Full Length Research Paper

# The role of Plasma Fibrinogen and Uric Acid Levels in relation to Myocardial Infarction risk in patients with the Metabolic Syndrome- Preliminary findings in the Littoral Region of Cameroon

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Background: Information on relationship between some biomarkers (uric acid and plasma fibrinogen) to the risk of myocardial infarction in patients with the metabolic syndrome among Africans is inexistent or scanty at best. Moreover there is a burden of myocardial infarction and metabolic syndrome on the population.

Aims: The objective of this study was to establish the correlation: plasma fibrinogen and uric acid to myocardial infarction risks in patients with the metabolic syndrome.

Method— A total 68subjects (34 patients and 34 healthy controls) aged between 18 -80 years were recruited for this study between November 2010 and March 2011.Patients for the study as well as the controls were recruited from Douala and were matched for sex ,age and place of residence. Trained nurses, using standard procedures, obtained blood pressure and anthropometric measurements and collected a venous blood sample for assay of plasma fibrinogen and uric acid. Serum uric acid was measured enzymatically after hydrolyzation to glycerol, Plasma glucose concentration was measured using an enzymatic reaction. Plasma fibrinogen was estimated by the Hook's procedure. Data were analyzed using SPSS version 15 ...

Results: Negative correlations were record between plasma fibrinogen and FBS, height, pulse, WHR and systolic pressure .Uric acid did not show any negative correlation with any of the indicators. There was no correlation between uric acid and fibrinogen (r=-0.056; p=0.599). As for uric acid, it showed very strong correlation with weight, height, waist circumference, diastolic pressure and body mass index.)

Plasma fibrinogen showed the strongest correlation with Hip circumference (r=0.362, p<0.001)) while with the waist circumference showed a relatively weaker though significant correlation (r=0.240, p=0.023) Plasma fibrinogen did not show any significant difference between patients group and control groups and did not show any significant correlation with the determinants of the metabolic syndrome

Conclusions: Uric acid and not plasma fibrinogen may be considered as a component of the metabolic syndrome. Further investigation is necessary to confirm the results of this preliminary study.

# INTRODUCTION

Metabolic syndrome is evolving into a pandemic, contributing to approximately 6-7% for all causes of mortality, 12–17% for cardiovascular disease, and 30–52% for diabetes in the population

(1).Metabolic syndrome significantly increases the risk for cardiovascular disease and chronic kidney disease (2) and from a recent prospective study, may play a role in the development of some tumors like vulvar and vaginal cancers (3).The increased risk for cardiovascular diseases can partly be caused by a

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prothrombotic state that exists because of abdominal obesity(2).

Various combinations of the following five risk factors constitute the basis for the different definitions of the Metabolic Syndrome: obesity (total body obesity measured by body mass index, or central obesity measured by waist-to hip ratio waist or circumference), atherogenic dyslipidemia (increased decreased high-density triglycerides, lipoprotein cholesterol); elevated blood pressure (systolic and diastolic, abnormal glucose tolerance (fasting blood glucose, 2-hour postprandial blood glucose), and insulin resistance measured by the homeostasis model assessment (HOMAIR) or fasting insulin( 5,6). Recent findings suggest that plasma fibrinogen and uric acid should be added to the list of determinants or biomarkers of MS (7, 8).

Clearly more studies are needed to better understand the role of uric acid in metabolic syndrome, but it seems likely that uric acid may have a role as both a marker and potential modifier of the metabolic syndrome (7).

The topical role of uric acid and its relation to cardiovascular disease, renal disease, and hypertension is rapidly evolving. Its important role both historically and currently in the clinical clustering phenomenon of the metabolic syndrome (MS), type 2 diabetes mellitus (T2DM), atheroscleropathy, and non-diabetic atherosclerosis is of great importance (9).

Hyper-uricaemia is associated with the MS and its prevalence is comparable in both genders and in subjects with and without hypertension(10).

The possible predictors of hyperuricaemia include centripetal obesity, significant smoking history and elevated serum TG

Elevated serum uric acid levels (SUA) have been associated with an increased risk of cardiovascular diseases and the metabolic syndrome (MS) and are often reported to be higher in females than in males (10). Hyperuricemia is only weakly associated with renal function, but is strongly associated with M S with or without a low Glomerular Filtration Rate (GFR). (11). It has been demonstrated that hyperuricemia predicts cardiovascular events in the general population, the hypertensive population, and patients with pre-existing Furthermore CVD. hyperuricemia predicts the development of future hypertension (12)].

High plasma fibrinogen levels are associated with an increased risk for myocardial infarction (MI). (13,14) To date, it is not clear whether an elevated plasma fibrinogen level itself increases the risk for MI (risk factor) or whether the elevated level is merely a reflection of the presence of preclinical atherosclerosis or of an association with a true risk factor (risk indicator). Atherosclerosis has multiple underlying causes. Unless every one of these risk factors is

corrected, aging adults will continue to suffer epidemic levels of vascular diseases, including heart attack and stroke..

A number of studies show elevated fibrinogen to be a major risk factor for coronary heart disease (heart attacks) and cerebrovascular disease (strokes), which together account for about 60% of deaths in the elderly. In fact, fibrinogen may possibly be the major risk factor, "contributions" of homocysteine, exceeding the cholesterol and other lipids in the pathogenesis of these diseases. Elevated fibrinogen levels have also been associated with a number of other diseases, including cancer, diabetes and hypertension (15). For coronary heart disease, the fibrinogen level is significant for both men and women and elevated fibrinogen level is a predictor of cardiovascular disease that should be added to the cardiovascular risk factor profile.(15)

In middle-aged healthy Japanese men without MS, not only severe, but also mild hyperuricemia may be a significant independent risk factor for endothelial dysfunction in subjects without MS, whereas only severe hyperuricemia(but not mild hyperuricemia) appeared to exacerbate endothelial dysfunction in similar subjects with MS (8). According to (7) it seems likely that uric acid may have a role as both a marker and potential modifier of the metabolic syndrome although these investigators recommended that more studies be carried out it seems likely that uric acid may have a role as both a marker and potential modifier of the metabolic syndrome. Working on Korean adults,(16) 2009, reported that hyperuricaemia Lee was independently associated with hepatic steatosis regardless of BMI category or the presence of the MS. Metabolic syndrome, previously considered as a disease of rich countries has become an important health harzard in Africa as rapid urbanization has encouraged sedentary lifestyles and modified dietary patterns(31). Recent studies in Cameroon and other sub Saharan countries have considered anthropometric determinants but limited the biochemical indices to glucose and dyslipidemia. It is known that elevated uric acid and plasma fibrinogen play in myocardial infarction.(,13,14,15). Several studies have indicted seum uric acid and plasma fibrinogen as risk factors of MS(11,7) but very few of these studies have been conducted in Africa(10) where the diet is predorminantly carbohydrates rich and proteins/lipids poor. No such studies has been carried out in Cameroon.

The aim of this study was to determine the roles of uric acid and plasma fibrinogen in relation to myocardial infarction risk in patients with the MS.

# METHODS

Patient type		Systoloic	Diastolic	Pulse	Waist circumference	Hip circumference	Height	Weight
Patient	N	34	34	34	34	34	34	34
	Mean	141.1176	86.1765	77.8529	109.2941	119.9412	1.6821	95.5894
	Std. Error of Mean	3.21143	2.39595	1.79429	1.64741	1.67977	.01280	2.48450
	Minimum	107.00	50.00	51.00	82.00	97.00	1.57	63.21
	Maximum	176.00	115.00	102.00	130.00	140.00	1.82	134.60
	Std. Deviation	18.72572	13.97068	10.46244	9.60596	9.79468	.07462	14.48699
	Median	144.5000	84.0000	79.5000	108.5000	119.5000	1.6700	94.8000
Control	N	34	34	34	34	34	34	33
	Mean	117.3235	72.0588	72.0588	90.4412	106.0294	1.6641	73.8579
	Std. Error of Mean	2.40607	1.69193	1.79519	1.97588	1.92760	.02298	2.41609
	Minimum	93.00	47.00	58.00	74.00	91.00	1.00	53.70
	Maximum	142.00	93.00	99.00	126.00	140.00	1.80	105.90
	Std. Deviation	14.02967	9.86559	10.46767	11.52124	11.23977	.13401	13.87938
	Median	118.0000	71.5000	69.0000	88.0000	105.0000	1.7000	68.8000
Total	Ν	68	68	68	68	68	68	67
	Mean	129.2206	79.1176	74.9559	99.8676	112.9853	1.6731	84.8858
	Std. Error of Mean	2.46537	1.69186	1.30835	1.71932	1.52712	.01310	2.17915
	Minimum	93.00	47.00	51.00	74.00	91.00	1.00	53.70
	Maximum	176.00	115.00	102.00	130.00	140.00	1.82	134.60
	Std. Deviation	20.33000	13.95144	10.78893	14.17787	12.59293	.10803	17.83713
	Median	128.0000	78.0000	75.0000	100.0000	112.5000	1.6900	86.5000
IST test		<0.001	<0.001	0.026	<0.001	<0.001	0.498	<0.001

Table 1: Components of the Metabolic Syndrome in patients and controls

A total 68subjects(34 MS patients and 34 healthy controls) aged between 8-80 were randomly selected by systematic sampling from their primary health care area were recruited for this study between November 2010 and March 2011.Patients for the study as well as the controls were recruited from Douala and was marched for sex, age and place of residence. Trained nurses obtained blood pressure and anthropometric measurements and collected a venous blood sample for assay of plasma fibrinogen , uric acid and the lipid profile.

Waist circumference was measured with a Gulick II spring-loaded measuring tape (Gay Mills, WI) midway between the inferior angle of the ribs and the superior iliac crest (ie at the high point of the iliac crest) at minimal respiration to the nearest 0.1 cm. Hip circumference was measured at the outermost points of the greater trochanters. WHR was recorded to the nearest 2 decimal places.

Serum uric acid and lipid profile were assayed using the Kenza 120TX Biochemistry analyser of Bilabo Diagnostics . Plasma glucose concentration was measured using an enzymatic reaction. Plasma fibrinogen was estimated by the Hook's procedure based on Von Clauss (1957) studies, validated by Destaing F(1960).

#### **Statistical Analysis**

Data were analyzed using SPSS version 15. Categorical variables were analysed using chi-square tests. Independent t-test was used to compare continuous variables among the group with hyperuricaemia and with the normouricaemic group. Pearson correlation coefficient determination was performed to evaluate the degree of association between uric acid, plasma fibrinogen and various clinical and biochemical parameters. Quantitative data are expressed as mean and standard deviation (SD). P values of < 0.05 were considered to be statistically significant.

				Plasma	Total	High density	Low density lipoprotei n cholester	Trialyceri
Patient t	уре	FBS	Uric Acid	Fibrinogen	cholesterol	cholesterol	ol	de
Patient	Ν	34	34	34	32	32	32	32
	Mean	1.2862	62.9412	3.1468	2.0700	.4594	1.4616	.7756
	Std. Error of Mean	.05860	2.81909	.09316	.03196	.01872	.03156	.04808
	Minimum	.87	34.00	1.66	1.66	.31	1.04	.38
	Maximum	2.31	97.00	4.53	2.86	.79	1.98	1.51
	Std. Deviation	.34171	16.43797	.54320	.18077	.10589	.17851	.27196
	Median	1.1950	58.0000	3.2250	2.0700	.4350	1.5000	.7600
Control	Ν	34	34	34	32	32	32	32
	Mean	.9232	51.3176	2.9659	2.0697	.4994	1.4191	.6966
	Std. Error of Mean	.01745	2.71106	.10684	.02772	.02564	.03630	.09105
	Minimum	.71	24.00	1.48	1.57	.32	.85	.31
	Maximum	1.22	90.00	4.53	2.54	.94	1.79	3.17
	Std. Deviation	.10176	15.80804	.62300	.15679	.14505	.20537	.51505
	Median	.9250	51.0000	2.9350	2.0550	.4600	1.4500	.5800
Total	Ν	68	68	68	64	64	64	64
	Mean	1.1047	57.1294	3.0563	2.0698	.4794	1.4403	.7361
	Std. Error of Mean	.03758	2.06672	.07121	.02098	.01595	.02401	.05131
	Minimum	.71	24.00	1.48	1.57	.31	.85	.31
	Maximum	2.31	97.00	4.53	2.86	.94	1.98	3.17
	Std. Deviation	.30989	17.04259	.58720	.16786	.12758	.19207	.41050
	Median	1.0100	56.0000	3.0800	2.0600	.4550	1.4700	.6500
IST test		<0.001	0.004	0.206	0.994	0.212	0.380	0.445

### RESULTS

### CASE CONTROL STUDY:

### Sample description

A total of 68 subjects comprising 34 patients diagnosed positive for the metabolic syndrome and 34 negative controls matched for age, sex, and area of residence were used for this case control study for which the case summary statistics are summarized in Table 32. There was however no significant association between participant type and sex ( $\chi$ 2-test:  $\chi$ 2=0.179; DF=1; p=0.673), participant type and marital status ( $\chi$ 2-test:  $\chi$ 2=4.602; DF=1; p=0.100) and participant type and level of education ( $\chi$ 2-test:  $\chi$ 2=2.429; DF=3; p=0.488)(Tables 1,2,3, 4,35)

In the same line, 30 (66.7%) of patients were aged above 41 years against a significantly lower proportion for the control group 15 (33.3%) ( $\chi$ 2-test:  $\chi$ 2=10.000; df=1; p=0.002).

There was however no significant association between participant type and sex ( $\chi$ 2-test:  $\chi$ 2=0.179; df=1; p=0.673), participant type and marital status ( $\chi$ 2-test:



Figure 1: Comparative Boxplots of biochemical indices for patient and control groups

There was a significant difference between patients and controls for fasting blood sugar and uric acid but plasma fibrinogen and HDLc like the other lipid fractions did not show any significant differences between the patients and controls.

Table 3: Components of	Netabolic Syndrome in	patients and controls
	5	1

Patient type		Systoloic	Diastolic	Pulse	Waist circumference	Hip circumference	Height	Weight	
Patient	Ν		34	34	34	34	34	34	34
	Mean		141.1176	86.1765	77.8529	109.2941	119.9412	1.6821	95.5894
	Std. Mean	Error	of 3.21143	2.39595	1.79429	1.64741	1.67977	.01280	2.48450
	Minimu	um	107.00	50.00	51.00	82.00	97.00	1.57	63.21
	Maxim	um	176.00	115.00	102.00	130.00	140.00	1.82	134.60
	Std. De	eviation	18.72572	13.97068	10.46244	9.60596	9.79468	.07462	14.48699
	Mediar	۱	144.5000	84.0000	79.5000	108.5000	119.5000	1.6700	94.8000
Control	Ν		34	34	34	34	34	34	33
	Mean		117.3235	72.0588	72.0588	90.4412	106.0294	1.6641	73.8579
	Std. Mean	Error	of 2.40607	1.69193	1.79519	1.97588	1.92760	.02298	2.41609
	Minimu	um	93.00	47.00	58.00	74.00	91.00	1.00	53.70
	Maxim	um	142.00	93.00	99.00	126.00	140.00	1.80	105.90
	Std. De	eviation	14.02967	9.86559	10.46767	11.52124	11.23977	.13401	13.87938
	Mediar	ſ	118.0000	71.5000	69.0000	88.0000	105.0000	1.7000	68.8000
Total	N		68	68	68	68	68	68	67
	Mean		129.2206	79.1176	74.9559	99.8676	112.9853	1.6731	84.8858
	Std. Mean	Error	of 2.46537	1.69186	1.30835	1.71932	1.52712	.01310	2.17915
	Minimu	um	93.00	47.00	51.00	74.00	91.00	1.00	53.70
	Maxim	um	176.00	115.00	102.00	130.00	140.00	1.82	134.60

	Std. Deviation	20.33000	13.95144	10.78893	14.17787	12.59293	.10803	17.83713
	Median	128.0000	78.0000	75.0000	100.0000	112.5000	1.6900	86.5000
IST test		<0.001	<0.001	0.026	<0.001	<0.001	0.498	<0.001

 $\chi$ 2=4.602; df=1; p=0.100) and participant type and level of education ( $\chi$ 2-test:  $\chi$ 2=2.429; df=3; p=0.488).

# Determinants of Metabolic Syndrome in patients and control group

As indicated in Table 1 below, height and plasma fibrinogen were the only parameters for which there were no significant difference between patients and control groups (p=0,436 and 0.835) The remaining indicators[systolic blood pressure, diastolic blood pressure, pulse, waist circumference, hip circumference, weight, fasting blood glucose and uric acid all showed significant differences (p<0,001) between the patients and control groups.

# Correlations between other determinants of MS with UA and Plasma Fibrinogen[PF]

Negative correlations were registered between plasma fibrinogen and FBS, height, pulse, WHR, systolic pressure. Uric acid did not show any negative correlation with any of the indicators. No correlation uric between acid and fibrinogen (r=-0.056; p=0.599).Plasma fibrinogen showed the strongest correlation with Hip circumference (r=0.362, p<0.001)) while with the waist circumference showed a relatively though weaker significant correlation (r=0.240,p=0.023)As for uric acid, it showed very strong

correlation with weight, height, waist circumference, and diastolic pressure and Body mass index.

### Indicators of MS in patients and controls

For all the key indicators of MS [elevated blood sugar, hypertensive condition, High BMI, elevated WHR, Elevated waist circumference and elevated uric acid] there was a significant difference between the patients and the controls as shown in Table 5. Plasma fibrinogen did not show any significant difference between patients and control groups.

# Life style options in Patients and Controls

Of the 14 lifestyle options (Table 5) considered in this study, only 5 showed somewhat significant differences

between the patients and the control group. These include sedentary lifestyle, consumption of less than one litre of water a day, having gained more than 10kg of body weight within the past 10 years (p<0.001), diabetes history and consumption of rapid sugars.

### DISCUSSION

The present study investigated the role of uric acid and plasma fibrinogen in relation to myocardial infarction risks in patients with the metabolic syndrome. Uric acid and not plasma fibrinogen may be considered as a component of the metabolic syndrome. .Negative correlations were registered between plasma fibrinogen and FBS, height, pulse, WHR, systolic pressure .Uric acid did not show any negative correlation with any of the indicators. There was no correlation between uric acid and fibrinogen (r=-0.056; p=0.599). As for uric acid. it showed very strong correlation with weight, height, waist circumference, diastolic pressure and body mass index. Plasma fibrinogen showed the strongest correlation with Hip circumference (r=0.362, p<0.001)) while with the waist circumference showed a relatively weaker though significant correlation (r=0.240, p=0.023) Plasma fibrinogen did not show any significant difference between patients and control groups and did not show any significant correlation with the determinants of the metabolic syndrome. Our results contrast sharply those of other investigators (19) who reported that hyper fibrinogenemia could be considered a component of the MS, but also that this fact explained the increased cardiovascular risk associated with hyperinsulinemia or insulin resistance. Another contrasting result to ours was reported by (20) who observed that patients without metabolic syndrome and in the lowest quartile of plasma fibrinogen levels had the lowest incidence of fatal/nonfatal coronary events or death, whereas those with metabolic syndrome and in the highest quartile of plasma fibrinogen levels had the highest incidence of these events. A number of studies (13,14,15,19,20) show elevated fibrinogen to be a major risk factor for coronary heart disease (heart attacks) and cerebrovascular disease (strokes), which together account for about 60% of deaths in the elderly. In fact, fibrinogen may possibly be the major risk factor, exceeding the "contributions" of homocysteine, cholesterol and other lipids in the pathogenesis of these diseases. Besides elevated fibrinogen levels have also been associated with a number of other diseases, including cancer, diabetes and hypertension .High plasma fibrinogen

	Uric acid			Plasma fibrinogen			
	r	р	n	r	р	Ν	
FBS (g/l)	0.218*	0.039	90	-0.077	0.470	90	
Weight (kg)	0.338**	0.001	89	0.130	0.226	89	
Height (m)	0.389**	<0.001	90	-0.110	0.300	90	
HC (cm)	0.093	0.381	90	0.362**	<0.001	90	
WC (cm)	0.278**	0.008	90	0.240*	0.023	90	
Pulse (b/min)	0.064	0.552	90	-0.029	0.785	90	
Diastolic (mmHg)	0.261*	0.013	90	0.055	0.607	90	
Systolic (mmHg)	0.338**	0.001	90	-0.028	0.794	90	
WHR	0.159	0.137	89	-0.076	0.478	90	
BMI (Kg/m <sup>2</sup> )	0.388**	<0.001	90	0.204	0.056	90	

Table 4.	Correlations	between other	determinants of MS with	UA and Plasma	fibrinogen[PF]
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\* Correlation is significant at the 0.05 level \*\* Correlation is significant at the 0.01 level. No correlation between uric acid and fibrinogen (r=-0.056;

p=0.599).

Indicators Syndrome	of Metaboli	<sup>C</sup> Patients	Control	Chi-square (P-Value)
	FBS 0.7 -1.1	5(11.1%)	37(82,2%)	
Diabetes	FBS 1.1 -1.26	20(44.4%)	8(17.8%)	<0.001
	FBS > 1.26	20(44.4%)	0(0.0%)	-
Hypertensive condition		18(40.0%)	1(2.2%)	<0.001
Highl BMI		32(71.1%)	11(25.0%)	<0.001
Elevated WHF	8	32(71.1%)	10(23.8%)	<0.001
Elevated Wai 2005	st circumference IDI	F 45(100.0%)	25(55.6%)	<0.001
Elevated plasma fibrinogen		2(4.4%)	3(6.7%)	0.645
Elevated uric a	icid	19(42.2%)	12(26.7%)	<0.001
MS IDF FBS (a	at 1.0 g/l)	20(44.4%)	0(0.0%)	<0.001

Table 5. Lifestyle options in	Patients and Controls
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Life style option indicators	Patients	Control	Chi-square (P-Value)
Sedentary life style	34(79.1%)	24(53.3%)	0.011
Smoker	3(6.1%)	4(9.5%)	0.774
Consume less than 1 liter of water per day	3(6.7%)	10(22.2%)	0.014
Gained more than 10 Kg over the past ten years	40(90.9%)	21(48.8%)	<0.001
Unhealthy dietary pattern	33(73.3%)	28(65.1%)	0.403
Unhealthy source of protein	33(73.3%)	30(68.2%)	0.593
Does not have parents who have live more than 75 years	11(24.4%)	8(18.6%)	0.562
Cardiovascular history	25(59.5%)	19(46.3%)	0.405
Diabetes history	26(57.8%)	15(34.1%)	0.015
Obesity history	26(59.1%)	19(43.2%)	0.297
Gout history	13(29.5%)	12(27.3%)	0.877
Consume rapid sugar	28(62.2%)	16(35.6%)	0.018
Healthy sleeping pattern	28(62.2%)	25(56.8%)	0.552
Heavy alcohol consumer	12(27.9%)	10(27.7%)	0.576

levels are associated with an increased risk for myocardial infarction (MI)(13,14) .To date, it is not clear whether an elevated plasma fibrinogen level itself increases the risk for MI (risk factor) or whether the elevated level is merely a reflection of the presence of preclinical atherosclerosis or of an association with a true risk factor (risk indicator)

Atherosclerosis has multiple underlying causes. Unless every one of these risk factors is corrected, aging adults will continue to suffer epidemic levels of vascular diseases, including heart attack and stroke. Recent scientific studies have validated that atherosclerosis is indeed a multi factorial process, and that taking aggressive preventive actions can dramatically reduce one's chances of dying from cardiovascular disease (13 .14) More recent studies have shown strona association between serum inflammatory parameters(including plasma fibrinogen levels, alpha 1 2 globulin, and accelerated erythrocyte and sedimentation rate) and diagnosed diabetes mellitus type 2(Music et al, 2010). Since elevated blood sugars levels is a key component of metabolic syndrome and also a risk factor for cardio vascular diseases, this fact confirms the strong likelihood that hyper in itself fibrinogenemia be considered as a component of the MS. Unfortunately our findings seem to contrast sharply with this fact since we observed negative correlations between plasma fibrinogen on one hand and fasting

blood sugar, height, pulse, WHR and systolic pressure on the other. Vorster et al,(1998)(24) reported ethnic variation in plasma fibrinogen levels. They observed that the mean fibrinogen (thrombin time coagulation method) of men and women were higher than published data for Europeans but slightly lower than values of black Americans. Ethnic factors might explain our results. Thus more studies with a larger sample size in another multiethnic city in Cameroon might be necessary.

Uric acid did not show any negative correlation with any of the indicators of MS. Instead, our results showed very strong correlation with weight, height, waist circumference, diastolic pressure and body mass index. This further confirms the fact that elevated uric acid could be considered a determinant or component of metabolic syndrome. This is consistent with the findings of several investigators (21,12,10,7). Working on Nigerian subjects, some investigators reported that hyper-uricaemia was associated with the MS and its prevalence comparable in both genders and in subjects with and without hypertension. Their findings revealed that possible predictors of hyperuricaemia include centripetal obesity, significant smoking history and elevated serum triglyceride(10). This is consistent with our findings as waist circumference, diastolic hypertension and BMI all show very strong correlation with the MS. Other investigators (22) reported that hyperuricemia was strongly associated with M S which is consistent with our findings .Elevated serum uric acid levels (SUA) have been associated with an increased risk of cardiovascular diseases and the metabolic syndrome (MS) and are often reported to be higher in females than in males(10). It has been demonstrated that hyperuricemia predicts cardiovascular events in the general population, the hypertensive population, and patients with pre-existing CVD. Furthermore hyperuricemia predicts the development of future hypertension (12)] .The strong correlation observed in our study between uric acid levels and the cardiac risk factors which are also deterninants of MS further strengthens the predictive value of uric acid for cardiovascular events.

Working on 26,903 multi-ethnic subjects, (18) reported that, the risk of MS on MI is generally comparable to that conferred by some, but not all, of its component risk factors. They emphasized that the characterization of risk factors, especially continuous variables, as dichotomous will underestimate risk and decrease the magnitude of association between MS and MI (18) . Inflammation and metabolic syndrome have additive and not multiplicative joint effects on coronary events and death. Other investigators (23) from their cross sectional studies suggested that plasma viscosity was associated with increased clustering of metabolic markers in middle-aged men of high socio-economic status.

In another study (26) Heart Failure (HF) was frequent among Cameroonian patients treated for hypertension and was regularly associated with co-morbidities[ renal (24.3%), impairment overweight and obesity (20.7%), chronic obstructive pulmonary disease (17.1%), gout (16.4%), anaemia (15.7), diabetes mellitus (13.5%), atrial fibrillation (12.9%), stroke (9.3%), and ischaemic heart disease (5.7%).] In Nigeria, hypertension and rheumatic heart disease are among the principal causes of heart failure (24,27) . Climate, genetic, and socioeconomic factors are possible reasons for these differences, but they operate between and within developing countries.(27).

In our study, the lifestyle options showing significant associations with MS [sedentary lifestyle, consumption of less than one liter of water a day, having gained more than 10kg of body weight within the past 10 years(p<0.001), diabetes history and consumption of rapid sugars] are all risk factors for cardiovascular pathologies. According to (28), resistance training was effective in improving body composition of middle-aged obese sedentary males. Only aerobic training was effective in raising HDL cholesterol and they recommended more studies were warranted to assess the effects of exercise on plasma fibrinogen and micro albuminuria .Both aerobic and resistance training must be recommended to both urban and peri urban dwellers in Cameroon in order to lower the HDLcholesterol and burn fats. The effect of urbanization. migration of youths from the urban to the rural areas with the attendant lifestyle maladjustments might exarcebate the problem. Sedentary lifestyle is eating deep into the peri-urban and rural populations of Cameroon especially with the recent introduction of motocycle taxi which now convey low income periurban dwellers to their farms and now rival four wheeled taxis in the urban centers. Thus appropriate lifestyle adjustments on the modifiable risk factors of metabolic syndrome should be the focal point for both prevention of cardiovascular diseases the as recommended by some investigators. (29)

There was no significant difference in the levels of plasma fibrinogen between MS patients and control group. Besides our study thus shows a very strong correlation between uric acid levels and the determinants of metabolic syndrome, most of which determinants are also myocardial risk factors. No such relationship was obtained with plasma fibrinogen levels contrary to other reports. This suggests that further research probably with a larger sample size in another city is necessary to confirm the results from this preliminary finding.

### CONCLUSION

Uric acid and not plasma fibrinogen may be considered as a component of the metabolic syndrome. Further investigation is necessary to confirm the results of this preliminary study.

### **Authors' Contribution**

GKT contributed in designing the study, collecting data, running the laboratory work, analyzing the data, and drafted the paper. SPC, JD and AN contributed in the pilot study and data collection. VPKT contributed in designing the entire project and supervised it all through. All the authors have read the final manuscript and approved the submission.

#### REFERENCES

E S Ford (2005). Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: a summary of the evidence," Diabetes Care, vol. 28, no. 7, pp. 1769–1778.

Vykoukal Daynene, Mark G Davies (2011). Metabolic Syndrome and Outcomes after Renal Intervention Review Article Cardiology Research and Practice Vol. 2011, Article ID 781035, 4 pages doi:10.4061/2011/781035

Nagel G, H Concin, T Bjørge, K Rapp1, J Manjer, G Hallmans, G Diem, C ggström, A Engeland, M Almquist, H Jonsson, R Selmer, T Stocks, S Tretli, H Ulmer, P Stattinand A. Lukanova Metabolic syndrome and rare gynecological cancers in the Metabolic syndrome and Cancer project (Me-Can) Annals of Oncology, Vol. 22, 6 Pp. 1339-1345

Kassi E, Panagiota Pervanidou, Gregory Kaltsas, George Chrousos (2011). Review Metabolic syndrome: definitions and controversies.BMC Medicine, 9:48doi:10.1186/1741-7015-9-48

Alberti KG, Zimmet P, Shaw J (2006). Metabolic Syndrome—new world-wide definition. A consensus statement from the International Diabetes Federation. Diabetes Med.;23:469–480.. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.Executive summary of the Third Report of the National Cholesterol Education Program.

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001;285:2486–2497

Lanaspa A, Miguel Y, Sautin Yuri, Ahsan Ejaz, A; Madero Magdalena Le, MyPhuong Manitius, Jacek Gabriela Sanchez-Lozada, Laura, Nakagawa, Takahiko J. Johnson, Richard Uric acid and Metabolic Syndrome: What is the Relationship? Current Rheumatology Reviews, Vol. 7, (2). May 2011, pp. 162-169(8).

Tomiyama Hirofumi, Yukihito Higashi Bonpei Takase, Kohichi Node, Masataka Sata Teruo Inoue, Yutaka Ishibashi, Shinichiro Ueda, Kenei Shimada and Akira YamashinaRelationships Among Hyperuricemia, Metabolic Syndrome, and Endothelial Function. Am. J. Hyperten. 2011; 24 7, 770–774. doi:10.1038/ajh.2011.55

Hayden Melvin R, Suresh C Tyagi (2005). Uric acid: A new look at an old risk marker for cardiovascular disease, metabolic syndrome, and type 2 diabetes mellitus: The urate redox shuttle Nutrition & Metabolism 2004, 1:10 doi:10.1186/1743-7075-1-10]

Ogbera AO, Azenabor AO, (2010). Hyperuricaemia and the metabolic syndrome in type 2 DM. Diabetol Metab Syndr. Apr 20;2:24.

Lai-Chu See, Chang-Fu Kuo, Fang-Hsiu Chuang, Yu-Ming Shen, Yu-Shien Ko, Yu-Ming Chen, Kuang-Hui Yu (2010) Hyperuricemia and metabolic syndrome: associations with chronic kidney disease Clinical Rheumatology Volume 30, Number 3, 323-330

Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, Tuttle KR, Rodriguez-Iturbe B, Herrera-Acosta J, Mazzali M (2003). Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease? Hypertension, 41(6):1183-1190. PubMed Abstract | Publisher Full Text

[Li JJ, Chen JL (2005). Inflammation may be a bridge connecting hypertension and atherosclerosis. Med Hypotheses.;64(5):925-9.]

Liao JK (2005). Clinical implications for statin pleiotropy. Curr Opin Lipidol. Dec;16(6):624-9.

Kannel William B, Philip A Wolf, William P, Castelli Ralph B (1987). 'Agostino Fibrinogen and Risk of Cardiovascular Disease The Framingham Study JAMA.:258(9):1183-1186.

Lee K (2009). Relationship between uric acid and hepatic steatosis among Koreans. Diabetes Metab. Dec;35(6):447-51

Palmer IM, Schutte AE, Huisman HW (2010). Uric acid and the cardiovascular profile of African and Caucasian men. J. Hum Hypertens. O24(10):639-45. Epub 2010 Feb 11.

Mente M, Salim Yusuf, Shofiqul Islam, Matthew J. McQueen, Supachai Tanomsup, Churchill L. Onen, Sumathy Rangarajan, Hertzel C Gerstein, Sonia S Anand, (2010). Metabolic Syndrome and Risk of Acute Myocardial Infarction J Am Coll Cardiol,; 55:2390-2398 G Imperatore, G Riccardi, C Iovine, A A Rivellese, O Vaccaro (1998).

Plasma fibrinogen: a new factor of the metabolic syndrome. A population-based study. diacare.21.4.649 Diabetes Care vol. 21 no. 4 649-654

Ramkumar Nirupama, Maureen A. Murtaugh, Alfred K. Cheung, Srinivasan Beddhu, ((2007). Lack of Synergistic Effects of Metabolic Syndrome and Plasma Fibrinogen on Coronary Events and Mortality in Moderate CKD American Journal of Kidney Diseases Volume 49, Issue 3, Pages 356-364, Vol. 49, (3). pp 356-364,

Lyu LC, Hsu CY, Yeh CY, Lee MS, Huang SH, Chen CL (2003). A case-control study of the association of diet and obesity with gout in Taiwan. Am. J. Clin. Nutr. 78(4):690-701. PubMed Abstract | Return to text

Carroll S; Cooke C B; Butterly R J (2007). Plasma viscosity, fibrinogen and the metabolic syndrome: effect of obesity and cardiorespiratory fitness. Volume 11 - Issue 1 Med Arh.;61(1):7-10.

Vorster HH, Jerling JC, Steyn K, Badenhorst CJ, Slazus W, Venter CS, Jooste PL, Bourne LT (1998). Plasma fibrinogen of black South Africans: the BRISK study. Public Health Nutr. 1(3):169-76.

Mendez GF, Cowie MR (2001). The epidemiological features of heart failure in developing countries: a review of the literature. Int. J. Cardiol.;80:213–219. [PubMed]

Kingue S, Binam F, Pouth SFB, B Ouankou, MD Muna, WFT (2000). La maladie coronaire au Cameroun. Aspects épidémiologiques et cliniques (à propos de 30 observations. Cardiologie Tropicale -Tropical Cardiology Vol. 26 No. 101 pp. 7-11

Dzudie A, Kengne AP, Mbahe S, Menange A, Kenfack M, Kingue S (2008). Chronic heart failure, selected risk factors and co-morbidities among adults treated for hypertension in a cardiac referral hospital in Cameroon. Eur. J. Heart Failure;10:367–372

Ladipo GO, Froude JR, Parry EH (1977). Pattern of heart disease in adults of the Nigerian Savannah: a prospective clinical study. Afr. J. Med. Med Sci.;6:185–192. [PubMed]

Zunzunegui-Pastor MV, F. M. Gómez-Trujillo, JS Luque-Martin, JJ Sánchez-Luque, C Ortiz-Garciéa, MJ Ferreras-Duarte, I García-Caravaca ((2001). Should the routinary determination of plasmatic fibrinogen as vascular risk indicative parameter be included in the metabolic syndrome patient? Am. J. Hypertens 14, 60A–61A; doi:S0895-7061(01)01633-8

Ogbera Anthonia O (2010). Prevalence and gender distribution of the metabolic syndrome Diabetol Metab Syndr; 2: 1.

Rees D, Kallner A, McMichael T, Raivio K, Ritchie P, Wake M, Titanji V, Gutierrez-Espeleta E, Nath I (2007). Final Draft Report *ICSU Scoping Group 15-3-0*, 1-13.

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