STUDY OF SERUM MAGNESIUM LEVEL IN TYPE 2 DIABETES MELLITUS

Nagarajan K[1]

[1]Professor, Department of Medicine, Sri Manakula Vinayagar Medical College and Hospital.

ABSTRACT

BACKGROUND
A high prevalence of magnesium deficiency is reported in diabetics. Magnesium depletion has a negative impact on glucose homoeostasis and insulin sensitivity in type 2 diabetic patients as well as on the evolution of complications such as retinopathy, nephropathy, neuropathy and arterial atherosclerosis.

The aim of this study is to estimate the prevalence of hypomagnesaemia in patients with type 2 diabetes mellitus and its correlations with microvascular complications of diabetes like retinopathy, nephropathy, neuropathy.

MATERIALS AND METHODS
Patients with type 2 diabetes admitted in Thanjavur Medical College and Hospital over a period of one year from October 2011 to October 2012 formed the study population. The sample size was 100 patients. Serum magnesium concentration was measured by calmagite dye method.

RESULTS
The study revealed that prevalence of hypomagnesaemia in study subjects was 35%. Sex, age and duration of diabetes were not significant predictors of serum magnesium. Significant association was found between hypomagnesaemia and diabetic retinopathy, nephropathy, neuropathy. Significant correlations were not found with comorbidities such as ischaemic heart disease and hypertension. Low serum magnesium concentrations are common in type 2 diabetics. Magnesium deficiency is conclusively associated with diabetic retinopathy, nephropathy, neuropathy.

KEYWORDS
Magnesium, Diabetes Mellitus, Retinopathy, Nephropathy, Neuropathy.

BACKGROUND
India is frequently referred to as the diabetic capital of the world as it has the highest number of cases in the world.

Worldwide, in the last 2 decades, incidence has suddenly increased from 30 million cases in 1985 to 171 million in 2000. Recent data suggests that prevalence of DM by the year 2030 could be 360 million. DM is worldwide in distribution and the incidence of both types is rising.[1,2]

The distribution of both T1DM and T2DM varies worldwide, due to relative difference in genetic and environmental factors in different parts of the world. Recent data shows it is associated with 10-30% reduction of life expectancy, most common cause of blindness in the age group of 20 to 65 years, 25-fold increased risk of non-traumatic lower limb amputations and increased incidence of end stage renal disease approximately 1000 patients per year.[2] India had around 31.7 million cases in year 2000 which is expected to rise alarmingly to around 79.4 million in 2030 by which time every fifth diabetic subject in the world would be an Indian.[3]

In Tamilnadu, the prevalence in 2008 is 18.6% in urban areas and 9.1% in rural areas.[3]
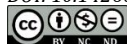
Although DM can cause hypomagnesaemia, low serum magnesium level is a risk factor for DM. Magnesium is an essential element for several enzymes that plays important role in glucose metabolism. Animal studies found that low magnesium has a negative effect on post receptor signalling of insulin. Some studies have also found that magnesium supplementation improves insulin action and glucose metabolism in diabetics.[4]

Magnesium is involved in multiple levels of insulin secretion. Magnesium deficiency can modify the Na+ K+ ATPase channel that maintains sodium, potassium and glucose transport.[5]

In DM, there is a direct correlation between serum magnesium concentration in blood and cellular glucose disposal that is independent of insulin secretion.[6]

Low serum magnesium level has direct correlation with microvascular complications (Retinopathy,[7] neuropathy. nephropathy) and macrovascular complications (IHD and cerebrovascular disease).[8]

In elderly type 2 DM, Paolisso hypothesised that oral magnesium supplementation for 4 weeks results in decreased fasting blood sugar level, increased plasma and RBC magnesium level.[9]

In this study, serum magnesium concentration in type 2 DM patients is correlated with occurrence of microvascular complications.

Objectives
The Aims of this study are
1. Measurement of serum magnesium concentration in diabetes mellitus.

2. To estimate the prevalence of hypomagnesaemia in patients with type 2 diabetes mellitus.
3. Its correlation with microvascular complications like retinopathy, nephropathy, neuropathy.

MATERIALS AND METHODS
Source of Data
Patients with type 2 diabetes admitted in Thanjavur Medical College & Hospital, Thanjavur who satisfied the inclusion criteria and consented to participate in the study were included.

Period of Study
October 2015-October 2015.

Type of Study
Descriptive Study
100 patients were randomly selected of which 50 were male and 50 were female. Cases with renal failure, acute coronary syndromes, patients on diuretics, alcoholics or with malabsorption were excluded. None were taking magnesium supplements or magnesium containing antacids. Informed consent was obtained.

Inclusion Criteria
All patients both males and females above 13 years of age group with type 2 diabetes mellitus admitted in Internal Medicine units of Thanjavur Medical College & Hospital, Thanjavur.

Exclusion Criteria
1. Patients with chronic renal failure.
2. Acute myocardial infarction in last 6 months.
3. Patients on diuretics.
4. Patients with history of alcohol abuse.
5. Patients receiving magnesium supplements or magnesium containing antacids.
6. Malabsorption or chronic diarrhoea.
7. Age <13 years.

Data Collection
The 100 diabetics (50 Men & 50 Women) were included in the study. Detailed history - including duration of diabetes, treatment mode, symptoms suggestive of diabetic neuropathy, associated diseases such as hypertension and ischaemic heart disease was obtained as per the proforma.

Detailed physical and neurological examination was done. Retinopathy was assessed by direct ophthalmoscopy.

Samples were collected for estimation of fasting blood glucose and magnesium level. Postprandial blood sugar was measured two hours after a standard meal. Nerve conduction study was done on selected patients by an experienced neurologist with symptoms & signs suggestive of neuropathy. Blood urea, serum creatinine and 24-hour urinary albumin were estimated. Serum magnesium was estimated by calmagite dye method. HbA1C measurement done by a modified calorimetric method.

Calmagite Dye Method- Test Principle
Under alkaline conditions, magnesium combines with calmagite dye to form a red colour which is read spectrophotometrically at 530 nm. Formation of colour depends on magnesium levels. To eliminate the interference of calcium during estimation, EDTA is included in the reagent. Cyanide reduces heavy metal interference. Surfactant reduces protein interference.

Test Procedure
Three test tubes labelled Blank, Standard and Test are prepared as in table.

In test tubes	Blank	Standard	Test
Calmagite	1 mL	1 mL	1 mL
Standard sample	-	10 mL	-
Patient's sample	-	-	10 mL
Distilled water	10 mL	-	-

This test tubes are incubated at room temperature (22-28°C). The absorbance of Test (A) T, Standard (A) S and Blank (A) B are read at 530 nm in TSB spectrophotometer. Magnesium concentration is calculated by the following formula.

$$\text{Magnesium concentration (mEq/L)} = (A_T - A_B \ / \ A_S - A_B) \times 2$$

Serum magnesium concentration is expressed in mg/dL by linearity of 1 mEq/L = 1.2 mg/dL.

According to Magnesium Levels Patients were classified into-
1. Normal 1.7 to 2.4 mg/dL,
2. Low <1.7 mg/dL,
3. High >2.4 mg/dL.

Patients were also categorised on the basis of duration of diabetes, presence of ischaemic heart disease or hypertension, mode of treatment, presence/absence of retinopathy, neuropathy and nephropathy, and glycaemic control (FBS and HbA1C).

Cases with Diabetic Retinopathy were Further Divided into
a. Non-proliferative diabetic retinopathy.
b. Proliferative diabetic retinopathy.

Diabetic Nephropathy was graded depending on 24-hour Urinary Excretion of Albumin as follows-
No nephropathy, < 30 mg/24 hours.
Microalbuminuria 30 – 299 mg/24 hours.
Macroalbuminuria (clinical proteinuria) >= 300 mg/24 hours.

Statistical Analysis
The statistical analysis was done by SPSS 15 software. MS Word and Excel were used to generate tables and charts. Following tests were used-
- Chi square test.
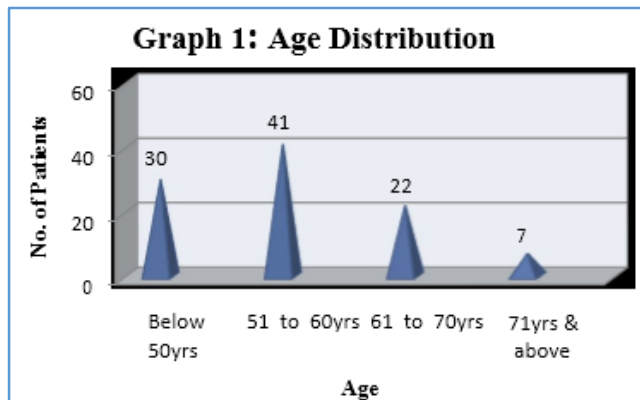- Student T test.
- One way ANOVA test.

Statistical results were considered significant at P<0.05.

RESULTS

100 cases of type 2 DM (50 males, 50 females, mean age 56.87 years) comprised the study group.

| Age (in years)	No. of Patients (n = 100)
Below 50 yrs.	30
51 to 60 yrs.	41
61 to 70 yrs.	22
71 yrs. & above	7
Table 1. Age Distribution	

Graph 1: Age Distribution

Patients were distributed across the age spectrum of 42 to 78 years. Mean age 56.87 years. Most patients (n=41) were present in 51-60 years age group. Youngest patient was 42 years old.

| Characteristics	
No. of Subjects	100
Age (Years)	56.87 (42 - 78)
Men	50
Women	50
Duration of diabetes (Years)	8.89 (2-23)
Medication	
Oral Hypoglycaemics	67
Insulin and Oral Hypoglycaemics	33
Diet only	0
Comorbidities	
Hypertension	30
Ischaemic Heart Disease	15
Diabetic Retinopathy	
NPDR	33
PDR	2
Diabetic Neuropathy	15
Diabetic Nephropathy	
Microalbuminuria	29
Macroalbuminuria	4
Poor glycaemic control	39
Table 2. Characteristics of Study Population	

Graph 2: Characteristics of study Population
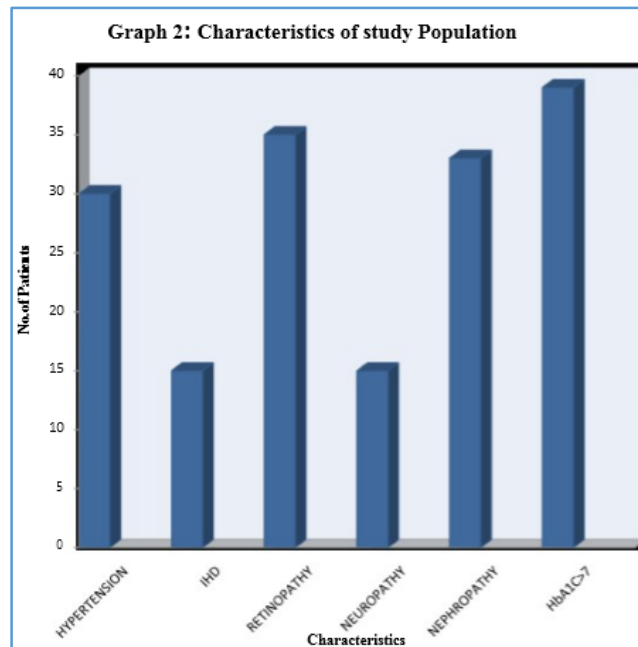
The average duration of diabetes in study population was 8.89 years and range was 2 years to 23 years. 67 patients received only oral hypoglycaemic agents and 33 patients received both. 30 patients had hypertension and 15 patients had ischaemic heart disease and 55 patients had no comorbidities. Total 35 patients had diabetic retinopathy. Total of 15 patients had diabetic neuropathy. 33 patients had nephropathy.

Sex	Magnesium		Statistical Inference
	Normomagnesaemia (n = 65)	Hypomagnesaemia (n = 35)	
Male	34 (52.3%)	16 (45.7%)	X^2 = .396
			Df = 1
			.529 >0.05
			Not Significant
Female	31 (47.7%)	19 (54.3%)	

Sl. No	MG	Mean	S.D	Statistical Inference
1	Male (n = 50)	0.492	0.7253	T=-.622 df=98
				.536>0.05
2	Female (n = 50)	0.584	0.7547	Not Significant
	Table 3. Prevalence of Hypomagnesaemia			

Graph 3: Prevalence of Hypomagnesemia

- Hypomagnesemia 35%
- Normomagnesemia 65%
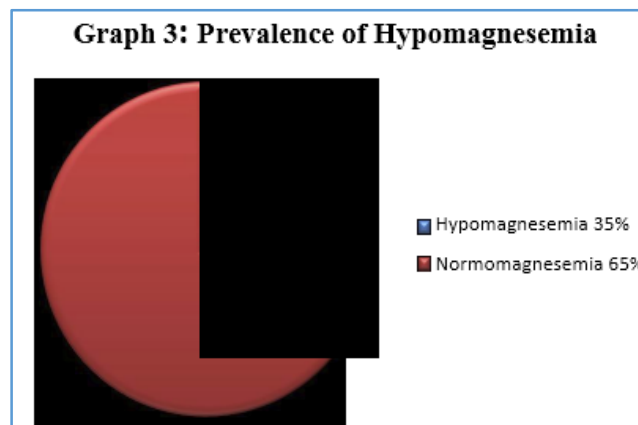
Hypomagnesaemia was found in 35 patients. 65 patients had normomagnesaemia No patient had hypermagnesaemia. No correlation was found between hypomagnesaemia in men and women (45.7% and 54.3%% respectively).

Sl. No	MG	Mean	S.D	SS	Df	MS	Statistical Inference
1	Between Groups			32.917	4	8.229	F = 13.265 0.060 (>0.05) Not Significant
2	Below 5 yrs. (n = 45)	0	0				
3	6 to 10 yrs. (n = 25)	0.804	0.7898				
4	11 to 15 yrs. (n = 15)	1.4333	0.4012				
5	16 to 20 yrs. (n = 10)	1.22	0.646				
6	21 yrs. & above (n = 5)	0	0				
7	Within Groups			20.979	95	0.221	
	Table 4. Prevalence of Hypomagnesaemia and Duration of Diabetes One way ANOVA						

Sl. No.	Duration (Years)	Magnesium		Statistical Inference
		Normomagnesaemia (n = 65)	Hypomagnesaemia (n = 35)	
1	Below 5 yrs.	46 (70.8%)	2 (5.7%)	X^2 = 4.568 Df = 4 0.067 (>0.05) Not Significant
2	6 to 10 yrs.	12 (18.5%)	13 (37.1%)	
3	11 to 15 yrs.	1 (1.5%)	11 (31.4%)	
4	16 to 20 yrs.	2 (3.1%)	8 (22.9%)	
5	21 yrs. & above	4 (6.2%)	1 (2.9%)	

Graph 4: Prevalance of Hypomagnesemia and Duration of Diabetes
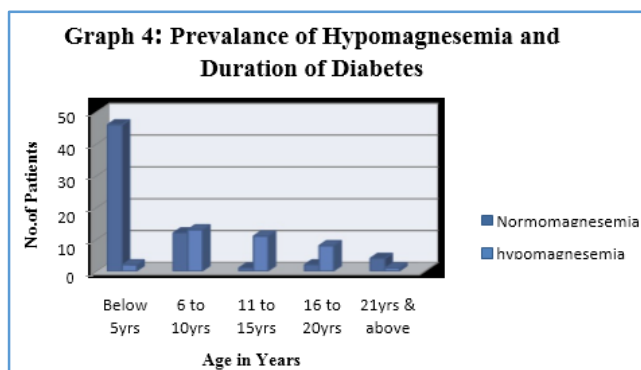
Chi- square ($\chi 2$) value is 4.568. So, the correlation is insignificant p value. So the duration of diabetes does not significantly predict serum magnesium concentration. One way ANOVA F=13.265 in between groups also shows no significant correlation. The mean duration was 8.89 years (2-23).

Graph 5: Prevalence of Hypomagnesemia

Insulin treated - Non-insulin treated

Sl. No	FBS	Magnesium		Statistical Inference
		Normomagne-saemia (n = 65)	Hypomagne-saemia (n = 35)	
1	Below 90	8 (12.3%)	2 (5.7%)	X^2=45.582 Df=5 0.000 (<0.05) Significant
2	91 to 100	11 (16.9%)	0	
3	101 to 110	10 (15.4%)	9 (25.7%)	
4	111 to 120	19 (29.2%)	1 (2.9%)	
5	121 to 130	13 (20%)	2 (5.7%)	
6	131 & above	4 (6.2%)	21 (60%)	
	Table 6. Prevalence of Hypomagnesaemia and Fasting Blood Sugar			

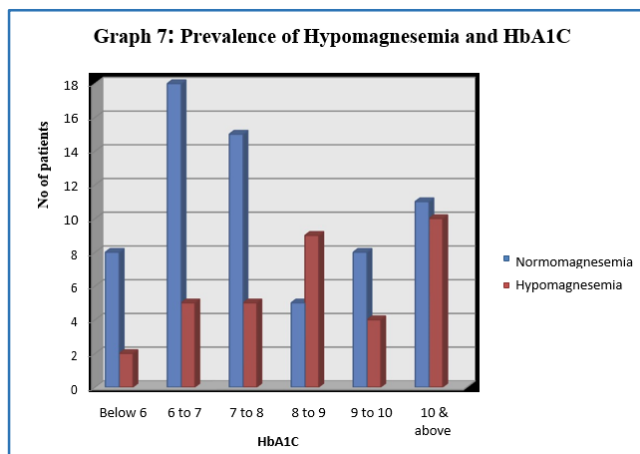Graph 6: Prevalence of Hypomagnesemia and Fasting Blood Sugar
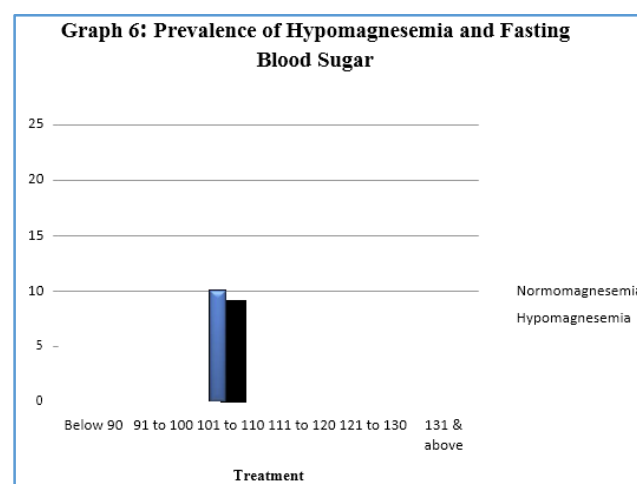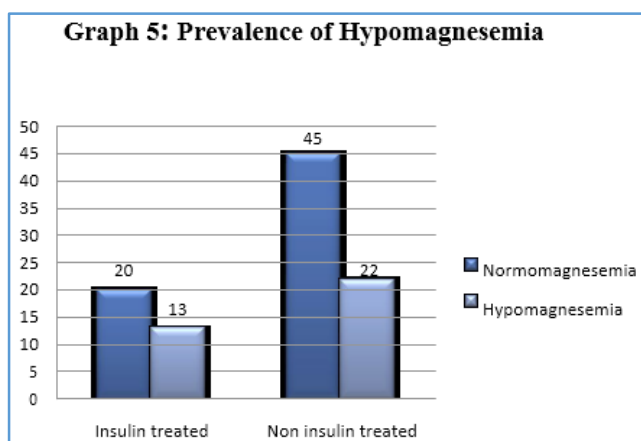
Prevalence of hypomagnesaemia is high when fasting blood sugar >131(60%). The chi-square ($\chi 2$) value is 45.582. So the correlation is significant. So, fasting blood sugar can significantly predict serum magnesium concentration.

| Sl. No. | HbA1C | Magnesium | | Statistical Inference |
		Normomagne-saemia (n = 65)	Hypomagne-saemia (n = 35)	
1	Below 6	8 (12.3%)	2 (5.7%)	X^2 = 10.408 Df = 5 0.064 (>0.05) Not Significant
2	6 to 7	18 (27.7%)	5 (14.3%)	
3	7 to 8	15 (23.1%)	5 (14.3%)	
4	8 to 9	5 (7.7%)	9 (25.7%)	
5	9 to 10	8 (12.3%)	4 (11.4%)	
6	10 & above	11 (16.9%)	10 (28.6%)	
Table 7. Prevalence of Hypomagnesaemia and HbA1c				

Serum magnesium concentration showed no significant association with HbA1C. Chi square X^2=10.408, Df=5, P value 0.064. Higher prevalence of hypomagnesaemia observed in HbA1c > 10 (28.6%).
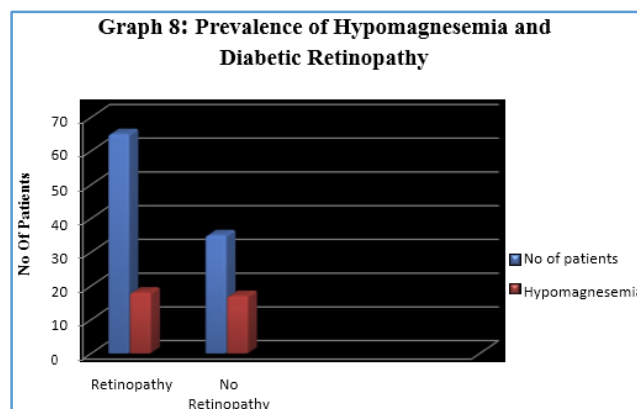

Graph 7. Prevalence of Hypomagnesaemia and HbA1C

	No. of Patients	Percentage
Particulars	(n = 100)	100%
No Retinopathy	65	65
NPDR	33	33
PDR	2	2
Table 8. Prevalence of Hypomagnesaemia and Diabetic Retinopathy		

| Sl. No. | Retino-pathy | Magnesium | | Statistical inference |
		Normomagne-saemia (n = 18)	Hypomagne-saemia (n = 17)	
1	NPDR	18 (100%)	15 (88.2%)	X^2 = 4.746 Df=1 0.034(<0.05) Significant
2	PDR	0	2 (11.8%)	

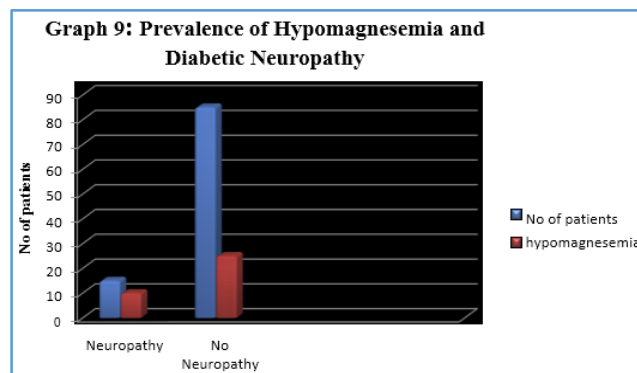

Observations revealed a definite correlation between hypomagnesaemia and diabetic retinopathy. The chi-square (χ2) value is 4.746. Df =1 .p value is 0.034.

Particulars	No. of patients (n = 100)	Percentage (100%)
No neuropathy	85	85
neuropathy	15	15

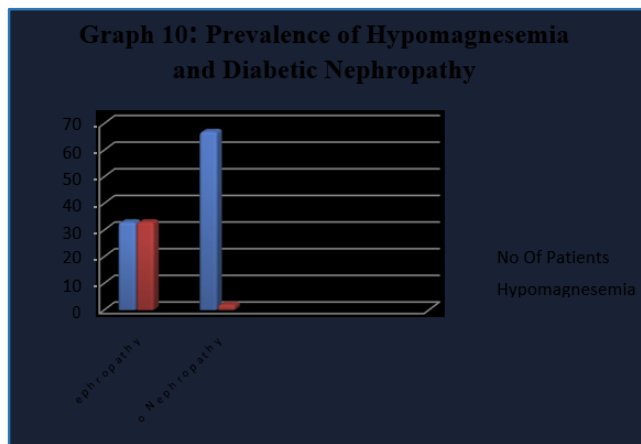| Sl. No. | Neuropathy | Magnesium | | Statistical Inference |
		Normomagne-saemia (n = 65)	Hypomagne-saemia (n = 35)	
1	Negative	60 (92.3%)	25 (71.4%)	X^2 = 7.778 Df = 1 0.005(<0.05) Significant
2	Positive	5 (7.7%)	10 (28.6%)	
Table 9. Prevalence of Hypomagnesaemia and Diabetic Neuropathy				

Observations revealed a definite correlation between hypomagnesaemia and diabetic neuropathy. The chi-square (χ2) value is 7.778, Df =1, p value is 0.005.

	No. of Persons	Percentage
Particulars	(n = 100)	(100%)
No albuminuria	67	67
Macroalbuminuria	4	4
Microalbuminuria	29	29
Table 10. Prevalence of Hypomagnesaemia and Diabetic Nephropathy		

Sl. No.	Nephropathy	Magnesium		Statistical Inference
		Normoma-gnesaemia (n = 65)	Hypomagne-saemia (n = 35)	
1	No albuminuria	65 (100%)	2 (5.7%)	X^2 = 91.471 Df = 2 0.000(<0.05)
2	Macroalbuminuria	0	4 (11.4%)	
3	Microalbuminuria	0	29 (82.9%)	Significant

Graph 10: Prevalence of Hypomagnesemia and Diabetic Nephropathy

Observations revealed a definite correlation between hypomagnesaemia and diabetic nephropathy. The chi-square (χ^2) value is 91.471. Df = 2.

Sl. No.	IHD	Magnesium		Statistical Inference
		Normomagne-saemia (n = 65)	Hypomagne-saemia (n = 35)	
1	Absent	54 (83.1%)	31 (88.6%)	X^2 = .539 Df = 1 0.463(>0.05) Not Significant
2	Present	11 (16.9%)	4 (11.4%)	

Table 11. Prevalence of Hypomagnesaemia and Ischaemic Heart Disease

Serum magnesium concentration showed no significant relation with ischaemic heart disease, X^2=.539, Df=1. P value is insignificant.

Graph 11: Prevalence of Hypomagnesemia and Ischemic Heart Disease
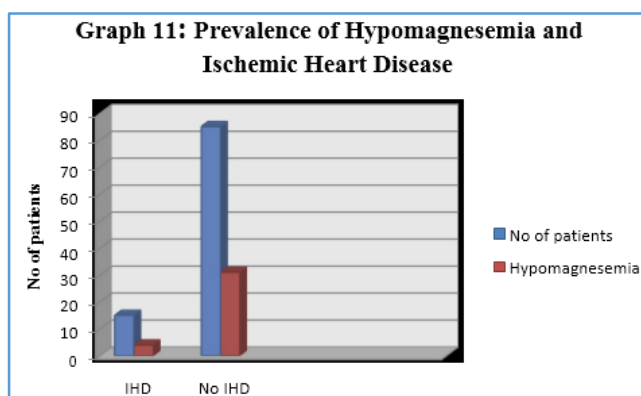
| Sl. No.	SHT	Magnesium		Statistical Inference
		Normo-magnesaemia (n = 65)	Hypomagne-saemia (n = 35)	
1	Absent	43 (66.2%)	27 (77.1%)	X^2 = 1.308 Df = 1 0.253(>0.05)
2	Present	22 (33.8%)	8 (22.9%)	Not Significant

Table 12. Prevalence of Hypomagnesaemia and Hypertension

Serum magnesium concentration showed no significance with systemic hypertension, X^2=1.308, Df=1. P value is insignificant.

Graph 12: Prevalence of Hypomagnesemia and Hypertension
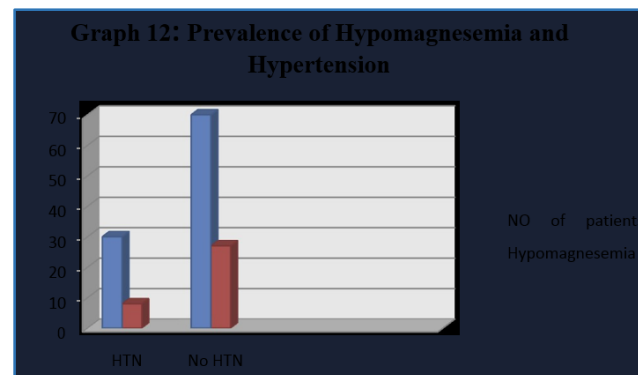
	Hypo-magnesaemia (No = 35)	Normo-magnesaemia (No = 65)
Complications		
IHD	4 (11.4%)	11 (16.9%)
HTN	8 (22.9%)	22 (33.8%)
Retinopathy	35 (48.6%)	18 (27.7%)
Neuropathy	10 (28.6%)	5 (7.7%)
Nephropathy	33 (94.3%)	0
FBS 131 & Above	21 (60%)	4 (6.2%)

Table 13. Distribution of Complications

Graph 13: Distribution of complications
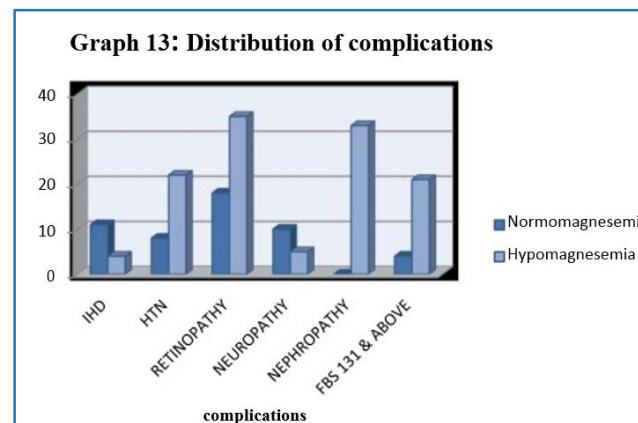
Among the persons with low serum magnesium level prevalence rates of IHD, HTN, Diabetic neuropathy, Diabetic nephropathy, Diabetic Retinopathy and Fasting glucose levels were compared to normomagnesaemic group. Study shows increased incidence of Diabetic neuropathy, Diabetic nephropathy, Diabetic Retinopathy in hypomagnesaemic patients compared to normomagnesaemics.

DISCUSSION

Reports of increased prevalence of low plasma magnesium concentrations among diabetic patients and possible association of hypomagnesaemia with diabetic complications prompted this study.

Marked magnesium deficiency has been reported in the previous case series of type-2 DM. However, some workers have also reported normal and even high levels. The present study shows serum magnesium level of 35 cases with type 2 DM, below the reference range. This confirms to the reported prevalence of low plasma magnesium status in T2DM in several studies, which ranged from 13.5% to 47.7%. Prevalence of hypomagnesaemia in T2 DM in our study was similar to that reported by Nadler et al.[10] Walti MK et al[11] also reported a 37.6% prevalence of hypomagnesaemia in T2DM versus 10.9% in nondiabetic controls in Zurich, Switzerland.

Serum magnesium concentration though less sensitive is a highly specific indicator of low magnesium status.

Low dietary intake is an unlikely cause of impaired magnesium status in diabetes. A dietary assessment conducted in Europe showed that only 5.4 percentage of the diabetics and 9.1 percentage of the control group had magnesium intake less than reference range. In addition, recently it has been shown that controlled diabetics have absorption of magnesium to a similar as healthy controls. Increased urinary magnesium excretion due to hyperglycaemia and osmotic diuresis may contribute to hypomagnesaemia in diabetes.

Serum levels of magnesium have been found by several investigators to correlate inversely with fasting blood glucose concentration and the percentage of HbA1C. Schlienger et al[12] hypothesised that patients with uncontrolled diabetics showed low serum magnesium concentration. The present study also revealed statistically significant correlation between serum magnesium levels and fasting blood sugar (Prevalence of hypomagnesaemia high in fasting blood sugar value above 131 mg% is 60%), but there is no significant correlation with HbA1C. However, a higher prevalence of hypomagnesaemia is observed in HbA1c >10 (28.6%) in the present study.

Hypomagnesaemia is reported to be both a cause and result of poor glycaemic control. Magnesium is necessary for glucose entry into the cell and act as a cofactor for enzymes of carbohydrate metabolism. In addition, magnesium deficiency has been shown to promote insulin resistance in multiple studies.

Nadler et al[10] have reported that insulin sensitivity decreases even in nondiabetic individuals after induction of magnesium deficiency. Likewise, elderly subjects were shown to have improved glucose tolerance when they received magnesium supplements. Thus, hypomagnesaemia by itself results in poor glycaemic control.

Conversely, hyperglycaemia and osmotic diuresis may lead to increased urinary magnesium excretion and hypomagnesaemia in diabetics. However, high prevalence of hypomagnesaemia is reported in type 2 diabetics with good glycaemic control. So although poor glycaemic control is associated with magnesium deficiency, it is not simply induced by hyperglycaemia and is not corrected by improvement in metabolic control alone.

Sex, age duration of diabetes were not the significant predictors of serum magnesium levels. Yajnick et al in 1984 reported that among patients with DM, serum magnesium levels depend on age and males had higher serum magnesium levels than females. The increasing magnesium levels with age were probably due to impaired renal function and the sample size (87 diabetics, 30 nondiabetics) was relatively small to confirm male preponderance. In our study, patients with impaired renal functions were excluded. Our results confirm to the recent reports that have not shown any significant associations between sex, age and duration of diabetes with serum magnesium levels.

Significant differences, in serum magnesium concentrations have been reported between the insulin treated and non-insulin treated diabetics. Our study revealed prevalence of hypomagnesaemia is low in insulin treated patients compared to non-insulin treated patients. ((37.1% v/s 62.9%). Yajnik et al reported that insulin treated diabetics have significantly lower serum magnesium levels compared to non-insulin treated ones. However, the difference was statistically not significant. Walti MK et al have reported that diabetes treatment (insulin or OHA) did not significantly predict hypomagnesaemia. Insulin mediates entry of magnesium from plasma to RBC. In a recent study, Alzaida et al[13] have found that cellular uptake of magnesium is normally stimulated by insulin. So, insulin treatment may enhance cellular magnesium uptake and result in increased prevalence of hypomagnesaemia.

In our study, no association was found between incidence of ischaemic heart disease and hypomagnesaemia. However, several observational studies have reported hypomagnesaemia is associated with higher risk of ischaemic heart disease. As part of atherosclerosis risk in communities study, a cohort of 15,792 subjects were studied over 7 years and an increasing relative risk of coronary artery disease with decreasing serum magnesium was reported.[14] How a low serum magnesium predisposes to coronary artery disease is not identified yet. However, in our study, no difference in prevalence of hypomagnesaemia was found between those with ischaemic heart disease and others. Similarly, no difference in prevalence of hypomagnesaemia was found between the hypertensive and non-hypertensive subjects.

Trials have shown that magnesium deficiency has been associated with diabetic microvascular disease. In our study, increased prevalence of hypomagnesaemia was observed in diabetics with microvascular complications and mean serum concentration of magnesium in diabetics with microvascular complications was comparatively lower than in diabetics with no microvascular complications.

Hypomagnesaemia has been reported in patients with diabetic retinopathy, with lower magnesium levels predicting a greater risk of severe diabetic retinopathy. Our observations revealed a definite association between diabetic retinopathy and lower serum magnesium levels. There was a significant difference in prevalence of hypomagnesaemia in diabetics with retinopathy and without retinopathy (48.6% Vs. 27.7%; P< 0.005). These observations are similar to other reports. Grafton et al[15] have proposed the inositol transport theory to explain this association. But the exact reason remains obscure.

Hypomagnesaemia is seen in cases with diabetic neuropathy, with lower magnesium levels predicting a greater risk of severe diabetic neuropathy. The present study

revealed patients with diabetic neuropathy had a slightly higher prevalence of hypomagnesaemia compared to those without neuropathy (28.6% v/s 7.7%). Rodriguez- Moran and Guerrero-Romero hypothesised that low serum magnesium level causes high incidence of diabetic foot ulcers.

Hypomagnesaemia is seen in cases with diabetic nephropathy. Decreased magnesium levels predict an increased risk of severe diabetic nephropathy. The present study shows patients with diabetic nephropathy had a slightly increased incidence of hypomagnesaemia than in those without nephropathy (94.3% v/s 0%). Corsonello et al demonstrated decreased serum magnesium in type 2 DM with nephropathy compared to normal patients. Recent study shows that decreased magnesium concentration is associated with rapid loss of renal function in patients with type 2 DM.

In summary, the present study has demonstrated that hypomagnesaemia is common in type 2 diabetics and magnesium deficiency is conclusively associated with diabetic retinopathy, neuropathy, nephropathy.

Diabetic patients need regular monitoring of serum magnesium concentration. If low level is reported, magnesium supplementation is necessary.

CONCLUSIONS
1. Prevalence of hypomagnesaemia in type 2 Diabetics is 35%.
2. Prevalence of hypomagnesaemia is higher in patients with microvascular diabetic complications than in those without microvascular complications.
3. Hypomagnesaemia is significantly associated with retinopathy, neuropathy and nephropathy.
4. No significant association was seen with ischaemic heart disease and hypomagnesaemia.
5. No significant association was seen with hypertension and hypomagnesaemia.
6. Prevalence of hypomagnesaemia was high in patients with fasting blood sugar >131 mg/dL.
7. No significant differences were seen among both sexes.
8. No significant association of hypomagnesaemia was seen with duration of diabetes and HbA1c levels.

Summary
Present study was conducted to estimate the prevalence of hypomagnesaemia in type 2 diabetics and to study the possible association of hypomagnesaemia with diabetic complications and comorbidities.

The study included 100 type 2 diabetic cases, with no factors significantly altering the serum magnesium levels. Fasting serum magnesium levels were estimated and correlation was done with variable parameters.

The results found that prevalence of low magnesium levels (<1.7 mg/dL) in type 2 diabetic cases were 35% and magnesium deficiency was significantly associated with diabetic retinopathy, neuropathy and nephropathy. No significant correlation was identified with other parameters - IHD, hypertension, duration of diabetes and treatment modality. Causes of hypomagnesaemia are multifactorial. Available data suggests that low magnesium levels are associated with adverse clinical outcomes. So it is prudent in

clinical practice to periodically monitor plasma magnesium concentrations in diabetic patients and have the condition corrected whenever possible.

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