

Full Length Research Paper

Glycosuria as a screening tool for diabetes mellitus in school children in Port Harcourt, Nigeria

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Abstract

Diabetes mellitus (DM) is the most common endocrine disorder of childhood and adolescence. Urine testing for glycosuria is an important screening tool for DM, especially in mass screening programmes. The screening of secondary school children in Port Harcourt for DM was done using glycosuria as a screening tool. Also, the relationship between risk factors for DM and glycosuria was determined. In November 2008, a cross sectional study of 1008 students aged 10 to 18 years from 12 secondary schools in Port Harcourt was carried out. Structured questionnaires were completed by the investigators. Urine glucose was determined using a dipstick urinary multistix strip. The prevalence of glycosuria was 0.7% with males having a higher prevalence (1.2%) than females (0.2%). Glycosuria was found more amongst children aged 16 to 17 years (1.1%), children from private schools (0.9%) and children in social class IV (3.3%). The relationship between elevated blood pressure and glycosuria was statistically significant. There was no statistically significant relationship between glycosuria and body mass index categories, waist hip ratio, acanthosis nigricans and family history of diabetes. Glycosuria occurs in apparently healthy secondary school children. As such, regular screening of secondary school children for DM using urine glucose testing should be incorporated into the school health programme.

Keywords: Glycosuria, diabetes, screening, secondary school children, Port Harcourt

INTRODUCTION

Diabetes mellitus (DM) is a common, chronic metabolic syndrome characterized by hyperglycemia as a cardinal biochemical feature (Alemzadeh et al., 2007). The major forms of diabetes are divided into those caused by deficiency of insulin secretion due to pancreatic β -cell damage (type 1 DM), and those that are consequences of insulin resistance occurring at the level of skeletal muscle, liver and adipose tissue, with various degrees of β -cell impairment (type 2 DM) (Alemzadeh et al., 2007). Type 1 DM is the most common endocrine-metabolic disorder of childhood and adolescence, with important consequences for physical and emotional development (Alemzadeh et al., 2007). Type 2 DM was previously thought to occur only in adults. However, there are increasing reports of type 2 DM in children globally

(Rosenbloom et al., 1999). The onset of type 2 DM is often insidious, without symptoms to alert the patient or clinician about the illness. Risk factors for childhood type 2 DM include obesity, overweight, family history, evidence of insulin resistance such as acanthosis nigricans, hypertension, dyslipidaemia and polycystic ovarian syndrome (American Diabetes Association, 1997)

DM may cause life threatening metabolic complications like diabetic ketoacidosis (Alemzadeh et al., 2007) which contributes to death in 1 out of 200 children presenting with DM (Schober et al., 1997). Diabetes is also an important risk factor for other leading causes of death such as coronary heart disease, congestive heart failure and cerebrovascular disease in adults. World Health Organization. Definition, diagnosis, and classification of diabetes mellitus and its complications

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(WHO, 1999). Individuals with DM confront lifestyle alterations that include an absolute daily requirement for exogenous insulin or oral hypoglycaemic drugs, the need to monitor their own glucose control, to pay attention to dietary intake and good hygiene (Alemzadeh et al., 2007). Diabetes mellitus is a major challenge to public health in the 21st century because of the cost of care of the increasing number of people with diabetes (Zimmet et al., 2001).

Currently, majority of the asymptomatic people who have diabetes mellitus have not been diagnosed and may remain undiagnosed for many years (Davis et al., 1997). The progression of the complications is worse when the illness starts from childhood. Identifying asymptomatic children early in the disease process will allow early institution of lifestyle changes and medical therapy of this disease to reduce its cost as well as to avoid enormous consumption of resources.

Screening for diabetes mellitus is an important measure in detecting the pre-diabetic stages and undiagnosed diabetes in order to avert pernicious effects of hyperglycaemia and associated complications (American Diabetes Association, 2003). Fasting plasma glucose is the preferred method of screening and World Health Organization's gold standard for confirmation of DM (Alemzadeh et al., 2007; American Diabetes Association, 2003). However, it is not favourable for non-hospital studies and not recommended for routine use. Urine testing for glycosuria is an important screening tool for DM. Glucose is usually detected when the renal threshold for blood glucose is exceeded (180 mg/dL; 10 mmol/L) (Alemzadeh et al., 2007). It is highly specific but less sensitive than most blood tests for DM. In population-based screening using quantitative urine dipstick in the USA, a "trace positive" dipstick result or greater has a reported sensitivity of 23-64% and specificity of 98-99% for DM (Modan and Harris 1994). In a high-risk adult population, quantitative assays of urine glucose achieved high sensitivity (81%) with high specificity (98%), comparable to both fasting plasma glucose and glycosylated protein assays (Modan and Harris 1994). It has been shown that screening for diabetes mellitus using urine glucose is invaluable in some clinical settings and has been utilized in mass screening of school children for DM (Afoke et al., 1992). It is particularly useful in resource poor countries in Sub Saharan Africa because it is simple, affordable, reliable, safe, feasible and requires less sophisticated technique and manpower (Van der Sande et al., 1999). In Japan, mass urine screening programme (for both type1 and 2 DM.) for school children has been mandatory since 1994. (Yokota et al., 2004). A study by Afoke et al (Afoke et al., 1992) on the prevalence and clinical picture of type1 DM in Nigerian Igbo school children aged 7-15 years was

able to identify 12 new diabetic cases with a crude prevalence rate of 0.33/1000 following screening with urine glucose. There is however no available information about screening for DM in children using glycosuria in Port Harcourt and other parts of Nigeria.

This study was carried out to raise awareness of DM especially type 2 DM and the risk factors for DM in children and also serve as a basis for developing health education and control programmes for prevention and treatment of DM as well as controlling the risk factors. Data generated from the study can serve as a reference standard for prevalence of glycosuria and other risk factors for DM in Nigerian adolescents using Port-Harcourt as a case study.

OBJECTIVES

To determine the prevalence of glycosuria in secondary school children aged 10-19 years in Port Harcourt City Local government Area, Nigeria. Also to determine the relationship between various risk factors for diabetes mellitus and glycosuria these children.

METHODS

In November 2008, a cross sectional study of 1008 students aged 10 to 19 years from 12 secondary schools in Port Harcourt was carried out. A total of 84 children were recruited from each secondary school. In each school, one arm per class was selected by simple random sampling. In the selected arm, 14 children were recruited by the simple random sampling of balloting. Structured questionnaires were completed by the investigators. Measurement of weight, height, waist-hip circumference and blood pressure was done and the school children were examined for acanthosis nigricans. The Body Mass index (BMI) was calculated using the formula $\text{weight (kg)/height (m)}^2$. Urine glucose was determined using a dipstick urinary multistix strip.

Children with glycosuria, risk factors for DM (obesity/overweight, family history, hypertension and acanthosis nigricans) and any abnormal findings in the urine were referred to Paediatric Endocrine Clinic of UPTH for follow up and management.

The data obtained were analysed using the Statistical Package for Social Sciences (SPSS) software version 14 and Epi Info version 6.04.

The approval of the Ethics and Research Committee of the UPTH was obtained before proceeding with the study. Permission was also obtained from the, Rivers State Ministry of Education and the Principals of the selected schools. Informed written consent was obtained

Table 1. General characteristics of study population

Gender	School Type		Total n (%)
	Private n (%)	Public n (%)	
Male	336 (50)	168(50)	504 (50)
Female	336 (50)	168 (50)	504 (50)
Social class			
I	92(13.7)	43(12.8)	135(13.4)
II	351(52.2)	131(39.0)	482(47.8)
III	189(28.1)	125(37.2)	314(31.2)
IV	34(5.1)	27(8.0)	61(6.0)
V	6(0.9)	10(3.0)	16(1.6)
Total	672(100)	336(100)	1008(100)

Table 2. Distribution of study group by age group and gender

Age (years)	Gender		Total n (%)
	Male n (%)	Female n (%)	
10-11	87 (17.3))	67 (13.3)	154 (15.3)
12-13	140 (27.8)	148 (29.4)	288 (28.6)
14-15	154 (30.5)	168 (33.3)	322 (31.9)
16-17	82(16.3)	94 (18.6)	176 (17.5)
18-19	41(8.1)	27 (5.4)	68 (6.7)
TOTAL	504(100)	504(100)	1008(100)

$\chi^2 = 4.56, P > 0.05$

from the parents /or guardians of the children recruited for the study.

RESULTS

General characteristics of study subjects

A total of 1008 children were recruited for the study. They comprised of 672 (66.7%) from private school and 336 (33.3%) from public school. There were equal number of males and females (50%) in both public and private schools (Table 1). More than half 443(65.9%) of the children in private schools and 174 (51.8%) in the public schools belonged to the upper socio economic class (Social Classes I and II). Forty(6%) of the children in private schools and 37(11%) of those in public schools belonged to the lower socio economic class.

Distribution of study population by age group and gender

The ages of the children ranged from 10 to 19 years with a mean of 14.25 ± 2.23 years. The mean age of males

was 14.23 ± 2.32 years, while that of females was 14.27 ± 2.13 years. The mean age difference between sexes was not statistically significant. t -value= -.283, $P > 0.05$. Table 2 shows the distribution of the subjects by age group and gender. The subjects were divided into 5 groups, 10-11, 12-13, 14-15, 16-17, and 18-19 years respectively. The highest number of the subjects belonged to the 14-15 year age group (31.9 %) while 18-19 age group had the least number of subjects (6.7 %). Male subjects were found more in the 10-11 and 18 -19 age groups while females were more in the 12-13, 14-15 and 16-17 age groups. The sex difference in the various age groups was not statistically significant $\chi^2 = 4.56, P > 0.05$.

Glycosuria

Prevalence of Glycosuria according to Gender

Table 3 shows the incidence of glycosuria. Seven (0.7%) subjects had glycosuria. These were 6 males (1.2%) and one female (0.2%). However, gender difference was not statistically significant. $\chi^2 = 2.302, P > 0.05$. Four (66.7%) out of the 6 males with glycosuria had 50mg of glucose in

Table 3. Prevalence of Glycosuria according to Gender

Gender	Glycosuria		
	Positive n (%)	Negative n (%)	Total n (%)
Male	6(1.2)	498(98.8)	504(100)
Female	1(0.2)	503(99.8)	504(100)
Total	7(0.7)	1001(99.3)	1008 (100)

$$\chi^2 = 2.302, P > 0.05$$

Table 4. Age characteristics of subjects with glycosuria

Age (years) Glycosuria	10-11 n (%)	12-13 n (%)	14-15 n (%)	16-17 n (%)	18-19 n (%)	Total n (%)
Positive (%)	1 (0.6)	2(0.7)	2(0.6)	2(1.1)	0	7(0.7)
Negative (%)	153 (99.4)	286(99.3)	320(99.4)	174(98.9)	68(100)	1001(99.3)
Total	154(100)	288(100)	322(100)	176(100)	68(100)	1008(100)

Fisher's Exact Test = 0.145, p > 0.05

Table 5. Relationship between type of School, Social Class and Glycosuria

School Type	Glycosuria			P value
	Positive n (%)	Negative n (%)	Total n (%)	
Private	6 (0.9)	666 (99.1)	672 (100)	> 0.05
Public	1(0.3)	335(97.3)	336 (100)	> 0.05
Total	7(0.7)	1001(99.3)	1008(100)	
$\chi^2 = 0.45$	P > 0.05			
Social Class				
I	2(1.5)	133(98.50)	135(100)	> 0.05
II	1(0.2)	481(99.8)	482(100)	> 0.05
III	2(0.6)	312(99.4)	314(100)	> 0.05
IV	2(3.3)	59(96.7)	61(100)	> 0.05
V	0(0)	16(100)	16(100)	> 0.05
Total	7(0.7)	1001(99.3)	1008(100)	

Fisher's exact test = 4.12, P > 0.05

the urine while 2(37.3%) had 100mg of glucose in the urine. The only female subject with glycosuria had 50mg of glucose in the urine.

Age characteristics of subjects with glycosuria

The mean age of the male subjects with glycosuria was higher (14.50±2.35years) than those without glycosuria (14.23±2.32years). In the females, the mean age of the subjects without glycosuria was (14.27±2.14 years) while the only female with glycosuria was 13.00 years. Overall the total mean age for those with glycosuria (14.29±2.22years) was higher than the subjects without glycosuria (14.25±2.22years). This age difference was however not statistically significant t-value=. 045, P>0.05

As shown in table 4, none of the subjects within the age group 18-19 years had glycosuria. Glycosuria was seen more among subjects 16-17 years (1.1%). The relationship between age and glycosuria was not statistically significant $\chi^2=0.145$, P> 0.05.

Relationship between type of school, socio economic class and glycosuria

Table 5 shows the relationship between the type of school, Social Class of subjects and glycosuria. Out of the 7 subjects with glycosuria 6 (0.9%) were from 5 private schools while 1 (0.3%) was from a public school. This difference was however not statistically significant χ^2

Table 6. Mean Weight, Height and BMI of subjects with glycosuria

Parameter	Glycosuria				Total Positive Mean(\pm SD)	Total Negative Mean(\pm SD)	Pvalue
	Positive		Negative				
	Male Mean(\pm SD)	Female Mean*	Male Mean(\pm SD)	Female Mean(\pm SD)			
Weight(kg)	55.50 \pm 13.08	57.00	47.38 \pm 11.36	49.16 \pm 10.8	55.71 \pm 11.95	48.33 \pm 14.14	>0.05
Height(m)	1.65 \pm 0.11	1.65	1.57 \pm 0.12	1.57 \pm 0.77	1.65 \pm 0.10	1.57 \pm 0.10	<0.05
BMI(kg/m ²)	20.26 \pm 3.19	20.95	18.95 \pm 2.78	20.00 \pm 3.67	20.36 \pm 2.93	19.49 \pm 3.30	>0.05

* One female had no standard deviation

Table 7. Mean WC, HC & WHR of subjects with glycosuria

Parameter	Glycosuria				Total Positive Mean(\pm SD)	Total Negative Mean(\pm SD)	Pvalue
	Positive		Negative				
	Male Mean(\pm SD)	Female Mean*	Male Mean(\pm SD)	Female Mean(\pm SD)			
WC(cm)	71.00 \pm 5.87	83.00	67.68 \pm 7.16	70.33 \pm 8.11	72.71 \pm 7.02	69.04 \pm 7.76	>0.05
HC(cm)	75.00 \pm 3.46	89.00	73.45 \pm 8.95	78.59 \pm 10.76	77.00 \pm 6.16	76.04 \pm 10.20	>0.05
WHR	0.95 \pm 0.05	0.93	0.93 \pm 0.06	0.90 \pm 0.08	0.94 \pm 0.05	0.91 \pm 0.74	>0.05

= 0.45, $P > 0.05$. The prevalence of glycosuria was higher 2(3.3%) in the subjects with Social Class IV. However, the relationship between glycosuria and Social Class was not statistically significant $\chi^2 = 4.12$, $P > 0.05$. None of the subjects from lower social class V had glycosuria.

Anthropometry of subjects with glycosuria

Table 6 shows the mean weight, height and BMI of subjects with and without glycosuria. The mean weight of the subjects with glycosuria was higher than those without glycosuria in the males. The weight of the only female with glycosuria was 57.0kg which was higher than the mean weight of the females without glycosuria. The difference in the weight between all the subjects with glycosuria (55.71 \pm 11.95kg) and those without glycosuria (48.33 \pm 14.14kg) was not statistically significant t -value=1.763, $P > 0.05$. The mean height of the subjects with glycosuria (1.65 \pm 0.10m) was significantly higher than those without glycosuria (1.57 \pm 0.10m) t -value=2.001, $P < 0.05$. The mean BMI in the males as well as females subjects with glycosuria was higher than those without glycosuria. The overall mean BMI between those with glycosuria (20.36 \pm 2.93) and those without glycosuria (19.49 \pm 3.30) was not statistically significant t -value=0.704, $P > 0.05$.

Mean Waist Circumference, Hip Circumference and Waist Hip Ratio of subjects with glycosuria

Table 7 shows the mean waist and hip circumferences

(WC&HC) as well as the mean waist hip ratio (WHR) of the subjects with and without glycosuria. The WC, HC and WHR of the male subjects with glycosuria were higher than those without glycosuria. The WC, HC and WHR of the only female with glycosuria were higher than those of the female subjects without glycosuria. However, there was no statistical significant difference between the mean WC, HC and WHR of the subjects with glycosuria and those without glycosuria. t -value = 1.257, 0.251 and 1.110 for WC, HC and WHR respectively, $P > 0.05$.

Relative frequency of risk factors for DM in subjects with glycosuria

Table 8 shows the relative frequency of risk factors for DM in subjects with glycosuria. Glycosuria was found more in subjects with elevated blood pressure 3(4.1%) followed by subjects with positive family history of DM 2(1.7%). The only subject with acanthosis nigricans had no glycosuria.

DISCUSSION

This study showed that the incidence of glycosuria among secondary school children in Port Harcourt was 0.7%. The incidence of glycosuria noted in this study was higher than that reported by Bai et al., (Bai et al., 1991) in their study of glycosuria and diabetes mellitus in children and adolescents in South India. They reported an incidence rate of 0.038% in 10,513 children aged between 3 and 20 years. This difference could be due to

Table 8. Relative frequency of risk factors for DM in subjects with glycosuria

Risk factors	Glycosuria		
	Positive n (%)	Negative n (%)	Total n (%)
Obesity/overweight(BMI)	1(1.0)	98(99.0)	99(100)
Abnormal (WHR)	1(0.3)	383(99.7)	384(100)
Family history of DM	2(1.7)	113(98.3)	115(100)
Elevated blood pressure	3(4.1)	71(95.9)	74(100)
Acanthosis nigricans	0	1(100)	1(100)

the larger sample size studied with age groups including children below 5 years who are less likely to develop diabetes unlike in the present study that involved only adolescents. Racial difference could also play a part in the difference in the prevalence rate of glycosuria noted in the Indian study. A similar incidence of 0.8% was however reported by Yang et al (Yang et al., 2004) in China among women aged 40-70 years although, the study involved only females that are older who are more likely to have type 2 DM than the subjects in the present study.

The prevalence of glycosuria was higher in males (1.2%) than females (0.2%) with a ratio of 6:1. The higher prevalence of glycosuria in males in this study supports previous studies that noted a higher prevalence of DM in males (Wild et al., 2000; Nyenwe et al., 2003; Dahiru et al., 2008). Nyenwe and colleagues in Port Harcourt reported a crude prevalence rate of type 2 DM in adults as 6.8% with male and female rates of 7.7 and 5.7% respectively. Similarly, Dahiru et al., in their study of the prevalence of diabetes in a semi urban community in Northern Nigeria reported a prevalence rate of 2.0% with all the diabetic cases being males. No obvious explanation has been given for the higher rate of diabetes in males in Nigeria and it may be assumed that genetic and hormonal differences between the males and females may be contributing factors. Other studies in Asia have however reported a higher incidence of DM in females (Urakami et al., 2005; Wei et al., 2003). In Japan, the overall annual incidence of 2.63/100,000 of type 2 DM was reported in school children in Tokyo Metropolitan area from 1972 to 2002 with a male-to-female ratio of 1.0:1.2 (Urakami et al., 2005). Wei et al in Taiwan, reported that the overall rates of newly identified DM were 9/100,000 boys, and 15.3/100,000 girls. The higher female preponderance reported in their studies was attributed to higher prevalence of obesity among the females. These children were all identified through urine glucose screening (Urakami et al., 2005; Wei et al., 2003). The prevalence of glycosuria has been noted to be highly predictive of development of DM and hence its comparison with the present study.

The mean age of the males with glycosuria was 14.5±2.35 years while the only female with glycosuria was 13 years. Glycosuria was found more in subjects aged 16-17 years. Although there is paucity of data to

compare with this finding, (Wei et al., 2003) in Taiwan however noted that the highest rate of new cases of DM in school children identified through screening for glycosuria occurred within 13-18 years which is the peak of adolescence. During this period, there is heightened sex steroids and growth hormone which are antagonistic to insulin production and action (Alemzadeh et al., 2007). Children in private schools had more subjects with glycosuria than those in public schools, although this difference was not statistically significant. Generally, children in private school are more likely to come from higher socio economic class but this trend is not apparent in this study area where both low and high social classes attend private schools. The highest prevalence of glycosuria in this study was from lower social class IV. The finding that glycosuria was more in children from lower socio class IV supports previous studies that found a higher incidence of diabetes and its complications among people in lower socio economic classes than people from higher economic classes (Koskinen et al., 1996; Pill et al., 1995). Lack of healthy diet, high prevalence of infections which may trigger pathogenesis of type 1 DM is more apparent in children in lower socio economic class. Also in the USA, obesity a risk factor for type 2 DM, is reported more in poor black Americans due to poor dietary habit (Olson 1999).

In this study, subjects with glycosuria were observed to have higher mean weight than those without glycosuria. This finding is comparable with previous reports that increasing weight is a risk factor for hyperglycaemia and diabetes because of its predisposition to obesity and insulin resistance (Alemzadeh et al., 2007; Chang et al., 2006; Unwin et al., 1997). The mean height of the subjects with glycosuria was significantly higher than the subjects without glycosuria. This finding is similar to other studies which have also shown that children who became diabetic were considerably taller than non-diabetic children. In some children this increase in height was evident up to three years before diagnosed diabetes. It was assumed that the height increase may be caused by changes in metabolism that occur before the onset of diabetes (John et al., 2002; Price and Burden, 1992). Increased stature may represent an phenomenon of the islet-cell hyperplasia and hyperinsulinaemia, which characterize the preclinical stages of diabetes. (Price and Burden,

1992; Homo-Delarche 1997). The mean BMI of subjects with glycosuria was also higher than those without glycosuria. This finding is similar to studies that found a higher prevalence of hyperglycaemia and diabetes among those with higher BMI (Chang et al., 2006; Unwin et al., 1997). BMI is directly related to obesity which is a major risk factor for type 2 DM due to insulin resistance leading to hyperinsulinaemia and finally to type 2 DM (Alemzadeh et al., 2007). The mean waist circumference, Waist Hip Ratio (WHR) of all the subjects with glycosuria was higher than those without glycosuria. Studies have also shown (Caro, 2009) that these variables like the BMI are also directly related to obesity, a major risk factor for type 2 DM (Unwin et al., 1997).

Glycosuria was found in only 1.6% of the overweight and none of the obese subjects. The relationship of glycosuria with overweight and obesity was however not statistically significant. Only one (0.3%) subject with abnormal WHR had glycosuria. The lack of association between glycosuria and obesity may be as a result of delayed development of insulin resistance (in the presence of adequate beta cell function) which can manifest later in adult life if appropriate measures to control obesity are not instituted. A long term follow up may however determine the period between the onset of insulin resistance and glycosuria in obese and overweight children. The lack of finding of significant association between glycosuria and obesity may also indicate that although obesity is a risk factor for DM, but not all obese children may develop DM. Similarly, Feneli et al (Feneli et al., 2008) in Greece did not find any significant relationship between obese children and hyperglycaemia or DM. The finding of approximately one third of the obese children in that study having hyperinsulinism points to the fact that insulin resistance is the earlier abnormality in glucose homeostasis in obesity. (Feneli et al., 2008) Obese and overweight children are more likely to be overweight adults and are at increased risk for type 2 DM and other cardiovascular complications as adult (Alemzadeh et al., 2007; Guo et al., 1994). Several studies have suggested that preventing obesity may be important in reducing the development of type 2 DM (American Diabetes Association, 2000; Bloomgarden, 2004). The prevention of obesity on the long term may require very early interventional strategies such as regular exercise, dietary control instituted in childhood and adolescence (Alemzadeh et al., 2007; Helmich, 1991).

Family History of DM was found in 115 (11.4%) of subjects in this study. The family history of DM was either in the father or mother but none had in both parents. This result was higher than that reported by (Puepet and Ohwovoriole, 2008) (3.5%) in a non-diabetic population aged 15 years and above in Jos. In this present study, 2(28.6%) out of the 7 subjects with glycosuria had a positive family history of DM. Although this study was on

glycosuria, it supports the finding in previous studies that family history of DM is seen in only 23.8% of diabetic probands. Puepet and Chuhwak (2002), Osuntokun et al (Osuntokun et al., 1971) in Ibadan however, reported a lower prevalence of family history of DM even among probands of type 2 DM. Low prevalence of family history of DM in subjects with diabetic probands may suggest that genetic factor may not be an important risk factor for development of type 2 DM in these populations. Ignorance about DM and the fact that Africans are usually reluctant to reveal family history of illness may also be responsible for the low family history of diabetes (Puepet and Ohwovoriole, 2008) It has been reported that offsprings of fathers with type 1 DM are nearly three times more likely to have type 1 DM than the offsprings of affected mothers (Anochie et al., 2004). In another study, maternal and paternal DM conferred equivalent risk for occurrence of type 2 DM in offspring (Meigs et al., 2000).

Acanthosis nigricans (AN) has been considered to be a predictive marker for type 2 diabetes. It is one of the signs of insulin resistance often associated with obesity. In this study, only one (0.1%) subject, a female had AN and she was also found to be obese. Nguyen et al (Nguyen et al., 2001) in the USA, had reported AN in overweight subjects. In their study of 139 children, 35 (25%) had AN with a greater body weight than the children without AN. However, the only subject in this study with AN had no glycosuria presently, but this may precede the development of type 2 DM in future which could be prevented if properly managed. The subject with AN however, had more than one risk factors for DM (elevated BP, obesity and family history of DM) and will benefit from a follow up. There is however paucity of data on AN among children in Nigeria as well as other regions in sub-Saharan Africa to compare with this study. This paucity of data may either be due to rarity of AN in these regions, or mis-diagnosis because its characteristic appearance could be misdiagnosed as a skin disease or infection rather than a metabolic disorder.⁴⁰ Furthermore, AN may lighten over time if the underlying insulin resistance is adequately treated making the diagnosis difficult.

This study was able to identify children with glycosuria as well as identifying those with various risk factors for DM (overweight, obesity, positive family history of diabetes, hypertension and acanthosis nigricans) and their association with glycosuria. All the children with glycosuria as well as those with risk factors for DM were referred to a paediatric endocrinologist for further evaluation.

REFERENCES

- Acanthosis nigricans and insulin resistance [online] (2002). [cited 2008 Nov 18];62(2). Available at: URL:www.tdh.state.tx.us/phpep/.
 Afoke AO, Ejeh NM, Nwonu EN, Okafor CO, Udeh NJ, Ludwingsson J (1992). Prevalence and clinical picture of IDDM in Nigerian Igbo Schoolchildren. *Diabetes Care* 15:1310-1312.

- Alemzadeh R, Wyatt DT (2007). Diabetes mellitus in children. In: Kliegman RM Behrman RE, Jenson HB, Stanton BF, editors. *Nelson Textbook of Paediatrics*. 18th ed. Philadelphia: Saunders. pP. 2404-24 31.
- American Diabetes Association (ADA) (1997). Report of the Expert Committee on the diagnosis and classification of diabetes mellitus. The Expert Committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*. 20:1183-1197.
- American Diabetes Association (2003). Position statement. Screening for type 2 Diabetes. *Diabetes Care*. 26 Suppl1:S21-S24.
- American Diabetes Association (2000). Type 2 diabetes in children and adolescents. *Diabetes Care*. 23:381-92.
- Anochie IC, Alikor EA, Unachukwu CN (2004). Diabetes mellitus in an infant: an unusual presentation. *Nig J Clin Prac* 7: 97-98.
- Bai PV, Krishnaswami CV, Chellamariappan M, Kumar GV, Subramaniam JR (1991). Glycosuria and diabetes mellitus in children and adolescents in south India. *Diabetes Res Clin Pract* 13:131-5.
- Bloomgarden ZT(2004). Type 2 diabetes in the young: the evolving epidemic. *Diabetes Care* 27: 998-1010.
- Caro JF (2002). Definitions and classification of obesity (online). (cited 2009 Jun 23); Available from: URL:<http://www.endotext.org/obesity1/obesityframe1.htm>
- Chang LY, Li HY, Wei JN, Chuang LM (2006). Type 2 diabetes and obesity in children and adolescents: experience from studies in Taiwanese population. *Curr Diabetes Rev* 2:185-93.
- Dahiru T, Jibo A, Hassan AA, Mande AT (2008). Prevalence of diabetes in a semi –urban community in Northern Nigeria. *Nig J Med* 17:414-16.
- Davis TM, Stratton IM, Fox CJ, Holman RR, Turner RC. U.K (1997). Prospective diabetes study. Effect of age at diagnosis on diabetic tissue damage during the first 6 years of NIDDM. *Diabetes Care* 20:1435-441.
- Feneli K, Aspasia F, Erini P, Stefanos M (2008). Prevalence of insulin resistance syndrome among Greek obese children adolescents. *Child J Med* 3:30-33.
- Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM (1994). The predictive value of childhood BMI for overweight at age 35 years. *Am J Clin Nutr* 59:810-19.
- Helmich SP, Ragland DR, Leung RW, Paffenbarger RS. Physical activity and reduced occurrence of NIDDM. *N Engl J Med* 1991;325:147-151.
- Homo-Delarche F. B-cell behavior during the prediabetic stage. *Diabetes Metab*.1997;23:181-94
- John H, Diliberti JH, Carver K, Parton E, Totka J, Mick G et al., (2002). Stature at time of diagnosis of type 1 diabetes mellitus. *Pediatrics* 109:479-483.
- Koskinen SVP, Martelin TP, Valkonen T (1996). Socioeconomic differences in mortality among diabetic people in Finland: five year follow up. *BMJ* 313:975-8.
- Meigs JB, Cupples LA, Wilson PWF (2000). Parental transmission of type 2 diabetes: The Framingham offspring study. *Diabetes* 49: 2201-2207.
- Modan M, Harris MI (1994). Fasting plasma glucose in screening for NIDDM in the U.S. and Israel. *Diabetes Care* 17:436-9.
- Nguyen TT, Pathomvanich A, Uwaifo GI, Sebring NG, Reynolds JC, Yanovski JA (2001). Relation of acanthosis nigricans by hyperinsulinemia and insulin sensitivity in overweight African American and white children. *J Pediatric* 2001; 138: 474-480.
- Nyenwe EA, Odia OJ, Ihekwa AE, Ojule A, Babatunde S (2003). Type 2 diabetes in adult Nigerians: a study of its prevalence and risk factors in Port Harcourt, Nigeria. *Diabetes Res Clin Pract*. 62:177-185.
- O'Dea K, Patel M, Kubisch RN, Hopper J, Traisnede MS (1993). Obesity, diabetes and hyperlipidaemia in a central Australian aboriginal community with a long history of acculturation. *Diabetes Care* 16:1004-1010.
- Olson CM (1999). Nutrition and health outcomes associated with food insecurity and hunger. *J Nutr* 129;521-524.
- Osuntokun BO, Akinkugbe FM, Francis TT, Reddy S, Taylor GOL (1971). Diabetes mellitus in Nigeria. A study of 832 patients. *West Afr Med J* 20:295-312.
- Pill R, Peters TJ, Robling MR (1995). Social class and preventive health behaviour: a British example. *J Epidemiol Com Health* 49:28-32.
- Price DE, Burden AC (1992). Growth of children before onset of diabetes. *Diabetes Care* 15:1393-1395.
- Puepet FH, Chuhwak EK (2002). Study of family history among parents and siblings of Nigerian diabetics in Jos: A preliminary report. *J Trop Med* 4:6-9.
- Puepet, FH, Ohwovoriole, AE (2008). Prevalence of risk factors for diabetes mellitus in a non-diabetic population in Jos, Nigeria. *Nig J Med* 17:71-74.
- Rosenbloom AL, Joe JR, Young RS, Winter WE (1999). Emerging epidemic of type 2 diabetes in youth. *Diabetes Care* 22:345-54.
- Schober E, Schneider U, Friedl HP, Unsinn K (1997). Early mortality in childhood diabetes in Austria: a population based cohort study. *Eur J Pediatr* 156:15-17.
- Unwin N, Harland J, White M, Bhopal R, Winocour P, Stephenson P et al., (1997). Body mass index, waist circumference, waist-hip ratio, and glucose intolerance in Chinese and European adults in Newcastle, UK. *J Epidemiol Com Health* 51:160-166.
- Urakami T, Kubota S, Nitadori Y, Harada K, Owada M, Kitagawa T (2005). Annual incidence and clinical characteristics of type 2 diabetes in children as detected by urine glucose screening in the Tokyo Metropolitan area. *Diabetes Care* 28:1876-1881.
- Van der Sande MA, Walraven GE, Bailey R, Rawley JT, Banyan WA, Nyan OA (1999). Is there a role for glycosuria testing in sub-Saharan Africa? *Trop Med Int Health* 4:506-513.
- Wei JN, Sung FC, Lin CC, Lin RS, Chiang CC, Chuang LM (2003). National surveillance for type 2 diabetes mellitus in Taiwanese children. *JAMA* 290:1345-1350.
- Wild S, Roglic G, Green A, Sicree R, King H (2004). Global prevalence of diabetes estimates for the year 2000 and 2030. *Diabetes Care* 27:1047-1053.
- World Health Organization (1999). Definition, diagnosis, and classification of diabetes mellitus and its complications. Report of WHO consultation. Part1: diagnosis and classification of diabetes. Department of non-communicable disease surveillance, Geneva.
- Yang G, Shu XO, Jin F, Elasy T, Li HL, Huang F et al. (2004). Soyfood consumption and risk of glycosuria: a cross-sectional study within the Shanghai women's health study. *Eur J Clin Nutr* 58:615-20.
- Yokota Y, Kikuchi N, Matsuura N (2004). Screening for diabetes by Urine glucose testing at School in Japan. *Pediatr Diabetes* 5:212-218.
- Zimmet P, Alberti KG, Shaw J (2001). Global and societal implications of the diabetes epidemic. *Nature* 414:782-787.