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Lipid Profile in Metabolic Syndrome Associated with Diabetes, Hypertension, Chronic Kidney Disease and Apparent Health

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ABSTRACT:

Background: There is dyslipidaemia in metabolic syndrome (MetS). Lipid profiles in MetS associated with different health conditions may not be obvious. This study investigated lipid profiles in MetS associated with type 2 diabetes (DM-MetS), hypertension (HBP-MetS), chronic kidney disease (CKD-MetS) and apparent health (AH-MetS).

Methods: 540 patients were recruited for this study; 183 T2D, 136 HBP, 84 CKD patients and 137 AH subjects. They were outpatients and workers in the University of Nigeria Teaching Hospital Enugu. Nigeria. FPG, TC, HDL-C, TG as well as anthropometric measurements were determined using standard methods. Data analyses were done using GraphPad Prism version 2 statistical programme. MetS was diagnosed using NCEP-ATP 111 criteria

Results: Study showed 135 DM, 64 HBP, 31 CKD and 52 AH subjects had MetS, (prevalence rates: 36.9 %, 14.7 %, 18.4 %, and 37.9 % respectively). Only 38% MetS subjects had hypertriglyceridaemia while 66% with hypertriglyceridaemia had MetS. Corresponding figures for low HDLC were 40, and 77%. CKD-MetS had higher mean value of TG and TC than others; (2.65 ± 0.16 mmol/l; F = 11.4; P =0.0001; 6.07±0.02mmol/l; p = 0.001). Variations in TC were observed across groups, (p =0.0001). HDL-C was highest in AH-MetS, (1.51 ±0.07 mmol/l) and differed with mean value of DM-MetS, (1.23 ± 0.04mmol/l, p=0.01) only. Following the pattern of TC, LDLC was lowest among DM-MetS, (2.75 ± 0.06 mmol/l) and highest among CKD-MetS, (3.65 ± 0.28 mmol/l, p =0.001) with variations across groups, (F = 6.35; p= 0.0004).

Conclusion: Dyslipidemia profile varied with associated disorders. Presence of MetS is not a strong factor for development of lipid disorders in the study population.

KEYWORDS: metabolic syndrome, diabetes, chronic kidney disease, hypertension, lipid profile

INTRODUCTION

The insulin resistance/metabolic syndrome (MetS) is characterized by the variable coexistence of hyperinsulinaemia, obesity, dyslipidaemia, and hypertension.[1 ,2] Workers in the diabetes field have named the syndrome 'insulin resistance syndrome' in keeping with their view that the underlying factor of the syndrome is insulin resistance while obesity is an exacerbating factor [3, 4], (Defronzo and Ferrannini, 1991; Balkau and Charles, 1999). MetS is comprised of a combination of risk factors for coronary heart disease, as well as for diabetes, fatty liver, and includes dyslipidaemia of the high-triglyceride-low high density lipoprotein cholesterol type and central obesity[5-7] [3, 4, 5]. Alone each component of the cluster conveys increased cardiovascular risk, but in combination, they become much more powerful [8], (Kaplan, 1989). The combination of cardiovascular risk factors and diabetes elevates risk more than the accumulation of risk factors without diabetes (Stamler *et al*, 1993) [9]. The metabolic syndrome with normal glucose tolerance identifies the subject as a member of a group at a very high risk of future diabetes [10], (WHO, 1999). The metabolic syndrome develops slowly over time, often over a course of 20 years or more. Individuals over the age of 35 years have one in three chances of having the syndrome without knowing it[11] (Mykkanen *et al*, 1993). This is because its signs and symptoms which include weight gain; rising blood pressure, blood lipids and blood glucose, and feeling of sluggishness are also

accepted signs of aging. The syndrome shares common features with type 2 diabetes, Cushing's syndrome and growth hormone deficiency [12-13], (Alexander *et al*, 2003; Summer and Nelson, 2005).

Health conditions associated with MetS include diabetes, hypertension and chronic kidney disease and it also occurs in apparently healthy individuals. Lipid disorders are major diagnostic feature of MetS. However, lipid profile in different health conditions associated with MetS is not yet well understood especially among blacks. This is necessary in view of the "TG paradox" seen in blacks whereby normal triglyceride values were recorded in the presence of insulin resistance [14] [6]. Applying the same criteria in the treatment of lipid disorders in MetS associated with different health conditions may be misleading if there are differences. Several studies of different ethnic groups suggest different patterns of clustering of the metabolic disorders in metabolic syndrome [15-18], (Meigs *et a*l, 1997; Gray *et al*, 1998; Chen *et al*, 1999; Sakkinen *et al*, 2000). In this study, lipid profiles in MetS associated with DM, HBP, CKD and AH were examined.

MATERIALS AND METHODS

The study was a cross-sectional study done between March and August. A total of 540 patients were recruited for this study. This was made up of 183 type 2 diabetics, 136 hypertensives, 84 renal failure patients and 137 apparently healthy subjects recruited from the immediate community including Hospital staff. The patients were all registered in the clinics of the University of Nigeria Teaching Hospital, Enugu. Patients visiting the clinics for the first time, those aged less than 35 years and those who had broken their fast at the time of sample collection were left out. Left out also were those on lipid lowering drugs and pregnant and lactating mothers. The apparently healthy subjects were men and women of age 35 years and above who at the time of sample collection were not registered in any clinic for treatment or receiving any medication. They have not had any major illness in the last six months and were not complaining of any discomfort that might be suspected to be illness at the time of sample collection. The lower age limit was chosen to coincide with the age above which the metabolic syndrome is most prevalent [19], (Mykkanen *et al*, 1993).

Ethical approval was obtained from the Hospital Ethic Committee of the University of Nigeria teaching Hospital and written informed consent was obtained in each case before sample collection.

Fasting blood samples, (3ml), were collected from subjects between 8 and 11 am each day using standard methods [20][7] and allowed to clot at room temperature. The sample was spun and sera harvested and used for the determination of triglyceride, (TG), total cholesterol, (TC), and high density lipoprotein cholesterol, (HDLC). Low density and very low density cholesterol values were calculated using Friedwald formula [21] [8]. The method of Buccolo and David [22] [9] was used in assay of TG, HDLC by the method of Allain et al [23] [10] and TC by the cholesterol oxidase method. Cromatest^{R} Mono-reagent kits were used for the biochemical determinations.

Analyses of data were done with GraphPad prism version 2 statistical programme. MetS was diagnosed according to the NCEP-ATP 111 criteria [24] [11].

RESULTS

The study showed 135 (men 68, women 67) DM, 64 (men 26, women 38), HBP, 31 (men 20, women 11), CKD, and 52 (men 15, women 37), AH subjects had the MetS giving prevalence rates of 36.9 % (32.4, 42.9), 14.7 % (12.1, 17.1), 18.4 % (23.2, 13.4) and 37.9 % (22.0, 53.6) respectively. 38% of the subjects with the MetS had hypertriglyceridaemia while 66% of the subjects with hypertriglyceridaemia had the MetS. Corresponding figures for total hypercholesterolaemia and low HDLC were 5.0, 40, and 24 and 77% respectively.

Lipid profile of subjects with the MetS associated with DM, HBP, CKD and AH varied considerably and significantly (Table 1). CKD subjects had significantly higher mean value of TG than other subjects (2.65 ± 0.16 mmol/l; F = 11.4; P =0.0001) while those of the other subjects did not differ significantly from each other, (p >0.05). The concentration of TC was lowest among the MetS subjects with DM, (4.4 ±0.11mmol/l) and highest among those with CKD, (6.07±0.02mmol/l; p = 0.001). Significant variations were observed across the groups, (p =0.0001). HDL-C was highest in the AH-MetS subjects (1.51 ±0.07 mmol/l) and differed significantly with mean value from DM-MetS subjects, 1.23 ± 0.04mmol/l, (p=0.01) but not with those of CKD-MetS and HBP-MetS subjects. Following the pattern of TC, LDLC was lowest among the DM-MetS subjects, (2.75 ± 0.06 mmol/l) and highest among the CKD-MetS subjects, (3.65 ± 0.28 mmol/l, p =0.001) with significant variations across the groups, (F = 6.35; p= 0.0004). VLDLC pattern followed that of TG; CKD-MetS subjects had higher values than others (1.11mmol/l; F = 11.78; p = 0.0001) followed by AH-MetS, DM-MetS and HBP-MetS (p= 0.001). Among the male subjects, (Table 2), TG, followed the pattern in the general population. Significant difference in TC was recorded only between DM-MetS, (4.74 ± 0.10 mmol/l) and CKD-MetS, (5.78 ± 0.16 mmol/l)

subjects, (p= .01). HDLC was highest in the apparently healthy subjects but no significant difference was recorded among the groups, (p>0.05). No significant difference was recorded for LDLC between the groups while VLDLC showed considerable significant differences between only CKD-MetS and the other groups, (F = 18.60; p = 0.0001).

Among the female subjects, (Table 3), no significant difference in TG was recorded among the groups, (F = 2.68, p > 0.05). TC showed significant differences between the values for CKD-MetS, (7.10 ± 0.26mmol/l) and the other groups, (4.97 ± 0.09, 5.24 ± 0.21, 5.10 ± 0.27 mmol/l respectively) only. HDLC varied only between HBP-MetS and AH-MetS subjects with apparently healthy subjects showing higher values, (1.55 ± 0.10mmol/l) than the hypertension subjects, (1.21 ± 0.05mmol/l; p = 0.01). LDLC in line with the pattern recorded for TC in addition to that between HBP-MetS, (3.13 ± 0.15 mmol/l) and apparent health, (2.90 ± 0.17 mmol/l) (p = 0.01). VLDLC showed no significant variations.

DISCUSSIONS

Dyslipidaemia in MetS varied appreciably with associated disorders. Among the criteria in the definition of metabolic syndrome is dyslipidaemia of the high triglyceride, low high density lipoprotein type. This combination is the hallmark of the metabolic syndrome and risk factor for ischaemic heart disease [25] [12]. With hyperinsulinaemia, hepatic output of triglyceride-rich very low density lipoprotein, (VLDL) increases. Cholesterol ester transfer protein, (CETP), transfers cholesterol from high density lipoprotein (HDL) in exchange for triglycerides. As a result, the concentration of HDL-C falls while that of triglycerides increases. In addition, there is a shift in the LDL particle diameter to smaller and denser LDL-cholesterol fractions termed 'pattern B particles', which are the most potent and damaging kind. They adhere to and penetrate the vascular wall more easily, exert a pro-coagulant effect by causing increased production of PAI-1 and are oxidized more easily [26,27] [1314]. Oxidized LDL particle combine with macrophages to form foam cells and build atheromatous plaques. Dyslipidaemia is most severe in CKD-MetS and involves mainly TG and TC which values were significantly higher in CKD-MetS than in other combinations. The increase in lipids in CKD-MetS could be because in renal failure, low molecular weight proteins are lost. The body responds by increasing the production of higher molecular weight proteins that include mainly VLDL and LDL. These contain mainly TG and cholesterol and their values are raised in CKD-MetS more than in other conditions. HDLC correlates inversely with the TG and this is said to be highly atherogenic [28][15]. Low HDL-C levels and high triglyceride levels are an independent risk factor for cardiovascular disease [29] [16]. Cholesterol ester transfer protein, (CEPT), transfers cholesterol from HDL to VLDL in exchange for TG. Hence serum TG increases while HDLC decreases.

Hyperlipidaemia of the high TG-low HDLC type is the hallmark of MetS and it predisposes to ischaemic heat disease [30, 31] [12, 17]. Changes in triglycerides and HDL-C play an important role in the development of MetS, insulin resistance, and are major mediators in the atherogenic process. In addition, studies have identified the lipid profile as an insulin resistance and cardiovascular risk marker [32 -34] [18-20]. It is difficult to evaluate the predictor potential of raised TG and low HDLC in MetS. This is due to the fact that the cutoff points for these parameters in MetS lie within their reference ranges; that is to say that values within the reference range could still predict MetS in an individual. In this study only 38% of the subjects with the MetS had hypertriglyceridaemia while 66% of the subjects with hypertriglyceridaemia had the MetS. Corresponding figures for total hypercholesterolaemia and low HDLC were 5.0, 40, and 24 and77% respectively. It would appear that the development of the MetS is not a strong factor for the development of lipid disorders in the study population. The reverse, however, appear more likely. This agrees with the view that hypertriglyceridaemia causes the misdiagnosis of MetS in blacks [35 -37] [21-23]. Previous studies have also recorded that only a fraction of blacks with metabolic syndrome ever meet the TG threshold [14, 34,38] [6, 24 -,25]

Insulin resistance appears to be the primary mediator of metabolic syndrome and T2D and it is also seen in hypertension. Lower values for TG were recorded for DM-MetS, HBP-MetS subjects than for AH-MetS subjects. Dyslipidaemia in MetS was more severe in men than women. The variation in TG was less pronounced in women than men and vice versa for TC. Since apparently healthy subject also have the MetS, the syndrome may not be a consequence of the metabolic disorders associated with it but rather their causative factor. The individual components of the MetS produce adverse clinical consequences leading to organ damage. For example, adipose tissue dysfunction lead to release of pro-inflammatory cytokines that promote insulin resistance. Insulin resistance is a causative factor of several metabolic disorders.

CONCLUSION

Dyslipidemia profile varied with associated disorders. Presence of MetS is not a strong factor for development of lipid disorders in the study population. Subjects with dyslipidaemia and/or these complications, especially CKD, should also be screened and treated for dyslipidaemia to prevent enhanced cardiovascular events.

AUTHORS' CONTRIBUTIONS

ISIO designed the study and drafted the manuscript. BNE, CO, NM participated in the sample collection and analysis; EC and AE participated in the identification of subjects and sample collection. BNE, ENI and ON reviewed the manuscript. All authors read and approved the final version of the manuscript.

REFERENCES

1) Reilly MP*, Rader DJ. The metabolic syndrome: more than the sum of its parts?*
2) Circulation 2003*;* 108:1546–*51.*
3) Ayyobi *AF*, Brunzell JD. Lipoprotein distribution in the metabolic syndrome, type 2 diabetes mellitus, and familial combined hyperlipidemia. *Am J Cardiol* 2003*;* 92:27–33J.
4) De-Fronzo, R.A; Frrrannim, E; (1991). Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerotic cardiovascular disease. *Diabetes Care* 14 (3): 173 – 174.
5) Balkau, B; Charles, M.A; (1999). Comments on the provisional report from the WHO Consultation. European Group for the Study of Insulin Resistance, (EGIR). *Diab Med 16*(3): 442 – 443.
6) National Cholesterol Education Programme. 3rd Report of the NCEP on Detection and treatment of high blood Cholesterol in Adults (Adult Treatment Panel 111): final report. *Circulation* 2002; 106: 3143 -3421.
7) Flegal, K.M; Caroll, M.D; Ogden, C.L; Johnson, C.L. Prevalence and trend in obesity among US adults 1999-2000. *JAMA* (2002); 288: 1723 – 1727.
8) Alexander, C.M; Landsman, T.B; Teutsh, S.M; Haffner, S.M;. National Cholesterol Education Programme, (NCEP) – defined metabolic syndrome, diabetes and prevalence of coronary heart disease among National Health and Nutrition Examination Survey, (NHANES) 111 participants age 50 years and older. *Diabetes* (2003); *52* (8): 1210 – 1214.
9) Kaplan, N.M; (1989). The deadly quartet: upper body adiposity, glucose intolerance, hypertriglyceridaemia and hypertension. *Arch Intern Med* 149: 1514 – 1520.
10) Stamler, J; Stamler, A; Neaon, J.D; (1993). Blood pressure, systolic and diastolic and cardiovascular risk; US Population Data. *Arch Intern Med* 153: 598 – 615.
11) World Health Organization (1999). Definition, diagnosis and classification of diabtes mellitus. Geneva, WHO department of Non-Communicable disease Surveillance
12) Mykkanen, L; Kuusisto, J; Pyoralas, K; Laakso, M; (1993). Cardiovascular risk factors as predictors of type 2 (non-insulin dependent) diabetes mellitus in the elderly subjects. *Diabetologia* 36: 553 – 559.
13) Alexander, C.M; Landsman, T.B; Teutsh, S.M; Haffner, S.M; (2003). National Cholesterol Education Programme, (NCEP) – defined metabolic syndrome, diabetes and prevalence of coronary heart disease among National Health and Nutrition Examination Survey, (NHANES) 111 participants age 50 years and older. *Diabetes 52* (8): 1210 – 1214.
14) Summer, S.A; & Nelson, D.H; (2005). A role for sphingolipids in producing the common features of type 2 diabetes, metabolic syndrome X and Cushing's syndrome. *Diabetes 53*(30: 591- 602.
15) Sophia S.K, Yu, Darleen C Casttilo, Amber B Courville, Anne E Summer. The Triglyceride Paradox in people of African descent. *Metab Syndr Relat Disord*. (2012);10(2): 77 – 82
16) Meigs, J.B; D'Agostino, R.B.S; Wilson, P.W; Gupples, L.A; Nathan, D.M; Singer, D.E; (1977). Risk variable clustering in the insulin resistance syndrome: the Framingham Offspring Study. *Diabetes 46* (10): 1594 – 1600.
17) Gray, R.S; Fabsitz, R.R; Cowan, L.D; Lee, E.T; Howard, B.V; Savage, P.J; (1998). Risk factor clustering in the insulin resistance syndrome: The Strong Heart Study. *Am J Epidemiol* 148:869 – 878.
18) Chen, W; Srinivasan, S.R; Elkasabany, A; Berenson, G.S; (1999). Cardiovascular risk factor clustering features of insulin resistance syndrome, (Syndrome X) in a biracial (blck-white) population of children, adolescents and young adults: The Bogahisa Heart Study. *Am J Epidemiol* 150: 667 – 674.
19) Sakkinen, PA, Wahl, P, Cushman, M, Lewis, MR, Tracy, RP, (2001). Clustering of procoagulation, inflammation and fibrinolysis variables with metabolic factor in insulin resistance syndrome. *Am J Epidemiol* 152: 897 – 907.
20) Mykkanen, L; Kuusisto, J; Pyoralas, K; Laakso, M; (1993). Cardiovascular risk factors as predictors of type 2 (non-insulin dependent) diabetes mellitus in the elderly subjects. *Diabetologia* 36: 553 – 559.
21) Dacie, J.V; Lewis, S.M; (1975). Practical Haematology. 5th Edition ELBS and Churchill Livingstone. P. 1-20
22) Friedewald, W.T; Levy, R. I; Fredrickson, D.S. Estimation of the concentration of low density lipoprotein cholesterol without the use of the preparatory ultracentrifuge. *Clin Chem* . (1972);18: 499-502

23) Buccolo, G; David, H. Quatitative determination of serum triglycerides by the use of enzymes. *Clin Chem* (1973); 19(4): 476 – 482.
24) .Allain, C.C; Poon, L.S; Clau, C.G.S; Richmond, W; Fu, P.D. Enzymatic determination of total cholesterol. *Clin Chem* (1974); 20(3): 470 – 475.
25) National Cholesterol Education Programme (NCEP).Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in adults, (Adult Treatment Panel 111) final report. *Circulation* (2002); 106 (25): 3143 – 3421.
26) Dows, J.R. Clerafeld, M. Weiss, S. Whitney, E. Shapiro, D.R. Beere, P.A. Langendorf, A. Stein,
27) Libby, P. Current concepts of the pathogenesis of the acute coronary syndrome. *Circulation* (2001); 104 (3): 365 – 372.
28) Krentz, A.J. Lipoprotien abnormalities and their consequences for patients with type 2 diabetes. *Diab Obes Metab* (2003); 5: 519 – 527.
29) Choi SH and Ginsberg HN. Increased very low density lipoprotein (VLDL) secretion, hepatic steatosis, and insulin resistance. *Trends in Endocrinol Metab.* 22:353-363
30) Pedroza-Tobias, A., Trejo-Valdivia, B., Sanchez-Romero, L.M. *et al.* Classification of metabolic syndrome according to lipid alterations: analysis from the Mexican National Health and Nutrition Survey 2006. *BMC Public Health* (2014); 14**,** 1056
31) Jeppesen, J; Hein, H.O; Saudicani, P; Cyntelberg, F. High triglyceride and low high density lipoprotein cholesterol and blood pressure and risk of ischaemic heart disease. *Hypertension* (2000); 36: 226 – 229.
32) Mazza A, Tikhonoff V, Schiavon L, Casiglia E: Triglycerides + high-density-lipoprotein-cholesterol dyslipidaemia, a coronary risk factor in elderly women: the CArdiovascular Study in the ELderly. *Intern Med J*. (2005); 35 (10): 604-610.
33) McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, Simon J, Krauss RM: Is there a simple Way to identify insulin-resistant individuals at increased risk of cardiovascular disease?. *Am J Cardiol*. (2005); 96: 399-404
34) McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G: Use of metabolic markers to identify overweight individuals who are insulin resistant. *Ann Intern Med*. (2003); 139 (10): 802-809.
35) Giannini E. Testa R. The metabolic syndrome: All criteria are equal, but some criteria are more equal than others. *Arch Intern Med*. 2003; 163:2787–2788.
36) Osei K. Metabolic syndrome in blacks: Are the criteria right? *Curr Diab Rep*. 2010; 10:199–208.
37) Sumner AE. Zhou J. Doumatey A, et al. Low HDL-cholesterol with normal triglyceride levels is the most common lipid pattern in West Africans and African Americans with metabolic syndrome: Implications for cardiovascular disease prevention. *CVD Prev Control*. 2010; 5:75–80.
38) Ervin RB. Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003–2006. *Natl Health Stat Report*. 2009:1–7.
39) Park YW. Zhu S. Palaniappan L, et al. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988–1994. *Arch Intern Med*. 2003; 163:427–436.

Table 1. Showing the values (mmol/l), (Mean ± SEM) of the lipid profiles of MS subjects with DM, (DM-MS), hypertension, (HBP-MS), chronic kidney disease, (CKD-MS) and apparent health, (AH-MS).

	DM-MS (n= 135)	HBP-MS (n= 64)	CKD-MS (n= 31)	AH-MS (n= 52)	F; p-values.
Triglycerides(TG)	1.87 ± 0.04	2.01 ± 0.05	2.65 ± 0.16	2.04 ± 0.06	11.4; 0.0001
Total Cholesterol (TC)	4.44 ± 0.11	5.09 ± 0.16	6.07 ± 0.22	5.16 ± 0.18	15.15; 0.0001
HDLC	1.23 ± 0.04	1.30 ± 0.05	1.23 ± 0.05	1.51 ± 0.07	4.79; 0.0029
LDLC	2.75 ± 0.06	3.09 ± 0.15	3.65 ± 0.28	2.86 ± 0.18	6.35; 0.0004
VLDLC	0.88 ± 0.02	0.83 ± 0.15	1.11 ±0.05	0.93 ± 0.03	11.78; 0.0001

Table 2. Showing the values (mmol/l), (Mean ± SEM) of the lipid profiles of male MS subjects with DM, (DM-MS), hypertension, (HBP-MS), chronic kidney disease, (CKD-MS) and apparent health, (AH-MS).

	DM-MS (n= 68)	HBP-MS (n= 26)	CKD-MS (n= 20)	AH-MS (n= 15)	F; p-values.
Triglycerides(TG)	1.76 ± 0.04	1.77 ± 0.06	2.31 ± 0.10	1.72 ± 0.05	17.8; 0.0001
Total Cholesterol TC	4.74 ± 0.10	4.72 ± 0.16	5.78 ± 0.16	5.22 ± 0.11	2.75; 0.029

HDLC	1.18 ± 0.05	1.42 ± 0.09	1.29 ± 0.08	1.44 ± 0.99	2.59; 0.056
LDLC	2.96 ± 0.22	2.64 ± 0.14	3.15 ± 0.21	2.91 ± 0.12	0.41; 0.747
VLDLC	0.79 ± 0.02	0.830± 0.03	1.07 ±0.06	0.77 ± 0.02	18.6; 0.0001

Table 3. Showing the values (mmol/l), (Mean ± SEM) of the lipid profiles of female MS subjects with DM, (DM-MS), hypertension, (HBP-MS), chronic kidney disease, (CKD-MS) and apparent health, (AH-MS).

	DM-MS (n= 67)	HBP-MS (n= 38)	CKD-MS (n= 11)	AH-MS (n= 37)	F; p-values.
Triglycerides(TG)	1.87 ± 0.07	1.69 ± 0.07	1..77 ± 0.06	1.82 ± 0.06	2.68; 0.0491
Total Cholesterol TC	4.97 ± 0.09	5.24 ± 0.21	7.10 ± 0.26	5.10 ± 0.27	11.79; 0.0001
HDLC	1.26 ± 0.05	1.21 ± 0.05	1.25 ± 0.04	1.55 ± 0.10	4.13; 0.0078
LDLC	2.64 ± 0.09	3.13 ± 0.15	3.80 ± 0.16	2.90 ± 0.17	15.96; 0.0001
VLDLC	0.88 ± 0.02	0.75 ± 0.03	0.80 ±0.02	0.83 ± 0.02	2.648; 0.5450