Dyslipidaemia in hypertensive obese type 2 diabetic patients in Jamaica

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Abstract

Introduction: Hypertension and obesity are common problems among diabetic patients accelerating progression of vascular diabetic complications.
Material and methods: A two-stage stratified random sampling design was used, and individuals aged 15 years and over were interviewed. This cross-sectional study evaluated lipid abnormalities of 117 obese type 2 diabetic patients (28 males and 89 females), and 56 hypertensive obese type 2 diabetic patients (22 males and 34 females). 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 using standard biochemical methods.
Results: Hypertensive obese type 2 diabetic females had significantly higher mean serum concentrations of TC ($p = 0.043$), TG ($p = 0.046$), LDL-C ($p = 0.040$), TC/HDL-C ratio ($p = 0.001$) and LDL-C/HDL-C ratio ($p = 0.003$) compared with hypertensive obese non-diabetic females. Similar results were found in hypertensive obese type 2 diabetic males compared with hypertensive obese non-diabetic males. Hypertensive obese type 2 diabetic females had significantly higher serum TC, TG and TC/HDL-C ratio ($p < 0.05$) than hypertensive obese type 2 diabetic males. Hypertensive obese type 2 diabetic females had significantly higher mean serum concentrations of TG ($p = 0.03$) and TC ($p = 0.01$) than obese type 2 diabetic females. There was a significant association between blood glucose and LDL-C concentrations in type 2 diabetic subjects ($r = 0.36$; $p < 0.05$).
Conclusion: Obese hypertensive type 2 diabetic females are exposed more profoundly to risk factors including atherogenic dyslipidaemia compared with males.

Key words: dyslipidaemia, obesity, diabetes, lipids, cholesterol, hypertension, Jamaica.

Introduction

Type 2 diabetes mellitus is one of the most common chronic diseases the world over, and the number of people with diabetes mellitus has risen sharply in recent years [1]. The World Health Organization (WHO) estimated

the worldwide prevalence of diabetes mellitus in adults to be around 173 million in 2002 and predicted that there will be at least 350 mln people with type 2 diabetes by 2030 [2]. The largest share of this increase will occur in developing countries, which is expected to show the same current pattern of concentration of cases in the 45-64-year age group [3]. In the Americas, it is estimated that 34 mln persons have diabetes mellitus [4]. The prevalence of diabetes mellitus is high in Jamaica and the Caribbean and many patients have poor metabolic control [5, 6]. Heart disease is the leading cause of death in persons with diabetes mellitus [7]. Cardiovascular disease in diabetes mellitus is multi-factorial and risk factors include hypertension, dyslipidaemia, insulin resistance and central obesity [8].

Obesity can be described as an imbalance between energy intake and expenditure such that excess energy is stored in fat cells, which enlarge or increase in number. Obesity is defined as a body mass index (BMI) of > 30 kg/m^2, according to WHO criteria [9]. Obesity and overweight are significant public health problems worldwide, affecting an estimated 1 billion persons and contributing to hypertension, type 2 diabetes mellitus, cardiovascular disease, and death [10-12]. Its prevalence in developed countries, such as the United States, is as high as 26.6% in men and 32.2% in women above age 20 years [13].

In the United Kingdom, 12% of adult females are obese and 24% overweight, but the rate of increase is faster [14]. The gradient in obesity in women (Nigeria 5%, urban Cameroon 13%, Jamaica 18%, Barbados 30% and Chicago 36%) was found to be closely correlated with economic development and with the prevalence of hypertension [15]. Ragoobirsingh *et al.* reported that Jamaica has a point prevalence of obesity with truncal 36.2% and gynoid 34.1%, in the 15 and over age group [16]. In Barbados, data show an increase in obesity prevalence across four surveys, which were done in 1969, 1981, 1987, and 1992 [17]. The Wildey population study of Barbadians aged over 40 showed prevalence in women three times that of men, 30% vs. 10% [18].

The Caribbean has demonstrated an increasing prevalence of type 2 diabetes mellitus associated with obesity due to the recent transition to high consumption of energy-dense foods and increasing inactivity [19]. In Jamaica, the adjusted prevalence rates (95% CI) are 9.5% (7.0-12.0) for men and 15.7% (13.2-18.3) for women in a population of over 2.6 m [20]. The characteristic pattern of lipid abnormalities in patients with diabetes mellitus consists of moderate elevation in triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C) concentrations, and an increase in small dense low-density lipoprotein cholesterol (LDL-C) particles [21]. To our knowledge, there has not been any study that has examined lipid abnormalities of obese patients with type 2 diabetes mellitus, and hypertension in Jamaica. This study was designed to investigate the pattern of lipid abnormalities in obese and hypertensive type 2 diabetes mellitus patients in the Jamaican adult population.

Material and methods

Sample design

We carried out a transverse study at the University of the West Indies, with the purpose of comparing the lipid profile of obese type 2 diabetic, and hypertensive obese type 2 diabetic subjects with their obese non-diabetic and hypertensive obese 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) [22], 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. 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. Informed consent was obtained after the nature of the procedures had been fully explained to the participants. Patients who did not complete the investigations needed for this study, and those on statins or fibrates, were excluded from the study. In addition we excluded persons with disorders that predispose to hyperlipidaemia, such as hypothyroidism and nephrotic syndrome.

At the homes of the participants a fasting blood sample was taken. The fasting blood glucose concentration for each group was determined by using Reflolux S type 1 172 115 glucometers (Boehringer Mannheim, Germany) [23]. All subjects with a fasting blood glucose of 6.1 mmol/l or above were asked to attend a nearby health facility, the day after an overnight fast (12-14 hours), without consuming anything that morning. At the health facility, an abbreviated glucose tolerance test (GTT) was conducted on each subject. Fasting blood glucose (FBG) was measured on arrival at the clinic, followed by a drink containing 75 g of glucose. Two hours later a second blood glucose measurement was taken. A fasting blood glucose of 6.7 mmol/l

or above, or a two-hour postprandial blood glucose of 11.1 mmol/l or above, was viewed as indicative of diabetes mellitus [24]. The plasma glucose concentration of the fasting and postprandial blood samples were determined based on the glucose-oxidase method using a multi-channel Abbott Spectrum Autoanalyzer (Abbott Laboratories, Abbott Park, USA). Glucose oxidase catalyses 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 [25]. Obesity was defined as a body mass index (BMI) of > 30 kg/m^2, according to WHO criteria [9]. Blood pressure (systolic and diastolic) measurements were taken using a mercury sphygmomanometer with standard technique by a trained health professional [26]. Patients were allowed to rest for 30 min. All measurements were made in the morning between 9:00 and 10:00 a.m. The mean of two blood pressures was recorded while the patients were seated. Hypertension was defined as a systolic blood pressure reading greater than 140 mmHg and/or a diastolic reading greater than 90 mmHg.

Biochemical analysis

Biochemical assays on the serum were performed with a multi-channel Abbott Spectrum Autoanalyzer (Abbott Laboratories, Abbott Park, USA). Parameters that were determined include: total cholesterol (TC), TG, HDL-C, LDL-C and very-low-density lipoprotein cholesterol (VLDL-C). Total cholesterol 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 [27]. 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 [28]. Triglyceride determination was carried out by an analytical method based on the sequence of reactions described by Fossati *et al.* [29]. In this direct colorimetric procedure, serum triglycerides 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 [29]. The methods adopted by the automated instrument for the determination of the above parameters are according to the instructions of the manufacturer, Abbott Laboratories (Abbott Diagnostics, Illinois, USA). Serum LDL-C was calculated according to computational procedures of Friedewald *et al.* [30] (LDL = TC – HDL-C – TG/2.2 [mmol/l]). Calculation of VLDL-C was 1/5 of TG concentration.

Statistical analysis

Values for the continuous variables are expressed as mean ± SD. Comparisons of males and females with obesity and type 2 diabetes mellitus, and those with hypertension, obesity and type 2 diabetes mellitus, against their obese non-diabetic and hypertensive obese non-diabetic counterparts, respectively, were performed using unpaired Student's t test for independent samples; a level of $p < 0.05$ was considered as statistically significant. Independent observations were assumed using Fisher's exact test and 0.05 was taken to be the cut-off for acceptability of significance levels. The study parameters showed non-Gaussian distribution and statistical significance was assessed by the Mann-Whitney U test [31]. Statistics were computed using SPSS 11.5 (SPSS Inc., Chicago, Illinois, United States).

Results

In the island-wide study, 2108 subjects were studied, 402 type 2 diabetic (19.1%) and 1706 non-diabetic subjects (80.9%). There were 117 obese type 2 diabetics and 56 obese hypertensive type 2 diabetics. Of the non-diabetic subjects, 1179 were non-diabetic without other diseases. There were 156 obese non-diabetics and 117 hypertensive obese non-diabetics (Table I).

Obese type 2 diabetic males had significantly higher serum concentrations of TG than their obese non-diabetic counterparts ($p = 0.039$; Table II).

Table I. Distribution of type 2 diabetes mellitus subjects according to gender

Characteristic	Gender		
	Male *N* (%)	Female *N* (%)	Total *N*
Obese type 2 diabetics	28 (24.0)	89 (76.0)	117
Obese non-diabetics	59 (37.7)	97 (62.3)	156
Hypertensive obese type 2 diabetics	22 (39.6)	34 (60.4)	56
Hypertensive obese non-diabetics	41 (35.2)	76 (64.8)	117
Total	150	296	446

χ^2 *(df = 3) = 6.79, p = 0.005*

Similarly, obese type 2 diabetic females had significantly higher serum concentrations of TG than obese non-diabetic females ($p = 0.019$; Table III).

Hypertensive obese type 2 diabetic males had significantly higher mean serum concentrations of TC ($p = 0.031$), TG ($p = 0.007$), LDL-C ($p = 0.044$) and TC/HDL-C ratio ($p = 0.004$) compared with hypertensive obese non-diabetic males (Table IV). Hypertensive obese type 2 diabetic females had significantly higher mean serum concentrations of TC ($p = 0.043$), TG ($p = 0.046$), LDL-C ($p = 0.04$), TC/HDL-C ratio ($p = 0.001$) and LDL-C/HDL-C ratio ($p = 0.003$) compared with hypertensive obese non-diabetic females (Table V).

Hypertensive obese type 2 diabetic females had significantly higher serum concentrations of TC, TG and TC/HDL-C ($p < 0.05$) than hypertensive obese type 2 diabetic males. Hypertensive obese type 2 diabetic females had significantly higher mean serum concentrations of TG ($p = 0.03$) and TC ($p = 0.01$) than obese type 2 diabetic females.

There was a significant association between blood glucose and LDL-C concentrations in type 2 diabetic subjects ($r = 0.36$; 95% confidence limits = $0.23 < r < 0.47$; $p < 0.05$). In addition, there was a significant correlation between blood glucose and TC concentrations in type 2 diabetic subjects ($r = 0.33$; 95% confidence limits = $0.26 < r < 0.49$; $p < 0.05$).

Table II. Comparison of the lipid profile between obese type 2 diabetic and obese non-diabetic males

Lipid profile [mmol/l]	Obese type 2 diabetic Mean ± SD	Obese non-diabetic Mean ± SD	*p*
Triglyceride	1.65 ±0.61	1.45 ±0.79	0.039
LDL-C	3.23 ±0.96	2.78 ±1.00	0.161
VLDL-C	0.39 ±0.04	0.38 ±0.19	0.984
HDL-C	0.90 ±0.20	1.04 ±0.47	0.361
Total cholesterol	5.44 ±1.48	5.19 ±1.02	0.159
LDL/HDL ratio	3.84 ±0.98	3.03 ±0.67	0.100
TC/HDL ratio	6.83 ±1.73	5.63 ±0.60	0.089

Table III. Comparison of the lipid profile between obese type 2 diabetic and obese non-diabetic females

Lipid profile [mmol/l]	Obese type 2 diabetic Mean ± SD	Obese non-diabetic Mean ± SD	*p*
Triglyceride	1.67 ±0.89	1.51 ±1.20	0.019
LDL-C	3.20 ±1.21	3.10 ±1.18	0.650
VLDL-C	0.43 ±0.30	0.39 ±0.09	0.699
HDL-C	1.07 ±0.65	1.29 ±0.36	0.216
Total cholesterol	5.80 ±1.54	5.68 ±1.68	0.721
LDL/HDL ratio	4.87 ±2.84	3.14 ±1.53	0.060
TC/HDL ratio	7.08 ±4.07	5.75 ±2.40	0.099

Table IV. Comparison of the lipid profile between hypertensive obese type 2 diabetic and hypertensive obese non-diabetic males

Lipid profile [mmol/l]	Hypertensive obese type 2 diabetic Mean ± SD	Hypertensive obese non-diabetic Mean ± SD	*p*
Triglyceride	1.96 ±1.14	1.53 ±1.34	0.007
LDL-C	3.29 ±1.21	2.99 ±1.18	0.044
VLDL-C	0.43 ±0.90	0.35 ±0.07	0.538
HDL-C	1.07 ±0.43	1.08 ±0.43	0.835
Total cholesterol	5.37 ±1.61	4.67 ±1.38	0.031
LDL/HDL ratio	3.29 ±1.51	3.10 ±1.15	0.581
TC/HDL ratio	6.98 ±2.14	5.70 ±0.20	0.004

Table V. Comparison of the lipid profile between hypertensive obese type 2 diabetic and hypertensive obese non-diabetic females

Lipid profile [mmol/l]	Hypertensive obese type 2 diabetic Mean ± SD	Hypertensive obese non-diabetic Mean ± SD	*p*
Triglyceride	2.00 ±0.34	1.76 ±0.43	0.046
LDL-C	3.42 ±1.18	2.87 ±0.92	0.041
VLDL-C	0.50 ±0.00	0.40 ±0.29	0.747
HDL-C	1.02 ±0.37	1.50 ±0.23	0.495
Total cholesterol	8.70 ±1.30	6.12 ±1.38	0.043
LDL/HDL ratio	4.01 ±1.68	3.56 ±1.25	0.003
TC/HDL ratio	8.16 ±2.50	5.90 ±1.62	0.001

Discussion

Diabetes mellitus is often associated with cardiovascular morbidity and this may partly be explained by the abnormal lipid profile which is sometimes a feature of diabetes. We studied the pattern of lipid abnormalities in a population of type 2 diabetic, obese and hypertensive subjects drawn from the 14 parishes in Jamaica. The magnitude of the detected abnormalities showed that TG was the parameter that was most affected as it showed the most significant mean difference between the values of those patients in the obese type 2 diabetic group, and their counterparts in the obese non-diabetic group for both males and females, thus implying a higher cardio-metabolic risk. The hypertriglyceridaemia associated with obesity and insulin resistance in type 2 diabetes was thought to be secondary to the effects of elevated plasma insulin levels causing increased hepatic fatty acid

esterification, which forms triglyceride [32]. It has been proposed that insulin resistance leads to elevated TG levels in obesity through decreased adipose tissue lipoprotein lipase activity [33].

The major risk factors in type 2 diabetes are glycaemic status, dyslipidaemia and hypertension. Type 2 diabetic patients have a high frequency of atherogenic dyslipidaemia especially for TC and LDL-C [34]. The parameters TC, TG, LDL-C and TC/HDL-C ratio showed significant mean differences between values in the hypertensive obese diabetic subjects compared with the hypertensive obese non-diabetics for both males and females. This indicates that the presence of type 2 diabetes is responsible for the more atherogenic pattern. The finding of elevated LDL-C in hypertensive obese type 2 diabetic subjects is of crucial importance as it represents the first alarming risk for cardiovascular diseases. According to the National Cholesterol Education Program (NCEP) guidelines LDL-C is the main target of cardiovascular disease prevention [35]. Elevated cholesterol though not usually regarded as highly predictive of cardiovascular disease was noted in a significant number of the participants in this study, particularly females. The significance of screening for total cholesterol in this study lies in the fact that it could serve as a valuable screening measure for dyslipidaemia.

The significant elevation of TC/HDL-C ratio in hypertensive obese diabetic males and females is a key finding. A study by Kinosian *et al.* showed that the TC/HDL-C ratio is a better predictor of atherosclerosis and cardiovascular disease than any other single lipid [36]. Individuals with increased TC/HDL-C ratio were shown to exhibit resistance to insulin-stimulated disposal and to have higher blood pressure, increased TG concentration and hyperinsulinaemia. Each of these factors being a part of the metabolic syndrome is an independent risk factor for cardiovascular disease [37]. The hypertensive obese diabetic females in this study had the highest TC/HDL-C ratio and mean TG concentration, and are therefore at the greatest risk for cardiovascular disease.

Several large epidemiological and clinical studies have found the LDL-C/HDL-C ratio to be an excellent predictor of coronary heart disease risk and an excellent monitor for the effectiveness of lipid-lowering therapies [38-41]. Therefore it was important to evaluate the LDL-C/HDL-C ratio of the participants in this study. The increased LDL-C/HDL-C ratio observed in the hypertensive obese type 2 diabetic females and males in this study indicates an increased risk of developing coronary heart disease [42]. In the PROSPER trial, a retrospective analysis of 6,000 patients, the ratio of LDL-C/HDL-C was the most powerful measure of cardiovascular disease risk in elderly people [43]. The researchers concluded that changes in LDL-C/HDL-C ratio as a result of statin treatment appeared to account for the beneficial effects of therapy and suggested that statin therapy could usefully be targeted to those with an LDL-C/HDL-C ratio > 3.3 [43]. It is important to note that while the mean LDL-C/HDL-C ratio of the hypertensive obese type 2 diabetic males in this study was 3.29, the value for the females was 4.01. This indicates that the females are at greater risk of developing cardiovascular disease and could benefit more from statin therapy.

Hypertension is one of the most important treatable causes of morbidity and mortality and accounts for a large proportion of cardiovascular diseases in the elderly in Jamaica [44]. The age-and sex-adjusted prevalence in Jamaica is 24% [45], with somewhat higher levels in women than in men. In a paper published from the data in this study, Ragoobirsingh *et al.* reported that Jamaica has a point prevalence of hypertension of 30.8% in the 15-and-over age group. The main risk factors for hypertension are being female, advancing age, obesity, having diabetes and having a family history of hypertension [46]. We report a higher significant mean difference between TC and TG in hypertensive obese type 2 diabetic females compared with obese type 2 diabetic females. The presence of hypertension may be contributory to the greater prevalence of dyslipidaemia observed in the former group. In explaining this scenario, insulin resistance or compensatory hyper-insulinaemia has been associated with hypertension and dyslipidaemia in cross-sectional studies [47, 48]. Insulin resistance often leads to increased intracellular hydrolysis of triglycerides and release of fatty acids into the circulation. The resultant inability of fat cells to store triglyceride is the initial step in the development of dyslipidaemia. Other plausible explanations for the contributory effect of hypertension to abnormal lipid profiles in diabetes mellitus include use of antihypertensive agents [49]. Beta blockers and diuretics, especially thiazide diuretics, have been found to negatively affect the lipid profile and glucose tolerance. Thiazides in high dosage and loop diuretics can increase serum LDL-C, TG, TC/HDL-C ratio and often TC [50]. These blood pressure lowering agents are commonly used for the purpose of blood pressure control in patients with diabetes and hypertension.

Obesity, and especially intra-abdominal fat, is strongly associated with insulin resistance, glucose intolerance, and diabetes mellitus, and is a growing problem in Jamaica [51]. Contributory to this is the westernization of traditional Jamaican diets, with fruits, vegetables, and whole grains being replaced by readily-accessible fast foods, which are high in saturated fat, sugars and refined carbohydrates. In

another paper published from the data in this study, Ragoobirsingh *et al.* reported that Jamaica has a point prevalence of obesity, truncal 36.2% and gynoid 34.1%, in the 15-and-over age group. Both were affected by increasing age, being female, level of education attained and smoking status [52]. Gender was found to be a possible determinant of the pattern of lipid profile levels in participants in the study. In particular, obesity was more prevalent among females [52]. We report a higher significant mean difference between TC, TG and TC/HDL-C ratio in hypertensive obese type 2 diabetic females compared with hypertensive obese type 2 diabetic males, indicating a higher prevalence of lipid abnormalities and greater atherogenic risk. King *et al.* reported that the risk of coronary heart disease in diabetic females is related to age, reproductive and hormonal status as well as HDL-C, TC and TG [53].

A high dietary energy and fat intake is likely to be a major contributing factor to the high prevalence of obesity and evidence of dyslipidaemia in the Jamaican population, particularly those living in urban areas. There is the westernization of traditional Jamaican diets, with fruits, vegetables, and whole grains being replaced by readily-accessible fast foods, which are high in saturated fat, sugars and refined carbohydrates. However, a high dietary fat intake alone cannot account for the extent of the problem. In Jamaica, there is a cultural acceptance of obesity, especially in women [54]. Fewer than half of the subjects recognized poor diet and/or overweight as contributing to their diabetes. Firmly rooted social and economic factors actively encourage over-eating and sedentary behaviour and discourage alterations in these patterns [55]. Physical inactivity among the participants may be a significant contributory factor of their obesity, as a national study recently conducted in Jamaica revealed that only 21.6% of the sample participated in planned exercise [56]. Furthermore, indiscriminate eating patterns could have a direct and unfavourable influence on weight management and glycaemic control.

Despite the importance of these findings of dyslipidaemia and increased atherogenic risk in hypertensive obese type 2 diabetics, and obese type 2 diabetics in a representative sample of the Jamaican population, there are some limitations in the study. We did not acquire any information about the hormonal status of our women subjects. Thus, we do not know whether these women went through earlier menopause and whether this was related to redistribution of fat. Physical activity was not considered and the amount of alcohol and tobacco use was not quantified in our study. These 3 lifestyle factors have been reported to be quantitatively correlated with plasma lipid levels [57, 58]. Also, data on the socioeconomic status of the patients were not available, to check for its influence on the dyslipidaemic state of the participants. The study could have been strengthened by the measurement of anthropometric variables such as higher waist circumference, BMI and waist-to-hip ratio, as these have been found to be significantly associated with dyslipidaemia in males and females.

In conclusions, obesity is a significant problem in the Caribbean countries, including Jamaica, and it is associated with a high prevalence of hypertension, particularly in women. Our study provided evidence for the presence of significant dyslipidaemia in obese type 2 diabetes mellitus and hypertensive obese type 2 diabetes mellitus patients. Future research should focus on intervention strategies aimed at reducing obesity and its morbid sequelae. These strategies should be culturally specific, with a particular focus on children and women, and should include dietary and behavioural aspects, weight reduction, increased physical activity, improved glycaemic control, and increased HDL cholesterol levels, along with reduced LDL cholesterol.

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