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Article

Relationship Between Serum Magnesium and Uric Acid Concentrations in Patients with Type 2 Diabetes Mellitus

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Abstract: Diabetes type 2 is a significant public health issue, Impacting adults worldwide. The search aim to evaluate the serum level of Mg and UA and also to investigate the relationship between the two variables with other variables used in the research in type 2 diabetes patients. Sixty samples were included in this study, they were divided into two groups; controls group which involved 30 persons , and patients group involved 30 persons , the sample collection has been attended from Abu Ghureb General hospital and some private laboratories in Al-Fallujah city, for the sampling period was from December 2022 to April 2023 and the ages of all samples was ranged from 30-90 year. The study quoted the levels of Magnesium, Uric acid and Glucose in serum. The findings indicated that there is statistically significant difference between infected and non-infected individuals with regard to weight gain and age increase, therefore, there is a higher possibility of getting diabetes as age increase and weight gain is increased. Concerning height, we do recognise that those who are more tall are people that are not as prone to diabetes as those who are shorter. When it comes to body mass indexes, this results in an enhanced chance of acquiring diabetes. The infected fasting person has a higher blood sugar level than healthy people. As for both magnesium, uric acid , and glucose , the percentage of magnesium in infected people is lower than that of healthy people. That people who have diabetes have high levels of both uric acid and glucose.

Keywords: Type 2 diabetes mellitus; Serum magnesium; Uric acid; Insulin resistance; Fasting serum glucose.

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1. Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease caused by insulin resistance in the peripheral tissues (skeletal muscle, adipose tissue, and liver) with concurrent progressive pancreatic β-cell dysfunction and inadequate insulin secretion. Recent evidence indicates that dysfunction of insulin receptor signaling in the pancreatic β-cell is a direct cause of β-cell dysfunction and a driver of disease progression. Experimental studies have provided evidence using insulin receptor knockdown in INS-1 pancreatic β-cells that disruption of insulin signalling negatively impacts on the function of pancreatic β-cells, highlighting the importance of insulin receptor activity in glucose homeostasis [1]. As it is becoming more common, T2DM is one of the most serious public health problems in the world, and the worldwide burden of T2DM is projected to more than double by 2045. Currently, there are ten classes of antidiabetic medications licensed for the treatment of T2DM, demonstrating the difficulty of T2DM management and the need for personalized treatment [2].

In T2DM, hyperglycaemia causes microvascular complications such as diabetic nephropathy, retinopathy and neuropathy over time, which significantly affects patients' quality of life, morbidity and mortality. Apart from these classic complications, sexual dysfunction has become an important, but often underappreciated microvascular complication. The pathogenesis is polyetiological and consists mainly of endothelial dysfunction, vascular impairment, autonomic neuropathy, and chronic inflammation caused by the high blood sugar level for extended periods of time [3].

Type 2 diabetes is a multifactorial disease that has a genetic predisposition, environmental factors, obesity, and unhealthy lifestyle behaviors. In addition to pharmacological therapy, in-depth behavioral changes are needed to slow the course of the disease, avoid complications, and enhance quality of life in the long run [4]. The disease arises when insulin secretion by the pancreas is inadequate to offset insulin resistance, a pathological state in which there is a reduced physiological response of peripheral tissues to insulin in the blood causing an inability to take up glucose and maintenance of hyperglycemia [5].

Body mass index (BMI) is the most common anthropometric measurement, which is simple and feasible both clinically and epidemiologically, and is one of the strongest modifiable risk factors for T2DM. However, BMI is an indirect measurement of body fat that cannot differentiate fat and lean body mass. Therefore, BMI can be inaccurate in certain groups of people, including children, older adults, athletes, people who are losing or gaining a lot of weight, and women who are pregnant. More recently, therefore, there has been a focus on developing standards based on direct measures of body fat, instead of surrogate anthropometric indices [6]. Physical inactivity and high BMI have been consistently found to be independent risk factors for T2DM, and the relative contribution of these factors is still being investigated [7]. Clinical studies have demonstrated that the BMI is significantly higher in people with diabetes compared with healthy individuals, and obesity is closely linked with diabetes [8]. Additionally, BMI and waist-to-hip ratio (WHR) are independent risk factors for developing T2DM, suggesting that both general and central obesity play a role in insulin resistance and metabolic dysfunction [9].

Magnesium is the fourth most common mineral present in the human body and the second most abundant intracellular cation and is involved in over 300 enzyme reactions related to glucose metabolism, insulin signalling, and energy production. Deficiency of magnesium has been linked to poor glycaemic control, increased insulin resistance, and increased risk of diabetic microvascular complications, specifically diabetic retinopathy. Hence, the daily consumption of sufficient amount of magnesium has been suggested as a possible approach for better metabolic control and the control of diabetes related complications [10]. The serum level of magnesium rarely reflects total body level, as much of the magnesium is found intracellularly. Therefore, intracellular magnesium levels in erythrocytes or mononuclear cells or skeletal muscle is more accurate to determine magnesium status, and the level of magnesium in mononuclear cells is most closely correlated with that in muscle [11].

T2DM is associated with several biochemical abnormalities that have a role in its progression and metabolic disturbances. In clinical studies, salivary amylase activity has been shown to change: low amylase activity has been found in obese people and the secretion of pancreatic amylase has been found to be less in insulin-dependent diabetics. These results indicate a relationship between glucose metabolism and digestive enzyme activity, but the involved mechanisms and their clinical relevance are not completely understood [12]. The clinical hallmark of T2DM is insulin resistance, which is a state of insulin resistance due to the dysfunction of numerous intracellular signaling pathways and ultimately leads to the impaired utilization of glucose, chronic hyperglycemia and progressive metabolic abnormalities [13].

2. Methodology

Specific The study was designed as a cross-sectional study and carried out in the hospitals of Abu Ghraib and some private laboratories in Iraq from December 2022 to April 2023. The study sample comprised 60 subjects aged from 30 to 90 years who fulfilled the inclusion criteria. With informed consent, venous blood was taken from each person (around 5 mL) using a sterile disposable syringe in an aseptic manner. The blood was allowed to clot and then centrifuged to remove the serum. The serum obtained was split into two portions, one of which was immediately used for biochemical analysis, and the other was placed in two sterile 250-µL Eppendorf microcentrifuge tubes and stored at −20°C until further biochemical analysis was performed as needed.

Statistical analysis:

IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA) was used for the statistical analysis. Continuous variables are expressed as mean ± standard deviation (SD) and categorical variables are expressed as frequencies and percentages. The Shapiro–Wilk test was used to determine the normality of data. Independent-samples t-test was used for normally distributed data while the Mann–Whitney U test was used for non-normally distributed data for comparison between groups. Chi-square test or Fisher's exact test, as appropriate, was used to assess any association between categorical variables. Pearson's or Spearman's correlation coefficient was used to determine the correlation between continuous variables based on the distribution of the data. A P value of < 0.05 was regarded as statistically significant for the two tailed P value test.

Ethical approval:

The study was carried out according to the ethical principles of the Declaration of Helsinki and received the approval of the proper Institutional Ethics Committee prior to the recruitment of the participants. All participants gave written informed consent after full information concerning the goals and procedures of the study were provided. All personal and clinical information was kept confidential and private throughout the study, while all data collected was utilized for scientific research purposes only.

3. Results

Comparison of age between patients with type 2 diabetes mellitus and healthy control subjects.

Table 1 shows that there was no statistically significant difference in mean age between the patients with Type 2 Diabetes Mellitus (40.83 ± 9.52 years) and healthy control group (38.93 ± 10.23 years) (P = 0.459). These results suggest that the study groups were not significantly different in age, putting age as a confounding factor at minimal risk in further analysis comparing serum Mg level with uric acid level.

Table 1. Baseline Demographic Characteristics of Patients with Type 2 Diabetes Mellitus and Healthy Controls Included in the Study.

Parameter	Controls	T2DM patients	p-value
	Mean ± S.D	Mean ± S.D	
Age (Years)	38.93± 10.232	40.83± 9.520	0.459

Comparison of body weight and height between patients with type 2 diabetes mellitus and healthy control subjects.

Overall, anthropometric measures are significantly different between study groups as shown in Table 2. The mean body weight of diabetic patients with type 2 diabetes mellitus (T2DM) was significantly higher than that of the healthy control group (77.72 ± 17.56 vs. 67.20 ± 10.28 kg, P = 0.020). In contrast, the mean height was significantly lower

among patients with T2DM compared with controls (160.92 ± 14.09 vs. 165.83 ± 10.49 cm, P = 0.009) [14]. These results suggest that there are marked variations between diabetic and healthy people with respect to their body composition, including body weight.

Table 2. Comparison of Anthropometric Characteristics Between Patients with Type 2 Diabetes Mellitus and Healthy Controls Included in the Study.

Parameter	controls	T2DM patients	p-value
	Mean ± S.D	Mean ± S.D	
Weight	67.20±10.280	77.72±17.563	0.020
Heigh	165.83±10.488	160.92±14.090	0.009

Parameter	controls	T2DM patients	p-value
	Mean ± S.D	Mean ± S.D	
BMI (kg/m^2)	24.4591±3.13591	30.6666±9.77843	0.002

Comparison of fasting serum glucose (FSG) concentrations between patients with type 2 diabetes mellitus and healthy controls.

As seen in the table 3, the level of fasting serum glucose (FSG) was significantly higher in patients of type 2 diabetes mellitus when compared to healthy controls (169.20 ± 86.16 vs 86.37 ± 11.30 mg/dL). This difference was highly statistically significant (P<0.001) and confirmed that there was significant hyperglycemia among those with T2DM and justified a clinical distinction between the diabetic and non-diabetic patients [15], [16].

Table 3. Comparison of Fasting Serum Glucose Levels Between Patients with Type 2 Diabetes Mellitus and Healthy Control Participants.

Parameter	controls	patients	p-value
	Mean ± S.D	Mean ± S.D	
FSG (mg/dL)	86.37 ± 11.303	169.20 ± 86.163	0.0001

Comparison of serum magnesium levels between patients with type 2 diabetes mellitus and healthy controls.

Table 4 shows that the serum magnesium level was significantly decreased in patients with type 2 diabetes mellitus when compared to healthy controls (0.53 ± 0.75 vs. 1.30 ± 0.32 mg/dL), suggesting a considerable loss of magnesium status in diabetic patients [17]. The results of this study indicate that hypomagnesemia could be related to T2DM and this could suggest that magnesium deficiency could be involved in the metabolic abnormalities seen in diabetic patients.

Table 4. Comparison of Serum Magnesium Concentrations Between Patients with Type 2 Diabetes Mellitus and Healthy Control Participants.

Parameter	Controls	patients	p-value
	Mean ± S.D	Mean ± S.D	
Mg (mg/dL)	1.3010± 0.31984	0.5267 ± 0.75239	>0.0001

Comparison of serum uric acid levels between patients with type 2 diabetes mellitus and healthy controls.

As shown in Table 5, the serum uric acid levels were significantly higher in patients with type 2 diabetes mellitus than in healthy controls (3.58 ± 1.78 vs. 2.18 ± 0.81 mg/dL). The difference was highly statistically significant (P < 0.0001) suggesting more

hyperuricemia in patients with T2DM. The results indicate that, hyperuricemia is a potential marker of the metabolic dysfunction in type 2 diabetes mellitus and may be linked with the metabolic abnormalities in the disease [18], [19].

Table 5. Comparison of Serum Uric Acid Concentrations Between Patients with Type 2 Diabetes Mellitus and Healthy Control Participants.

Parameter	controls	patients	p-value
	Mean ± S.D	Mean ± S.D	
Uric acid (mg/dL)	2.1833± 0.81414	3.5800 ± 1.77578	> 0.0001

Correlation analysis of serum magnesium with fasting serum glucose, uric acid, age, weight, height, and body mass index.

Table 6 shows significant negative correlation between serum magnesium and serum uric acid (r = –0.424, P = 0.001) and body mass index (BMI) (r = –0.345, P = 0.007) - the lower the magnesium the higher the uric acid and the higher the BMI. On the other hand, the serum magnesium showed significant positive correlation with height (r = 0.379; P = 0.003) [20], [21]. There were no significant correlations between serum magnesium and fasting serum glucose (r = −0.198, P = 0.129), age (r = −0.178, P = 0.174), or body weight (r = −0.142, P = 0.278) suggesting that these were not significantly associated with serum magnesium in patients with type 2 diabetes mellitus.

Table 6. Correlation Between Serum Magnesium Levels and Clinical, Anthropometric, and Biochemical Parameters in Patients with Type 2 Diabetes Mellitus.

Magnesium (mg/dL)	r	p-value
FSG (mg/dL)	-0.198	0.129
Uric acid (mg/dL)	-0.424	0.001
Age (Years)	-0.178	0.174
Weight (cm)	-0.142	0.278
Height (cm)	0.379	0.003
BMI (kg/m^2)	-0.345	0.007

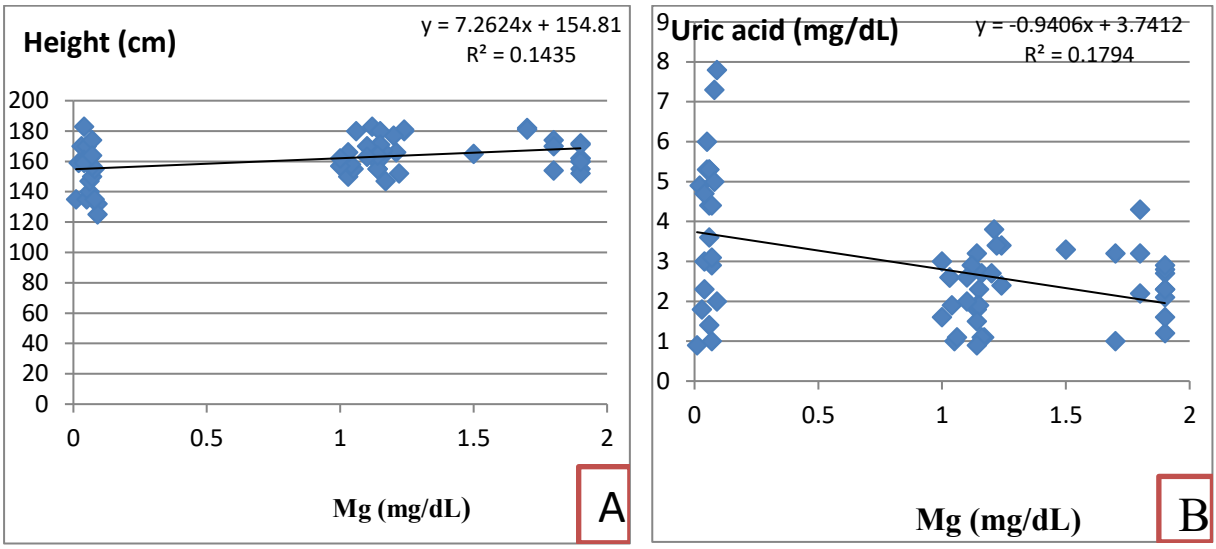

Figure 1. Correlation between Serum Magnesium (Mg) and Height (A), and between Serum Magnesium (Mg) and Uric Acid (B).

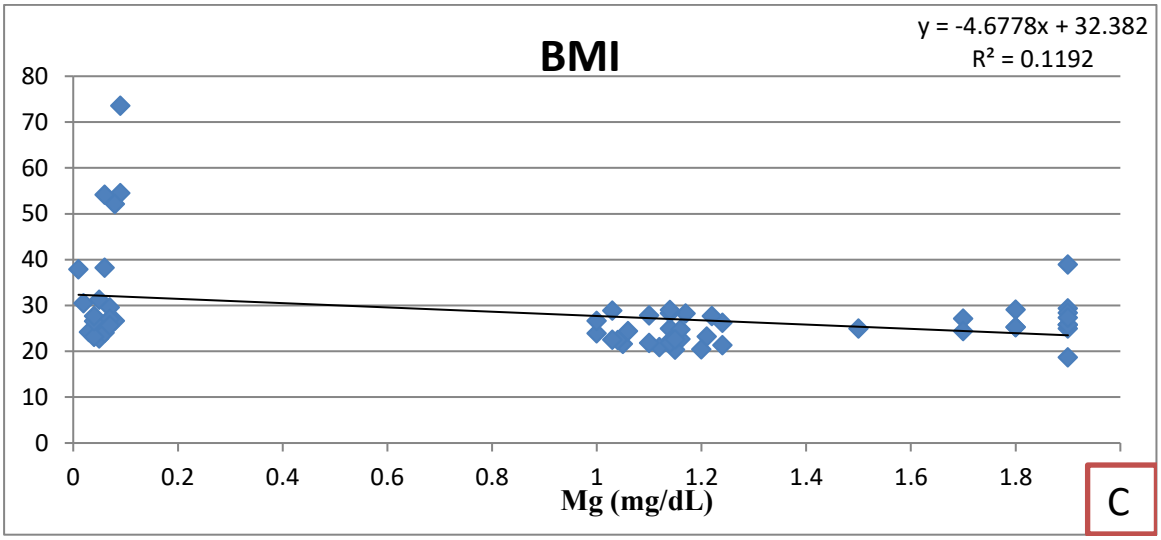

Figure 2. Correlation of Mg with BMI.

Correlation analysis of serum uric acid with fasting serum glucose, magnesium, age, weight, height, and body mass index.

As shown in the Table 7, there was a significant positive correlation between serum uric acid and fasting serum glucose (r = 0.362, P = 0.004) and serum BMI (r = 0.336, P = 0.009) where increased uric acid levels were associated with worse glycemic control and greater adiposity [22]. In contrast, serum uric acid negatively correlated with serum magnesium (r = −0.424, P = 0.001) and height (r = −0.320, P = 0.003). The age and the body weight were not significantly related to serum uric acid (r = −0.176, P = 0.174, r = 0.123, P = 0.348, respectively).

Table 7. Correlation Between Serum Uric Acid Levels and Clinical, Anthropometric, and Biochemical Parameters in Patients with Type 2 Diabetes Mellitus.

Uric acid (µg/dL)	r	p-value
FSG (mg/dL)	0.362	0.004
Magnesium (mg/dL)	-0.424	0.001
Age (Years)	-0.176	0.174
Weight (cm)	0.123	0.348
Height (cm)	-0.320	0.003
BMI (kg/m²)	0.336	0.009

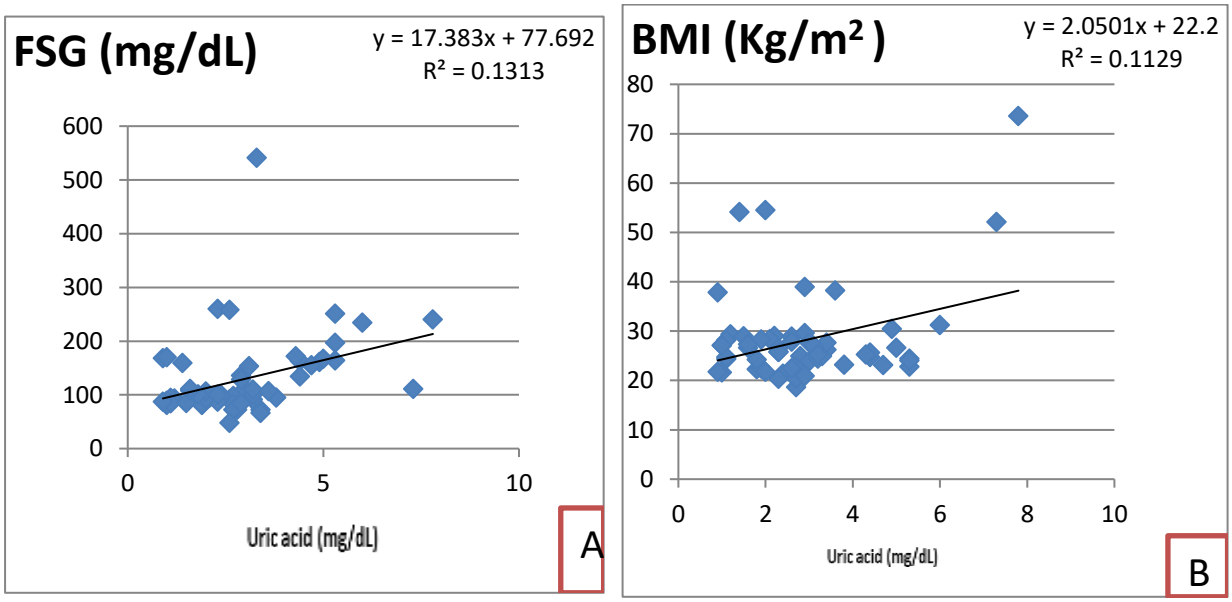

Figure 3. Correlation of Serum Uric Acid with Fasting Serum Glucose (FSG) (A) and Body Mass Index (BMI) (B).

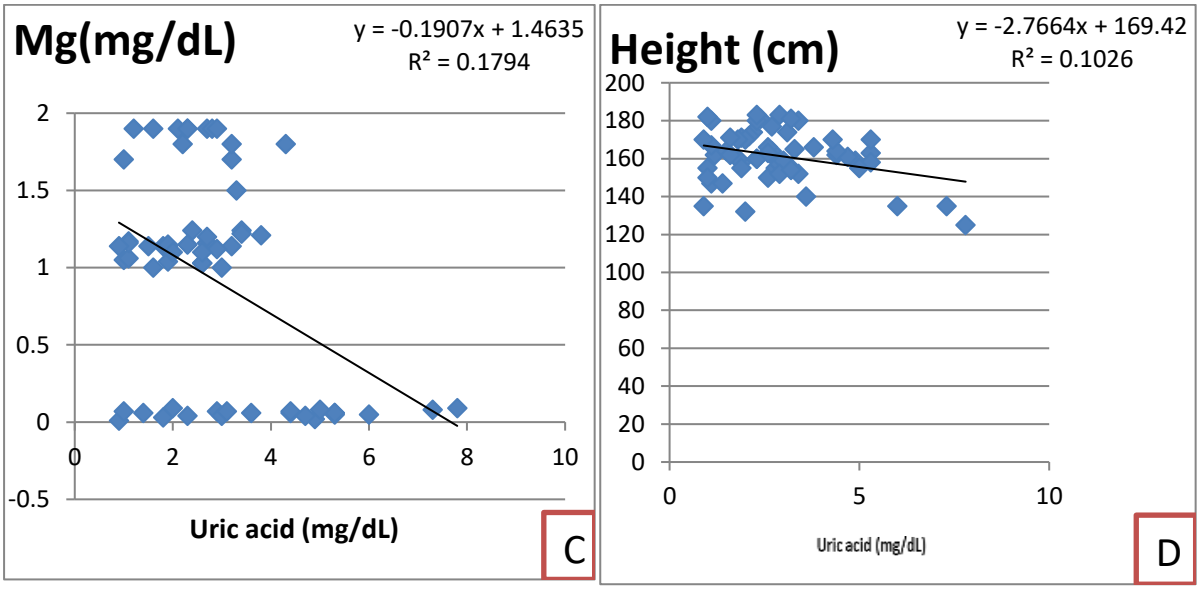

Figure 4. Correlation between Serum Uric Acid and Fasting Serum Glucose (FSG) (A), Body Mass Index (BMI) (B), Magnesium (Mg) (C), and Height (D).

Diagnostic performance of demographic, anthropometric, and biochemical variables for identifying patients with type 2 diabetes mellitus.

The receiver operating characteristic (ROC) analysis of the variables investigated is presented in Table 8. Fasting serum glucose (FSG) produced the best results with an area under the curve (AUC) of 1.000 ($P < 0.0001$). Body mass index (AUC = 0.747, P = 0.001), serum uric acid (AUC = 0.741, P = 0.001) and body weight (AUC = 0.675, P = 0.020) had good discriminatory ability to distinguish patients with type 2 diabetes mellitus from healthy controls. Conversely, the levels of serum magnesium (AUC = 0.206, $P < 0.0001$) and height (AUC = 0.337, P = 0.030) had inverse discriminatory performance, with lower levels in diabetic patients [23], [24]. Age did not show good diagnostic accuracy (AUC = 0.566, P = 0.379) and was not significantly associated with disease status.

Table 8. Receiver Operating Characteristic Analysis of Clinical and Biochemical Parameters for Discriminating Patients with Type 2 Diabetes Mellitus.

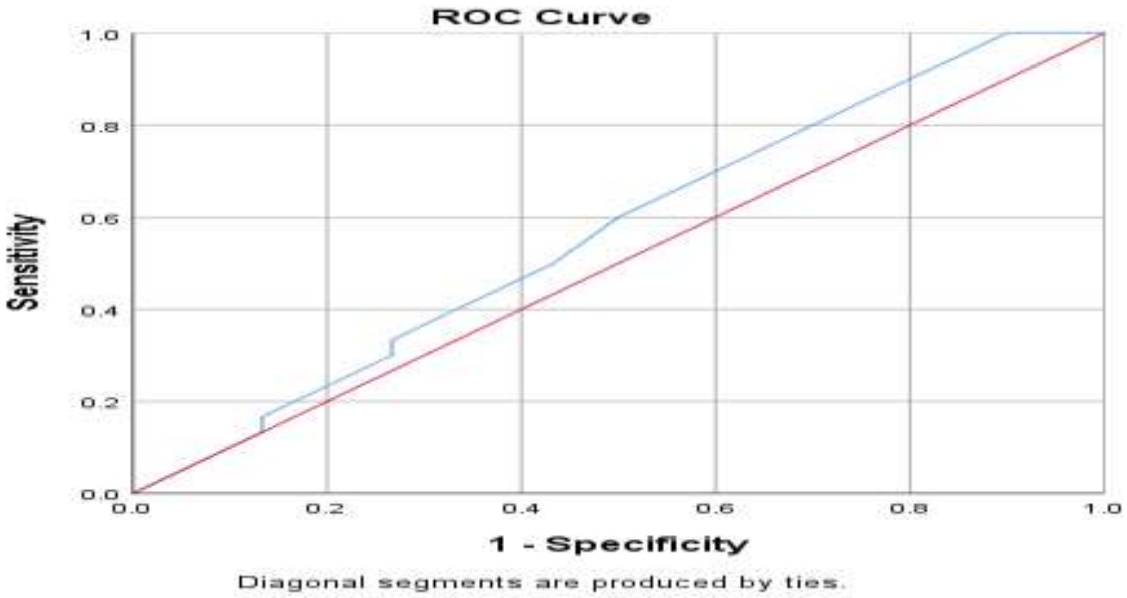

Figure 5. The Receiver Operating Characteristic Curves Showing AUC between Sensitivity and Specificity for Studied Parameter.

4. Discussion

In the present study, serum magnesium and uric acid (UA) levels in patients with type 2 diabetes mellitus (T2DM) was investigated and their relationship with fasting serum glucose (FSG), body mass index (BMI) and some anthropometric and demographic parameters was analyzed. The main findings were a significantly lower serum magnesium level and significantly higher serum uric acid level and FSG level in patients with T2DM than in healthy controls. Furthermore, the levels of uric acid and BMI were inversely correlated with serum magnesium, while the level of the FSG was positively correlated with serum uric acid and BMI [25]. These results suggest that there is a mutual relationship among the magnesium homeostasis, purine metabolism, obesity, and glycemic dysregulation in T2DM.

The mean ages of the patients and the controls did not differ significantly, suggesting that these two groups were not significantly different on the demographic variable of age. Age matching is important because of possible changes in magnesium status, renal uric acid handling, insulin sensitivity and body composition with age. The non-significant correlation of age with either magnesium or uric acid in the current study also shows that

the biochemical differences observed was more closely correlated with the diabetes-related metabolisms rather than age itself [26]. However, other studies have shown an elevation of serum uric acid with age, especially in the elderly male population, that can be attributed to number of factors including reduced renal function, body composition, drug use, and increased metabolic syndrome. This disagreement could be due to the small number of patients, the wide age distribution and the absence of stratification based on sex, renal function, diabetes duration and medication in the current study.

Body weight was significantly greater in the T2DM patients than in the controls, as has been previously reported as an association of excess adiposity with insulin resistance. Adipose tissue dysfunction contributes to the release of free fatty acids and pro-inflammatory mediators, an impairment in insulin receptor signaling and the induction of hepatic glucose production. These mechanisms lead to an increased insulin need and eventually to β-cell exhaustion and chronic hyperglycaemia [27]. The significantly higher BMI of diabetic patients and the acceptable ROC performance is biologically plausible. A similar study has been done in the past, which also found obesity as an important factor associated with poor glycemic control and hypomagnesemia in patients with T2DM. A recent study also indicated that BMI decreased as the serum magnesium concentrations increased, suggesting that there is an inverse association between magnesium status and adiposity.

The lower mean height of the patients should be taken with caution. The difference in adult height reported in the literature after the establishment of T2DM may be due to differences in sex distribution, childhood nutrition, socioeconomic status, ethnicity, or sampling variation over a short period of time. BMI also takes into consideration height, so a slight difference in body weight can make a significant difference in the BMI even if the mean height is lower. Thus, it is not likely that this inverse relationship between height and diabetes status is a clinically useful causal linkage. AUC under 0.50 for height suggests that the patient group was not typical of positive diagnostic performance, but does not represent a negative diagnostic performance [28]. Finding of this magnitude would need adjustment for other anthropometric variables, such as sex, as well as in a multivariable analysis before it could be given independent importance.

As anticipated, FSG was significantly higher in the patients with T2DM than in controls. This difference is the intrinsic metabolic defect of diabetes, which is characterized by insulin resistance and increasing dysfunction of β-cells, leading to the inability to suppress hepatic glucose production and decrease the uptake of glucose into the skeletal muscle and adipose tissue. The wide range of SD values within the patient group implies that there is a high degree of heterogeneity in glycemic control, possibly due to variations in glycemic control over time, compliance, diet, antidiabetic medications, and complications . The ROC analysis gave an AUC of 1.000 for FSG, though this is a very good AUC, it should be interpreted in the light of the selection of participants. When FSG is used to diagnose or classify the study groups, it is considered to induce incorporation bias and predictably overestimate diagnostic performance if it is used as a predictor in ROC analysis. This result, therefore, does not imply that FSG is a newly-identified biomarker but classifies the group.

In the patients with T2DM, the serum magnesium level was significantly decreased which corroborates with high number of studies indicating a high prevalence of hypomagnesemia in diabetes. There has been a global meta-analysis that reported high levels of magnesium deficiency among T2DM patients but the prevalence varied by region, laboratory cut-off, glycemic control, type of diet and co-morbidity. Lecube et al. also observed a significant decrease in magnesium levels in diabetics as compared to non-diabetics and negative correlations with fasting glucose, HbA1c, insulin resistance and BMI. Other researchers have shown that as glycemic control deteriorates and as diseases progress, magnesium deficiency increases [29].

There are several possible mechanisms that might explain the depletion of magnesium in T2DM. Glucose is filtered and leads to osmotic diuresis that may lead to greater urinary loss of magnesium. Insulin resistance can also affect the reabsorption of magnesium in the kidneys and lower insulin activity can cause a disruption in cellular uptake of magnesium. Other factors that decrease magnesium levels are gastrointestinal losses, diuretic use, use of proton-pump inhibitors, gastrointestinal side effects of metformin, and diabetic tubular dysfunction. Magnesium deficiency can then affect insulin receptor phosphorylation, post-receptor signaling, glucose transporter activity and the activity of enzymes in the glucose metabolism. This establishes a vicious cycle whereby diabetes leads to a loss of magnesium and magnesium deficiency causes insulin resistance and metabolic instability [30].

The present study found a negative, but non-significant, correlation between serum magnesium and FSG. This direction is similar to that proposed in relation to the depletion of magnesium and its relation to glycemic control, whereas no statistical significance was found in studies that reported significant inverse relationships between magnesium and fasting glucose or HbA1c . This difference could be due to the small study size, the high range of FSG, the fact that one fasting value was used, and that factors influencing magnesium intake (medications, renal function, dietary magnesium intake and diabetes duration) were not taken into account. A single FSG result might not have been as reliable as HbA1c to reflect chronic glycemic exposure. Magnesium assays also have to be taken into consideration as differences exist between the two. Not all magnesium measured in the serum is available for other uses in the body, and thus can be normal despite depletion from the intracellular compartment [31].

This strong inverse relationship between magnesium and BMI suggests that there may be an association between insulin resistance and magnesium deficiency in obesity. Hypomagnesemia can lead to chronic low-level inflammation, oxidative stress, changes in adipokine levels and decreased insulin signaling. On the other hand, some diets that are linked to obesity may be high in energy density but low in foods rich in magnesium, such as whole grains, legumes, nuts and vegetables. This discovery is in line with the reports which indicated that magnesium levels were lower in the obese and inverse relationship between magnesium and BMI. Others studies however had indicated that diabetes is associated more with hypomagnesemia than obesity or metabolic syndrome without diabetes. These differences could be due to differences in population characteristics, and how renal magnesium wasting, dietary intake, and glycemic control were accounted for.

The serum uric acid level was significantly elevated in patients with T2DM. The results are similar to the epidemiological data indicating that hyperuricemia is linked to metabolic syndrome, insulin resistance and subsequent T2DM [32]. A meta-analysis showed that those with higher serum uric acid were at greater risk for T2DM in the future, but with some level of heterogeneity and residual confounding. Uric acid can also play a role in metabolic dysfunction through contributing to oxidative stress, endothelial dysfunction, inflammation, mitochondrial injury and reduced nitric oxide availability. Such effects may decrease the glucose uptake mediated by insulin, and result in insulin resistance in both liver and peripheral tissues.

The positive association of serum uric acid with FSG is similar to the findings of other studies showing that an increase in the severity of glycemic status is associated with an increase in serum uric acid [33]. The insulin-resistant phase of T2DM is associated with hyperinsulinemia, which can decrease renal urate excretion by stimulating the reabsorption of urate by the tubular system, increasing serum uric acid. High fructose consumption also can cause simultaneous rise of uric acid production, de novo lipogenesis and insulin resistance in the liver. A few studies, however, have claimed an inverse or non-linear relationship between uric acid and glucose with the latter being more severe or poorly controlled. When glycosuria is severe, glucose in the renal tubules may cause

uricosuria, which decreases the serum uric acid. Thus, the relationship can vary with the stage of disease, with an increase in the blood uric acid level associated with insulin resistance and early diabetes, but a decrease in advanced uncontrolled hyperglycemic diabetics due to increase urate excretion in the urine. Some of the results are conflicting and may be due to variations in renal function, medication regimen, sex, diet, and severity of the diabetes.

The positive correlation of uric acid and BMI is also in line with the metabolic function of obesity. An increase in adiposity is associated with higher purine turnover, higher XO activity, higher uric acid production in the liver and insulin-dependent decrease in renal urate clearance. Dietary habits that increase uric acid include obesity, high consumption of fructose-sweetened beverages and purine-rich food. Based on the observed associations between BMI, FSG and uric acid, it can therefore be postulated that elevation in uric acid could represent a component of a larger insulin-resistant metabolic phenotype, and not just a biochemical change [34].

A very interesting finding was the negative correlation of magnesium and uric acid, which was highly significant. This is consistent with an earlier study that found an inverse association between serum Mg and uric acid levels, and proposed that this combination was used to predict someone's metabolic risk [35]. In T2DM similar studies have shown low levels of magnesium and high levels of uric acid, particularly in those with microalbuminuria or poor glycaemic control. Increased oxidative stress, inflammation, endothelial dysfunction and insulin resistance due to magnesium deficiency may be pro-uric acid factors. Concurrently, high levels of uric acid can lead to an impaired endothelial nitric oxide production, damage renal microvasculature, and impair magnesium handling. However, since the cross sectional design says nothing about the effects of magnesium deficiency on uric acid or how uric acid affects magnesium metabolism, or whether both are associated with poor glycemic and renal status, one cannot reach any conclusions about the cause of the elevation of uric acid.

The results of the ROC analysis revealed moderate discriminatory power for BMI and uric acid in the serum between diabetic patients and controls. This confirms their link to T2DM, but will not make them independent tests. Renal function, diet, sex, genes coding for urate transporters, medication, and hydration impact uric acid, and BMI is not able to distinguish between visceral fat and lean mass. Reversing the classification direction, that is diabetic patients having higher serum magnesium, would result in an AUC of about 0.79, which might yield some useful inverse discrimination; the actual values were quite a bit lower than 0.50, however. The reported magnesium confidence interval needs to be verified, however: “0.74–0.337” does not make sense and probably should be taken to mean “0.074–0.337”. Similarly, values < 0.0001 are considered highly significant, and should be reported as $P < 0.0001$, not $P > 0.0001$. For making any claim of clinical diagnosis, cutoff values, sensitivity, specificity, and validation in an independent population are necessary.

5. Conclusion

Together, these results suggest that the decreased magnesium and elevated uric acid levels are associated with T2DM and that these abnormalities are associated with obesity and metabolic dysfunction. Measurement of magnesium and uric acid may be useful for getting additional information about the metabolic status of patients, but neither should be used as a substitute for the use of the basic diagnostic and monitoring tools like fasting glucose, HbA1c, renal function and the evaluation of urinary albumin. Further larger studies should include dietary intake, duration of diabetes, HbA1c, insulin resistance indices, eGFR, albuminuria, diabetes medication, and sex-stratified study. These studies would decide if the magnesium-uric acid connection is obese, glycemic control and renal

function independent and if balancing magnesium deficiency can enhance uric acid metabolism or diabetes-related parameters.

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