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**Original Research Article**

# Evaluation of Serum 1.5-Anhydroglucitol as a Short-Term Glycemic Marker in Iraq Patients with Type 2 Diabetes: A Cass-Control Study

Al-Baker Ali Mukheef[1]*, Prof. Sawsan Hassan Kazem[2]

[1]Department of Pathological Analysis, College of Health and Medical Technology, Al-Furat Al-Awsat Technical University, Kufa, Iraq
[2]Al-Furat Al-Awsat Technical University, Babylon Technical Institute, Babylon, Iraq

***Corresponding Author:** Al-Baker Ali Mukheef
Department of Pathological Analysis, College of Health and Medical Technology, Al-Furat Al-Awsat Technical University, Kufa, Iraq

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**Abstract**: ***Background*:** Diabetes Mellitus type 2 (T2DM) is an epidemic metabolic condition that is typified by persistent hyperglycemia. Although the gold standard in the evaluation of long-term glycemic control is glycated hemoglobin (HbA1c), the polyol 1.5-anhydroglucitol (1.5-AG), a naturally occurring polyol, is reabsorbed in the renal tubules and is competitively inhibited by glucose, thus it may represent early glucose fluctuations. Change in 1.5-AG is a reflection of the variation in blood glucose over one to two weeks. ***Objective*:** The objective of the study was to establish the relationship between serum 1,5-AG and glycemic control variables (HbA1c, FBS) in T2DM patients. ***Materials and Methods*:** The study was a cases control study where 60 healthy controls were used, and 60 T2DM patients were used. 1.5-AG has been demonstrated to indicate change in blood glucose over a period of one to two weeks. An ELISA kit was used to measure serum 1, 5-AG. Fasting glucose, insulin (with a Maglumi device), insulin resistance index (HOMA-IR), renal function test (B.Urea and S.Cr) and GH-900Plus HbA1c Analyzer. ***Results:*** Serum 1.5-AG was also found to be lower in the T2DM patients than the control group (9.31 ± 4.73 mg/L vs. 22.39 + 8.20 mg/L; $p<0.001$). There was a considerable negative correlation between (1.5-AG and FBS, HbA 1c and HOMA-IR). The receiver operating characteristic (ROC) curve analysis indicated the diagnostic accuracy of (1.5-AG on T2DM (AUC = 0.94)) was high and the best cutoff value of 11.95mg/L offered 85.0% sensitivity and 98.3% specificity. ***Conclusion*:** It is possible to consider that 1.5-AG is a good marker to study levels in people with type 2 diabetes during the period of one to two weeks. More research will be required to ascertain the use of these chemicals in diagnosis. The blood glucose variations in the patients with T2DM. Specifically the people who are below 180mg/dl and those above 180mg/dl of blood sugar.

**Keywords:** Diabetes Mellitus, 1.5-AG, Glycemic Control, Hyperglycemia, HbA1c, HOMA-IR.

## INTRODUCTION

Diabetic Mellitus type 2 is chronic disease, where effects are experienced by people over the age of 40 years and in this case, the disease becomes divided into two significant categories: Type 1 which is dominant among people and Type 2 among older people. One of the most common metabolic illnesses is type 2 diabetes mellitus. This can be explained by the fact that it occurs when the two most prevalent causes are used together, which is the reduced level of insulin production of the pancreatic 3-cells, as well as the insensitivity of the cellular tissue in the body to insulin [3]. Most markers are currently employed to detect and measure its progression, and one of them is the glycated hemoglobin (HBA1C) that indicate the degree of saturation of the tissues with sugar within 2-3 months, which is a good standard of Diabetic Mellitus type 2 progression. Conversely, 1,5- anhydro glucitol is also termed as a good index to predict the degree of progression and sequalae of Diabetic Mellitus. between patients whose 1, 5- anhydro glucitol index may be classified in either of the 180 mg/dL or above 180 mg /dl of blood sugar levels. It is an established fact that 1,5 -AG is metabolically inactive, polyol that competes glucose to be reabsorbed in the kidney. Thus, the elevated blood sugar will be reflected by the low levels of 1,5 -AG. In the blood, this index has bioavailability of the same (1 -2 weeks). It is reported that 1,5 -AG is non-specific

and sensitive compared to HBA1c and indicates the levels of blood sugar in patients with D.M.M.but 1,5 -AG is good biomarker that should be considered to control the levels of blood sugar and it is used regularly nowadays.

# MATERIALS AND METHODS

**Ethical Approval and Design of the Study**

This case-control study was done to compare the level of 1.5-AG in the blood of patients with T2DM and a Control group of healthy patients. The IRB of Al-Furat Al-Awsat Technical University, College of Health & Medical Techniques, Kufa, Iraq ethically approved the study procedure (Reference No: 7/37/4466 on date September 10th 2025).

**Recruitment and Selection of Participants**

This was done in 120 patients in total (60 patients with T2DM and 60 healthy control volunteers who were matched by age and sex). The recruitment was done at Al-Qasim General Hospital Babylon Province, Iraq between Augusts and December 2025. Participants T2DM group Subjects were aged above 30 years and had a diagnosis of type 2 diabetes per the ADA diagnostic criteria of T2DM position statement. All participants were excluded on the basis of chronic kidney disease (identified by the level of urea and serum creatinine), other glucose metabolic disorders such as type 1 or gestational diabetes, use of SGLT-2 inhibitors or diuretics, and history of chronic liver or heart diseases.

**Collection and Pre-Treatment of Samples**

The samples were taken in the morning (5 mL) before an 8-h to 12-h overnight fast and in venous blood. 2 aliquots were prepared in all the samples. A sample of the first aliquot (2 to 3 mL) was placed on a gel tube to separate the clot. The samples were clotted at room temperature and centrifuged subsequently (10-20) minutes at (3000-4000) rpm. The serum was decanted and stored at -20 o C to be used later. The second sample (23 mL) was transferred to an EDTA tube in order to estimate glycated hemoglobin (HbA1c).

**Biochemical Analyses**

The level of serum 1.5-AG (BIOLABO system, France), FBG and serum Creatinine were measured using a commercial ELISA Kit (BT LAB, Blocsaszy Technology, China) as per the directions of the manufacturer. This was done through a laboratory procedure, which involved a colorimetric biochemical spectrophotometric analysis of serum lipids concentrations, such as Triglycerides, High-Density Lipoprotein, Total Cholesterol and Low-Density Lipoprotein [NS BIOTEC, Egypt]. HbA1c was measured using a GH-900Plus HbA1c analyzer. Chemiluminescence immunoassay (CLIA) was used to measure serum insulin using a Maglumi instrument (Shenzhen New Factories Biomedical Engineering Co., Ltd. [SNIBE], China).

**Insulin Resistance Indices Calculation**

The Triglyceride-Glucose (TyG) index and the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) were used to measure insulin resistance. The following formulas were used to compute these indices;

$$\text{HOMA-IR} = (\text{Fasting Insulin [}\mu\text{IU/mL]} \times \text{Fasting Glucose [mmol/L]}) / 22.5$$

$$\text{TyG Index} = \ln (\text{Fasting Triglycerides [mg/dL]} \times \text{Fasting Glucose [mg/dL]} / 2)$$

**Statistical Analysis**

The study made use of SPSS 21.0, developed by IBM Corp. and located in Armonk, NY, USA. To display the results, the means were accompanied with the standard deviation. The Kolmogorov-Smirnov test was employed to ascertain if the data followed a regularly distributed distribution. Appropriate statistical methods, such as logistic regression, were used to investigate group differences. Using Pearson's test, we identified the associations between serum 1.5-AG and other biochemical markers. We considered a p-value of less than 0.05 to be statistically significant.

# RESULTS

It was a cross sectional study with 120 subjects, 60 of them were known diabetics with type-2 (patients) and the rest 60 other healthy controls in the age bracket. Table 1 displays the demographics and clinical data from the study's baseline population. There were no significant differences between the mean ages in the T2DM and control group (p=0.130) [Table I] which was (56.00±10.26 years and 53.08±10.79) years, respectively. The percentage of men involved as well as their place of residence were also similar in the two. Nevertheless, the T2DM patients had higher BMI compared to the controls (29.05 ± 6.89 vs. 24.71 ± 3.67 kg/m 2; p = 0.001).

**Table 1: Study Participants' Clinical and Demographic Features**

| Variable | T2DM Group (n=60) | Control Group (n=60) | p-value |
|---|---|---|---|
| **Age (years)** | 56.00 ± 10.26 | 53.08 ± 10.79 | 0.130 |
| **Gender** | | | |
| **Male, n (%)** | 27 (45.0%) | 28 (46.7%) | 0.855 |
| **Female, n (%)** | 33 (55.0%) | 32 (53.3%) | |
| **Residency** | | | |
| **Urban, n (%)** | 44 (73.3%) | 37 (61.7%) | 0.172 |
| **Rural, n (%)** | 16 (26.7%) | 23 (38.3%) | |
| **Disease Duration (years)** | 6.13 ± 0.90 | N/A | Not application |
| **BMI (kg/m²)** | 29.05 ± 6.89 | 24.71 ± 3.67 | **<0.001** |
| **Creatinine (mg/dL)** | 0.57 ± 0.18 | 0.73 ± 0.17 | **<0.001** |
| **Blood Urea (mg/dL)** | 28 ± 4.7 | 27 ± 4.5 | 0.336 |
| **Lipid Profile** | | | |
| **Total Cholesterol (mg/dL)** | 210.31 ± 9.30 | 158.55 ± 5.8 | **<0.001** |
| **Triglycerides (mg/dL)** | 310.65 ± 29.8 | 127.47 ± 11.4 | **<0.001** |
| **LDL-C (mg/dL)** | 166.69 ± 8.9 | 112.57 ± 4.7 | **<0.001** |
| **HDL-C (mg/dL)** | 29.00 ± 1.5 | 40.17 ± 7.60 | **<0.001** |
| **Glycemic Markers** | | | |
| **HbA1c (%)** | 7.84 ± 1.74 | 5.34 ± 0.29 | **<0.001** |
| **Fasting Blood Sugar (mg/dL)** | 132.86 ± 23.9 | 83.66 ± 7.99 | **<0.001** |
| **Insulin (µIU/mL)** | 13.80 ± 3.41 | 6.43 ± 2.11 | **<0.001** |
| **TYG Index** | 8.68 ± 0.63 | 8.34 ± 0.31 | 0.267 |
| **HOMA-IR** | 4.03 ± 2.10 | 1.34 ± 0.59 | **<0.001** |
| **1,5-Anhydroglucitol (mg/L)** | 9.31 ± 4.73 | 22.39 ± 8.20 | **<0.001** |

*Values are shown as mean ± SD or n (%). An independent t-test was used to get the P-values. Values that are bolded indicate the significance of the statistic ($p < 0.05$). N/A stands for not applicable.*

The glycemic parameters were all significantly higher in the T2DM group as expected. On the contrary, the peripheral blood concentrations of 1, 5-AG were significantly lower than those of normal controls in people with type 2 diabetes ($p<0.001$). Figure 1: Serum 1.5-AG levels in various glycemic control (Good<6.5, Fair (6.5-8), and Poor>8) categories by the level of HbA1c. The data has been given as mean ± SD, $p < 0.05$ stating the statistical significance between groups.

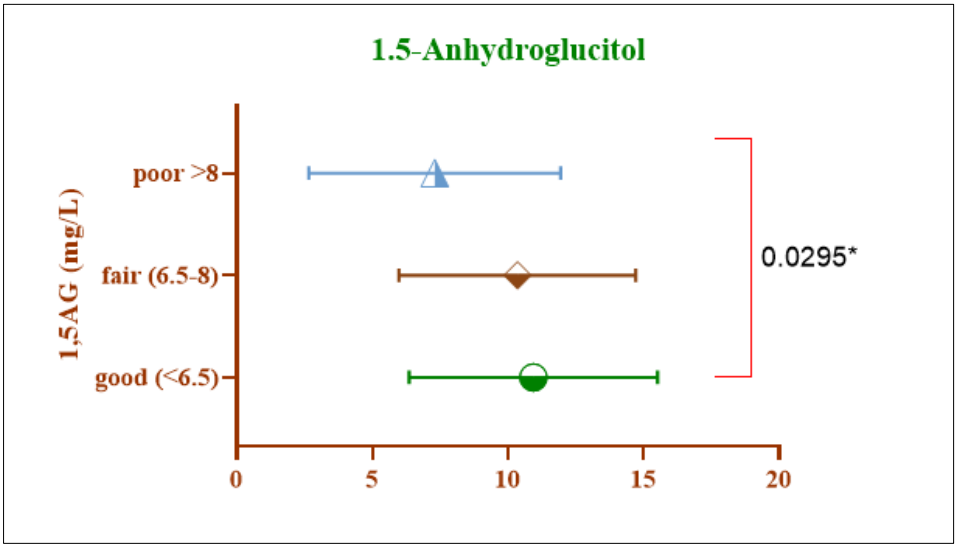


**Figure 1: 1, 5-Anhydroglucitol concentrations in groups of glycemic control in T2DM patients**

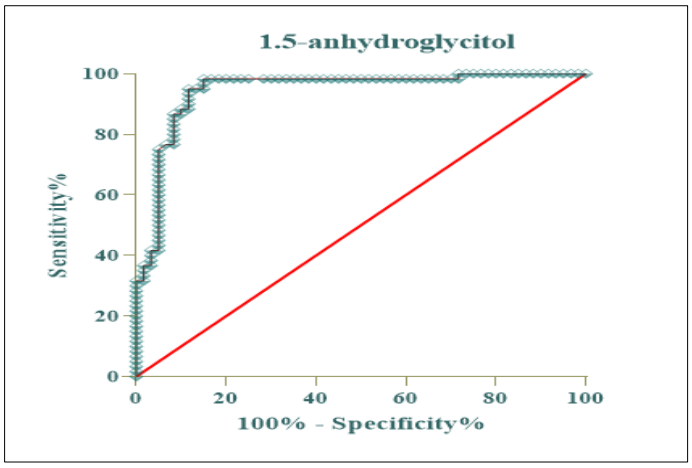


**Figure 2: Analysis of the receiver operator characteristic curve in order to identify one potential diagnostic cutoff value**

The support capacity of blood concentration of 1.5-AG in distinguishing between T2DM patients and healthy-individuals was measured using the ROC curve as illustrated in figure 2. The AUC of this analysis was 0.94 95% CI: 0.88-0.97 that represents a splendid degree of discriminative power in this analysis cohort. Optimal cutoff value of 1.5-AG was determined as 11.95 mg/L that had a sensitivity and specificity of 85.0% and 98.3 respectively. The Youden index which is a single statistic that could be used to compare between statuses of and results of dichotomous or binary diagnostic test (0.833) also indicated the strong reflects short-term glycemic excursion potential of 1.5-AG on T2DM. Table 2 shows the results of the Pearson correlation coefficients between the levels of serum 1.5-AG and other clinical and metabolic parameters in the study group. The subsequent analysis indicates that 1.5-AG correlates negatively with the fasting blood sugar (FBS; r= -0.332, p=0.009), HbA1c (r= -0.271, p=0.03), and triglyceride (TG; r= -0.436, p=0.001) in a statistically significant manner. Moreover, 15-AG has a strong negative correlation with total cholesterol (r =-0.281, p = 0.02) and a moderate negative-correlation with HOMA-IR(r =-0.205, p= 0.11) but it was not found to be significant.

**Table 2: Pearson correlation coefficients of serum 1.5-anhydroglucitol with clinical and metabolic parameters in the study participants.**

| | CH mg/dl | HDL mg/dl | LDL mg/dl | TG mg/dl | FBSmg/dl | Homa IR | HBA1c% | Insulin mIU/L | AGE | Cr mg/dl | urea mg/dl |
|---|---|---|---|---|---|---|---|---|---|---|---|
| HDL mg/dl | 0.947 P<0.001 | 1 | | | | | | | | | |
| LDL mg/dl | 0.911 P<0.001 | 0.841 P<0.001 | 1 | | | | | | | | |
| TG mg/dl | 0.320 P=0.012 | 0.282 P=0.029 | -0.026 P=0.843 | 1 | | | | | | | |
| FBSmg/dl | -0.281 P=0.029 | -0.208 P=0.110 | -0.304 P=0.018 | -0.007 P=0.96 | 1 | | | | | | |
| Homa IR | -0.275 P=0.033 | -0.111 P=0.396 | -0.310 P=0.016 | 0.025 P=0.84 | 0.600 P<0.001 | 1 | | | | | |
| HBA1c% | -0.003 P=0.980 | 0.001 P=0.993 | 0.004 P=0.97 | -0.057 P=0.66 | 0.731 P<0.001 | 0.231 P=0.076 | 1 | | | | |
| Insulin mIU/L | -0.084 P=0.524 | -0.015 P=0.907 | -0.053 P=0.68 | -0.034 P=0.79 | -0.169 P=0.196 | 0.615 P<0.001 | -0.310 P=0.016 | 1 | | | |
| AGE | 0.132 P=0.313 | 0.093 P=0.478 | 0.124 P=0.34 | 0.092 P=0.48 | -0.111 P=0.400 | -0.093 P=0.47 | -0.130 P=0.32 | -0.078 P=0.55 | 1 | | |
| Cr mg/dl | 0.064 P=0.628 | 0.131 P=0.317 | 0.008 P=0.95 | 0.116 P=0.37 | -0.163 P=0.212 | -0.004 P=0.97 | -0.229 P=0.07 | 0.001 P=0.99 | 0.207 P=0.11 | 1 | |
| urea mg/dl | -0.092 P=0.486 | -0.038 P=0.77 | -0.120 P=0.36 | -0.069 P=0.60 | 0.085 P=0.51 | 0.016 P=0.90 | 0.177 P=0.17 | -0.066 P=0.61 | -0.059 P=0.65 | -0.090 P=0.49 | 1 |
| B.M.I | -0.003 P=0.982 | -0.043 P=0.741 | 0.060 P=0.64 | -0.091 P=0.48 | -0.032 P=0.80 | -0.028 P=0.82 | -0.050 P=0.70 | -0.022 P=0.86 | -0.089 P=0.50 | 0.054 P=0.67 | -0.289 P=0.02 |
| 1,5AGmg/L | -0.281 P 0.02 | -0.012 P=0.926 | 0.083 P=0.53 | -0.436 P=0.001 | -0.332 P=0.009 | -0.205 P=0.11 | -0.271 P=0.03 | 0.076 P=0.56 | 0.206 P=0.11 | 0.106 P=0.41 | 0.012 P=0.93 |

Pearson correlation coefficient.

A logistic regression model was built to obtain independent determinants of T2DM. The age and BMI adjusted 1.5-AG, HbA1c and HOMA-IR were all found to be highly significant independent complementary predictors of T2DM status (p less than 0.001). The model was able to show excellent supportive power of 98.3% and cross-validated AUC of 0.996. Table 3 indicates odds ratios (OR) and coefficients.

**Table 3: Multivariate Analysis of Logistic Regression**

| Predictor | Odds Ratio (OR) | 95% CI for OR | p-value |
|---|---|---|---|
| **HBA1c%** | 5.667 | (4.427-7.255) | <0.001 |
| **Homa IR** | 3.805 | (2.997-4.831) | <0.001 |
| **AGE (years)** | 1.053 | (1.021-1.085) | <0.001 |
| **BMI (kg/m²)** | 1.167 | (1.101-1.231) | <0.001 |
| **1.5AG (mg/L)** | 0.246 | (0.191-0.315) | <0.001 |

Bold values denote statistical significance; CI stands for Confidence Interval.

**Table 4: Comparison of Serum 1, 5-Anhydroglucitol (mg/L) according to Fasting Blood Sugar (FBS) threshold (180 mg/dL) in Diabetic Patients**

| | < 180 FBS | >180 FBS |
|---|---|---|
| Number of values | 40 | 20 |
| Minimum | 3.820 | 3.230 |
| 25% Percentile | 7.059 | 4.348 |
| Median | 9.065 | 5.470 |
| 75% Percentile | 11.40 | 7.888 |
| Maximum | 25.67 | 11.95 |
| Mean | 9.937 | 6.160 |
| Std. Deviation | 4.824 | 2.545 |

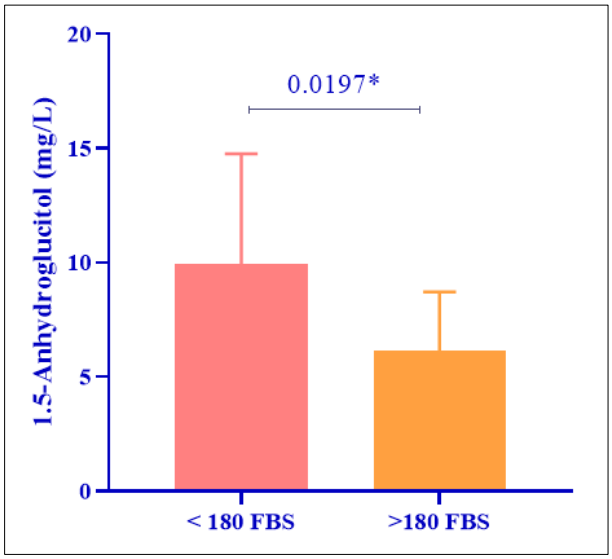


**Figure 3: Serum 1, 5-anhydroglucitol level (mg/L) in diabetic patients when (FBS<180 mg/dL versus FBS >180 mg/dL). Data are presented as Mean ± SD. (Significant at p = 0.0197).**

Discussion There has been a general reporting of ethnic disparities in risks to diabetes [10]. Therefore, the aim of this study was to assess comparatively new marker that has had little coverage in the literature particularly in Iraq and more specifically in Babylon, Evidence indicates that HbA1c test is not the best test to use in monitoring diabetes due to the fact that there has been a poor control of diabetes management and poor glycemic control. Consequently, there is the impending necessity to identify a different short term biomarker such as 1.5-anhydroglucitol to ease the frequent assessment of glycemic excursion in patients with type 2 diabetes mellitus [11]. 1.5-AG is a nonmetabolizable glucose analog, which is chemically stable and attainable in dietary sources [13]. The process of this glucose excretion in the urine is attributed to the inhibition of renal reabsorption of 1.5-AG in the verge of being fully inhibited when the plasma glucose had surpassed the renal load limit (say 180 mg/dL) [14]. Moreover, the T2DM methods were more sensitive and specific when 1.5-AG

was used with FBG [15]. Lower levels of insulin were linked to lower 1.5-AG levels in both prediabetes and T2DM patients with lower 3D-cell mass and lower insulin production. This implies that 1.5-AG might be a noninvasive 8 -cell saving signifier, which is not sufficiently illustrated by conventional glycemic indicators [16]. It turned out that the gender or age could not have an apparent impact on the disease status of the chosen demographic variables as the results showed no statistically significant differences between the diabetic and non-diabetic patients regarding gender and their age at the time of diagnosis [17-19]. Nonetheless, the BMI values were much higher in people with diabetes, as it should be anticipated due to the obvious relationship between obesity and type 2 diabetes mellitus [20, 21]. Furthermore, we noted a tendency that indicated a dyslipidemic trend of diabetics. It negatively correlated the alteration in HDL-C and positively and negatively with the alteration in LDL-C, cholesterol total and triglyceride. Such lipid profile is more in line with the generally accepted changes correlated with IR and IGT/Diabetes [22]. Also, glycemic indices including the levels of insulin in the plasma, HbA1C, and FBS were significantly elevated in diabetics, and the alterations can be mainly attributed to the metabolic defect existing in the disease. Evidence of insulin resistance was clearly seen in diabetic patients as depicted by TyG and HOMA-IR. This observation was also confirmed and established through logistic regression. However, some with high HOMA-IR levels did not read as highly insulin resistant as well as there were no statistically significant relationships that were found between low serum 1.5 AG levels. It is important to note that our findings indicated that there was a significant negative relationship between 1.5-anhydroglucitol and FBS and the 1.5AG expression had been significantly lower in diabetics compared to healthy controls. It is also similar to the findings of other researchers that 1.5-AG, 1.5-anhydroglucitol, and HBA1c are valid measures of short-term and transient hyperglycemia and glucose variability in diabetic patients [23]. In the evaluation of recent and short-term hyperglycemia in diabetic patients, this also confirms the fluctuation of glucose and the possible therapeutic effects of 1.5-AG as a supplementary measure, along with the HbA1c [24]. 1.5-AG together with triglycerides ($r=-0.436$ and $p=0.001$) and 1.5-AG and cholesterol ($p<0.05$). Conversely, the negative correlation indicates that individuals who have higher cholesterol or triglycerides have a higher likelihood of being hyperglycemic thereby reducing the levels of 1.5-AG [25]. In this relationship, it may be demonstrated that the interdependence of lipid and glucose metabolism is a factor in the occurrence of metabolic diseases.

Creatinine and urea were both normal kidney enzymatic functions. As this implies that kidneys has been working well, any alteration in 1.5-AG, to a significant level, is attributed to glucose metabolism and not renal impairment [9]. In this research, it was found that the level of blood sugar and 1.5-Anhydroglucitol have a negative/opposite association, with the levels of the latter being decreasing as the levels of blood sugar exceed the threshold value of sugar in blood (>180mg/dl), this finding corroborates with [26]. In this case, this biomarker plays a crucial role in forecasting the over 180 mg/dl or under 180mg/dl sugar level using the levels of 1.5-AG that are low when the level of blood sugar is above 180mg/dl. Within the framework of the country of Iraq, where the precision of the HbA1c measurements can be distorted by the impact of such conditions as anemia and hemoglobinopathies, 1.5-AG can be viewed as an attractive option of glycemic control monitoring. This potential to offer a quick test of glycemic condition in the short-term may enable clinicians and patients to make more timely therapeutic alteration, which may enhance patient interactions and outcomes as proposed in the introduction of our paper.

**Study Limitations**

Small sample size and single-center methodology that limits the generalizability of the results. No consideration was given to confounding factors like drugs and past medical history. Our results need further prospective, multi-center, large-scaled investigations to verify our reports and establish the cause-effect relationship between type 2 diabetes and 1.5-AG concentrations as a case-control study suggests association and not causality.

# CONCLUSION

Conclusively, the present study confirms the usefulness of serum 1.5-AG as a sensitive reflector of temporary excursions of hyperglycemia in one or two weeks as well as demonstrates a significant reduction in these amounts in T2DM patients. The negative correlation of 1.5-AG with the well-established determinants of metabolic risk including HbA1c, glucose fasting blood cholesterol and triglyceride points has a very strong negative value, which demonstrates their excellent potential as a useful complementary parameter to be used to assess global assessment of recent glycemic control. The lipid parameters associated may indicate an undiscovered role of 1.5-AG in the complex pathophysiology, which links glucose and lipids in diabetic states. Nevertheless, interindividual heterogeneity in the 1.5-AG concentration of diabetic patients implies that other variables that influence the levels of the drug should be explored to be utilized in the future as baseline of pharmaceutical treatment and diagnosis and management of diabetes.

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