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USE OF CARDIAC BIOMARKERS FOR PREDICTION OF CORONARY HEART DISEASE IN FAMILY MEMBERS OF DIABETICS IN JOS NIGERIA

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ABSTRACT

Diabetes is known as a strong etiological source of Coronary Heart Disease (CHD). Family history of diabetes and heart disease are key risk factors of Diabetes Mellitus Type-2 (DMT2) and Cardiovascular Disease especially CHD. The underlying disorders in diabetes and CHD are scientifically and clinically linked to genetic predispositions, lifestyle and socioeconomic factors. This study aimed at assessing some biomarkers of CHD in people with family history of diabetes. A total of 160 participants: 120 (75%) with family history of diabetes and 40 (25%) without history of diabetes, were enrolled in the study. Plasma and serum from venous blood samples were used in spectrophotometric assaying for Fasting Plasma Glucose, Lipids (Total Cholesterol, Triglycerides, High Density Lipoprotein Cholesterol and Low Density Lipoprotein Cholesterol); while high sensitivity C - reactive protein (hs-CRP) was measured using Enzyme Linked Immunosorbent Assay (ELISA) technique. Anthropometric parameters: weight and height were taken for Body Mass Index (BMI) while Blood Pressure (BP), was taken using a sphygmomanometer and stethoscope. The results showed mean of: Age (21-40 years), Systolic and Diastolic Blood Pressure (127.5±19.43 and 85.0±4.5 respectively), Fasting Blood Sugar (5.01±1.15), BMI (26.6±5.98), Triglycerides (1.12±0.71), hs-CRP (6.7±2.78). BMI and BP were statistically significant ($p<0.05$) with age while all other parameters were not statistically significant ($p>0.05$). We concluded that there is risk of CHD in relations of Diabetics; and that the Framingham risk tools, BMI, Lipid Profile Ratios, Family history and CRP levels could be used routinely in predicting Coronary Heart Disease.

KEYWORDS: Coronary heart disease; Family History; Diabetes mellitus; Genetics; Asymptomatic risk.

INTRODUCTION

Coronary Heart Disease (CHD) is the most common cause of morbidity and mortality in the modern world (Rungoe *et al.*, 2015; Sekhri *et al.*, 2014; Krishnan, 2012) with prevalence of 21.9% (Al-Mamari, 2009); and is predicted to rise to 26.3% by the year 2030. It has been linked to genetic and environmental factors (Baixeras *et al.*, 2014). Robert and Stewart (2012) reported that, predisposing genetics is responsible for 50% of CHD susceptibility. Family history of Diabetes Mellitus is also a significantly proved pivot of Metabolic Syndrome thus CHD (Das *et al.*, 2012).

Oxidative stress is said to be cardinal cause of Atherogenesis, Ischemic-Reperfusion Injury and Cardiac remodeling (He and Zuo, 2015); and to a large extent, pathologically resulting from increased levels of Reactive Oxygen Species-ROS (Belló-Klein *et al.*, 2014; Marchi *et al.*, 2013).

Commonly, CHD begins with the narrowing of blood vessels resulting from Atherosclerosis (Weber and Noels, 2011). CHD is triggered when waxy substances called-plaques build up on the intima of the Blood Vessels meant to supply blood and oxygen to the heart muscles. CHD usually contributes to an increased incidence and poor prognostic outcome of Myocardial Infarction and stroke (Efimov *et al.*, 2001).

Coronary Heart Disease (CHD) and associated risk factors may include positive family history of early CHD, Diabetes Mellitus Type 2 (DMT2), Hypertension, Obesity, Sedentary lifestyle and Dyslipidaemia (Rungoe *et al.*, 2015; Gopalan *et al.*, 2015). Though, dyslipidaemia is a modifiable risk factor, it is the basic aetiology of atherosclerosis and a potential contributor in the pathogenesis of other forms of cardiovascular diseases (Lim, 2013). Some school of thought had suggested that income level may have some link to CVD, but this has been proved wrong in relation to the prevalence of CVD risk and its screening; advocating for the need for screening and diagnosis of modifiable risk factors at all levels of society (Oguoma *et al.*, 2015). Metabolic syndrome triggers atherosclerosis and diabetes, which play major roles in the development of cardiovascular disease (American Heart Association, 2015).

These risk factors are common among adults both in the developed and developing countries (Awosan *et al.*, 2013). Normoglycemic range is strongly and independently associated with the risk of developing cardiovascular disease. Fasting normoglycaemia may help in identifying apparently healthy individuals of asymptomatic metabolic abnormalities who are at increased risk of cardiovascular disease before the progression to pre-diabetes and overt diabetes mellitus (Kivity *et al.,* 2012).

Biomarkers of CHD include the Framingham Heart Study tools (Age above 20 years, Blood Pressure, Gender, Increased levels of Total cholesterol, Reduced High Density Lipoprotein Cholesterol (HDL-c), Increased Low Density Lipoprotein Cholesterol (LDL-c) and cigarette smoking), Increased Triglycerides, Chronic hyperglycaemia, Obesity and Overweight, Hypertension, elevated C-Reactive Protein level (CRP). Clinically, heart disease is indicated in an individual with LDL-c >3.36mmol/L, hs-CRP>3µg/dl (Ridker, 2003)**.** Thus, the use of CRP with cholesterol values significantly predicts and prevents cardiovascular disease globally. Hyperlipidaemia> 2.59 mmol/L sets an individual at a high risk for myocardial infarction and thus an abrupt cardiac death while Blood Pressure >130/85 (mmHg) worsens the development of CVD. Recently, lipid ratios have emerged as options for better prediction; TC: HDL-c predicts carotid intima-media thickness (Millán *et al.*, 2009). TG/HDL-c ratio >4 independently predicts development of Coronary Artery Disease even when little is yet known about its association with the severity of the lesion (Luz *et al.,* 2008). Gender difference may sometimes contribute to the development of CVD at some points. Females generally have more body fat percent but lesser Physical Working Capacity (PWC) within the limit of different age groups, but there is no significant difference in Total Cholesterol and HDL-c levels compared to that of males (Dikko, 2015).

METHODOLOGY

Study population/Area

The study was conducted among family members of diabetic patients attending Jos and Bingham University Teaching Hospitals and Plateau State Specialist Hospital, all in Jos Plateau State, Nigeria. A total of 160 participants **-** 120 with family history of diabetes and 40 individuals without diabetes history were enrolled in the study irrespective of age and gender using random sampling technique.

Exclusion criteria

Persons who had pregnancy, psychiatric disorder, renal disorder, hepatic disorders, smoking, alcoholism, cancer and those who did not consent were excluded from the study.

Ethical clearance

Ethical clearances were obtained from the Ethical Committees of the three hospitals where this study was carried out.

Sample collection

Fasting blood samples were collected by standard venipuncture into Fluoride oxalate tubes (for glucose) and plain containers for other biochemical assays. Blood for glucose test was well mixed in the bottle to avoid clotting, while the one for other tests was allowed to clot and centrifuged at 3000rpm for 5 minutes from which the serum was obtained into cryovails. Plasma samples for Glucose and lipids were analyzed immediately within 2-3 hours after collection while the serum samples were stored at -20^{o}C in which CRP was analyzed within 30 days using the Enzyme Linked Immunosorbent Assay (ELISA) technique.

Biochemical analysis

Plasma glucose was analyzed using Glucose Oxidase Method as Adopted by Trinder 1979. Total Cholesterol using Trinder's colour system of Peroxidase/Phenol/4 Aminoatipyrine), HDL-c was analyzed using the combination of precipitation and enzymatic method, LDL-c was calculated using the Friedewald equation except for those with Triglycerides>4.52mmol/L which were estimated directly. Lipid profile ratios to HDL-c were calculated as TC/HDL-c, TG/HDL-c and LDL/HDL-c. High sensitive C-Reactive Protein (hs-CRP) was measured using immune-enzymometric assay. All test kits were commercially available as products of Bt Laboratory and hs-CRP *Product code: 3125-300*. In each analysis, the manufacturer's instructions were strictly adhered to.

Anthropometric measurements

Blood Pressure (BP) was taken using Sphygmomanoter and Stethoscope while Anthropometric measurement of Body Mass Index (BMI) was calculated as weight (kg) divided by height square (m^2).

Statistical analysis

All data generated from the different tests were subjected to statistical analysis using SPSS software version 21.0. Statistical significance level was set at P<0.05 to obtain Mean and Standard Deviation of Mean. Independent Paired Student T-test was used to test for statistical difference between means.

RESULTS AND DISCUSSION

This study determined Cardiac Biomarkers of Coronary Heart Disease (CHD) in family members of persons with diabetes. Table 1 showed that there was no statistical difference between the Test participants and the Control participants (P>0.05). This could imply that all these parameters assessed are potential risks in any individual. It could also mean that family history of diabetes may not play a major role in developing CHD even though it is a predictive risk tool. The assessed risks were generally high and asymptomatic in both test participants and controls.

Table 1: Comparism of means of Test participants and Controls.

Mean ± Standard deviation				
Characteristics	**Tests (n=120)**	**Controls (n=40)**	**t-Test**	**P-value**
BMI	26.8±5.859	25.9±6.346	-1.020	0.309
SBP	128.8±20.698	123.4±14.473	1.545	0.124
DBP	85.2±17.778	84.5±12.547	0.239	0.812
FPG	5.0±1.279	5.0±0.880	0.301	0.763
TC	4.5±0.961	4.4±0.876	0.291	0.771
$HDL-_C$	1.4±1.144	1.3±0.666	0.601	0.549
$LDL-_C$	2.6±1.638	2.6±1.194	0.179	0.858
TG	1.0±0.688	1.2±0.778	1.706	0.090
TC:HDL	6.3±13.876	4.0±1.924	1.028	0.306
TG:HDL	2.6±15.152	2.6±1.506	0.591	0.556
LDL:HDL	4.6±12.817	2.6±1.506	0.985	0.326
CRP	6.9±2.703	6.8±2.677	0.978	0.744

Key: BMI= Body Mass Index, SBP= Systolic Blood Pressure, DBP= Systolic Blood Pressure, FPG= Fasting Plasma Glucose, TC= Total Cholesterol, LDL-c= Low Density Lipoprotein-cholesterol, $HDL-_C$= High Density Lipoprotein-cholesterol, TG= Triglycerides, TC:HDL= Total Cholesterol High Density Lipoprotein ratio, TG:HDL= Triglycerides High Density Lipoprotein ratio, LDL:HDL= Low Density Lipoprotein High Density Lipoprotein ratio, CRP= C-Reactive Protein.

Table 2: Prevalence (%) of Risks and Asymptomatic Disorders.

Risk Marker	Frequency (%)	Disorder (undiagnosed)	Frequency (%)
Overweight	70(43.8)	Obesity	24(15)
Pre-hypertension	27(16.9)	hypertension	49(30.6)
Pre-diabetes	16(10)	hyperglycaemia	10(6.3)
Risk of inflammation	124(77.5)	Vascular inflammation	19(11.9)
		Dyslipidamia	
		Hyperlipidaemia	7(4.4)
		HDL	76(47)
		LDL	34(21.3)
		(TG)	34(21.3)
		TG: HDL-c	19(11.9)
		TC: HDL-c	55(34.4)
		LDL-c: HDL-c	56(35)

Key: *TG: HDLc= Triglycerides High Density Lipoprotein Cholesterol ratio, TC: HDLc= Total Cholesterol High Density Lipoprotein Cholesterol ratio, LDL-c: HDL-c= Low Density Lipoprotein-Cholesterol High Density Lipoprotein-Cholesterol ratio.*

From table 2, it was observed that there are increased risks and asymptomatic disorders of the Biomarkers of CHD among the participants. The prevalence of asymptomatic hypertension was 30.6%. This prevalence strongly agrees with similar reports of 31.2% by Dahiru and Ejembi (2013) and Awosan *et al.*, 2013 who reported a prevalence of 33.7% both in Zaria, Kaduna State, Nigeria. The prevalence observed is also within the ranges 8%-46.4% following the report of Okechukwu *et al.*, 2012 in Nigeria. Meanwhile the prevalence of individuals at risk of hypertension was 16.9%. This suggests that a considerable percentage (16.9%) of the population is at risk of developing hypertension with time.

Similarly, the results from table 2 also revealed that the prevalence of cardiovascular risk was 77.5% using (CRP) 3≤10µg/dl. This suggests that there is a very high prevalence for developing heart disease after a decade among the participants. This finding is in agreement with Weil (2016) who showed that CRP 3≤10µg/dl predicts a 10year development of cardiovascular disease. More so, this result is in agreement with Ridker (2003) who reported that the use of hs-CRP could screen a large population in primary prevention for risk of diabetes and cardiovascular disease. The prevalence of undiagnosed Fasting Hyperglycaemia was 6.3%. The prevalence obtained is similar to that reported by Gezawa *et al., 2015* as 7.0%. Furthermore, 10% of the participants are at risk of high normoglycaemic range. This result tallies with the report of 10.3% of undiscovered diabetes by Puepet *et al.,* 2004 following his first report of 3.1% in 2002.

Triglycerides was 21.3%, indicating a high prevalence of the risk of Myocardial Infarction. Following the observations in table 1, it could be observed that the prevalence of asymptomatic Coronary Artery Disease was 11.9% especially Myocardial Infarction. This result agrees with Luz *et al.,* 2008, who showed that TG/HDL-c ratio>4 is known to be a powerful independent predictor for developing Coronary Artery Disease even when little is yet known about its association with the severity of the lesion.

The prevalence of obesity and overweight were found to be 15% and 43.8% respectively. This is in agreement with similar prevalence of 17.2% and 31.1% for obesity and overweight respectively among urban Nigerians (Okafor *et al.,* 2014). We observed hypercholesterolaemia to be 4.4%; knowing that increased Total Cholesterol (TC) may be clinically associated with risk factors of Nephrotic syndrome, Diabetes Mellitus, Obstructive Jaundice, Coronary thrombosis, Angina Pectoris and Hyperthyroidism among others.

Generally, the marked increased differences in the rate of risks and undiagnosed conditions in these results could be due to changes in dietary habits, with the adoption of westernized diets such as increased consumption of social and fast foods which are gaining popularity around the city of Jos. Sedentary lifestyle and low energy-demanding vocations, enhanced easy means of transportation systems either by private or mass. Improved technologies; use of computers, internet, live television broadcasts, electronic and programmed games have set the Nigerian population less ambulatory. Fast food available in restaurants, consuming garnished meals with asymptomatic high salt and saturated fats are often rich in sugar content accompanied with increased bottled drinks.

From table 3, it was discovered that, among the individuals at risk, apart from Fasting Plasma Glucose, females were more at risk than males. Similarly, in the distribution across overt asymptomatic disorder, apart from High Density Lipoprotein-cholesterol (HDL-c) and Triglycerides (TG) in which there were marginal differences in frequencies; females had higher frequency than males. There was a significant relationship between both risk and asymptomatic disorders and gender which were more common in females than males.

There was a statistical relationship between BMI, BP and gender ($P<0.05$); and females were more susceptible than males. The results could imply that females are generally at more risk of developing diabetes and heart disease than males. This result disagrees with Lara (2015) who implicated estrogen to have a preventive mechanism against the development of atherosclerosis, although that report was strictly based on sex and age especially with respect to pre-menopausal phase of life.

Also, the mean values of FPG and the Lipids were within their physiologic ranges. The mean values of BMI, SBP, DBP and CRP were within the pathologic risk ranges which may suggest risk of heart disease. These suggest that most of the participants are at risk of Obesity, Hypertension and Inflammatory disorder within the Cardiovascular System consecutively. Furthermore, only BMI and BP were statistically significant ($P<0.05$) as dependent biomarkers of Coronary Heart Disease on age.

Comparing results in table 3 and table 2, although CRP, FPG, Lipid Profile and the ratios did not show significant relationships with age ($P>0.05$), they were able to group the individuals into overt hyperglycaemia, dyslipidaemia and at risk of heart disease especially following the use of C-RP with 77.5%. This could imply that these Biomarkers are independent of age and therefore, they could potentially ignite the development of diabetes and heart disease at any age in life. This agrees with the findings of Oguoma *et al.,* 2015 who reported a statistical significance between BMI, BP and Age.

Table 3: Frequency of risks and prevalence of disorders with respect to sex and their significance.

Marker	At risk (%)		Overt (%)		χ^2 value	P-value
	Males (%)	Females (%)	Males (%)	Females (%)		
BMI	23(14.4)	47(29.4)	5(3.0)	19(11.9)	0.000	0.000**
BP	11(6.9)	16(10.0)	14(8.8)	35(21.9)	15.915	0.017*
FPG	10(6.3)	6(3.8)	5(3.1)	5(3.1)	447.492	0.478
C-RP	-	-	56(35.0)	68(42.5)	107.660	0.115
TC	-	-	2(1.3)	5(3.0)	348.257	0.372
HDL	-	-	35(21.9)	41(21.6)	748.004	0.799
LDL	-	-	57(35.6)	71(44.4)	364.172	0.389
TG	-	-	18(11.3)	16(10)	261.193	0.279
TG: HDL-c-		-	8(5)	11(6.9)	736.770	0.787
TC: HDL-c	-	-	25(15.6)	30(18.8)	658.131	0.703
LDL-c: HDL-c-		-	26(16.3)	31(19.4)	850.047	0.908

Key: *BMI= Body Mass Index, BP= Blood Pressure, FPG= Fasting Plasma Glucose, CRP= C-Reactive Protein, TC= Total Cholesterol, HDL= High Density Lipoprotein, LDL= Low Density Lipoprotein, TG= Triglycerides, TG:HDL-c= Triglycerides High Density Lipoprotein-cholesterol ratio, TC:HDL= Total Cholesterol High Density Lipoprotein-cholesterol ratio, LDL-c:HDL-c= Low Density Lipoprotein-cholesterol High Density Lipoprotein-cholesterol ratio, *Statistically significant, ** Statistically very significant.*

Table 4: Mean ± Standard Deviation (SD) and P-value of biomarkers across various age group

AGE (Years)	BMI (kg/m^2)	SBP (mmHg)	DBP (mmHg)	FPG	TC (mmolL^{-1})	TG (mmolL^{-1})	HDL (mmolL^{-1})	LDL (mmolL^{-1})	CRP (µg/dl)
0-20	22.4±4.0	117.3±15.81	80.0±3.0	4.6±0.92	4.1±0.42	0.9±0.64	1.4±1.0	2.3±1.41	7.5±2.78
21-40	24.1±6.0	123.6±16.71	85.0±8.0	5.1±1.3	4.5±0.85	1.1±0.69	1.3±0.7	2.8±1.41	6.7±2.78
41-60	27.3±5.9	136.7±21.60	85.0±5.0	5.0±0.95	4.6±1.13	1.2±0.75	1.4±1.2	2.6±1.70	6.7±2.59
>60	26.8±4.9	135.0±18.30	90.0±2.0	5.0±1.46	4.6±1.15	1.2±1.0	2.1±1.6	2.0±1.803	7.6±3.08
Total Mean ± Standard deviation									
21-40	25.6±6.1	127.5±19.43	85.0±4.5	5.0±1.5	4.5±0.94	1.1±0.71	1.4±1.17	2.6±1.536	6.7±2.78
F-value	4.621*	7.620**	3.981*	0.537	1.055	0.901	1.963	0.250	0.714
P-value	0.004	0.000	0.048	0.658	0.370	0.442	0.122	0.667	0.545

Key: BMI= Body Mass Index, SBP= Systolic Blood Pressure, DBP= Systolic Blood Pressure, FPG= Fasting Plasma Glucose, TC= Total Cholesterol, TG= Triglycerides, HDL= High Density Lipoprotein, LDL= Low Density Lipoprotein,*= statistically significant, **= Statistically Very significant.

Table 4, shows that the mean age of the participants was 21-40 years. It could be postulated that it is the average age of the population that may develop (risks of) overt disease after 10 years (with time). This result is in agreement with Jaagus *et al.*, 2010 who reported that symptoms of heart disease usually start manifesting clinically above 40 years. This result also agrees with American Heart Association (2015), from which it was reported that 5.5-6.5% symptoms of heart disease begins at age above 40 years for both men and women.

CONCLUSION

Based on the results of this study, we concluded that there is a high prevalence of asymptomatic heart disease and its risks among relatives of Diabetics in Plateau State, Nigeria. The Framingham risk tools, BMI, Lipid Profile Ratios, CRP levels and family history can be used in predicting Coronary Heart Disease.

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