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Lipid Profile and Atherogenic Indices in Obese Hypertensive and Diabetic Patients

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Abstract: ***In a bid to achieve this objective of examining the lipid profile and atherogenic indices in obese hypertensive and diabetic patients, as well as compare these with the apparently healthy subjects, two hundred and sixty-four (264) subjects were recruited. They consist of group A (59 obese diabetic subjects), group B (59 obese hypertensive subjects), group C (41 obese diabetic and hypertensive subjects), group D (41 obese only or non complicated obese subjects) and group E (60 non obese non diabetic/hypertensive or normal control). Using standard procedures, weight, height, blood pressure and fasting blood glucose were measured and used to classified patients and serum lipid profile and atherogenic indices were determined. Results showed that the non complicated obese group (group D) has increased propensity to develop worsen lipid profile (dyslipidemia) and atherogenic diseases compared to the apparently healthy group (group E) and complicated obese groups (group A, B and C). Judging from the finding of this study, it was observed that non complicated obese group (group D) are more likely to die of CAD followed by the obese diabetic, obese diabetic and hypertensive and then lastly the obese hypertensive individual.***

Keywords: Diabetes, Hypertension, Obesity, co-morbidity, lipid profile, atherogenic indices

1. Introduction

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems[1]. According to Kaufer et al[2], it is the condition under which adipose tissue is increased and can be defined as an increase in body weight that results in excessive fat accumulation. It is a chronic disease of multi-factorial origin that develops from the interaction of social, behavioral, psychological, metabolic, cellular, and molecular factors. Barness et al.[3], tag it as a leading preventable cause of death worldwide, with increasing prevalence in adults and children and is viewed by authorities as a serious public health problem of the 21st century.

Obesity is said to place additional burdens on health care expenditure because are associated with an increased risk for many medical problems[4]. In fact, it is said to be related to several chronic conditions such as diabetes, hypertension, cardiovascular diseases, obstructive sleep apnoea, certain type of cancers and osteoarthritis[1]. Of interest in this study, is the association of obesity with diabetes and/or hypertension which according to Porte and Kahn[5] and Goodman et al[6] the mechanism is not fully understood. Despite the advance in technology, predicting in obese individuals those that will develop diabetes mellitus and/or hypertension is still highly obscure[6].

One of these problems of interest is diabetes which is one of the most common chronic diseases, affecting about 150 million people worldwide[7]. The other is hypertension, whose prevalence is projected to increase globally, especially in the developing countries[8]. Although a lot has been said with respect to the wastefulness of obesity and metabolic condition associated with obesity, bothersome however, obesity and associated conditions still cause government and nongovernmental organisation so many funds. One opportunity for elucidating these mechanisms most likely involves identifying biomarkers that result from obesity and that independently enhance susceptibility for diabetes and/or hypertension. By implication, the search for biomarker to predict obese individual that will develop diabetes or hypertension in the future is an area that requires attention as such biomarkers will refine risk assessment and aid in diabetes and hypertension prevention in an obese individual. In this regards, C-reactive protein, an inflammatory marker, have been extensively studied and shown to be variably associated with obesity or cardiovascular endpoints[9, 10, 11].

The objective of this study is therefore to examine the lipid profile and atherogenic indices of obese hypertensive and diabetic patients, as well as compare these with the apparently healthy subjects.

2. Materials and Methods

Study Area: This study was conducted in Ekpoma, Benin City, Kwale and Asaba, all in the south-south zone of Nigeria.

Study Design: This study is a cross-sectional study involving simple random sampling and cohort sampling for subjects recruitment.

Ethical Consideration: Ethical approval was obtained from the LGAs Chairmen of health ethical committee and research proposal was considered and approved by the postgraduate school board of studies before the commencement of this study. The community/ Village Heads of the participants were duly informed and permission sorted for and was given after the aims and objectives of the study were explained to them. Also, informed consent was

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sought and obtained from the respondents before enrolment into the study.

Subjects and Grouping: Subjects were then classified as obese using BMI ≥ 30, diabetic using fasting plasma glucose (FBG) ≥ 7mmol/l (126mg/dl), hypertensive using blood pressure of ≥ 140/90mmHg and all subjects for the study was then classified into the following five (5) groups to meet the objective of the study.
Group A: Obese Diabetic (OD)
Group B: Obese Hypertensive (OH)
Group C: Obese Diabetic/Hypertensive (ODH)
Group D: Obese only (ONDH)
Group E: Non Obese Non Diabetic/Hypertensive [normal control(C)]
A total of two hundred and sixty-four (264) subjects were recruited for the study distributed as follow:
A=59, B=59, C=41, D=45, E= 60

Inclusion Criteria: All Subjects with BMI ≥30kg/m^2 and not on any form of medication were recruited as obese subjects while those with BMI between 18 and less than 25 kg/m^2 were recruited as non obese subjects.

Exclusion Criteria: All Subjects who are obese and non obese already diagnosed as been hypertensive and/or diabetic who are on medications and Subjects with BMI of 25 to less than 30 ie overweight were excluded from this study.

Duration of Study: The study was conducted from July, 2009 to June, 2012.

Sample Collection

Blood Pressure Measurement: Resting blood pressure was taken in a sitting position after a 5-10 minutes rest using a mercury sphygmomanometer according to standard procedures at least four different times and the mean recorded.

BMI Measurement: Heights were measured in standing position, with shoulder and buttocks against the wall, the subject looking straight ahead with joined feet, and arms hanging on both sides with a graduated tape. In addition, body weights were measured with a calibrated beam scale. These were used to calculate the BMI which is weight (kg)/height (m^2). Also the waist circumference (WC) of each individual was measured. WC was obtained using a graduated tape when subjects are in standing position. It was measured as the narrowest circumference of the trunk. However, when the narrowest circumference cannot be identified, the measurement was taken as the level of the last rib.

Preliminary Fasting Blood Glucose (FBG) Measurement: Preliminary measurement of FBG in all the subjects was done using Gluco-meter at least two different times with the mean value recorded. Subjects were asked to fast overnight (no food, drink, alcohol or smoking).

Blood Sample collection: About 5mls of fasting blood was collected from each subject following standard laboratory procedures.

Sample analysis: 0.5ml of the blood collected was immediately used for preliminary FBG estimation and 2.0mls immediately placed in fluoride oxalate container for FBG estimation spectrophotometrically as these served as basis for comparing FBG levels. 2.5mls was placed into lithium heparin bottle for lipid profile (TC, HDL-c, LDL-c and TG). The plasma was separated by centrifugation at 5000rpm for 15minutes. Samples was preserved at +4◦c to +8◦c till analysis.

Laboratory Analysis:

Glucose Estimation: The glucose Oxidase (GOD-POD) spectrophotometric method as described by Trinder[12] was employed.

Lipid Profile: Serum Triglyceride (TG) and Total Cholesterol (TC) concentrations were determined as described by Erickson *et al*[13], while High Density Lipoprotein Cholesterol (HDL-C) and Low Density Lipoprotein Cholesterol (LDL-C) were determined according to the method of Nicholls *et al*[14]. Very Low Density Lipoprotein Cholesterol (VLDL-C) was calculated using the Friedewald *et al* [15].
(e) Atherogenic Indices (Cardiac Risk Ratio-CRR and Atherogenic Index of the Plasma-AIP) Estimation was calculated as:
CRR=TC/HDLc
AIP=Log (TG/HDLc)

Data analysis: Data were presented as mean±S.D (standard deviation) and then analyzed using Statistical Package for Social Sciences (SPSS) at a P value of 0.05 and 95% level of confidence. Where suitable, simple statistics, pair sample t test and the one way analysis of variance were performed. A $p < 0.05$ was considered significant.

3. Results

Table 1 showed the status of lipid profile of obese hypertensive and diabetic patients com pared with the apparently healthy individual. TC levels was highest in non complicated obese group (Group D; 5.12±1.03 mmol/l) compared to apparently healthy subjects (group E; 5.06±1.20 mmol/l) and complicated obese groups (group A, B, and C); however, the difference are not significant. Comparatively, TC was higher in obese-hypertensive group (group B; 4.99±0.95 mmol/l) compared to obese-diabetic group (group A; 4.90±0.99 mmol/l) but lowest in co-existence of obese-hypertensive and diabetic group (group C; 4.85±0.79 mmol/l).

TG was highest in the control (apparently health subject; group E; 1.30±1.27 mmol/l) compared to the obsess groups irrespective of a co existing diseases. Obese only group (group D; 1.06±1.40 mmol/l) presented lower TG level however, it was not significantly different compared to the control. Compared to the control, TG level was significantly lower in obese hypertensive group (group B; 0.44±0.61

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mmol/l) and in obese diabetic and hypertensive group (group C; 0.75±0.74 mmol/l) compared to the control.

The level of HDL-c was lower in non complicated obese group (group D; 1.16±0.58 mmol/l) compared to the control group (group E; 1.23±0.69 mmol/l). On the other hand, complicated obese groups (group A, B and C) presented higher HDL-c levels compared to the control (group E; 1.23±0.69 mmol/l) with the obese hypertensive presenting (group B; 1.90±0.76) the highest level compared to the obese diabetic (group A; 1.34±0.57 mmol/l). However, the difference in HDL-c between groups were not significantly different ($p<0.05$).

The level of VLDL-c was higher in the control group (0.59±0.58 mmol/l) compared to the obese groups irrespective of complication. It was observed that the present of complication in obesity (group A, B and C) further reduces VLDL-c level and this was significant in obese hypertensive group (group B; 0.20±0.27 mmol/l) and in obese diabetic and hypertensive group (group C; 0.34±0.33 mmol/l) compared to the control and non complicated obese group (group D; 0.48±0.63 mmol/l).

The level of LDL-c was lower in the control group (3.24±1.51 mmol/l) compared to non complicated obese group (3.53±1.34 mmol/l) however the different is not significantly. On the other hand, LDL-c was observed to the further lower in complicated obese groups (group A, B and C) than the control with the obese hypertensive group (2.89±0.97 mmol/l) presenting a lower value than the obese diabetic group (3.13±0.97 mmol/l).

Table 1: Mean± S.D of lipid profile of the studied subjects

Parameter (mmol/l)	A	B	C	D	E
TC	4.90± 0.99 [a]	4.99± 0.95 [a]	4.85± 0.79 [a]	5.12± 1.03 [a]	5.06± 1.20 [a]
TG	0.92± 1.03 [a]	0.44± 0.61 [b]	0.75± 0.74 [c]	1.06± 1.40 [a]	1.30± 1.27 [a]
HDL-c	1.34± 0.57 [a]	1.90± 0.76 [a]	1.51± 0.68 [a]	1.16± 0.58 [a]	1.23± 0.69 [a]
VLDL-c	0.42± 0.46 [ac]	0.20± 0.27 [b]	0.34± 0.33 [ab]	0.48± 0.63 [c]	0.59± 0.58 [c]
LDL-c	3.13± 0.97 [a]	2.89± 0.97 [b]	2.99± 0.86 [ab]	3.53± 1.34 [ac]	3.24± 1.51 [ac]
Number of Subjects	59	59	41	45	60

Key: A= Obese Diabetic, B= Obese Hypertensive, C= Obese Diabetic/Hypertensive, D= Obese Non Diabetic/Hypertensive, E= Non Obese Non Diabetic/Hypertensive [normal control(C)].
All values are expressed as mean ± standard deviation.
Means in a row with different superscripts are significantly different at the $p<0.05$ level.

Table 2 showed the atherogenic indices of obese hypertensive and diabetic patients compared with the apparently healthy individual. The level of CRR was higher in the non complicated obese group (group D; 5.75±0.50 mmol/l) compared to the control (5.71±0.47 mmol/l) although not significant. Comparatively, the present of complication in obesity (group A-4.30±0.27mmol/l; group B- 3.22±0.25 mmol/l; group C-4.08±0.38mmol/l) presented a significant lower CRR level compared to non complicated obese group with the obese hypertensive group presenting the lowest CRR level.

On the level of AIP, non complicated obese group (group D; 0.38±0.12 mmol/l) presented a higher level compared to the control (0.15±0.08 mmol/l). AIP was observed to increase in obese complications groups (groups A, B and C) with the highest among the obese hypertensive (group B; 0.94±0.1 mmol/l) followed by the co-morbidity of diabetic and hypertension (group D; 0.55±0.11 mmol/l) and then the obese diabetic (group A; 0.41±0.10 mmol/l).

Table 2: Mean± S.D of atherogenic indices (CRR and AIP) of the studied subjects

Parameter (mmol/l)	Sub-groups of the studied subjects				
	A	B	C	D	E
CRR	4.30± 0.27 [a]	3.22± 0.25 [b]	4.08± 0.38 [c]	5.75± 0.50 [d]	5.71± 0.47 [d]
AIP	0.41± 0.10 [ac]	0.94± 0.10 [b]	0.55± 0.11 [c]	0.38± 0.12 [a]	0.15± 0.08 [d]
Number of Subjects	59	59	41	45	60

Key: A= Obese Diabetic, B= Obese Hypertensive, C= Obese Diabetic/Hypertensive, D= Obese Non Diabetic/Hypertensive, H= Non Obese Non Diabetic/Hypertensive [normal control(C)].
All values are expressed as mean ± standard deviation.
Means in a row with different superscripts are significantly different at the $p<0.05$ level.

4. Discussion

Considering the fact that dyslipidemia usually involve elevated plasma levels of TG, TC, LDL-c, VLDL-c and a low level of HDL-c, it is therefore justified to claim base on the findings of this study "that the non complicated obese state has the greatest dylipidemic and atherogenic potentials compared with the non obese and the complicated obese states". The findings of this study showed that the non complicated obese case (group D) has increased propensity to develop worsen lipid profile (dyslipidemia) and atherogenic diseases compared to the non obese and apparently healthy group (group E) and complicated obese groups (group A, B and C). That the non complicated obese group has increased potential to develop dyslipidemia and atherogenic potential compared to the non obese apparently healthy in this study is expected and understandable. In line with this, the increased atherogenic impact in obesity may be related to the increase dyslipidemia characterized by an increase in plasma triglycerides, large very low density lipoprotein (VLDL) particles, small dense low density lipoprotein (LDL) particles as well as low concentrations of high density (HDL) cholesterol as previously reported by Taskinen[16].

Worrisome is the reduced impact of dyslipidemia and atherogenic potentials in obese diabetic, obese hypertensive, and obese diabetic and hypertensive groups compared with the non complicated obese group. For example, the findings that obese diabetics (group A) have a better lipid profile status and a better artherogenic impact than the non complicated obese group (group D) disagrees with the finding of Abdul Hamid et al[19] who reported obese diabetics subjects to showed statistically significant increase in the

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levels of serum total lipids, serum total cholesterol, serum triglycerides, and serum LDL –cholesterol when compared to obese control.

Similarly, obese hypertensive case was observed to have a reduced dyslipidemic and atherogenic potential compared to the non complicated obese case. While excess body weight independent of hypertension, is associated with several other cardiovascular risk factors such as increased blood levels of LDL-c, low levels of HDL-c[20], the present study showed that obesity associated with hypertension has a reduced potential on dyslipidemia and atherogenic indices. On the other hand, because many of the reported complication in obesity are also associated with hypertension, it could be anticipated that the obese hypertensive patient would have an exaggerated outcome of dyslipidemia and atherogenic impact. Paradoxically, however, it has been reported that lean hypertensive patients might have a worse cardiovascular prognosis than obese hypertensive patients[21, 22]. This apparent advantage to obese hypertensive patients appears to persist even when other risk factors are taken into account[23]. This has led to speculation that obese hypertension and lean hypertension represent two genetically distinct forms of hypertension[23]. Except for TC where obese hypertensive group appear to present a non significant higher level than obese diabetic group, obese diabetic group presented significantly higher levels of TG, VLDL-c and LDL-c and a non significant lower HDL-c compared to obese hypertensive group. By implication, obese hypertensive case has a lesser dyslipidemic impact compared to obese diabetic case.

Another observation that is of great consideration in the present study is the better lipid profile and atherogenic indices in co-morbidity of diabetes and hypertension (group C) which in our expectation should present the most horrible dylipidemic and atherogenic impact. In fact, the dylipidemic and atherogenic potentials in co-morbidity of diabetes and hypertension are better than those observed in the non complicated obesity case (group D) and in the obese diabetic case (group A). Interestingly, the co-morbidity of diabetes and hypertension (group C) presented weaker dyslipidemic potential compared to the control (non obese and apparently healthy group E). However, the co-morbidity of diabetes and hypertension (group C) presented more powerful indicator of Atherogenic Index of the Plasma but lesser cardiac risk ratio. The finding of this study in the co-morbidity of diabetes and hypertension case disagrees with what is expected as the co-morbidity of diabetes and hypertension is expected to act in synergism to bring a poorer outcome in lipid profile. This assertion is based on the fact that the occurrence of diabetes and hypertension are multiplicative risk factors for macro- and microvascular disease, resulting in increased risk of cardiac death, coronary heart disease, congestive heart disease, cerebrovascular disease, and peripheral vascular disease[24].

Considering the fact that LDL is considered as an important risk factor in the development of CAD[25], it can be supposed from the findings of this study, that individuals who are obese without any associated diseases (such as hypertension or and diabetic) are more likely to die of CAD followed by the obese diabetic, obese diabetic and hypertensive and then lastly the obese hypertensive individual. Interestingly, this supposition is confirmed considering the level of cardiac risk ratio presented in this study (table 2). Although cardiac risk ratio is higher in obese diabetic group compared to obese hypertensive group, atherogenic index of plasma is higher in the obese hypertensive than obese diabetic. This observation therefore implicate that while obese diabetic may be more likely exposed to cardiovascular risk, obese hypertensive may be more likely expose to atherogenic diseases. In line with is assertion, epidemiological studies have established a strong association between hypertension and coronary artery disease[26].

5. Conclusion

One of the major risk factor for the development of CVD is dyslipidemia, which may be primary associated with hypertension, diabetes mellitus and obesity. The results showed that comparatively, obese hypertensive group presented higher TC and HDL-c levels while the obese diabetic group presented higher TG, VLDL-c and LDL-c levels. Interestingly, the co-morbidity of diabetic and hypertension presented a mid value between diabetic and hypertensive levels in the lipid profile parameters except for TC were it was lowest. The co-morbidity of diabetic and hypertension is expected to worsen the lipid profile but this was not the case in this study.

References

[1] Haslam, D.W. and James, W.P. (2005): Obesity". Lancet; 366 (9492): 1197–1209
[2] Kaufer M, Tavano L, Ávila H (2001). Obesidad en el adulto. In Nutriología Médica, 1st ed.; Casanueva, E., Kaufer, M., Pérez, A., Arroyo, P., Eds.; Editorial Médica Panamericana: México, México, Kahn BB, Flier JS (2000). Obesity and insulin resistance. J. Clin. Invest. 106:473–81
[3] Barness, L.A., Opitz, J.M. and Gilbert-Barness, E. (2007): Obesity: genetic, molecular, and environmental aspects". American Journal of Medicine and Genetic; A 143A (24): 3016–3034.
[4] Rahmouni K, Correia MLG, Haynes WG, Mark AL (2005). Obesityassociated hypertension: new insights into mechanisms. Hypertension. 45:9–14.
[5] Porte, D. and Kahn, S.E. (2001). Beta-cell dysfunction and failure in type 2 diabetes: potential mechanisms. Diabetes; 50(Suppl 1)**:**S160-S
[6] Goodman, E., Dolan, L.M. and Morrison, J.A. (2005). Factor analysis of clustered cardiovascular risks in adolescence: obesity is the predominant correlate of risk among youth. Circulation; 111:1970–1977..
[7] Matthaei S, Stumvoll M, Kellerer M, Haring HU (2000). Pathophysiology and pharmacological treatment of insulin resistance. Endoc.r Rev. 21:585–618.
[8] Murray CJ, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet. 1997; 349(9063):1436–1442.
[9] Lemieux, I., Pascot, A. and Prud'homme, D. (2001). Elevated C-reactive protein: another component of the atherothrombotic profile of abdominal obesity. Arteriosclerosis Thrombosis and Vascular Biology; 21:961–967.

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[10] Warnberg, J., Moreno, L.A. and Mesana, M.I. (2004). Inflammatory mediators in overweight and obese Spanish adolescents. The AVENA Study. International Journal of Obesity and Related Metabolic Disorder 28(suppl 3):S59–63.
[11] Rogowski, O., Shapira, I. and Toker, S. (2005). Obesity-related correlation between C-reactive protein and the calculated 10-y Framingham Coronary Heart Disease Risk Score. International Journal of Obstetric; 29:772–777.
[12] Trinder P (1969). Determination of glucose in blood using glucose oxidase with alternate oxygen acceptor. Annals of clinical biochemistry, 6:24.
[13] Erickson, S.K., S.R. Lear, M.A. Barker and T.A. Musliner, 1990.Regulation of cholesterol metabolism in the ethionine-induced premalignant rat liver. J. Lipid. Res., 31: 933-945.
[14] Nicholas S.B; Lipid disorders in obesity. Curr Hypertens Rep. 1 1999:131-136.
[15] Friedwald, W.T., Levy, R.I and Fredrickson, D.S (1972). Estimation of the concentration of low density lipoprotein cholesterol in the plasma, without use of the preparative ultracentrifuge. Clinical chemistry; 18:499-502.
[16] Taskinen MR (2005) Type 2 diabetes as a lipid disorder. Curr Mol Med 5: 297–308.
[17] Abdul Hamid Z., Farooq A.W., Mool Brahman W., Bashir A.L., Shariq R.M., Nissar A.S. (1995). Serum Lipid Profile in Non-insulin-dependent Diabetes Mellitus Associated with Obesity. INT. J. DIAB. DEV. COUNTRIES;, VOL. 15; 9-13.
[18] Nicholls, D.G., S.A. Cunningham and E. Rial, 1986.The Bioenergetic Mechanisms of Brown Adipose Tissue Thermogenesis. In: Trayhurn, P. and D.G. Nicholls (Eds.), Brown Adipose Tissue. Edward Arnold, London, pp: 52-85.
[19] Goldbourt U, Holtzman E, Cohen-Mandelzweig L, Neufeld H.N; Enhanced risk of coronary heart disease mortality in lean hypertensive men. Hypertension. 10 1987:22-28.
[20] Barrett-Connor E, Khaw K; Is hypertension more benign when associated with obesity?. Circulation. 72 1985:53-60.
[21] Carman W.J, Barrett-Connor E, Sowers M, Khaw K; Hypertension/risk factors. higher risk of cardiovascular mortality among lean hypertensive individuals in Tecumesh, Michigan. Circulation. 89 1994:703-711.
[22] Epstein M, Sowers JR. Diabetes mellitus and hypertension. Hypertension. . 1992; 19: 403–418.
[23] Criqui, M.H, Cowan, L.D and Tyroler, H.A (1987). Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality: results from the lipid research clinics follow-up study. American Journal of Epidemiology 126:629-637.
[24] Liu Y, Zhang B, Chen JY, Chen PY. The relationship between fasting triglyceride level and prevalence and severity of angiographic coronary artery disease in 16,650 patients from the TRUST study in the statins era. Eur Heart J. 2013;34 (Suppl 1):P1550.

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