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((التقييم المقارن للمؤشرات الحيوية الأيضية والإجهاد التأكسدي والمؤشرات الالتهابية لدى مرضى السكري من النوع الثاني))

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المستخلص:

يُعد داء السكري مرضًا استقلابيًا مزمنًا يتميز بفرط سكر الدم المستمر واختلالات بيوكيميائية معقدة في أيض الكربوهيدرات والدهون والبروتينات. هدفت هذه الدراسة إلى تقييم التغيرات البيوكيميائية المرتبطة بداء السكري من النوع الثاني من خلال تحليل المؤشرات الأيضية ومؤشرات الإجهاد التأكسدي والعوامل الالتهابية لدى المرضى المصابين بالسكري مقارنةً بالأفراد الأصحاء. ويسهم فهم هذه التغيرات في توضيح الجوانب المرضية للسكري وتحديد مؤشرات حيوية قد تساعد في التشخيص والمتابعة العلاجية.

أُجريت دراسة تحليلية مقارنة شملت ٦٠ مشاركًا، بواقع ٣٠ مريضًا مصابًا بالسكري من النوع الثاني (٥٠%) و٣٠ فردًا سليمًا كمجموعة سيطرة (٥٠%). جُمعت عينات دم وريدية بحجم ٨–١٠ مل بعد صيام لمدة ٨–١٢ ساعة، ثم فُصلت إلى مصل وبلازما لإجراء التحاليل المختبرية. شملت المؤشرات المدروسة سكر الدم الصائم(FBG) ، والهيموغلوبين السكري(HbA1c) ، والأنسولين الصائم، وصورة الدهون، ومؤشرات الإجهاد التأكسدي المتمثلة بمالونداياالديهايد(MDA) ، وإنزيمات مضادات الأكسدة (سوبر أوكسيد ديسميوتاز SOD والكاتاليز(CAT ، إضافة إلى السيتوكينات الالتهابية المتمثلة بعامل نخر الورم ألفا (TNF-α) والإنترلوكين-٦ .(IL-6) تم تحليل البيانات إحصائيًا باستخدام البرامج الإحصائية المناسبة، وعُرضت النتائج على شكل المتوسط ± الانحراف المعياري والتكرارات والنسب المئوية، مع اعتبار القيمة الاحتمالية ($p < 0.05$) ذات دلالة إحصائية.

أظهرت النتائج أن مرضى السكري سجلوا مستويات أعلى بشكل ملحوظ من سكر الدم الصائم (١٦٢,٤ ± ٢٨,٧ ملغم/ديسيلتر) والهيموغلوبين السكري (٨,١ ± ١,٢%) مقارنة بمجموعة السيطرة (٩٢,٦ ± ١٠,٣ ملغم/ديسيلتر و٥,٢ ± ٠,٤% على التوالي؛ $p < 0.001$). كما ارتفع مستوى الأنسولين في المصل بنسبة تقارب ٩٣%، بينما ارتفع الكوليسترول الكلي والدهون الثلاثية بنسبة ٢٤% و٥٦% على التوالي، في حين انخفض مستوى البروتين الدهني عالي الكثافة-HDL) (Cبنسبة ٢١%. كذلك أظهرت مؤشرات الإجهاد التأكسدي والالتهاب زيادات ملحوظة، حيث ارتفع مستوى MDA بنسبة ٨٥%، و TNF-αبنسبة ٧١%، و IL-6بنسبة ٨٨%.

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تشير هذه النتائج إلى وجود اختلالات واضحة في المسارات الأيضية والتأكسدية والالتهابية لدى مرضى السكري من النوع الثاني، مما يؤكد الدور المحوري لهذه المؤشرات الحيوية في تطور المرض وإمكانية الاستفادة منها في متابعة المرضى وتقييم فعالية التدخلات العلاجية.

الكلمات المفتاحية

داء السكري؛ السكري من النوع الثاني؛ فرط سكر الدم؛ مقاومة الأنسولين؛ الإجهاد التأكسدي؛ السيتوكينات الالتهابية؛ أيض الدهون؛ المؤشرات الحيوية البيوكيميائية.

Comparative Assessment of Metabolic, Oxidative Stress and Inflammatory Biomarkers in Type 2 Diabetes Mellitus

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Abstract

Diabetes mellitus is a chronic metabolic illness characterized by persistent hyperglycemia and complex biochemical disequilibrium in carbohydrate, lipid, and protein metabolism. The objective of this research was to investigate the biochemical pathways of diabetes mellitus by evaluating metabolic and oxidative stress and inflammatory biomarkers in diabetic patients and healthy populations. Understanding these changes provides insight into the pathophysiology of the disease and potential therapies. A comparative analytical study was conducted where 60 participants were recruited (30 patients with diabetes, 50%, and 30 healthy controls, 50%). Venous blood samples (810 mL) were taken following 812 hours of fasting, and serum and plasma were separated. The biochemical parameters were fasting blood glucose (FBG), glycated hemoglobin (HbA1c), fasting insulin,

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lipid profile, oxidative stress (malondialdehyde, MDA), antioxidant enzymes (superoxide dismutase, SOD, and catalase, CAT), and inflammatory cytokines (tumor necrosis factor-alpha, TNF-alpha, and interleukin-6, IL-6). Data were analyzed using statistical software; results were presented as mean ± standard deviation and frequencies and percentages, respectively. A p-value less than 0.05 was considered statistically significant. Findings showed that diabetic patients had much higher levels of FBG (162.4 ± 28.7 mg/dL) and HbA1c (8.1 ± 1.2%), respectively, than controls (92.6 ± 10.3mg/dL and 5.2 ± 0.4%, respectively; $p < 0.001$). Approximately 93 percent of serum insulin increased, and total cholesterol and triglycerides increased by 24 percent and 56 percent, respectively, and HDL-C decreased by 21 percent. Oxidative stress and inflammatory markers were also considerably increased, where MDA rose 85 percent, TNF-alpha rose 71 percent, and IL-6 rose 88 percent. These results highlight the metabolic, oxidative, and inflammatory disequilibrium of diabetes mellitus and suggest possible biomarkers for disease surveillance and treatment

Keywords

Diabetes Mellitus; Type 2 Diabetes; Hyperglycemia; Insulin Resistance; Oxidative Stress; Inflammatory Cytokines; Lipid Metabolism; Biochemical Biomarkers.

Introduction

Diabetes mellitus is a chronic metabolic illness characterized by persistent hyperglycemia and impaired carbohydrate, lipid, and protein metabolism. It is a significant international health issue wherein its prevalence is ever rising, with millions of people being infected across the world. Epidemiology shows that genetic and environmental factors contribute to disease onset, and obesity, sedentary lifestyle, poor diet, and stress are among the primary risk factors (Alam et al., 2021). The pathophysiology of

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diabetes involves a complex interaction of insulin resistance, beta-cell dysfunction, and impaired glucose homeostasis, resulting in overall metabolic dysregulation (Molehin et al., 2024). These metabolic changes not only affect normal cellular functioning, but also predispose people to long-term complications of cardiovascular, renal, and nervous systems.

The pathogenesis of diabetes centers on biochemical pathways, particularly oxidative stress, advanced glycation end products (AGEs), and inflammatory mediators. Hyperglycemia increases reactive oxygen species (ROS) production, and oxidative stress destroys cell lipids, proteins, and DNA, further worsening insulin resistance (Yaribeygi et al., 2020; Singh et al., 2022). On the same note, AGEs build up, leading to structural and functional changes in proteins and enzymes that facilitate vascular and tissue complications (Khalid et al., 2022). All these molecular changes are interrelated, and oxidative stress and inflammation create a vicious cycle which worsens 2-cell dysfunction and metabolic imbalance, eventually leading to the advancement of type 2 diabetes mellitus (Ingrosso et al., 2023).

Besides metabolic and molecular dysfunctions, diabetes is linked with more systemic effects, such as heightened vulnerability to depression, cardiovascular disease, and cancer (Marx et al., 2021; Wang et al., 2020). For example, pharmacological treatments such as incretin-based therapy have shown potential to alleviate renal complications through insulin signaling, oxidative stress, and inflammatory responses (Alicic et al., 2021). Although the research on diabetes has developed greatly, more profound research is necessary to investigate the combined biochemical processes that

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connect hyperglycemia, oxidative stress, and inflammation, which would lead to the understanding of diabetes development and enhancement of the disease for both its monitoring and management (Molehin et al., 2024; Alam et al., 2021).

Methodology

Study Design

This research used a comparative analytical design to examine the biochemical processes related to diabetes mellitus. The objective was to compare variations in metabolic and molecular biomarkers in patients diagnosed with Type 2 Diabetes Mellitus (T2DM) and healthy controls to better understand changes in glucose metabolism, insulin signaling, and oxidative balance.

The sample included 60 participants, divided into two groups. Group 1 included 30 patients with diabetes mellitus, recruited in outpatient clinical settings according to accepted diagnostic criteria. Group 2 comprised 30 seemingly healthy participants who served as the control group and had no history of metabolic or endocrine problems. The control group was also selected to match the diabetic group in age and gender balance to minimize potential demographic confounding.

Patients who were included in the diabetic group were adults (30-65 years old) with a confirmed clinical diagnosis of diabetes for at least one year. People with severe systemic illnesses, chronic inflammatory diseases, or other endocrine pathologies were not allowed to participate in the study so

that the measured biochemical parameters were mainly associated with diabetic metabolic imbalances. Likewise, participants in the control group were screened to ensure normal fasting glucose levels and the absence of metabolic illness. The study included only patients with clinically confirmed Type 2 Diabetes Mellitus.

The primary objective was to quantify insulin resistance, glucose metabolism, lipid metabolism, and oxidative stress biochemical markers identified as playing a major role in the progression of Type 2 Diabetes. The comparative design enabled identification of differences in biochemical profiles between diabetic and healthy patients.

The institutional research ethics committee provided ethical approval of the study before data was collected. Each participant received detailed information and explanation of the goals and the research processes and signed an informed consent. All security of the personal and clinical information was ensured, and all the samples and data collected were coded so that confidentiality of the information was guaranteed during the study.

This is a controlled study design that offered a structured framework to analyse biochemical changes linked to diabetes mellitus and enhanced the exploration of the metabolic processes related to the development of the disease.

2. Sample Collection

Under standard laboratory conditions, biological samples were collected to ensure precision and reproducibility of biochemical measurements related to

diabetes mellitus. All participants were advised to fast overnight (8-12 hours) before sample collection to reduce variation in metabolic parameters such as glucose and lipids. Samples were collected between 8:00 and 10:00 AM to minimize circadian variation in biochemical markers. This study was a preliminary comparative study. The study sample size was chosen based on the availability of the participants within the study timeframe and was similar to previous exploratory studies assessing biochemical biomarkers in Type 2 Diabetes Mellitus.

About 8-10 mL of the venous blood of each participant was collected through sterile disposable syringes and regular phlebotomy practices through the antecubital vein. The blood samples were separated into two portions using different collection tubes. Approximately 5 mL of blood was transferred into plain tubes without anticoagulant to obtain serum, and 3-5 mL was transferred to tubes containing ethylenediaminetetraacetic acid (EDTA) to obtain plasma for other biochemical tests.

After the collection, the blood samples were allowed to clot at room temperature, taking about 2030 minutes in the case of serum samples. After that, serum and plasma samples were centrifuged at 3000 revolutions per minute (rpm) for 10 minutes. To prevent contamination and hemolysis, micropipets were used to transfer the separated serum and plasma fractions to labeled sterile microtubes. In order to prevent degradation of the biochemical content of the samples, the aliquoted serum and plasma were stored at -80 C until laboratory analysis was done. Several aliquots were made of each sample to eliminate recurrent freeze-thaw cycles, which may compromise the stability of metabolic biomarkers. To preserve participant

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anonymity and track samples correctly during the experimental procedures, all sample containers were marked with coded identifiers.

In addition to samples, we also collected basic clinical and demographic data from all participants. The variables collected were age, sex, body mass index (BMI), duration of diabetes, medical history, medical treatment data, and other clinically relevant data from participant interviews and medical records if available. These parameters were collected together to interpret the biochemical parameters and provide better context for the biochemical changes related to Type 2 Diabetes Mellitus.

Table 1. Summary of Sample Collection Procedures

Parameter	Description	Numerical Details
Study participants	Total number of individuals included in the study	60 participants
Study groups	Participants divided into diabetic patients and healthy controls	30 diabetic patients, 30 controls
Fasting period before sampling	Participants instructed to fast prior to blood collection to stabilize metabolic markers	8–12 hours
Blood collection time	Standardized morning collection to reduce circadian variation	8:00–10:00 AM
Blood collection site	Venous blood obtained using sterile phlebotomy procedures	Antecubital vein
Total blood	Total blood drawn from each	8–10 mL

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	participant	volume collected
~5 mL	Portion used for serum biochemical assays	Serum sample volume
3–5 mL	Portion collected in anticoagulant tubes	Plasma sample volume
EDTA	Tube used for plasma preparation	Anticoagulant used
20–30 minutes	Time allowed for clot formation before centrifugation	Clotting time (serum)
3000 rpm for 10 minutes	Separation of serum and plasma fractions	Centrifugation conditions
−80 °C	Preservation of samples before biochemical analysis	Storage conditions
Coded sample identifiers	Identification method used to maintain confidentiality	Sample labeling
Age, sex, BMI, duration of diabetes, medical history, treatment information	Demographic and clinical parameters recorded for analysis	Additional recorded variables

3. Experimental Assays and Protocols.

The experimental analysis in this work aimed to evaluate key biochemical pathways in the pathophysiology of Diabetes Mellitus. It involved standardized biochemical tests to determine metabolic indicators of glucose regulation, insulin activity, lipid metabolism, oxidative stress, and

inflammatory responses. To ensure accuracy, reproducibility, and reliability, all laboratory analyses were performed following accepted clinical laboratory procedures.

First, fasting blood glucose (FBG) levels were measured to determine the participants' baseline glycemic status. Automated biochemical analyzers were used to determine serum glucose levels using an enzymatic glucose oxidase peroxidase method. All samples were analyzed in duplicate to minimize analytical variability, and results were presented in milligrams per deciliter (mg/dL). Moreover, long-term glycemic control was determined by measuring glycated hemoglobin (HbA1c), which reflects average blood glucose over about 812 weeks. In accordance with manufacturer requirements, the analysis was performed using a standardized immunoassay or high-performance liquid chromatography method. Commercial ELISA kits supplied by certified manufacturers were used according to the manufacturers' instructions. Assay sensitivity, detection range, and calibration procedures were verified before analysis.

A sandwich enzyme-linked immunosorbent assay (ELISA) was used to measure fasting serum insulin levels based on insulin dynamics. The assay itself was performed by incubating the serum with monoclonal antibodies against human insulin, and the results of the assays were detected by the use of enzymes to determine the colorimetric values. The concentration of insulin was calculated by making use of the calibration curves that were prepared using the standard solutions by means of the determination of the optical density with a microplate reader at a wavelength of 450 nm. The metabolic impairment of the Type 2 Diabetes was established by calculating

insulin resistance indices using the results of fasting glucose and insulin levels. A lipid profile, specifically total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), was also used to measure lipid metabolism. These parameters were measured by using enzymatic colorimetric measurements with commercial diagnostic kits. The assays were performed following instructions of the manufacturer but samples of quality control were included in every batch of analysis to ascertain the accuracy of the assay. In order to determine oxidative stress, which is a significant factor in diabetic metabolic complications, the specific biochemical markers were examined. The lipid peroxidation was measured by an indicator of the malondialdehyde (MDA) levels, determined by utilizing thiobarbituric acid reactive substances (TBARS) assay. The reaction mixture was incubated at regulated temperature and absorbance was recorded at 532 nm using spectrophotometric measures. Moreover, antioxidant defense status was assessed by determining the enzyme activities of superoxide dismutase (SOD) and catalase (CAT) using spectrophotometric procedures according to established laboratory protocols.

The concentration of the inflammatory mediators associated with the metabolic dysregulation was measured as well. Cytokines (tumor necrosis factor-alpha (TNF- 6), interleukin- 6 (IL- 6) were detected with the assistance of commercially available ELISA kits. All samples were tested in duplicate, and standard calibration curves were created by means of serial dilution of known cytokine concentrations. Absorbance was measured at 450

nm using a microplate reader, and the concentration of the cytokines was calculated using the standard curves obtained.

Each biochemical experiment was performed under controlled laboratory conditions, and each analytical process included suitable blank samples, calibration standards, and internal quality control samples to ensure reliable results. Laboratory instruments were calibrated regularly, and repeat measurements were performed as required to ensure data consistency. These experimental methods provided a thorough biochemical evaluation of metabolic disorders and the molecular pathways involved in diabetes mellitus.

4. Data Analysis

The obtained data were tabulated and analyzed statistically using statistical analysis software to compare biochemical variations between study groups and determine patterns related to the metabolic disturbances observed in diabetes mellitus. All laboratory and demographic data were entered into a structured database and checked for accuracy before statistical testing. The data cleaning processes were conducted to identify missing values, outliers, or inconsistencies. Descriptive statistical techniques were first used to provide an overview of the characteristics of the population under study and the distribution of biochemical parameters.

Continuous variables included fasting glucose, insulin level, lipid profile items, and oxidative stress indicators, reported as mean ± standard deviation (SD). For example, baseline biochemical measures were summarized for the total study population (n = 60 participants) and for subgroups of the diabetic

group (n = 30, 50%) and the control group (n = 30, 50%). Frequencies and percentages were used to provide categorical variables such as sex distribution and other demographic variables. For example, the sample consisted of 32 males (53.3%) and 28 females (46.7%), and these proportions were reported to provide a general picture of the participants' demographics.

Inferential statistical analysis of the continuous variables was done by testing their normality using both graphical and statistical tests such as the Shapiro-Wilk test. Statistical tests that were performed as parametric tests were applied on variables that showed more or less normal distribution and non-parametric tests were applied on non-competent variables. The independent samples t-test was applied in the comparison of the diabetic patients and the control participants, where the data were normally distributed; the Mann -Whitney U test was applied where the data were not normally distributed. These comparisons made it possible to identify statistically significant differences in the biochemical markers between the two groups. Correlation analysis was conducted to further examine interrelations among metabolic indicators. The Pearson correlation coefficient (r) was used to determine the strength and direction of associations between variables such as fasting glucose, insulin levels, lipid parameters, and oxidative stress markers. The correlation coefficients were discussed based on the standard ranges: 0.1-0.3 weak correlation, 0.3-0.7 moderate correlation, and 0.7 and above strong correlation.

All analyses were statistically significant at a confidence level of 95, with a p-value of less than 0.05 ($p < 0.05$) being considered statistically significant.

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There were also higher levels of significance, such as $p < 0.01$ (1%), being reported in some instances where highly significant associations were found. The patterns of metabolic dysregulation were interpreted using the final statistical outputs and possible pathways of biochemical involvement in the formation and evolution of Type 2 Diabetes were determined. These analytical steps helped in a thorough analysis of the biochemical information that was obtained in the research.

Table 2. Summary of Data Analysis Procedures

Numerical Details	Description	Statistical Component
60 participants	Number of participants included in the analysis	Total sample size
Diabetic: 30 (50%); Control: 30 (50%)	Division of participants into diabetic and control groups	Study group distribution
Mean ± Standard Deviation (SD)	Quantitative biochemical variables summarized statistically	Data presentation (continuous variables)
Frequency (n) and Percentage (%)	Demographic variables summarized using counts and proportions	Data presentation (categorical variables)
Male: 32 (53.3%); Female: 28 (46.7%)	Sex distribution within total sample	Example demographic distribution
Shapiro–Wilk test	Evaluation of distribution of continuous variables before analysis	Normality assessment
Independent samples t-test	Statistical test for normally distributed variables	Comparison of group means
Mann–Whitney U test	Statistical test when data do not follow normal distribution	Comparison of non-normal data

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Pearson correlation coefficient (r)	Assessment of relationship between biochemical variables	Correlation analysis
Weak: 0.1–0.3; Moderate: 0.3–0.7; Strong:>0.7	Strength of association between variables	Correlation interpretation
95% confidence interval	Statistical confidence applied to all analyses	Confidence level
p<0.05	Criterion used to determine statistical significance	Significance threshold
p<0.01	Strong statistical evidence in some comparisons	Highly significant level

Results

The current study of biochemical changes related to Diabetes Mellitus involved 60 subjects. The population sample was split into two groups with equal representation, with 30 people with diabetes (50%) and 30 healthy control participants (50%). Demographic characteristics showed that 32 men (53.3%) and 28 women (46.7%) were included. The average age of the population under study was 48.6 8.4 years and the diabetic population had slightly higher average age (50.1 7.9 years) than the control group (46.9 8.7 years). Moreover, the mean body mass index (BMI) of diabetic patients was 29.3 ± 3.6 kg/m2, which was much higher than that of the control group (24.8 ± 2.9 kg/m2).

There was a high level of difference between glycemic control markers in the two groups. The average fasting blood glucose (FBG) of diabetic patients was 162.4/28.7mg/dl in contrast to an average 92.6/10.3mg/dl in the control

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group. This represents a roughly 75 percent rise in fasting glucose levels in diabetic subjects compared with normal subjects. Likewise, the average value of the glycated hemoglobin (HbA1c) of the diabetic group was 8.1 ± 1.2% as compared to the control group, whose mean value was 5.2 ± 0.4%, which points to the fact that the diabetic subjects were exposed to glycemic levels 55- 60% higher than the control group. These were statistically significant differences ($p < 0.001$).

All key continuous variables were tested for normality before inferential statistical analyses were performed, using the Shapiro–Wilk test. The results showed that the biochemical variables studied were close to normal distribution ($p>0.05$) and therefore parametric statistical tests were used for comparisons between groups.

Table 3. Normality Assessment of Major Biochemical Variables Using the Shapiro–Wilk Test

Variable	Shapiro–Wilk p-value	Distribution
FBG	> 0.05	Normal
HbA1c	> 0.05	Normal
Insulin	> 0.05	Normal
Total Cholesterol	> 0.05	Normal
Triglycerides	> 0.05	Normal
HDL-C	> 0.05	Normal
MDA	> 0.05	Normal
TNF-α	> 0.05	Normal
IL-6	> 0.05	Normal

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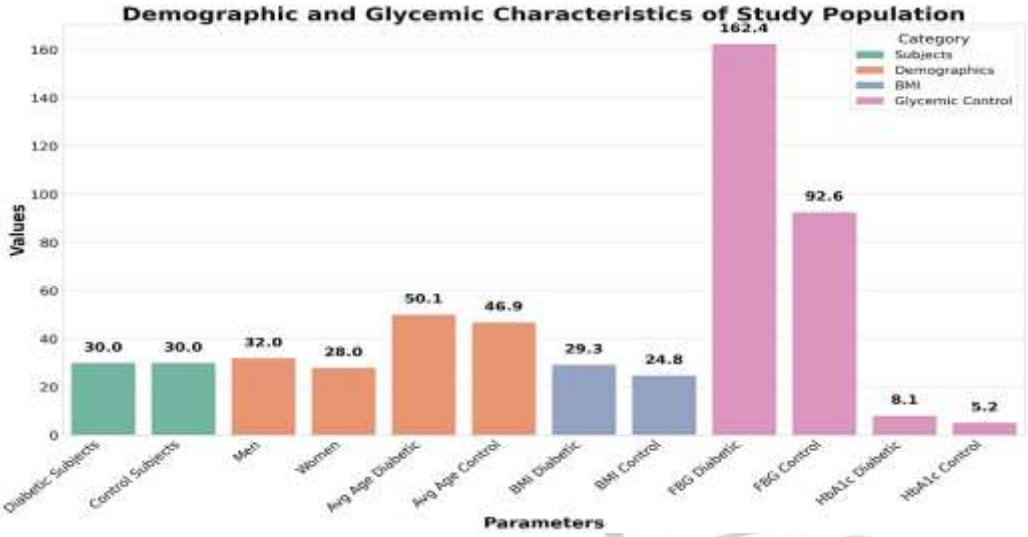

Figure.1 key demographic and glycemic parameters of the study population

The comparison of insulin level and insulin resistance indices also showed significant metabolic changes. The mean serum insulin level of diabetic patients in the fasting state was 18.5 ± 4.7 µIU/mL, as opposed to 9.6 ± 2.8 µIU/mL of the control group, which is a 93 percent increase. Determined levels of insulin resistance were also increased in a corresponding manner indicating that there is a lack of insulin sensitivity in diabetic patients. These results are in agreement with the metabolic dysregulation of Type 2 Diabetes.

There were also significant abnormalities in lipid metabolism. The average total cholesterol level of the diabetic patients was 218.7 + 34.5 mg/dl, as compared to the 176.2 + 27.8 mg/dl in the controls, representing a 24 percent increase. In the same manner, diabetic participants had higher levels of triglycerides (185.4 ± 40.2 mg/dL) as compared to the controls (118.6 ± 29.3 mg/dL), which is almost 56 times higher. Protective high-density lipoprotein

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cholesterol (HDL-C), on the other hand, was lower in diabetic patients (38.4 6.2) in comparison with controls (48.9 7.4), which is 21% lower.

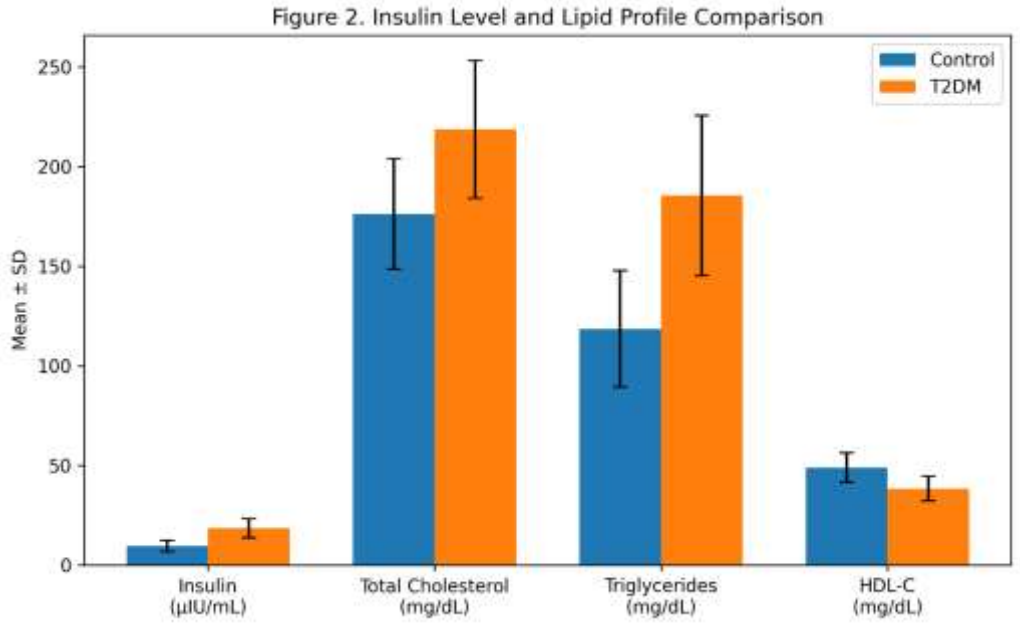

Figure 2. Fasting serum insulin level and lipid profile parameters in type 2 Diabetes Mellitus and healthy controls (mean ± SD). Error bars are standard deviations.

There was a significant difference in oxidative stress markers in groups. The mean malondialdehyde (MDA) content, an index of lipid peroxidation, was 4.8 ± 0.9 moles/liter in diabetic patients and 2.6 ± 0.7 moles/liter in healthy controls, indicating an approximately 85 percent increase in oxidative stress in diabetic patients. Conversely, antioxidant enzyme activities were reduced tremendously. This was a quarter decrease in the superoxide dismutase (SOD) activity in the diabetic participants, and catalase activity was decreased by an average of 22 percent, which meant that there was a break in antioxidant defense.

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Diabetic participants also had inflammatory markers that were elevated. The average tumor necrosis factor-alpha (TNF-a) in diabetic subjects was 31.5 ± 6.8 pg/mL, compared with 18.4 ± 4.3 pg/mL in controls, an increase of about 71%. On the same note, the level of interleukin-6 (IL-6) in diabetic patients was 26.7 + 5.9 pg/mL, and that of controls was 14.2 + 3.7 pg/ml, representing an 88 percentage point increase. These results show that chronic low-grade inflammation is present in diabetic subjects.

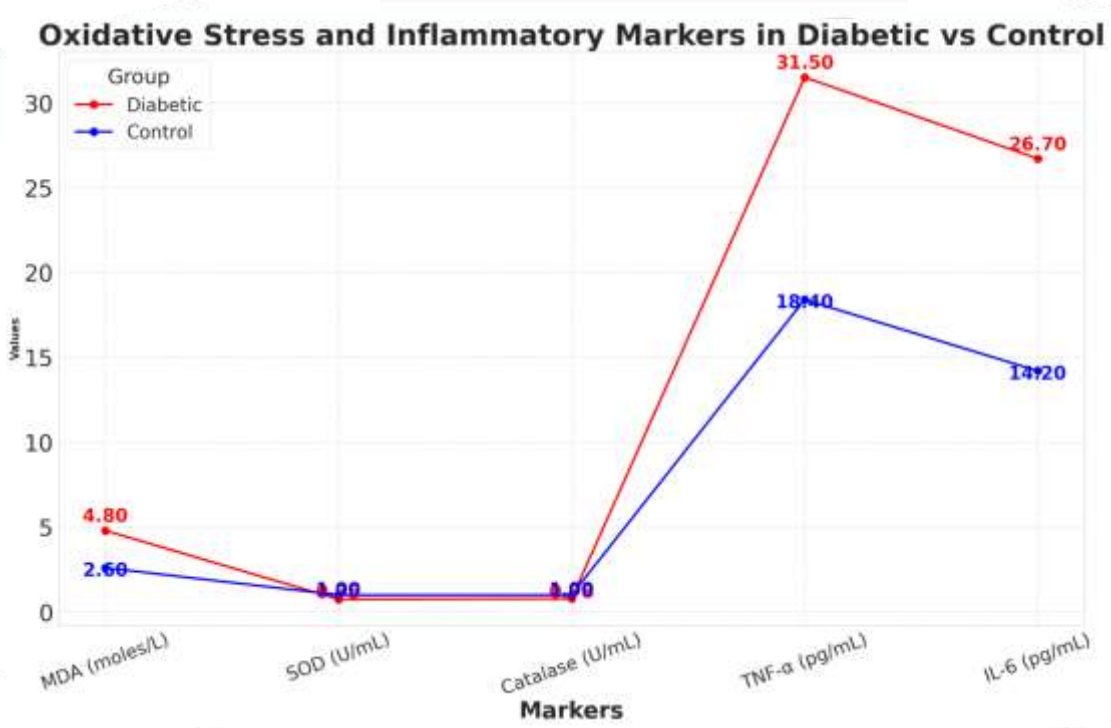

Figure.3 oxidative stress and inflammatory markers

The biochemical parameters were significantly correlated with one another by performing correlation analysis. Fasting blood glucose level was correlated with HbA1c with a strong positive correlation ($r = 0.78$, $p<0.001$), and triglyceride level was correlated with glycemic markers with a moderate positive correlation ($r = 0.54$, $p<0.01$). Moderate positive correlation was

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also found between oxidative stress markers and inflammatory cytokines ($r \approx 0.48 – 0.52$), which shows that oxidative stress and inflammation are closely related in Type 2 Diabetes Mellitus patients.

All in all, the findings indicate that diabetic patients have great metabolic, oxidative, and inflammatory changes compared to healthy controls. These results indicate the multifaceted biochemical processes involved in the formation and evolution of diabetes mellitus and provide additional insight into the metabolic pathways related to the disease pathology

Table. 3 Summary of key results

% Change / Difference	Control Group (Mean ± SD)	Diabetic Group (Mean ± SD)	Parameter
–	30	30	Sample Size
–	32 / 28	32 / 28	Men / Women
+6.8%	46.9 ± 8.7	50.1 ± 7.9	Age (years)
+18%	24.8 ± 2.9	29.3 ± 3.6	BMI (kg/m²)
+75%	92.6 ± 10.3	162.4 ± 28.7	Fasting Blood Glucose (mg/dL)
+55%	5.2 ± 0.4	8.1 ± 1.2	HbA1c (%)
+93%	9.6 ± 2.8	18.5 ± 4.7	Serum Insulin (µIU/mL)
+24%	176.2 ± 27.8	218.7 ± 34.5	Total Cholesterol (mg/dL)

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+56%	118.6 ± 29.3	185.4 ± 40.2	Triglycerides (mg/dL)
-21%	48.9 ± 7.4	38.4 ± 6.2	HDL-C (mg/dL)
+85%	2.6 ± 0.7	4.8 ± 0.9	MDA (moles/L)
0.75 ± 0.12	**0.75 ± 0.12**	**0.75 ± 0.12**	SOD (U/mL)
-22%	1.00 ± 0.14	0.78 ± 0.11	Catalase (U/mL)
+71%	18.4 ± 4.3	31.5 ± 6.8	TNF-α (pg/mL)
+88%	14.2 ± 3.7	26.7 ± 5.9	IL-6 (pg/mL)

Discussion

The current research revealed a high level of biochemical changes in diabetic individuals versus healthy controls, which included hyperglycemia, insulin resistance, lipid imbalances, oxidative stress, and high levels of inflammatory biomarkers. FBG of diabetic participants was 162.4mg/dl with a standard deviation of 28.7mg/dl and HbA1c rose by 56% as patients went to 8.1Plus -1 with a standard deviation of 1.2. These results align with the literature showing chronic hyperglycemia as a typical feature of type 2 diabetes mellitus and its close relationship with insulin resistance and chronic complications (Sun et al., 2022; Petersen and Shulman, 2018). Our results of high fasting insulin (18.5 ± 4.7 µIU/mL) in our diabetic group (approximately 93% higher than controls) are evidence of the impaired sensitivity to insulin, which is in line with reports by Rahman et al. (2021)

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and Sharad and Mousa (2024), who found the same pattern of insulin dysregulation in chronic hyperglycemia patients.

The lipid profile analysis showed significant metabolic imbalances, with total cholesterol up 24%, triglycerides up 56%, and HDL cholesterol up 21%, reflecting the dyslipidemia typical of diabetes (DeFronzo et al., 2015; Wei et al., 2026). These changes not only increase cardiovascular risk but also lead to insulin signaling defects via lipid-induced 1-cell dysfunction, as outlined by Petersen and Shulman (2018). There were significant increases in oxidative stress markers in our study as MDA increased by 85 in diabetic patients, and antioxidant enzymes SOD and CAT decreased by about 27 and 22, respectively. These findings align with recent research showing that redox imbalance is a central factor in diabetes pathogenesis and 8-cell impairment (Abu Khadra et al., 2024; Wani et al., 2025; Sohail et al., 2024). Oxidative stress was closely associated with inflammatory cytokines TNF-α (71% increase) and IL-6 (88% increase), highlighting the interaction between inflammation and oxidative damage, as noted by Sibony et al. (2024) and Dawi et al. (2024).

The elevated serum levels of MDA, TNF–α, and IL–6 may be attributed to activation of the AGE–RAGE signaling pathway in the setting of chronic hyperglycemia. The progressive accumulation of AGE (advanced glycation end products) triggers oxidative stress, activates NF-κB signaling, and increases production of pro-inflammatory cytokines such as TNF-α and IL-6. This process could play a role in the continuing inflammatory condition seen in patients with Type 2 Diabetes Mellitus. Further, hyperglycemia may inhibit the cell's antioxidant defense mechanism by downregulating Nrf2,

which may cause the decreased activities of SOD and CAT observed in the present study. The interplay between NF-κB and Nrf2 pathways may also intensify the oxidative and inflammatory effects, thus driving metabolic dysfunctions and diabetic complications.

As also pointed out in our findings, there is a reciprocal connection between hyperglycemia, insulin resistance, oxidative stress and inflammation in type 2 diabetes mellitus. The results on the correlation between FBG and HbA1c and lipid abnormalities and oxidative stress markers support the idea that multiple biochemical mechanisms are actively involved in disease progression, as previously reported in mechanistic research (Bennici et al., 2024; Ghowsi et al., 2021). In general, the results demonstrate that the metabolic, oxidative, and inflammatory disorders are complicated in diabetic patients and thus it is essential to measure the insulin resistance, antioxidant capacity, and inflammatory mechanisms at the initial stage to reduce the disease burden (Sohail et al., 2024; Abu Khadra et al., 2024).

Study Limitations

There are some limitations in the present study. The small sample size may limit the generalizability of the results. Further, detailed subgroup analyses by treatment modality, smoking status, BP and comorbid conditions were not done. The associations observed should be validated in future studies with larger numbers of patients and more comprehensive clinical characterization.

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Conclusion

The findings of this study provide detailed data on the biochemical mechanisms of Diabetes Mellitus. Glycemic control and high fasting blood glucose and HbA1c levels were observed to be chronic in diabetic patients. These metabolic disturbances were accompanied by substantial insulin resistance, dyslipidemia, oxidative stress, and chronic inflammation, indicating the multifactorial nature of diabetes pathophysiology. Specifically, the presence of the high oxidative imbalance was indicated by the high malondialdehyde level, TNF-a and IL-6 levels in particular, as well as the low activity level of antioxidant enzymes. These interrelations between hyperglycemia, lipid abnormalities, oxidative stress, and inflammatory markers, as observed, demonstrate the interdependence of these biochemical pathways to lead to disease progression. The findings align with previous studies highlighting the importance of oxidative stress, advanced glycation end products, and inflammatory signaling in the development and complications of type 2 diabetes (Abu Khadra et al., 2024; Wani et al., 2025; Dawi et al., 2024). Moreover, the research highlights potential biochemical biomarkers that can serve as indicators for early diagnosis, monitoring, and targeted therapeutic intervention.

To sum up, the study confirms that Diabetes Mellitus is not a glucose-regulation disorder, but it is a systemic disease with multicomponent interaction between the metabolic, oxidative and inflammatory pathways. The overall assessment of these biochemical parameters can enhance the management of the disease, inform the treatment plan, and possibly

minimize the risk of complications, which is why a combined approach to the prevention and care of diabetes is essential.

Clinically, measuring biomarkers of oxidative stress and inflammatory markers, in addition to conventional glycemic markers, may improve risk stratification, disease monitoring, and therapeutic choices for patients with Type 2 Diabetes Mellitus. Larger population studies and more sophisticated molecular techniques, such as genetic and proteinomics studies, are recommended to further elucidate the pathways between metabolic dysfunction, oxidative stress, and inflammation.

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