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Original Article

Lipid Profile of Type 2 Diabetic and Hypertensive Patients in the Jamaican Population

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ABSTRACT

Aims: Previous studies have shown that diabetes mellitus (DM) increases the risk of cardiovascular diseases in females to a greater extent than in males. In this cross-sectional study, we evaluated the lipid profiles of type 2 diabetic males and females.

Materials and Methods: The study included 107 type 2 diabetic patients (41 males and 66 females), and 122 hypertensive type 2 diabetic patients (39 males and 83 females), aged 15 years and older. Total cholesterol (TC), triglycerides (TG), low density lipoprotein-cholesterol (LDL-C), very low density lipoprotein-cholesterol (VLDL-C) and high density lipoprotein-cholesterol (HDL-C) concentrations were assayed for each group using standard biochemical methods.

Results: The mean TC, TG, VLDL-C, HDL-C and LDL-C concentrations, TG/HDL and LDL/HDL ratios were higher in type 2 diabetic and hypertensive type 2 diabetic patients compared with non-diabetic, and hypertensive non-diabetic control subjects, although these were not significant ($P > 0.05$). Hypertensive type 2 diabetic females had significantly higher serum TC (7.42 ± 1.63 mmol/L) than hypertensive non-diabetic males (5.76±1.57 mmol/L; $P < 0.05$). All the other lipid and lipoprotein parameters except HDL-C were non-significantly higher in females with type 2 DM and those with hypertension and type 2 DM, compared with type 2 diabetic and hypertensive type 2 diabetic males, respectively ($P > 0.05$).

Conclusion: This study demonstrated that dyslipidemia exists in our type 2 diabetic population with greater TC in hypertensive type 2 diabetic females compared with hypertensive type 2 diabetic males. This suggests that hypertensive type 2 diabetic females are exposed more profoundly to risk factors including atherogenic dyslipidemia compared with males.

Keywords: Females, hypertension, lipids, lipoprotein, males, type 2 diabetes mellitus

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INTRODUCTION

Diabetes mellitus (DM) is one of the leading health problems in the Caribbean, contributing significantly to morbidity and mortality and adversely affecting both the quality and length of life.[1] In 2000, diabetes mellitus was the third leading cause of mortality in the Caribbean region, accounting for approximately 10% of all deaths. Those aged 45–64 years were particularly affected.[2] In Jamaica, the prevalence of DM is high. Among the survey participants who were 25–74 years old in the city of Spanish Town, the prevalence was estimated at 13.4%.[3] A third of these participants had DM that had not been detected previously. These estimates were somewhat lower than those from an earlier population survey in Jamaica, in which the overall prevalence was 17.9%, with a 48% frequency of undiagnosed diabetes.[4]

In Caribbean populations, DM often co-exists with obesity, hypertension and dyslipidemia. Dyslipidemia is common in DM, as both insulin deficiency and resistance affects enzymes and pathways of lipid metabolism.[5] Characteristic abnormalities in lipids in type 2 DM include elevated triglyceride (TG) levels, decreased athero-protective high density lipoprotein-cholesterol (HDL-C) levels, and increased levels of small dense low density lipoprotein-cholesterol (LDL-C).[6,7]

Lipid abnormalities are commonly found in persons with DM but there is paucity of recent literature relating to local incidence in Jamaica. In this study, we evaluated the lipid profile of persons with type 2

DM, and those with both hypertension and type 2 DM, in a sample of the Jamaican adult population.

MATERIALS AND METHODS

Sample design

Jamaica has an area of 4411 square miles and is divided into 14 parishes, each of which was visited by the survey team. The districts in each parish were randomly selected. We made a transverse study at The University of the West Indies, with the purpose of comparing the lipid profile of type 2 diabetic and hypertensive type 2 diabetic subjects with their non-diabetic and hypertensive non-diabetic counterparts, respectively, in the Jamaican population. This design was adopted from the Jamaican Labour Force Surveys (LFS) by the Statistical Institute of Jamaica (STATIN),[8] a statutory body in Jamaica with responsibility for census and other official population studies. The design adopted for the LFS was a two-stage stratified sampling design, with the first stage being a selection of areas, Enumeration Districts (ED) of the population census and the second stage being a selection of dwellings. Each dwelling in the sampling universe had an equal probability of being selected for inclusion in the first stage. Informed consent was obtained after the nature of the procedures had been fully explained to participants. At the homes visited, only individuals, 15 years and over, were interviewed and included in the study. A questionnaire was administered by members of the health team to each participant; this included personal, medical and family histories. There were no exclusion criteria, except those patients who did not complete the investigations needed for this study.

All the subjects with a fasting blood glucose (FBG) of 6.1 mmol/L or above were asked to attend a nearby health facility, the following day, after an overnight fast (12–14 hours) and not consuming anything that morning. At the health facility, an abbreviated glucose tolerance test (GTT) was conducted on each subject. A fasting blood glucose (FBG) was done on arrival at the clinic. Then the subjects were asked to take a drink containing 75 g of glucose. Two hours later, a second blood glucose determination was done. An FBG of 6.7 mmol/L or above, or a 2-hour postprandial blood glucose of 11.1 mmol/L or above was viewed as indicative of DM.[9]

Standard blood pressure (systolic and diastolic) measurements were done using the auscultatory technique which involves the use of a mercury sphygmomanometer by a trained health professional.

Biochemical analysis

Biochemical assays on the serum were performed with a multi-channel *Abbott Spectrum autoanalyzer* (*Abbott* Laboratories, *Abbott* Park, Chicago, IL, USA). Parameters that were determined include: FBG, total cholesterol (TC), TG, HDLC, LDL-C and very low density lipoprotein cholesterol (VLDL-C). The FBG concentration for each group was determined by using the Reflolux S type 1172115 glucometers (Boehringer Mannheim, Germany).[10] This is based on the glucose-oxidase method. Glucose oxidase catalyzes the oxidation of glucose to gluconic acid. The generation of hydrogen peroxide is indirectly measured by oxidation of *o*-dianisidine in the presence of peroxidase.[11] TC was determined by an enzymatic method. The cholesterol esters are hydrolyzed to free cholesterol by cholesterol esterase. The free cholesterol is then oxidized by cholesterol oxidase to cholesten-3-one with the simultaneous production of hydrogen peroxide. The hydrogen peroxide produced couples with 4-aminoantipyrine and phenol, in the presence of peroxidase, to yield a chromogen with maximum absorbance at 505 nm.[12] HDL-C was measured by an enzymatic method on the supernatant obtained after selective precipitation of apolipoprotein B-containing lipoproteins with phosphotungstic acid, in the presence of magnesium ions and centrifugation.[13] Serum LDL-C was calculated according to computational procedures of Friedewald *et al*.[14] [LDL = TC − HDL-C − TG/2.2 (mmol/L)]. TG was determined by an analytical method based on the sequence of reaction described by Fossati and colleagues.[15] In this direct colorimetric procedure, serum TGs are hydrolyzed by lipase, and the released glycerol is assayed in a reaction catalyzed by glycerol kinase and L-alpha-glycerol-phosphate oxidase in a system that generates hydrogen peroxide. The hydrogen peroxide is monitored in the presence of horseradish peroxidase with 3,5-dichloro-2-hydroxybenzenesulfonic acid/4-aminophenazone as the chromogenic system. The absorbance of this chromogen system is measured at 510 nm.[15] The methods adopted by the automated instrument for the determination of the above parameters are according to the manufacturer's (Abbott Laboratories, Abbott Park, Chicago, IL, USA).

Statistical analysis

Values for the continuous variables are expressed as mean ± SD. Comparisons of males and females with type 2 DM, and those with hypertension and type 2 DM against their non-diabetic and hypertensive non-diabetic counterparts were performed using unpaired students *t* tests for independent samples; a level of $P < 0.05$ was considered

as statistically significant. *Independent* observations were assumed using the *Fisher* exact *test* and 0.05 was taken to be the cutoff for acceptability of significance levels. The study parameters showed non-Gaussian distribution and statistical significance was assessed by the Mann-Whitney U test.[16] Statistics were computed using SPSS 11.5 (SPSS Inc., Chicago, IL, USA).

RESULTS

In this island-wide study, 2108 subjects were studied: 402 type 2 diabetic (19.1%) and 1706 non-diabetic subjects (80.9%). Of the type 2 diabetes patients, 107 had no other disease. Of these, 66 were females (61.8%) and 41 males (38.2%). One hundred and twenty-two of the type 2 diabetics were hypertensives, of which 83 were females (68.0%) and 39 were males (32.0%). Two hundred and fifty-four of the non-diabetic subjects were hypertensives, of which 193 were females (76.0%) and 61 were males (24.0%) [Table 1].

The mean TC, TG, VLDL-C, LDL-C and HDL concentrations, and TG/HDL and LDL/HDL ratios were higher in type 2 diabetics and hypertensive type 2 diabetic patients, compared with non-diabetic and hypertensive non-diabetic control subjects, respectively, although these were not significant ($P > 0.05$) [Table 2]. The mean LDL-C concentration was higher in the type 2 diabetic females (3.64±1.34 mmol/L) than in non-diabetic females (3.08±1.17 mmol/L), but the difference was also not significant ($P = 0.183$) [Table 3]. The mean TC concentration was higher in hypertensive diabetic females (7.42±1.63 mmol/L) than in hypertensive non-diabetic females (6.03±1.61 mmol/L), but the difference was not significant ($P = 0.087$) [Table 4].

Hypertensive type 2 diabetic females had significantly higher serum concentrations of TC (7.42±1.63 mmol/L) than their male counterparts (5.76±1.57 mmol/L; $P < 0.05$). There was a significant association between blood glucose and LDL-C concentrations in type 2 diabetic subjects ($r = 0.36$; $r^2 = 0.13$; 95% confidence limits = $0.23 < R < 0.47$; $P < 0.05$). In addition, there was significant correlation between blood glucose and TC concentrations in type 2 diabetic subjects ($r = 0.33$; $r^2 = 0.15$; 95% confidence limits = $0.26 < R < 0.49$; $P < 0.05$).

Table 1: Distribution of type 2 DM subjects according to gender

Characteristic	Gender		Total *N* (%)
	Male *N* (%)	Female *N* (%)	
Non-diabetics	413 (35.0)	766 (65.0)	1179 (71.0)
Type 2 diabetics	41 (38.2)	66 (61.8)	107 (6.4)
Hypertensive type 2 diabetics	39 (32.0)	83 (68.0)	122 (7.3)
Hypertensive non-diabetics	61 (24.0)	193 (76.0)	254 (15.3)
Total	554	1108	1662

χ^2 (df = 3) = 6.79, $P = 0.900$

Table 2: Comparison of the mean lipid profile of type 2 diabetic and non-diabetic males

Lipid parameters	Diabetics Mean ± SD	Non-diabetics Mean ± SD	*P value*
Triglyceride	1.47±0.27	1.42±0.87	0.404
LDL	2.89±1.04	2.77±0.84	0.802
VLDL	0.30±0.00	0.28±0.19	0.835
HDL	0.93±0.48	1.04±0.27	0.733
TC	5.10±1.46	4.50±0.73	0.344
LDL/HDL	2.92±2.08	2.57±1.77	0.634
TC/HDL	5.92±2.76	5.17±2.15	0.121

LDL-C (mmol/L), VLDL-C (mmol/L), HDL-C (mmol/L), TC (mmol/L)

Table 3: Comparison of the mean lipid profile of type 2 diabetic and non-diabetic females

Lipid parameters	Diabetics Mean ± SD	Non-diabetics Mean ± SD	*P value*
Triglyceride	1.66±1.88	1.59±1.37	0.061
LDL	3.64±1.34	3.08±1.17	0.183
VLDL	0.32±0.09	0.28±0.31	0.793
HDL	1.02±0.43	1.11±0.26	0.624
TC	5.62±1.78	5.40±1.45	0.913
LDL/HDL	3.80±1.89	3.40±2.25	0.609
TC/HDL	5.73±2.25	5.47±2.56	0.705

LDL-C (mmol/L), VLDL-C (mmol/L), HDL-C (mmol/L), TC (mmol/L)

Table 4: Comparison of the mean lipid profile of hypertensive type 2 diabetic and hypertensive non-diabetic females

Lipid parameters	Hypertensive diabetics Mean ± SD	Hypertensive non-diabetics Mean ± SD	*P value*
Triglyceride	1.68±1.00	1.55±1.42	0.123
LDL	3.36±1.17	3.02±1.18	0.289
VLDL	0.31±0.09	0.30±0.28	0.881
HDL	1.08±0.48	1.16±0.40	0.354
TC	7.42±1.63	6.03±1.61	0.087
LDL/HDL	3.24±1.68	2.90±1.60	0.533
TC/HDL	5.90±3.25	5.30±2.11	0.525

LDL-C (mmol/L), VLDL-C (mmol/L), HDL-C (mmol/L), TC (mmol/L)

DISCUSSION

The study revealed that the lipid and lipoprotein profiles of type 2 diabetic and hypertensive type 2 diabetic patients were not statistically different from those of non-diabetic and hypertensive non-diabetic subjects, respectively. These findings are not in consonance with previous studies which suggest that lipoprotein abnormalities are higher in diabetic than in non-diabetic subjects.[17,18] The results showed gender differences in TC concentrations of hypertensive type 2 diabetic females and hypertensive type 2 diabetic males.

In this study, the mean TC concentrations were 5.10 and 5.62 mmol/L among the type 2 diabetic males and females, respectively. These values are lower than those reported by Khandekar *et al.* (5.52 mmol/L for type 2 diabetic males and 5.97 mmol/L for type 2 diabetic females).[19] Onyemelukwe *et al.* also reported slightly higher values in persons with type 2 DM compared with controls [Table 5].[20]

HDL acts by enhancing the removal of cholesterol from the peripheral tissues and so reduces the body's cholesterol pool. Type 2 DM was usually associated with low plasma levels of HDL-C.[21] There were also lower mean HDL-C concentrations in hypertensive type 2 diabetic males and hypertensive type 2 diabetic females. Low HDL-C concentrations are often accompanied by elevated TG levels as seen in this study and others,[22] and this combination has been strongly associated with an increase in risk of coronary heart disease (CHD).[23-25]

The study indicated a weak, yet positively significant association between elevated blood glucose concentrations and low concentrations of HDL-C. Hyperglycemia progressively increases the transfer of cholesterol esters from HDL-C to VLDL-C particles.[26] The denser LDL particles acquire a large proportion of these HDL esters, further diminishing the HDL-C levels. In addition, HDL-C is a ready substrate for hepatic lipase which converts it into smaller particles that are readily cleared from the plasma.[27] The relative insulin deficiency that occurs in type 2 diabetes impairs the action of lipoprotein lipase and results in lower HDL-C levels and higher TG levels, which may improve with improved glycemic control.[28] Thus, HDL hypocholesterolemia in type 2 diabetes patients is mainly due to insulin resistance-linked lipoprotein lipase deficiency and a reduction in HDL_2 sub-fraction which is secondary to increased HDL-C catabolism.[29]

In Nigeria, hypertension and type 2 DM occur in 10–15% and 2–4% of the population, respectively.[30] Both the conditions coexist frequently, the prevalence of hypertension among the diabetics being 20–40%.[31] They are independent risk factors for dyslipidemia.[32] The UK Prospective Diabetes Study found that mean TC and LDL-C concentrations in those with type 2 DM may not differ significantly from those in non-diabetic subjects.[33] Cook *et al.*,[34] in their report on gender differences in the pattern of dyslipidemia, noted that elevated LDL-C and reduced HDL-C concentrations were more commonly documented in females than in males. In this study, elevated TC, TG, HDL-C and LDL-C concentrations were comparable in type 2 diabetics and non-diabetics, as well as hypertensive type 2 diabetics and hypertensive non-diabetics for both genders. However, TC was significantly higher in hypertensive type 2 diabetic females than in hypertensive type 2 diabetic males. Most of the participants in the study lived in the rural areas in Jamaica. Most of the men were farmers or employed in jobs that required a lot of physical activity. On the other hand, most of the females were housewives and might have been engaged in work that would require less physical activity. This may have accounted for the lower TC in hypertensive type 2 diabetic males compared with that of their female counterparts.

Table 5: Comparison of the mean lipid profile of hypertensive type 2 diabetic and hypertensive non-diabetic males

Lipid parameters	Hypertensive diabetics Mean ± SD	Hypertensive non-diabetics Mean ± SD	*P value*
Triglyceride	1.49±0.44	1.40±0.78	0.956
LDL	2.84±0.74	2.83±1.01	0.943
VLDL	0.30±0.04	0.29±0.19	0.884
HDL	0.90±0.17	1.03±0.45	0.659
TC	5.76±1.57	5.68±1.18	0.911
LDL/HDL	3.41±2.80	3.32±2.93	0.875
TC/HDL	5.47±1.01	4.80±0.56	0.526

LDL-C (mmol/L), VLDL-C (mmol/L), HDL-C (mmol/L), TC (mmol/L)

The TC/HDL-C ratio is a sensitive and specific index of cardiovascular risk.[35] Apart from HDL-C, the ratio of TC/HDL-C is regarded as a predictor of CHD risk, especially with values >6.0.[36] In this study, the mean TC/HDL-C ratios in male and female type 2 diabetics, and hypertensive diabetics were less than 6.0, ranging from a low of 5.47 to a high of 5.93. Onyemelukwe *et al.*[20] reported an atherogenic ratio of 4.4 in type 2 diabetics in Nigeria. The mean ratio considered to be atherogenic in the United States is 5.0 and the values in our diabetic population are above this figure.

In Singapore, fasting serum TG levels, but not HDL-C and LDL-C concentrations, were found to be higher among persons with type 2 DM than those of non-diabetics.[37] In this study, the fasting TG concentrations were higher, although not significant, in type 2 diabetic and hypertensive type 2 diabetic patients, compared with non-diabetic and hypertensive non-diabetic subjects, respectively. The mean TG levels in type 2 diabetic and hypertensive type 2 diabetic patients ranged from 1.57 to 1.68 mmol/L. High TG levels cause increased transfer of cholesteryl esters from HDL-C and LDL-C to very VLDL-C via cholesteryl ester transfer protein, thus forming cholesteryl ester depleted, small dense LDL-C particles.[38] These small dense lipoprotein particles are taken up by arterial wall macrophages, resulting in atherogenesis.[39]

Our study has some limitations. Firstly, this is a cross-sectional design, which reflects only associations between

blood lipids and risk factors. Other variables such as body mass index (BMI) and waist circumference could have been investigated; the studied groups could have been age- and sex-matched. Due to a limited sample size of 224, we cannot rule out that there may be additional gender-related differences that we did not have sufficient statistical power to detect. Despite these limitations, the results of this study give the lipid profile of type 2 diabetics and hypertensive type 2 diabetics in the Jamaican population, and can be used for advanced lipid profile research and intervention. Diabetic educators have the unique opportunity to promote positive health behaviors and improve lipid profiles among these two groups of patients.

CONCLUSION

This study demonstrated that dyslipidemia exists in the type 2 diabetic population in Jamaica with greater TC in hypertensive type 2 diabetic females compared with that of hypertensive type 2 diabetic males. Hypertensive type 2 diabetic females are exposed more profoundly to risk factors including atherogenic dyslipidemia compared with males. Therefore, lipid profiling for all persons with type 2 DM should be a routine test. All persons with type 2 diabetes must be started on primary prevention by encouraging healthy lifestyle diets so as to reduce the risk of CHD and atherosclerosis. Further studies should be undertaken to establish the dietary pattern of type 2 diabetic patients in Jamaica and other factors that may lead to hyperlipidemia.

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