Original Research Paper



CORRELATION OF SERUM MG²⁺ LEVELS WITH DURATION OF T2DM

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ABSTRACT INTRODUCTION: Diabetes is the most common serious metabolic disease. The hallmark of Diabetes is elevated blood glucose concentration, just one of many biochemical & physiological alterations that occur in the disease. In Diabetics there is a DIRECT association between serum magnesium levels & cellular glucose disposal independent of insulin secretion. This change in glucose disposal is found to be associated with INCREASED SENSITIVITY of the tissues to insulin in the presence of sufficient magnesium levels. Magnesium deficiency has been found to be related with DIABETIC MICROVASCULAR DISEASE. Hypomagnesaemia has been reported to occur in 25-38% patients with T2DM without metabolic control. Hence the present study is conducted to find any correlation with DURATION of T2DM & Serum Magnesium¹

KEYWORDS: T2DM, Serum Mg²⁺,T2DM Duration

MATERIALS & METHODS:

INCLUSION: T2DM 50 Subject

EXCLUSION: T2DM On Insulin,T2DM with Hypothyroid, Hypertension, Nephropathy, Neuropathy, Retinopathy, CHF & Renal diseases.

CONTROL: 50 Healthy subjects

Fasting /Postprandial blood glucose measured :Automated calorimeter Serum Magnesium levels measured : Calmagnite dye method, Normal=1.8-2.5mg/dl

STATISTICAL ANALYSIS: Student t test

RESULTS:

Table no:1 Showing serum Mg²⁺ levels compared with that of duration of T2DM, as longer the duration the less is the serum Mg²⁺ levels in study group compared with that of control group ie 1.46mg/dl compared to 2.51mg/dl in control group.

DURATION OF STUDY GROUP	SERUM Mg ²⁺ LEVELS (mg/dl)	CONTROL GROUP SERUM Mg ²⁺ levels (mg/dl)
2-5 YEARS	2.26mg/dl	2.51mg/dl
10-15 YEARS	2.16mg/dl	2.51mg/dl
_ 20 years	1.46mg/dl; p Value significant < 0.0001	2.51mg/dl

Table no:2 Showing the comparison of fasting along with postprandial blood glucose compared with that of duration.

The longer the duration ie more than 20years the FBG-232.41±17.02 and PPBG-269.38±14.12

DURATION T2DM	FASTING BLOOD GLUCOSE mg/dl	POSTPRANDIAL BLOOD GLUCOSE mg/dl
2-5YEARS	101±14.0mg/dl	130.22±8.2mg/dl
10-15YEARS	162±12.05mg/dl	200±12.01mg/dl
_ 20YEARS	232.41±17.02 mg/dl	269.38±14.12mg/dl

American Diabetes Association Criteria: Normal FBS: >126mg/dl, PPBG:>200mg/dl 2 (9 recheck ADA criteria of blood glucose levels)

Table no.3 Comparison of Serum Mg2+ with duration & blood glucose levels

DURATION OF T2DM	FASTING BLOOD GLUCOSE mg/dl	POSTPAND RIAL BLOOD GLUCOSE mg/dl	SERUM Mg ²⁺ mg/dl	p Value
2-5years	101±14.0	130.22 ±8.2	2.26mg/dl	0.38
10-15years	162±12.05	200± 12.01	2.16mg/dl	0.25
	232.41± 17.02	269.38±14.1 2	1.46mg/dl	<0.0001

DISCUSSION.

- Diabetes- a potential epidemic in India with 62 billion currently. According to WILD 2 et al prevalence of diabetes estimated to double globally from 171 million to 366 MILLION BY 2030 with MAXIMUM rise in India.
- Magnessium --4th most abundant cation in human body 2nd most abundant intracellular cation An important COFACTOR for many enzymatic reactions especially phosphate transfer along with regulation of glucose metabolism3
- Present study exhibit longer the duration along with raise FBG & PPBG had a low serum Mg2+ correspondingly A.G.Kulkarni et al concluded serum Mg2+ levels was lower in T2DM. Mohamed Murtuza Kause et al showed serum Mg2+ levels lower in T2DM.
- 4. Similarly Sharma A et al study concluded poor glycemic control was ASSOCIATED with hypomagnesaemia.
- Mg2+ in insulin sensitivity: hypomagnesemia is associated with increased insulin resistance in T2DM 5-7
- 6. The crystal structure of insulin receptor tyrosine kinase shows two Mg2+ ions bind to tyrosine kinase domain as shown in Fig No.1 8 Mg2+ being ESSENTIAL autophosphorylation of β -subunit of insulin receptor.
- 7. Depending on target tissue, direct substrate of insulin receptor may recruit the receptor. Insulin receptor substrate phosphorylate downstream signalling pathway which are (PIP3,PKC,Grb2)13 resulting in glucose uptake, glycogenesis, lipid synthesis, cell growth and cell differentiation.
- Alternatively , insulin receptor act independently via Srchomology 2domain called MAP13 resulting in cell signalling and regulation of cell proliferation9-12 as shown in Fig No.1

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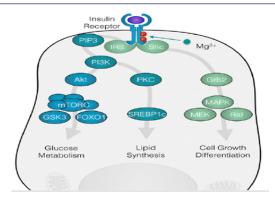


Fig.no: 1 Mg2+ regulates insulin signalling pathway.

CONCLUSION:

- Serum Mg²⁺ levels decreased with increased duration T2DM.
- Mg²⁺ deficiency usually presents as chronic latent Mg²⁺ deficit rather than clinical hypomagnesemia³
- HYPOMAGNESEMIA CAUSES INSULIN RESISTANCE WHICH FURTHER REDUCES SERUM Mg²⁺ LEVELS IN T2DM CREATING A VICIOUS CYCLE⁴
- Accurate measurement of intracellular and extracellular Mg²⁺ levels- A TOOL TO TRANSLATE KNOWLEDGE of magnesium and glucose in insulin metabolism INTO CLINICAL PRACTICE
- However the picture is far from complete and more research would be enforced to distinguish the complex yet dynamic role of Mg2+ in T2DM



Mg²⁺ deficiency is a CAUSE or EFFECT OF T2DM..?

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 Philadelphia. Saunders. 2006
- MAP- Mitogenic activated protein, PKC-protein kinase c, Grb2- growth factor receptor bound-2 an adaptor protein for cell communication, PIP3phosphatidylinositol3,4,5triphosphate
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