Triglyceride paradox in Nigerians living with HIV

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Original article

ABSTRACT

Objective: To achieve early detection of type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVDs), the concept of metabolic syndrome (MetS) was designed as a mathematical construct with diagnosis made when three out of five features are present. However, racial differences exist in the predictive value of MetS in identifying risk of T2DM and CVD. Whilst the prevalence of MetS is higher in Whites than Blacks, Blacks show a higher prevalence of T2DM, hypertension and CVDs. This discordance may be due to the fact that while Whites display the classic pattern of elevated triglyceride (TG) and low HDL-C, Blacks usually have normal TG in the presence of low HDL-C, the so-called "triglyceride paradox". We determined the presence of triglyceride paradox in people living with Human Immunodeficiency Virus (PLWHA) in whom there is little or no earlier report of this phenomenon.

Methodology: This cross-sectional study evaluated the lipid pattern of 265 PLWHA.

Results: Low HDL-C with normal TG was found in 127 (47.9%) while 19 (7.2%) had low HDL-C and elevated TG. Low HDL-C with normal TG was found in 7 (29.2%) and 15 (55.6%) of sub-cohorts with diabetes and hypertension respectively. MetS was present in 31 (11.7%) participants and low HDL-C and normal TG was the most common lipid pattern 17 (54.8%) in them.

Conclusion: Low HDL-C and normal TG was the most common lipid pattern. This calls for longitudinal studies to re-define the cut-off point of TG level used in PLWHA in order to improve the predictive value of MetS in the early diagnosis of CVDs and T2DM.

Key words: Lipid pattern, triglyceride paradox, people living with HIV.

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Triglycérides paradoxe de Nigérians vivant avec le VIH

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RÉSUMÉ

Objectif: Atteindre détection précoce du diabète de type 2 (T2DM) et les maladies cardiovasculaires (CVDs), la notion de syndrome métabolique (mets) a été conçu comme une construction mathématique avec diagnostic fait lorsque trois des cinq fonctions sont présents. Toutefois, les différences raciales existent dans la valeur prédictive des mets dans l'identification des risques de T2DM et MCV. Tandis que la prévalence de la norme est plus élevé chez les Blancs que chez les Noirs, les Noirs montrent une prévalence plus élevée de T2DM, hypertension et CVDS. Cette discordance peut être due au fait que tandis que les Blancs afficher le schéma classique d'élévation des triglycérides (TG) et faible le HDL-C, les Noirs ont généralement normal TG en présence de faible HDL-C, la prétendue "triglycéride paradoxe". Nous avons déterminé la présence de triglycérides paradoxe chez des personnes vivant avec le virus de l'immunodéficience humaine (VIH/SIDA) dans lesquels il y a peu ou aucun rapport antérieur de ce phénomène.

Méthodologie: Cette étude transversale évalué les lipides pattern de 265 personnes vivant avec le VIH/sida.

Résultats: Bas niveau de cholestérol HDL -C avec la normale TG a été trouvé dans 127 (47,9 %) tandis que 19 (7,2 %) avaient un faible le HDL-C et de l'élévation des TG. Faible le HDL-C avec la normale TG a été trouvé dans 7 (29,2 %) et 15 (55,6 %) des sous-cohortes avec le diabète et l'hypertension respectivement. Mets était présent dans 31 (11,7 %) les participants et faible le HDL-C et TG normale était la plus courante des lipides modèle 17 (54,8 %) en eux.

Conclusion: Faible le HDL-C et TG normale était la plus courante des lipides pattern. Cela appelle des études longitudinales pour re-définir le point de coupure de TG niveau utilisé dans PVS afin d'améliorer la valeur prédictive des mets dans le diagnostic précoce de CVDS et T2DM.

Mots clés: modèle des lipides, des triglycérides paradoxe, les personnes vivant avec le VIH.

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INTRODUCTION

In 2008, the estimated global death from cardiovascular diseases (CVDs) was 17 million and this accounted for approximately 30% of the global 57 million deaths (1). Diabetes mellitus (DM) accounted for additional 1.3 million deaths (1). The recently published Global Burden of Disease Study 2010 (GBD 2010) report indicated that in the age group 15 – 49 years, CVDs accounted for 10.7% and 12.8% of global deaths in females and males respectively (2). Reports from middle- and high-income countries also suggest that there is increased risk of CVDs among people living with human immunodeficiency virus/acquired immune deficiency syndrome (PLWHA) (3-6). Although reports from sub-Saharan Africa (SSA), the region with the greatest burden of human immunodeficiency virus (HIV) (7) showed that tuberculosis and other pulmonary infections, sepsis and advanced HIV disease are the leading causes of death in PLWHA, cardiovascular risk factors (CVRFs) and CVDs are fairly prevalent in this population (8-11). In order to prevent the double jeopardy of deaths from infectious and CVDs in our population of PLWHA, attempts must be made to diagnose CVD early and to institute effective treatment.

In a bid to achieve early detection of T2DM and CVDs, the concept of metabolic syndrome (MetS) was designed as a mathematical construct with diagnosis of MetS made when three out of five features i.e. obesity, elevated blood pressure (BP), low high-density lipoprotein cholesterol (HDL-C), elevated blood glucose and hypertriglyceridaema are present (12). Although MetS carries a five-fold risk of T2DM and a two-fold risk of CVDs, there are racial differences in the predictive value of MetS in identifying risk of T2DM and CVD (13,14). For instance, whilst the prevalence of MetS is higher in Whites than Blacks, Blacks show a higher prevalence of T2DM, hypertension and CVDs (15,16). One explanation put forward to explain this discordance in the prevalence of MetS and presence of T2DM and CVD is that while Whites display the classic pattern of elevated triglyceride (TG) and low HDL-C (referred to as dyslipidaemia of insulin resistance), Blacks usually have normal TG in the presence of low HDL-C (12,17). This lipid paradox in insulin resistant Blacks has been referred to as *"triglyceride paradox"* by some workers (12,17). This has made many researchers to call for the lowering of the present cut-off point of TG level used in the diagnosis of MetS in people of African descents in other to improve the predictive value of MetS in the early diagnosis of CVDs and T2DM (21,17). Although TG paradox has been documented in people of African descent there is limited or no data in PLWHA (18-21). We therefore examined the prevalence of TG paradox in our population of PLWHA without any prior documentation of CVD or CVRFs before the commencement of the study.

MATERIALS AND METHODS

The ethical approval for the study was obtained from the Ladoke Akintola University of Technology Teaching Hospital Research Ethics Committee. The participants involved gave both verbal and written consent after the study was explained to them. The study involved 265 consecutive PLWHA seen over a 4 month period (September 2011 to January 2012) at our dedicated PLWHA clinic. The details of the methodology were as documented in our earlier publication (11). Patients who refused to give consent, those with prior history of hypertension, DM, use of lipid-lowering medication, CVD, liver disease, thyroid disease, ischaemic heart disease (IHD) and cerebrovascular disease and acute illness necessitating admissions were excluded from the study. We used the World Health Organization (WHO) STEPS questionnaire developed for non-communicable diseases (NCDs) (22). Information obtained included age, gender, educational status, smoking history, alcohol intake, level of physical activity, and family history of NCDs such as hypertension, DM, IHD, and CVD. Anthropometric variables obtained included weight (kg) in light clothing with the shoes off, height (m) using a stadiometer, waist circumference (WC) (m) using a tape measure in light contact but not compressing the skin midpoint between the lowest rib and the iliac crest and hip circumference (HC) (m) at the levels of the greater trochanters. The body mass index was calculated from weight /height^2 (kg/m^2). Three blood pressure (BP) and pulse rate (PR) readings were obtained from each participant using the A&D UA 767 automated manometer which had been validated by the British Hypertension Society (23); and the average of the two last readings was used for statistical analysis. Intake and duration of highly active antiretroviral therapy (HAART) were noted.

We obtained fasting blood glucose and lipid profile of the participants using the glucose oxidase method and commercially available reagents (Randox laboratories Ltd, UK) respectively. Components of MetS were defined as follows: abnormal waist circumference (≥ 102 cm in males and ≥ 88 cm in females, impaired fasting glucose (FPG ≥ 6.1 mmol/L), low HDL-C (<1.2 mmol/L in females, < 1.0 mmol/L in males) high BP (systolic blood pressure (SBP) ≥ 130 mm Hg and /or diastolic blood pressure (DBP) ≥ 85 mm Hg) elevated TG (≥ 1.7 mmol/L) (24). Metabolic syndrome was defined as the presence of three or more of the component traits according to the ATP III criteria.[24] Diabetes was defined as fasting plasma glucose ≥ 7 mmol/L (25) and hypertension was defined as systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg (26).

Statistical Analysis

Categorical variables are presented as percentages and Chi-square was used to assess the degree of association between categorical variables. Quantitative variables are expressed as means ± standard deviation (SD) and differences between two means were assessed using the student's t-test. All analyses were done using the Statistical Package for Social Sciences software, version 16 (SPSS, Chicago, IL).

RESULTS

Out of a population of 265 PLWHA, 179 (67.5%) were females. The mean age of the participants was 38.7 ± 8.7 years and the males were significantly older than the females (41.7 ± 8.4 vs. 37.2 ± 8.5 years, p < 0.001). Current smoking and alcohol intake were found in 5 (1.9%) and 19 (7.2%) participants respectively. Two hundred and fourteen (80.8%) were on HAART out of which 194 (90.7%) were on zidovudine, lamivudine and nevirapine while 12 (5.6%) were on tenofovir-based regimen. The mean duration of HAART intake was 17.3 ± 11 months (range 1 – 63 months). The lipid patterns of the population according to gender and HAART use are as shown in Table 1. There were no significant gender differences in the mean values of TC, TG, HDL-C, and LDL-C in the study participants. However, low HDL-C was significantly more common in females when compared to males (60.3% vs. 44.2%, p = 0.013). There were no significant statistical differences in the mean values of TC, TG, HDL-C and LDL-C in those on HAART when compared to HAART-naïve participants.

Low HDL-C with normal TG was found in 127 (47.9%) participants. Only 19 (7.2%) of participants with low HDL-C had elevated TG (Table 1). Low HDL-C with normal TG was seen in 97 (45.3%) of those on HAART and 30 (58.8%) of HAART-naïve participants indicating that this pattern was the most common lipid combination in our cohort. In the sub-cohort without hypertension and DM, the most common lipid combination was low HDL-C with normal TG which occurred in 103 (49.1%) participants. Twenty seven (10.2%) of the study participants had hypertension, 24 (9.1%) had DM and 4 (1.5%) had a combination of hypertension and DM. In the sub-cohort of participants with hypertension, 15 (55.6%) had low HDL-C and normal TG while none had low HDL-C and elevated TG. In the sub-cohort with DM, 7 (29.2%) had low HDL-C and normal TG while 6 (25.9%) had low HDL-C and elevated TG. Two of the patients with a combination of hypertension and DM had low HDL-C with normal TG while the other two had normal HDL-C and normal TG. Of the 31 (11.7%) participants with MetS, only 11 (35.5%) had elevated TG. Low HDL-C with normal TG was seen in 17 (54.8%) of participants with MetS.

DISCUSSION

In the total study population and sub-cohorts with hypertension, and DM, the most common lipid pattern was low HDL-C and normal TG. This is consistent with earlier reports in Black populations which showed that Blacks generally usually have normal TG in the presence of low HDL-C (12,17,18). This has been referred to as "TG paradox" by many workers because TG level is expected to be elevated in the presence of low HDL-C (12,17). A few mechanisms have been proposed to explain normal TG in the presence of low HDL-C. The activity of lipoprotein lipase (LPL), the enzyme responsible for clearing TG-rich particles from the circulation is inversely related to the activity of hepatic lipase (HL), the enzyme that clears HDL-C (27). Thus, in the presence of insulin resistance, LPL activity decreases and HL activity increases leading to elevated TG and low HDL-C. Lipoprotein lipase activity has been found to be higher in Blacks compared to Whites (27). Also, apolipoprotein CIII (which inhibits LPL activity) levels are lower in Blacks than in Whites (28). In addition, LPL activity is not inhibited by insulin resistance in Blacks, such that Blacks are able to clear TG from the circulation even when there is insulin resistance (27,28). Thus, there is a greater clearance of TG-rich lipoprotein in Blacks than Whites.

The most common lipid pattern in participants with MetS was low HDL-C with normal TG which was seen in 17 (54.8%) participants. This is in keeping with reports that showed that unlike Caucasians who tend to have elevated TG, Blacks with MetS usually have normal TG in the presence of low HDL-C (12,27). This relative absence of dyslipidaemia of insulin resistance in Blacks may explain the lower-than-expected prevalence of MetS though Blacks show a higher prevalence of T2DM, hypertension and CVDs. Thus, the cut-off thresholds used to define hypertriglyceridaemia and low HDL-C in Blacks may need to be redefined in order to improve the predictive value of MetS in identifying people with high risk of developing DM and cardiovascular diseases. Lipid abnormalities noted in PLWHA before the advent of HAART included reduced levels of TC, HDL-C and LDL-C and hypertriglyceridaemia (29). With the introduction of HAART, non-nucleoside reverse transcriptase inhibitors (NNRTIs) and protease inhibitors (PIs) (with the exception of atazanavir) were noted to cause elevated TC, HDL-C, and TG (29). In our study, we did not find any significant difference in the mean levels of TC and the different fractions among those on HAART when compared with HAART-naïve participants. In addition, the most common lipid pattern was low HDL-C and normal TG in those on HAART and HAART-naïve participants indicating that HAART use did not significantly alter this lipid pattern in our patients. The short duration of HAART use in our patients (mean duration, 17.3 ± 11 months [range 1 – 63 months]) may, however, have prevented the full manifestation of lipid abnormalities associated with HAART.

In conclusion, the most common lipid pattern in our population of PLWHA was low HDL-C and normal TG which has been referred to as *triglyceride paradox* by some workers. However, it must be borne in mind that the absence of elevated TG does not rule out the presence of risk for CVD and T2DM. This finding calls for prospective longitudinal studies to define the cut offs for elevated TG and its clustering effect with other cardiometabolic risk factors on predicting risk for CVD and T2DM in the Black population (12,20). This in turn will help to improve the predictive value of MetS in the early diagnosis of CVDs and T2DM and improve clinical outcomes in these patients.

Conflict of Interest

The authors declare no conflict of interest

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REFERENCES

1. World Health Organization. Global Status Report on Non-communicable Diseases 2010. Available at www.who.int/nmh/publications/ncd_report_full_en.pdf . Accessed December 11, 2013.
2. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990 – 2010: a systematic analysis for the Global Burden of Diseases Study 2010. Lancet 2012; 380: 2224 – 2260.
3. Lewden C, Salmon D, Morlat P, Bevilacques S, Jougla E, Bonnet F, et al: Mortality 2000 study group. Causes of death among human immunodeficiency virus (HIV) – infected adults in the era of potent antiretroviral therapy: emerging role of hepatitis and cancers, persistent role of AIDS. Int J Epidemiol 2005; 34: 121 – 130.
4. Glass TR, Ungsedhapand C, Wolbers M, Weber R, Vernazza PL, Rickenbach M, et al. and the Swiss HIV Cohort Study. Prevalence of risk factors for cardiovascular disease in HIV-infected patients over time: the Swiss HIV Cohort Study. HIV Medicine 2006; 7: 404 – 410.
5. Savès M, Chêne G, Ducmetrière P, Leport C, Le Moal G, Amouyel P, et al. for French WHO MONICA Project and the APROCO (ANRS EP 11) Study Group. Risk factors for coronary heart disease in patients treated for human immunodeficiency virus infection compared with the general population. Clin Infect Dis 2003; 37: 292 – 298.
6. Friis-Møller N, Sabin CA, Weber R, d' Arminio Monforte A, El-Sadr WM, Reiss P et al. Data Collection on Adverse Events of Anti-HIV Drugs (DAD) Study Group. Combination antiretroviral therapy and the risk of myocardial infarction. N Engl J Med 2003; 349: 1993 – 2003.
7. UNAIDS World AIDS Day Report 2012. Available from: . [accessed on April 30, 2013]
8. Muronya W, Sanga E, Talama G, Kumwenda JJ, van Oosterhout JJ. Cardiovascular risk factors in adult Malawians on long-term antiretroviral therapy. Trans R Soc Trop Med Hyg 2011; 105: 644 – 649
9. Sani MU, Mohammed AZ, Adamu B, Yusuf SM, Samaila AA, Borodo MM. AIDS mortality in a tertiary health institution: a four year review. J Natl Med Assoc 2006; 98: 862 – 866.
10. Lucas S. Causes of death in the HAART era. Curr Opin Infect Dis 2012; 25: 36 – 41.
11. Ayodele OE, Akinboro AO, Akinyemi SO, Adepeju AA, Popoola AA. Prevalence of traditional cardiovascular risk factors and evaluation of cardiovascular risk using three risk equations in Nigerians living with human immunodeficiency virus. North Am J Med Sci 2013; 5: 680 – 688.
12. Sumner AE, Zhou J, Doumatey A, Imoisili OE, Amoah A, Acheampong J, 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.
13. Alberti KGMM, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Danato KA et al. Harmonizing the metabolic syndrome: a Joint Interim Statement of the of the International Diabetes

Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation 2009; 120: 1640 – 1645.

14. Grundy SM. Metabolic syndrome pandemic. Arterioscler Thromb Vasc Biol 2008; 28: 629 – 636.
15. Cowie CC, Rust KF, Byrd-Holt DD et al. Prevalence of diabetes and impaired fasting glucose in adults in the US population: National Health and Nutrition Examination Survey 1999 – 2002. Diabetes Care 2006; 29: 1263 – 1268.
16. Lloyd-Jones D, Adams R, Carnethon M, et al. Heart disease and stroke statistics – 2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009; 119: e21 – e181.
17. Yu SSK, Castillo DC, Courville AB, Sumner AE. The triglyceride paradox in people of African descent. Metab Syndr Relat Disorders 2012; 10: 77 – 82.
18. Sumner AE. Ethnic differences in triglyceride levels and high-density lipoprotein lead to underdiagnosis of the metabolic syndrome in black children and adults. J Pediatr 2009; 155: S7.e7 – 11.
19. Sumner AE, Finley KB, Genovese DJ, Criqui MH, Boston RC. Fasting triglyceride and the triglyceride-HDL cholesterol ratio are not markers of insulin resistance in African Americans. Arch Intern Med 2005; 165: 1395 – 1400.
20. Lin SX, Carnethon M, Szklo M, Bertoni A. Racial/ethnic differences in the association of triglycerides with other metabolic syndrome components: the Multiethnic Study of Atherosclerosis. Metab Syndr Relat Disord 2011; 9: 35 – 40.
21. Sumner AE, Cowie CC. Ethnic differences in the ability of triglyceride levels to identify insulin resistance. Atherosclerosis 2008; 196: 696 – 703.
22. World Health Organization STEPS Instrument. Available at www.who.int/entity/chp/steps/STEPS_instrument_v2.1.pdf Accessed January 2, 2010
23. British Hypertension Society. Validated monitors. Available at www.bhsoc.org/blood_pressure_list.stm/bp_monitors/automatic/stm. Accessed on January 10, 2012.
24. 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.
25. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2013; 36 Suppl 1: S67 – 74.
26. Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Bohm M et al. 2013 ESH/ESC Guidelines for the management of arterial hypertension. J Hypertens 2013; 31: 1281 -1357.
27. Sumner AE, Vega GL, Genovese DJ, Finley KB, Bergman RN, Boston RC. Normal triglyceride levels despite insulin resistance in African Americans: role of lipoprotein lipase. Metabolism Clinical and Experimental 2005; 54: 902 – 909.
28. Florez H, Mendez A, Casanova-Romero P, Larreal-Urdaneta C, Castillo-Florez S, Lee D, et al. Increased apolipoprotein C-III levels associated with insulin resistance contribute to dyslipidemia in

normoglycemic and diabetic subjects from a triethnic population. Atherosclerosis 2006; 188: 134 – 141.

29. Blanco F, Róman JS, Vispo E, Lopez M, Salto A, Abad V, et al. Management of metabolic complications and cardiovascular risk in HIV-infected patients. AIDS Rev 2010; 12: 231 – 241.

Table 1: Biochemical and clinical profile of the study population

Characteristics	Total	Gender		P value	HAART		P value
		Male (n=86)	Female (n=179		No (n=51)	Yes (n=214)	
Mean TC± SD (mmol/L)	4.50 ± 1.49	4.35 ± 1.42	4.58 ± 1.52	0.24	4.47 ± 1.64	4.52 ± 1.45	0.85
Mean HDL-C± SD (mmol/L)	1.21 ± 0.61	1.22± 0.62	1.21 ± 0.61	0.89	1.17 ± 0.75	1.22 ± 0.58	0.59
Mean TG± SD (mmol/L)	1.03 ± 0.56	1.03 ± 0.58	1.04 ± 0.55	0.96	0.94 ± 0.51	1.06 ± 0.57	0.16
Mean LDL-C± SD (mmol/L)	2.83 ± 1.23	2.68 ± 1.21	2.90 ± 1.23	0.18	2.88 ± 1.29	2.82 ± 1.22	0.75
Total Cholesterol				0.806			0.710
Normal	176 (66.4)	58 (67.4)	118 (65.9)		35 (68.6)	141 (65.9)	
Elevated	89 (33.6)	28 (32.6)	61 (34.1)		16 (31.4)	73 (34.1)	
HDL-C				0.013			0.222
Normal	119 (44.9)	48 (55.8)	71 (39.7)		19 (37.3)	100 (46.7)	
Low	146 (55.1)	38 (44.2)	108 (60.3)		32 (62.7)	114 (53.3)	
TG				1.000			0.832
Normal	231 (87.2)	75 (87.2)	156 (87.2)		44 (86.3)	187 (87.4)	
Elevated	34 (12.8)	11 (12.8)	23 (12.8)		7 (13.7)	27 (12.6)	
LDL-C				0.921			0.881
Normal	190 (71.7)	62 (72.1)	128 (71.5)		37 (72.5)	153 (71.5)	
Elevated	75 (28.3)	24 (27.9)	51 (28.5)		14 (27.5)	61 (28.5)	
Elevated Blood Sugar	60 (22.6)	18 (20.9)	42 (23.5)	0.645	12 (23.5)	48 (22.4)	0.866
Elevated Blood Pressure	56 (21.1)	23 (26.7)	33 (18.4)	0.121	7 (13.7)	49 (22.9)	0.149
Metabolic syndrome	31 (11.7)	4 (4.7)	27 (15.1)	0.013	5 (9.8)	26 (12.1)	0.640
Normal HDL-C, normal TG	104 (39.2)	43 (50.0)	61 (34.1)	0.013	14 (27.5)	90 (42.1)	0.055
Low HDL-C, normal TG	127 (47.9)	32 (37.2)	95 (53.1)	0.016	30 (58.8)	97 (453)	0.083
Low HDL-C, elevated TG	19 (7.2)	6 (7.0)	13 (7.2)	0.933	2 (3.9)	17 (7.9)	0.317
Normal HDL-C, elevated TG	15 (5.7)	5 (5.8)	10 (5.6)	0.932	5 (9.8)	10 (4.7)	0.154

Key: TC: Total cholesterol; SD: standard deviation; HDL-C: high density lipoprotein - cholesterol; TG: triglyceride; LDL-C: low density lipoprotein-cholesterol.

Table 2: Lipid pattern of the study population

Lipid Pattern	No hypertension or diabetes (n=210)	Hypertension (n=27)	Diabetes (n=24)	Hypertension and diabetes (n=4)	Metabolic syndrome (n=31)
Normal HDL-C, normal TG (%)	82 (39.0)	11 (40.7)	9 (37.5)	2 (50.0)	3 (9.7)
Low HDL-C, normal TG (%)	103 (49.1)	15 (55.6)	7 (29.2)	2 (50.0)	17 (54.8)
Low HDL-C, elevated TG (%)	13 (6.2)	0 (0.0)	6 (25.9)	0 (0.0)	10 (32.3)
Normal HDL-C, elevated TG (%)	12 (5.7)	1 (3.7)	2 (8.3)	0 (0.0)	1 (3.2)

Key: TC: Total cholesterol; HDL-C: high density lipoprotein-cholesterol; TG: triglyceride; LDL-C: low density lipoprotein cholesterol.