Assessment of Lipid Profiles, Liver Function, Kidney Markers, and Antioxidant Levels in Diabetic Patients at Ahmadu Bello University Teaching Hospital and Specialist Hospital in Zaria, Kaduna State

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Abstract

Background: Diabetes mellitus is a persistent metabolic-condition characterized by an increase level of blood glucose due to the abnormalities in insulin secretion by the β-cell of the pancreas and or resistance toward the exploit of hormone. Insulin linked with disturbances in the carbohydrates, proteins and lipids metabolism which leads to long term complications.

Methods: A total of 240 equal proportions of male and female subjects were involved in this study. Face to face interview was used to collect the data and other possible clinical information associated with diabetes mellitus. Fasting venous blood specimens were collected to assess serum lipid profiles, liver function, kidney indices and antioxidant status. Weight and height of the participants were measured.

Results: The result shows lipid abnormalities of significant increase in the levels of LDL, TC, TG with significant decrease in the level of HDL, liver profile abnormalities of significant increase in the level of AST, ALT, and ALP followed by significant increase in the level of total protein and a significant decrease in the level of DB, TB and Albumin, kidney abnormalities of significant increase in the levels of urea, creatinine, chloride and significant decrease in the level of sodium and bicarbonate, antioxidant enzymes of significant decrease in the level of SOD and Catalase and significant decrease in the level of lipid peroxidation product (MDA) according to age groups and gender.

Conclusion: The complications of diabetes mellitus were high in the patients attending Ahmadu Bello University Teaching Hospital (ABUTH) and Specialist Hospital Zaria, among middle aged and elderly with Type II Diabetes Mellitus patients. Gender, age, higher body mass index (BMI), lipid profile abnormalities, kidney function abnormalities, liver profile abnormalities and decrease in antioxidant markers were the risk factors associated with diabetes mellitus that may lead to cardiovascular diseases, diabetic nephropathy, NAFLD and the generation of free radicals beyond the scavenging abilities of endogenous antioxidant defense, that can result in oxidative stress related diabetes, which can increase morbidity and mortality in diabetes mellitus.

Keywords: Diabetes Mellitus; Lipid Profiles; Liver Function; Kidney Indices and Antioxidant Status

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Introduction

Diabetes mellitus which is a metabolic disorder with a combination of hereditary and environmental that results in abnormally high blood sugar levels and it is the third leading cause of death after heart disease and cancer in many developed countries, affecting about 2 - 3% of general population [13]. These days, humans are suffering not only on the disease itself but also from the diabetes-related complications. These complications are significantly important because of their effects on the functions of several organs in the body [19].

People around the globe are being affected by Diabetes mellitus which poses major public health concern and socioeconomic challenges [12]. According to a declaration made in 2010 by the former United Nations Secretary-General Ban Ki-moon, he described diabetes and other non-communicable diseases (NCDs) as "a public health emergency in slow motion". This is because they now present a greater threat than infectious diseases such as HIV/AIDS, malaria, and tuberculosis [14]. Globally, diabetes as a chronic metabolic disorder of multiple etiologies is assuming epidemic proportions [30] with an estimated 415 million adults affected in the world [20] and 14.2 million adults aged 20 - 79 years have diabetes in the African region. There are more than 1.56 million cases of diabetes in Nigeria, and by 2040 this figure will be more than double [20]. While the rates of both type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus are growing, T2DM has a disproportionately greater contribution to the rising prevalence of DM globally compared to T1DM [5]. One consequence of the growing rates of DM is a considerable economic burden both for the patient and the healthcare system. The economic burden associated with DM is substantial both in terms of the direct costs of medical care as well as indirect costs of diminished productivity tied to diabetes related morbidity and mortality [4].

Diabetes mellitus occurs throughout the world but is more common (especially type 2) in more developing countries. The greatest increase in rates has however been seen in low- and middle-income countries, [45] where more than 80% of diabetic deaths occur [28]. The fastest prevalence increase is expected to occur in Asia and Africa, where most people with diabetes will probably live in 2030 [27].

The pathogenesis of type I and type II diabetes mellitus are different, but hyperglycemia and it is associated complications are common in all diabetic conditions [10]. However, epidemiological studies and clinical trials strongly held the notion that hyperglycemia is the principal cause of the complications. Effective blood glucose control is the key for preventing or reversing diabetes complication and improving quality life in patient with diabetes. Thus, sustained reduction in hyperglycemia will decrease the risk of developing microvascular and macrovascular complications [18]. Insulin and oral hypoglycaemic agents (sulphonylureas, biguanides and thiazolidinedione) and α-glucosidase inhibitors are the main agents for the treatment of diabetes and are effective in controlling hyperglycaemia [10]. However, the practical applicability (administration) of these therapeutic agents' remains restricted owing to their limited action, secondary failure rates and accompanying side effects [24]. Diabetes is also managed or controlled by non-pharmacologic methods, such as diet and exercise. Despite the great strides made in the understanding and management of diabetes, the disease and disease related complications remain unabated [10].

Aim of the Study

The study was aim to assess the lipid profiles, liver function, kidney markers, and antioxidant levels in diabetic patients attending Ahmadu Bello University Teaching Hospital and Specialist Hospital in Zaria, Kaduna State.

Materials and Methods

Materials

Equipment

Equipment used included the following: Centrifuge 5480 (Eppendorf), Spectrophotometer (300) SP, Water bath, Grant instrument ltd, Cambridge, England.

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Human subject

Blood samples were collected from diabetic patients in Ahmadu Bello University Teaching Hospital (ABUTH) and Specialist hospital Zaria.

Chemicals

Sodium potassium tartrate, potassium iodide, succinic acid, bromocresol green, sodium azide, Brij 35, mercuric nitrate $[Hg(NO_3)_2]$, nitric acid (HNO_3), diphenylcarbazone, ethanol, sodium chloride (NaCl), hydrogen chloride (HCl), sodium hydroxide (NaOH) trichloroacetic acid, thiobarbituric acid (TBA), methionine, nitro-blue tetrazoline (NBT), EDTA, hydrogen peroxide (H_2O_2).

Methods

Physical examination

The weight and height of each study subject were measured by using analog digital scale without shoes. The height was measured by instructing each subject's feet pointed outward; legs straight and knee together; arms at sides; head, shoulder blades, buttocks, and heels touching measurement surface; looking straight ahead; and shoulder relaxed. The body mass index (BMI) was calculated by using the formula, weight over height square, and the results were recorded.

Collection and storage of samples

Exactly 5ml blood was collected from each study subject by a trained medical laboratory technologist after overnight fasting following the standard operating procedure guideline. The collected blood specimen was kept at room temperature for ~30 minutes for clot formation. After clot formation, the blood was centrifuged at 3000 rpm for 5 minutes by using centrifuge. Then, the serum was separated from the whole blood and stored at −20°C before analysis. All the samples for the analysis were analyzed at Ahmadu Bello University Teaching Hospital Zaria, Kaduna State.

Experimental design

Exactly 240 participants were used for the analysis (200 diabetic and 40 control participants) and were divided based on their age and gender.

Age groups (years)	Male	Female
Control	20	20
20 - 29	20	20
30 - 39	20	20
40 - 49	20	20
50 - 59	20	20
60 and above	20	20

Table

Determination of biochemical parameters

Serum total cholesterol (TC) was determined by enzymatic method [48], serum triglyceride was determined by the method of [52], determination of serum HDL-C was done by enzymatic method of [52], determination of serum LDL-C was calculated using Friedewald formula [49], serum alanine aminotransferase and serum aspartate aminotransferase were determined based on the method explained

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by [50], serum alkaline phosphatase was determined according to the method of [52], serum total bilirubin determination was done by the method of [51], determination of serum total protein was using biuret method, determination of serum albumin by the method of bromo cresol green (BCG), determination of serum urea was done using Urease method [53], determination of serum creatinine by the method of [24], determination of serum sodium and potassium was done by the method explained by [57] and determination of serum chlorides and bicarbonate were using [57], determination of catalase activity was done by [56], determination of lipid peroxidation product as evidenced by the formation of thiobarbituric acid reactive substances (TBARS) by the method of [57], serum superoxide dismutase (SOD) activity was determined by the method of [58].

Statistical analysis

Statistical analysis was done using SPSS software (version 25.0 for windows; SPSS, Chicago, IL, USA). Descriptive data were expressed as a Mean ± Standard Error of Mean (SEM) and analyzed using one way analysis of variance (ANOVA) followed by post Hoc LSD test (fisher 1935). Categorical variables were expressed as numbers or percentage and analyzed using the chi-square test. Multivariate logistic regression models were used to identify the determinant factors associated with diabetes complications. $P < 0.05$ was considered as statistically significant.

Results

Age and gender of the participants

Out of the 240 individuals included in the study, 200 were diagnosed with diabetes while 40 were healthy control subjects. A noteworthy distinction in age was observed between male and female participants, with average ages of 43.52 ± 14.99 and 42.84 ± 14.19 years, respectively.

Age and gender of diabetic patients

Table 1 shows the age and gender of diabetic patients attending Ahmadu Bello University Teaching Hospital and Specialist Hospital Zaria, Kaduna State. Age >20 years.

Age (years)/Gender	20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> years
Male	26.80 ± 9	41.25 ±	45.52 ± 5	52.60 ±	66.65 ± 7
Female	25.96 ± 7	54.05 ±	48.30 ±	51.95 ± 1	61.65 ±

Table 1: Age and gender of diabetic patients.

Values were expressed as mean ± SEM; n = 20, Control mean age 32.44 ± 1.57.

BMI values for diabetic patients

Body mass index (BMI) of diabetic patients was highest in diabetic patients than in the normal control participants, compared by sex; the BMI was highest in males (Table 2). Table 2 shows the BMI of diabetic subjects in different age groups for the patients attending ABUTH and Specialist Hospital Zaria age > 20 years. Body mass index (BMI) was calculated as the ratio of weight to height squared (kg/m^2). Subjects with a BMI ≥ 18 kg/m^2 and < 25 kg/m^2 were classified as normal, a BMI ≥ 25 kg/m^2 and < 28 kg/m^2 were classified as overweight and those with BMI ≥ 28 kg/m^2 were classified as obese [45] standards).

Lipid profile of diabetic patients

Table 3 shows the result of serum triglycerides, total cholesterol, high density lipoprotein (HDL), low density lipoproteins (LDL) of diabetic subjects. The result indicated a significant increase (P < 0.01) of the serum triglycerides in the age groups 40 - 49, 50 - 59 and 60

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BMI	20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above years
Male	25.13 ± 0	26.17 ± 8	27.45 ± 13	29.15 ± 38	31.54 ±
Female	23.08 ± 56	24.25 ± 1	21.47 ± 5	26.88 ± 15	28.62 ± 1.00

Table 2: *BMI values for diabetic patients.*

Values are expressed as mean ± SEM; n = 20, Control Body Mass Index (BMI) value 20.23 ± 0.63.

above years as compared with the normal control group and was higher in males than in females. In the same way, there was no significant difference (P < 0.05) of the serum level of triglycerides in the age groups 20 - 29, 30 - 39, years as compared with the control group.

There was a significant increase (p < 0.01) of the serum total cholesterol level in the age groups 40 - 49, 50 - 59 and 60 > above years but it was (P < 0.05) of the serum total cholesterol in the age groups 30 - 39 years and was higher in females than in males as compared with the control groups. However, there was no significant difference (P < 0.05) of the serum level of total cholesterol in the age groups 20 - 29 years as compared with the control group.

There was a significant decrease (P < 0.01) of the serum level of HDL in the age groups 40 - 49, 50 - 59, and 60> above years as compared with the control group, but was higher in females than in males and there was no significant difference (P < 0.05) of the serum level of HDL in the age groups 20-29, and 30-39 years as compared with the control group.

The result also shows that, there was a significant increase (P < 0.01) of the serum level of LDL in the age groups 40 - 49, 50 - 59 and 60 and above years as compared with the control group and was higher in males than in females. In the same way, there was no significant difference (P < 0.05) of the serum LDL level in the age groups 20 - 29 and 30 - 39 years as compared with the control group.

Lipid Profile		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60 > above years
TG (mg/dl)	Male	137.74 ± 3.73	142.45 ± 4.30	290.15 ± 7.56**	531.88 ± 8.54**	871.51 ± 9.85**
	Female	121.17 ± 5.03	133.11 ± 7.29	289.93 ± 5.72**	534.36 ± 7.89**	863.04 ± 11.32**
TC (mg/dl)	Male	138.44 ± 7.74	147.81 ± 10.17*	302.19 ± 8.51**	447.69 ± 7.68**	591.75 ± 7.37**
	Female	153.91 ± 8.33	164.16 ± 9.88*	300.70 ± 4.68**	463.13 ± 7.03**	585.29 ± 7.11**
HDL (mg/dl)	Male	57.74 ± 1.49	50.22 ± 2.71	26.82 ± 1.17**	23.0 ± 0.61**	15.5 ± 0.77**
	Female	58.32 ± 1.71	51.33 ± 2.96	30.58 ± 1.03**	24.56 ± 1.01**	16.03 ± 0.94**
LDL (mg/dl)	Male	55.30 ± 5.77	62.32 ± 10.48	217.55 ± 9.12**	318.53 ± 8.15**	402.29 ± 7.88**
	Female	47.25 ± 2.19	52.49 ± 2.50	202.31 ± 6.58**	308.83 ± 6.52**	397.32 ± 6.54**

Table 3: *Serum lipid profiles of diabetic patients.*

Value are expressed as a mean SEM, mean values indicated by asterisk (*) along the groups are significantly different at P 0.05.

Lipid profile control values: TAG 120.3 ± 3.48, TCH 125.21 ± 4.58, HDL 56.32 ± 2.02, LDL 75.12 ± 3.25.

Key: TG: Triglycerides, TC: Total Cholesterol; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein.

Lipid peroxidation products (MDA) of diabetic patients

Table 4 shows the result of serum lipid peroxidation product (Malondialdehyde or MDA) of diabetic subjects. The result shows a significant increase (P < 0.05) of the serum level of MDA in the age groups 40 - 49, 50 - 59 and 60> above years as compared with the

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control group but were higher in males than in females. There was no significant difference (P0.05) of the serum level of MDA in the age groups 20 - 29 and 30 - 39 years as compared with the control group.

Lipid Peroxidation Product		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above Years
MDA (mg/dl)	Male	107.170.36	110.941.74	137.052.00**	156.212.29**	179.683.32**
	Female	105.880.52	109.911.11	135.821.82**	154.201.74**	176.353.08**

Table 4: Lipid peroxidation products (MDA) of diabetic patients.

Values are expressed as a mean SEM mean values indicated by asterisk (*) alone the groups are significantly different at (P0.05).

MDA Control value: MDA 105.960.48.

Key: MDA: Malondialdehyde.

Liver function enzymes of diabetic patients

Table 5 shows the result serum alanine amino transferase (ALT), aspartate amino transferase (AST) and alkaline phosphatase (ALP) of diabetic and control subjects according to gender. The result shows significant increase ($P < 0.01$) of the serum level of ALT, AST and ALP in the age groups 50 - 59 and 60> above years as compared with the control group but was higher in females than in males. There was no significant difference ($P < 0.05$) of the serum level of ALT, AST and ALP in the age groups 20 - 29, 30 - 39 and 40 - 49 years as compared with the control group.

LFT Enzymes		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above years
ALT (I/U)	Male	37.911.07	38.371.26	40.490.96	45.490.96**	49.862.20**
	Female	39.301.14	41.661.58	42.380.82	47.991.28**	54.591.43**
AST (I/U)	Male	39.170.78	39.740.84	40.340.77	48.430.98**	50.300.92**
	Female	39.900.64	40.940.66	41.190.58	50.100.67**	52.720.53**
ALP (I/U)	Male	141.440.88	142.570.63	143.240.55	146.770.99**	149.760.65**
	Female	143.970.42	144.820.76	144.770.87	149.050.99**	152.370.81**

Table 5: Liver function enzymes of diabetic patients.

Values are expressed as a mean SEM mean values indicated by asterisk (*) alone the groups are significantly different at ($P < 0.05$).

Liver Function Enzymes control values: ALT 36.71.37, AST 38.410.88, ALP 141.50.77.

Key: ALT: Alanine Amino Transferase, AST: Aspartate Amino Transferase, ALP: Alkaline Phosphatase.

Liver function of diabetic patients

Table 6 shows the result of serum direct bilirubin (DB), total bilirubin (TB), total protein (TP) and albumin. There was a significant decrease ($P < 0.05$) of the serum level of DB and TB in the age groups 50 - 59 and 60> above years as compared with the control group. Also, there was no significant difference ($P < 0.05$) of the serum level of DB and TB in the age groups 20 - 29, 30 - 39 and 40 - 49 years.

The result also shows a significant decrease ($P < 0.01$) of the serum level of albumin in the age group 60 > above years but was ($P < 0.05$) in the age group 50 - 59 years as compared with the control group. In the same vein, there was no significant difference ($P < 0.05$) of the serum level of albumin in the age groups 20 - 29, 30 - 39 and 40 - 49 years.

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There was a significant increase (P < 0.01) of the serum level of TP in the age groups 20 - 29, 30 - 39, 40 - 49, and 50 - 59 years as compared with the control group but was (P < 0.05) in the age group 60 > above years as compared with the control group and were higher in males than in females.

LFT		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above years
DB (mg/dl)	Male	2.660.02	2.600.01	2.590.01	2.530.02*	2.520.01*
	Female	2.700.02	2.640.01	2.610.01	2.570.01*	2.560.01*
TB (mg/dl)	Male	2.020.09	1.970.04	1.960.03	1.780.03*	1.750.06*
	Female	2.060.09	2.060.07	2.030.09	1.820.02*	1.780.05*
Albumin (g/dl)	Male	4.080.06	4.010.05	3.970.05	3.900.05*	3.730.09**
	Female	4.170.05	4.140.05	4.070.07	4.010.06*	3.870.05**
TP (g/dl)	Male	11.790.17**	11.100.11**	9.880.10**	9.350.11**	7.880.08*
	Female	12.020.16**	11.210.12**	10.030.10**	9.490.10**	7.950.07*

Table 6: Liver function of diabetic patients.

Values are expressed as a mean SEM mean values indicated by asterisk (*) alone the groups are significantly different at (P0.05).

Liver function control values: DB 2.880.2, TB 2.070.11, Albumin 4.20.05, TP 7.440.1.

Key: DB: Direct Bilirubin, TB: Total Bilirubin and TP: Total Protein.

Serum urea and creatinine of diabetic patients

Table 7 shows the result of serum urea and creatinine of diabetic subjects. The result shows that, there was a significant increase (P0.01) of the serum level of creatinine and urea in the age groups 40 - 49, 50 - 59 and 60 > above years as compared with the control group and were higher in males than in females. There was no significant difference (P0.05) of the serum level of creatinine and urea in the age groups 20 - 29 and 30 - 39 years as compared with the control group.

Kidney Profile		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above years
Creatinine (mg/dl)	Male	0.850.04	1.000.06	2.380.08**	3.560.10**	6.380.13**
	Female	0.680.03	0.890.06	1.930.08**	3.320.08**	5.970.09**
Urea (mg/dl	Male	28.850.70	29.180.63	89.342.67**	132.173.01**	168.573.25**
	Female	27.621.03	28.530.80	85.933.21**	111.091.77**	160.563.45**

Table 7: Serum creatinine and urea of diabetic patients.

Values are expressed as a mean ± SEM, mean values indicated by asterisk (*) along the groups are significantly different at p < 0.05.

Kidney profile control values: Creatinine 0.730.02, Urea 27.870.93.

Serum electrolyte (Na+, Cl-, and HCO3-) of diabetic patients

Table 8 shows the result of serum electrolytes (sodium, chloride and bicarbonate) of diabetic subjects. The result indicated a significant decrease (P0.01) of the level of serum sodium (Na^+) and Bicarbonate (HCO_3^-) in the age groups 40 - 49, 50 - 59 and 60 > above years as compared with the control group but were higher in males than in females. However, there was no significant difference (P0.05) of the serum level of sodium and bicarbonate in the age groups 20 - 29, and 30 - 39 years as compared with the control.

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The result also indicated a significant increase (P0.01) of the serum level of Chloride (Cl^-) in the age groups 40 - 49, 50 - 59 and 60 > above years as compared with the control group but were higher in males than in females. However, there was no significant difference (P0.05) of the serum level of Chloride in the age groups 20 - 29 and 30 - 39 years as compared with the control group.

Electrolytes		20 - 29 years	30 - 39 years	40 - 40 years	50 - 59 years	60> above years
Na^+ (Mmol/l)	Male	138.680.67	137.340.60	120.290.62**	112.880.54**	102.590.64**
	Female	139.030.57	138.540.98	122.830.86**	113.030.80**	102.241.00**
Cl^- (Mmol/l)	Male	95.590.97	100.241.87	133.332.58**	163.271.12**	207.178.23**
	Female	87.611.36	95.140.90	124.713.41**	157.571.45**	195.206.74**
HCO_3^- (Mmol/l)	Male	27.690.41	27.490.53	19.290.28**	15.740.23**	12.030.43**
	Female	27.010.52	26.480.75	18.80.28**	15.100.30**	11.140.39**

Table 8: *Serum electrolyte (Na^+, Cl^-, and HCO_3^-) of diabetic patients.*

Values are expressed as a mean ± SEM, mean values indicated by asterisk (*) along the groups are significantly different at p < 0.05.

Electrolytes control values: Na^+ 139.400.87, Cl^- 87.242.16, HCO_3^- 28.60.57.

Key: Na^+: Sodium, Cl^-: Chloride, HCO_3^-: Bicarbonate.

Antioxidant enzymes of diabetic patients

Table 9 shows the result of serum superoxide dismutase (SOD) and catalase in diabetic subjects. The result indicated a significant decrease (P0.01) of serum superoxide dismutase (SOD), and catalase in the age groups 40 - 49, 50 - 59 and 60 > above years as compared with the control but was higher in males than in females. There was no significant difference (P0.05) in the serum level of SOD, and Catalase in the age groups 20 - 29, and 30 - 39 years as compared with the control groups.

Antioxidant Enzymes		20 - 29 years	30 - 39 years	40 - 49 years	50 - 59 years	60> above years
SOD (U/L)	Male	167.680.78	167.160.56	147.971.02**	140.531.21**	135.710.65**
	Female	166.510.59	166.330.66	145.950.84**	138.641.04**	133.620.75**
Catalase (U/L)	Male	114.320.74	112.081.26	109.340.91**	94.560.78**	88.421.04**
	Female	107.820.81	106.571.05	102.271.54**	94.160.72**	83.710.58**

Table 9: *Antioxidant enzymes of diabetic patients.*

Values are expressed as a mean SEM mean values indicated by asterisk (*) alone the groups are significantly different at (P0.05).

Antioxidant enzymes control values: SOD 169.20.48Catalase 111.680.57.

Key: SOD: Superoxide Dismutase.

Multivariate logistic regression result

Table 10 shows the result of independent predictor variables by using multivariate adjusted odd ratio for factors predictive or that was associated with the complications of diabetes mellitus in the patients attending ABUTH and Specialist hospitals Zaria. From the result of this study males had 1.449 times (AOR 1.449, P < 0.046) increased odds of having diabetes mellitus over females. While patients aged > 40years had 1.6E8 times (AOR 1.6 E8, P < 0.997) increased odds over those aged of < 40 years. Subjects with higher BMI had higher odds

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of having DM. Over weight and obese diabetic patients were 1.381 and 2.048 times (AOR 1.381, P < 0.01 for overweight and AOR 2.048, P < 0.01 for obesity) respectively more likely to develop diabetes mellitus compared to other diabetic patients.

Furthermore, there is association between DM and other complications notably for a lipid profile abnormalities: Triglycerides (AOR 1.045, P < 0.01), Cholesterol (AOR 1.027, P < 0.01), and LDL (AOR 7.328, P < 0.095), Kidney failure abnormalities: Creatinine (AOR 43.66, P < 0.01), Urea (AOR 1.122, P < 0.01), and Chlorite (AOR 1.126, P < 0.01), Liver profile abnormalities: AST (AOR 1.250, P < 0.01), ALT (AOR 1.191, P < 0.01), and ALP (AOR 1.330, P < 0.01), Antioxidant status: MDA (AOR 1.180, P > 0.01).

Explanatory Variables	β	Odds Ratio	OR 95%CI	p value
Male sex (vs. females)	0.371	1.449	1.007-2.085	0.046
Age (years)				
20 - 29	-1.386	0.250	0.115-0.543	< 0.01
30 - 39	-1.386	0.250	0.115-0.543	< 0.01
40 - 49	21.203	1.6E8	0.00	0.997
50 - 59	21.203	1.6E8	0.00	0.997
60> above	21.203	1.6E8	0.00	0.997
Overweight	0.323	1.381	0.788-2.421	0.260
Obesity	0.717	2.048	1.215-3.450	0.007
Triglycerides	0.042	1.043	1.027-1.060	< 0.01
Cholesterol	0.027	1.027	1.020-1.034	< 0.01
HDL	-0.136	0.873	0.847-0.900	< 0.01
LDL	1.992	7.328	0.00-8.5E31	0.0958
Creatinine	3.776	43.662	13.258-143.794	< 0.01
Urea	0.115	1.122	1.068-1.179	< 0.01
Na^+	-0.314	0.730	0.665-0.802	< 0.01
CL^-	0.118	1.126	1.085-1.168	< 0.01
HCO_3^-	-0.541	0.582	0.510-0.664	< 0.01
SOD	-0.279	0.756	0.697-0.821	< 0.01
Catalase	-0.200	0.819	0.778-0.862	< 0.01
MDA	0.166	1.180	1.126-1.236	< 0.01
AST	0.223	1.250	1.174-1.331	< 0.01
ALT	0.175	1.191	1.132-1.253	< 0.01
ALP	-0.285	1.330	1.219-1.452	< 0.01
TB	-1.506	0.222	0.099-0.499	< 0.01
DB	-1.557	0.211	0.077-0.580	< 0.01
TP	-0.186	0.830	0.711-0.970	< 0.01
Albumin	-2.322	0.098	0.035-0277	< 0.01

***Table 10:** Multivariate adjusted odd ratio (95% CI) for factors predictive of diabetes mellitus complications.*

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Discussion

In this present study, an attempt was made to determined diabetes mellitus (DM) and its related complications in the diabetic patients attending Ahmadu Bello University Teaching Hospital and Specialist Hospital Zaria, Kaduna State.

Age and gender distribution of diabetes mellitus

There was an equal distribution of age and gender, DM complications increased with advancing age, this could be because aging is often accompanied by decline in lean body mass and increase in body fat particularly by visceral adiposity which may contribute to the development of insulin resistance [23]. Aging is also known to induce a decrease of insulin sensitivity and inadequate response of β-Cell functional mass when there is insulin resistance [8]. Aging is associated with a decrease of β-Cell proliferate capacity and enhances sensitivity to apoptosis [43]. Males suffers more from DM complications than females, the explanation for this discrepancy has consistently been fast changing life style due to over whelming influence of Westernization Cum. Urbanization and with economic and technological development [12]. In most developing countries which tends to decrease physical activity in males as they went to work in the offices while females became responsible for all house work and even animal grazing work.

BMI pattern of diabetes mellitus

The present study shows that there was a significant difference of DM between different body mass indexes (BMI). In this study in both men and women, the BMI of DM subject was 19.9% for normal 39.6% for overweight and 50.5% for obesity. This data support the notion that overweight and obesity in particular are strong risk factors for type 2 diabetes a finding that is consistent with those report previously in various racial/ethnic populations [29]. In comparable study from India, it was reported that T2DM patients who had obesity along with hypertriglyceridemia and high LDL-C and non-HDL-C levels exhibited poorly controlled HbA1c levels compared with a group with normal lipid profiles [40]. Moreover, several reports have shown a significant influence of lipid levels on HbA1c level and cardiovascular complications possibly a consequence of increased insulin resistance [46].

Diabetes mellitus and liver function enzymes

Diabetes mellitus is often simply considered as a syndrome of disordered metabolism with abnormally high blood glucose levels (hyperglycemia) [12]. Besides, the microvascular and macrovascular complications in DM a compromised immune state is also a condition that increase the susceptibility of diabetics patient to a different infections.

The present study determined the most common liver abnormalities found in different diabetic progression stages among middle aged and elderly DM patients attending Ahmadu Bello University Teaching Hospital (ABUTH) and Specialist Hospital Zaria were elevated serum level of AST, ALT and ALP. The level of AST, ALT, and ALP increases with ages. The serum level of AST, ALT, and ALP was found to be significantly higher in diabetic patients group in comparison with the normal control group.

This finding is consistent with the result obtained from various researchers, it was shown that individuals with T2DM have higher incidence of liver function test abnormalities than individuals who do not suffer from DM [17]. Aminotransferases such as ALT and AST Activities are sensitive indicators of liver cell injury and are helpful in recognizing hepatocellular disease, chronic mild elevation of liver enzymes is frequently found in type 2 diabetic patients [15].

In this study though not statistically significant level, values of transaminases where higher in females than in male's diabetic patients, Similar finding was observed in a study conducted from western part of Nepari which reported higher derangement of liver enzymes in female diabetic subjects than that of diabetic males [2]. Also the finding reported from Nigeria [17]. Similar studies were also reported significant differences in transaminases level in gender wise distribution [7]. Whereas finding by [17] shows a significant difference of AST level alone between the two genders in diabetic subjects [15].

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Insulin resistance activates lipolysis resulting in accumulation of non- esterified fatty acid. This enhanced fat accumulation in liver and is known to be directly toxic to hepatocyte [15]. This attributes increase in transaminases and diminished synthetic capacity of the liver [23]. One of the hepatic manifestations of DM with metabolic syndrome is NAFLD and more specifically ALT has been used as a marker of NAFLD [31].

Diabetes mellitus and liver function

In this study, data emphasizes statistically significant rise in transaminases and significantly lower level of direct bilirubin (DB), total bilirubin (TB), albumin and elevated level of total protein (TP).

The present study, revealed a low serum level of bilirubin but main finding of present study is that elevated bilirubin levels are associated with better control of type 2 DM patients who are otherwise healthy. The first study that revealed low level of bilirubin is by [1], which reported negative correlation between bilirubin in blood flow and coronary artery diseases [39]. Following studies were confirmed their result by revealing inverse association between circulating bilirubin levels and stroke [38], ischemic heart disease and coronary claudication [39]. Moreover, it has been proposed as a better predicator of heart disease than HDL cholesterol [1]. Rate of cardiovascular diseases increased in uncontrolled type 2 DM, thus lower levels of serum bilirubin in poorly controlled subjects [2].

Chronic oxidative stress causes worse diabetic control via defective insulin secretion and gene expression [6]. In 1993 wolf suggested that DM was linked to oxidative stress. Another study revealed that micro and macrovascular complications of diabetes mellitus were strongly correlated with oxidative stress [26]. Bilirubin is an endogenous antioxidant and decrease the levels of free oxygen radicals, thus, ameliorate oxidative stress [11]. Therefore, free oxygen radicals may increase by lowering of bilirubin levels, thus, causing worse diabetic regulation. Hyperglycemia in Type 2 DM is supposed as the main cause of diabetic complications. Glycation of serum proteins, which increase in hyperglycemic states, may have an important role in such conditions. Bilirubin should play an important role in glycation of protein. These also explain the association of diabetic regulation and lower levels of bilirubin.

It was shown in a previous studies, that risk of cardiovascular diseases and diabetes increases with lower level of serum albumin [22]. In another paper, level of serum albumin has been associated with nephropathy; the test is clinically used to diagnose kidney disease in diabetic individuals. Renal damage increases with increasing age of diabetic individuals [17]. It is well known fact that diabetic nephropathy is associated with albuminuria, microalbuminuria being the earliest indicator of development of diabetic nephropathy. Low albumin levels observed in these patients can be attributed to the associated renal diseases. Study by [22] suggests lowered albumin level in diabetics [6].

Comparatively, high total protein found in this study is supported by findings of various studies [46]. This elevation could be attributed to the elevation of various acute phase proteins CRP, α-1 acid glycoprotein, plasminogen, compliment c_3, ceruloplasmin [29]. Similar findings have also been reported that increase in total protein may be due to the elevation of acute phase proteins, globulins, and fibrinogen compounded by a decrease in fractional synthetic rate of albumin due to insulin resistance deficiency. Thus, low levels of albumin in the body due to some other etiology may result in over estimation of HbA1c in diabetics as shown by [29] which can be due to competition between serum albumin and haemoglobulin glycation. Routine measurement of serum of albumin with HbA1c may be helpful to interpret the discrepancy between the degree of glycemic control and evaluation of diabetic complications [25].

Diabetes mellitus and kidney profile

Diabetic nephropathy is a disease that occurs as a result of diabetes. An international study has reported that diabetes control worsened with longer duration of the disease, with neuropathy as the most common complications followed by cardiovascular complication, renal complications, retinopathy and foot alters. Diabetic nephropathy can occur in both type 1and type 2 diabetes mellitus [3].

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In this study, the most common kidney abnormalities detected in different diabetic progression stages among middle aged and elderly patients attending ABUTH and Specialist Hospital Zaria was elevated serum creatinine and urea.

The result of this study are in agreement with various studies which showed that raised serum creatinine and blood urea levels in diabetic patients may indicate a pre-renal problem [21]. In this study, high serum creatinine levels were seen in males than females which could be because of the presence of high muscle mass in males compared to females as reported earlier [5].

This study results shows that poorly controlled blood sugar level would cause increase in the blood urea level and thus increase the chances of patients suffering from diabetics nephropathy, this is in agreement with the findings of earlier study report that hyperglycemia is one of the major causes of progressive renal damage [1].

Furthermore, this increase in the blood urea and serum creatinine indicates the progression towards diabetic nephropathy and estimation of serum creatinine has greater prognostic ability compared with that of blood urea for predicting the adverse outcome [5]. Therefore, increased blood urea and serum creatinine levels in diabetics, clearly indicate prolonged hyperglycemia which cause irretrievable damage to nephrons of the kidney [7], over time high blood sugar levels damage millions of nephrons, and tiny filtering units with each kidney as a result kidneys are unable to maintain the fluid and electrolyte homeostasis. Serum creatinine is filtered by the glomerulus. Therefore, serum creatinine level is used as an indirect measure of glomerular filtration as glomerular filtration rate (GFR) diminishes; there is a rise in plasma concentration of serum creatinine and blood urea. Raised serum creatinine and reduced GFR has become fairly reliable indicators of kidney dysfunction. Serum levels of urea and creatinine can be used as a prognostic markers and predictors of renal damage in diabetic patients [1].

Diabetes mellitus and electrolyte imbalance

Diabetes mellitus is also a metabolic syndrome that is characterized by electrolyte variability as a result of high concentration of glucose in the extracellular fluid and low concentration in the intracellular fluid. The increase concentration of glucose in the extracellular creates in appropriate concentration gradients that tend to move fluid from the intracellular to the extracellular. The increase of water concentration in the extracellular distorts the fine "electrolyte balance" hence leading to polydipsia and polyuria. This cascade of malfunction further instigates the rennin-angiotensin pathway and the hypothalamus to balance the derangement. All these further distort the fine ion balance because membrane transportation is involved and both utilize the same membrane for metabolic activities [41].

In this study, the most common electrolytes imbalance detected in different diabetic progression stages among middle aged and elderly patients attending ABUTH and Specialist Hospital Zaria was a significant decrease in the serum level of sodium (Na^+) and bicarbonate (HCO_3). Increased urination in DM leads to loss of electrolytes and water and result in the imbalance which disturbs sodium and potassium levels in the body. In the present study, it was found that sodium level in DM was found to be low when compared with control [41]. Studies suggest that uncontrolled DM can induce hypovolemic hyponatremia due to osmotic diuresis. Furthermore in diabetic ketoacidosis urinary electrolyte loss magnify the renal sodium wasting [5]. The most common cause of hypotonic-hyponatremia in patients with diabetes is osmotic dieresis-induced hypovolemia [41]. It should be mentioned that in patients with diabetes ketoacidosis the excretion of β-hydroxybutyrate and acetoacetate obligate urine sodium loses resulting in aggravation of hypovolemia [37]. Hypovolemia can also be due to diabetes mellitus-associated complications, such as diarrhea and vomiting. Serum sodium levels in poorly controlled patients with DM vary, since these levels are the result of hyperglycemia-induced hyponatremia (dilutional hyponatremia), osmotic dieresis-induced hypotonic losses (losses of water in excess of electrolytes), which tend to increase serum sodium levels, and hypovolemia-induced decrease in serum sodium levels [41].

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Significant decrease in the serum level of bicarbonate (HCO_3) clearly showed the deficit of bicarbonate resulting from its massive utilization for buffering. HCO_3 is a useful buffer that is spent in maintaining equilibrium in the production of hydrogen ions [41].

Elevated serum chlorite levels were found in diabetic patients and this might be due to diabetic ketoacidosis. Ketoacidosis causes reduction in blood pH which further disturbs acid- base balance and leads to the elevation of chlorite. Therefore, the association between blood glucose and serum electrolyte is multifactorial in which it is related to a number of other factors, which include age and associated condition.

Diabetes mellitus and lipid profiles

Lipid profile varies and is highly associated with diabetes mellitus and both are important parameters for dyslipidemia, hypertension, hyper-insulinemia and coronary heart disease. Accelerated coronary and peripheral vascular atherosclerosis is very common and serious complications of long term diabetes mellitus [32]. Due to economic growth and changing of life style in developing countries the prevalence of abnormal serum lipid profile is increasing particularly in population with chronic illness, with less activity. Dyslipidemia is the most important independent predictor of cardiovascular disease (CVD), in diabetic patients which leads to high morbidity and mortality of diabetic patients [1].

In this study, the most common lipid abnormalities detected in diabetic patients attending ABUTH and Specialist Hospital Zaria among middle aged and elderly patients was elevated triglycerides, cholesterol, LDL followed by low serum level of HDL.

As with the triglycerides, improvement in glycaemic control leads to an increase in the level of HDL-C and suggest the evidence for a role for poor glycaemia in decreasing the level of this lipoproteins. Other researchers also associated the high triglyceride level to the poor glycemic control of diabetes and obesity [32]. Abbate and Brunzel reported that the increase in triglycerides in poorly control diabetic patients was related to the decrease of activities of adipose tissue and muscle lipoprotein lipase activity [1].

Epidemiological study has shown evidence for relationship of serum lipid profile with CAD risk in multiple risk factor intervention trial (MR FIT), CAD risk decline with progressive lower serum cholesterol level [42].

Further supportive relationship between CAD risk and dyslipidemia comes from various recent primary and secondary prevention trials with lipid lowering therapy [36]. Heart protein study (HPS) had shown that lipid lowering with statin therapy is efficacious in patients with diabetes to reduce the risk of CAD [35], (prove IT) trial has demonstrated that intensive LDL-C lowering will reduce the major coronary events [42]. The National Cholesterol Education Program Adult Treatment Panel III (NCEPATP III) guidelines recommend LDL cholesterol goal of < 100 mg/dl.

A study reported that a reduced HDL-C is a powerful predicator for premature coronary heart disease [1]. According to Goldberg HDL-C converted to VLDL-C particles and denser LDL particles acquire a large proportion of these HDL esters. This process decreases the HDL-C level [16]. Besides, HDL-C is ready substrates for hepatic lipase which converts it into smaller particles which are readily cleared from the plasma [42].

Furthermore, multiple factors such as obesity, overweight are associated with dyslipidemia, interestingly, age > 50 years was significantly associated with dyslipidemia in diabetic patients which could be attributed to work pressure and lack of physical activity [9]. As expected, obesity was significantly associated with dyslipidemia in T2DM patient. In a patient comparable study from India, it was reported that T2DM patients who had obesity along with hypertriglyceridemia and high LDL-C and Non-HDL-C level exhibited poorly controlled HbA1c level compared with a group with normal lipid profiles [34]. Moreover, several reports have shown a significant influence of lipid level on HbA1c levels and cardiovascular complications possible as a consequence of increase insulin resistance [9].

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Diabetes mellitus and antioxidant enzymes

Diabetes does not only alter the metabolism of carbohydrates, lipid and protein but also the chemistry of macromolecules [44]. Poorly controlled diabetes accelerates the chemical modification of protein and their functions which could lead to the development of diabetic complications [47]. In this context, several hypotheses have been made in order to understand the origin of the complications observed in diabetic patients. These hypothesis include mitochondria damage, mitochondrial effect in oxidative phosphorylation, increased formation of advanced glycation end product (AGES), increased activity of polyol pathway, hypoxia, alteration of lipoprotein metabolism, increased protein kinase activity, alteration of growth factors and cytokine activities and increased oxidative and reductive stress [36]. Although oxidative stress appears as one of the metabolic events associated to diabetes and its complications [18]. Oxidative stress seems to be increased in a system where the rate of free radicals production is increased and or the antioxidant mechanisms are impaired [47].

The present study, determined the antioxidant enzymes abnormalities in different diabetic progression stages among middle aged and elderly patients attending ABUTH and Specialist Hospital Zaria, Kaduna state with low level of serum SOD and catalase.

This result shows decrease in the activity or level of SOD and catalase. Similar result were found in the previous studies by Fatani using animal model, hyperglycemia is known to inactivate the activities of anti-oxidative enzymes in diabetic animal models, which may involve non enzymatic glycosylation [47]. Anti-oxidants enzymes as well as non-enzymatic anti-oxidants are first line of defense against ROS-Induced oxidative damage in a living organism [36].

Diabetes mellitus and lipid peroxidation product (MDA)

An increase in glucose level induced diabetes, the overproduction of oxygen free radicals which consequently increases the protein and lipid peroxidation. Malondialdehyde (MDA) is highly toxic by- products partly produced by oxidation and derived from lipid. MDA reacts both irreversibly and reversibly with proteins and phospholipids with profound effects [18].

In this study, high statistical significant differences were observed in the level of MDA than in control group similar results were found in previous studies by [33] the observed high level of plasma MDA in diabetic patients reflects lipid peroxidation which is the consequence of oxidative stress. The increase in the level of MDA correlates which hyperglycemia in these patients because of self-oxidation of glucose and could generate free radicals [10].

The result of multivariate logistic regression analyses showed that diabetes were statistically significantly and positively associated with sex, age, overweight, obesity, lipid profile, kidney profile, antioxidants and liver profile abnormalities. In the multivariable logistic models, male sex, older age > 40 years, overweight, obesity, level of triglycerides, cholesterol and HDL for cardiovascular diseases (CVD), level of creatinine and urea for kidney damage, AST, ALT, and ALP for liver damage and the level of Malondialdehyde (MDA) for the lipid peroxidation products were all statistically and positively associated with an increased risk of diabetes mellitus complications.

Conclusion

The complications of diabetes mellitus was high in the patients attending Ahmadu Bello University Teaching Hospital (ABUTH) and Specialist hospital Zaria among middle aged and elderly type 2 diabetes mellitus patients. Gender, aging, higher body mass index (BMI), lipid profile abnormalities, kidney function profile abnormalities, liver profile function abnormalities and antioxidant status were the risk factors associated with diabetes mellitus that lead to cardiovascular diseases, diabetic nephropathy, NAFLD and the generation of free radicals beyond the scavenging abilities of endogenous antioxidant defense, that can result in oxidative stress related diabetes, which can increase morbidity and mortality in diabetes mellitus. Effective control of blood sugar can stop or prevent the progression of diabetes mellitus to all these complications.

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Acknowledgement

Conflict of Interest

There is no conflict of interest.

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