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Correlation between body mass index, gender, age, family history and the prevalence of diabetes mellitus in Akwa Ibom State, Nigeria

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ABSTRACT

Objective: To study the relationship between body mass index (BMI), gender, age, family history and incidence of diabetes mellitus.
Methods: A total of 445 human subjects volunteered to participate in this study. They were divided into two groups, the test group (diabetics) containing 224 subjects and the control (non-diabetics) containing 221 subjects. Samples (blood and urine) were collected from the subjects on their normal diabetic clinic visit for check-up. The family history and other related data of subjects were obtained from their hospital case note and through the research questionnaire. Analysis was done to determine the blood sugar concentration in both groups. Their weight, height, waist circumference, mid-arm circumference and hip circumference were measured to determine their BMI.
Results: The mean age was significant ($P < 0.01$) at 50 years and above. Diabetes mellitus was significantly ($P < 0.01$) higher among the females. The range of the BMI was between < 20 kg/m^2 and > 40 kg/m^2. The diabetics had a higher BMI than the control subjects in all the BMI ranges used ($P < 0.01$ and $P < 0.001$) except those in the 30–39 kg/m^2 BMI range. The diabetics had significantly higher fasting and random blood sugar concentration ($P < 0.01$) than the non-diabetics. The result on family history showed that 42.5% of the diabetics had no idea of the history of diabetes in their family, 32.14% had first degree relatives with diabetes, and 25% of them had no family history of diabetics.
Conclusions: BMI, female gender, advancing age and positive family history were all directly correlated with the incidence of diabetes mellitus.

1. Introduction

Some disease conditions have been found to be traditionally linked to the body mass index (BMI), gender, age and family history, with diabetes mellitus not exempted, but rather, the strong association of these indices to the etiology of diabetes mellitus has continued to be of immense interest in biomedical studies. All of these indices are more or less inter-related. For instance, body fat increases as the age advances and the muscle diminishes. In the aged, body weight and BMI remain unchanged[1]. Sex or gender difference is known to impose significant influence on physio-chemical processes. The genetic constituent of each individual is the predominant determinant of their peculiarity both in molecular and phenotypic events or outcome.

BMI is a measure of weight adjusted for height, calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Studies have shown that BMI is correlated to more direct measures of body fat seen in underwater weighing and dual-energy X-ray absorptiometry. BMI is a simple, inexpensive, and non-invasive surrogate measure of body fat.

The resulting condition of increased BMI is obesity which is a frequent comorbid condition[2]. BMI and type 2 diabetes are known to go hand in hand and many patients with established type 2 diabetes suffer from poor BMI[3]. Reasonably, accurate studies have shown that BMI level correlates with body fat and future health risks[4]. High BMI may predict future morbidity and death. Therefore, BMI is an appropriate measure for screening for obesity and its health risks. If a poor BMI is diagnosed early, it can

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The study protocol was performed according to the Helsinki declaration and approved by University of Uyo Teaching Hospital. Informed written consent was obtained from the subjects.

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aid potentially in preventing diseases and give warning about any health problem.

In countries undergoing epidemiological transition, a complex picture related to increased food consumption and the accompanying changes towards sedentary life style is frequently found[1]. Consequently, Nigeria as many other developing countries has been experiencing a rapid phase of urbanization and industrialization in recent decades. The prevalence of obesity varies significantly across the world and it is an undesirable outcome of changing lifestyle and behavior.

Obesity is known to induce insulin resistance due to decrease in insulin receptors as the weight increases[5]. Insulin facilitates the uptake of glucose into cells, especially adipocytes and myocytes. Reduction in insulin receptors sensitivity invariably results in the increase of blood glucose level[6]. We therefore expect that BMI should correlate with the level of blood glucose but there has been differing reports; a Scottish study has shown no significant statistical correlation between the random blood sugar level and BMI[7].

The greater risk of diabetes among Asians and the developing countries like Nigeria is identified by making lower BMI cut-off values[8]. This susceptibility is linked to the family history as a combination of genetic susceptibility plus adoption of a high caloric, low activity lifestyle of the developing world[9]. The interplay between genetic and environmental factors develops heterogenous phenotype of obesity[10].

Family history has been shown to be a risk factor for a majority of chronic disease of public health significance including cardiovascular disease, type 2 diabetes mellitus *etc*. The family history of specific disease reflects the consequences of genetic susceptibility, shared environment and common behaviours[11]. Family has been recognized in clinical medicine as an important yet non-modifiable disease risk factor that may influence the probability of a suspected diagnosis[12]. However, collection and interpretation of family history have rarely been applied in the practice of preventive medicine to assess disease risk and influence early detection and prevention strategies[13,14].

The family history of diabetes is not only a risk factor for the disease but it is also positively associated with the risk awareness and risk reducing behaviours[12]. It may provide useful screening tool for the detection and prevention of diabetes[15]. Whereas most previous studies were limited to the association between diabetes mellitus, obesity and BMI and other markers of obesity; the integral correlation between BMI, family history, gender, age and diabetes mellitus prevalence has not been established at the time of this work. The scope of this study therefore is elaborated to wholistically correlate these inter-related factors.

2. Materials and methods

2.1. Sources of samples

Two centres were used; one major and one supplementary centre were chosen for this study. The University of Uyo Teaching Hospital, Uyo, Akwa Ibom State, Nigeria was the major centre and supplementary centre was St. Luke's Hospital, Anua, Uyo.

Subjects numbering 445 participated in this study comprising the test group (224, diabetics) and control (221, non-diabetics). There was no application of inclusion/exclusion criteria as basically all those attending their routine diabetic clinic appointment in the various centres were cooperative and willing to take part in the study. Both the test and the control subject were Nigerians with variation in tribal/ethnic groups. However, indigenes of Akwa Ibom State were the majority in the study population.

Application for permission to carry out the study was forwarded to the Ethics and Rules Committee of the respective study centres. The study protocol was performed according to the Helsinki declaration and approved by University of Uyo Teaching Hospital. The period and the purpose of the study were cleairly stated and informed written consent was obtained from the subjects. The rules with respect to person were strictly complied. Both oral interview and questionnaires were employed. Subjects were interviewed and samples and respective measurement were taken during their weekly clinic visit. The research questionnaires were duly filled during same visit. Family history was gotten from patient's hospital case notes for the diabetic and non-diabetic control respectively. The control group was randomly selected from those on routine medical/surgical check-up, and was confirmed to be non-diabetic.

2.2. Collection, measurement and treatment of samples

2.2.1. Urine

Universal urine bottles were issued to subjects to put in 3–5 mL of urine using the hospital's convenience. The urine samples were kept at normal room temperature (25 °C). Urinalysis was carried out on the samples within 1 h of collection.

2.2.2. Blood

Venepuncture was used in collecting blood samples. A tourniquet was tied above the point chosen for venepuncture. The area was clean with methylated spirit swab (cotton wool). Blood was withdrawn with 5 mL syringe. The blood was emptied into the fluoride bottle for random blood sugar and fasting blood sugar estimation.

2.3. Urinalysis

A strip of combi-10 (glucose test strip) was dipped into the urine in the universal urine container. Excess urine was wiped off with cotton wool. The colour changes on the strip were compared with the standard colour on the bottle within 30 s. The various indices were determined and recorded accordingly using standard

methods.

2.4. Blood sugar estimation and patient's hospital case note

The blood for estimation of blood glucose was collected into fluoride bottle. The one stop glucometer was switched on and the specified glucose strip inserted into the meter. A drop of blood was placed on the sample spot. The meter automatically started timing, and after 45 s the result was displayed in mg/dL.

Patient's hospital case note is a file in the hospital which contains information about the age, sex, the family history and other information about the patient.

2.5. Measurement of parameters

2.5.1. Measurement of body weight

The measurement of body weight was carried out using bathroom weight measurement scale. The scale has an inbuilt scale calibrated in kg. The subjects were made to stand erect on the scale and look forward without their shoes on. The weights were read directly from the scale. The measure of the weight was done in light clothing as this could alter the reading.

2.5.2. Measurement of height

Standing height was measured using a wooden ruler graduated in m and cm. The subjects were made to stand erect, look forward and ruler was placed at the back of the subject and height was read from the head of the subject at the side of the ruler calibrated in m. However, the height was taken without shoes on and with legs placed together as these could alter the reading.

2.5.3. Measurement of mid arm circumference

A measuring tape was used to measure the distance from shoulder joint to the elbow joint and half of the distance between the two joint was considered the mid-arm circumference of the subject. The measuring tape was used to obtain the circumference of the mid-arm in cm.

The subjects were expected to remove all clothing from the region of the mid-arm before the circumference was taken. This was to prevent errors in measurement.

2.5.4. Measurement of waist circumference

Subjects were asked to stand erect and a measuring tape calibrated in centimetre was used to measure the circumference of the waist. The subjects were expected to remove all clothing from the region of the waist before the circumference was taken. This is to avoid or prevent errors in measurement.

2.5.5. Measurement of hip circumference

A measuring tape was used to measure the hip circumference. The subjects were asked to stand erect and the measuring tape calibrated in cm was used to measure the circumference of the hip.

2.6. Statistical analysis

Data were presented as mean ± SEM. The *Chi*-square and student's *t*-test were employed to compare two sets of data. *P* value of less than 0.05 was considered statistically significant.

3. Results

3.1. Blood sugar

The means of the blood sugar concentration were shown by fasting blood sugar and the random blood sugar for diabetics and control groups. The diabetics had significantly higher fasting and random blood sugar concentrations ($P < 0.01$) compared to the non-diabetics (Figure 1).

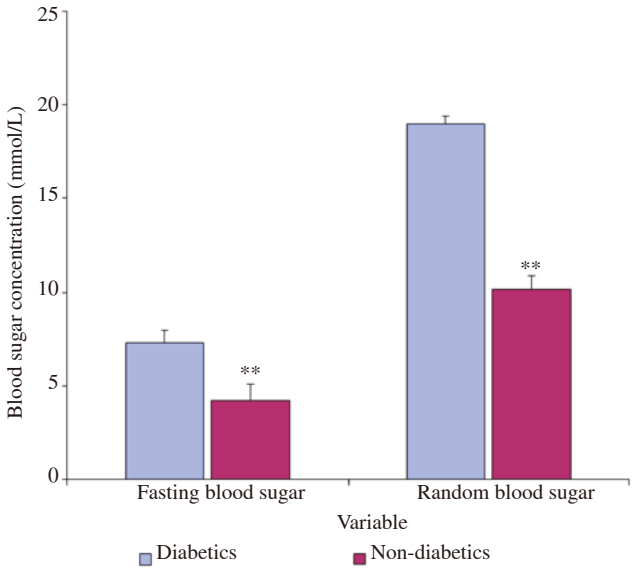

Figure 1. Comparison of blood sugar concentrations in diabetic and non-diabetic subjects.
**: $P < 0.01$ compared to diabetics.

3.2. Diabetic with family history

The data showed that 42.85% of the diabetics had no idea of history of diabetes in their family. About 32.14% had first degree relatives with diabetes, while 25.00% of them had no family history of diabetes.

3.3. BMI among the diabetics and control population

The range of the BMI was between those less than 20 kg/m^3 and greater than 40 kg/m^3. Diabetic subjects had a higher BMI than control subjects in all the BMI ranges used ($P < 0.01$) except those in the 30–39 kg/m^3 range (Figure 2).

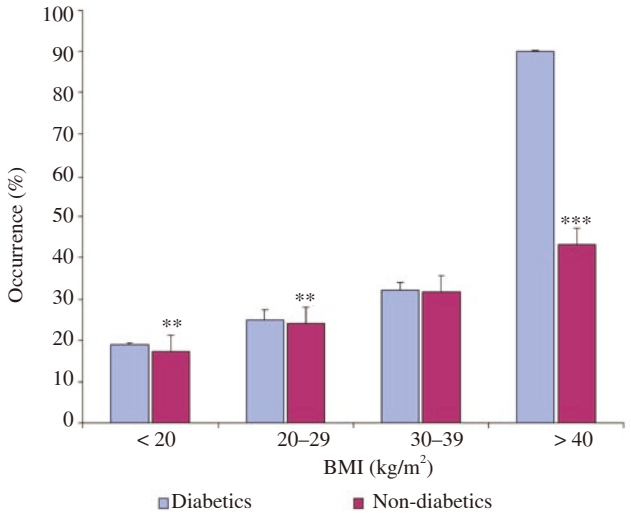

Figure 2. BMI distribution of diabetic and non-diabetic subjects. **: $P < 0.01$, ***: $P < 0.001$ compared to diabetics.

4. Discussion

Parameters considered in this study were gender, age, family history and BMI vis-a-vis their relationship with the incidence of diabetes mellitus in the study population. There was altogether positive pattern in the correlation between these parameters and the prevalence of diabetes mellitus with the sex distribution tilting towards the female gender.

The BMI results showed higher range for diabetics in conformity with the reports of Saikumar *et al.* and Considine *et al.*[4,16]. Higher BMI index is a reliable indicator of obesity. In the pathogenesis of type 2 diabetes mellitus, obesity could be one of the most essential acquired risk factor that can be modified[17]. Visceral obesity is linked with multiple endocrine disturbances, including low growth hormone and elevated cortisol in women and low testosterone secretion in males[18]. Adipose tissue secretes leptin which influences appetite and energy expenditure by signaling the body's state of adiposity to the brain[19]. Leptin deficiency has been reported to cause both severe insulin resistance and obesity[4]. In obese and insulin-resistant subjects and also in individuals with a genetic predisposition to type 2 diabetes mellitus, leptin levels were found to be elevated[16,20].

The result of the mean blood sugar concentration shows an increase in glycemic levels for both the fasting blood sugar and random blood sugar of diabetics. This result coincides with a similar report by Saikumar *et al.* who showed that the glycemic level was found to be higher in diabetes[4]. In a study of 253 subjects, BMI and blood glucose level were positively correlated among Nigerian undergraduates[2].

Innocent *et al.* reported that the relationship between diabetic incidence and gender was positively and strongly correlated among female, suggesting that the difference in sex may be due to increased muscular activities in male than female who are more sedentary[2]. Muscular activities result in the activation of the peptide hormone adiponectin that causes the cascade activation of AMP-activated protein kinase which inhibit and prevent acetyl-CoA carboxylase from synthesizing malonyl-CoA for biosynthesis of fatty acid in the hepatocytes[21]. AMP-activated protein kinase once activated also increases the uptake of glucose and fatty acid from the myocytes into the hepatocytes for metabolism[6].

The alterations in cortisol metabolism and the local activation of cortisol in adipose tissue provide an important link between glucocorticoids and the development of the metabolic syndrome in clinically obese individuals[22]. Cortisol induced insulin resistance is partly explained by its metabolic effect in opposing insulin action[23]. Apart from their antagonising action on insulin sensitivity, glucocorticoids inhibit pancreatic beta cells from secreting insulin[24]. The diabetic patients have increased physical and mental stress. In response to stress, the catecholamines are secreted by the adrenal medulla and sympathetic nerve endings. The catecholamines decrease the insulin effect on glucose utilization and lead to elevation of blood glucose[23].

Reporting a family history depends on the prevalence of the disease, the number of family members, diagnostic facilities available, characteristics of health seeking for patients, and on how familiar they are with the diagnosis among their family members. Most of these factors are likely to be common in an urban environment[25]. A report by van der Sande *et al.* shows a positive link of a familial history to the occurrence of disease such as diabetes and obesity[26]. In the result of this study, 32.14% of the diabetics had a family history of the condition, 25.00% did not have a family history of the condition and 42.85% had no idea at all. The hereditary component of type 2 diabetes and insulin resistance has been studied extensively, with most results showing greater insulin resistance in offspring of diabetics[27,28]. This finding is against the report of Goran *et al.* which hypothesized that a positive family history of type 2 diabetes does not have any significant effect on insulin resistance and associated risk factors for type 2 diabetes particularly in young children[25].

Except for the maturity onset diabetes of the young, the mode of inheritance of type 2 diabetes is unclear. Maturity onset diabetes of the young is inherited as an autosomal dominant trait, which may result from mutations in glucokinase gene on chromosome 7p[29]. Glucokinase is a key enzyme of glucose metabolism in the beta cell and the liver[30]. The receptor gene for insulin is located on chromosome 19, which encodes a protein that has alpha and beta subunits, and a transmembrane tyrosine kinase domain[3]. Mutations affecting the insulin receptor have been identified and their association with type 2 diabetes mellitus and type A insulin resistance is recognised[29].

Obesity and family history are more than just risk factors; they have a casual effect in the development of type 2 diabetes mellitus with a genetic susceptible background predisposing succeeding generation. The evolution from obesity to diabetes mellitus results from a succession of pathological events such as augmentation of the adipose mass leading to increased lipid oxidation; insulin resistance

which is noticed in obesity early is shown by euglycemic clamp, as a resistance to insulin mediates glucose storage and oxidation, blocking the glycogen cycle. Unused glycogen prevents further glucose storage leading to hyperglycemia which is the hallmark of diabetes mellitus.

The incidence of diabetes mellitus was higher in the females. Genetic inheritance or previous family history, obesity and advancing age was all positively correlated with the prevalence of diabetes mellitus in this region.

Conflict of interest statement

We declare that we have no conflict of interest.

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