Section: Biochemistry

Original Research Article

EVALUATION OF SERUM MAGNESIUM LEVELS IN RELATION TO INSULIN RESISTANCE USING THE TRIGLYCERIDE-GLUCOSE INDEX IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: A HOSPITAL-BASED CROSS-SECTIONAL ANALYTICAL STUDY

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ABSTRACT

Background: Type 2 diabetes mellitus is a complex metabolic disorder characterized by chronic hyperglycaemia due to impaired insulin secretion and insulin resistance. Insulin resistance is a central abnormality in type 2 diabetes mellitus and is associated with dyslipidaemia, cardiovascular risk and metabolic complications. The triglyceride-glucose index is a simple and cost-effective surrogate marker of insulin resistance. Magnesium plays an important role in insulin secretion, insulin receptor activity and glucose metabolism. Low serum magnesium levels may contribute to poor glycaemic control and increased insulin resistance in patients with type 2 diabetes mellitus. The aim is to evaluate serum magnesium levels in relation to insulin resistance using the triglyceride-glucose index in patients with type 2 diabetes mellitus.

Materials and Methods: This hospital-based cross-sectional analytical study was conducted in the Department of Biochemistry, Guntur Medical College, in collaboration with Government General Hospital, Guntur. The study included 170 patients with type 2 diabetes mellitus aged 30–65 years. Patients attending the outpatient department who fulfilled the inclusion and exclusion criteria and gave informed consent were enrolled. Blood samples were analysed for HbA1c, fasting blood sugar, post-prandial blood sugar, fasting insulin, fasting triglycerides, serum magnesium, total cholesterol, HDL and LDL. The triglyceride-glucose index was calculated. Data were entered in Microsoft Excel and analysed using SPSS version 25.0. Since the data were not normally distributed, median and interquartile range were used. Mann–Whitney U test, Kruskal–Wallis test and Spearman correlation were applied. A p-value less than 0.05 was considered statistically significant.

Results: The study included 170 patients with type 2 diabetes mellitus, comprising 88 males and 82 females. The median age was 48 years, median HbA1c was 7.70, median fasting blood sugar was 159.50, median post-prandial blood sugar was 303.00, median insulin was 17.90, median TyG index was 9.71 and median serum magnesium was 1.62. Serum magnesium showed statistically significant negative correlations with HbA1c (rho = -0.895, p <0.0001), TyG index (rho = -0.736, p <0.0001), post-prandial blood sugar (rho = -0.896, p <0.0001) and fasting blood sugar (rho = -0.873, p <0.0001). TyG index showed statistically significant positive correlations with fasting blood sugar (rho = 0.841, p <0.0001), post-prandial blood sugar (rho = 0.853, p <0.0001) and triglycerides (rho = 0.785, p <0.0001).

Conclusion: Serum magnesium showed a strong negative correlation with glycaemic parameters and TyG index in patients with type 2 diabetes mellitus. Lower magnesium levels were associated with poorer glycaemic control and increased insulin resistance. The TyG index showed strong positive correlations with fasting blood sugar, post-prandial blood sugar and triglycerides, supporting its role as a simple surrogate marker of insulin resistance. Routine assessment of serum magnesium and TyG index may be useful in the metabolic evaluation of patients with type 2 diabetes mellitus.
Keywords: Type 2 diabetes mellitus, serum magnesium, triglyceride-glucose index, TyG index, insulin resistance, HbA1c, fasting blood sugar, post-prandial blood sugar.

INTRODUCTION

Type 2 diabetes mellitus is a complex metabolic disease characterized by chronic hyperglycaemia resulting from impaired insulin secretion, insulin action or both. It is one of the most common non-communicable diseases and is associated with significant morbidity and mortality due to microvascular and macrovascular complications. The burden of type 2 diabetes mellitus is increasing globally as well as in India, making early identification of metabolic abnormalities clinically important.[1,2]

Insulin resistance is a major metabolic abnormality in type 2 diabetes mellitus. It refers to reduced biological response of target tissues to normal circulating insulin concentrations. Insulin resistance is commonly associated with dyslipidaemia, hypertension, central obesity and cardiovascular disease. It often precedes overt hyperglycaemia by several years and plays a central role in the development and progression of type 2 diabetes mellitus.[3,4]

The hyperinsulinemic-euglycemic clamp is considered the gold standard method for assessment of insulin resistance. However, it is expensive, technically demanding and not feasible for routine clinical practice or large-scale studies. Therefore, simple and cost-effective surrogate markers are needed for assessing insulin resistance. The triglyceride-glucose index, calculated from fasting triglycerides and fasting plasma glucose, has emerged as a practical surrogate marker of insulin resistance. Higher TyG index values have been associated with increased risk of type 2 diabetes mellitus, cardiovascular disease and metabolic complications.[5-8]

Magnesium is the fourth most abundant mineral in the human body and an important intracellular cation. It acts as a cofactor in several enzymatic reactions and plays a significant role in insulin secretion, insulin binding, glucose transport, insulin receptor phosphorylation and downstream intracellular signalling. Magnesium deficiency can impair insulin action and contribute to insulin resistance.[9,10]

In patients with type 2 diabetes mellitus, low serum magnesium levels may occur due to increased urinary loss from glycosuria, poor dietary intake, impaired absorption, insulin resistance and drug-related factors. Hypomagnesemia has been associated with poor glycaemic control, increased HbA1c levels, dyslipidaemia and diabetic complications.[10,11] Despite its clinical importance, serum magnesium is not routinely assessed in many patients with diabetes.

There is limited regional data regarding the relationship between serum magnesium levels and insulin resistance assessed by TyG index among patients with type 2 diabetes mellitus. Evaluation of this association is clinically relevant because both serum magnesium and TyG index are simple, inexpensive and easily available biochemical measures. The present study was therefore undertaken to evaluate serum magnesium levels in relation to insulin resistance using the TyG index in patients with type 2 diabetes mellitus attending Government General Hospital and Guntur Medical College, Guntur.

Aim: To evaluate serum magnesium levels in relation to insulin resistance using the triglyceride-glucose index in patients with type 2 diabetes mellitus.

Objectives

1. To assess insulin resistance in type 2 diabetes mellitus using the triglyceride-glucose index.
2. To estimate and assess the correlation between serum magnesium levels and glycaemic parameters in type 2 diabetes mellitus patients.
3. To explore the relationship between serum magnesium levels and insulin resistance in patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

Study design: The present study was conducted as a hospital-based cross-sectional analytical study to evaluate serum magnesium and insulin resistance assessed using the triglyceride-glucose index in patients with type 2 diabetes mellitus.

Study duration: The study was carried out over a period of 18 months.

Study setting: The study was conducted in the Department of Biochemistry, Guntur Medical College, in collaboration with Government General Hospital, Guntur, where patients attending the outpatient department were recruited.

Study population: The study population comprised patients with type 2 diabetes mellitus attending the outpatient department of Government General Hospital, Guntur.

Sample size: A total of 170 patients with type 2 diabetes mellitus were included in the study.

Source of data: Outpatients attending the Department of General Medicine, Government General Hospital, Guntur, who fulfilled all inclusion and exclusion criteria and provided informed consent, were included as research participants.

Inclusion Criteria

Patients fulfilling the following criteria were included:

1. Patients with type 2 diabetes mellitus.
2. Age range 30–65 years.
3. Patients who gave consent to participate in the study.

Exclusion Criteria

Patients with the following conditions were excluded:

1. Patients on magnesium supplementation.
2. Patients with chronic kidney disease or renal failure.
3. Patients on diuretic therapy.

Biochemical investigations:

The following biochemical parameters were assessed using Beckman automated systems:

1. HbA1c.
2. Fasting blood sugar.
3. Two-hour post-prandial blood glucose.
4. Fasting insulin.
5. Complete lipid profile.
6. Serum magnesium.
7. TyG index.

Estimation of HbA1c: HbA1c was estimated by immunoturbidimetric method using AU 480 automated clinical chemistry analyser. HbA1c in the sample forms soluble antigen-antibody complexes with specific anti-HbA1c antibodies. Excess antibodies react with polyhapten reagents, producing agglutinated complexes that are measured turbidimetrically. The absorbance change provides an estimation of HbA1c concentration, expressed as percentage of total haemoglobin.

Estimation of plasma glucose: Fasting plasma glucose and two-hour post-prandial glucose were measured using the hexokinase enzymatic method in fully automated AU 480 analyser. Hexokinase phosphorylates glucose with ATP to form glucose-6-phosphate, which is oxidized by glucose-6-phosphate dehydrogenase with formation of NADPH. The absorbance of NADPH is measured at 340 nm and is proportional to glucose concentration.

Estimation of lipid profile: Total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides were estimated using Beckman Coulter reagent kits and enzymatic colorimetric methods in AU 480 fully automated analyser.

Total cholesterol was measured by enzymatic cholesterol esterase oxidase method. HDL cholesterol was measured using homogeneous enzymatic method. LDL cholesterol was measured using direct homogeneous two-step enzymatic assay. Triglycerides were estimated by enzymatic colorimetric method.

Estimation of serum magnesium: Serum magnesium was estimated colorimetrically using xylidyl blue reagent on Beckman Coulter AU 480 clinical chemistry analyser. Magnesium in the sample reacts with xylidyl blue in alkaline medium to form a coloured complex. The analyser photometrically measures the colour intensity, which is proportional to magnesium concentration.

Estimation of insulin: Fasting insulin was estimated by immunoassay method using ACCESS 2 analyser. The method is a sandwich-type assay using monoclonal antibodies directed against insulin epitopes. The measured signal is proportional to the insulin concentration in the sample.

Calculation of TyG index: Fasting plasma glucose and triglycerides were used to calculate the TyG index, which serves as a surrogate marker of insulin resistance. Higher TyG index values indicate reduced insulin sensitivity and altered glucose-lipid metabolism.

TyG index = ln [fasting triglycerides × fasting glucose]:

Statistical analysis: The collected data were entered into Microsoft Excel and analysed using Statistical Package for Social Sciences software, version 25.0. Shapiro–Wilk test was performed for normality assessment. Since the data were not normally distributed, continuous variables were expressed as median and interquartile range. Categorical variables were expressed as frequencies and percentages.

Mann–Whitney U test was used for comparison between males and females. Kruskal–Wallis test was used for comparison of study variables across HbA1c categories. Spearman correlation was used to assess associations among serum magnesium, glycaemic parameters, TyG index and other biochemical variables. A p-value less than 0.05 was considered statistically significant.

RESULTS

This study included 170 patients with type 2 diabetes mellitus, comprising 88 males and 82 females. Blood samples were collected from all study participants and analysed for HbA1c, fasting blood sugar, post-prandial blood sugar, fasting insulin, fasting triglycerides, serum magnesium, total cholesterol, HDL and LDL. Age was documented for each study participant. The Triglyceride-Glucose index was calculated and documented. All values were presented in the master chart.

The whole study data was documented in Microsoft Excel sheet and statistical analysis was done using SPSS IBM software, version 25.0. Males and females were labelled as 1 and 2, respectively, in the

Excel sheet and SPSS. Shapiro–Wilk test was performed for normality assessment. The study data were not normally distributed; therefore, median and interquartile range were calculated. Non-parametric tests were performed for statistical interpretation. Mann–Whitney U test was used for comparison between males and females. Kruskal–Wallis test was used for comparison of study variables across different HbA1c categories. Spearman correlation was used to assess correlation among study variables. A p-value less than 0.05 was considered statistically significant.

Table 1: Descriptive statistics of the study participants, n=170

Parameter	Minimum	Maximum	Median	IQR (Q1–Q3)
Age (years)	30.00	65.00	48.00	37.25–56.00
HbA1c (%)	5.72	10.00	7.70	6.96–8.60
FBS (mg/dl)	110.00	215.00	159.50	138.25–187.00
PPBS (mg/dl)	230.00	379.00	303.00	271.25–338.00
Insulin (µIU/ml)	5.20	29.80	17.90	12.58–21.70
Triglycerides (mg/dl)	73.3	540.6	220.45	158.05–279.73
TyG index	8.80	10.40	9.71	9.51–10.00
Magnesium (mg/dl)	1.36	1.89	1.62	1.54–1.71

The median age of the study participants was 48 years. The median HbA1c was 7.70, median fasting blood sugar was 159.50, median post-prandial blood sugar was 303.00, median insulin was 17.90, median triglycerides was 9.30, median TyG index was 9.71 and median serum magnesium was 1.62.

Table 2: Comparison of study variables among different HbA1c categories, n=170

Parameter	HbA1c <7	HbA1c 7–8	HbA1c >8	p-value
Magnesium	1.75 (1.71–1.78)	1.65 (1.60–1.68)	1.53 (1.46–1.56)	<0.0001
FBS	122.50 (114.25–130.75)	162.00 (137.00–170.00)	192.00 (181.00–204.50)	<0.0001
PPBS	252.50 (243.25–263.00)	295.00 (286.00–304.00)	341.00 (332.50–361.50)	<0.0001
Insulin	18.40 (14.08–21.30)	19.80 (16.00–22.00)	18.30 (10.90–21.70)	0.670
HbA1c	6.44 (6.11–6.64)	7.53 (7.34–7.74)	8.76 (8.46–9.22)	<0.0001

Serum magnesium showed a statistically significant decreasing trend across increasing HbA1c categories. Fasting blood sugar and post-prandial blood sugar showed statistically significant progressive increases across HbA1c categories. Insulin did not show a statistically significant difference across HbA1c categories.

Table 3: Correlation of serum magnesium with HbA1c, TyG index, PPBS and FBS

Correlation	Variable 1, Median (IQR)	Variable 2, Median (IQR)	Spearman rho	p-value
Magnesium with HbA1c	Magnesium: 1.63 (1.54–1.71)	HbA1c: 7.70 (6.96–8.60)	-0.895	<0.0001
Magnesium with TyG index	Magnesium: 1.63 (1.54–1.71)	TyG index: 9.30 (9.19–9.43)	-0.736	<0.0001
Magnesium with PPBS	Magnesium: 1.63 (1.54–1.71)	PPBS: 303.00 (271.25–338.00)	-0.896	<0.0001
Magnesium with FBS	Magnesium: 1.63 (1.54–1.71)	FBS: 159.50 (138.25–187.00)	-0.873	<0.0001

Serum magnesium showed statistically significant negative correlations with HbA1c, TyG index, post-prandial blood sugar and fasting blood sugar. The strongest negative correlation was observed between magnesium and post-prandial blood sugar, followed by magnesium and HbA1c, magnesium and fasting blood sugar, and magnesium and TyG index. These findings indicate that lower serum magnesium levels were associated with poorer glycaemic control and increased insulin resistance.

Table 4: Correlation of TyG index with FBS, PPBS and triglycerides

Correlation	Variable 1, Median (IQR)	Variable 2, Median (IQR)	Spearman rho	p-value
TyG index with FBS	TyG index: 9.30 (9.19–9.43)	FBS: 159.50 (138.25–187.00)	0.841	<0.0001
TyG index with PPBS	TyG index: 9.30 (9.19–9.43)	PPBS: 303.00 (271.25–338.00)	0.853	<0.0001
TyG index with triglycerides	TyG index: 9.30 (9.19–9.43)	Triglycerides: 215 (165–257)	0.785	<0.0001

TyG index showed statistically significant positive correlations with fasting blood sugar, post-prandial blood sugar and triglycerides. The strongest positive correlation was observed between TyG index and post-prandial blood sugar, followed by TyG index and fasting blood sugar, and TyG index and triglycerides. These findings indicate that higher TyG index values were associated with higher glucose and triglyceride levels, suggesting increased insulin resistance.

Table 5: Correlation between HbA1c categories with magnesium and post-prandial blood sugar

Parameter	HbA1c <7	HbA1c 7–8	HbA1c >8	Spearman rho	p-value
HbA1c categories	6.44 (6.11–6.64)	7.53 (7.34–7.74)	8.76 (8.46–9.22)	-0.853	<0.0001
Magnesium	1.75 (1.71–1.78)	1.65 (1.60–1.68)	1.53 (1.46–1.56)		
HbA1c categories	6.44 (6.11–6.64)	7.53 (7.34–7.74)	8.76 (8.46–9.22)	0.930	<0.0001
Post-prandial blood sugar	252.50(243.25263.00)	295.00 (286.00–304.00)	341.00 (332.50–361.50)		

Serum magnesium levels showed a decreasing trend across increasing HbA1c categories. A statistically significant strong negative correlation was observed between HbA1c categories and magnesium levels. Post-prandial blood sugar levels increased progressively across HbA1c categories, with a statistically significant strong positive correlation. This indicates that worsening glycaemic control was associated with lower serum magnesium levels and higher post-prandial blood sugar levels.

Table 6: Comparison of all parameters among males and females, n=170

Parameter	Male, Median (IQR)	Female, Median (IQR)	p-value
TyG index	9.69 (9.51–9.93)	9.73 (9.52–10.00)	0.508
HbA1c	7.56 (6.90–8.52)	7.79 (7.10–8.71)	0.356
FBS	152.00 (136.50–179.75)	164.50 (146.50–189.75)	0.123
PPBS	293.00 (269.75–330.25)	308.50 (284.25–341.00)	0.105
Total cholesterol	216.00 (182.75–237.00)	210.50 (183.25–245.75)	0.966
Triglycerides	220.45 (160.68-273.80)	216.75 (154.23–286.95)	0.863
HDL	44.00 (38.00–53.25)	44.00 (35.00–50.00)	0.230
LDL	140.00 (103.75–167.00)	132.00 (105.00–159.75)	0.187
VLDL	29.50 (20.00–43.00)	30.50 (17.00–39.75)	0.325
Magnesium	1.64 (1.55–1.71)	1.60 (1.52–1.70)	0.120
Insulin	19.65 (12.73–24.42)	17.00 (12.32–20.68)	0.098
Age	51.00 (37.00–58.25)	44.50 (38.00–53.00)	0.055

No statistically significant difference was observed between males and females for TyG index, HbA1c, fasting blood sugar, post-prandial blood sugar, total cholesterol, triglycerides, HDL, LDL, VLDL, magnesium, insulin and age, as all p-values were greater than 0.05.

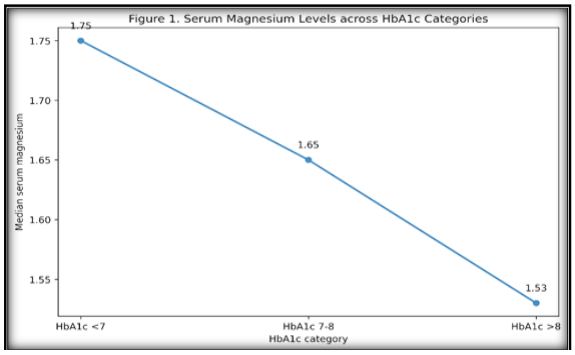

Figure 1: Serum Magnesium Levels across HbA1c Categories

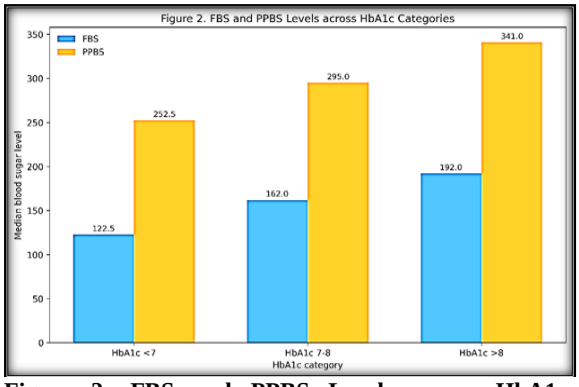

Figure 2: FBS and PPBS Levels across HbA1c Categories

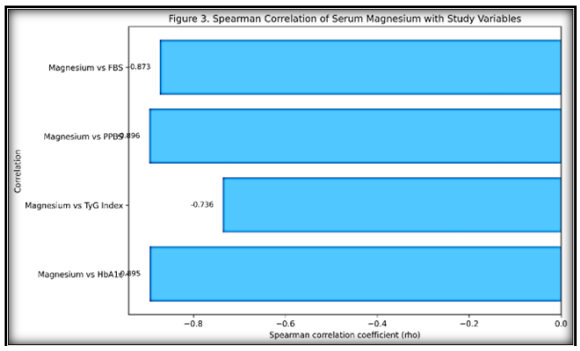

Figure 3: Spearman Correlation of Serum Magnesium with Study Variables

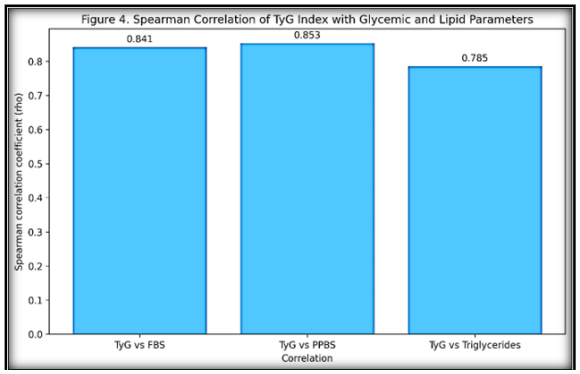

Figure 4: Spearman Correlation of TyG Index with Glycemic and Lipid Parameters

Summary of Results: In the present study of 170 patients with type 2 diabetes mellitus, 88 were males and 82 were females. The median age was 48 years, median HbA1c was 7.70, median fasting blood sugar was 159.50, median post-prandial blood sugar was 303.00, median insulin was 17.90, median TyG index was 9.71 and median serum magnesium was 1.62.

Serum magnesium showed a statistically significant decreasing trend across increasing HbA1c categories. Fasting blood sugar and post-prandial blood sugar increased significantly across HbA1c

categories. Serum magnesium showed strong negative correlations with HbA1c, fasting blood sugar, post-prandial blood sugar and TyG index. TyG index showed strong positive correlations with fasting blood sugar, post-prandial blood sugar and triglycerides. HbA1c categories showed a strong negative correlation with serum magnesium and a strong positive correlation with post-prandial blood sugar. No significant gender-wise differences were observed in the study variables.

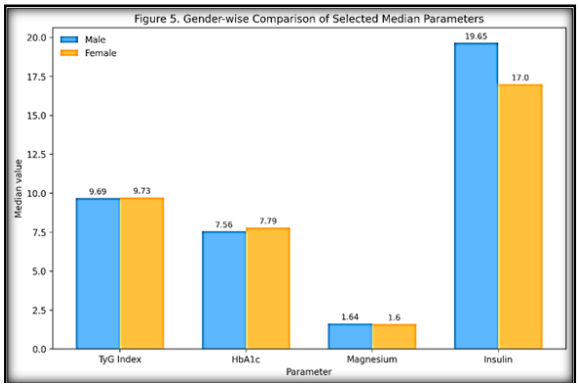

Figure 5: Gender-wise Comparison of Selected Median Parameters

DISCUSSION

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by insulin resistance, impaired insulin secretion and persistent hyperglycaemia. Insulin resistance plays a central role in the development and progression of type 2 diabetes mellitus and is commonly associated with dyslipidaemia, central obesity, hypertension and increased cardiovascular risk. In recent years, serum magnesium and the triglyceride-glucose (TyG) index have received increasing attention as simple biochemical indicators related to glycaemic control and insulin resistance in patients with type 2 diabetes mellitus.

Magnesium is an essential intracellular cation and acts as a cofactor in several enzymatic reactions involved in glucose metabolism, insulin secretion, insulin receptor activity and intracellular signalling. Low serum magnesium levels have been associated with impaired insulin action, worsening glycaemic control and metabolic syndrome.[12,13] Magnesium deficiency may contribute to insulin resistance by reducing insulin receptor tyrosine kinase activity, impairing post-receptor signalling, altering glucose transport and increasing oxidative stress and inflammatory activity.[14,15] Therefore, assessment of serum magnesium in patients with type 2 diabetes mellitus may provide clinically useful information regarding metabolic control and insulin resistance.

In the present study, 170 patients with type 2 diabetes mellitus were included, comprising 88 males and 82 females. The median age of the study participants was 48 years. The median HbA1c was 7.70, median fasting blood sugar was 159.50, median post-prandial blood sugar was 303.00, median insulin was 17.90, median TyG index was 9.71 and median serum magnesium was 1.62. These findings indicate that the study population consisted of patients with variable glycaemic control and measurable insulin resistance using the TyG index.

Serum magnesium and glycaemic control: A major finding of the present study was the strong inverse association between serum magnesium and glycaemic parameters. Serum magnesium showed a statistically significant negative correlation with HbA1c (rho = -0.895, p <0.0001), indicating that lower magnesium levels were associated with poorer long-term glycaemic control. Serum magnesium also showed strong negative correlations with fasting blood sugar (rho = -0.873, p <0.0001) and post-prandial blood sugar (rho = -0.896, p <0.0001). Among these, the strongest negative correlation was observed between magnesium and post-prandial blood sugar.

These findings are consistent with previous studies that reported an inverse relationship between serum magnesium levels and glycaemic indices in patients with type 2 diabetes mellitus. Dasgupta et al. observed that hypomagnesemia was common among patients with type 2 diabetes mellitus and was associated with poor glycaemic control.[16] Saha et al. reported that lower serum magnesium levels were associated with higher fasting blood sugar, post-prandial blood sugar and HbA1c.[17] Mishra et al. also found an inverse relationship between serum magnesium and HbA1c, suggesting that declining magnesium levels may worsen glycaemic control.[18] Palathingal and Thomas similarly reported a significant association between serum magnesium and glycaemic control among diabetic patients.[19]

The biological explanation for this relationship is supported by the role of magnesium in insulin secretion and action. Magnesium is required for ATP-dependent enzymatic reactions, insulin receptor phosphorylation and cellular glucose uptake. Magnesium deficiency reduces the activity of insulin receptor tyrosine kinase and impairs intracellular signalling pathways, thereby reducing insulin sensitivity.[20,21] In addition, chronic hyperglycaemia may increase urinary magnesium loss through osmotic diuresis, further reducing serum magnesium levels. Thus, poor glycaemic control and hypomagnesemia may reinforce each other in patients with type 2 diabetes mellitus.

Serum magnesium and insulin resistance assessed by TyG index: The present study showed a statistically significant negative correlation between serum magnesium and TyG index (rho = -0.736, p <0.0001). This indicates that lower serum magnesium levels were associated with higher TyG index values, suggesting increased insulin resistance. Although the correlation between magnesium and TyG index was slightly weaker than its correlation with HbA1c, fasting blood sugar and post-prandial blood sugar, it remained statistically significant and clinically relevant.

This finding supports earlier evidence that magnesium deficiency is linked to insulin resistance and metabolic dysfunction. Guerrero-Romero and Rodríguez-Morán reported that low serum magnesium levels were associated with insulin resistance and metabolic syndrome.[12] Barbagallo and Dominguez described the role of magnesium in insulin action and highlighted that magnesium deficiency may impair insulin-mediated glucose uptake.[20] Kostov also emphasized that magnesium deficiency interferes with insulin signalling and contributes to insulin resistance.[15] Gommers et al. further described the relationship between magnesium homeostasis and insulin resistance, supporting the metabolic significance of magnesium status in diabetes.[22]

The TyG index is calculated using fasting triglycerides and fasting glucose and serves as a practical surrogate marker of insulin resistance. It is particularly useful in routine clinical and resource-limited settings because it does not require insulin assays or complex clamp-based methods. In the present study, the inverse relationship between serum magnesium and TyG index suggests that magnesium deficiency may be associated not only with poor glycaemic control but also with insulin resistance.

TyG index and glycaemic parameters: The TyG index showed statistically significant positive correlations with fasting blood sugar, post-prandial blood sugar and triglycerides. In the present study, TyG index was positively correlated with fasting blood sugar (rho = 0.841, p <0.0001), post-prandial blood sugar (rho = 0.853, p <0.0001) and triglycerides (rho = 0.785, p <0.0001). These findings indicate that higher TyG index values were associated with higher glycaemic and triglyceride levels, reflecting increased insulin resistance.

The strongest positive correlation was observed between TyG index and post-prandial blood sugar, followed by fasting blood sugar and triglycerides. This suggests that the TyG index captures the combined metabolic disturbance of glucose and lipid metabolism in patients with type 2 diabetes mellitus. As insulin resistance increases, glucose uptake by peripheral tissues decreases, hepatic glucose production increases and lipolysis is enhanced, contributing to hyperglycaemia and hypertriglyceridaemia.

Simental-Mendía et al. described the TyG index as a reliable marker of insulin resistance.[23] Guerrero-Romero et al. validated the TyG index against the hyperinsulinemic-euglycemic clamp and supported its utility as a simple insulin resistance marker.[24] Vasques et al. compared the TyG index with HOMA-IR and demonstrated its usefulness in identifying insulin resistance.[25] Lee et al. reported that higher TyG index values were associated with increased risk of diabetes.[26] Zhang et al. also reported that TyG index was associated with metabolic syndrome and adverse metabolic risk.[27] These studies support the present finding that TyG index is strongly associated with glycaemic and triglyceride-related abnormalities in type 2 diabetes mellitus.

HbA1c categories, magnesium and post-prandial blood sugar: The present study also evaluated magnesium and post-prandial blood sugar across different HbA1c categories. Serum magnesium showed a progressive decline with increasing HbA1c categories. In patients with HbA1c <7, the median magnesium was 1.75; in HbA1c 7–8, it was 1.65; and in HbA1c >8, it decreased to 1.53. The correlation between HbA1c categories and magnesium was strongly negative and statistically significant (rho = -0.853, p <0.0001). This finding indicates that worsening glycaemic control was associated with lower serum magnesium levels.

Post-prandial blood sugar increased progressively across HbA1c categories. In patients with HbA1c <7, median PPBS was 252.50; in HbA1c 7–8, it was 295.00; and in HbA1c >8, it increased to 341.00. The correlation between HbA1c categories and PPBS was strongly positive and statistically significant (rho = 0.930, p <0.0001). This result is expected because HbA1c reflects long-term glycaemic exposure and is influenced by both fasting and post-prandial glucose levels.

The progressive fall in magnesium levels across increasing HbA1c categories is clinically important. It suggests that patients with poorer glycaemic control are more likely to have lower magnesium levels. This may be due to increased renal magnesium loss, insulin resistance, altered intracellular magnesium transport and dietary insufficiency. Previous evidence also supports the link between magnesium deficiency, poor glycaemic control and diabetic complications.[28,29]

Gender-wise comparison: In the present study, no statistically significant difference was observed between males and females for TyG index, HbA1c, fasting blood sugar, post-prandial blood sugar, total cholesterol, triglycerides, HDL, LDL, VLDL, magnesium, insulin and age. All gender-wise p-values were greater than 0.05. This indicates that the observed relationships between magnesium, glycaemic parameters and TyG index were not primarily driven by gender differences in the study group.

The absence of significant gender-wise differences supports the interpretation that serum magnesium and TyG index may be useful metabolic indicators across both male and female patients with type 2 diabetes mellitus. However, the p-value for age was 0.055, which was close to statistical significance, suggesting that future studies with larger sample sizes may further evaluate age and gender-based metabolic differences.

Pathophysiological interpretation: The findings of the present study can be explained by the close biological relationship between magnesium metabolism, insulin action and glucose homeostasis. Magnesium is essential for ATP stabilization and phosphorylation reactions. Insulin receptor

activation requires magnesium-dependent tyrosine kinase activity, and downstream insulin signalling depends on phosphorylation of insulin receptor substrate proteins and activation of the PI3K-Akt pathway.[30,31] Magnesium deficiency may impair these processes, reducing GLUT4 translocation and cellular glucose uptake.

Low magnesium levels may also increase intracellular calcium levels, which can interfere with insulin-mediated glucose transport and worsen insulin resistance.[32] Magnesium deficiency has also been linked to mitochondrial dysfunction, oxidative stress and chronic low-grade inflammation, all of which contribute to impaired insulin signalling and metabolic dysregulation.[33,34] Therefore, the strong negative correlations observed between magnesium and glycaemic parameters in the present study are biologically plausible.

In type 2 diabetes mellitus, chronic hyperglycaemia may further reduce magnesium levels through osmotic diuresis and urinary magnesium wasting. This creates a vicious cycle in which hyperglycaemia promotes magnesium loss, and magnesium deficiency further worsens insulin resistance and glycaemic control. The present findings support this bidirectional relationship.

Clinical implications: The findings of this study have important clinical implications. Serum magnesium estimation is simple, inexpensive and widely available. The strong inverse relationship between serum magnesium and HbA1c, fasting blood sugar and post-prandial blood sugar suggests that magnesium status may be considered as part of routine metabolic assessment in patients with type 2 diabetes mellitus. Patients with poor glycaemic control may benefit from evaluation of serum magnesium, especially when insulin resistance or metabolic complications are suspected.

The TyG index also has practical clinical value because it can be calculated using fasting glucose and triglyceride levels, which are routinely measured in diabetic patients. Its strong positive correlation with fasting blood sugar, post-prandial blood sugar and triglycerides supports its use as a cost-effective marker of insulin resistance. In resource-limited settings, TyG index may serve as a useful alternative to complex insulin resistance assessment methods.

The combination of serum magnesium and TyG index may help identify diabetic patients at higher metabolic risk. Low magnesium levels together with high TyG index may indicate poor glycaemic control and increased insulin resistance. Such patients may require closer monitoring, dietary counselling, improved glycaemic control and evaluation of micronutrient status.

CONCLUSION

The present study demonstrated that serum magnesium levels were strongly and negatively correlated with glycaemic parameters and TyG index in patients with type 2 diabetes mellitus. Lower serum magnesium levels were significantly associated with higher HbA1c, fasting blood sugar, post-prandial blood sugar and TyG index, indicating poorer glycaemic control and increased insulin resistance.

TyG index showed strong positive correlations with fasting blood sugar, post-prandial blood sugar and triglycerides, supporting its role as a simple, cost-effective and practical surrogate marker of insulin resistance in type 2 diabetes mellitus. Serum magnesium levels also decreased progressively across increasing HbA1c categories, while post-prandial blood sugar increased significantly with worsening glycaemic control.

Overall, the study supports the clinical utility of routine serum magnesium estimation and TyG index calculation in the metabolic evaluation of patients with type 2 diabetes mellitus. Monitoring serum magnesium along with glycaemic indices may help identify patients at higher risk of insulin resistance and poor metabolic control.

Strengths of the study:

1. The study included 170 patients with type 2 diabetes mellitus, providing a reasonable sample size for assessing biochemical correlations.
2. The study evaluated both glycaemic parameters and insulin resistance using the TyG index.
3. Serum magnesium was assessed along with HbA1c, fasting blood sugar, post-prandial blood sugar, fasting insulin and lipid profile, allowing comprehensive metabolic evaluation.
4. Non-parametric statistical tests were used appropriately because the data were not normally distributed.
5. The study provides region-specific data from patients attending Government General Hospital and Guntur Medical College, Guntur.

Limitations:

1. The cross-sectional study design prevents establishment of causal relationship between serum magnesium deficiency and insulin resistance.
2. The study was conducted at a single centre, which may limit generalizability.
3. Serum magnesium reflects extracellular magnesium and may not accurately represent intracellular magnesium stores.
4. Insulin resistance was assessed using TyG index, a surrogate marker, and was not validated against the hyperinsulinemic-euglycemic clamp in the present study.
5. Dietary magnesium intake, duration of diabetes, antidiabetic drug use, lipid-lowering therapy and other possible confounders were not fully assessed.

Future recommendations: Future studies should include larger multicentric cohorts to validate the relationship between serum magnesium, glycaemic control and insulin resistance in patients with type 2 diabetes mellitus. Longitudinal studies are needed to

clarify whether low magnesium levels precede worsening insulin resistance or occur as a consequence of poor glycaemic control. Studies assessing intracellular magnesium, ionized magnesium or magnesium loading tests may provide more accurate information regarding magnesium status.

Randomized controlled trials are required to evaluate whether magnesium supplementation can improve insulin sensitivity, TyG index, HbA1c and long-term diabetic outcomes. Future research should also evaluate the role of other micronutrients such as zinc and vitamin D along with magnesium in diabetes management.

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