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RESEARCH ARTICLE

ANALYSIS OF SERUM MAGNESIUM LEVEL IN TYPE 2 DIABETES MELLITUS PATIENTS

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ABSTRACT

Background: Magnesium (Mg) deficiency is a common problem in diabetic patients. Hypomagnesemia increase the incidence of diabetes mellitus and occurrence of diabetic complications[1] Diabetic peripheral neuropathy affects 50% of all diabetic patients. Patients with peripheral neuropathy are considered at risk of insensate foot ulcerations. At least half of foot ulcers, the end stage of such neuropathy should be preventable by early diagnosis and effective management. **Aim:** The aim of this study was to estimate serum magnesium levels in patients with type 2 diabetes mellitus and correlate it with complications of type 2 diabetes mellitus - glycemic control and foot ulcer. **Materials and methods:** This is a cross sectional study done in stanley Medical College and Hospital, serum magnesium level, fasting blood sugar were estimated by standard methods and compared in type 2 diabetic patients and controls. **Result:** Serum magnesium level in diabetic population was significantly low in comparison to controls .In particular diabetic patients with foot ulcer had the lowest level in serum magnesium. There was negative correlation between fasting blood sugar and serum magnesium in diabetic patients. Duration of diabetes and serum magnesium were inversely related. **Conclusion:** Hence it is concluded that the lower levels of serum magnesium may have a bearing on the complication and morbidity in patients of DM, and estimation of serum levels of magnesium may be helpful to monitor the severity of complications in diabetic patients.

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INTRODUCTION

Micronutrients have been investigated as potential, preventive and therapeutic agents for type 2 diabetes mellitus and their complications. In particular, diabetes has shown to be associated with abnormalities in the metabolism of zinc, chromium, copper, magnesium and manganese. Out of these, magnesium has been investigated as a clinically significant electrolyte, for a long term global policy to lower the burden of diabetes mellitus, with new findings and researches. Magnesium, the fourth most common cation in the body, is established as a central electrolyte in a large number of cellular metabolic reactions, including DNA and protein synthesis, neurotransmission, and hormone receptor binding. It is a component of GTPase and a cofactor for Na+ / K+- ATPase, adenylate cyclase and phosphofructokinase (Arnaud, 2008). Diabetes is an iceberg disease. The prevalence of diabetes in adults was around 4% worldwide (Park, 2002).

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The recent WHO report suggests that over 19% of worlds diabetic population currently resides in India. One third of all diabetic patients have significant peripheral neuropathy and/or peripheral vascular disease - the main risk factor for foot ulceration. Diabetic foot problems are the most common causes of hospitalization in diabetic patients and it imposes heavy financial burden on the health care system. There are several studies on the role of trace elements like magnesium deficiency in the etiology of diabetes and its complications. The probable cause of Hypomagnesaemia in Diabetes are decreased Magnesiumintake, enhanced renal loss, reduced renal reabsorption. Magnesium deficiency leads to impairment of insulin action, worsening of insulin resistance and development of diabetic complications[3].Many studies have reported the association between hypomagnesemia and adverse complications of type 2 Diabetes like coronary artery disease, hypertension, diabetic retinopathy, nephropathy, neuropathy and foot ulcerations.

MATERIALS AND METHODS

This study was conducted on 30 cases of Type 2 Diabetes mellitus with foot ulcer (foot ulcer of any size present for

atleast 3 months in one or both foot), 30 cases of type 2 Diabetes mellitus without foot ulcers and 20 healthy volunteers of both sexes with age group ranging from 50 – 69 years from the out patients of Department of Diabetology, with due permission of the Head of the Department and the Dean, stanley Medical College Hospital, written consent was obtained from all the subjects. Ethical clearance was obtained. Detailed history was taken and clinical examination was done. Serum magnesium estimation was done by Calmagite dye method by semi-auto analyzer in department of Bio-chemistry Stanley Medical College. The Lab investigations ,fasting blood sugar and urine routine were done in the Department of Biochemistry, Stanley Medical College hospital.

RESULTS AND OBSERVATIONS

The different parameters were analyzed using one way ANOVA F-test, Bonterferoni t-test, Chi-square test, student t-test and correlations were analyzed by Pearson Correlation Coefficient. In our study on diabetic patients with foot ulcers there were 19 males and 11 females, with mean age of 61.05. In diabetic patients without foot ulcers there were 17 males and 13 females with mean age of 58.17. In control group there were 11 males and 9 females with mean age of 60.05. There was statistically significant variation in the level of serum magnesium between the controls (mean – 2.35meq/L), DM without foot ulcers (Mean – 1.95 meq/L) and DM with foot ulcers (Mean – 1.67 meq/L). The level of serum magnesium was lower in DM without foot ulcer and lowest in DM without foot ulcers when compared to the control (Table 1).

DISCUSSION

Magnesium depletion has shown to have a negative impact on glucose homeostasis and insulin sensitivity in patients with Type 2 diabetes. The impairment of insulin sensitivity may be related to a defective tyrosine kinase activity of the insulin receptor. Also in human nondiabetic subjects, low plasma Mg has been associated with relative insulin resistance, glucose intolerance, and hyperinsulinemia (Ramsey *et al*., 1999) (Rosolova *et al* 1997). Relationship between Mg deficiency and insulin resistance is a vicious circle. Low Mg status contributes to the development of insulin resistance, which in turn attenuates Mg uptake in insulin sensitive tissues (Rodriguez, 2001).

More than 30% of ambulatory diabetic patients without renal insufficiency were hypomagnesemic on a multifactorial basis (Phuong, ?). The main cause appears to be increased urinary loss accompanying glycosuria induced osmotic diuresis. In this study, it was observed that the mean serum magnesium level was low (1.95 meq/l) in diabetic patients in comparison to control subjects (2.35 meq/l). This indicates the association of hypomagnesemia with diabetes mellitus. These results were in accordance with the observation of Stutzman and Amatuzia (Garfinked, 1998), Jackson and Meier (Tosiello, 1996), Jain *et al*. (1979) Mc Nair *et al* and Vyas *et al*. (1953). All had reported strongly association of hypomagnesemia with diabetes mellitus. The mean serum magnesium in cases with foot ulcers was 1.67 meq/l which was much lower than those without foot ulcer 1.95meq/l.

Table 1. Comparison of serum magnesium

	N	Serum. Magnesium		Oneway NOVA F-test	Multiple comparison by Bonferroni t-test
		Mean (Meq/L)	Std Deviation		
Control	20	2.3520	.114	F=216.3 P=0.001 significant	1 & 2, 1 & 3, 2& 3 significant
DM with Foot ulcer	30	1.6700	.082		
DM without Foot ulcer	30	1.9550	.137		

Table 2. Correlations between fasting blood sugar & serum magnesium

Group			Serum Magnesium
DM with Foot ulcer	blood sugar	Pearson Correlation	-.885(**)
		Sig. (2-tailed)	.001
		N	30
DM without Foot ulcer	blood sugar	Pearson Correlation	-.618(**)
		Sig. (2-tailed)	.001
		N	30

**Correlation is significant at the 0.01 level (2-tailed).

Table 3. Correlation between dm duration and serum magnesium

Group			Serum Magnesium
DM with Foot ulcer	DM duration	Pearson Correlation	-.228
		Sig. (2-tailed)	.199
		N	30
		Sig. (2-tailed)	.000
		N	30

** Correlation is significant at the 0.01 level (2-tailed).

In groups DM with foot ulcers and without foot ulcers there was negative correlation between fasting blood sugar and serum magnesium levels ie higher the fasting blood sugar level lower the serum magnesium level (Table 2). There was an inverse relationship between the duration of diabetes and serum magnesium levels,(ie) longer the duration of diabetes lower, the serum magnesium level. This observation was statistically significant.

These findings are in accordance with those of Rodriguez Morann[2] in which there was strong relationship between serum magnesium depletion and the presence of foot ulcers. Magnesium is an effector of inositol transport. It increases the affinity of the transporter for inositol. Hypomagnesemia causes a decrease in the affinity of the inositol transport protein for inositol. Decreased levels of intracellular myoinositol lead to reduced phosphoinositide synthesis and decreased production

of diacylglycerol. This in turn impairs Protein kinase C activation and may lead to decreased Na^+/K^+ ATPase activity. In nerve, the inhibition of Na^+/K^+-ATPase activity may reduce Na^+ extrusion and therefore lead to increased intracellular Na^+ concentration which on its part inhibits depolarization and slows nerve conduction rate. It was postulated that polyol pathway hyperactivity could be the mechanism linking hyperglycemia to neuropathy. Activation of polyol pathway also may promote oxidative stress by depleting NADH, a cofactor for both aldose reductase and glutathione reductase which lead to a decrease in reduced glutathione and increase in oxidized glutathione. Reduced glutathione is an important scavenger of reactive oxygen species, thus the decrease in reduced glutathione may induce or exacerbate intracellular oxidative stress.This allows hypomagnesemia and the polyol pathway to be rationalized into a single mechanism for the aetiology of diabetic complications (Jackson, 1968)**.** In this study there was negative correlation between fasting blood sugar and serum magnesium in diabetic patients. Jackson and Meier (Jackson, 1968) also had observed that hypomagnesemia was commonly associated with poor control of diabetes specially when the fasting blood sugar was more than 180 mg %. Thus it was observed that a strong association exists between hypomagnesmia and duration of diabetes and poor glycemic control. In particular, Diabetic patients with foot ulcers had the lowest levels of serum magnesium, indicating that hypomagnesemia is important risk an factor in the pathogenesis of foot ulcers.

Conclusion

In our study there is strong association between hypomagnesaemia and Type 2 Diabetes mellitus, The serum levels of magnesium were significantly lower in patients with food ulcers than those without foot ulcers. There is strong association between poor glycaemic control and hypomagnesaemia. Duration of diabetes and serum magnesium was inversely related. The reduced serum magnesium level could be an early predictor of the course and complications of diabetes mellitus. So it is prudent to monitor serum magnesium level in the diabetic population and treat the condition appropriately. The treatment of the patients with diabetes requires a multidisciplinary approach whereby every potential complicating factor , such as serum mg, must be monitored closely and treated. Further large series observational and interventional studies are necessary to establish the facts of current study.

Conflict of Interest: Nil.

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Ethical Clearance: Taken.

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