Kumar DA, Priya VS, Jaiprabhu J, Swaroopa R. (May 2015). Low serum magnesium and zinc levels in type 2 diabetes mellitus: A pilot study. *Jour of Med Sc & Tech*; 4(2); Page No: 148 – 151.



Original Article

Open Access

Low serum magnesium and zinc levels in type 2 diabetes mellitus: A pilot study

*D Anil Kumar¹, V Shanmuga Priya², J Jaiprabhu², R Swaroopa³

¹Associate Professor, ²Assistant professor, Department of Biochemistry, Vinayaka Missions Medical College, Karaikal, Pondicherry, India.

³Associate Professor, Department of Pathology, Vinayaka Missions Medical College, Karaikal, Pondicherry, India.

Abstract

Diabetes mellitus is associated with alterations in the metabolism of zinc (Zn) and magnesium (Mg). Direct associations of these two trace elements with Diabetes mellitus have been observed by many researchers. The aim of the present study was to estimate the serum levels of these trace elements in patients with type 2 diabetes. The study population consists of total ninety participants, out of whom 45 subjects were type 2 diabetic patients and 45 were healthy age matched controls. All the participants were in the age group of 40– 55 years. Blood samples were collected for the estimation of Fasting glucose, serum magnesium and zinc. The present study showed that mean serum zinc level was significantly low in diabetics as compared to control subjects. Our study recommends supplementation of with Zn and Mg may have beneficial role in Diabetes Mellitus as diabetic patients have lower serum magnesium and zinc levels when compared to healthy individuals.

Keywords: Diabetes, serum magnesium, serum zinc, fasting glucose, Atomic absorption spectrophotometry

*Corresponding Author: Dr D Anil Kumar, Associate Professor, Department of Biochemistry, Vinayaka Mission Medical College and Hospital, Pondicherry, India. E-mail: munna_dr_74@rediffmail.com

Received: January 22, 2015. Accepted: March 7, 2015. Published: May 20, 2015. This is an openaccess article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

Diabetes mellitus is the leading cause of morbidity and mortality all over the world [1]. In 2011 the number of diabetes cases throughout the world is around 366 million and by 2030 it is expected to rise up to 552 million. Eighty percent of people with diabetes live in low- and middle-socioeconomic society. The greatest number of people with diabetes is between 40 and 59 years of age. In India, 61.3 million people were affected with diabetes in 2011 and it is estimated that by 2030 the number may reach to 101.2 million. Direct associations of trace elements with Diabetes mellitus have been observed in many research studies [2]. The metabolism of several trace elements has been reported to be altered in diabetes mellitus. Insulin

action on reducing blood glucose was reported to be potentiated by some trace elements as chromium, magnesium, vanadium zinc. manganese, molybdenum and selenium [3]. The proposed mechanism of trace elements enhancing insulin action includes activation of insulin receptor sites, serving as cofactors or components for enzyme systems involved in glucose metabolism [4, 5] increasing insulin sensitivity and acting as antioxidants preventing tissue per oxidation [6]. These trace elements might have specific roles in the pathogenesis, progress and complications of this disease [7]. Some of these trace elements may act as antioxidants and prevent membrane peroxidation while others act directly on glucose metabolism.

Magnesium is the fourth most abundant mineral in your body. Insufficient cellular magnesium levels set the stage for deterioration of proper metabolic function which causes many health problems. This important mineral is required by more than 300 different enzymes in your body, which play important roles in the following biochemical processes, many of which are crucial for proper metabolic function [8]. Zn is second to iron as the most abundant essential trace element in the body, is a component of many enzymes, and plays an important role in the maintenance of several tissue functions, including the synthesis, storage and release of insulin [9]. Zn modulates the immune system and Volume 4. Issue 2

Kumar DA, Priya VS, Jaiprabhu J, Swaroopa R. (May 2015). Low serum magnesium and zinc levels in type 2 diabetes mellitus: A pilot study. Jour of Med Sc & Tech; 4(2); Page No: 148 – 151.

its dysfunction in diabetes mellitus may be related in part to the status of Zn. Zn excretion may be increased in catabolic illness is like DM [10]. Magnesium is the second most prevalent intracellular cation, with a role as a cofactor in the phosphorylation of glucose and in other enzymatic reactions. It is important in oxidative phosphorylation, glycolysis, cell replication. nucleotide metabolism and protein synthesis [11]. Magnesium deficiencies have been implicated in carbohydrate insulin resistance, intolerance. dyslipidemia and complications of diabetes [12]. It is not known whether differences in trace elements status are a consequence of diabetes and alternatively hyperglycemia or whether their deficiencies contribute to the expression of the disease.

Zinc is required for insulin synthesis and storage and insulin is secreted as zinc crystals, it maintains the structural integrity of insulin [14]. Magnesium is a cofactor in the glucose transporting mechanisms of the cell membrane and various enzymes in carbohydrate oxidation. It is also involved at multiple levels in insulin secretion, binding and enhancing the ability of insulin to activate tyrosine kinase [15]. Magnesium deficiencies have been implicated in insulin resistance, carbohydrate intolerance, dyslipidemia and complications of diabetes [16, 17]. Lower serum levels of these elements have been reported in the diabetic state. It is unknown whether difference in trace elements status is a consequence of diabetes and hyperglycemia or alternatively whether their deficiencies contribute to the expression of the disease. The objective of this study was to determine the serum levels of magnesium and zinc in type 2 diabetic patients in comparison with healthy age matched controls.

Materials and Methods

This pilot study was conducted at Vinayaka Missions Medical College, Karaikal, Pondicherry, during the period of August 2014 to October 2014. The study population consists of Ninety (52-male, 38-female) subjects, out of whom 45 subjects were type 2 diabetic patients and 45 subjects were apparently healthy age matched controls. All the participants were in the age group of 40- 55 years. The study was approved by institutional ethics committee and informed written consent was obtained from each subject before recruitment into the study. Subjects with obesity, pregnancy, renal

disease, hypertension, and finally those taking nutritional supplements, laxatives, diuretics, or alcohol were excluded from the study.

Blood samples were collected by vein puncture from the patients after an overnight fast, into fluoride oxalate tubes for fasting glucose (FBS), and in plain tubes for serum magnesium and zinc determination. All the samples were centrifuged at 3500 rpm to separate serum. Fasting glucose was estimated on the same day using the enzymatic glucose oxidase peroxidase (GOD - POD) method. Samples were stored at -20°C till the estimation of magnesium and zinc. For the estimation of serum magnesium and zinc, ashed serum dissolved in dilute HCl was used and were analysed by atomic absorption spectrometry.

Results

Forty five diabetic patients were selected for the study, Out of these forty five subjects, 31 (68.8%) were male and 14 (31.2%) were females. Forty five age matched healthy subjects were selected as a control group. Out of them, 21 (46.6%) were male and 24 (53.3%) were females. Mean fasting blood sugar was $89.57 \pm 13.5 \text{ mg/dl}$ in healthy subjects while diabetic patients show 118.53 ± 24.56 mg/dl in diabetic patients (p < 0.001) [Figure 1]. Mean serum zinc was significantly lower in diabetic patients 79.54 \pm 14.60 when compared to healthy subjects 85.42 \pm 12.54, p < 0.001) [Figure – 2]. Statistically significant difference was found in serum magnesium level in both groups with mean of 1.76 ± 0.35 mg/dl in diabetic patients and 2.14 ± 0.26 mg/dl in healthy subjects (p = 0.001, Table 1). The difference in serum zinc and magnesium levels was statistically significant when compared between the groups (p =(0.0001) (p = 0.11).

Age 44.87 ± 6.84 45.21 ± 4.25 0.77 (in years)-Gender-Male 31 21 Female 14 24 FBS 118.53 ± 24.56 89.57 ± 13.5 0.0001 (mg/dl)Magnesium 1.76 ± 0.35 2.14 ± 0.26 0.0001 (mg/dl)Zinc 79.54 ± 14.60 85.42 ± 12.54 0.043 (µg/dl)Table 1: Shows comparison of all the parametersbetween the controls and the cases.	Parameter	Cases (n= 45)	Controls (n= 45)	p value
(in years) Image: second	Age	44.87 ± 6.84	45.21 ± 4.25	0.77
Gender Image: space	(in years)			
Male 31 21 - Female 14 24 - FBS 118.53 \pm 24.56 89.57 \pm 13.5 0.0002 (mg/dl) - - - Magnesium 1.76 \pm 0.35 2.14 \pm 0.26 0.0002 (mg/dl) - - - Zinc 79.54 \pm 14.60 85.42 \pm 12.54 0.043 (µg/dl) - - - Table 1: Shows comparison of all the parameters between the controls and the cases.	Gender			
Female 14 24 FBS 118.53 \pm 24.56 89.57 \pm 13.5 0.0001 (mg/dl) 1.76 \pm 0.35 2.14 \pm 0.26 0.0001 (mg/dl) 79.54 \pm 14.60 85.42 \pm 12.54 0.043 (µg/dl) 1.76 \pm 0.001 1.10 \pm 0.001 1.10 \pm 0.001 Table 1: Shows comparison of all the parameters between the controls and the cases. 1.10 \pm 0.101	Male	31	21	-
FBS (mg/dl) 118.53 \pm 24.56 89.57 \pm 13.5 0.0001 Magnesium (mg/dl) 1.76 \pm 0.35 2.14 \pm 0.26 0.0001 Zinc (mg/dl) 79.54 \pm 14.60 85.42 \pm 12.54 0.043 (µg/dl) Table 1: Shows comparison of all the parameters between the controls and the cases.	Female	14	24	
(mg/dl) Image: Constraint of the second	FBS	118.53 ± 24.56	89.57 ± 13.5	0.0001
Magnesium (mg/dl) 1.76 ± 0.35 2.14 ± 0.26 0.0001 Zinc 79.54 ± 14.60 85.42 ± 12.54 0.043 (µg/dl) Table 1: Shows comparison of all the parameters between the controls and the cases.	(mg/dl)			
(mg/dl)79.54 \pm 14.6085.42 \pm 12.540.043(µg/dl)Table 1: Shows comparison of all the parameters between the controls and the cases.	Magnesium	1.76 ± 0.35	2.14 ± 0.26	0.0001
Zinc 79.54 ± 14.60 85.42 ± 12.54 0.043 (µg/dl)Table 1: Shows comparison of all the parameters between the controls and the cases.	(mg/dl)			
(μg/dl) Table 1: Shows comparison of all the parameters between the controls and the cases.	Zinc	79.54 ± 14.60	85.42 ± 12.54	0.043
Table 1: Shows comparison of all the parametersbetween the controls and the cases.	(µg/dl)			
between the controls and the cases.	Table 1: S	hows compariso	on of all the pa	rameters
	between the	controls and the	cases.	

Kumar DA, Priya VS, Jaiprabhu J, Swaroopa R. (May 2015). Low serum magnesium and zinc levels in type 2 diabetes mellitus: A pilot study. *Jour of Med Sc & Tech*; 4(2); Page No: 148 – 151.



Figure 1: Shows comparison of age and fasting blood sugar between controls and cases.



Figure 2: Shows comparison of serum magnesium and zinc levels between controls and cases.

Discussion

Many trace elements are important for human function. Numerous studies metabolic have demonstrated the essential roles of trace elements as chromium, zinc, magnesium, selenium, vanadium, molybdenum and manganese in insulin action and carbohydrate metabolism. The actual role of these trace elements in he pathogenesis and progress of diabetes is still unclear [17, 18]. In the present study, it was observed that mean serum zinc level was significantly low in diabetics as compared to control subjects. Similar observations are reported by Masood N et al., Sharma et al., [18, 19] who also observed significantly lower serum zinc level in diabetics than in control subjects. Diwan, et al. [20] reported that serum magnesium levels were significantly low in diabetic patients when compared with control group. Lower serum magnesium level in diabetics than in controls was also reported by Tripath, et al [21].

It has been reported by many authors, that hypomagnesemia is a possible risk factor in the development and progress of diabetic retinopathy. Mg deficiency has been linked to diabetes, insulin resistance and metabolic syndrome. Induction of Mg deficiency reduced insulin sensitivity in individuals without diabetes, whereas Mg supplementation during a 4-week period has been shown to improve glucose handling in elderly individuals without diabetes [18, 22]. Significant lowering of serum magnesium has been shown in patients on long term treatment with insulin and those recovering from diabetic ketoacidosis.

The possible explanation for the significant decrease of zinc and magnesium levels in type 2 diabetes mellitus patients, may be diabetic patients excrete more zinc in urine than non-diabetics or may be decreased gastrointestinal absorption of zinc [18-21]. Disturbed metabolism of zinc metallo enzymes and abnormal binding of zinc to tissue proteins have also been suggested as possible causes. The low plasma zinc levels in diabetics suggest that the hyperzincuria is of renal origin. Renal tubular defect in handling zinc and glucose-induced, osmotic diuresis are other possibilities. Zinc is useful in the synthesis, storage, and secretion of insulin. Zn deficiency is associated with metabolic disturbances including impaired glucose tolerance, insulin degradation, and reduced pancreatic insulin content. Zn may improve glycaemia, and a restored Zn status in patients with type 2 diabetes may counteract the deleterious effects of oxidative stress, helping to prevent complications associated with diabetes. Zn has been reported to have beneficial antioxidant effects in persons with type 2 diabetes. This is particularly important in light of the deleterious consequences of oxidative stress in persons with diabetes [22 - 24].

Conclusion

Our study revealed low Serum zinc and magnesium levels in type-2 diabetics, when compared with control subjects. Similar results were reported by earlier authors. Hence supplementing Zn and Mg may have beneficial role in Diabetes Mellitus. However, larger studies in general populations are needed to evaluate the cost effective beneficial effects of Zn and Mg supplementation of Diabetes Mellitus and the related complications.

References

1. Power AC. Diabetes mellitus. In: Harrison's principles of internal medicine.17th ed. New York: McGraw-Hill; 2005. p. 2275.

Kumar DA, Priya VS, Jaiprabhu J, Swaroopa R. (May 2015). Low serum magnesium and zinc levels in type 2 diabetes mellitus: A pilot study. *Jour of Med Sc & Tech*; 4(2); Page No: 148 – 151.

- 2. Nourmohammadi I, Shalmani IK, Shaabani M, Gohari L. Zinc, copper, chromium, manganese and magnesium levels in serum and hair of insulin-dependent diabetics. Arch Iranian Med 2000; 2:88-100.
- 3. Candilish DJ. Minerals. J Am Coll Nutr 2000; 17:286-310.
- 4. Vincent JB. Quest for the molecular mechanism of chromium action and its relationship to diabetes. Nutr Rev 2000; 58:67-72.
- Murray RK, Grannner PA, Rodwell VW (eds). Metabolism of carbohydrates. In: Harpers Biochemistry. 25th ed. Appleton and Lange, 2000.p.190-5.
- 6. Kruse-Jarres JD, Rukgauer M. Trace elements in diabetes mellitus. peculiarities and clinical validity of determinations in blood cells. *J Trace Elem Med Biol*. 2000; 14:21-7.
- Hussain F, Arif MM, Sheikh MA, Nawaz H, Jamil A. Trace elements status in type 2 diabetes. Bangladesh J Med Sci. 2009; 8:52–6.
- 8. Winterberg B, Bocchichchio M, Horsdorf TH, Lahi H, Lison AE, Zimkley H. Zinc in treatment of diabetic patients. *Trace Elem Med.* 1989; 6:173–7.
- Roth HP, Kirchgessner M. Zinc and insulin metabolism. *Biol Trace Elem Res.* 1981; 3:13– 32.
- Shenkin A, Baines M, Fell GS, et al. Vitamins and trace elements. In: Burtis CA, Ashwood ER, Bruns DE, editors. Teitz textbook of clinical chemistry and molecular diagnostics. 4th ed. New Delhi:Elsevier; 2006. 1137–9.
- 11. Endres DB, Rude RK. Mineral and bone metabolism. In: Burtis CA, Ashwood ER, Bruns DE, editors. Teitz textbook of clinical chemistry and molecular diagnostics. 4th ed. New Delhi: Elservier; 2006. p.1909–12.
- Resnick LM, Gupta RK, Bhargava KK, Hgruenspan H, Alderman MH, Laragh J. Cellualar ions in hydertenson, diabetese, and obesity. A nuclear magnetic resonance spectrospic study. *Hypertension* 1991; 17:951-7.
- 13. Chausmer AB. Zinc, insulin and diabetes. J Am Coll Nutr 1998; 17:109-15.
- 14. Suarez Z. Decreased insulin sensitivity in skeletal muscle of hypomagnesemic rats. Diabetologia 1993; 36:82.
- 15. Zargar AH, Bashir MI, Masoodi SR, Laway A, Wani AI, Khan AR, *et al.* Copper, Zinc and Magnesium evels in type-1 diabetes mellitus. *Saudi Med J* 2002; 23:539-42.
- 16. Al-Maroof RA, Al-Sharbatti SS. Serum zinc levels in diabetic patients and effect of zinc

supplementation on glycemic control of type-2 diabetics: *Saudi Med J* 2006; **27**:344-50.

- 17. Anetor JI, Senjobi A, Ajose OA, Agbedana EO. Decreased serum magnesium and zinc levels: artherogenic implications in type-2 diabetes mellitus in Nigerians. *Nutr Health* 2002; **16**: 291-300.]
- Masood N, Baloch GH, Ghori RA, Memon IA, Memon MA, Memon MS. Serum Zinc and Magnesium in Type-2 Diabetic Patients. *Journal* of the College of Physicians and Surgeons Pakistan. 2009; 19 (8): 483-486.
- Sharma A, Dabla S, Agrawal RP, Barjatya H, Kothari RP, Kochar DK. Serum magnesium: an early predictor of course complications of diabetes mellitus. *J Indian Med Assoc* 2007. 105:16-20
- 20. Diwan AG, Pradhan AB, Lingojwar D, Krishna KK, Singh P, Almelkar SI. Serum zinc, chromium and magnesium levels in type-2 diabetes. *Int J Diabet Dev Countries* 2006; 26:122-3.
- 21. Tripathy S, Sumathi S, Raj GB. Minerals nutritional status of type-2 diabetic subjects. *Int J Diab Dev Countries*. 2004; 24:27-8.
- 22. Duzguner V, Kaya S. Effect of zinc on the lipid peroxidation and the antioxidant defense systems of the alloxan-induced diabetic rabbits. *Int J Free Radical Biol Med.* 2007; 42:1481–6.
- 23. Roussel AM, Kerkeni A, Zouari N, Mahjoub S, Matheau JM, Anderson RA. Antioxidant effects of zinc supplementation in Tunisians with type 2 diabetes mellitus. *Int J Am Coll Nutr.* 2003; 22:316–21.
- 24. Tosiello L. Hypomagnesaemia and diabetes mellitus. *Arch Intern Med.* 1996; 156:1143–8.