Academic Science Journal

Some Biochemical Markers in Type 1 Diabetes Patients

Dhifaf abdul adheem* and **Ekhlas Abdallah Hassan**

Department of Chemistry, College of Science, University of Diyala

* Dhdf5589@gmail.com

Received: 14 February 2024 **Accepted: 30 May 2024**

Published: July 2025

DOI: https://dx.doi.org/10.24237/ASJ.03.03.860B

Abstract

Type 1 diabetes mellitus (T1DM) is characterized by hyperglycaemia in aggregate with biochemical adjustments in glucose, lipid profile and glycated haemoglobin (HbA1c). This examine objectives to assess glucose, lipid profile, glycated haemoglobin (HbA1c), UREA and Creatinine in kind 1 diabetic subjects. Type 1 sufferers have been selected from the subjects attending National Center of Diabetes (AL- Mustansiria University) the variety of patients are 90 the variety of male are 47 the variety of female are 43. Fasting blood sugar (FBS), lipid profile had been determined by means of enzymatic strategies, glycated haemoglobin (HbA1c) became measured through (HPLC), UREA and Creatinine (by kinetic technique) in sufferers had been as compared with healthy controls there wide variety are 30. kind 1 diabetic patients showed statistically good sized boom inside the ranges of HbA1c, urea, creatinine , FBS, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C), whereas statistically good sized decreased degree was determined in triglycerides (TGs) and very low- density lipoprotein(VLDL). In addition, HDL/LDL ratio have been significantly higher than the control subject was.

In conclusion: Most patients with uncontrolled diabetes have dyslipidaemia, and HbA1C used as a dual marker. To control blood sugar levels as well as dyslipidaemia, both of which are major causes of cardiovascular disease. In the advanced stages, urea and creatinine levels rise,

causing kidney failure. Complications of diabetes: Good blood sugar control leads to improved lipid status and less Complications of diabetes.

Keywords: Type 1 diabetes mellitus, biochemical markers, lipid profile, Haemoglobin A1c

Introduction

A metabolic disorder called diabetes is characterized by consistently high blood sugar levels. It may result from either insufficient or insufficient production of insulin. Interestingly, insulin plays a key role in anabolism and influences how proteins, fats, and carbs are metabolised [1]. Insulin resistance is the main source of the metabolic issues associated with diabetes, which mostly impact the skeletal muscles, adipose tissue, and liver. The type and duration of diabetes may have an impact on the severity of symptoms. Dysuria, increased appetite, polydipsia, visual issues, and weight loss can all be brought on by high blood sugar. This is especially true for children and individuals who have no insulin at all. In the early stages of the disease, some people with type 2 diabetes may not exhibit any symptoms at all [2]. Uncontrolled diabetes can cause a number of consequences if left unchecked, including coma, disorientation, and in extreme situations, death from ketoacidosis or nonketotic hyperosmolar syndrome [3]. According to 2014 World Health Organization (WHO) statistics: 8.5% of adults over the age of 18 have diabetes. Diabetes caused 1.5 million deaths in 2019, with 48% of those deaths occurring among people under the age of 70. Moreover, diabetes has been connected to 460,000 fatalities from kidney disease and approximately 20% of deaths from cardiovascular disease, both of which were brought on by high blood glucose levels. Between 2000 and 2019, the conventional death rates associated with diabetes rose by 3%. Lower-middle income countries have seen a 13% increase in mortality due to diabetes. The likelihood that a person between the ages of 30 and 70 will die from any of the four main noncommunicable diseases diabetes, cancer, heart disease, or chronic respiratory disease decreased by 22% globally between 2000 and 2019 [4].

Type 1 diabetes (T1D) is caused by the autoimmune destruction of insulin-producing pancreatic β-cells. The classic trio of T1D symptoms is polydipsia, polyphagia, and polyuria. Most often the disease is diagnosed in children and adolescents, who usually demonstrate the abovementioned combination of symptoms and a marked hyperglycemia that necessitates

lifelong exogenous insulin replacement. The study of T1D pathogenesis was mostly based on two animal models of the disease: the nonobese diabetic mouse and the BioBreeding-diabetes-prone rat, both of which are characterized by progressive T-cell-mediated destruction of β-cells. However, the differences between rodent models and the human situation limited the transferability of the obtained results. In humans, autoantibodies were present in 70–80% of patients at the time of diagnosis. Immunosuppressive and immunointerventive approaches for preventing T1D did not result in preservation of β-cell function or acted only temporally. [1]

Materials and Methods

Ninety people with insulin-treated type 1 diabetes mellitus were chosen from among the students who attended the National Centre of Diabetes (AL-Mustaniania University) between August 2023 and November 2023. The individuals' ranged in age from 18 to 40 years and the average disease duration was 25.36±5.34 years. The full medical history of each patient was obtained, including details about their weight, age, height, marital status, duration of diabetes, and family history of type 1 diabetes mellitus. There were no expectant mothers or those on medication that could change their hormone levels among the patients.

Healthy Subjects

The study's control group consisted of thirty healthy persons, both male and female, ages 18 to 30. The controls had to meet the following medical requirements in order to be included: they could not have diabetes, hypertension, or any recent illnesses.

Sample Collection

After a 12- to 15-hour fast, ten millilitres of blood were withdrawn from the patient and the control subject between hours of 8:00 and 11:00 in the morning. By separating the initial portion of the blood sample into two aliquots and one of them treated with 1.5 mg/ml of ethylene diamine tetra acetic acid (EDTA), the HbA1c can be ascertained in less than three hours. To extract the serum from the second half, the sample placed into a plain tube and allowed to coagulate at 22°C (in the room temperature). After centrifugation, the serum then collected at 3000 rpm. The serum was divided into several 500 µl portions, each of which was frozen at -20° C in Eppendorf tubes until it was required.

Anthropometries Measurements

A straightforward calculation of the Body Mass Index (BMI) involves weight is divided by length. It is believed to be the most reliable indicator of metabolic and cardiovascular disease when compared to BMI.

Measurement of Fasting serum Glucose (F.S.G):-

The glucose measuring equation is based on the Barham and Trindoer (1972)[5] approach, which assumes that glucose oxidase oxidizes glucose enzymatically to gluconate, producing hydrogen peroxide in the process. Subsequently, phenol and peroxide react to generate quinidine, which is measured spectrophotometrically at 505 nm. The information was presented as milligrams/dl.

N.V (Fasting) = 65 - 110 mg/dl = 3.6 - 6.1 mmol/L [6].

Measurement of Glycated Hemoglobin HbA1c %:-

Using the BIO-Rad Variant Hemoglobin A1C software, one can measure their level of glycated hemoglobin.

The Variant Hemoglobin A1C program uses high Performance Liquid Chromatography (HPLC) Based on ion exchange to automatically and precisely separate hemoglobin A1C (HbA1C)[7].

Serum Lipid Profile Assay:-

Measurement of Serum Total Cholesterol (TC):-

An already-made laboratory kit was used to measure total serum cholesterol, with the enzymatic hydrolysis method serving as the basis for the determination.

Reference normal TC value = 140 – 200 mg /dl [8].

Measurement of Triglyceride (T.G):-

Enzymes hydrolysis of triacylglycerol (TAG) to glycerol and fatty acids (FAs).

Reference normal TG value < 150 mg /dl [9].

Measurement of High serum Cholesterol (HDL – C):-

When phos-photungstic acid added to a pH 6.2 solution containing magnesium chloride, chylomicron fraction, low-density lipoprotein cholesterol (LDL), and very low-density lipoprotein cholesterol (VLDL), a quantitative precipitation occurs. The amount of cholesterol

in the HDL fraction detected Dhane of the top after centrifugation measured using a cholesterol kit [10]

Reference normal HDL-C value = 40 – 60 mg /dl [11].

Measurement of Low Density Cholesterol (LDL – C):-

LDL – cholesterol mathematically, the total cholesterol, triglycerides and HDL – cholesterol levels can be calculated using Friedwald's formula:-

$$\text{LDL-C} = \text{TC} - \frac{\text{T.G}}{5} - \text{HDL-C} \quad \textbf{[12]}$$

The formula is only valid at TG concentration of less than (5.32 mmol/L) (400 mg/dL).

Optimal LDL value = 100 –129 mg/dl . [13]

Measurement of Very Low Density cholesterol (VLDL):-

VLDL levels were calculated as one - fifth of the serum TG

$$\text{VLDL mmol / L} = \frac{\text{T.G}}{5} \quad \textbf{[14]}$$

Measurement of Serum urea: The urea concentration in the sample calculation using the following general formula:

$$\text{Bloodurea (mg/dL)} = \left(\frac{\text{A of Sample}}{\text{A of Standard}}\right) \times 50 \text{Concentraton of Standard}$$

Measurement of Serum creatinine: The creatinine concentration in the sample calculated using the following general formula

$$\text{creatininee (mg/dL)} = \left(\frac{\text{A2 - A1 of Sample}}{\text{A2 - A1 of Standard}}\right) \times 2(\text{Concentraton of Standard})$$

P-ISSN: 2958-4612
E-ISSN: 2959-5568

Statistical Methods

Using SPSS version 25, the statistical tool for social sciences, the research was carried out once the data were entered into a computer database structure. This made it possible to confirm the existence of a statistically significant variance in the median difference between more than two groups. BMI, FSG, HbA1c, urea, and lipid profile. The parametric statistical technique used in the analysis of variance to ascertain the significance of the average difference was an independent t-test between the two groups.

Results and Discussion

Results of metabolic factors (FSG and HbA1c) in patients and healthy controls. The study compared the metabolic traits (FSG and HbA1c) of the sick group and the healthy group. The results are shown in Tables (1) and (2), Figures (1) and (2). The mean fasting serum glucose (FSG) for the patient and control groups was 92.68 ± 5.73 mg/dl and 297.85 ± 99.19 mg/dl, respectively. Patients with type 1 diabetes had statistically significant increases in HbA1c and FSG. Results of (Triglycerides, Cholesterol, VLDL, LDL and HDL) in patients and healthy controls.

Using the lipid profile approach, triglycerides, cholesterol, VLDL, LDL, and HDL were compared between the patient and control groups. Figures (1) and (2), as well as Tables (1) and (2), present the findings. The patients' and controls' mean blood triglyceride levels were 138.07±31.985 mg/dl and 145.2±13.2 mg/dl, respectively. In comparison to the controls, the patients' triglyceride levels were lower. The mean blood cholesterol readings for the patient and control groups were 173.36±30.62 mg/dl and 106.49±23.0 mg/dl, respectively. These results suggest that the unwell group's cholesterol is higher. Very-low-density lipoprotein (VLDL) mean blood levels in the patient group were found to be 28.09 ± 5.2 mg/dl, a value that was less than that of the control group (29.14 ± 2.64 mg/dl). The sick group had mean values of 100.18 ± 21.09 mg/dl while the control group had mean values of 35.73 ± 12.14 mg/dl for low-density lipoprotein (LDL). Furthermore, the patients' HDL/LDL ratio and HDL/HDL increase were substantially higher than the control group.

Academic Science Journal

Results of Renal function profile (Urea and Creatinine) in patients and healthy controls.

The results of a comparison of the renal function profiles (creatinine and urea) between the patient group and the control group are shown in Tables (1) and (2) as well as Figures (1) and (2). The control group's average blood urea level was 21.95 ± 3.72 mg/dl, but the patients' was 26.43 ± 6.93 mg/dl. The sick group's mean blood creatinine levels were 0.62 ± 0.15 mg/dl, compared to 0.526 ± 0.151 mg/dl for the control group. Because of their high urea and creatinine levels, the patients were at risk for renal failure. They were also diagnosed with type 1 diabetes.

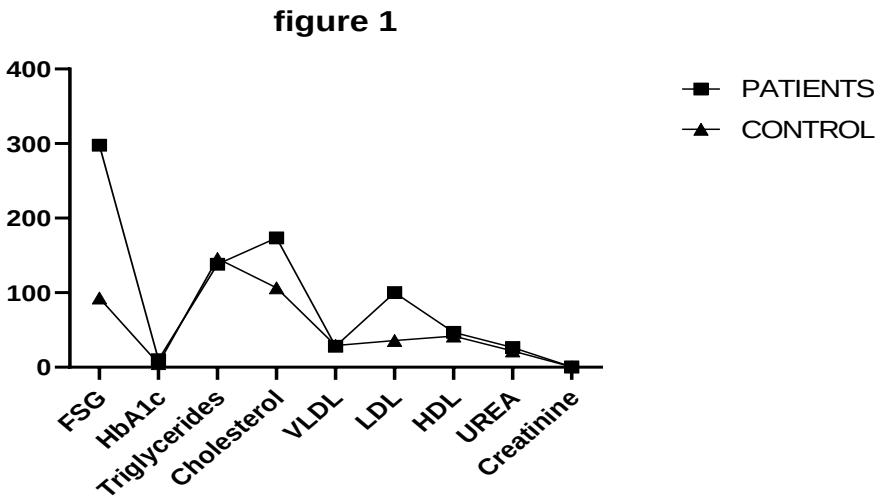

Figure 1: Characteristic Curve Analysis for the Calculation of Biomarkers for Control and Patients

Table 1: The Mean of Biomarkers of Patients and Control Subjects.

Biomarkers	PATIENTS	CONTROL
FSG	297.8539	92.6823
HbA1c	10.04699	4.77
Triglycerides	138.0795455	145.7006667
Cholesterol	173.3863636	106.4907667
VLDL	28.09091	29.14013
LDL	100.1818	35.73663
HDL	46.78409	41.88267
UREA	26.43023	21.956
Creatinine	0.62093023	0.52693333

Academic Science Journal

Table 2: Demographic characteristics of patients and control subjects.

The P value	0.0248
Significantly different (P < 0.05)?	Yes
One- or two-tailed P value	Two-trees
t, duff	t=2.756, df=8
Geometric interval (B / A)	0.6556
SD of log(ratio)	0.1997
SEM of log(mean)	0.06656
95% confidence interval ranged	From 0.4604 to 0.9334
R squared (partial square)	0.4869
Correlation coefficient (R)	0.9698
P value (one tailed)	<0.0001
Are two participants more effective?	Yes

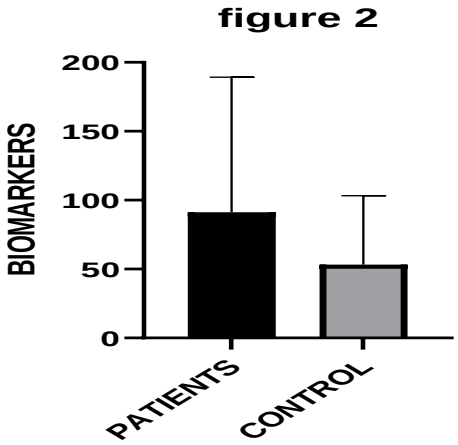

Figure 2: The means of biomarkers of patients and control groups

Conclusion

To define clinically acceptable cut-off values and validate their application in various diabetes patient populations, it is imperative to investigate the long-term associations between these alternative glycaemic markers and the risk of diabetic sequelae.

Source of funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest: The authors declare that there is no conflict of interest.

Ethical clearance: The samples were gained according to Local Research Ethics Committee approval in the College of Science, University of Diyala, and No.EC-3 in 6/8/2023.

References

[1] A. Kaplan, K. A. Glucose, Clin Chem, (The CV Mosby Co. St Louis, Toronto, Princeton, 1984), 436

[2] U. Turpeinen, U. H. Stenman, Analysis of HbA1C and some Hb variants by HPLC, In Analytical and Preparative Separation Methods of Biomacromolecules (CRC Press, 2020), 1–11.

[3] W. Richmond, The development of an enzymic technique for the assay of cholesterol in biological fluids, Clin. Sci. Mol. Med. 46(1), 6P–7P(1974), DOI(https://doi.org/10.1042/cs046006pa)

[4] F. Martinello, E. L. da Silva, Ascorbic acid interference in the measurement of serum biochemical parameters: in vivo and in vitro studies, Clin. Biochem., 39(4), 396–403(2006), DOI(https://doi.org/10.1016/j.clinbiochem.2005.11.011)

[5] K. G. M. Bouafou, B. A. Konan, A. Meite, K.G. Kouame, S. Kati-Coulibally, Substitution de la farine de poisson par la farine d'asticots séchés dans le régime du rat en croissance: risques pathologiques?. Int. J. Biol. Chem. Sci. 5(3), (2011)

[6] M. Choudhary, A. Kochhar, J. Sangha, Hypoglycemic and hypolipidemic effect of *Aloe vera* L. in non-insulin dependent diabetics, J. Food Sci. Technol. 51, 90–96(2014), DOI(https://doi.org/10.1007/s13197-011-0459-0)

[7] E. Voutilainen, E. Hietanen, Isolation and Determination of Lipoproteins, In Regulation of Serum Lipids By Physical Exercise, (CRC Press, 2018), 11–18

[8] S. Tomo, S. Sankanagoudar, R. Shukla, P. Sharma, Validation of a novel method for determination of low-density lipoprotein cholesterol levels in Indian patients with type 2 diabetes, Diabetes Metab. Syndr. Clin. Res. Rev. 16(4), 102448(2022), DOI(https://doi.org/10.1016/j.dsx.2022.102448)

[9] S. G. Fiances, G. G. David, Basic and Clinical Endocrinology: Disorder of lipoprotein catabolism in female infertility, (McGraw-Hill, New York, 20, 2001), 7230–5

[10] A. Poznyak, A.V. Grechko, P. Poggio, V. A. Myasoedova, V. Alfieri, A. N. Orekhov, The diabetes mellitus–atherosclerosis connection: The role of lipid and glucose

metabolism and chronic inflammation, Int. J. Mol. Sci., 21(5), 1835(2020), DOI(https://doi.org/10.3390/ijms21051835)

[11] M.C. Rossi, A. Nicolucci, A. Ozzello, S. Gentile, A. Aglialoro, A. Chiambretti, D. Cucinotta, Impact of severe and symptomatic hypoglycemia on quality of life and fear of hypoglycemia in type 1 and type 2 diabetes, Nutr. Metab. Cardiovasc. Dis., 29(7), 736–743(2019), DOI(https://doi.org/10.1016/j.numecd.2019.04.009)

[12] D. W. E. Fakhir Yousuf, A. Z. Syed, S. Kumar, I. Sharif, A. Ali, Urinary Clinical Manifestation in Type I and II Diabetes; An Observational Study, J. Pharm. Negat. Results, 149–156(2023), DOI(https://doi.org/10.47750/pnr.2023.14.04.20)

[13] GBD 2019 Ageing Collaborators, Global, regional, and national burden of diseases and injuries for adults 70 years and older: systematic analysis for the Global Burden of Disease 2019 Study, BMJ 376 (2022), DOI(https://doi.org/10.1136/bmj-2021-068208)

[14] L. S. Ashoor, T. M. A. Rajab, M. S. Yones, H. Q. Munshid, Visfatin in diabetes mellitus as a key role factor in osteoarthritis, Biochem. Cell. Arch., 22(1), (2022)