Patterns of lipid changes among type 2 diabetes patients in Sudan

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أنماط التغيُّرات الشحمية بين المرضى بالسكَّري من النمط الثاني في السودان

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الخلاصة: أجريت هذه الدراسة، في السودان، لتقصي أنماط ومحددات الاضطرابات الشحمية في مجموعة شملت 250 من المرضى البالغين بالسكَّري من النمط الثاني مع شواهدهم الأصحّاء في مجموعات من الفئة العمرية التي بلغت 60 عاماً وتنوَّعت بين الذكور والإناث. ولقد حصل الدارسون على معطيات سكانية وسريرية للمرضى، إضافةً إلى قيامهم بقياس مَنْسَب كتلة الجسم ومحيط الخصر، وعينات من الدم على الريق لتقدير الغلوكوز ومُرْتَسَم الشحميات. وكان متوسط المدة الزمنية للإصابة بالسكَّري 9.4 عاماً، مع ضعف التحكم في مستويات السكر لدى 46.0٪ من المرضى. وبمقارنة النتائج مع الشواهد، وجد الدارسون أن هنالك زيادة يُعْتَدُّ بها إحصائياً في مستوى الغليسيريدات الثلاثية، وانخفاضاً في مستويات كولسترول البروتين الشحمي المرتفع الكثافة. كما أظهرت سائر قيم الشحميات زيادة طفيفة مقارنة بالشواهد، كما تبيَّن وجود ترابُطاً يُعْتَدُّ بها إحصائياً بين الغليسيريدات الثلاثية وبين كولسترول البروتين الشحمي المرتفع الكثافة وبين زيادة العمر، والجنس المؤنث، والسمنة، والنشاط البدني، وضعف التحكم في مستويات السكر لدى السكريِّين.

ABSTRACT This study investigated the patterns and determinants of lipid disorders among a group of 250 type 2 adult diabetic patients and 60 age- and sex-matched healthy controls in Sudan. Demographic and clinical data, body mass index and waist circumference, and fasting blood samples were taken to estimate the glucose and lipid profile. The mean duration of diabetes was 9.4 years and 46.0% of patients had poor glycaemic control. Compared to controls we detected a statistically significant increase in triglycerides level and a decrease in HDL-C levels. Other lipid values showed a slight increase compared to controls. There was a statistically significant association of triglycerides and HDL-C with increasing age, female sex, obesity, physical inactivity and poor glycaemic control of diabetes.

Caractéristiques des anomalies lipidiques chez des patients diabétiques de type 2 au Soudan
RÉSUMÉ Cette étude a porté sur les caractéristiques et les déterminants des troubles lipidiques dans un groupe de 250 adultes atteints de diabète de type 2 et dans un groupe témoin de 60 sujets sains du même âge et du même sexe au Soudan. Les données démographiques et cliniques, l'indice de masse corporelle et le tour de taille des patients ont été établis, et des prélèvements de sang à jeun ont été réalisés afin d'évaluer leur profil glycémique et lipidique. L'ancienneté moyenne du diabète était de 9,4 ans et chez 46,0 % des patients, la glycémie n'était pas bien contrôlée. La comparaison avec les témoins nous a permis de détecter une augmentation statistiquement significative du taux de triglycérides et une diminution des taux de cholestérol HDL (C-HDL). Les autres valeurs lipidiques étaient légèrement augmentées par rapport aux témoins. On observait une association statistiquement significative entre les triglycérides et le C-HDL d'une part, et l'augmentation de l'âge, le sexe féminin, l'obésité, la sédentarité et le mauvais contrôle de la glycémie d'autre part.

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Introduction

There are 2 major types of lipids in the blood: cholesterol and triglycerides. They are carried on 4 types of lipoproteins: chylomicrons, low-density lipoproteins (LDL), very-low-density lipoproteins (VLDL) and high-density lipoproteins (HDL). The chylomicrons are raised only after eating a fat-containing meal. The primary function of lipid particles is that of fat transport: after feeding, to the liver and adipose storage areas, or during fasting, when lipids are recalled from depots to peripheral tissues for energy requirements. Cholesterol is an essential component in cell membranes and steroid hormones.

Nowadays, the term "dyslipidaemia" is increasingly being used to describe abnormal changes in lipid profile, replacing the old term "hyperlipidaemia". Dyslipidaemia encompasses changes in HDL-cholesterol (HDL-C), the size and density of LDL-cholesterol (LDL-C) and the total cholesterol/HDL-C ratio (TC/HDL-C) [*1*]. The term diabetic dyslipidaemia comprises a triad of raised triglycerides, reduced HDL-C and excess of small, dense LDL particles [*2,3*]. The lipid abnormalities are prevalent in diabetes mellitus because insulin resistance or deficiency affects key enzymes and pathways in lipid metabolism [*1*]. In particular, the following processes are affected: apoprotein production, regulation of lipoprotein lipase, action of cholesteryl ester, transfer proteins and hepatic and peripheral actions of insulin [*1*]. Even more, it has been proposed that the composition of lipid particles in diabetic dyslipidaemia is more atherogenic than other types of dyslipidaemia. This means that even normal lipid concentrations might be more atherogenic in diabetic than in nondiabetic people [*4*].

Only in recent years have researchers in the Eastern Mediterranean Region and Africa started to study lipid changes among diabetic and nondiabetic people. In a Saudi study comprising 1155 diabetic type 2 patients, dyslipidaemia was reported among 46% (second only to hypertension) [*5*]. Al Muhtaseb's study of Arab women indicated an incidence of 50% of lipid disorders among type 2 diabetic patients [*6*]. The only Sudanese study indicated a high incidence of lipid changes among diabetic patients that are comparable to the regional literature [*7*].

Nowadays, in Sudan and elsewhere in Africa, diabetes is no longer a "rare" or "Western" disease and deserves some of the attention and resources that are now diverted to communicable diseases. The causal association between atherosclerosis and dyslipidaemia is well established. In diabetes the associated hyperglycaemia, obesity and insulin changes highly accelerate the progression to atherosclerosis [*2*]. Atherosclerosis accounts for up to 80% of deaths in diabetic patients due to coronary heart disease (CHD) and cerbrovascular or peripheral vascular disease [*2,3*].

There is a relative lack of local and regional literature on the problem of diabetic dyslipidaemia. By studying diabetic dyslipidaemia among a group of our patients we hoped to draw attention of the medical community to the occurrence and magnitude of this problem in Sudan. In particular, the macrovascular complications of diabetes are becoming increasingly apparent among our hospital patients in Omdurman. We believed that our study would add a badly needed body of information on lipid changes among our diabetic population, bearing in mind that the only similar study was done 13 years ago [*7*]. Thus future research on this subject will refer not only to literature derived from patients living in industrialized environments largely different from ours. We aimed to investigate the patterns

and determinants of lipid disorders among type 2 diabetic patients. In particular, we investigated the association of lipid disorders with age, sex, weight, physical activity and duration of and glycaemic control of diabetes.

Methods

Sample and setting

Our study was carried out at a public medical clinic for chronic diseases (run by the second author) in Omdurman, Sudan, in the period September to October 2004. Our clinic is part of a public multi-specialty clinic comprising medical and surgical referral clinics, serving patients under the umbrella of the Sudan Insurance Authority. Most of our patients have diabetes or hypertension or both. The diabetic patients registered at the clinic come for monthly follow-up visits for assessment of glycaemic control and treatment of any intercurrent illness. The aims, nature and procedures of the study were fully explained to the potential study population and a clear verbal consent was required. The potential population of our study was all the adult patients with type 2 diabetes attending our clinic. We excluded patients with factors that might affect blood lipids, such as renal or hepatic disease, alcohol intake and the use of drugs such as thiazide diuretics and oral contraceptives.

Data collection

The study had 3 steps of data collection: a questionnaire; measurements of blood pressure, height, weight and waist circumference; and collection of blood samples to estimate blood lipids and glucose.

Questionnaire

The data required from our patients included age, sex, duration of diabetes (< and ≥ 10 years), types of antidiabetic treatment and degree of physical activity. We graded physical activity according to the number of episodes of exercise undertaken per week. The patients were categorized as active (≥ 3 times per week) or inactive (< 3 times per week), according to the recommendations of the American Heart Association [*8*].

Measurements

Blood pressure was measured using a mercury sphygmomanometer with the subject sitting down after a 5-minute rest. The patients were classified as having high blood pressure if they had systolic blood pressure > 160 mmHg and diastolic blood pressure > 95 mmHg, or if they were already receiving antihypertension drug therapy.

Body mass index (BMI) was based on the formula of weight (in kilograms) divided by the square height (in metres). The results of this formula classified our patients as underweight (BMI < 18 kg/m^2), normal (BMI 18–24.9 kg/m^2), overweight (BMI 25–29.9 kg/m^2) and obese (BMI > 30 kg/m^2). The weight was measured in kilograms, with participants wearing clothes but no shoes. A lever balance scale was used and was zeroed immediately before each session and was regularly checked using a known fixed weight. The same scale was used to weigh all the study participants. The height of the participants without shoes was measured to the nearest centimetre. The participants were asked to stand upright with their back against the stand, heels together and eyes directed forward so that the top of the tragus of the ears was horizontal with the inferior orbital margin and the measuring plate was lowered onto the scalp to give the correct level. We used the same instrument for all the patients.

Waist circumference was measured with the patient standing up, and the waist was

measured at the level of the umbilicus as the smallest girth between the lower margin of the ribs and the iliac crests.

Blood samples were taken from each patient after they had been fasting for 10–12 hours.

The control group consisted of 60 age- and sex-matched healthy subjects (recruited from the medical school staff and students), who were not suffering from an acute illness or a metabolic disease that affected blood lipids and had no family history of hypercholesterolaemia. We measured BMI and lipid profile for all controls.

Laboratory data

Blood samples were collected by venipuncture in EDTA-coated tubes and then centrifuged for plasma immediately. The blood glucose level was measured immediately.

The remaining plasma was stored at 4 ºC for further analysis for total cholesterol, triglycerides, HDL-C and LDL-C using commercial kits (Spinreact, Spain). VLDL-C was calculated according to the formula: VLDL-C = TC – (LDL-C + HDL-C).

The TC/HDL-C ratio was calculated by dividing the 2 means of each group. The measurement of chylomicrons was not included in the study as they rise only shortly after eating a fat-containing meal and are then normally completely metabolized. Abnormal lipid values were defined as: total cholesterol > 6 mmol/L, triglycerides > 1.6 mmol/L, HDL-C < 1.05 mmol/L, LDL-C > 3.8 mmol/L,VLDL-C > 1.05 mmol/L and TC/HDL-C ratio > 6.

Patients with fasting blood glucose < 8 mmol/L were classified as having fair glycaemic control and ≥ 8 mmol/L as poor glycaemic control.

Statistical analysis

The data were presented as mean and standard deviation (SD), and were analysed using *SPSS*, version 10. The test for significance was Student's *t*-test. A *P*-value of 0.05 or less was considered statistically significant.

Results

Our study included 250 patients (95 male and 155 female), mean age 52 (SD 13) years (range 31–80 years). The female patients were slightly younger than the male patients: mean age 51 (SD 11) years versus 54 (SD 12) years. According to the BMI criteria, 112 patients (44.8%) were normal weight, 74 (29.6%) were overweight and 64 (25.6%) were obese, with an overall mean BMI of 28.7 (SD 6.4) kg/m^2. The female patients were more overweight than the male patients with a mean BMI of 29.6 (SD 3.0) kg/m^2 versus 27.7 (SD 2.5) kg/m^2. According to the waist circumference, 21 patients (8.4%) had abdominal obesity. Only 66 of our patients (26.4%) were physically active and 184 were inactive (73.6%). Female patients were less active than male patients.

The mean duration of diabetes among our patients was 9.4 (SD 5.3) years (range 1–33 years). The female patients had earlier onset of diabetes than the male patients: mean duration 9.5 (SD 5.2) years versus 8.9 (SD 5.3) years. Of our patients 24 (9.6%) were on no treatment, 32 (12.0%) were using diet control, 146 (58.4%) were taking oral hypoglycaemic agents and 48 (19.2%) were on insulin. Glycaemic control of diabetes was classified as fair for 135 patients (54.0%) and poor for 115 (46.0%). The female patients had worse control than males (49.0% versus 41.0% had poor control). The mean systolic blood pressure was 140 (SD 19) mmHg and mean diastolic blood pressure was 84 (SD 10) mmHg. According to the criteria adopted, 74 patients (29.6%) were hypertensive and 175 (70.4%) normotensive.

Table 1 summarizes the characteristics of patients (n = 250) and controls (n = 60). The control group had lower BMI and blood pressure than the patients, and were more physically active. The difference in systolic and diastolic pressure was statistically significant ($P < 0.05$).

The mean value of blood lipids among the patients and controls are shown in Table 2. The levels of total cholesterol, triglycerides, LDL-C and VLDL-C were higher among patients than controls. The difference for triglycerides was statistically significant ($P < 0.05$). The HDL-C was significantly lower in patients ($P < 0.05$).

The distribution of abnormal lipid values among individual patients showed some variations. Abnormal total cholesterol was detected in 74 patients (29.6%), raised triglycerides in 122 (48.8%), low HDL-C in 65 (26.0%), high LDL-C in 38 (15.2%), high VLDL-C in 78 (14.0%) and TC/ HDL-C ratio > 6 in 35 patients (14%). According to these figures almost half the patients had some lipid abnormality, i.e. diabetic dyslipidaemia. Among the controls, raised total cholesterol was recorded in 8 patients (13.3%), raised triglycerides in 9 (15%), low HDL-C in 4 (6.6%), high LDL-C in 8 (13.3%), high VLDL-C in 6 (10.0%). The TC/HDL-C ratio was > 6 in 3 patients.

Table 3 shows the distribution of mean lipid values by 10-year age groups among the diabetic patients. The levels of total cholesterol showed a progressive increase with age up to the age of 60 years. The difference in the values of total cholesterol, triglycerides and HDL-C between the youngest and oldest age groups were statistically significant ($P < 0.05$).

The association between the mean lipid values among our patients and their sex, diabetes duration, glycaemic control, physical activity, BMI and waist circumference is shown in Table 4. With the exception of duration of diabetes, the mean lipid values showed clear differences. Females, the obese, the physically inactive and the poorly controlled patients showed higher levels of total cholesterol, triglycerides, LDL-C, VLDL-C and TC/HDL-C ratio, and lower levels of HDL-C. Among these groups the differences in triglycerides and HDL-C were statistically significant ($P < 0.05$), but not for other parameters.

Discussion

Our study in Sudan indicated that nearly half our diabetic patients had some disorder in their lipid profile. This figure is to

Table 1 **Demographic and clinical characteristics of diabetic patients and healthy controls**

Characteristic	Patients (n = 250)	Controls (n = 60)
Mean (SD) age (years)	53 (11)	52 (10)
Male/female (No.)	95/155	22/38
Mean (SD) body mass index (kg/m^2)	28.7 (6.0)	26.0 (4.4)
Mean (SD) waist circumference (cm)	96 (8.4)	93 (8.5)
Mean (SD) systolic blood pressure (mmHg)	140 (19)	127 (12)
Mean (SD) diastolic blood pressure (mmHg)	84 (10)	72 (9)
Physically active/inactive (No.)	66/184	21/39

n ***= number of patients; SD = standard deviation.***

Table 2 **Distribution of mean lipid values among diabetic patients and healthy controls**

Variable	Mean (SD) values		*P*-value
	Patients (*n* = 250)	Controls (*n* = 60)	
Total cholesterol (mmol/L)	5.69 (0.28)	5.20 (0.15)	NS
Triglycerides (mmol/L)	1.71 (0.27)	1.16 (0.21)	< 0.05
HDL-C (mmol/L)	0.93 (0.15)	1.11 (0.18)	< 0.05
LDL-C (mmol/L)	3.68 (0.21)	3.45 (0.14)	NS
VLDL-C (mmol/L)	1.01 (0.26)	0.81 (0.11)	NS
TC/HDL-C	6.11 (1.86)	4.69 (0.13)	NS

n ***= number of patients; SD = standard deviation; NS = non-significant. HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; VLDL-C = very-low-density lipoprotein cholesterol; TC/HDL-C = total cholesterol/HDL-C ratio.***

some extent lower than that reported in international studies. For example, 70% of the Americans and up to 85% of Finnish diabetic patients were reported to have lipid abnormalities [*2,9*]. The Canadian Heart Association reported that up to half the diabetic patients had low HDL-C [*2*]. The difference in dietary habits and climate are claimed to justify the difference in lipid profile between our study and international studies. The findings of other regional studies are somewhat similar to our study. Nigerian and Kuwaiti studies show an incidence of lipid disorders of around 50% among diabetic patients [*10,11*].

In accordance with other studies, the most common recognized abnormality was hypertriglyceridaemia (which was reported in nearly half the patients). Some studies on triglyceride levels showed a significant

Table 3 **Distribution of mean lipid values by age group among 250 diabetic patients**

Variable	Mean (SD) values			
	31–40 years (*n* = 25)	41–50 years (*n* = 82)	51–60 years (*n* = 60)	71–80 years (*n* = 15)
Total cholesterol (mmol/L)*	5.53 (0.21)	5.68 (0.23)	5.81 (0.25)	5.78 (0.52)
Triglycerides (mmol/L)*	1.66 (0.11)	1.69 (0.25)	1.74 (0.34)	1.90 (0.18)
HDL-C (mmol/L)*	1.13 (0.23)	1.01 (0.12)	0.88 (0.17)	0.86 (0.27)
LDL-C (mmol/L)	3.43 (0.25)	3.65 (0.29)	3.81 (0.12)	3.75 (0.17)
VLDL-C (mmol/L)	0.82 (0.19)	0.94 (0.22)	1.09 (0.31)	0.96 (0.14)
TC/HDL-C (mmol/L)	4.89 (0.17)	5.60 (0.90)	6.63 (0.14)	6.72 (0.19)

n ***= number of patients; SD = standard deviation.***
P < 0.05.
HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; VLDL-C = very-low-density lipoprotein cholesterol; TC/HDL-C = total cholesterol/HDL-C ratio.

Table 4 **Association of some patients' characteristics to the mean values of their blood lipids**

Characteristic	Mean (SD) values					
	Total cholesterol (mmol/L)	Triglycerides (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	VLDL-C (mmol/L)	TC/HDL-C (mmol/L)
Sex						
Male*	5.51 (0.62)	1.21 (0.33)*	0.88 (0.14)*	3.52 (0.39	0.98 (0.15)	6.26 (0.44)
Female	5.90 (0.73)	1.81 (0.25)	0.81 (0.41)	3.75 (0.27)	1.15 (0.32)	7.28 (0.27)
Diabetes duration						
< 10 years	5.71 (0.34)	1.68 (0.10)	0.88 (0.11)	3.71 (0.45)	0.98 (0.19)	6.48 (0.31)
≥ 10 years	5.66 (0.38)	1.70 (0.21)	0.92 (0.12)	3.66 (0.27)	1.02 (0.13)	6.15 (0.19)
Glycaemic control*						
Fair	5.52 (0.13)	1.59 (0.26)*	1.01 (0.19)*	3.53 (0.12)	0.93 (0.15)	5.52 (0.13)
Poor	5.87 (0.34)	2.11 (0.15)	0.81 (0.11)	3.79 (0.22)	1.11 (0.21)	7.24 (0.30)
Physical activity						
Active	5.59 (0.12)	1.53 (0.25)*	1.04 (0.34)*	3.61 (0.29)	0.98 (0.33)	5.37 (0.35)
Inactive	5.87 (0.69)	1.69 (0.69)	0.86 (0.26)	3.71 (0.28)	1.11 (0.15)	6.82 (2.65)
Body mass index*						
Normal	5.31 (0.13)	1.52 (0.53)*	1.11 (0.20)*	3.53 (0.41)	0.89 (0.61)	4.78 (0.06)
Overweight	5.53 (0.27)	1.81 (0.45)	0.99 (0.31)	3.71 (0.53)	0.94 (0.44)	5.58 (0.87)
Obese	5.98 (0.73)	1.92 (0.21)	0.83 (0.15)	3.84 (0.31)	1.11 (0.35)	7.20 (0.48)
Waist circumference*						
No abdominal obesity	5.61 (0.23)	1.62 (0.12)*	0.99 (0.21)*	3.51 (0.19)	0.95 (0.17)	5.66 (1.09)
Abdominal obesity	6.21 (0.32)	1.95 (0.20)	0.78 (0.18)	3.89 (0.25)	1.21 (0.05)	7.96 (1.77)

SD = standard deviation.

****P < 0.05.***

HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; VLDL-C = very-low-density lipoprotein cholesterol; TC/HDL-C = total cholesterol/HDL-C ratio.

sex-dependant pattern, with male diabetic patients having higher levels than female patients. This might be related to excessive smoking or alcohol consumption. This factor was not applicable in our study as we excluded smokers and alcohol consumers. Instead, the levels of triglycerides were remarkably higher among female patients. The possible explanation for this is the dominance of overweight and obesity (both generalized and abdominal) among our female patients. It has been found that hypertriglyceridaemia is reported 4 times more in overweight and obese patients [*12*]. Some researchers associate the high triglyceride level to the poor glycaemic control of diabetes [*13*]. This hypothesis is supported by the reduction of the triglyceride level with the improvement of glycaemic control (in the absence of weight gain) [*13*]. The increase in triglycerides in poorly controlled patients was related to the decrease of activities of adipose tissue and muscle lipoprotein lipase activity [*12*]. In contrast to triglycerides and HDL-C levels, the LDL-C level was not significantly different between patients with poor and fair control, in accordance with other studies [*2*].

Our study showed lower HDL-C levels compared with controls. The importance

of this parameter stems from the growing evidence that reduced HDL-C is a powerful predicator for premature CHD [*14*]. Our study indicated a positive association between poor glycaemic control and the low level of HDL-C. Hyperglycaemia progressively increases the transfer of cholesterol esters from HDL-C to VLDL-C particles [*1*]. The denser LDL particles acquire a large proportion of these HDL esters, further diminishing the HDL-C level. In addition, HDL-C is a ready substrate for hepatic lipase which converts it into smaller particles, which are readily cleared from the plasma [*15*]. As with the triglycerides, improvement in glycaemic control leads to an increase in the levels of HDL-C, supporting the evidence for a role for poor glycaemia in decreasing the level of this lipoprotein. Apart from HDL-C, the ratio of total cholesterol to HDL-C is regarded as a predictor of CHD risk, especially with values > 6 [*16*].

As in other studies, the level of VLDL-C was lower than controls [*1*]. Poor insulinization results in increased lipolysis in adipocytes. The resulting increase in fatty acid transport to the liver, which is a common abnormality in type 2 diabetes, may cause an increase in VLDL-C. Insulin directly degrades the apo B (which is the major protein of VLDL particles) [*17*] and thus insulin may increase secretion of apo B (and then VLDL).

A brief glance at our study sample indicates that the female sex was dominant. Our clinic was held in the morning, a time that women (who were mostly housewives) prefer to attend. In accordance with some studies, our study showed that dyslipidaemia occurs more in diabetic women [*12*]. Two previous studies reported higher cholesterol in Arab women [*6,7*]. Erasmus et al. reported a high level of triglycerides and low HDL-C in diabetic women in Nigeria [*18*]. Our findings might be surprising to some clinicians, who tend to request more lipid profile tests and prescribe more lipid-lowering drugs to their male patients compared to women [*19*]. Our study indicates a positive association between age and lipid abnormalities, with higher levels among older than younger ages. This fact, to some extent, may hold true for nondiabetic subjects. For example, total cholesterol and LDL-C may increase with age in those with otherwise good health. Some studies indicate that the level of triglycerides decreases after the sixth decade, stating reasons such as lower food intake and absorption and increased rate of catabolism [*20*]. The age factor may show a different pattern when lipid disorders first appear in childhood or early adulthood. Here, lipid disorders do not remain constant over time. Such patients may have normal lipid values later in life [*1*].

The association between weight gain and lipid disorders is beyond doubt, to the extent that even a slight reduction by 5%–10% of weight may positively influence lipid values in diabetic patients [*21*]. Using BMI or waist circumference as indices of weight, our study patients were predominantly above the normal weight. The obese patients had higher levels of triglycerides and lower levels of HDL-C. In fact our patients showed a gradual increase in the levels of cholesterol and triglycerides from normal to overweight to obesity BMI values. The effects of obesity on lipid metabolism are mediated by insulin resistance, resulting in higher levels of triglycerides, and low HDL-C [*22*]. A proportion of our patients had abdominal obesity and showed more marked prevalence of abnormal lipid values than those with generalized obesity. Abdominal obesity is associated with qualitative and quantitative lipid changes [*23,24*]. Such changes include, for example,

overproduction of VLDL-C and production of smaller and denser LDL particles. Some researchers even suggest that measuring waist circumference might be the "cheapest and easiest" method to determine the likelihood of a patient having small, dense LDL particles [*21*].

The results of our study indicate a clear association between glycaemic control of diabetes and appearance of dyslipidaemia. The positive improvement in lipid profile with fair glycaemic control is evident from many studies [*25*]. The epidemiological analysis of the United Kingdom Prospective Diabetes Study data has shown that a continuous relationship exists between the risks of microvascular complications and degree of glycaemic control [*25*]. The association of hyperglycaemia and hypertriglyceridaemia can be explained by the decrease in the adipose tissues and muscle lipoprotein lipase activity [*26*]. Hyperglycaemia (together with increased free fatty acids) tends to increase the hepatic secretion of VLDL-C [*26*]. In uncontrolled diabetic patients, it has been reported that the activity of lipoprotein lipase and hence clearance of VLDL-C in the circulation is diminished due to insulin resistance [*27*]. The level of total cholesterol is usually normal or near normal if glycaemic control is adequate, and worsening of control raises the level [*16*].

The increase of lipid abnormalities with age in our study may erroneously raise a suspicion of a possible association with duration of diabetes. But looking at those with < and ≥ 10 years duration of diabetes, the distribution of lipid values was similar between the 2 groups. Furthermore, some researchers detected lipid abnormalities in prediabetic subjects with insulin resistance (but normal plasma glucose) [*28*]. This finding might suggest a role for abnormalities of insulin action, independent of blood glucose level [*28*]. In support of this hypothesis, an oral hypoglycaemic agent called thiazoladinedione, which improves insulin actions on peripheral tissues, highly improves the lipid profile (in contrast to other hypoglycaemic drugs) [*29*]. We rather advocate the view that there is a dissociation between duration of diabetes and dyslipidaemia. In fact the occurrence of dyslipidaemia depends on factors such as insulin action on peripheral tissues and liver, apoprotein production and regulation of lipoprotein lipase [*1*]. The duration of diabetes seems to play only a minor role in modifying these factors. In addition, clinicians tend to neglect looking for other secondary causes of dyslipidaemia, such as renal disease, alcohol consumption and certain drugs. These factors may accelerate the progression of dyslipidaemia at any stage of diabetes (i.e. irrespective of duration).

Our study clearly shows the dominance of physical inactivity among our study population and the increased prevalence of lipid disorders. The difference in weight is only a partial explanation, as a proportion of inactive patients maintain a normal weight. Some studies indicate that physical inactivity *per se* may initiate some lipid changes, in particular reduced HDL-C levels [*30*].

Conclusion

Our study showed a high prevalence of lipid disorders among diabetic patients in Sudan. There was a positive association between lipid disorders and age, sex, weight, physical activity and glycaemic control. Thus lipid disorders occurred more among the aged, the female, the obese, the less active and the poorly-controlled diabetic patients. The high prevalence of lipid disorders in our study suggests that they might be playing a major role in the development of atherosclerosis in Sudanese patients. The optimal care of diabetic patients should include pe-

riodic screening for lipid abnormalities. Aggressive lifestyle changes, such as weight reduction and physical exercise should be initiated first. Addition of lipid-lowering drugs should be considered if such changes do not achieve effective lipid control. The optimum treatment with antidiabetic drugs to obtain fair glycaemic control should go hand-in-hand with lipid-lowering drugs. Further research on lipid disorders in diabetic patients are recommended to study type 1 patients, children and adolescents with type 2 diabetes and prediabetic subjects.

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