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Research Article

**MICRONUTRIENT DEFICIENCY (MAGNESIUM) AND ITS
RELATION TO THE CAUSATION OF DIABETES MELLITIS -
A CASE CONTROL STUDY ON DIABETIC PATIENTS OF
BAHAWAL VICTORIA HOSPITAL, BAHAWALPUR,
PAKISTAN**

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Abstract:

Hyperglycemia causes a multi-factor disorder known as diabetes. Poor control of the diabetes and hypomagnesaemia are critically associated as confusions are caused about Diabetes Mellitus (DM). Insulin receptor is affected by the key component of Magnesium. In the patients of diabetes, the factor of hypomagnesaemia is involved in resistance of the insulin through the interference of the insulin receptors integration. An inactive insulin receptor phosphorylation is responsible for this act; therefore, the whole process can be viewed as a central system through which hypomagnesaemia is added to the resistance of insulin in the cells.

Objectives: *This research was aimed at the probe that if low levels of the magnesium in the patients of diabetes in comparison of those patients never diagnosed with diabetes.*

Settings: *Research was held at Quaid-e-Azam Medical College, Bahawalpur (Department of Biochemistry)*

Material & Method: *Source of the collected data was BVH, Bahawalpur. Design of the research was observational and descriptive. Research was completed in the period of six months.*

Conclusion: *It is concluded that there is an association of hypomagnesaemia with Type I & II DM as diabetes cases reflect clear decreased magnesium levels. Its consequences in severity of disorders can be reduced in a drastic way through the diets of magnesium fortified administrated to diabetes cases.*

Keywords: *Frequency, Diabetics, Type-I Diabetes Mellitus (T1DM), Type-II Diabetes Mellitus (T2DM), Hypomagnesemia and Bahawalpur.*

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INTRODUCTION:

Hyperglycemia causes a multi-factor disorder known as diabetes. It is well established through various research studies that level of the magnesium is reduced in the diabetes cases in contrast to the control cases of non-diabetic patients. Poor control of the diabetes and hypomagnesaemia are critically associated as confusions are caused about Diabetes Mellitus (DM). Ill effects of the DM have been faced by above 300 million its prevalence estimated to increase up to 600 million after next thirty years, relation of the T2DM is associated with hypomagnesemia since 1940. Insulin receptor is affected by the key component of Magnesium. Under the impact of certain conditions there is a prompt in the generation of glycogen, lipid union and increase in glucose respectively in liver, fat tissue and muscles. Insulin resistance is counted as the regular result of the reduced insulin receptor affectivity because of the insulin subunits. In diabetic cases, the factor of hypomagnesaemia is involved in resistance of the insulin through the interference of the insulin receptors integration. An inactive insulin receptor phosphorylation is responsible for this act; therefore, the whole process can be viewed as a central system through which hypomagnesaemia is added to the resistance of insulin in the cells. Increase in glucose in skeletal muscles is because of GLUT4 upregulation that is a glucose transport protein mechanism. Recent research conducted on rats having streptozotocin instigated diabetes, GLUT4 articulation was expanded through the magnesium supplementation for the articulation of rodent muscles, it also brought level of serum glucose down to a typical range. Moreover, in the presence of the hypomagnesaemia negative impacts in the case of T2DM (that basically interfered sensitivity of the insulin receptor), hypomagnesaemia similarly stimulates a disabled insulin emission through pancreatic B-cells and in the same way T1DM is also ensnared.

In pancreatic B cells, particles of Mg²⁺ impact glucokinase action rate through the adenine nucleotides cofactor. In our research study we focused on the power and recurrence of hypomagnesaemia in Type I & II diabetic cases and its association with the glycemic record. In the past few decades, hypomagnesaemia is surely associated to T2DM. Its cases and hypomagnesaemia are destined to an endless circle that insulin resistance is caused by hypomagnesaemia. Low levels of Magnesium in diabetes are associated to low Magnesium incorporate eating regimens, Osmotic Diuretics that causes increases rate of Mg renal discharge, insulin insensitivity that affects

intracellular Magnesium. A decreased level of Mg may also lead to an endothelial breakdown. A happening of hypomagnesaemia is attributed to the incidence of 13.54 – 47.7 percent in T2DM cases. Reduced dietary admission, adjusted insulin digestion, autonomic brokenness, osmotic diuresis, glomerular hyper filtration, hypophosphatemia intermittent metabolic acidosis and hypokalemia may also be nominated as the contributors. There is another link of the poor glycemic control, hypertension, coronary vein ailment, neuropathy, diabetic retinopathy, foot ulceration and nephropathy. Ladies have been observed with high rate of hypomagnesaemia in comparison to men with a ratio of (2:110).

MATERIAL & METHODOLOGY:

Sample Size: Sample size included diabetic and non-diabetic respectively 59 and 24 cases, non-diabetic cases were considered as controls.

Data source: Source of the collected data was BVH, Bahawalpur.

Study Design: Design of the research was observational and descriptive.

Period: Research was completed in the period of six months.

Data Collection: Multiple questions were included in the questionnaire of our research inquiring about lifestyle, diet and illness complications.

Laboratory Procedure: In our research we took a quantity of (5 ml) blood sample of the patients in the vaccinators without the addition of any additive. Centrifuge process was carried out on the samples at the rate of (3000 rpm) in a centrifuge machine. Eppendorf tube was used for the collection of the serum and these tubes were numbered respectively including the basic information of the patient such as name. Analysis of level of magnesium was also carried out with the help of the spectrophotometric method in the department of the Biochemistry. Further analysis was carried out through SPSS-20.

RESULTS:

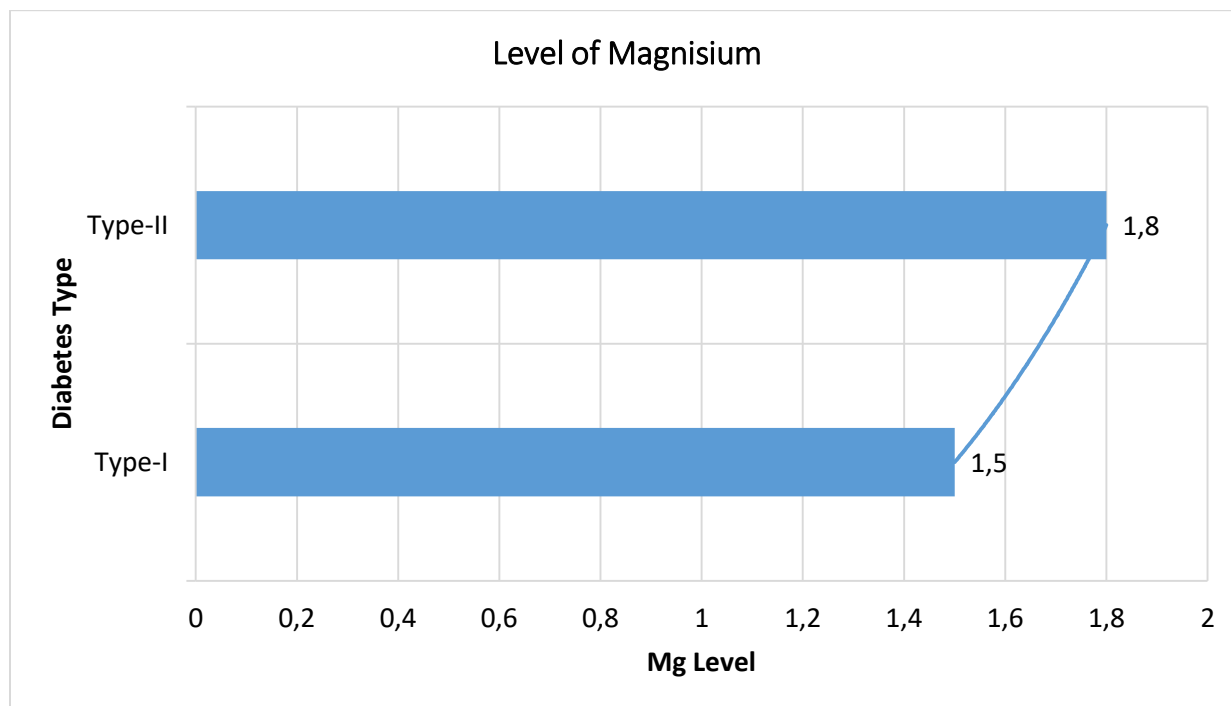
Research sample was 59 patients including females and male with the respective proportions of 41 and 59 percent. Furthermore, the proportion of the T2DM and T1DM were respectively 47 cases (80%) and 12 cases (20%). In the control group we included twenty non-diabetic cases. It was observed that level of random glucose serum was extraordinarily high, that was 334.4 ± 46.8 in the T1DM cases having a number of twelve. A moderate increase was observed in the T2DM with forty-seven cases as (253 ± 16.8) . Random glucose serum was observed normal in the non-diabetic cases with a number of twenty-four cases. Levels of the HbA1c were observed high in the

T1DM cases as (11.9 ± 0.9) . Same was significantly high in the cases of T2DM as (12.6 ± 2.9) and the level of the HbA1c was observed normal in non-diabetic patients. Level of The serum magnesium were observed low in the patients of T1DM as (1.5 ± 0.1) , same was low in the T2DM cases as (1.8 ± 0.3) ; contrarily normal in non-diabetic cases as (2.0 ± 0.1) . Table clears the concept of the mild – moderate diabetes cases in terms of their type and comparison to the controls as there is a close correlation of the levels of HbA1c and magnesium in serum. Good glycemic control (5.8) in controls associates with good magnesium levels of body as 2 mg/dl. Whereas, poor glycemic control in the patients of diabetes

(high HbA1c of 11.9 in Type-I & 12.6 in Type-II cases) is responsible for the intense hypomagnesaemia respectively 1.5 & 1.8 mg/dl. Type I and II cases significantly reflect the increase in the level of glucose. Level of hypomagnesaemia in our cases indicate a positive association between diabetes severity & hypomagnesaemia status. Normal glucose level and magnesium level equal or greater to 2 mg/dl in controls clearly establishes hypomagneseemia implicated role in the cases of diabetes. Type I & II also reflect a decrease in magnesium level that indicates need of magnesium for insulin synthesis but it also plays a vital role in the sensitivity of insulin receptor.

Table 1. Shows the magnesium levels, random sugar levels and glycosylated hemoglobin levels (HbA1c) of the cases and controls

Group	Cases		Controls
Value	Mean \pm SD		
Type	Type-I	Type-II	Number
Number	12	47	23
Serum Glucose	334.4 ± 46.8	253 ± 16.8	104 ± 1.4
HbA1c	11.9 ± 0.9	12.6 ± 2.9	5.8 ± 0.1
Mg	1.5 ± 0.1	1.8 ± 0.3	2.0 ± 0.1



DISCUSSION:

Magnesium is considered as an intracellular cation, as there is total (< 2 %) body concentration in ECF. Numerous research studies have also reflected about the deficiency of magnesium as the new risk factor in the T2DM cases and a presentation of the strong T2DM relation with low level of Mg serum. Similarly, in other research studies there is a relation

of the magnesium effect on secretion of insulin through pancreas and peripheral tissues cause insulin responsiveness. A Libyan research observed concentration of Mg as $(0.74 \pm 0.10 \text{ mmol/L})$. The cases using metformin, β adrenergic receptor and proton pump inhibitors were observed with a decreased level of plasma Mg^{+2} . Our research reflected for Type-I & II respectively (1.5 ± 0.1) and

(1.8 ± 0.3) but for the controls (2.0 ± 0.1). In the result of an examination held in U.K. the level of Serum Mg level in T2DM in terms of its association with glycosylated hemoglobin, 38% cases presented level of serum Mg as < 1.8 mg/dl with no change in the already admitted and new patients respectively 41 and 35 percent. Our research found about T1DM level of Mg as (1.5 ± 0.1) & T2DM as (1.8 ± 0.3) and Mg level with diabetes presented negative association with duration that was measured through HbA1c and close negative association in terms of blood sugar fasting. There was no significant relation between level of Mg^{2+} and age or BMI. It was observed in our study that values of HbA1c in T1DM including SD was (11.9 ± 0.9) and same for T2DM as (12.6 ± 2.9); whereas, in controls as (5.8 ± 0.1).

Diabetes occurrence can be prevented through the supplementation of Magnesium as observed in the model of a rat with T2DM. Numerous other research studies have presented the link of regular Mg intake association with reduced insulin response as there is a direct association between Mg level and diabetes; whereas, glucose is independent of the secretion of insulin. Magnesium is considered as an intracellular cation, as there is total ($< 2\%$) body concentration in ECF. Numerous research studies have also reflected about the deficiency of magnesium as the new risk factor in the T2DM cases and a presentation of the strong T2DM relation with low level of Mg serum. In other observational research studies the hazard of atherosclerosis has a concrete connection; however, low level of serum Mg^{2+} & T2DM. In the vitro views it has been demonstrated that magnesium impact on insulin discharge by pancreas and responsiveness to insulin is because of fringe-tissues. Another study held on controls observed hypomagnesaemia in T2DM is associated to Insulin Resistance, mean age and SD in T2DM cases was (50.5 ± 11.2) and in sound controls mean age and SD was (46.8 ± 11.7). Serum Mg level were observed by some scholars having converse association with blood glucose fasting concentration and HbA1c concentration. Our research exposed a higher hypomagnesaemia predominance with HbA1c more than 7% & FBS more than 130 mg/dl. A research held at Hyderabad (LUMHS) observed that Hypomagnesaemia in DM cases in terms of geographical distribution, rural area was more involved as (76%), mean random level of blood glucose as (220.52 ± 13.68), in contrast to the observations of our research for T1DM it was (334.4 ± 46.8) while in T2DM observed as (253 ± 16.8). Other results are also as reflected in Table-I and Figure-I in terms of Mg level in Type I & II DM, glycemetic control, HbA1c and Mean \pm SD. In few other clinical investigations, the supplementation of the Mg can enhance the reaction of the insulin in

individuals suffering from diabetes and an immediate association is present between level of serum Mg and transfer of cell glucose that is insulin secretion free. Glucose transfer adjustment appeared to be recognized with expanded tissue affectability in the closest Mg level that is considered sufficient.

CONCLUSION:

Hypomagnesaemia is linked with aggravation and development of Type I & II DM severity. Its consequences in severity of disorders can be reduced in a drastic way through the diets of magnesium fortified administrated to diabetes cases. Furthermore, in the upcoming research studies hypomagnesaemia hazardous implications scope may be focused for the complication alleviation in diabetes cases.

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