

## Correlation of Serum Magnesium and Lipid Parameters in Patients of Type 2 Diabetes Mellitus

Sadia Khalid, Sabahat Haider, Madeeha Afaq, Zainab ul Ghazali, Maheen Shuja

### ABSTRACT

**Objective:** To ascertain the relationship between serum magnesium and lipid parameters in type 2 diabetes mellitus patients.

**Study design and Settings:** analytical study in the department of pathology Sheikh Zayed Hospital, Rahim Yar Khan.

**Methodology:** 100 adults were recruited by non-probability consecutive sampling with T2DM. The fasting venous blood serum magnesium, fasting plasma glucose, HbA1c, total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol were taken. The level of association among serum magnesium and lipid parameters was determined through Pearson correlation and linear regression.

**Results:** Mean age was 53.2  $\pm$  9.1 years and 54% were male. The average serum magnesium was 1.71  $\pm$  0.29 mg/dL; 47 of them had suffered hypomagnesemia. There were strong negative associations observed between serum magnesium and total cholesterol ( $r=-0.41$ ), triglycerides ( $r=-0.46$ ), LDL cholesterol ( $r=-0.43$ ) and HbA1c ( $r=-0.34$ ) and a positive association between serum magnesium and HDL cholesterol ( $r=0.39$ ), all  $p<0.001$ . Hypomagnesemic patients had increased triglycerides, LDL cholesterol, total cholesterol and HbA1c and decreased HDL cholesterol.

**Conclusion:** Reduced serum magnesium had a significant negative lipid profile connected with T2DM. Periodic magnesium monitoring could be employed to detect diabetic patients at increased cardiometabolic risk.

**Keywords:** Type 2 diabetes mellitus; serum magnesium; hypomagnesemia; lipid profile; triglycerides; LDL cholesterol; HDL cholesterol; dyslipidaemia; HbA1c; insulin resistance.

### How to cite this Article:

Khalid S, Haider S, Afaq M, Ghazali ZU, Shuja M. Correlation of Serum Magnesium and Lipid Parameters in Patients of Type 2 Diabetes Mellitus. J Bahria Uni Med Dental Coll. 2026;16(4):1037-43 DOI: <https://doi.org/10.51985/JBUMDC20261102>

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### INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a disease of metabolism that is also correlated with unremitting hyperglycaemia, which unfolds as a result of varying forms of insulin resistance and dysregulations of insulin secretions. Its burden rate in the world continues to increase particularly in low- and

middle-income countries with an upward trend on the rate of urbanization, sedentary activities, diet-rich high energy diets and obesity resulting in a rise in cardiometabolic disease. It has been documented that hundreds of millions of adults today worldwide have diabetes and the number will continue to grow in the coming decades. T2DM has more than just clinical implications on hyperglycaemia since it is a multisystem disease and associated with dyslipidaemia, hypertension, renal failure, retinopathy, neuropathy, cardiovascular disease and premature mortality. Since cardiovascular disease is the most frequent and frequent cause of death in diabetes, revealing biochemical indicators that reflect atherogenic lipid changes continues to have the priority in the scenario of preventive treatment.<sup>1</sup>

The high triglycerides, increased levels of small dense LDL and decreased levels of HDL cholesterol and fluctuating levels of the total cholesterol are the classical manifestations of diabetic dyslipidaemia. Insulin resistance, excesses in the production of very-low-density lipoproteins in the liver, malfunction in lipoprotein lipase, broken adipokines signalling and dysfunctional low-grade inflammation precipitate this lipid pattern. Qualitative LDL changes and elevated triglyceride-rich remnant particles raise risks to the vessels even in the conditions of the absence of LDL cholesterol significantly raised. This is why the regular testing of lipids is one of the fundamental elements of

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Received: 02-07-2026  
Accepted: 29-09-2026

1st Revision: 18-08-2026  
2nd Revision: 15-09-2026

diabetes care. Nevertheless, all differences in cardiovascular risk have not been explained by common lipid tests in diabetes patients and other modifiable metabolic factors have been explored.<sup>2,3</sup>

The fourth most abundant cation present in the human body and the essential cofactor in over 300 enzyme reactions, such as ATP metabolism, glucose transport, insulin receptor signalling and vascular tone reactions. Osmotic diuresis, Glycosuria, reduction of tubular reabsorption, diet deficiency, drug action and chronic inflammation in diabetic people can cause magnesium depletion. Hypomagnesemia has the potential to decrease the activity of insulin receptor tyrosine kinase and exacerbate insulin resistance, establishing a vicious cycle where hyperglycaemia increases urinary magnesium loss and low magnesium further worsens control of glucose. Population-based evidence has shown that the lower serum magnesium level is associated with higher insulin insensitivity and higher risk of T2DM.<sup>4,5</sup> The relationship between the metabolism of magnesium and lipid is biologically plausible. Magnesium has the potential to influence lipid profile through the effects to insulin action, lipoprotein lipase, lecithin-cholesterol acyltransferase, HMG-CoA reductase activity, oxidative stress and endothelial functioning. The condition of lowered magnesium level could thus be one of the reasons of increased triglycerides and LDL cholesterol and lower HDL cholesterol. Recent reviews and meta-analyses indicate that magnesium supplementation can potentially enhance certain glycaemic and lipid effects, but outcomes vary with population (and other factors), with supplement dose, and baseline magnesium status and duration of intervention.<sup>6,7</sup>

In diabetic populations, clinical studies have claimed that hypomagnesemia is prevalent as is related to poor glucose control, microvascular illness and cardiovascular difficulties. Magnesium Supplement A randomized study of 1300 patients with insulin-treated T2DM found no clear evidence of insulin sensitivity in response to magnesium Supplement, although some studies have found a beneficial effect of magnesium Supplement on fasting glucose levels and select lipid fractions in specific situations.<sup>8-10</sup> The aim of the current research was to identify the association between serum magnesium and lipid parameters in the patients with T2DM who attend the Department of Chemical Pathology, Sheikh Zayed Medical College/Hospital, Rahim Yar Khan. The practical clinical question that was focused on in the study was: is a lower serum magnesium correlate with an atherogenic lipid pattern in the regular diabetic patient? Having shown such correlation would help justify magnesium testing as an easy adjunct in assessing risks and would result in a framework to be used in future interventional research to determine whether lipid-related cardiovascular risks can be reduced with magnesium deficiency correction in T2DM.

## METHODOLOGY

The specimen of the analytical study was a cross-sectional study that was done within the Department of Pathology/Chemical Pathology, Sheikh Zayed Medical College/Hospital, Rahim Yar Khan between 16th March 2026 and 15th June 2026 following the approval of the institutional review board and the Research Evaluation Unit (Ref no. 129/IRB/SZMC/SZH dated 28-02-2025). Participants of the study were grown-ups with type 2 diabetes mellitus who approached the clinic to undergo a regular blood test. The approved synopsis formed the basis of the study title, setting and design whereby the objective found was the determination of the correlation of serum magnesium and lipid parameters in T2DM patients. All the participants signed an informed consent that was written before the process of blood sampling and the confidentiality was ensured during the data collection. The paper adhered to the ethics of the Declaration of Helsinki. The size of a sample was estimated with the help of calculating a correlation coefficient. We took  $r=0.30$  as the minimum clinically important correlation between serum magnesium and lipid parameters based on previous reports of moderate negative relationships between magnesium and atherogenic lipids. The required sample size based on 80% power ( $Z_{\beta}=0.84$ ) and 2-sided 95% confidence level ( $Z_{\alpha}=1.96$ ) on the formula of  $n=[(Z_{\alpha} + Z_{\beta})/0.5 \ln(1+r)/(1-r)]^2 + 3 = 85$ . To address incomplete samples and laboratory rejection and missing data, it was estimated that the sample should be supplemented by roughly 15% and the final sample of 100 patients was considered. The sampling was non-probability consecutive and follows the approved protocol.

Eligible patients included those who were aged above 30 years, diagnosed with T2DM beyond one year and had insulin or oral antidiabetic therapy. Both male and female patients were included. Patients were not included in case of receiving medications, which could influence glucose or lipid metabolism, such as systemic or local steroid treatment, anti-obesity medication, glucagon-like peptide-1 receptor agonist, statins, fibrates, omega-3 fatty acids or cholesterol absorption inhibitor. Patients on proton-pump inhibitors, diuretics or recent magnesium supplements and patients with diarrhoea or malabsorption were also outlived since they could change the serum magnesium levels. The data about the age, sex, years with diabetes, their level of diabetes control, hypertension, their smoking status, their location, their monthly earnings, their height, weight and their body mass index were entered on a structured proforma to every participant. 20 mL of venous blood (after an 8-12 fast) was transferred in 5 mL standard aseptic. HbA1c and serum samples (separated by centrifugation to determine magnesium and lipid profile) were collected in EDTA tubes and serum, respectively. The modified xylidyl blue colorimetric technique was chosen to measure serum magnesium. The fasting plasma glucose levels and lipid profiles such as a total

cholesterol, triglycerides, HDL cholesterol and LDL cholesterol levels were determined on an automated chemical analyser by standard enzymatic procedures. Directly measured or calculated based on laboratory policy when triglycerides fell within valid range LDL cholesterol was measured or calculated. The analysis of data was performed with the help of SPSS 25.0. Quantitative variables were reported in terms of the mean and Standard deviation and qualitative variables in terms of frequency and percentage. Hypomagnesemia was considered as serum magnesium of less than 1.70 mg/dL. The Shapiro-Wilk test and histograms were used to test normality. Lipid and glycaemic parameters of patients with low and normal magnesium were compared using independent-sample t test. Pearson correlation was used to determine correlation of serum magnesium with total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, fasting plasma glucose and HbA1c. Linear regression was adopted to examine independent predictor of serum magnesium on lipid parameters upon modifying age, sex, BMI, duration of diabetes and HbA1c. A p value of = 0.05 or less was taken to be statistically significant.

## RESULTS

The number of analyzed patients with T2DM was 100. The mean age was 53.2  $\pm$  9.1 years, with ages ranging from 33 to 72 years. It comprised of 54 male patients and 46 female patients. The mean duration of diabetes was 7.6  $\pm$  4.5 years, and the mean BMI was 28.1  $\pm$  4.2 kg/m<sup>2</sup>. Individuals who had led diabetes in relation to HbA1c classification were 42 as compared to those with uncontrolled diabetes 58. The number of hypertensive and current smokers was 38 and 25 respectively. The majority of the patients lived in urban (57 percent) and 43 percent in rural regions. It showed that such population of the study was made up of typical middle aged and elderly people with T2DM in which the burden of obesity, poor glycaemic control and blood pressure was great. The average magnesium level in the serum was 1.71  $\pm$  0.29 mg/dL. Forty-seven patients (47.0%) had hypomagnesemia, while 53 patients (53.0%) had normal serum magnesium levels. The mean fasting plasma glucose was 168.4  $\pm$  47.8 mg/dL and mean HbA1c was 8.2  $\pm$  1.6%. Mean total cholesterol was 204.6  $\pm$  38.9 mg/dL, mean triglycerides were 184.8  $\pm$  76.5 mg/dL, mean LDL cholesterol was 128.2  $\pm$  35.1 mg/dL and mean HDL cholesterol was 39.8  $\pm$  8.6 mg/dL. Therefore, atherogenic with increased triglycerides and LDL cholesterol and almost a low level of HDL cholesterol were average lipid profile. Lipid parameter derived and based on magnesium status comparison provided a worse result to the lipid parameters of the patients who were magnesium deficient. In the low magnesium group, the mean total cholesterol was 222.3  $\pm$  34.6mg/dL as compared to the normal magnesium group where it was 188.9  $\pm$  34.8mg/dL ( $p < 0.001$ ). Mean triglycerides were 221.2  $\pm$  73.5 mg/dL versus 152.5  $\pm$  58.2 mg/dL ( $p < 0.001$ ), and mean LDL cholesterol was 144.1

$\pm$  32.4 mg/dL versus 114.1  $\pm$  31.2 mg/dL ( $p < 0.001$ ). Contrastingly, patients with hypomagnesemia (36.4  $\pm$  7.6 mg/dL) had a lower level of HDL cholesterol in comparison to that of patients with normal levels of magnesium (42.8  $\pm$  8.3 mg/dL;  $p < 0.001$ ). HbA1c was also higher in the hypomagnesaemia group (8.8  $\pm$  1.5% versus 7.6  $\pm$  1.4%;  $p < 0.001$ ). The Pearson correlation analysis found that all the lipid fractions and serum magnesium had statistically significant relationships. The serum magnesium had a moderate negative correlation with total cholesterol ( $r = -0.41$ ,  $p = 0.001$ ), triglycerides ( $r = -0.46$ ,  $p = 0.001$ ) and LDL cholesterol ( $r = -0.43$ ,  $p = 0.001$ ). The HDL cholesterol was positively related with a significant degree of correlation ( $r = 0.39$ ,  $p = 0.001$ ). HbA1c ( $r = -0.34$ ) and fasting plasma glucose ( $r = -0.29$ ) were also inversely related to serum magnesium ( $p = 0.001$ ;  $p = 0.004$ ). These results indicate not only did lower magnesium correlate with poorer glycaemic control, but also a poorer lipid profile. Adjusting serum magnesium was also found to have independent predictability of triglycerides and LDL cholesterol in relation to age, sex, BMI, duration of diabetes and HbA1c. The difference of 0.1mg/dl increase in serum magnesium was observed to decrease the triglycerides and LDL cholesterol by an average of 9.8 and 4.1 respectively. Magnesium in serum was also a predictor of HDL cholesterol independent of other factors with higher magnesium levels predicting greater values in the HDL. The triglycerides and LDL cholesterol variance accounted by the model was 31 and 27 percent, respectively. These findings confirm the study hypothesis which includes that serum magnesium has a significant correlation with lipid parameters in T2DM.

Table 1. Baseline demographic and clinical characteristics of patients with T2DM (n=100)

| Variable                                    | Value          |
|---------------------------------------------|----------------|
| Age (years), mean $\pm$ SD                  | 53.2 $\pm$ 9.1 |
| Male sex                                    | 54 (54.0%)     |
| Female sex                                  | 46 (46.0%)     |
| Duration of diabetes (years), mean $\pm$ SD | 7.6 $\pm$ 4.5  |
| BMI (kg/m <sup>2</sup> ), mean $\pm$ SD     | 28.1 $\pm$ 4.2 |
| Controlled diabetes                         | 42 (42.0%)     |
| Uncontrolled diabetes                       | 58 (58.0%)     |
| Hypertension                                | 38 (38.0%)     |
| Current smoker                              | 25 (25.0%)     |
| Urban residence                             | 57 (57.0%)     |
| Rural residence                             | 43 (43.0%)     |

Table 2. Biochemical profile of study participants (n=100)

| Parameter                      | Mean +/- SD    | Minimum | Maximum |
|--------------------------------|----------------|---------|---------|
| Serum magnesium (mg/dL)        | 1.71 +/- 0.29  | 1.05    | 2.38    |
| Fasting plasma glucose (mg/dL) | 168.4 +/- 47.8 | 88      | 312     |
| HbA1c (%)                      | 8.2 +/- 1.6    | 5.9     | 12.4    |
| Total cholesterol (mg/dL)      | 204.6 +/- 38.9 | 132     | 312     |
| Triglycerides (mg/dL)          | 184.8 +/- 76.5 | 72      | 438     |
| LDL cholesterol (mg/dL)        | 128.2 +/- 35.1 | 58      | 226     |
| HDL cholesterol (mg/dL)        | 39.8 +/- 8.6   | 23      | 64      |

Table 3. Comparison of lipid and glycaemic parameters according to magnesium status

| Parameter                      | Low Mg (n=47)  | Normal Mg (n=53) | Mean difference | p value |
|--------------------------------|----------------|------------------|-----------------|---------|
| Total cholesterol (mg/dL)      | 222.3 +/- 34.6 | 188.9 +/- 34.8   | 33.4            | <0.001  |
| Triglycerides (mg/dL)          | 221.2 +/- 73.5 | 152.5 +/- 58.2   | 68.7            | <0.001  |
| LDL cholesterol (mg/dL)        | 144.1 +/- 32.4 | 114.1 +/- 31.2   | 30.0            | <0.001  |
| HDL cholesterol (mg/dL)        | 36.4 +/- 7.6   | 42.8 +/- 8.3     | -6.4            | <0.001  |
| Fasting plasma glucose (mg/dL) | 183.9 +/- 49.4 | 154.7 +/- 42.3   | 29.2            | 0.002   |
| HbA1c (%)                      | 8.8 +/- 1.5    | 7.6 +/- 1.4      | 1.2             | <0.001  |

Table 4. Pearson correlation of serum magnesium with lipid and glycaemic parameters

| Variable correlated with serum magnesium | Correlation coefficient (r) | p value | Interpretation    |
|------------------------------------------|-----------------------------|---------|-------------------|
| Total cholesterol                        | -0.41                       | <0.001  | Moderate negative |
| Triglycerides                            | -0.46                       | <0.001  | Moderate negative |
| LDL cholesterol                          | -0.43                       | <0.001  | Moderate negative |
| HDL cholesterol                          | 0.39                        | <0.001  | Moderate positive |
| Fasting plasma glucose                   | -0.29                       | 0.004   | Weak negative     |
| HbA1c                                    | -0.34                       | 0.001   | Moderate negative |

Table 5. Multivariable linear regression showing independent association of serum magnesium with lipid parameters

| Dependent variable | Adjusted beta for serum Mg | 95% CI          | p value | Model R2 |
|--------------------|----------------------------|-----------------|---------|----------|
| Total cholesterol  | -38.6                      | -58.9 to -18.3  | <0.001  | 0.24     |
| Triglycerides      | -98.0                      | -135.6 to -60.4 | <0.001  | 0.31     |
| LDL cholesterol    | -41.4                      | -60.2 to -22.6  | <0.001  | 0.27     |
| HDL cholesterol    | 9.2                        | 4.2 to 14.1     | <0.001  | 0.21     |

## DISCUSSION

This study found a significant relationship between serum magnesium and lipid parameters in patients with T2DM. Almost half of the study population was hypomagnesemic and low magnesium level was linked with increased total cholesterol, triglycerides and LDL cholesterol levels and reduced HDL cholesterol. These results have clinical implications since diabetic dyslipidaemia is a leading cause of cardiovascular morbidity, and mortality. The pattern observed here indicates that magnesium deficiency might not necessarily be just existing with diabetes but in association with the biochemical environment that favors atherogenic

dyslipidaemia. The correlation did not diminish with the age, sex, BMI, diabetes length and HbA1c implying that magnesium and lipid metabolism have an independent relationship. Hypomagnesemia was common in the current analysis and this was found to be 47%. It aligns with a systematic review by Pitliya et al., who revealed that, in observational studies, hypomagnesaemia is prevalent in T2DM, but with varying rates based on population, lab cut-off and renal function.<sup>11</sup> Abdullah et al. also found a high burden of hypomagnesemia by T2DM in a resource-poor setting, with obesity and glycaemic control surging together.<sup>12</sup>

The negative correlation between serum magnesium and

HbA1c and fasting glucose is consistent with Li et al., who determined that low serum magnesium correlates with the combination of increased insulin resistance and increased diabetes risk in a large adult population.<sup>13</sup> Our study did not directly assess complications but the combination of low magnesium, high glycaemic control, and diverse lipidemia is a well-established risk constellation of long-term vascular disease. The likely action mechanism is that magnesium deficiency will affect insulin receptor phosphorylation, and post-receptor signalling, resulting in a decrease in glucose uptake, hepatic glucose production, and insulin resistance. Our study revealed the strongest correlation to be between magnesium and triglycerides ( $r=-0.46$ ). This observation promotes some prior clinical observations that low magnesium correlates with dyslipidaemia characterized by high triglycerides.<sup>14</sup> Asbaghi et al. evaluated randomized trials and discovered that magnesium supplementation decreased LDL cholesterol but inconsistently influenced triglycerides, total cholesterol and HDL cholesterol in T2DM patients.<sup>15</sup> The imbalance between interventional and observational findings could be attributed to differences in baseline magnesium deficiency, dose of magnesium supplement, duration of treatment and previous lipid-lowering therapy. In cross-sectional, magnesium can indicate a more general metabolic condition, such as insulin resistance, dietary quality and renal magnesium wasting.

We also found that serum magnesium is negatively correlated to LDL cholesterol and this is in line with the systematic review of cardiovascular risk carried out by Oost et al. who clarified that hypomagnesaemia can be a contributor to vascular risk by dyslipidaemia, endothelial dysfunction, inflammation and arrhythmogenic processes.<sup>16</sup> In another cohort, Oost et al. reported inverse associations of serum magnesium with heart failure, atrial fibrillation and microvascular complications in T2DM.<sup>17</sup> This would mean that magnesium is more than just lipid control in the context of cardiometabolic health. The increase in LDL cholesterol in the low magnesium group could represent the way that magnesium depletion leads to cardiovascular risk. Another observation that is of importance is the positive correlation between magnesium and HDL cholesterol. In diabetes, HDL may change its quality and activity; it has anti-inflammatory properties and a reverse cholesterol transporter. A characteristic of diabetic dyslipidaemia is low HDL cholesterol. Magnesium can possibly indirectly stimulate HDL by enhancing insulin recruitment and bringing down the exchange of the triglyceride-rich lipoproteins. Other minor studies of diabetic patients have shown similar positive associations but not all. Inacio et al. demonstrated a correlation between diminished magnesium and unfavorable lipid profile in diabetes, but also correlated magnesium status with diabetic retinopathy.<sup>18</sup> Despite the fact that this study included type 1 diabetes, the evidence supports the larger association of magnesium and vascular metabolic risk.

There is ambivalent interventional evidence. In a meta-analysis comparing the selected glycaemic outcomes of oral magnesium supplementation in T2DM, Asbaghi et al. reported that magnesium supplementation was clinically relevant, whereas magnesium failed to enhance insulin-treated patients with low magnesium with established insulin resistance.<sup>19</sup> means that magnesium supplementation is unlikely to invariably benefit glycaemic in diabetic and high-risk populations; nevertheless, hypercholesterolaemia and blood pressure have been shown to be improved.<sup>20</sup> Our paper, however, must be taken as being a piece of evidence of association and not demonstration that supplementation will correct dyslipidaemia.

Low- vs normal magnesium groups had significantly higher total cholesterol, triglycerides, and LDL cholesterol in the low magnesium group. These results are in accord with recent local and regional reports mentioned in the accepted synopsis with studies indicating negative correlations of magnesium with triglycerides, cholesterol and LDL cholesterol and positive correlation with HDL cholesterol. Correlations in the current model were moderate as opposed to very strong, biologically sensible since the lipid parameters are affected by numerous factors such as diet, obesity, glycaemic control, hepatic insulin resistance, renal and renal medications. The protocol also was enhanced by the exclusion of statin and fibrate users, making it possible to see the association between magnesium and lipids in nature.<sup>21,22</sup> According to the regression analysis, serum magnesium alone was an independent predictor of triglycerides and LDL cholesterol. This is in line with mechanistic evidence suggesting magnesium influences lipid-method related enzymes and insulin-mediated inhibition of lipid metabolism. Increase in magnesium deficiency of insulin resistance leads to magnesium deficiency, which in turn enhances the free fatty acid flux of adipose tissue, elevates liver VLDL synthesis and triglycerides. The harmful effects of a larger triglyceride can lower HDL by exchanging cholesterol ester transfer protein, and induce small dense LDL. The following mechanism could be the reason of the negative correlation between magnesium and triglycerides and LDL cholesterol and positive correlation between magnesium and HDL cholesterol in the current study.

The results also lend weight to an individualistic approach to the future intervention investigations. Participants with verified hypomagnesemia, reported dietary intake and muscular magnesium loss, and kidney functioning and complementary treatment should be favored in trials. Recurring levels of magnesium would explain that in the cases of persistent deficiency, the lipid abnormalities would be more closely related, compared to the baseline sample. Secondly, measuring apolipoprotein B, non-HDL cholesterol, remnant cholesterol, inflammatory biomarkers, and insulin resistance may also offer a more comprehensive picture of the biological mechanism between magnesium deficiency

and cardiovascular risk in T2DM and enhance the clinical analysis of the results.

The size of the group differences is clinically significant. Hypomagnesemia patients exhibited a significantly higher level of triglycerides and LDL cholesterol and a decreased level of HDL cholesterol. The fact that these differences cannot prove causality indicates that magnesium status is potentially useful in helping to identify a subgroup of patients with a less favorable metabolic phenotype. When dyslipidaemia is also unproportional or challenging to treat, serum magnesium may be a physiological supplement to HbA1c, renal function, medication history, and lipid profile in routine practice. Nevertheless, magnesium measurement is advised to be used as a supplement to the known heart disease risk evaluation and evidence-based lipid-lowering therapy. The current results also should be interpreted as a background of magnesium homeostasis in diabetes. The level of magnesium in serum is an indication of the fraction in circulation only and could be at a reference level with the intracellular storage being depleted. Therefore, the associations observed could be understatement of the actual association between magnesium deficiency and abnormal lipid metabolism. Continuous hyperglycaemia fosters osmotic diuresis and regulation of magnesium in the urine, whilst insulin resistance can limit the uptake of magnesium in the cells. Simultaneously, magnesium deficiency can further disrupt insulin signalling, enhance oxidative stress, and stimulate inflammatory processes that negatively influences the synthesis and clearance of lipoproteins. Such a two way interaction could be the reason as to why the patient with lower magnesium exhibited both worse glycaemic regulation and a more atherogenic lipid profile.

The research has a number of strengths. It employed fasting blood, standardized biochemical testing, and clear-cut exclusion criteria to minimize drug and condition confounding of magnesium or lipid metabolism. It also performed the group comparisons and correlation/regression analyses also offering a more detailed evaluation of magnesium-lipid relationship. Limitations: Nevertheless, there are drawbacks that need to be addressed. There was no causality in the cross-sectional design. Serum magnesium is a small part of body magnesium and may not be an accurate measure of intracellular magnesium. Dietary magnesium intake, renal function markers, urinary magnesium excretion, apolipoproteins and inflammatory markers were not included in the model analysis.

Irrespective of these shortcomings, there are practice implications. Serum magnesium estimation might be helpful in the adjunctive laboratory parameters in patients with T2DM and predominantly dyslipidaemia and weakly controlled glucose levels. The detection of hypomagnesemia may initiate dietary advice, revisitation of magnesium-reducing medicines, urinary testing and supplemental administration where clinically right. Future prospective

research highly needs to determine whether the amelioration of magnesium deficiency enhances the lipid profile, glycaemic control and cardiovascular outcomes in patients with T2DM. Participants in randomized controlled studies should be stratified (according to baseline magnesium status, renal capability, lipid lowering treatment and diabetes duration) to determine the group who may gain the most.

## CONCLUSION

In patients with T2DM serum magnesium revealed significant negative relationship with total cholesterol, triglycerides, LDL cholesterol, fasting plasma glucose and HbA1c amount and positive relationship with HDL cholesterol. An atherogenic lipid profile was related to hypomagnesemia, which was frequent. Serum magnesium can be regarded as a simple adjunctive biochemical indicator to diagnose diabetic individuals with increased metabolic and cardiovascular risk.

**Conflict of Interest:** None

**Fundings:** No funding source

**Use of Artificial Intelligence (AI) Disclosure:** There is no AI tool used.

### Authors Contribution:

**Sadia Khalid:** Conception and design, acquisition of data analysis and interpretation of data, drafting and critical revision, final approval of the version to be published.

**Sabahat Haider:** Conception and design, acquisition of data analysis and interpretation of data, drafting and critical revision, final approval of the version to be published

**Madeeha Afaq:** Conception and design, acquisition of data analysis and interpretation of data

**Zainaul ul Shazali:** drafting and critical revision, final approval of the version to be published

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