) | 2(2.5%) | 3(10.3%) | 17(10.1%) | 1(16.7%) | 10(14.5%) |
| | *p*-value | 0.013 | | | | 0.212 | | |
| Total Cholesterolemia | Normal cholesterolemia | 66(98.5%) | 68(100%) | 72(90.0%) | 27(93.1%) | 159(94.1%) | 5(83.3%) | 69(100%) |
| | Hyper cholesterolemia | 1(1.5%) | 0(0.0%) | 8(10.0%) | 2(6.9%) | 10(5.9%) | 1(16.7%) | 0(0.0%) |
| | *p*-value | 0.014 | | | | 0.047 | | |
| HDL Cholesterolemia | Normal HDL-cholesterolemia | 38(56.7%) | 50(73.5%) | 50(62.5%) | 13(44.8%) | 109(64.5%) | 2(33.3%) | 40(58.0%) |
| | Hypo HDL-cholesterolemia | 29(43.3%) | 18(26.5%) | 30(37.5%) | 16(55.2%) | 60(35.5%) | 4(66.7%) | 29(42.0%) |
| | *p*-value | 0.041 | | | | 0.022 | | |
| LDL-Cholesterolemia | Normal LDL-cholesterolemia | 64(95.5%) | 68(100%) | 68(85.0%) | 25(86.2%) | 156(92.3%) | 5(83.3%) | 64(92.8%) |
| | Hyper LDL-cholesterolemia | 3(4.5%) | 0(0.0%) | 12(15.0%) | 4(13.8%) | 13(7.7%) | 1(16.7%) | 5(7.2%) |
| | *p*-value | 0.003 | | | | 0.709 | | |
| Triglyceridemia | Normal triglyceridemia | 54(80.6%) | 56(82.4%) | 62(77.5%) | 22(75.9%) | 133(78.7%) | 6(100.0%) | 55 (79.7%) |
| | Hypertriglyceridemia | 13(19.4%) | 12(17.6%) | 18(22.5%) | 7 (24.1%) | 36(21.3%) | 0(0.0%) | 14(20.3%) |
| | *p*-value | 0.844 | | | | 0.446 | | |
| Glycemia | Normal glycemia | 24(35.8%) | 21(30.9%) | 50(62.5%) | 21(72.4%) | 93(55.0%) | 2(33.3%) | 21(30.4%) |
| | Hypoglycemia | 7(10.4%) | 18(26.5%) | 12(15.0%) | 1(3.4%) | 21(12.4%) | 2(33.3%) | 15(21.7%) |
| | Hyperglycemia | 36(53.7%) | 29(42.6%) | 18(22.5%) | 7(24.1%) | 55(32.5%) | 2(33.3%) | 33 (47.8%) |
| | *p*-value | 0.000 | | | | 0.009 | | |
| ASAT | Normal ASAT | 64(95.5%) | 65(95.6%) | 70(87.5%) | 28(96.6%) | 155(91.7%) | 6(100.0%) | 66(95.7%) |
| | High ASAT | 3(4.5%) | 3(4.4%) | 10(12.5%) | 1(3.4%) | 14(8.3%) | 0(0.0%) | 3(4.3%) |
| | *p*-value | 0.129 | | | | 0.442 | | |
| ALAT | Normal ALAT | 67(100%) | 68(100%) | 77(96.2%) | 29(100%) | 166(98.2%) | 6(100%) | 69(100%) |
| | High ALAT | 0(0.0%) | 0(0.0%) | 3(3.8%) | 0(0.0%) | 3(1.8%) | 0(0.0%) | 0(0.0%) |
| | *p*-value | 0.101 | | | | 0.510 | | |

| Parameters | | Employment | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Civil servants | Formal sector workers | Informal sector workers | Retirees | | | |
| Creatininemia | Normal creatininemia | 22(53.7%) | 8(44.4%) | 101(69.7%) | 15(38.5%) | | | |
| | Hyper creatininemia | 19(46.3%) | 10(55.6%) | 44(30.3%) | 24(61.5%) | | | |
| | *p*-value | 0.001 | | | | | | |
| Hematuria | Hematuria (-) | 38(92.7%) | 16(88.9%) | 138(95.2%) | 33(84.6%) | | | |
| | Hematuria (+) | 3(7.3%) | 2(11.1%) | 7(4.8%) | 6(154%) | | | |
| | *p*-value | 0.146 | | | | | | |
| Proteinuria | Proteinuria (-) | 28(68.3%) | 9(50.0%) | 89(61.4%) | 24(61.5%) | | | |
| | Proteinuria (+) | 13(31.7%) | 9(50.0%) | 56(38.6%) | 15(38.5%) | | | |
| | *p*-value | 0.614 | | | | | | |
| Glycosuria | Glycosuria (-) | 35(85.4%) | 17(94.4%) | 126(86.9%) | 37(94.9%) | | | |
| | Glycosuria (+) | 6(14.6%) | 1(5.6%) | 19(13.1%) | 2(5.1%) | | | |
| | *p*-value | 0.401 | | | | | | |
| Kalemia | Normal kaliemia | 31(75.6%) | 10(55.6%) | 113(77.9%) | 32(82.1%) | | | |
| | Hypokaliemia | 4(9.8%) | 1(5.6%) | 8(5.5%) | 3(7.7%) | | | |
| | Hyperkaliemia | 6(14.6%) | 7(38.9%) | 24(16.6%) | 4(10.3%) | | | |
| | *p*-value | 0.202 | | | | | | |
| Natremia | Normal natremia | 25(61.0%) | 11(61.1%) | 99(68.3%) | 28(71.8%) | | | |
| | Hyponatremia | 2(4.9%) | 1(5.6%) | 3 (2.1%) | 0(0.0%) | | | |
| | Hypernatremia | 14(34.1%) | 6(33.3%) | 43(29.7%) | 11(28.2%) | | | |
| | *p*-value | 0.704 | | | | | | |
| Chloremia | Normal chloremia | 34(82.9%) | 16(88.9%) | 103(71.0%) | 37(94.9%) | | | |
| | Hypochloremia | 5(12.2%) | 0(0.0%) | 19(13.1%) | 1(2.6%) | | | |
| | Hyperchloremia | 2(4.9%) | 2(11.1%) | 23(15.9%) | 1(2.6%) | | | |
| | *p*-value | 0.022 | | | | | | |
| Total Cholesterolemia | Normal cholesterolemia | 34(82.9%) | 18(100%) | 144(99.3%) | 36(92.3%) | | | |
| | Hyper cholesterolemia | 7(17.1%) | 0(0.0%) | 1(0.7%) | 3(7.7%) | | | |
| | *p*-value | 0.000 | | | | | | |
| HDL Cholesterolemia | Normal HDL-cholesterolemia | 24(58.5%) | 5(27.8%) | 100(69.0%) | 21(53.8%) | | | |
| | Hypo HDL-cholesterolemia | 17(41.5%) | 13(72.2%) | 45(31.0%) | 18 (46.2%) | | | |
| | *p*-value | 0.004 | | | | | | |
| LDL-Cholesterolemia | Normal LDL-cholesterolemia | 34(82.9%) | 18(100%) | 137(94.5%) | 35(89.7%) | | | |
| | Hyper LDL-cholesterolemia | 7(17.1%) | 0(0.0%) | 8(5.5%) | 4(10.3%) | | | |
| | *p*-value | 0.051 | | | | | | |
| Triglyceridemia | Normal triglyceridemia | 26(63.4%) | 16(88.9%) | 119(82.1%) | 32(82.1%) | | | |
| | Hypertriglyceridemia | 15(36.6%) | 2(11.1%) | 26(17.9%) | 7(17.9%) | | | |
| | *p*-value | 0.042 | | | | | | |
| Glycemia | Normal glycemia | 30(73.2%) | 12(66.7%) | 57(39.3%) | 17(43.6%) | | | |
| | Hypoglycemia | 1(2.4%) | 1(5.6%) | 29(20.0%) | 6(15.4%) | | | |
| | Hyperglycemia | 10(24.4%) | 5(27.8%) | 59(40.7%) | 16(41.0%) | | | |
| | *p*-value | 0.003 | | | | | | |
| ASAT | Normal ASAT | 36(87.8%) | 18(100%) | 133(91.7%) | 39 (100%) | | | |
| | High ASAT | 5(12.2%) | 0(0.0%) | 12(8.3%) | 0(0.0%) | | | |
| | *p*-value | 0.096 | | | | | | |
| ALAT | Normal ALAT | 39(95.1) | 18(100%) | 144(99.3%) | 39(100%) | | | |
| | High ALAT | 2(4.9%) | 0(0.0%) | 1(0.7%) | 0(0.0%) | | | |
| | *p*-value | 0.137 | | | | | | |

ASAT: aspartate aminotransferase, ALAT : alanine aminotransferase.

## 3.7. Correlations between Some Hemodynamic and Biochemical Parameters during Hypertension

Bivariate analysis (Table 8) of the hemodynamic and biochemical parameters revealed positive / negative moderate correlations between BMI and heart rate; heart rate vs total cholesterolemia, LDL-cholesterolemia, ASAT, ALAT; creatininemia vs kaliemia, glycemia; kaliemia vs total cholesterolemia, glycemia; natremia vs chloremia, total cholesterolemia, triglyceridemia, LDL-cholesterolemia, ASAT; chloremia vs LDL-cholesterolemia, ASAT, ALAT; total cholesterolemia vs triglyceridemia; triglyceridemia vs LDL-cholesterolemia, ASAT. Positive high correlations were observed between total cholesterolemia and LDL-cholesterolemia; ASAT and ALAT.

**Table 8. Correlations between some hemodynamic and biochemical parameters during hypertension**

| Parameters | | BMI | Pulse | Creatinine | Potassium | Sodium | Chloride | T. Chol | HDL-c | TG | LDL-c | Glucose | ASAT | ALAT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Body mass index (BMI) | Pearson Correlation | 1 | **0.162**$^{**}$ | 0.052 | -0.026 | -0.017 | 0.037 | 0.019 | -0.040 | 0.043 | 0.066 | -0.055 | -0.046 | 0.080 |
| | P-value | | 0.003 | 0.337 | 0.634 | 0.760 | 0.496 | 0.724 | 0.456 | 0.427 | 0.221 | 0.310 | 0.396 | 0.140 |
| Pulse | Pearson Correlation | **0.162**$^{**}$ | 1 | 0.105 | -0.037 | 0.012 | 0.043 | **0.219**$^{**}$ | -0.027 | 0.037 | **0.189**$^{**}$ | -0.003 | **0.221**$^{**}$ | **0.286**$^{**}$ |
| | P-value | 0.003 | | 0.053 | 0.500 | 0.828 | 0.430 | 0.000 | 0.615 | 0.497 | 0.000 | 0.957 | 0.000 | 0.000 |
| Creatinine | Pearson Correlation | 0.052 | 0.105 | 1 | **0.123**$^{*}$ | 0.001 | 0.013 | 0.040 | 0.021 | 0.036 | -0.023 | **0.128**$^{*}$ | 0.025 | 0.060 |
| | P-value | 0.337 | 0.053 | | 0.022 | 0.979 | 0.810 | 0.455 | 0.694 | 0.507 | 0.666 | 0.017 | 0.651 | 0.267 |
| Potassium | Pearson Correlation | -0.026 | -0.037 | **0.123**$^{*}$ | 1 | 0.000 | -0.094 | **0.148**$^{**}$ | -0.025 | 0.075 | **-0.222**$^{**}$ | **0.228**$^{**}$ | 0.062 | 0.055 |
| | P-value | 0.634 | 0.500 | 0.022 | | 0.996 | 0.084 | 0.006 | 0.647 | 0.163 | 0.000 | 0.000 | 0.254 | 0.310 |
| Sodium | Pearson Correlation | -0.017 | 0.012 | 0.001 | 0.000 | 1 | **0.361**$^{**}$ | **0.123**$^{*}$ | -0.013 | **0.218**$^{**}$ | **0.109**$^{*}$ | 0.045 | **0.130**$^{*}$ | -0.018 |
| | P-value | 0.760 | 0.828 | 0.979 | 0.996 | | 0.000 | 0.022 | 0.815 | 0.000 | 0.043 | 0.402 | 0.016 | 0.740 |
| Chloride | Pearson Correlation | 0.037 | 0.043 | 0.013 | -0.094 | **0.361**$^{**}$ | 1 | 0.104 | 0.045 | 0.104 | **0.114**$^{*}$ | -0.099 | **0.361**$^{**}$ | **0.117**$^{*}$ |
| | P-value | 0.496 | 0.430 | 0.810 | 0.084 | 0.000 | | 0.053 | 0.409 | 0.054 | 0.035 | 0.066 | 0.000 | 0.030 |
| Total cholesterol | Pearson Correlation | 0.019 | **0.219**$^{**}$ | 0.040 | **-0.148**$^{**}$ | **0.123**$^{*}$ | 0.104 | 1 | 0.065 | **0.317**$^{**}$ | **0.856**$^{**}$ | 0.022 | 0.053 | 0.104 |
| | P-value | 0.724 | 0.000 | 0.455 | 0.006 | 0.022 | 0.053 | | 0.232 | 0.000 | 0.000 | 0.686 | 0.324 | 0.054 |
| HDL-cholesterol | Pearson Correlation | -0.040 | -0.027 | 0.021 | -0.025 | -0.013 | 0.045 | 0.065 | 1 | -0.026 | 0.040 | -0.050 | -0.010 | -0.040 |
| | P-value | 0.456 | 0.615 | 0.694 | 0.647 | 0.815 | 0.409 | 0.232 | | 0.633 | 0.462 | 0.359 | 0.857 | 0.458 |
| Triglycerides | Pearson Correlation | 0.043 | 0.037 | 0.036 | 0.075 | **0.218**$^{**}$ | 0.104 | **0.317**$^{**}$ | -0.026 | 1 | **0.219**$^{**}$ | 0.006 | **0.114**$^{*}$ | 0.048 |
| | P-value | 0.427 | 0.497 | 0.507 | 0.163 | 0.000 | 0.054 | 0.000 | 0.633 | | 0.000 | 0.917 | 0.034 | 0.376 |
| LDL-cholesterol | Pearson Correlation | 0.066 | **0.189**$^{**}$ | -0.023 | **-0.222**$^{**}$ | **0.109**$^{*}$ | **0.114**$^{*}$ | **0.856**$^{**}$ | 0.040 | **0.219**$^{**}$ | 1 | -0.027 | 0.031 | 0.094 |
| | P-value | 0.221 | 0.000 | 0.666 | 0.000 | 0.043 | 0.035 | 0.000 | 0.462 | 0.000 | | 0.624 | 0.565 | 0.083 |
| Glucose | Pearson Correlation | -0.055 | -0.003 | **0.128**$^{*}$ | **0.228**$^{**}$ | 0.045 | -0.099 | 0.022 | -0.050 | 0.006 | -0.027 | 1 | 0.023 | -0.013 |
| | P-value | 0.310 | 0.957 | 0.017 | 0.000 | 0.402 | 0.066 | 0.686 | 0.359 | 0.917 | 0.624 | | 0.665 | 0.813 |
| ASAT | Pearson Correlation | -0.046 | **0.221**$^{**}$ | 0.025 | 0.062 | **0.130**$^{*}$ | **0.361**$^{**}$ | 0.053 | -0.010 | **0.114**$^{*}$ | 0.031 | 0.023 | 1 | **0.595**$^{**}$ |
| | P-value | 0.396 | 0.000 | 0.651 | 0.254 | 0.016 | 0.000 | 0.324 | 0.857 | 0.034 | 0.565 | 0.665 | | 0.000 |
| ALAT | Pearson Correlation | 0.080 | **0.286**$^{**}$ | -0.060 | 0.055 | -0.018 | **0.117**$^{*}$ | 0.104 | -0.040 | 0.048 | 0.094 | -0.013 | **0.595**$^{**}$ | 1 |
| | P-value | 0.140 | 0.000 | 0.267 | 0.310 | 0.740 | 0.030 | 0.054 | 0.458 | 0.376 | 0.083 | 0.813 | 0.000 | |

$^{**}$Correlation is significant at $p < 0.01$; $^{*}$Correlation is significant at $p < 0.05$, ASAT : aspartate aminotransferase, ALAT : alanine aminotransferase, T. chol : total cholesterol, HDL-c : HDL-cholesterol, LDL-c : LDL-cholesterol, TG: triglycerides.

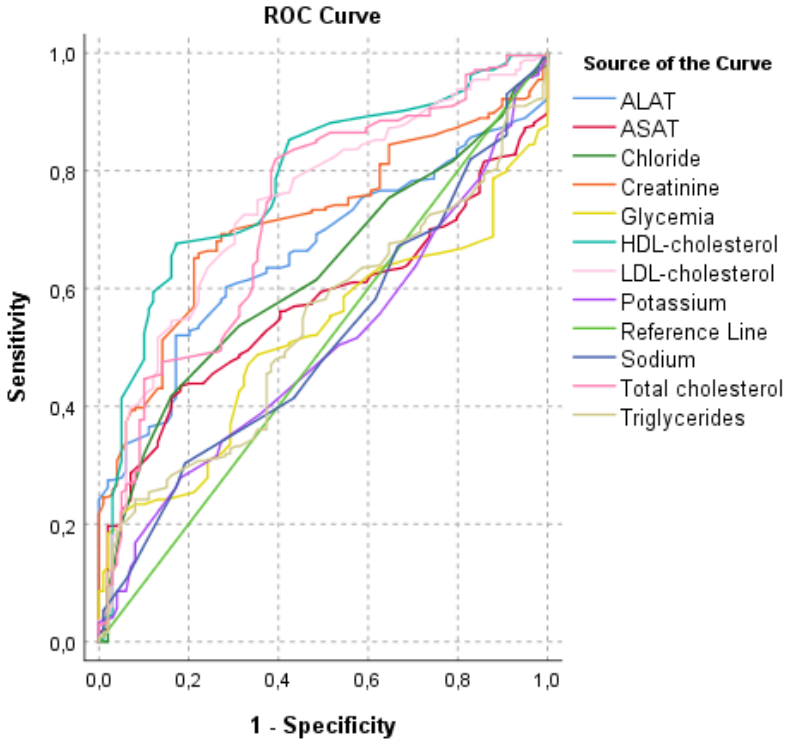


**Figure 1.** Receiver Operating Characteristic (ROC) curve analysis of biochemical parameters against hypertensive status (ASAT : aspartate aminotransferase, ALAT : alanine aminotransferase)

**Table 9. Summary statistics of areas under the curve for the ROC analysis of biochemical parameters against hypertensive status**

| Parameters | Areas under the curve | Standard error | *p*-value | 95% Confidence Interval | |
|---|---|---|---|---|---|
| | | | | Lower Bound | Upper Bound |
| Creatinine | 0.716 | 0.028 | 0.000 | 0.661 | 0.770 |
| Potassium | 0.500 | 0.033 | 0.995 | 0.436 | 0.564 |
| Sodium | 0.512 | 0.033 | 0.720 | 0.447 | 0.577 |
| Chloride | 0.623 | 0.031 | 0.000 | 0.563 | 0.684 |
| Total Cholesterol | 0.727 | 0.030 | 0.000 | 0.668 | 0.786 |
| HDL-cholesterol | 0.782 | 0.027 | 0.000 | 0.730 | 0.835 |
| Triglyceride | 0.535 | 0.032 | 0.279 | 0.472 | 0.598 |
| LDL-cholesterol | 0.743 | 0.029 | 0.000 | 0.688 | 0.799 |
| Gucose | 0.513 | 0.031 | 0.665 | 0.452 | 0.575 |
| ASAT | 0.568 | 0.031 | 0.025 | 0.508 | 0.629 |
| ALAT | 0.661 | 0.029 | 0.000 | 0.604 | 0.718 |

ASAT : aspartate aminotransferase, ALAT : alanine aminotransferase.

### 3.8. Association between Biochemical Parameters and the Hypertensive Status

Regarding the association between biochemical parameters and the hypertensive status, we performed the Receiver Operating Characteristic (ROC) curve analysis and summarised results in Figure 1 and Table 9. The areas under the curve (AUC) with their confidence intervals for creatininemia, chloremia, total cholesterolemia, HDL cholesterolemia, LDL cholesterolemia, ASAT and ALAT were above 0.5; supporting significant associations between increases in these biochemical parameters with the hypertensive status.

## 4. Discussion

High blood pressure is a serious public health problem worldwide. More than 60% of the risk factors for hypertension are associated with metabolic disturbances. [16] Hence, due to the importance of risk factors in the risk assessment, pathogenesis, and treatment of hypertension, we evaluated the metabolic abnormalities and risk factors for hypertension in patients attending the Bafoussam Regional Hospital in Cameroon. The findings of the present study showed that smoking (24.2%), family history of hypertension (63.9%), obesity (33.6%) and advanced age were frequent and significantly higher among hypertensive individuals as compared to normotensive participants; suggesting that the above factors are predictors of hypertension. However, alcohol consumption of at least 3 glasses per day was more represented in normotensive participants (66.7%) than in hypertensive patients (50.8%) ($p$ = 0.005); indicating that normotensive patients also present risk factors for hypertension. Indeed, the cause of hypertension is multifactorial [17] and is strongly associated with alcohol consumption status, education subcategories, age, body mass index, and being overweight or obese. [18,19,20,21] Hypertensive patients were significantly most represented between 50 - 80 years while the majority of normotensive participants have less than 50 years. It is known that the prevalence of hypertension increased with the age of the patients, [22] which is consistent with the results obtained in this study. Indeed, from the age of 50 and 60 years in women and men, respectively, it is important not only to monitor blood pressure permanently, but also to adopt a consistent lifestyle. [23,24] In the present study, obesity (body mass index) with a prevalence of 33.6% in hypertensive patients versus 11.1% in normotensive individuals has emerged as a risk factor for hypertension. The hypertensive effect of fat mass could be linked in part to the activation of the sympathetic system and the renin/angiotensin/aldosterone axis. Both systems are activated in overweight or obese individuals, promoting both an increase in peripheral arterial resistance and an increase in renal sodium retention. Sodium retention may also be promoted by hyperinsulinemia, often associated with overweight and obesity, which is a predictor of the development of hypertension. [14]

Cardiometabolic abnormalities such as hyperglycemia, hypertriglyceridemia, LDL hypercholesterolemia and HDL hypocholesterolemia were highly prevalent in hypertensive patients. This finding suggests that dyslipidemia, diabetes, obesity, arteriosclerosis, and stroke are major metabolic risk factors for hypertension as previously reported. [25,26,27,28] However, early reports have demonstrated that cardiometabolic disturbances alone can contribute to the pathogenesis of hypertension. [5] Thus, hypertension can be considered as a hypertensive syndrome caused by multiple cardiometabolic risk factors. Other studies demonstrated that more than 80% of hypertensive patients are complicated with different forms of metabolic abnormalities, while only 20% of hypertensive patients are not complicated with metabolic disturbances. [10,11] The cardiometabolic disturbances observed in this study are certainly related to tobacco consumption, sedentary lifestyle and low physical activity which were significantly predicted the odds of hypertension. Indeed, numerous cross-sectional and prospective epidemiological studies have shown a significant inverse association between regular physical activity and blood pressure. [27] Regular aerobic physical exercise reduces the risk of coronary heart disease. [29] This benefit would come from the decrease in blood pressure and the metabolic effects of physical exercise. [30]

The most prevalent cardiometabolic risk factors in hypertensive patients were hyperglycemia levels followed by dyslipidemia. In fact, hypertension is strongly associated with obesity, as well as a decrease in HDL-c. [31,32] A study conducted in Yaoundé (Cameroon) on 263 patients at risk or suffering from cardiovascular

diseases had revealed that the main metabolic disorder was HDL hypocholesterolemia (44.3%). [33] However, in this study, the prevalence of HDL hypocholesterolemia was significantly lower in hypertensive patients compared to normotensive participant. This would support the hypothesis that some therapies have limited the increase of the prevalence of HDL hypocholesterolemia in hypertensive patients. [34]

Overall, renal biochemical abnormalities were highly prevalent in hypertensive patients, especially ceatininemia (40.2%) and hypoglycemia (15.6%). These observations are alarming given that these parameters are markers of renal function impairment including glomerulonephritis and nephropathies, which cause high mortality in patients. [35] Hypoglycemia has been frequently associated with hypertension or its complications. [36] These results can be explained by the non-compliance with hygieno-dietary rules, which we have identified as risk factors for hypertension through the positive correlations that exist between these factors and hypertension, notably age, poor sport practice, and alcoholism. [37,38]

Of the 244 hypertensive patients, 40% are complicated by different forms of biochemical abnormalities. This result is lower compared with the report of Shanghai Hypertension Institute and Chongqing Hypertension Institute, who indicated that more than 80% of hypertensive patients are complicated by various forms of metabolic abnormalities, while only 20% of hypertensive patients are not complicated with metabolic disturbances. [10,11] Our results clearly suggested that strategies targeting the metabolic pathways may be therapeutic options for improving biochemical abnormalities and reducing blood pressure and risk of complications in metabolic hypertension. The high prevalence of metabolic disorders in hypertensive patients may be explained by high tobacco consumption and poor practice of physical activities, the lifestyle of population and family history of hypertension. Indeed, many studies have already demonstrated the beneficial effects of practice of physical activity on cardiometabolic risk factors and the harmful effects of alcoholism and tobacco on these same factors [39,40]

Hypertension is characterized by the hyperreactivity and remodelling of small resistance arteries. However, many studies demonstrate that hypertensive subjects with metabolic disturbances also suffer from macrovascular lesions, which cause decreases in vascular compliance, atherosclerosis and endothelial dysfunction. [41] The form of vascular damages is dependent on the types of metabolic risk factors. Dyslipidemia leads to macrovascular atherosclerosis, which induces monocyte adhesion and migration to the subendothelium, the uptake of oxidised low-density lipoprotein (LDL)-cholesterol by vascular smooth muscle cells and monocytes to form the foam cells, further development of fatty plaque, fibrosis and calcification, endothelial dysfunction and decreased vascular compliance. Hyperglycemia destroys the endothelium [42] and causes microvascular damage in the kidney and retina, although other mechanisms, such as activation of the protein kinase C, cellular polyol and hexosamine pathways as well as the formation of advanced glycation end products, could also be implicated. [43,44,45] The mechanisms responsible for obesity-induced vascular damage include endothelial dysfunction, insulin resistance, obstructive sleep apnoea, impaired baroreflex sensitivity, and the activation of the renin-angiotensin-aldosterone and sympathetic nervous systems. [46]

## 5. Conclusion

Overall, the results of the present study indicate that smoking, age group, obesity, sedentary lifestyle (behavioral risk factors), family history of hypertension (genetic risk factor), hypertriglyceridemia, hyperglycemia (cardiometabolic risk factors), hypernatremia, hyperchloremia, glycosuria, ceatininemia and hypoglycemia (renal metabolic disorders) are the main risk factors of hypertension in the study population. Of the 244 hypertensive patients, 40% are complicated by various forms of metabolic abnormalities. Hence, strategies targeting the metabolic pathways may be therapeutic options for improving biochemical abnormalities and reducing blood pressure and risk of complications in metabolic hypertension.

## Competing Interests

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

## References

[1] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19.1 million participants. Lancet 2017; 389(10064): 37-55.

[2] WHO (World Health Organization). A global brief on hypertension: silent killer, global public health crisis: World Health Day 2013. Geneva: WHO, 2013.

[3] WHO Steps Manual. International Society of Nephrology: Program for detection and management of chronic kidney disease, hypertension, diabetes and cardiovascular disease in developing countries (KHDC PROGRAM). Brussels, Int Soci Nephrol. 2005; 17-19.

[4] Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Worldwide prevalence of hypertension: a systematic review. J Hypertens. 2004; 22(1):11-9.

[5] Zhu Z, Wang P, Ma S. Metabolic hypertension: concept and practice. Front. Med. 2013; 7(2): 201-206.

[6] World Health Organization, Global Status Report on Noncommunicable Diseases 2010, World Health Organization, Geneva, Switzerland, 2011.

[7] Sharma AM, Pischon T, Hardt S, Kunz I, Luft FC. β-Adrenergic receptor blockers and weight gain. A systematic analysis. Hypertension 2001; 37: 250-254.

[8] Boschmann M, Engeli S, Adams F, Franke G. Influences of AT1 receptor blockade on tissue metabolism in obese men. Am. J. Physiol. Regul. Integr. Com. Physiol. 2006; 290: R219-R223.

[9] Mathai ML, Naik S, Sinclair AJ, Weisinger HS. Weisinger RS. Selective reduction in body fat mass and plasma leptin induced by angiotensin-converting enzyme inhibition in rats. Int. J. Obes. 2008; 32: 1576-1584.

[10] Chu S, Zhu D, Xiong M. Linkage analysis of candidate genes for glucose and lipid metabolism with essential hypertension. Nation Med J China (Zhonghua Yi Xue Za Zhi) 2001; 81(1): 20-22.

[11] Xu XS, Yang WT, Liu DY, Zhong J, Tian ZQ, Yan ZC, Ni YX, Chen J, Zhao ZG, Zhu ZM. Cardiovascular and Renal Damage in Hypertensive Complicated with Metabolic Disorders. Chin J Hypertens (Zhonghua Gao Xue Ya Za Zhi) 2006; 14(11): 894-898.

[12] Sowers JR, Epstein M, Frohlich ED. Diabetes, hypertension, and cardiovascular disease: an update. Hypertension 2001; 37(4): 1053-1059.

[13] Dekker JM, Girman C, Rhodes T, Nijpels G, Stehouwer CD, Bouter LM, Heine RJ. Metabolic syndrome and 10-year cardiovascular disease risk in the Hoorn Study. Circulation. 2005; 112(5): 666-673.

[14] Tanaka M, Itoh H. Hypertension as a Metabolic Disorder and the Novel Role of the Gut. Curr Hypertens Rep 2019; 21: 63.

[15] Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-densitylipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499-502.

[16] Lee SR, Cha MJ, Kang DY, Oh KC, Shin DH, Lee HY. Increased prevalence of metabolic syndrome among hypertensive population Ten years' trend of the Korean National Health and Nutrition Examination Survey. Int J Cardiol 2013; 166(3): 633-639.

[17] Borzecki A, Glickman M, Kader B, Berlowitz D. The effect of age on hypertension control and management," Am J Hypertens 2006; 19(5): 520-527.

[18] Manimunda SP, Sugunan AP, Benegal V, Balakrishna N, Rao MV, Pesala KS. Association of hypertension with risk factors & hypertension related behaviour among the aboriginal nicobarese tribe living in car nicobar island. India. Indian J Med Res 2011; 133(3): 287-293.

[19] Mahmood SE, Srivastava A, Shrotriya VP, Shaifali I, Mishra P, Prevalence and epidemiological correlates of hypertension among labour population, Nat J Community Med 2011;2(1): 43-48.

[20] Laxmaiah A, Meshram II, Arlappa N, Balakrishna N, Rao KM, Reddy ChG, Ravindranath M, Kumar S, Kumar H, Brahmam GN. Socio-economic & demographic determinants of hypertension & knowledge, practices & risk behaviour of tribal in India, Indian J Med Res 2015; 141(5): 697-708.

[21] Awino BO, Ogonda LA, Barno G. C, Ng'wena GM. Awareness status and associated risk factors for hypertension among adult patients attending yala sub-county hospital, siaya county, Kenya. Public Health Res 2016; 6(4): 99-105.

[22] Krzesinski J. Epidémiologie de l'hypertension artérielle. Rev Med Liege 2002 ;57(3): 142-47.

[23] Belmin J. Actualités sur l'hypertension artérielle. L'hypertension du sujet âgé. Presse Med 1999 ;28(16): 862-869.

[24] Llyod-Jones D, Evanx J, Levy D. Hypertension in adults across the age spectrum: current outcomes and control in the community. J Am Med Assoc 2005; 294(4): 466-472.

[25] Nielsen G, Andersen L. The association between high blood pressure, physical fitness, and body mass index inadolescents. Prev Med 2003; 36(2): 229-234.

[26] Fagard R. Physical activity, physical fitness and the incidence of hypertension. J Hypertens 2005; 23(2): 265-267.

[27] Cornelissen V, Fagard R. Effects of endurance training on blood pressure, blood pressure - regulating mechanisms, and cardiovascular risk factors. Hypertension 2005; 46(4): 667-675.

[28] Saha M, Sana N, Ranajit K. Serum lipid profile of hypertensive patients in the northern region of Bangladesh. J Biosci 2006; 14: 93-98.

[29] Arakawa K. Effect of exercise on hypertension and associated complications. Hypertens Res 1996;19(1): 87-91.

[30] Longo-Mbenza B. Diabète sucré et maladies cardiovasculaires. Cardiol Trop 1995; 21(82): 37-44.

[31] Landsberg L, Aronne L, Beilin L, Burke V, Igel L, Lloyd-Jones D, Sowers J. Obesity-related hypertension: pathogenesis, cardiovascular risk, and treatment. J Clin Hypertens 2013; 15: 14-33.

[32] Kannel W. Risk stratification in hypertension: new insights from the Framingham Study. Am J Hypertens 2000; 13: 3S-10S.

[33] Moor AVJ, Amougou S, Ombotto S, Ntone F, Wouamba D, Nonga B. Dyslipidemia in Patients with a Cardiovascular Risk and Disease at the University Teaching Hospital of Yaoundé, Cameroon. Int J Vasc Med 2017; Article ID 6061306.

[34] Hackam D, Khan N, Padwal R, Tobe S. Primary prevention of CVD: treating hypertension. BMJ Clin Evid 2010; 1-21.

[35] McCullough P, Li S, Jurkovitz C, Stevens L, Wang C, Collins A, Chen S, Norris KC, McFarlane S, Johnson B, Shlipak M, Obialo C, Brown W, Vassalotti J, Whaley-Connell A. CKD and cardiovascular disease in screened high-risk volunteer and general populations: the Kidney Early Evaluation Program (KEEP) and National Health and Nutrition Examination Survey (NHANES) 1999-2004. Am J Kidney Dis 2008; 51(2): S38-45.

[36] Wright R, Frier B. Vascular disease and diabetes: is hypoglycaemia an aggravating factor? Diabetes Metab Res Rev 2008; 24: 353-363.

[37] Stranges S, Wu T, Dorn JM, Freudenheim JL, Muti P, Farinaro E, Russell M, Nochajski TH, Trevisan M. Relationship of Alcohol Drinking Pattern to Risk of Hypertension: A Population-Based Study. Hypertension 2004; 44(6): 813-819.

[38] Aguilera M, de la Sierra A, Coca A, Estruch R, Fernández-Solá J, Urbano-Márquez A. Effect of alcohol abstinence on blood pressure: assessment by 24-hour ambulatory blood pressure monitoring. Hypertension 1999; 33(2): 653-657.

[39] Eman M, Gordon A. Dietary fruits and vegetables and cardiovascular diseases risk. Crit Rev Food Sci Nutr 2015; 57(9): 1950-1962.

[40] Bell J, Sabia S, Singh-Manoux A, Hamer M, Kivimaki M. Healthy obesity and risk of accelerated functional decline and disability. Int J Obes 2017; 41: 866-872.

[41] Caballero AE. Endothelial dysfunction in obesity and insulin resistance: a road to diabetes and heart disease. Obes Res 2003; 11(11): 1278-1289.

[42] Cersosimo E, DeFronzo RA. Insulin resistance and endothelial dysfunction: the road map to cardiovascular diseases. Diabetes Metab Res Rev 2006; 22(6): 423-436.

[43] Milicevic Z, Raz I, Beattie SD, Campaigne BN, Sarwat S, Gromniak E, Kowalska I, Galic E, Tan M, Hanefeld M. Natural history of cardiovascular disease in patients with diabetes: role of hyperglycemia. Diabetes Care 2008; 31(2): S155-160.

[44] Orasanu G, Plutzky J. The pathologic continuum of diabetic vascular disease. J Am Coll Cardiol 2009; 53(5): S35-S42.

[45] Gao L, Mann GE. Vascular NAD(P)H oxidase activation in diabetes: a double-edged sword in redox signalling. Cardiovasc Res 2009; 82(1): 9-20.

[46] Razani B, Chakravarthy MV, Semenkovich CF. Insulin resistance and atherosclerosis. Endocrinol Metab Clin North Am 2008; 37(3): 603-621.