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Survey of the Iron Status of Patients with Type 2 Diabetes Mellitus Attending Hospitals in Jos

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Authors' contributions

This whole work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Original Research Article

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ABSTRACT

Background and Objective: Epidemiological studies have shown that high body iron stores are associated with insulin resistance and type 2 diabetes. The aim of this study was to evaluate iron status of patients with type 2 diabetes mellitus (T2DM). Blood samples were collected from participants after overnight fast.
Materials and Methods: Two hundred (200) subjects comprising 130 type 2 diabetics attending Hospitals in Jos and 70 normal subjects as controls were involved in the survey. Questionnaires were used in the recruitment of participants. Serum ferritin (SF) was assayed by ELISA method, while other parameters were determined colorimetrically.
Results: Diabetics presented with higher mean age, BMI, and blood pressure than non diabetics. Also, diabetics had elevated serum ferritin, SI, TIBC, total cholesterol (TC), triglycerides, and TC/HDL ratio, lower serum HDL; elevated serum aminotransferases and creatinine than non diabetic subjects. There was a strong and significant positive correlation between serumn ferritin levels and each of six diabetes mellitus risk factors: systolic blood pressure, diastolic blood pressure, serum total cholesterol (TC), serum triglyceride (TG), HDL and TC/HDL.
Conclusion: This work has shown that type 2 diabetic subjects exhibited strong positive

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diagnostic features for the indices of iron status, dyslipidaemia, liver damage and kidney dysfunction compared to non diabetic subjects.

Keywords: Ferritin; type 2 diabetes; diabetes risk factors; hyperglycaemia.

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a predominant public health concern worldwide, accounting for 90% of the cases of diabetes globally [1,2]. The chronic hyperglycaemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels [3].

In diabetes, lipid abnormalities, anaemia, alteration of liver and kidney functional indices have been implicated as major risk factors to the progression of Diabetic complications [4].The pathogenesis of type 2 diabetes mellitus (T2DM) is complex and involves the interaction of genetic and environmental factors [5]. Individuals with T2DM show both insulin resistance and beta cell defects [6].

The relationship between T2DM and iron metabolism has gained interest in both research and clinical practice [7]. Several studies indicate that increased body iron stores and subclinical haemochromatosis have been associated with the development of glucose intolerance and type 2 diabetes [8]. Several studies have shown that moderate increases in iron stores below the levels found in patients with hereditary haemochromatosis (HH) were associated with significant elevations in blood glucose and insulin levels [9]. Increased serum ferritin, reflecting body iron overload, is often associated with measures of insulin resistance [8].

Genetic mutations in the haemochromatosis gene (HFE) make up the most common genetic cause of elevated serum ferritin levels [10,11]. Hereditary haemochromatosis (HH) is an autosomal recessive disorder characterized by enhanced intestinal absorption of dietary iron [12]. Defects in human haemochromatosis protein (HFE) cause iron overload due to reduced hepatic hepcidin secretion [13]. Genetic and acquired factors lead to increased iron absorption and storage in hepatocytes, with consequent alteration in the redox status [14].

Excess iron impairs pancreatic β cell function and causes β cell apoptosis [15]. Iron serves as a potent pro-oxidant in human body and participates in the generation of reactive oxygen species (ROS) such as hydroxyl radical [16]. The susceptibility of β-cells to iron-induced oxidative stress and the iron deposition in β-cells usually leads to apoptosis, and consequently, to insulin deficiency [15]. Iron deposition also induces insulin resistance by inhibiting glucose uptake in fat and muscle tissues, and reducing the capacity of liver to extract insulin, which results in an abnormal increase in hepatic glucose production [17].

Although several epidemiological studies have reported a strong association between elevated serum ferritin and increased risk for type 2 diabetes; more so, a link between serum ferritin concentration and insulin resistance or type 2 diabetes has been established [18]. However, it appears that little or no work on the relationship between iron status and type 2 diabetes mellitus has been done in Nigeria, hence, the need for this study.

This study was carried out to evaluate the iron status of patients with type 2 diabetes mellitus (T2DM) in Jos, for the purpose of ascertaining whether the hypothesis regarding the relationship between iron stores and type 2 DM, which has been established by studies in several countries of the world, is also applicable to Nigeria and Plateau state, in particular.

2. MATERIALS AND METHODS

2.1 Study Location/ Sampling Procedures

This study was carried out amongst diabetic patients attending the outpatient medical clinics of Jos University Teaching Hospital, Plateau Specialist Hospital, Jos, and ECWA Evangel Hospitals, Jos and amongst normal subjects as controls. A total of two hundred (200) subjects comprising of one hundred and thirty (130) type 2 DM patients (70 females and 60 males) aged 40-70 years and seventy (70) apparently healthy subjects as controls (37 females and 33 males) were recruited for the study. Patients with acute or chronic inflammatory or infective disease and serious diabetic complications were excluded from the study. Control subjects consist of individuals with no history of medical disorder on the basis of their clinical history and biochemical data.

Ethical clearance from hospital review boards and informed consents from patients/controls were obtained. One standard questionnaire was completed for each subject, which included subject's personal data, drug usage, disease history and physical examination. The designing of questionnaire and recruitment of participants was carried out under a close supervision by a clinician. Confidentiality was maintained in accordance with standard medical practice.

Blood pressure was measured by mercury sphygmomanometer in the right upper arm of the subject, who was seated for 5 minutes before the measurement. Blood pressure was measured twice, and the mean of these two measurements was used in the analysis. Weight was measured without shoes to the nearest 0.1kg on a weighing machine and height was measured to the nearest 0.1m with an anthropometric rod. Body mass index was calculated as weight in kilograms divided by the square of height in metres.

2.2 Sample Collection and Preservation

10ml of venous blood was collected from each volunteer after an overnight fast (2ml into fluoride oxalate bottle for glucose analysis, 2ml into EDTA bottle for some haematological parameters and the remaining into plain tubes for other biochemical analysis. Plasma glucose and haematological analysis were carried out immediately while blood in the plain tube was allowed to clot and centrifuged to separate the serum from the cells for subsequent analysis of other biochemical parameters. Sera were stored frozen at -20^{o}C, prior to assay. Frozen sera were completely thawed and well mixed and all reagents were allowed to attain room temperature.

2.3 Biochemical Analysis

Plasma glucose concentration was determined using glucose oxidase method [19]. Total cholesterol was determined by enzymatic point method [19]. Triglyceride (TG) was determined by enzymatic colorimetric method by hydrolysis of triglycerides [19]. High-density Lipoprotein-cholesterol (HDL-c) was measured colorimetrically in the supernatant fluid by enzymatic endpoint method (Randox test kit), following the precipitation of other lipoproteins (apo-B-100-containing lipoprotein) with a polyanion-divalent cation mixture (phosphotungstic and magnesium chloride) [20]. Low-density lipoprotein-cholesterol (LDL-c) was calculated by the Friedward equation summarised as follows [21]:

$$LDL = (Tc - HDL\text{-}c) - \frac{\text{Triglyceride}}{2.2} \text{ (mmol/L), where Tc = Total cholesterol}$$

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were assayed by the kinetic method of Karmen [22].

Serum iron (SI) was assayed by the colorimetric method by Stoockey [23] whereas total iron binding capacity (TIBC) was determined by the colorimetric method of Ramsay [24]. Serum ferritin was assayed using human ferritin enzyme immunoassay test kit (catalogue number: 6001) based on the method of White et al. [25].

Serum creatinine was determined by the Jaffes reaction methods [26].

Parked cell volume (PCV) was determined by the micro-haematocrit method [27]. Haemoglobin was assayed by the cyanmethaemoglobin method [27].

White blood cells (WBC) were determined by visual white cell counts [28].

2.4 Statistical Analysis

Statistical analysis was performed with SPSS software version 16.0. Data are expressed as the mean ± SEM. Independent sample t-test was used for comparison of means of variables between groups while Pearson correlation coefficient was used to determine the relationship between serum ferritin and the outcome variables. Statistical significance was considered when p value was less than 0.05.

3. RESULTS

The results of the physical anthropometric risk indices profiles in diabetic and control subjects are summarised on Tables 1. The mean age of the controls (41.9±0.5) was significantly (*P*=0.000) lower than that of the diabetics (52.7±0.8). Expectedly, the mean blood pressure (systolic & diastolic) of control subjects (124.4±1.5; 79.9±1.2 in mmHg) were significantly lower than in the diabetics (153.6±2.3; 91.6±1.1 in mmHg). There was statistical significant difference between male and female control subjects in height, BMI, and diastolic blood pressure all but serum ferritin being higher in males, while male and female diabetics differ significantly only in height, the mean height being higher in males.

The results of the serum glucose and the indices of iron status in diabetic and control subjects are summarised on Table 2. Expectedly, the mean glucose level was significantly (*P*=0.000) higher in diabetics (9.9±0.4 mmol/l) than in the control (4.4±0.1 mmol/l). Similarly, the mean values of the three iron status indices, serum ferritin, serum iron and total iron binding capacity (TIBC), were significantly higher in diabetics than in the control subjects. The differences in the mean PCV and haemoglobin values were not statistically significant (*P*=0.431 and p=0.401 respectively). There are statistically significant gender differences between male and female control subjects in respect of serum ferritin, serum iron, PCV and haemoglobin, with the females having the lower values in all four indices.

Table 1. Mean blood pressure and other physical anthropometric indices of DM subjects

Variables	Diabetic			Control			Total		
	Female (n=70)	**Male (n=60)**	***P* -value**	**Female (n=39)**	**Male (n=33)**	***P*- value**	**Diabetic (n=130)**	**Control (n=37)**	***P* value**
Age (years)	51.7±1.1	53.8±1.2	.329	42.2±0.7	41.5±0.7	.793	52.7±0.8	41.9±0.5[c]	.000
Syst BP (mmHg)	151.5±3.2	154.6±3.7	.393	123.3±2.0	125.6± 2.3	.484	153.7±2.3	124.4±1.5[c]	.000
Diast BP(mmHg)	90.0±1.5	92.7±1.8	.173	80.4±1.4[b]	79.3±2.1	.028	91.6±1.1	79.9±1.2[c]	.021
Weight(kg)	69.9±1.9	72.6±1.9	.461	71.9±2.3	69.7±1.9	.225	71.2±1.4	70.8±1.5	.083
Height(m)	1.57±0.01a	1.7±0.0	.808	1.6±0.0[b]	1.7±0.0	.953	1.6±0.0	1.6 ±0.0	.656
BMI(kg/m)	27.8±0.7	26.2±0.7	.472	29.3± 0.9[b]	25.9±0.9	.525	27.1±0.5	27.7±0.6	.410

Tabulated values are means ± S.E.M; Statistical significance between means was assessed using independent sample t-test, [a]= depicts significant difference between diabetic female versus diabetic male (P<0.05), [b]= depicts significant difference between control female versus control male (P<0.05), [c]= depicts significant difference between diabetic (total) and control (total) (P<0.05), Abreviations: syst BP= systolic blood pressure; diast BP=diastolic blood pressure; BMI=body mass index.

Table 2. Mean serum glucose, serum ferritin and other indices of iron status of DM subjects

Variables	Diabetic			Control			Total		
	Female (n=70)	**Male (n=60)**	***P* -value**	**Female (n=39)**	**Male (n=33)**	***P*-value**	**Diabetic (n=130)**	**Control (n=37)**	***P*- value**
Glucose (mmol/l)	9.4±0.5	10.5±0.6	.109	4.3±0.1	4.5±0.1	.002	9.9±0.4	4.4±0.1c	.000
Ferritin (ng/ml)	65.8±6.4	86.7±6.6	.471	33.8±4.7[b]	62.6±7.5	.043	75.7±4.7	47.9±4.5[c]	.012
SI(µmol/l)	40.8±2.1	46.7±2.2	.944	18.4±0.8[b]	23.0±1.4	.034	43.5±1.5	20.3±0.8[c]	.000
TIBC(µmol/l)	87.1±2.5	82.1±2.8	.492	68.3±3.00	67.3±3.1	.701	84.4±1.9	67.9±2.2[c]	.048
TS (%)	48.1±2.5	58.5±2.6	.823	27.5±1.5[b]	37.2±3.4	.002	52.9±1.8	31.5±1.7[c]	.000
PCV (%)	41.4±0.6	42.6±0.7	.749	41.5±0.7b	44.8±0.7	. 706	41.9±0.5	43.0±0.5	.431
Hb (g/l)	13.8±0.2	14.1±0.6	.036	13.6±0.2b	14.6±0.2	.366	13.7±0.1	14.0±0.2	.401

Tabulated values are means ± S.E.M; Statistical significance between means was assessed using independent sample t-test, [a]= depicts significant difference between diabetic female versus diabetic male (P<0.05), [b]= depicts significant difference between control female versus control male (P<0.05), [c]= depicts significant difference between diabetic (total) and control (total) (P<0.05), Abreviations: SI=serum iron; TIBC=total iron binding capacity; TS=transferrin saturation; PCV=packed cell volume; Hb=haemoglobin.

The results of serum lipids and other indicators of clinical complications of type 2 diabetes mellitus in diabetic and control subjects are summarised on Table 3. There are significant differences between the diabetic and control subjects in all four lipid parameters with the mean values for three of these, total cholesterol (TC), triglyceride (TG) and TC-HDL, being higher in diabetics than in the control subjects; the level of the exception, HDL, was lower in diabetics than the control. There are no significant gender differences in these parameters between the female and male subjects either among the diabetics or control subjects. The mean serum creatinine, a muscle tissue and renal function marker, of the diabetics (108.4 ± 1.8 µmol/l) was significantly higher (*P*=0.001) than in the control subjects (93.4 ± 1.8 µmol/l), a situation consistent with a higher state of muscle tissue catabolism or renal dysfunction in diabetics. There is a significant (*P*=0.022) gender difference in serum creatinine between female and male control subjects with the higher value in males.

The activities of the liver marker enzymes, aspartate and alanine amimotransferases, in the serum were significantly elevated in the diabetics as compared to the control subjects, suggesting a hepatotoxic side effect of diabetes in the subjects. There are no significant gender differences.

The results of the analyses of the relationship between serum ferritin levels and each of the other risk factors, physical anthropometric indices, blood glucose, indices of iron status, serum lipid indices and indicators of clinical complication of DM, in all subjects, diabetics and control subjects by Pearson's two-tailed correlation analysis are summarized on Table 4. There is a significant correlation between serum ferritin levels and systolic BP (*P*=0.040) and diastolic BP (*P*=0.017), respectively, among the diabetic subjects. A similar significant relationship obtains in control subjects and when control and diabetics are pooled.

Among diabetic subjects, there is a significant (*P*=0.005) correlation between serum ferritin levels and fasting plasma glucose level. There is no significant correlation between serum ferritin and any of the other iron status indices of DM among the diabetics. However, a significant correlation exists between serum ferritin and serum iron in the control subjects and total sample population.

There is a significant correlation between serum ferritin and each of three serum lipid risk factors, TC, TG and TC/HDL; the correlation with the fourth lipid index, HDL, is also statistically significant. A similar significant correlation between serum ferritin level and each of all but one, HDL exists among the control subjects and when control and diabetic subjects are pooled. No significant correlation exists between serum ferritin and any of markers of cytotoxic side effects, that is, serum aminotransferases (AST, ALT) and serum creatinine.

Table 3. Mean serum concentrations of lipids and other indicators of clinical complications of DM

Variables	Diabetic			Control			Total		
	Female (n=70)	**Male (n=60)**	***P*- value**	**Female (n=39)**	**Male (n=33)**	***P*- value**	**Diabetic (n=130)**	**Control (n=37)**	***P*-value**
TC (mmol/l)	5.4±0.2	5.6±0.2	.856	3.9±0.1	4.0±0.2	.404	5.5±0.1	4.00±0.1[c]	.000
TG (mmol/l)	2.0±0.1	2.1±0.1	.604	1.0±0.1	1.1±0.1	.668	2.1±0.1	1.1±0.1[c]	.000
HDL (mmol/l)	0.9±0.0	0.8±0.0	.329	1.4±0.1	1.4±0.1	.925	0.9±0.0	1.4±0.1[c]	.023
TC-HDL	6.4±0.2	6.8±0.2	.608	2.8±0.1	3.0±0.2	.414	6.6±0.2	2.9±0.1[c]	.000
AST (iu/l)	25.5±1.2	26.0±1.6	.253	22.7±2.2	21.2±1.5	.116	25.7±1.0	22.0±1.3[c]	.024
ALT (iu/l)	25.9±1.2	27.1±1.6	.126	22.0 ± 1.8	20.1±1 .9	.953	26.4±1.0	21.1±1.3[c]	.001
Creatinine (µmol/l)	107.8±2.6	109.3±2.5	.058	85.6 ± 2.3[b]	102.8±1.9	.022	108.4±1.8	93.4±1.8[c]	.001
WBC	4.6±0.1	4.6±0.1	.517	4.6 ± 0.1	4.2±0.1	.167	4.6±0.1	4.6±0.1	.711

Tabulated values are means ± S.E.M; Statistical significance between means was assessed using independent sample t-test, [a]= depicts significance difference between diabetic female versus diabetic male (P<0.05), [b]= depicts significance difference between control female versus control male (P<0.05), [c]= depicts significance difference between diabetic (total) and control (total) (P<0.05)
Abreviations: TC=total cholesterol; TG=triglyceride; HDL=high density lipoprotein; AST=aspartate aminotransferase; ALT=alanine aminotransferase; WBC=white blood cell

Table 4. Correlation between serum ferritin and some outcome variables

Variables	All subjects		Diabetics		Control	
	R	*P*-value	r	*P*-value	r	*P*-value
Age (years)	.163*	.028	-.003	.977	.086	.515
Weight (kg)	-.036	.625	-.038	.678	-.052	.693
Height(m)	.119	.111	.155	.085	-.107	.412
BMI (kg/m2)	.057	.448	.067	.460	.088	.501
Systolic BP (mmHg)	.306**	.000	.185*	.040	.318*	.015
Diastolic BP (mmHg)	.314**	.000	.215*	.017	.315*	.016
Glucose (mmol/l)	.355**	.000	.248**	.005	.872**	.000
SI (µmol/l)	.255**	.001	.128	.181	.131**	.315
TIBC (µmol/l)	.073	.347	.001	.991	-.161	.215
TS (%)	.225**	.003	.104	.275	.200	.122
PCV (%)	.015	.848	.021	.823	.174	.180
Hb (g/l)	.056	.459	.107	.251	.063	.637
TC (mmol/l)	.447**	.000	.368**	.000	.389**	.002
TG (mmol/l)	.375**	.000	.275**	.002	.360**	.004
HDL (mmol/l)	-.148*	.045	.098	.276	-.167	.211
TC/HDL	.422**	.000	.338**	.000	.434**	.001
WBC	.005	.948	-.042	.656	.249	.053
Creatinine(µmol/l)	.089	.237	.025	.781	.133	.308
AST (iu/l)	0.080	.286	.035	.705	.152	.243
ALT (iu/l)	.057	.449	.026	.773	.039	.767

*Pearson's correlation coefficient was used to determine the relationship between serum ferritin and the outcome variables, *Correlation is significant at the 0.05 level (2-tailed), **Correlation is significant at the 0.01 level (2-tailed), Abreviations: BMI=body mass index; BP=blood pressure; SI=serum iron; TIBC=total iron binding capacity; TS=transferrin saturation; PCV=packed cell volume; Hb=haemoglobin; TC=total cholesterol; HDL=high density lipoprotein; WBC=white blood cell; AST=aspartate aminotransferase; ALT=alanine aminotransferase.*

4. DISCUSSION

Serum levels of iron store markers, such as ferritin, TIBC and serum iron and the common indicators of clinical complications of type 2 diabetes, such as serum lipid profile, liver enzymes and creatinine, were determined in the present study. Significantly elevated serum ferritin levels in diabetics compared to control subjects recorded in the present study support similar findings from previous studies [29,30,31,32]. The exact mechanism through which elevated ferritin promotes the development of type 2 diabetes is uncertain. Elevated iron stores may induce diabetes through a variety of mechanisms, including oxidative damage to pancreatic beta cells, impairment of hepatic insulin extraction by the liver, and interference with insulin's ability to suppress hepatic glucose production [8]. I.e. iron deposition and iron-induced oxidative stress contribute to the pathogenesis of type 2 diabetes (T2D) through β-cells apoptosis, hepatic dysfunction, and insulin resistance [15]. The pancreatic beta cells are particularly susceptible to oxidative damage because of their weak antioxidant defense [9].

The causative role of elevated iron store levels in the onset of insulin resistance is well established by prospective data as well as evidence that blood donations improve insulin sensitivity by decreasing iron stores [16]. Studies have revealed that first-degree relatives of type 2 diabetes mellitus patients with normal glucose tolerance have higher ferritin concentrations than normal control subjects [33]. These observations may suggest a genetic predisposition to hyperferritinaemia in type 2 diabetes. Genetic mutations in the haemochromatosis gene (HFE) make up the most common genetic cause of elevated serum ferritin levels [34,35,11]. Hereditary hemochromatosis is an autosomal recessive disorder associated with the HFE genes and is characterized by enhanced intestinal absorption of dietary iron [36,12].

The systemic iron homeostasis is achieved by regulating iron absorption and storage and recycling mechanisms. The ferroportin-mediated efflux of Fe2+ from enterocytes and macrophages into the plasma is critical for systemic iron homoeostasis [37,38]. This process is negatively regulated by hepcidin, a liver-derived peptide hormone that binds to ferroportin and promotes its phosphorylation, internalization and lysosomal degradation [38,15]. Thus hepcidin acts to decrease the absorption of dietary iron and the release of recycled iron from macrophage stores by diminishing the effective number of iron exporters on the membrane of enterocytes or macrophages [39,40]. Under conditions of high iron, hepcidin-induced down regulation of ferroportin in duodenal enterocytes prevents dietary iron from entering the circulation [15]. Defects in human hemochromatosis protein (HFE) cause iron overload due to reduced hepatic hepcidin secretion [13,41].

Dyslipidaemia in diabetics compared to control subjects observed in this study is in agreement with the findings of other researchers [42,43]. Impaired lipid metabolism resulting from uncontrolled hyperglycaemia has been implicated in diabetes [44,45]. Type 2 DM is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities that are all recognized as predictors for coronary heart disease [46]. Resistance to insulin likely underlies the changes that occur in lipid parameters of type 2 DM and, usually, it is associated with higher concentrations of TC and TG, and lower concentrations of HDL-C [47,48]. The mechanism responsible for hypertriglyceridaemia may be an increased hepatic secretion of VLDL and a delayed clearance of TG-rich lipoproteins, which might mainly be due to increased levels of substrates for TG production, free fatty acids, and glucose. The latter could be secondary to decreased activity of lipoprotein lipase (LPL), a key enzyme for lipoprotein-TG [48].

The significantly elevated liver marker enzymes in diabetes compared to control subjects also obtained in the present study is in line with the report of previous researchers [49,50]. The occurrence of liver disease and raised liver enzymes is common in type 2 diabetes [51,52]. Elevated serum liver enzymes (aminotransferases and alkaline phosphatase) can reflect abnormalities in liver cells or in the bile duct [53,54]. For example, predominant elevation of aminotransferases typically indicates hepatocellular injury, whereas elevated alkaline phosphatase indicates cholestatic injury. Elevated alkaline phosphatase and aminotransferases can indicate a mixed pattern of injury [54]. AST is present in blood cells and many tissues, including liver, muscle, brain, pancreas, and lung. Although ALT is present in several organs and in muscle, the highest levels are in the liver, making it a more specific indicator of liver disease [54]. Alkaline phosphatase is active in many organs, mainly the liver and bones, but is also found in the small bowel, kidneys, and placenta. Diseases of the hepatobiliary system can cause moderate to marked elevations of alkaline phosphatase [54]. Mild chronic elevations of transaminases often reflect underlying insulin resistance [53].

The elevated serum creatinine level in diabetics compared to control subjects obtained in this study is in accord with the reports of several researchers [55,56]. Renal impairment associated with elevated serum creatinine levels is common in diabetes [57,58]. Diabetic nephropathy occurs in approximately one third of type 2 diabetics [59,60]. Raised plasma creatinine in diabetic patients may indicate a pre-renal problem such as volume depletion which may be due to impaired function of the nephrons [55]. Gender differences in serum creatinine between female and male control subjects observed in this study may be due to the difference in muscle mass of males and females [56]. An independent role for gender in the progression of renal disease in human has not been clearly established [56]. Since prospective and interventional studies have confirmed an aetiologic role of iron overload in the pathogenesis of insulin resistance and type 2 diabetes, in the future actively lowering body iron stores may become a tool in preventing type 2 diabetes in selected subjects with impaired glucose metabolism.

5. LIMITATION

Serum ferritin concentration is by far the most commonly used indicator of body iron stores in epidemiological studies [61]. However, the specificity of high circulating ferritin levels as a marker of increased body iron stores is somewhat limited because ferritin is an acute-phase reactant that may be elevated in inflammation and other disorders such as liver disease and cancer [62]. In addition, circulating ferritin is also increased with alcohol consumption and body mass index (BMI), and differs with gender [9]. It is thus unclear whether the association of ferritin with type 2 diabetes risk factors reflects these other abnormalities

Serum soluble transferrin receptor (sTfR) concentration has been suggested as a more accurate measure of available body iron [9]. Circulating sTfR levels correlate inversely with body iron stores and thus reflect, inversely, intracellular iron storage. The sTfR:ferritin ratio has been found to distinguish between subjects with similarly high ferritin levels, and sTfR is believed to be free of influence by acute or chronic inflammation, therefore it has been suggested that the sTfR:ferritin ratio is a better marker than ferritin alone to measure a wide range of iron levels to quantifiably reflect body iron over the entire spectrum of iron balance [16]. More studies on the association of iron stores with type 2 diabetes risk need to be carried out more extensively using sTfR:ferritin ratio in addition to serum ferritin as indicators of iron status.

6. CONCLUSION

From the foregoing, a simple blood test which measures ferritin levels can be used to predict development of diabetes in healthy people. This may help in identifying high risk people who would possibly benefit from lifestyle or therapeutic interventions that can lower iron stores in the body.

CONSENT

Informed consent was used in the recruitment of the participants and confidentiality was maintained in accordance with standard medical practice

ETHICAL APPROVAL

Ethical approval was given by the Ethics Committees of Jos University Teaching Hospital, Plateau Specialist Hospital and ECWA Evangel Hospital Jos.

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COMPETING INTERESTS

Authors have declared no competing interests exit.

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