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Assessment of Some Cardiac Markers in Apparently Healthy Active Tobacco Male Smokers in Nnewi, South Eastern, Nigeria.

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Abstract
Background: *Smoking is regarded as a social activity but with adverse effects. This study investigated the cardiac status of active tobacco smokers in Nnewi, South Eastern, Nigeria.*
Methods: *A total of 100 subjects compromising of 50 apparently healthy active and non- smokers respectively were recruited for the study. Their blood pressure readings and weight were obtained. Well – structured questionnaire was used to obtain their bio-data. Blood sample was collected from each subject and estimated for apolipoproteins (Apo A-1, Apo B), creatine kinase –MB (CK-MB) and Troponin I. Standard routine analyses were used for estimation of parameters. Data obtained were statistically analyzed using student t-test and Pearson r correlation.*
Results*: The results showed that the mean serum levels of ApoA-1, ApoB, CK-MB and Troponin I were significantly higher in active tobacco smokers than in the control groups at p< 0.05 respectively. Also the mean serum activity of CK-MB was significantly higher in active tobacco smokers than in the control groups at p< 0.05. Similarly, the mean levels of systolic and blood pressure (SBP, DBP) and weight (WT) were significantly higher in active tobacco smokers than in the control groups at p< 0.05 respectively. There was positive correlation between age vs ApoA-1, CK-MB vs WT, WT vs ApoA-1, WT vs ApoB and WT vs CK-MB in control groups than in active tobacco smokers at p <0.05 respectively. Similarly, there was negetive correlation between ApoA vs SBP, Troponin I vs age, CK-MB vs ApoA-1, SBP vs WT, SBP vs SBP, DBP vs ApoA-1, DBP vs DBP, WT vs age in control groups than in active tobacco smokers at p <0.05 respectively. There was positive correlation between age vs ApoA-1, Troponin I vs SBP and negative correlation between Apo B vs SBP, CK-MB vs DBP in active tobacco smokers respectively.*
Conclusion: *The study observed elevated levels in ApoA-1, ApoB, Troponin I, CK-MB, SBP, DBP and weight in the active smokers. There were increasing and attenuating associations of parameters observed in the group studied. These findings may suggest that the incidence of coronary heart disease may be associated with smoking. Hence, smoking is needed to be refrained from as part of modifiable risk factors in the control of heart diseases.*
Keyword*: smokers, CK-MB, ApoA-1, ApoB, Troponin I.*

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I. Introduction

Smoking is performed by burning a substance and the resulting smoke breathed in and absorbed into the bloodstream [1]. The substance burned is a dried leaves of the tobacco, which is rolled into small round cylinder called cigarette [1]. Cigarette smoking is the leading cause of preventable death and a major public concern [2].Tobacco is an agricultural product from fresh leaves of plant in the genus *Nicotiana*. Tobacco contains more than 200 chemicals that are known to be harmful to the body such as hydrogen cyanide, ammonia and carbonmonoxide [3]. Also, more than 50 of these chemicals are carcinogenic to health such as benzene, nickel, beryllium, aromatic amines, acetaldehyde, cadmium and cumene [3].The most important chemicals causing cancer are those that produce DNA damage since such damage appears to be the primary underlying cause of cancer [4, 5].

Reports have it that 7.3 % of men and 0.4 % of women currently used smoked tobacco, while 29 % of men and 0.9 % of women used smokeless tobacco globally. In Nigeria, 10 % of men and 1.1 % of women used tobacco products [6]. W.H.O. in [7], estimated that tobacco caused 5.4 million deaths.Atherosclerosis is the major cause of cardiovascular disease. It is a degenerative process, which hardened the arteries, causing plague formation a lipid accumulation within the intima of the arteries [8]. Hypertension is also a persistence blood pressure equal or above 140 mmHg in systole and or 90 mmHg in diastole. Hypertension leads to a two to a fourfold increase in cardiovascular events in comparison to a normotensive person of the same age group [9].Reports have it that physical fitness and weight loss reduces metabolic risk factors for cardiovascular diseases [10]. Troponin I is a cardiac protein biomarker used for detecting acute myocardial infarction (AMI) [11]. Also, CK-MB is found in heart muscle and it is released in AMI [12].Apolipoproteins are proteins that bind lipids to form lipoproteins whose main function is to transport lipids in the body [13].

II. Materials and methods

Active smokers that smoked at least one cigarette per day were randomly recruited from motor parks, public eateries, recreational centres and restaurants in Nnewi, Anambra State, Nigeria. Control group made up of non- smokers were randomly recruited from apparently healthy individuals in the same area of sampling. The subjects were given written informed consent, while the study design was approved by the ethics committee of Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria. Their blood pressure readings and weight were obtained. Well – structured questionnaire was used to obtain their bio-data. 3 ml of blood sample was collected from each subject, collected into plain tube and allowed to clot, centrifuged, the serum separated and analyzed for Apo A-1, Apo B, CK-MB and Troponin I.

2.1 Quality control measures

Quality control sera were ran along test in each batch of analysis these were compared with the reference values of the control sera. Standard deviation and coefficient of variation were calculated on them.

Methods of assay

Enzyme linked Immunosorbent Assay (ELISA) as described by Tiez [14] was used for Myoglobin and Troponin I. Spectrophotometric method as described by Tiez [15] was used for CK-MB. Data obtained were statistically analyzed using student t-test and Pearson r correlation. Students't-test was used to compare means. The analysis was performed with the use of Statistical *Package for Social Sciences* (SPSS) statistical software package, version 16.0. P <0.05 is considered statistically significant.

III.Results

In this study, the levels of ApoA-1, ApoB, CK-MB and Troponin I were significantly higher in active tobacco smokers than in the control groups at p< 0.05 respectively. Similarly, the mean serum activity of CK-MB was significantly higher in active tobacco smokers than in the control groups at p< 0.05. Again, the mean levels of SBP, DBP and weight (WT) were significantly higher in active tobacco smokers than in the control groups at p< 0.05 respectively. There was positive correlation between Age vs ApoA-1, CK-MB vs WT, WT vs ApoA-1, WT vs ApoB and WT vs CK-MB in control groups than in active tobacco smokers at p <0.05 respectively. Similarly, there was negetive correlation between ApoA vs SBP, Troponin I vs Age, CK-MB vs ApoA-1, SBP vs WT, SBP vs SBP, DBP vs ApoA-1, DBP vs DBP, WT vs Age in control groups than in active tobacco smokers at p <0.05 respectively. There was positive correlation between age vs ApoA-1, Troponin I vs SBP and a negative correlation between Apo B vs SBP, CK-MB vs DBP in active tobacco smokers respectively. Similarly, there was positive correlation between Age vs DBP, ApoA- vs Troponin I , SBP vs WT and a negative correlation between ApoB vs DBP, DBP vs WT in non- smokers respectively.

Table 1: Comparison of mean ± SD levels of ApoA-1, ApoB, Troponin I , CK-MB in groups studied.

Groups	ApoA-1	ApoB	Troponin I	CK-MB
Controls (n=50)	O.12 ± 0.02	0.78 ± 0.28	0.31 ± 0.22	24.65 ± 6.17
Smokers (n=50)	0.28 ± 0.13	1.31 ± 0.48	0.67 ± 0.78	44.02 ± 22.18
t-value	0.000	0.000	0.020	0.000
p-value	29.77	26.44	14.67	44.53

Table 2: Comparison of mean ± SD levels of Age, SBP, DBP and weight in groups studied.

Groups	Age	SBP	DBP	weight
Controls (n=50)	32.04 ± 8.01	124.78 ± 8.94	80.78 ± 5.65	74.88 ± 3.35
Smokers (n=50)	35.2 ± 11.17	130.72 ± 8.31	88.68 ± 10.49	76.84 ± 3.51
t-value	0.191	0.001	0.000	0.005
p-value	6.51	0.11	10.03	0.25

Table 3: Levels of association between parameters studied in controls (1) and active smokers (2).

Groups	Pearsons r correlation coefficient	F- value	P- value
Age_1 v $ApoA\text{-}1_2$	-0.036	0.803	<0.05
$ApoA\text{-}1_1$ v SBP_2	-0.022	0.88	<0.05
Troponin I_1 v Age_2	-0.023	0.876	<0.05
$CK\text{-}MB_1$ v $ApoA\text{-}1_2$	-0.047	0.748	<0.05
$CK\text{-}MB_1$ v WT_2	0.015	0.917	<0.05
SBP_1 v WT_2	-0.034	0.813	<0.05
SBP_1 v SBP_2	-0.016	0.914	<0.05
DBP_1 v $ApoA\text{-}1_2$	-0.017	0.908	<0.05
DBP_1 v DBP_2	-0.033	0.823	<0.05
WT_1 v Age_2	-0.009	0.953	<0.05
WT_1 v Apo $A\text{-}1_2$	0.005	0.97	<0.05
WT_1 v Apo B_2	0.032	0.826	<0.05
WT_1 v $CK\text{-}MB_2$	0.014	0.921	<0.05

Table 4: Levels of association between parameters studied within controls (1) and active smokers (2).

Groups	Pearsons r correlation coefficient	F- value	P- value
Age_1 v DBP_1	0.042	0.775	<0.05
$ApoA\text{-}1_1$ v Troponin I_1	0.011	0.94	<0.05
$ApoB_1$ v DBP_1	-0.022	0.933	<0.05
SBP_1 v WT_1	0.033	0.813	<0.05
DBP_1 v WT_1	-0.039	0.787	<0.05
Age_2 v $ApoA\text{-}1_2$	0.000	0.996	<0.05
$ApoB_2$ v SBP_2	-0.029	0.841	<0.05
Troponin I_2 v SBP_2	0.001	0.996	<0.05
$CK\text{-}MB_2$v DBP_2	-0.02	0.89	<0.05

IV. Discussion

In this study, the serum levels of apolipoprotein A-1 was significantly higher in active smokers. ApoA-1 is the major apolipoprotein in HDL [16] and also a co-factor for lecithin cholesterol acyl transferase (LCAT) [17], which is important in removing excess cholesterol from tissues to liver when incorporated into HDL [18]. The study showed higher concentration of ApoB in active smokers. ApoB is essential for the binding of LDL particles and excess ApoB is a main trigger in the atherogenic process [18].Apolipoprotiens help to transport lipids in blood [13]. Reports have it that smoking has been associated with significantly elevated serum concentrations of total cholesterol and triglycerides [19]. Also, several studies have observed higher levels of LDL and VLDL cholesterol in smokers [20]. These associations seem to be dose dependent [19]. Again, HDL level has been lowered in smokers; HDL is a powerful protective factor against the development of atherosclerosis [21, 22]. The difference is usually small, 5 mg/dl or less, but this difference represents a 10% decrease and would be expected to affect atherogenesis to a significant degree [20]. lso, the serum concentration of Troponin I was increased in active smokers. Increased Troponin I levels have been associated with acute and chronic heart failure, rhabdomyolysis, cardiomyopathy [23]. Magnus *et al*, [24], observed that Current smoking was associated with lower concentrations of troponin I (cTnI), suggesting that substances in tobacco smoke

might affect cardiomyocyte injury. Smoking is a major risk factor for cardiovascular disease, but it is not clear by which mechanism smoking exerts its detrimental effects on cardiovascular disease [25, 26]. Smoking cessation has been reported to be followed by a decrease in blood pressure [27, 28]. Higher serum activity of CK-MB was observed in active smokers. CK-MB is a cardiac enzyme marker used to assess heart diseases [29]. It is seen in myocardial infarction [30].he study revealed higher levels in SBP and DBP in active smokers. Higher level of adiposity is strongly associated with elevated blood pressure and is also considered a major risk factor for hypertension [31, 32].n this study, there was a positive association between weight in non-smokers when compared with serum level of Apo A-1 and serum activity of CK-MB in active smokers respectively. Also, there was a positive association between ApoA-1 in non-smoker when compared with the value of SBP of in active smokers. On the other hand, there was a negative association with SBP in non-smokers when compared with weight and SBP in active smokers respectively.Also, the study observed a negative association with ApoA-1 vs SBP, Troponin I vs Age, CK-MB vs ApoA-1, DBP vs ApoA-1, DBP vs DBP, weight vs Age in non-smokers when compared in active smokers respectively.he study revealed a positive association between Age vs DBP, ApoA-1 vs Tropinin I and SBP vs weight in non-smokers respectively. On the contrary, there was a negative correlation between ApoB vs DBP and DBP vs weight respectively.There was a positive association between Age vs ApoA-1 and Tropinin I vs SBP in active-smokers respectively. However, there was a negative correlation between ApoB vs SBP and CK-MB vs DBP in active-smokers respectively. The increasing and attenuating associations observed in this study, could serve as predictive or prognostic index for evaluating myocardial infarction.

V. Conclusion

The study observed elevated levels in ApoA-1, ApoB, Troponin I, CK-MB, SBP, DBP and weight in the active smokers. There were increasing and attenuating associations of parameters observed in the group studied. These findings may suggest that the incidence of coronary heart disease may be associated with smoking. Hence, smoking is needed to be refrained from as part of modifiable risk factors in the control of heart diseases.

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