

Dyslipidaemia Associated with Type 2 Diabetics with Micro and Macrovascular Complications among Ghanaians

Michael B. Adinortey · Ben E. Gyan · Jonathan Adjimani ·
Philomena Nyarko · Charity Sarpong · Francis Y. Tsikata ·
Alexander K. Nyarko

Received: 11 November 2010 / Accepted: 25 December 2010 / Published online: 12 January 2011
© Association of Clinical Biochemists of India 2011

Abstract In this study, differences in lipid levels amongst diabetics with and without complications were assessed to determine lipid disorders that are associated with diabetic complications other than cardiovascular diseases. A Cross sectional study design was employed. The study included 288 diabetics and 108 non diabetics with different types of complications such as hypertension, nephropathy, neuropathy, and retinopathy. The mean serum total cholesterol was higher in patients with complications compared to those without complications and the non-diabetic controls. The normotensive diabetic patients had the lowest total cholesterol among the diabetic patients' groups (4.65 ± 0.17 mmol/l) compared to the diabetics with hypertension (6.051 ± 0.20 mmol/l), retinopathy (6.26 ± 0.29 mmol/l), neuropathy (5.80 ± 0.17 mmol/l) and nephropathy patients

5.74 ± 0.26 mmol/l ($P < 0.05$). The prevalence of dyslipidaemia among diabetic subjects was between 19.2 and 84.0%. The study shows that, in addition to macrovascular complications, dyslipidaemia is common in type 2 diabetes mellitus patients with microvascular complications.

Keywords Type 2 diabetes mellitus · Ghanaians · Dyslipidaemia · Complications

Introduction

Diabetes mellitus is a heterogeneous metabolic disorder of lipid and carbohydrate metabolism. The condition is characterized by hyperglycaemia resulting from defective insulin secretion, resistance to insulin action or both [1, 2]. The prevalence reported for Ghana in the early 1990 s was 2% [3]. Subsequent studies by Amoah et al. indicate that prevalence rate rose from 0.4% in the 1950 s to 6.3% in 2000 and that type 2 accounted for 85–90% of diabetic cases in the Greater Accra region of Ghana [4]. Poor or inadequate control of persistent hyperglycaemia in diabetes mellitus leads to the development of micro and macro vascular complications often involving organ damage and finally causing death among people with diabetes mellitus [5, 6].

Several studies show that type 2 diabetes mellitus (T2DM) is often associated with lipoprotein disorders [7–11]. In Ghana, a number of studies conducted on lipoprotein disorders indicate that it is associated mostly with diabetic patients who also have hypertension, a macrovascular complication, and not other microvascular complications [12–15]. In Ghana, not much is known about the trends of lipoprotein disorders in type 2 diabetics with microvascular complications. Knowledge about the

M. B. Adinortey (✉)
Department of Biochemistry, School of Biological Sciences,
University of Cape Coast, Cape Coast, Ghana
e-mail: Michaelbuenor@yahoo.com

B. E. Gyan
Immunology Department, Noguchi Memorial Institute
for Medical Research, University of Ghana, Legon, Ghana

J. Adjimani
Department of Biochemistry, University of Ghana,
Legon, Ghana

P. Nyarko
Regional Institute for Population Studies, University of Ghana,
Legon, Ghana

C. Sarpong · F. Y. Tsikata
Tema General Hospital, Ghana Health Service, Tema, Ghana

A. K. Nyarko
Clinical Pathology Department, Noguchi Memorial Institute
for Medical Research, University of Ghana, Legon, Ghana

prevalence would draw specific attention to its management in patients with microvascular complication since the focus has been on macrovascular complications.

Methodology

Informed Consent and Ethical Approval

The Scientific and Technical Committee (STC) and Institutional Review Board (IRB) of the Noguchi Memorial Institute for Medical Research, University of Ghana, examined and approved the research proposal for this study. All participants provided written informed consent on a form approved by the STC and the IRB.

Recruitment of Participants

Sample size was calculated using 95% confidence interval, and a diabetes mellitus prevalence of 6.3% in Ghanaians. A total of 396 participants were recruited into the study comprising type 2 diabetic patients attending the Diabetes Clinic of the Tema General Hospital and healthy non-diabetic controls from the Accra-Tema metropolis of Ghana.

Inclusion Criteria

The study subjects comprised newly and previously diagnosed Ghanaian type 2 diabetes patients with fasting blood glucose (FBG) > 6.4 mmol/l, and normal hepatic function (indicated by normal levels of AST and ALT) and with or without complications of neuropathy, retinopathy, overt nephropathy, coronary artery diseases (hypertension or stroke) and attending the diabetes clinic of the Tema General Hospital, Ghana Health Service in the Greater Accra Region of Ghana. Apparently healthy normotensive non-diabetic controls were also recruited from the same hospital.

Exclusion Criteria

Among the non diabetic control group, those with high blood pressure and also on treatment were excluded from the study to obtain normotensives. Hepatic dysfunction indicated by AST and ALT levels more than twice the upper limit of normal reference values were left out. In addition patients who had deteriorating ailment or who had been severely ill within 3 months prior to the study were also excluded from the study. Similarly patients on thyroid stimulating drugs, corticosteroids lipid-lowering drugs, oral contraceptives, aspirin and sulphonamides were excluded to avoid false elevated HDL-Cholesterol level. Pregnant women were also excluded to avoid a false overt nephropathy.

Clinical Assessment of Subjects

All participants completed a questionnaire approved for the study. Each participant, after granting his or her informed consent, was given a full physical examination by a physician. The clinical history of each patient, disease condition and treatment history as well as demographic data were also recorded. The mean of two blood pressure (BP) readings, measured on the right arm after the participants had been seated for 5 min, was recorded. Diabetes mellitus was diagnosed and classified based on the World Health Organization Criteria [16]. The date of the initial diagnosis of diabetes was defined as the date of the first hospital visit because of diabetes mellitus. Current smoker meant anyone currently smoking tobacco. Dyslipidaemia was defined as serum total cholesterol > 5.2 mmol/l; serum LDL > 2.58 mmol/l; serum triglycerides (TG) > 1.7 mmol/l; and serum HDL < 1.03 mmol/l [17].

Diagnosis of Diabetic Complications

Complications were diagnosed based on laboratory results and full physical examination, treatment history and examination by the study Physician. The definitions adopted in assessing coronary artery disease status and risk were hypertension, systolic BP \geq 140 mmHg and/or diastolic BP \geq 90 mmHg or participant currently receiving treatment for hypertension [18]. Risk of coronary artery diseases was based on HDL-C < 0.75 mmol/l for males, <0.91 mmol/l for females, LDL-C > 3.37 mmol/l for males and females and TC/HDL-C > 4.17. For Neuropathy the definition included experience of 'pins and needles' numbness, loss of sensation, reduced ability to sense touch and vibration, bladder and bowel dysfunction, impotence, diarrhoea and change in blood pressure [19].

Overt nephropathy (proteinuria) was defined and diagnosed based on patient repeatedly having either a urinary albumin excretion rate of > 200 μ g/min or a presence of protein in a reagent strip urinalysis tests [20].

Retinopathy diagnosis was done based on evidence of macular oedema, blot haemorrhages, cotton wool spots, hard exudates, micro-aneurysms and other intraretinal microvascular abnormalities [21].

Biochemical Analysis

Biochemical tests were performed on blood donated by participants after an overnight fast. Fasting blood glucose (FBG), aspartate aminotransferases (AST) and alanine-aminotransferases (ALT) levels and serum lipids-triacylglycerol (TAG) total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) were determined using commercial assay kits (Randox Ltd Antrium UK). The

LDL—cholesterol was calculated using the Friedwald formula [22].

Urinalysis

Early morning urine specimens were collected from all participants into clean labelled plastic containers. Urine protein was determined immediately using urine strips (URO PAPER). The results were reported according to the colour chart provided by the manufacturer.

Anthropometric Measurements

Participants were weighed in light clothing without shoes and their heights were measured with a stadiometer. Body mass index (BMI) was calculated as kilogram per metre square.

Statistical Analysis

Data was analyzed with Pearson chi-square tests for categorical variables to determine group differences. Analysis of variance (ANOVA) was also used to compare multiple means. Data analysis was performed with the statistical package SPSS version 16.0 (SPSS, Chicago, IL) and the level of statistical significance was set at $P < 0.05$.

Results

General Characteristics of the Study Population

A total of 396 individuals gave their written/thumb-printed informed consent to participate in the study. The volunteers comprised 288 T2DM individuals and 108 non-diabetic

normotensive individuals (Table 1). There were significantly more female diabetics than male diabetics and also many more normotensive non-diabetics female volunteers than males. The male diabetics were significantly older than the females ($P < 0.05$). There was no statistical difference between the ages for the male and female normotensive non diabetic volunteers ($P > 0.05$). Table 1 also shows that apart from the female diabetics, majority of participants have had secondary education ($P < 0.05$). Furthermore, compared to the others, relatively higher male non diabetic participants have had tertiary education ($P < 0.05$). Alcohol consumption was found to be relatively higher among the normotensive non-diabetic than the diabetic participants. Alcohol consumption was higher among male diabetic participants compared to female diabetics. Conversely, among the normotensive non-diabetics, more female consumed alcohol compared to males (Table 1). There was no smoker among the female participants. However, there were more smokers (15.22%) among the normotensive non-diabetic male participants than there were among the diabetic males (2.97%) (Table 1). Both systolic and diastolic blood pressures (BP) for participants with diabetes mellitus, was significantly higher than those without diabetes ($P < 0.05$). The BMI for diabetics was significantly higher among diabetics compared to non-diabetics ($P < 0.05$). The female diabetics also had a significantly higher BMI than the males.

Biochemical Parameters for Non-Diabetic and Diabetics

Table 2 shows that the mean TC level for diabetics was significantly higher than for non diabetics ($P < 0.05$). The mean TAG level for diabetics was also significantly higher compared to non diabetics ($P < 0.05$). The highest HDL-C

Table 1 General characteristics of the study population

| Parameter | Non-diabetics | | Diabetics | |
|--------------------|--------------------|-----------------|--------------|---------------------------|
| | Male | Female | Male | Female |
| Number of persons | 46 | 62 ^b | 100 | 188 ^a |
| Age (years) | 52.70 ± 1.49 | 51.27 ± 1.48 | 56.83 ± 0.97 | 53.36 ± 0.74 ^a |
| % Alcohol drinkers | 13.04 | 29.03 | 6.93 | 3.1 ^c |
| % Smokers | 15.22 | – | 2.97 | – |
| Education (%) | | | | |
| Primary | 15.22 | 27.42 | 38.61 | 85.71 |
| Secondary | 65.22 | 64.52 | 51.49 | 11.11 ^c |
| Tertiary | 19.57 ^d | 8.06 | 9.90 | 3.18 |

The values for age are mean ± Standard error of mean (SEM)

Significant difference ($P < 0.05$) between ^a male and female diabetics, ^b non-diabetic males and females, ^c female diabetics and others, ^d male non-diabetics and others, ^e diabetics and non-diabetics

Table 2 Clinical and biochemical indices of normotensive non-diabetics controls and diabetics

| Parameter | Participant | | | | |
|--------------------------|---------------|---------------------------|---------------|----------------------------|-----------------|
| | Non-diabetics | | Diabetics | | |
| | Male | Female | Male | Female | Ref. range |
| BMI (kgm ⁻²) | 23.66 ± 0.28 | 25.07 ± 0.42 ^b | 26.86 ± 0.51 | 28.58 ± 0.40 ^{ac} | 18.8–24.9 |
| Systolic (mmHg) | 118.48 ± 0.82 | 118.00 ± 0.77 | 130.19 ± 1.47 | 128.94 ± 1.00 ^a | ≤120 |
| Diastolic (mmHg) | 77.39 ± 0.66 | 76.77 ± 0.60 | 85.94 ± 1.05 | 84.07 ± 0.62 ^a | ≤80 |
| FBS (mmol/l) | 4.99 ± 0.094 | 5.45 ± 0.22 | 9.65 ± 0.37 | 9.24 ± 0.28 ^a | 4.2–6.4 |
| AST (U/l) | 8.97 ± 0.42 | 9.28 ± 0.32 | 9.39 ± 0.43 | 9.09 ± 0.22 | Up12 |
| ALT (U/l) | 7.56 ± 0.30 | 8.04 ± 0.31 | 8.61 ± 0.42 | 8.09 ± 0.20 | Up12 |
| TC (mmol/l) | 4.37 ± 0.16 | 4.47 ± 0.13 | 5.50 ± 0.51 | 5.52 ± 0.12 ^a | 3.1–6.5 |
| TAG (mmol/l) | 1.08 ± 0.09 | 1.04 ± 0.06 | 1.62 ± 0.10 | 1.71 ± 0.08 ^a | 0.3–1.7 |
| LDL-C (mmol/l) | 2.51 ± 0.18 | 2.53 ± 0.16 | 3.59 ± 0.17 | 3.48 ± 0.13 ^a | <3.88 |
| HDL-C (mmol/l) | 1.37 ± 0.07 | 1.46 ± 0.06 | 1.18 ± 0.06 | 1.26 ± 0.04 ^a | M > 0.9 F > 1.6 |
| TC/HDL-C | 3.58 ± 0.26 | 3.49 ± 0.22 | 5.67 ± 0.28 | 5.63 ± 0.23 ^a | <3.88 |

The values are mean ± Standard error of mean (SEM)

Significant difference ($P < 0.05$) between ^a diabetics and non-diabetics, ^b non-diabetic males and females, ^c male and female diabetics

Table 3 Lipids levels in diabetic with or without complication

| Parameter | Diabetic patients | | | |
|----------------|-----------------------|--------------------------|--------------------|--------------------------|
| | Without complications | | With complications | |
| | Male ($n = 34$) | Female ($n = 49$) | Male ($n = 66$) | Female ($n = 139$) |
| TC (mmol/L) | 4.84 ± 0.20 | 4.46 ± 0.14 | 5.90 ± 0.18 | 5.88 ± 0.13 ^b |
| TAG (mmol/L) | 1.30 ± 0.14 | 1.33 ± 0.11 | 1.77 ± 0.13 | 1.85 ± 0.09 ^b |
| LDL-C (mmol/L) | 2.58 ± 0.26 | 1.92 ± 0.17 ^a | 4.16 ± 0.19 | 4.03 ± 0.14 ^b |
| HDL-C (mmol/L) | 1.66 ± 0.11 | 1.93 ± 0.07 ^a | 0.94 ± 0.03 | 1.01 ± 0.04 ^b |
| TC/HDL-C | 2.92 ± 0.15 | 2.3 ± 0.11 ^a | 6.28 ± 0.11 | 6.70 ± 0.25 ^b |

The values are mean ± Standard error of mean (SEM)

Significant difference ($P < 0.05$) between ^a male and female diabetics without complications, ^b diabetics with complications and without complications

level was recorded for the non diabetic group. The ratio of TC to HDL-C was highest among diabetics ($P < 0.05$). AST and ALT levels for both diabetics and non diabetics were not significantly different.

Lipid Levels in Diabetics with and without Complications

The results in Table 3 show that the mean total cholesterol (TC) and TAG levels for diabetics with complications were significantly higher than for those without complications ($P < 0.05$). Furthermore the mean HDL-C level for diabetics with complications was significantly lower compared to those without complications ($P < 0.05$). The converse was true for LDL-C levels, which was significantly higher for diabetics with complications ($P < 0.05$). The ratio of TC to HDL-C was also significantly higher among diabetics with complications ($P < 0.05$).

Lipid and Blood Pressure Levels of Diabetics with Micro and Macrovascular Complications

Table 4 shows that the mean TC levels for diabetics with macro- and microvascular complications were significantly higher than those without complications ($P < 0.05$). The mean TAG level for diabetics with macrovascular complications was also significantly higher compared to those without complications ($P < 0.05$). The participants with macrovascular complications recorded the highest LDL-C levels among the three groups. In contrast, the highest HDL-C level was recorded for diabetics without complications compared to the other two groups.

The ratio of TC to HDL-C was highest among diabetics with hypertension—a macrovascular complication ($P < 0.05$). Both systolic and diastolic BP for participants with hypertension were significantly higher than those with

Table 4 Lipid and blood pressure profile of diabetics with macrovascular, microvascular and without complications

| Parameter | Complications | | |
|------------------------------|--|---|-----------------------------------|
| | Macrovascular complications (<i>n</i> = 50) | Microvascular complications (<i>n</i> = 155) | No complications (<i>n</i> = 83) |
| Blood Lipid Levels | | | |
| TC (mmol/l) | 6.05 ± 0.20 | 5.83 ± 0.13 | 4.65 ± 0.17 ^a |
| TAG (mmol/l) | 1.85 ± 0.15 ^c | 1.82 ± 0.09 | 1.32 ± 0.13 ^{ab} |
| LDL-C (mmol/l) | 4.29 ± 0.23 | 4.00 ± 0.13 | 2.25 ± 0.22 ^{ab} |
| HDL-C (mmol/l) | 0.93 ± 0.04 | 1.01 ± 0.03 | 1.80 ± 0.09 ^a |
| TC/HDL-C | 7.09 ± 0.34 | 6.64 ± 0.23 | 3.07 ± 0.29 ^{ab} |
| Blood Pressure (mmHg) | | | |
| Systolic | 147.6 ± 1.58 ^d | 127.61 ± 1.00 | 121.88 ± 0.88 ^a |
| Diastolic | 95.4 ± 1.28 ^d | 83.68 ± 0.67 | 80.35 ± 0.59 ^a |

The values are mean ± Standard error of mean (SEM)

Significant difference ($P < 0.05$) between ^a diabetics without complications and others, ^b diabetics with complications and others, ^c no complications and macrovascular complication, ^d macrovascular and others (micro and no complications)

Table 5 Lipid and blood pressure profile of diabetics with retinopathy, neuropathy, nephropathy and hypertension

| Parameter | Retinopathy (<i>n</i> = 25) | Neuropathy (<i>n</i> = 82) | Nephropathy (<i>n</i> = 48) | Hypertension (<i>n</i> = 50) |
|------------------------------|------------------------------|-----------------------------|------------------------------|-------------------------------|
| Blood Lipids Levels | | | | |
| TC (mmol/l) | 6.26 ± 0.29 | 5.80 ± 0.17 | 5.74 ± 0.26 | 6.05 ± 0.20 |
| TAG (mmol/l) | 1.83 ± 0.19 | 1.94 ± 0.12 | 1.47 ± 0.17 | 1.85 ± 0.15 |
| LDL-C (mmol/l) | 4.40 ± 0.33 | 4.00 ± 0.16 | 3.96 ± 0.27 | 4.29 ± 0.23 |
| HDL-C (mmol/l) | 1.02 ± 0.09 | 0.96 ± 0.04 | 1.11 ± 0.07 | 0.93 ± 0.04 |
| TC/HDL-C (mmol/l) | 6.13 ± 0.19 | 6.043 ± 0.11 | 5.17 ± 0.17 | 6.51 ± 0.12 |
| Blood Pressure (mmHg) | | | | |
| Systolic | 128.0 ± 1.97 | 123.0 ± 0.84 | 133.33 ± 2.20 | 147.0 ± 1.58 ^a |
| Diastolic | 82.4 ± 1.19 | 81.22 ± 0.51 | 87.29 ± 1.45 | 95.44 ± 1.28 ^a |

The values are mean ± Standard error of mean (SEM)

^a Significant difference ($P < 0.05$) between hypertension and other complications

other complications and those without any complication ($P < 0.05$) Table 4.

Table 5 shows that the mean TC and LDL-C levels were relatively higher among diabetics with retinopathy compared to other complications ($P > 0.05$). TC/HDL-C ratio was observed to be highest in diabetic hypertensive. HDL-cholesterol was found to be relatively low in diabetic hypertensives. The blood pressure was significantly higher among hypertensives ($P < 0.05$). Diabetics with nephropathy recorded a significantly lower TAG levels ($P < 0.05$) compared to those with other complications.

Distribution of Lipid Disorders in the T2DM Patients with or without Complications

Table 6 compares the percentage frequency distribution of LDL-cholesterol, total cholesterol, triacylglycerol and

HDL-cholesterol in diabetes patients using ATP (Adult Treatment Panel) III classification. Among 288 T2DM patients, 28% had neuropathy, both nephropathy and hypertension registered 17% and diabetics with retinopathy constituted 9% whereas the remaining 29% had no complications.

In this study, using the ATP III cut-off marks, the prevalence of dyslipidaemias- (hypercholesterolemia, hypertriglyceridaemia, high LDL-cholesterol) was lower in diabetic patients without complications (19.2–26.6%), compared to those with complications (29.2–84%). Hypercholesterolemia in various complications was: 62.6% for nephropathy, 84% in retinopathy, 68.3% in neuropathy patients and 76% among those with hypertension. Hypertriglyceridaemia which was also found in all complications was highest (37.7%) in patients with diabetic neuropathy and lowest (18.8%) in those with nephropathy.

Table 6 Comparison of prevalence of low density lipoprotein-cholesterol, total cholesterol, high density lipoprotein-cholesterol and triglyceride disorders in T2DM with and without complications using ATP III classification

| ATP III Classification | T2DM Complications (%) | | | | |
|--|------------------------|----------------|----------------|----------------|----------------|
| | NU (n = 82) | NP (n = 48) | HP (n = 50) | RT (n = 25) | NC (n = 83) |
| LDL-cholesterol (mmol/l) | | | | | |
| Optimal value < 2.6 | 19.5 | 33.3 | 16.0 | 20.0 | 73.5 |
| Optimal to near optimal 2.6–3.3 | 13.4 | 4.2 | 6.0 | 0.0 | 7.2 |
| Border line high risk 3.4–4.1 | 12.2 | 10.4 | 18.0 | 12.0 | 7.3 |
| High risk 4.2–4.9 | 29.3 | 16.7 | 30.0 | 24.0 | 4.8 |
| Very high risk \geq 5.0 | 25.6 | 35.4 | 30.0 | 44.0 | 7.2 |
| Total cholesterol (mmol/L) | | | | | |
| Desirable value < 5.2 | 31.7 | 37.5 | 24.0 | 16.0 | 75.9 |
| Border line high risk 5.2–6.2 | 24.4 | 18.8 | 30.0 | 24.0 | 15.7 |
| High risk \geq 6.3 | 43.9 | 43.7 | 46.0 | 60.0 | 8.4 |
| HDL-cholesterol (mmol/L) | | | | | |
| Men higher risk < 1.03 | 29.5 | 27.1 | 28.0 | 24.0 | 9.6 |
| Women higher risk < 1.3 | 58.3 | 47.9 | 58.0 | 56.0 | 9.6 |
| Men normal 1.03–1.3 | 1.2 | 0.0 | 6.0 | 0.0 | 8.5 |
| Women normal 1.3–1.6 | 3.7 | 6.3 | 4.0 | 8.0 | 3.6 |
| All sexes protected \geq 1.6 against heart disease | 7.3 | 18.7 | 4.0 | 12.0 | 68.7 |
| Triglycerides (mmol/l) | | | | | |
| Normal < 1.7 | 45.1 | 70.8 | 46.0 | 56.0 | 73.5 |
| Border line to high risk 1.7–2.3 | 17.1 | 10.4 | 20.0 | 8.0 | 13.3 |
| High risk 2.4–5.6 | 37.8 | 16.7 | 34.0 | 36.0 | 13.3 |
| Very high risk \geq 5.7 | 0.0 | 2.1 | 0.0 | 0.0 | 0.0 |

NU neuropathy, NP nephropathy, HP hypertension, RT retinopathy, NC diabetics without any complication, ATP III adult treatment panel III

The prevalence of hypertriglyceridaemia in diabetics without complications was 13.3% (Table 6). High LDL-cholesterol disorders were recorded in the prevalence range of 62.5–80% in all complications with the least in nephropathy and the highest in retinopathy patients.

Discussion

It is well established that inadequate management and/or control of hyperglycaemia predisposes diabetic patients to a number of complications. The aim of our study was to determine the prevalence of dyslipidaemia (hypercholesterolemia, hypertriglyceridaemia and high LDL-cholesterol disorders) in diabetic patients visiting the Tema general hospital in Accra, Ghana using the revised National Cholesterol Education Program-Adult Treatment Panel (NCEP-ATP) III criteria. These criteria are used for the evaluation and treatment of lipid disorders [23]. We also investigated if any differences existed in lipid levels among diabetics with and without complications. The study showed that the major complications found in the subjects were hypertension, nephropathy, neuropathy and retinopathy. These findings are consistent with reports from other studies [12, 21, 24, 25]. The mean age of T2DM patients in this study was 55.1 ± 0.86 years and the mean duration was about

4 years. There appears to be no sex predilection for type 2 DM in this study which is similar to observations made by Fabian et al. and Vinter–Repalust et al. [25, 26]. The duration of diabetes is known to be related to the development of complications [24]. However, in this study no such association was found. The reason for this is not apparent. Meanwhile, it is reasonable to attribute it to delay in diagnoses. There was no significant difference in FBG levels between diabetics with complications and those without complications. This may be attributed to the fact that, both groups (diabetics with or without complications) were on oral hypoglycaemic agents such as daonil and metformin. The higher BMI recorded for diabetic patients in this study are consistent with previous reports [13, 15].

Dyslipidaemia is identified as a major risk factor that contributes significantly to the development of macrovascular and microvascular complications in T2DM patients. Patients with T2DM often present with adverse lipoprotein disorders [27]. In fact, hyperlipidaemia (high LDL-C, TC) is known to increase the risk for macrovascular diseases such as arterial hypertension [28]. It is believed to hasten the process of arteriosclerosis especially in the presence of hypertension and diabetes mellitus [29] since patients with hypertension and diabetes mellitus have a higher risk of developing coronary artery disease (CAD) [30]. The high TC, LDL-C, TAG levels and low HDL-C levels

recorded for subjects with hypertension as a complication compared to those without complications was similar to trends reported previously [14]. Other studies have recorded lower lipid levels in Ghanaian diabetics than the levels observed in this study [15, 31, 32]. The difference could be attributed to the sampling time for the various studies since some studies have demonstrated seasonal variation in cholesterol levels [33]. The LDL-C level recorded among Ghanaians is lower than Caucasian levels [17]. The population differences have previously been observed by Nyarko et al. [15]. In this study, the high TC/HDL-C ratio in the diabetics with complications compared to those without is consistent with known trends that high TC/HDL ratio indicates risk for CAD while a low TC/HDL-C indicates a low risk for CAD [34].

Our study revealed combined hyperlipidaemia, hypercholesterolaemia and hypertriglyceridaemia among type 2 diabetics. The cause of the lipid alteration among T2DM subjects has been attributed to differential insulin distribution which leads to increased very low density lipoprotein cholesterol (VLDL-C) and triacylglycerol production through hepatic hyperinsulinaemia accompanied by decreased catabolism of triacylglycerol-rich lipoprotein as a result of relative peripheral insulin deficiency [35]. Using the ATP III cut-off for hypercholesterolemia, prevalence in nephropathy, retinopathy, neuropathy, and hypertension patients are; 62.6, 84, 68.3, and 76%, respectively. Among those without any complications, 24.1% had hypercholesterolemia. Apart from the slightly higher hypertriglyceridaemia, the findings in this study compares favourably with other studies [8, 9]. A low-density lipoprotein cholesterol level was reported to be normal in diabetics by Ruderman and Haudenschild [36]. This is contrary to our findings of elevated LDL cholesterol levels in Ghanaian diabetic patients in this study, especially among the patients with diabetic retinopathy and hypertension. The observation that hypertriglyceridaemia among the diabetics with complications was highest in patients with diabetic neuropathy suggests hypertriglyceridaemia induced neuropathy as postulated by Hou et al. [37]. This implies that sustained lowering of triglyceride levels could delay the progression of neuropathy, as reported by Davis et al. [38] but would be unlikely to completely reverse it.

In this study, the prevalence range of dyslipidaemia was observed to be low in diabetics without complications (19.2–26.6%) was observed but high in patients with complications (29.2–84%). The high proportion of diabetic subjects (19.2–84.0%) with lipid abnormalities in this study is similar to what Emile et al. [39] found among their T2DM patients. The dyslipidaemia seen in the T2DM patients with hypertension could be due the effects of treatment. In other studies, treatment of hypertension with β -blockers, as well as high doses of thiazide diuretics have

been shown to exacerbate the dyslipidaemia in patients with hypertension and diabetes mellitus [40].

Conclusion

The study revealed that, dyslipidaemia is common in T2DM patients in Ghana with both micro and macrovascular complications. It further reveals disorders of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride, in patients with complicated type 2 diabetes mellitus.

References

- Gavin JR III, Alberti KGMM, Davidson MB, De Fronzo RA, Drash A, Gabbe SG, et al. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*. 1997;20:1183–97.
- Nair M. Diabetes mellitus, part 1: physiology and complications. *BJN*. 2007;16(3):184–8.
- Gyesie. A Living a healthy and normal life with diabetes. In: Health care tit-bits. Accra: Health Care Services Limited; 1992.
- Amoah AG, Owusu SK, Adjei S. Diabetes in Ghana: a community based prevalence study in Greater Accra. *Diab Res Clin Pract*. 2002;56(3):197–205.
- Morrish NJ, Wang SL, Stevens LK, Fuller JH, Keen H. Mortality and causes of death in WHO multinational study of vascular diseases in diabetes. *Diabetologia*. 2001;44(2):14–21.
- Gupta PB, Bhatt P, Thakker K. Vascular complications of type 2 diabetes mellitus. *Guj Med J*. 2002;59:9–12.
- Joslin EP. Arteriosclerosis and diabetes. *Ann Chim Med*. 1927;5: 1061–79.
- Abdul RAN, Olufunsho F. Hyperlipidaemia among Saudi diabetic patients—pattern and clinical characteristics. *Ann Saudi Med*. 1995;15(3):240–3.
- Akbar DH. Hyperlipidaemia in diabetic patients in Saudi Arabia. *Diabetes Int*. 2001;11(1):17–8.
- Agrawal R, Sharma P, Pal M, Kochar A, Kochar D. Magnitude of dyslipidemia and its association with micro and macro vascular complications in type 2 diabetes: a hospital based study from Bikaner (Northwest India). *Diabetes Res Clin Pract*. 2006;73(2): 211–4.
- Idogun ES, Unuigbo EI, Ogunro PS, Akinola OT, Famodu AA. Assessment of serum lipids in Nigerians with type 2 diabetes mellitus complications. *Pak J Med Sci*. 2007;23(5):708–12.
- Adufour KOM, Ofei F, Mensah Adufour J, Owusu SK. Diabetes in Ghana: a morbidity and mortality analysis. *Int Diab Dig*. 1993;4(3):90–2.
- Nyarko A, Adufour K, Ofei F, Kpodonu J, Owusu S. Serum lipid and lipoprotein levels in Ghanaians with diabetes mellitus and hypertension. *J Natl Med Assoc*. 1997;89:191–6.
- Eghan BA Jr, Acheampong JW. Dyslipidemia in outpatients at general hospital in Kumasi, Ghana: cross-sectional study. *Croat Med J*. 2003;44(5):576–8.
- Nyarko A, Asiedu-Larbi J, Ofosuene M, Asare-Anane H, Addy ME. Serum lipids and antioxidants in Ghanaian diabetic, hypertensive and healthy subjects. *Ghana Med J*. 2003;37(2):72–82.
- WHO. Diabetes mellitus. Technical Report Series 1985;727.
- National Cholesterol Education Program (NCEP). Expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486–97.

18. Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The sixth Report of the joint National Committee on prevention, detection, evaluation and treatment of high blood pressure. *Arch Intern Med.* 1997;157:2413–46.
19. Vijian S, Stevens DL, Herman WH, Funnel MM, Standiford CJ. Screening, prevention, counselling and treatment for the complications of type II diabetes. *J Gen Intern Med.* 1997;12:567–80.
20. Klein R, Klein BEK, Moss SE, DeMets DL. Proteinuria in diabetes. *Arch Intern Med.* 1988;148:181–6.
21. Robert N, Frank MD. Diabetic retinopathy. *N Engl J Med.* 2004;350:48–58.
22. Friedwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultra centrifuge. *Clin Chem.* 1972;18:499–502.
23. Talbert RL. Role of the national cholesterol education program adult treatment panel III guidelines in managing dyslipidemia. *Am J Health Syst Pharm.* 2003;60(2):3–8.
24. Davis MD. Diabetic retinopathy: a clinical overview. *Diabetes Care.* 1992;15:1844.
25. Fabian W, Majkowska L, Stefanski A, Moleda P. Prevalence of diabetes, antidiabetic treatment and chronic diabetic complications reported by the general practitioners. *Przegl Lek.* 2005;62(4):201–5.
26. Vinter–Repalust N, Jurkomo L, Katie M, Simunovic R, Petric D. Disease duration, patient compliance and presence of complications in diabetic patients. *Acta Med Croatica.* 2007;61(1):57–62.
27. Taskinem MR. Quantitative and qualitative lipoprotein abnormalities in diabetes mellitus. *Diabetes.* 1992;41:12–7.
28. De Fronzo RA, Ferrannini A. Multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerotic cardiovascular disease. *Diabetes Care.* 1991;14:173.
29. Sznajderman M. Hypertension and lipids. *Blood Press Suppl.* 1996;1:14–7.
30. Arauz-Pacheco C, Parrott MA, Raskin P. The treatment of hypertension in adult patients with diabetes. *Diabetes Care.* 2002;25:134–47.
31. Swanikar GR. Biochemical normals in Ghanaians. *Ghana Med J.* 1971;10:81–5.
32. Asibey-Berko E, Avorkliyah VM. Serum cholesterol levels of male blood donors at Korle-bu teaching hospital. *Ghana Med J.* 1999;33:104–7.
33. Dobson HM, Muir MM, Hume R. The effects of ascorbic acid on the seasonal variations in serum cholesterol levels. *Scott Med J.* 1984;29(3):176–82.
34. Kumar A, Sivakanesan R. Serum lipid profile abnormality in predicting the risk of myocardial infarction in elderly normolipidaemic patients in South Asia: a case-controlled study. *Internet J Altern Med.* 2009;6:2.
35. Reaven GM. Non insulin-dependent diabetes mellitus, abnormal lipoprotein metabolism and atherosclerosis. *Metabolism.* 1987;36:1–8.
36. Ruderman NB, Haudenschild C. Diabetes as an atherogenic factor. *Prog Cardiovas Dis.* 1984;26:373–412.
37. Hou R, Goldberg AC, Tobin GT. A case of severe neuropathy associated with hypertriglyceridemia. *Endocr Pract.* 2008;14(8):1020–2.
38. Davis TM, Yeap B, Bruce DG, Davis WA. Lipid-lowering therapy protects against peripheral sensory neuropathy in type 2 diabetes. Presented at American Diabetes Association 67th Scientific Sessions; June 22–26, 2007. Chicago, IL. Abstract.
39. Werk EE Jr, Gonzalez JJ, Ranney JE. Lipid level differences and hypertension effect in blacks and whites with type II diabetes. *Ethn Dis.* 1993;3(3):242–9.
40. Andrew JK, Clifford JB. Type 2 diabetes. London: Royal Society of Medicine Press; 1994. p. 107.