



A STUDY OF SERUM MAGNESIUM PROFILE IN TYPE – 2 DIABETES MELLITUS

General Medicine

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ABSTRACT

Serum hyperglycaemia resulting from defects in insulin secretion, insulin action (may be due to failure of complex insulin – receptor interaction) or both. According to the International Diabetes Federation (IDF), diabetes has affected at least 300 million people worldwide, and this number is expected to reach 450 million by the year 2030. There are mainly two types of diabetes Type-1 and Type-2, the latter type of diabetes mellitus is the most common form of diabetes accounting for 90% of the cases. In India, the scenario is not different when compared to the world scenario. The best strategies are not able to prevent the complications suggesting that alternative treatment strategies are needed. Insulin-dependent uptake of glucose is reduced in magnesium deficiency. Magnesium supplementation improves glucose tolerance. So one such treatment strategy under research is supplementation of magnesium and its role in primary prevention of diabetes and slowing the progression of complications that is related to the dreaded disease. So before anything it is best to understand the profile of serum magnesium in type 2 Diabetes mellitus. So this study is dedicated for this above said cause.

KEYWORDS

Diabetes, Magnesium, Balance, Serum Levels

INTRODUCTION:

Serum hyperglycaemia resulting from defects in insulin secretion, insulin action (may be due to failure of complex insulin – receptor interaction) or both.^[1]

According to the International Diabetes Federation (IDF), diabetes has affected at least 300 million people worldwide, and this number is expected to reach 450 million by the year 2030.^[2] there are mainly two types of diabetes Type-1 and Type-2, the latter type of diabetes mellitus is the most common form of diabetes accounting for 90% of the cases.^[3] In India, the scenario is not different when compared to the world scenario.

The best strategies are not able to prevent the complications suggesting that alternative treatment strategies are needed.^[4] Insulin-dependent uptake of glucose is reduced in magnesium deficiency. Magnesium supplementation improves glucose tolerance. Magnesium, is an essential mineral needed by human body.^[5] It is also the fourth most abundant cation in the human body.^[6] Magnesium orally produces diarrhoea; but intravenously it produces CNS depression.^[7] It is also the activator of many enzymes requiring ATP. Alkaline phosphatase, hexokinase, fructokinase, phosphofructokinase, adenylyl cyclase, cAMP dependent kinases, etc. need magnesium.^[7] It is believed to play a role in glucose homeostasis, insulin action, and the development of type 2 diabetes.^[8] It may influence insulin secretion by interacting with cellular calcium homeostasis.^[9] Magnesium can function as a mild, natural calcium antagonist. So the level of intracellular calcium is increased in Mg-deficiency subjects. This increased intracellular calcium may compromise the insulin responsiveness of adipocytes and skeletal muscles leading to the development of insulin resistance.^[10] Another study has also found that insulin deficiency or insulin resistance can affect the tubular absorption of Mg, leading to hypomagnesaemia in diabetic subjects.^[11] Thus, a vicious circle is formed by mutual influence between insulin resistance and hypomagnesaemia resulting in aggravation of insulin resistance.^[12] Mg has been reported to possess antioxidant property.^[13]

The dietary recommendation (Recommended Dietary Allowances/RDA) for magnesium is 400 to 420 mg daily for adult men and 310 to 320 mg daily for adult women.^[14]

The best strategies are not able to prevent the complications suggesting that alternative treatment strategies are needed. Insulin-dependent uptake of glucose is reduced in magnesium deficiency. Magnesium supplementation improves glucose tolerance. So one such treatment strategy under research is supplementation of magnesium and its role in primary prevention of diabetes and slowing the progression of

complications that is related to the dreaded disease. So before anything it is best to understand the profile of serum magnesium in type 2 Diabetes mellitus. So this study is dedicated for this above said cause.

Aims and Objectives:

To study the serum magnesium profile in Type 2 Diabetes Mellitus.

Materials and Methods:

This study was done in the Department of General Medicine, from Feb 2014 to Feb 2015.

This study was done in Shree Siddhartha Medical College, Agalakote, Tumkur.

One hundred subjects who were diagnosed to be diabetic were included for the study. They were compared with the normal 100 patients.

The complete history was taken and the relevant data was looked upon and noted. The blood was collected and was sent to the Department of Biochemistry for Magnesium level analysis.

Inclusion criteria:

Patients who are confirmed type – 2 diabetics.

Exclusion Criteria:

Patients who were on magnesium supplementation.

RESULTS:

Table 1: Age Distribution

Age	Males	Females
20-30 years	03	Nil
30-40 years	10	08
40-50 years	08	21
50-60 years	13	17
69-70 years	07	04
70-80 years	04	05

Table 2: Gender Distribution

Males	Females
45	55

Table 3: Mean Magnesium levels

	Normal	Diabetic	P
	Mean ± sd	Mean ± sd	Value
Mean Magnesium Levels	2.1±0.12	1.1±0.17	<0.001

Table 4: Mean serum magnesium levels

Age	Males	Females	Mean magnesium levels
20-30 years	03	Nil	1.7±0.17
30-40 years	10	08	1.62±0.18
40-50 years	08	21	1.15±0.18
50-60 years	13	17	1.11±0.43
69-70 years	07	04	1.12±0.63
70-80 years	04	05	1.12±0.11

DISCUSSION:

Magnesium is the fourth most abundant cation in the body and second most prevalent intra - cellular cation. Magnesium is mainly seen in intracellular fluid. Total body magnesium is about 25 grams, Sixty percent of which is complexed with calcium in bone. One-third of skeletal magnesium is exchangeable with serum. Magnesium orally produces diarrhoea; intravenously it produces CNS depression.

The requirement of the human body is about 400 mg/day for men and 300 mg/day for women. Above 600 mg may cause diarrhoea. More amount of magnesium is required during lactation. Some of the best sources of magnesium are cereals, beans, leafy vegetables and fish.

Normal serum magnesium is 1.8 - 2.2 mg/dL. Inside the RBC, the magnesium content is 5 mEq/L. In muscle tissue, is 20 mEq/L. About 7000 of magnesium exists in free state and remaining 30% is protein-bound (2500 to albumin and 500 to globulin). Serum must be separated from the clot as soon as possible or the level of magnesium will increase because of its elution from the red blood cells. Hemolyzed samples as well as blood collected with citrate, oxalate or EDTA are unacceptable for analysis. Homeostasis is maintained by intestinal absorption as well as by excretion by kidney. Magnesium is reabsorbed from loop of chlc and not from proximal tubules. Functions of Magnesium are, it is the activator of many enzymes requiring ATP. Alkaline phosphatase, hexokinase, fructokinase, phosphofructokinase, adenyl cyclase, CAMP dependent kinases, etc. need magnesium. Neuromuscular irritability is lowered by magnesium. Insulin-dependent uptake of glucose is reduced in magnesium deficiency. Magnesium supplementation improves glucose tolerance.

Hypomagnesemia is commonly seen in hospital patients. Conditions which require magnesium estimation are many. When serum magnesium level falls below 1.7 mg/dl, it is called hypomagnesemia. Vomiting, nasogastric suction, diarrhoea, liver cirrhosis, protein-calorie malnutrition and diuretic therapy are the common causes (see Box 35.18). Urinary loss can occur in alcoholism, osmotic diuretics, loop diuretics and amino-glycosides. Serum magnesium levels need not always reflect body content. Measurement of urinary magnesium excretion will distinguish between renal and gastrointestinal losses.

Deficiency of magnesium leads to neuromuscular hyper-irritability and cardiac arrhythmias. The magnesium deficiency symptoms are similar to those of calcium deficiency; but symptoms will be relieved only when magnesium is given. Acute symptomatic deficiency is treated by giving parenteral magnesium. Oral therapy may lead to diarrhoea, intravenous magnesium sulfate is given for the treatment.

Hypermagnesemia is uncommon and always due to excessive intake either orally (antacids) rectally (enema) or parenterally. Causes of hypomagnesaemia are many including excessive antacid treatment. Magnesium intoxication causes depression of neuro - muscular system, causing lethargy, hypotension, respiratory depression, bradycardia and weak tendon reflexes. In severe conditions acute rhabdomyolysis results. So there exists a fine balance which is definitely broken in diabetes mellitus so magnesium supplementation is recommended highly and may be helpful in treating the Diabetes Mellitus.

CONCLUSION:

Magnesium levels in diabetes is deranged and it has to be replenished which is ideal for the treatment of Diabetes Mellitus.

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