

## Survey of Serum Levels of Magnesium in Type 2 Diabetics Attending Clinics in Bayelsa State

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### Abstract

The aim of this study was to determine the serum magnesium levels in Type 11 diabetics attending clinics in Bayelsa state. A randomized, cross sectional study was adopted with 50 healthy non diabetic subjects, mainly blood donors (mean aged 48.88±1.46) as control group and 100 diabetics attending clinics in Bayelsa state (mean aged 49.42±2.42) as study group. Blood samples were obtained after at least 8 hours of fasting in plain tubes and serum glucose levels were determined by glucose oxidase method and serum magnesium using xylydyl blue reagent. Height and weight were measured using a tapeline to the nearest 0.5cm and a bath scale to the nearest 0.1kg respectively. Body mass index (BMI) was calculated as weight (kg)/height (m)<sup>2</sup>. Statistical analyses were conducted using graph prism pad 5.0 statistical software versions and Microsoft excel 2003. Mean serum magnesium value among diabetics was 1.18± 0.06mmol/l and 0.7± 0.06mmol/l among controls (p <0.05). Magnesium values were statistically significant between sexes (p<0.05) in both diabetics and non-diabetics. The study established that hypomagnesaemia was not found in the diabetics attending clinic in Yenagoa. Therefore we recommend that serum magnesium levels be determined in diabetics attending clinic periodically as symptoms may occur long before it can be established in the laboratory.

**Keywords:** type 2 diabetes, serum magnesium, fasting blood sugar (FBS), body mass index (BMI).

### 1. INTRODUCTION

It has been observed for some time worldwide, that there has been a reduction in micronutrients ingestion among the population due to changes in feeding habits (Lopez et al, 2004). Magnesium is one of the most abundant intracellular ions with an essential role in fundamental biological reactions, whose deficiency provokes biochemical and symptomatic alterations in the human organisms (Satis et al, 2000; Detra et al, 2002). Diabetes mellitus, characterized by metabolic disorders related, to high levels of serum glucose (American Diabetic Association, 2004), is perhaps the most associated disease to magnesium depletion in the intra and extra cellular components.

Low serum magnesium level have been related to abnormalities in insulin actions and decrease in insulin secretions, both of which are involved in the patho-physiology of Type 2 diabetes mellitus (Rasic et al, 2004). In addition, hypomagnesia is also related to the main risk factors for the development of diabetes, such as obesity, low-grade chronic inflammatory syndrome, aging, retinopathy, and thrombosis (Fernando et al, 2007). Since customary diet is the main source of magnesium and several prospective interventions and cross sectional studies have demonstrated a strong association between low serum magnesium levels and metabolic disorders of glucose and insulin, it has been hypothesized that low magnesium intake could be a risk factor for the development of type 2 diabetes mellitus (Lopez et al, 2004) as well as resulting in complications of diabetes (Correa et al, 2003).

Prevalence of diabetes mellitus in adult worldwide was estimated to be 4.0% in 1995 and expected to rise to 5.4% by the year 2025 i.e. from 135 million to 300 million. The estimates also indicate that diabetes will rise at 0.42% in the developed countries and 1.7% in the developing countries thereby imposing a substantial health burden (Fernando et al, 2007). Despite the HIV epidemic, the total number of people with diabetes in the sub-saharan region is expected to grow because of changing demography. A concerted multi-sectoral effort will be critical to ensuring improvement in healthcare delivery for people with diabetes in the region (Levitt, 2008).

Magnesium supplementation prevents the development of diabetes in rats (Balon et al, 1995). This is however, inconsistent with dietary intervention studies with magnesium as only a modest response reported on glycaemic control (Al-Delaimy et al, 2004; Song et al, 2004). Although the mechanism involving diabetes mellitus and hypomagnesaemia is unclear, magnesium supplementation did not reduce diabetic risk, it has a beneficial effect in the action of insulin and glucose metabolism (Lopez et al, 2004). According to Takaya et al, (2003) and Barbagallo et al, (2003), a poor intracellular magnesium concentration, as found in type 2 diabetes, may result in defective tyrosine-kinase activity at the insulin receptor level and exaggerate intracellular calcium ion concentration. Both events are responsible for the impairment in insulin action and a worsening of insulin in type 2 diabetic patients.

The almost universal involvement of magnesium in a wide variety of cellular processes critical to glucose metabolism, insulin action and cardiovascular functions has been well appreciated. The incidence of sub clinical magnesium deficiency is common in diabetes and cardiovascular disorders. However, limited attention has been drawn to the impact of magnesium deficiency on late diabetic complications, including cardiovascular disorders (Meludu et al, 2001). Until recently, the function of magnesium in biological processes was largely ignored to the point where it was described as the 'forgotten' ion (Meludu et al, 2001). Magnesium is primarily regarded as calcium antagonist and thus, alterations of intracellular or extracellular magnesium concentration may affect cell function through its effect on calcium handling (Chetan et al, 2002). Levels of magnesium in the plasma of healthy people are remarkably constant, being on the average of 1.7-2.4mg/dl (0.7-1.0mmol/l) (Chetan et al, 2002). It has been estimated that refining and processing of food causes a substantial loss of magnesium (Saris et al, 2000). For example, the refining and processing of wheat to flour, rice to polished rice and corn to starch droplets reduced magnesium concentration by 82, 83, and 97% respectively (Marrier et al, 1986; Popkin, 2001). Thus, modern food processing technology partially explains why a significant segment of the population has intake of magnesium below the recommended dietary amounts and may be predisposed to chronic, latent magnesium deficiency (Chetan et al, 2002). Drinking water on the other hand, remains an important source of magnesium. However, other factors including acid rain have caused a reduction in the magnesium level in the eco-system by causing an exchange with aluminum in the soil. Intensive farming, has also led to a reduction in magnesium concentration in the food chain (Classen et al, 1994).

According to World Health Organization (2002) estimates, about 60% of deaths in the world are now caused by non-communicable diseases. In 2005, an estimated 17.5 million people died of CVD representing 30% of all global deaths of which 80% were from low- and middle-income countries (WHO, 2007). By 2020, studies indicate that mortality by CVD is expected to increase by 120% for women and 137% for men (Yach et al, 2004). These findings highlight the need to explore the nature and magnitude of CVDs and other non-communicable diseases in developing countries.

Sub-Saharan Africa (SSA), consisting of those countries that are fully or partially located south of the Sahara Desert, are currently experiencing one of the most rapid epidemiological transitions characterized by increasing urbanization and changing lifestyle factors (Fezeu et al, 2006), which in turn have raised the incidence of NCDs. Studies indicate that urbanization and economic development have also led to the emergence of a nutritional transition characterized by a shift to a higher caloric content diet and/or reduction of physical activity (Popkin, 2003). Together, these transitions create enormous public health challenges, and failure to address the problem may impose significant burden for the health sector and the economy of sub-Saharan African countries (Asfaw, 2005).

In countries such as Nigeria, the prevalence of chronic diseases is increasing, while the threat of communicable and poverty-related diseases (malaria, infant mortality, cholera, malnutrition) still exists (Yach et al, 2004; Bonita et al, 2007; Kengne et al, 2009). This double burden of communicable and chronic NCDs has long-term public health impact as it undermines healthcare systems (Yach et al, 2004). Sub-Saharan African countries, similar to most developing countries, often do not have the public health infrastructure and finances to address both communicable and poverty-related illness and behavior/chronic related illnesses (Yach et al, 2004). In addition, there is reluctance on the part of health funding agencies and policy makers to divert scarce resources away from communicable diseases into other areas of disease burden, such as NCDs (Bonita et al, 2007; Unwin et al, 2001). Also, evidence suggests that the increasing burden of chronic diseases has grave consequences because very few people will seek treatment, leading to high morbidity and mortality rates from potentially preventable diseases (Duda et al, 2007). The objectives of this work are

1. Determine the serum magnesium levels in diabetics attending clinics in Bayelsa state.
2. To compare the serum magnesium values between diabetics and apparently healthy non diabetics in Bayelsa State.

## **2. MATERIAL AND METHOD**

### **2.1 Study Design**

The study design adopted is a cross sectional survey design which enables the researcher to randomize the study population.

### **2.2 Study Population**

50 healthy subjects (mainly blood donors) aged between 25 and 50 years with no history of diabetes as control group and 100 patients with diabetes attending clinics in Bayelsa state aged between 30 and 60 years as study group.

Thus, patients who met the inclusion and exclusion criteria were enrolled in the study. Inclusion criteria were -

- (a) People initially diagnosed with diabetes and met the W.H.O. criteria for diagnosis.
- (b) Patient(s) with stable disease without need for hospital admission for 3 months prior to assessment.

- (c) Diabetics on diet control only without late diabetic manifestations, not on diuretics or antibiotics and without alcohol consumption.
- (d) Diabetics with an established disease for at least 1 year.

Exclusion criteria were as follows:

- (a) Diabetics with an obvious damage to the brain or nervous system or any other concomitant disease that could affect the nervous system.

All patients (control and study group) were given a questionnaire on socio economic, demographic and clinical information after an informed consent.

Anthropometric determinations (weight and height) were measured using standard techniques and body mass index (BMI) calculated.

### 2.3 Measurements

Blood samples were obtained after at least 8 hours of fasting in sodium fluoride tubes and glucose analysis was done on the same day. Plasma glucose levels were determined by the glucose oxidase method and serum magnesium using xylydyl blue reagent.

#### 2.3.1 Covariates

Height and weight were measured with the subject standing using a tapeline to the nearest 0.5cm and a bath scale to the nearest 0.1kg respectively. Body mass index (BMI) was calculated as weight (kg)/height (m)<sup>2</sup>.

### 2.4 Sample Collections

Venous blood samples (5mls) of patients and controls were collected into plain tubes using standard aseptic conditions from participants who voluntarily agreed to participate after seeking their consent, and were allowed to clot, retracted and centrifuged at 3000rpm (revolution per minute) for 5 minutes without delay. The serum was separated into other two labeled clean, plain serum bottles avoiding haemolysis for serum glucose and serum magnesium assays.

### 2.5 Statistical Methods

Statistical analyses were conducted using graph prism pad 5.0 statistical software versions and Microsoft excel 2003. A two-way ANOVA was used to analyse magnesium values, fasting blood sugar (FBS), age and sex at p value <0.05 significant level between diabetics and control groups.

## 3. RESULTS

**TABLE 1: SEX DISTRIBUTIONS AMONG DIABETICS AND CONTROLS**

SEX	DIABETICS		CONTROLS	
	NO	%	NO	%
MALE	42	42	25	50
FEMALE	58	58	25	50
TOTAL	100	100	50	100

Table 1 shows the percentage of participants according to their sexes in the study, with diabetics having 42% males and 58% females while the controls have 50% for males and females respectively.

**TABLE 2 BIOCHEMICAL AND PHYSICAL PARAMETERS OF DIABETICS AND CONTROL**

	AGE	FBS (mmol/L)	Mg (mmol/L)	height(M)	weight(Kg)	BMI
DIABETICS	48.88 ± 1.46	5.96 ± 0.32	1.18 ± 0.06	1.62 ± 0.01	70.54 ± 1.3	26.75 ± 0.41
CONTRL	49.42 ± 2.42	5.59 ± 0.38	0.7 ± 0.06	1.61 ± 0.02	69.5 ± 1.8	26.64 ± 0.59
P-VALUE	0.22765812	0.43263972	<b>9.02858E-09</b>	<b>0.016572</b>	0.134699816	0.311063564

Table 2 shows the biochemical and physical parameters of the 100 diabetic patients and the 50 non-diabetic (control) subjects. Mean age was 48.88 and 49.42 in diabetics and controls respectively with no significant difference between their ages (p>0.05). Patients' BMI was slightly higher than the controls, but there was no statistical difference (p>0.05). Fasting blood sugar (FBS) level was higher among diabetics than the non-diabetics, although it was not statistically significant (p>0.05). Serum magnesium level was statistically significant among diabetics and the controls (p<0.05) with values higher among diabetics.

**TABLE 3 BIOCHEMICAL AND PHYSICAL PARAMETERS OF DIABETIC MALE SUBJECTS AND CONTROLS**

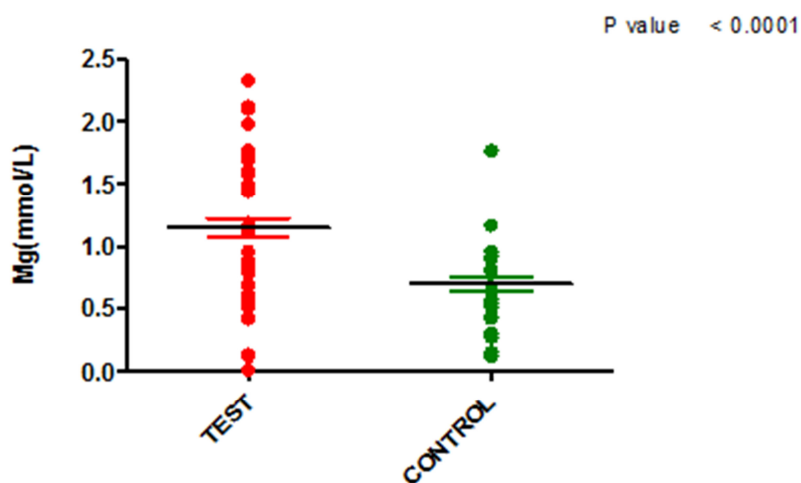
		<i>FBS</i>	<i>MG</i>			
	<i>AGE</i>	<i>(MMOL/L)</i>	<i>(MMOL/L)</i>	<i>HEIGHT(M)</i>	<i>WEIGHT(KG)</i>	<i>BMI</i>
<b>MALE</b>	52.07 ± 2.28	5.69 ± 1.26	1.22 ± 0.50	1.66 ± 0.07	72.38 ± 1.40	26.21 ± 0.63
<b>CONTRL</b>	42.4 ± 2.18	5.27 ± 1.14	0.78 ± 0.46	1.65 ± 0.06	71.4 ± 1.36	26.64 ± 0.59
<b>P-VALUE</b>	0.227658118	0.432639716	<b>1.21E-06</b>	<b>0.016572</b>	0.134699816	0.311063564

Table 3 shows comparison of mean values of the biochemical and physical parameters in male diabetics and male control subjects. There was no significant difference between their ages and BMI ( $p > 0.05$ ). Fasting blood sugar levels were lower in the control group compared with the diabetics but not statistically significant. Serum magnesium value and height were significantly lower in the control group than in the diabetics ( $p < 0.05$ ).

A similar comparison of these parameters in the females is shown in table 4. There was no significant difference between their ages, FBS, weight and BMI. However, serum magnesium level is significantly lower in the control females than the diabetic females ( $p < 0.05$ ).

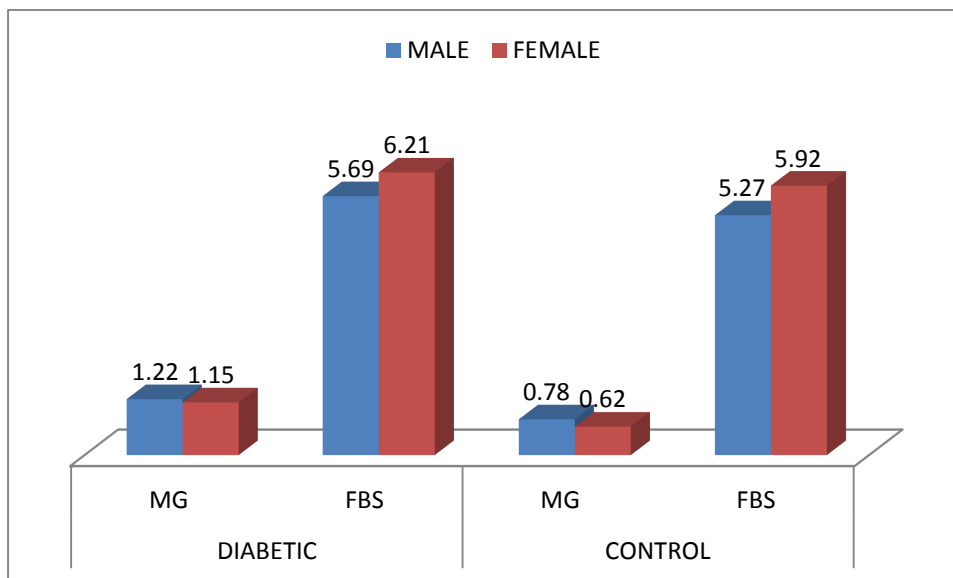
**TABLE 4 BIOCHEMICAL AND PHYSICAL PARAMETERS OF DIABETIC FEMALE SUBJECTS AND CONTROLS**

		<i>FBS</i>	<i>MG</i>			
	<i>AGE(YRS)</i>	<i>(MMOL/L)</i>	<i>(MMOL/L)</i>	<i>HEIGHT(M)</i>	<i>WEIGHT(KG)</i>	<i>BMI(KG/M<sup>2</sup>)</i>
<b>FEMALES</b>	46.57 ± 1.82	6.21 ± 1.31	1.15 ± 0.52	1.59 ± 0.08	69.2 ± 1.51	27.14 ± 0.5
<b>CONTRL</b>	56.44 ± 2.21	5.92 ± 1.12	0.62 ± 0.48	1.58 ± 0.09	67.6 ± 1.62	26.64 ± 0.59
<b>P-VALUES</b>	0.166940717	0.142723816	<b>2.90E-06</b>	0.196894509	0.452624439	0.26888739



**FIG 1: Aligned dot plot of serum magnesium concentration levels of diabetics and control study groups**

A visual inspection of the figure 1 above revealed an obvious indication of higher magnesium level in the test group than the control group. The P value  $< 0.0001$  is also less than 0.05 or 0.01 alpha level of significance, thus statistically significant. Median values of serum magnesium are higher in the test group than in the control group. Fig 1 shows higher magnesium levels in the test group (median value=1.18) than the control group (median value=0.7) with P values  $< 0.05$ , thus showing a statistically significant difference. Also, Fig. 1 shows a higher variability in serum magnesium level among the test group than the control group.



**FIGURE 2: A Bar chart showing concentrations of serum magnesium and fasting blood sugar among diabetics and controls.**

A visual inspection of the figure 2 above revealed an obvious indication of higher serum magnesium mean level in the diabetic group than the control group according to sex, with values higher in male subjects than in females.

#### 4. DISCUSSION

Hypomagnesaemia has been reported to occur at an increased frequency among patients with type 2 diabetes compared with their counterparts without diabetes (Lopez et al, 2004). Despite numerous reports linking hypomagnesaemia to chronic diabetic complications, attention to this issue is poor among clinicians (Meludu et al, 2001). Diabetes mellitus has remained one of the most burdensome chronic metabolic diseases with complications constituting a significant cause of morbidity and mortality among patients; with developing nations like Nigeria with poor resources having a substantially greater percentage (Yudkin, 2003). Several investigators have therefore addressed the topic of magnesium status and dietary magnesium intake, especially in diabetes mellitus in other nations but such studies are not found in Nigeria. Not all studies, however, observed a correlation between glycaemic control and serum Mg levels or improvement of diabetic control with Mg replacement (Lopez et al, 2004; Hans et al, 2003). The conflicting data may reflect different study designs and populations studied.

Results from this study showed serum magnesium in the control study group are within the normal range (0.75-1.25mmol/l) as published by (Chetan et al, 2001; Zinov et al, 2008 and Rubi, 2007). Serum magnesium values were higher among diabetics ( $1.18 \pm 0.06$ ) compared with the controls ( $0.7 \pm 0.06$ ). This may be due to their glycaemic control as reported by Yokota, (2005), that "in diabetics' poor glycaemic control is associated with serum magnesium deficiency". It is therefore our opinion that since these patients are attending clinic and are therefore under glycaemic control, no hypomagnesaemia is observed. However, recognizing the signs of diabetes-induced magnesium deficiency may be important but this was not done in this study, as the deficiency can occur long before it is reflected in the serum values (Ma et al, 1995).

Since the mechanism responsible for serum magnesium deficiency in diabetics is not completely known, and osmotic diuresis accounts for a portion of the magnesium loss, it is possible to suggest that hypomagnesaemia in diabetics may only be observed in cases of loss or no glycaemic control. Barbagallo et al, (2003) also reported that impaired insulin action or insulin deficiency as observed in type 2 Diabetes results in an intracellular magnesium deficiency while Huerta (2005), in his work said high serum magnesium levels may be due to insulin which increases absorption of magnesium in diabetics. Thus, the increased levels of serum magnesium ion concentrations as observed among the diabetics in this study may be due to various clinical variables such as kind of therapy practiced and renal function that may modify the status of magnesium in diabetic patients (Hans et al, 2002).

Epidemiological studies done by Kao et al (1999); Lopez-Ridaura et al (2004); and Song et al (2004), shows an inverse correlation between magnesium intake and the risk of developing diabetes mellitus. The authors suggested that an increased consumption of magnesium-rich food, such as whole grains, beans, nuts and green vegetables, might reduce the risk for T2DM. Although, this study examines serum magnesium levels among established type 2 diabetic patients compared with apparently healthy subjects as controls, such inverse relationship was not found and may be due to their glycaemic control through diets rich in magnesium ions such

as vegetables and legumes that may have led to the absence of risk among the diabetics and as well improve their magnesium levels. It can also be deduced from the findings of this report that hypomagnesaemia may not be found in all diabetics but may be a consequence of diabetic complications such as obesity, low grade chronic inflammatory disease syndrome, aging, retinopathy and thrombosis (Fernando et al, 2007). However, this did not hold true for the precursor states of diabetes, as no differences were observed between healthy controls and individuals with impaired glucose tolerance or impaired fasting glucose levels (Simmons et al 2010). Therefore, it may not be out of place to test for serum magnesium and fasting blood sugar among patients with poor glycaemic control as epidemiological evidence shows that complications may begin several years before clinical diagnosis (Stahl et al, 2001). As a consequence of the aforesaid observations, a controversy has ensued concerning the causal association between hypomagnesaemia and the risk for diabetes mellitus. It is therefore, necessary to analyze serum magnesium levels in diabetics across races to establish a strong relationship between serum magnesium and diabetes as observed in the study, as literatures are scarce in this regard.

## 5. Conclusion

Hypomagnesaemia among diabetics has been documented in various studies. However, in this study it was observed that serum magnesium values were higher among diabetics than the non-diabetic study group. It is clear that serum magnesium values may be reduced in uncontrolled diabetic subjects together with other contributing factors such as osmotic diuresis as well as environmental factors.

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