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Short Communication

EVALUATION OF SOME MICRONUTRIENT PATTERNS IN BLOOD SAMPLE OF TYPE 1 DIABETIC PATIENTS

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ABSTRACT

To investigate the relationship of micronutrients concentration in Type 1 Diabetic Patients and how their interactions affect the homeostasis in this hyperglycemic state, we evaluated the concentration of calcium, magnesium, zinc and glucose. We observed a concomitant increase in calcium with raised glucose level while magnesium and zinc levels where decreased. Using only Samples with plasma glucose above threshold (≥10.0mmol/L) the micronutrients were measured at time intervals of 0 to 30 mins, 60 mins, 120 minutes, 180 mins and 240 mins. It was observed that raised glucose induced significant ($P< 0.05$) elevation of calcium from 20.2 ± 1.4 mmol/L to 60.4 ± 21 mmol/L while reduction of magnesium and zinc were in the order 180 ± 7 to 58.4 ± 3 Φmol/L and 15.4 ± 0.5 to 4.3 ± 0.4 Φmol/L respectively. We conclude that variations in concentration of micronutrients may elicit clinical expression of common abnormal homeostatic environment expressed partly by the reduced levels of magnesium and zinc that complicates the diabetic process.

Keywords: Micronutrients, blood sample, type 1 diabetic patients.

INTRODUCTION

Studies on the relationship between diabetic conditions and micronutrient concentration have been carried out by other workers as shown by Lawrence *et al*. (2001). Our knowledge of the role ions play to effect cells response and the dynamics surrounding this relationship will require further scrutiny over time. Calcium, magnesium and zinc are known to contribute towards the maintenance of the intracellular ion homeostasis. Magnesium is known to play a key role in the process by serving as an important co-factor for activation of calcium and sodium pumps (Guillemett, 1991; Anetor *et al*. 2002). Moreover magnesium and calcium both play a significant role to stimulate the B – cell of pancrease to produce insulin. As shown (Boyd *et al*., 1989; Zureil *et al*., 2004) reduced magnesium level is a catalyst for induction of hyperinsulnism. This gives credence to the underlying fact that some of the sequelae of magnesium deficiency may be partly accountable for the inbalance of competition between calcium and magnesium along with other ions as they move into the cells.

Calcium is essentially known to play vital role in the intracellular transduction mechanism for insulin secretion from pancreatic B – cells, modulation in one of two ways as a regulator or as a signaling system for cellular enzyme activities.

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Serum Zinc is known to exert strong antioxidant behaviour as shown by Powel (2000). Being an important component of biomembranes and essential cofactor in a variety of enzymes (Bettger *et al*., 1981), zinc plays an important role in the synthesis and functions of insulin as well as membrane – stabilizing properties.

MATERIALS AND METHODS

At the Federal Medical Center Yenagoa, Nigeria, blood Samples were collected from known type 1 diabetic patients (n = 50) all having glucose levels above threshold (10.0 mmol/L), minimum 15.0mmol/L. The second group were non-diabetics (n = 50) whose fasting plasma glucose level ranged between 3.5mmol/L – 6.0mmol/L. Measurements of the micronutrients for the diabetic patients were compared with those of non-diabetic patients whose glucose concentration were varied by the addition of 5.0mmol/L, 10mmol/L, 20mmol/L and 30 mmol/L and a coordinated measurement taken at a time interval of t = o, t = 30, t = 60, t = 180 and t = 240 minutes.

Spectrophotometric method was used for the determination of glucose (oxidase method). Calcium was measured by the O-cresolphthalein complexone (CPC) method while the calgamite and Methylthymol method was used for magnesium. Zinc level was determined by Atomic absorption spectroscopy.

Table 1. Measured concentration of Ca^{2+}, Mg^{2+} and Zn^{2+} .

Time Min	0	30	60	120	180	240
Ca^{2+} (mmol/L)	2.2	2.8	3.6	4.8	6.7	6.2
Mg2+ (Φmol/L)	210	200	180	132	58.4	53.3
Zn^{2+} (Φmol/L)	15.4	14.6	13.2	12.3	10.1	8.6

Table shows time course of effects of elevated glucose 20 mmol/L on Ca^{2+}, Mg^{2+}, Zn^{2+} for the different ions demonstrated.

RESULTS

The sustained effects of raised glucose concentration on Ca^{2+}, Mg^{2+} and Zn^{2+} at glucose levels greater than 10.0 mmol/L over the course of 4 hours are recorded (Table 1).

Glucose was observed to elicit a co-ordinated elevation of calcium in which peak values was approximately 3 times higher than at base line 2.2mmol/L to 6.2 mmol/L (P=0.05 at t = 30, 60, 120, 180 and 240mins) as shown in table 1.

We equally observed concurrent suppression of mg^{2+} in which values dropped from a baseline of 210 **Φ**mol/L to 53.3 **Φ**mol/L almost representing a three fold decrease. Zinc values also show a moderate decrease in concentration over the four hours period under study (Table 2).

Table 2. Effects of varied glucose concentration Vs Time.

Ca^{2+} (mmol/L)	Mg^{2+} 2+ (Φmol/L)	Zn^{2+} (Φmol/L)
$t_0 = 2.8$	$t_0 = 220$	$t_0 = 15.4$
$t_{30} = 3.5$	$t_{30} = 180$	$t_{30} = 14.6$
$t_{60} = 6.8$	$t_{60} = 155$	$t_{60} = 13.2$
$t_{120} = 7.3$	$t_{120} = 140$	$t_{120} = 12.3$
$t_{180} = 6.0$	$t_{180} = 147$	$t_{180} = 10.1$
$t_{240} = 5.8$	$t_{240} = 140$	$t_{240} = 8.6$

The elevated glucose induced changes persisted over the period of observation. We however observed calcium level elicited a return close to the normal baseline value as shown in the table 3.

Table 3. Measured value of calcium vs glucose in 30 mins.

Ca^{2+} (mmol/L)	Glucose (mmol/L)
2.6	5.0
3.3	7.5
4.0	10.0
4.5	15.0
3.5	20.0
3.0	30.0

Effects of various extracellular glucose concentration in vitro (n = 6 for respective concentration on calcium in normal red cells samples incubated with glucose for 30 mins P = 0.05 Vs basal (5mmol/L) repeated measures analysis of variance.

STATISTICAL ANALYSIS

Values obtained for the response of each micronutrient (ion specie) to glucose were analyzed for statistical significance with the repeated measures of analysis of variance for both the effect of glucose over time at t = 0, 60, 120, 180, 240 mins over the range of concentrations tested at 5.0, 7.5, 10, 15, 20, 25 **Φ**mol/L. All values are reported as mean of ±SEM.

DISCUSSION

The clinical association of micronutrients in diabetic abnormalities related to insulin has been established according to Sigh *et al*. (1998).

Our present findings suggest that glucose itself may be a factor contributing both to cellular ion homeostasis and (cellular ion inbalance) and also to the risk of pathophysiology of hyperglycemic stress state. Glucose induced micronutrient effects were found to be existing effects that are both time and concentration dependent as shown in table 2 which corresponds to the renal glucose threshold for glucose excretion. Recent evidence suggests that inhibitory effect of glucose on membrane calcium Atpase has seen reported at concentrations similar to those associated here with elevated cystolic free calcium levels. Moreover, there is evidence to suggest an interaction of glucose with membrane bound phosphoinositide system. In skin microvascular and adipose tissue, glucose stimulated diacetyl and protein kinase activity in an insulin dependent manner (Guilemette *et al.*, 1991).

Both intracellular and extracellular calcium are increased in most tissues. The activities of the ATpase associated cation pumps which determined intracellular calcium level i.e calcium ATpase and (sodium + potassium) ATpase, are also altered. The nature of alteration is often tissue specific and may depend on the level of blood glucose.

Diabetics often have low concentrations of Ca^{2+}. There is a consistent association of low level Ca^{2+} level and risk and incidence of diabetes mellitus. Calcium is necessary for intracellular communication between the hormone insulin and tissues that utilizes insulin including sketetal muscle and fat tissues. Only a small amount of calcium is

needed for optimal insulin-related functions, but being a little bit off can cause serious health consequences including resistance and impaired insulin signaling. It is known that calcium supplementation on blood sugar level when properly regulated can elucidate the comparative effect like using the diabetes drug metformin (Joseph, 2007; Danit and Sharhar, 2007).

There is growing body of evidence to suggest that Mg^{2+} play play a pivotal role in reducing cardiovascular risk and may be involved in the pathogenesis of Diabetes mellitus. It is known that magnesium supplementation improved insulin sensitivity. Magnesium depletion may play a role in delaying the complications associated with type 1 diabetes mellitus onset and potentially reduce its devastating complication; cardiovascular disease, retinopathy and nephropathy. Both mean plasma and intracellular free mg^{2+} are lower in diabetes mellitus. Magnesium is a crucial co-factor involved in many enzymatic reactions involved in the metabolic process. Plasma levels were seen to be lower in normal persons. Levels of serum ionized Mg^{2+} and erythrocyte intracullar free Mg^{2+} are strong predisposing factors for development of the excess cardiovascular morbidity. Plasma Mg^{2+} as shown by Paolisso *et al*. (1989) declined and erythrocyte Mg^{2+} level rose significantly ($P<0.05$) in response to insulin in fasting healthy adults with no family history of diabetes. Reasons for low magnesium is adduced to the high renal excretory state, the effect imposed on it by its insensitivity to insulin and Mg^{2+} deficiency is associated with insulin resistance and increased platelets reactivity. Sasakis *et al*. (2009) and other colleagues have confirmed that magnesium deficiency decrease insulin mediated glucose disposal in non diabetic subjects, which is consistent with insulin resistance. Diabetes is known to effect zinc homeostasis since zinc plays a role in the synthesis and activation of insulin. Deficiency of Zn^{2+} results in oxidative stress which may damage the cells irreversibly exacerbating some of the classical complications of Diabetes. The current study was undertaken to understand the roles of some micronutrients in the diabetic process and has shown parts played by Ca^{2+}, mg^{2+} and Zn^{2+} in this hyperglycemic condition. The results strongly suggested that a well co-ordinated combination of these micronutrient could exhibit beneficial effect in managing diabetes mellitus.

REFERENCES

Anetor, J., Seujobi, A., Ajose, O. and Agbedana, E. 2002. Decreased Serum magnesium and zinc levels: atherogenic implications in type 2 diabetes mellitus in Nigeria. Nutr Health. 16:291-300.

Boyd, III AE., Rajan, AS. and Gaines, KL. 1989. Regulation of insulin Release by Calcium In: Insulin Secretion: Molecular and Cellular Biology of Diabetes Mellitus. (1):93:105.

Bettger, W. and O' Dell, B. 1981. A Critical Physiological role of Zinc in the Structure and Function of Biomembrane. Life Science. 28:1425-1438.

Danit, R. and Sharhar, S. 2007. Does Daily Calcium intake enhance weight loss among over weight Diabetic patients? Diabetes care. 3(3):485-493.

Guillemett, C., Poitras, M. and Boulay, G. 1991. Two Calcium System as distinguished on the basis of their my^{2+} dependency in a post nuclear particulate fraction of bovine adrenal cortex. Cell Calcium. 8:51-60.

Joseph, L. 2007. Diabetes Mealitus, A Disease of Abnormal Cellular Calcium Metabolism. American Journal of Medicine. 34:293-307.

Powell, S. 2000. The antioxidant Properties of Zinc. J Nutr. 130:14475-14545.

Paolisso, G., Sgambato, P. and Pissarielon, G. 1989. Improved insulin response and action by chronic magnesium administration in aged NIDDM Subjects. Diabetes Care. 12:262-269.

Sasaki, S., Oshime, T. and Matsura, H. 2009. Abnormal magnesium status in patient with cardiovascular disease. Clin Sci. (Colch). 98:175-181.

Sigh, R., Niar, M., Rastogi, S., Bajaj, S., Gaoli, Z. and Shoumin, Z. 1998. Current Zinc intake and risk of diabetes and coronary attery disease and factors associated with insulin resistance in rural and urban population of North India. J. AM. Coll. Nutr. 17:564-570.

Zureil, M., Galan, P., Bertrais, S., Mennen, L., Czeruichow, S., Blacher, J., Ducimetiere, P. and Henberge, S. 2004. Effects of long-term daily low dose supplementation with antioxidant, vitamins and mineral, on structure and function of large arteries. Arterioscler Thromb Vasc Biol. 24:1485-1495.

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