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ORIGINAL ARTICLE

Evaluation of Inflammatory Markers (CRP, IL-6, TNF-α) in Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a major global health concern characterized not only by chronic hyperglycemia but also by a state of persistent low-grade systemic inflammation. Inflammatory biomarkers such as C-reactive protein (CRP), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-α) are increasingly recognized for their role in insulin resistance, endothelial dysfunction, and the development of diabetic complications.

Objective: To evaluate the levels of inflammatory markers (CRP, IL-6, TNF-α) in patients with T2DM and to determine their association with glycemic control and clinical parameters.

Methods: This cross-sectional study was conducted at the University of Lahore Teaching Hospital from June 2022 to September 2023. A total of 110 participants were enrolled, including 55 T2DM patients and 55 healthy controls. Fasting blood samples were collected to measure CRP, IL-6, TNF-α, fasting blood glucose, and HbA1c. Data were analyzed using SPSS version 26. Independent t-tests and Pearson correlation analysis were applied, with $p<0.05$ considered statistically significant.

Results: T2DM patients exhibited significantly higher levels of CRP (7.18 ± 2.34 mg/L), IL-6 (10.24 ± 3.66 pg/mL), and TNF-α (11.88 ± 4.12 pg/mL) compared to controls ($p<0.001$). HbA1c showed a strong positive correlation with CRP ($r=0.64$), IL-6 ($r=0.59$), and TNF-α ($r=0.56$). Patients with poor glycemic control had significantly higher inflammatory marker levels than those with better control.

Conclusion: Inflammatory markers are significantly elevated in T2DM and are closely associated with poor glycemic control. These biomarkers may serve as useful indicators of disease severity and potential predictors of complications.

Keywords: Type 2 Diabetes Mellitus, CRP, IL-6, TNF-α, Inflammation, HbA1c, Biomarkers, Insulin Resistance.

INTRODUCTION

The Type 2 Diabetes Mellitus (T2DM) has become one of the fastest growing chronic non-communicable diseases in the globe and has become a significant public health issue, especially in the low and middle-income nations[1]. The International Diabetes Federation estimates that there were almost 589 million adults with diabetes living around the world in 2024, over 90 percent of which were caused by Type 2 Diabetes Mellitus[2]. The pressure is likely to increase significantly in the next decades because of the growing rates of obesity, sedentary living, urbanization, poor dietary habits, and ageing of the population[3]. Such an increase is particularly worrisome due to the fact that T2DM is not merely linked to chronic hyperglycemia but also to severe microvascular and macrovascular complications in the long term, such as nephropathy, neuropathy, retinopathy, ischemic heart disease, and stroke[4].

Conventionally, T2DM was viewed as a metabolic disease mainly due to insulin resistance and metabolic dysfunction in a progressive manner in the β-cell of the pancreas[5]. Nonetheless, present-day evidence has certainly shown that the disease is also closely associated with state of chronic low-grade systemic inflammation. This inflammatory background has now been considered to be a key biological process that causes the onset, development, and complications of T2DM[6]. Excess adipose tissue in metabolically unhealthy people is not just a storage organ of energy, but an active endocrine and immunological tissue, capable of releasing a multitude of pro-inflammatory cytokines, chemokines and acute-phase reactants. These inflammatory mediators disrupt insulin signaling pathways, aggravate glucose metabolism, suppress endothelial functioning, and encourage vascular damage[7].

C-reactive protein (CRP), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-α) among the inflammatory biomarkers have been particularly studied in relation to T2DM[8]. CRP is an acute-phase protein that is produced by the liver in reaction to cytokines of inflammation, especially IL-6 and is commonly used as a marker of systemic inflammation. High CRP levels have been consistently linked to insulin resistance, obesity, endothelial dysfunction and future diabetes[9]. The IL-6 is a versatile cytokine released by adipocytes, macrophages, endothelial cells and immune cells and has a great role in the production of glucose in hepatic, inflammatory signaling, and the stimulation of acute-phase proteins. TNF-α is a second potent pro-inflammatory cytokine which has direct effects on insulin resistance via inhibition of insulin receptor signaling, glucose uptake and promotion of lipolysis and oxidative stress[10]. A combination of these indicators indicates the inflammatory load underlying the metabolic abnormalities of T2DM[11].

This interaction between inflammation and diabetes is of clinical significance since it is believed that the process of inflammatory activation starts well before manifestations of the overt complications[12]. Even relatively early in the disease, persistent low-grade inflammation can hasten the process of β-cell exhaustion, augment glycemic fluctuations, and raise the danger of cardiovascular and metabolic issues even in the disease[13]. Thus, the detection of inflammatory abnormalities in diabetic patients can not only enhance the knowledge of the disease mechanisms but can also play a role in the early stratification of risks and the possibility of prognosis and the creation of specific treatment measures[14]. It is especially applicable in places such as Pakistan where the number of diabetics is increasing at an alarming rate and where most of the patients report late with inadequately managed disease and numerous metabolic comorbidities[15].

Although there is growing awareness regarding inflammation as a main cause of T2DM, local information assessing the use of inflammatory markers in diabetic patients is still scarce[16]. Inflammatory status is still not given much attention as most clinical practice is still more concerned with glycemic indices like fasting blood glucose and HbA1c. The evaluation of CRP, IL-6, and TNF-α inflammatory biomarkers in T2DM patients could offer a better insight into the disease severity and metabolic dysregulation[17]. This information can also be specifically applicable to high-risk people whose closer observation and more detailed metabolic care may be of benefit[18].

Thus, the given work was done to examine the serum levels of CRP, IL-6 and TNF-alpha in Type 2 Diabetes Mellitus patients and compare them with healthy controls[19]. Moreover, the research sought to determine the association of these inflammatory molecules with glycemic condition and chosen clinical features and therefore the possible use of these as biochemical surrogates of inflammatory load in T2DM[20].

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MATERIALS AND METHODS

The analytical study was a cross-sectional study that was carried out in the University of Lahore Teaching Hospital, Lahore, Pakistan, between the months of June 2022 and September 2023. The research was aimed at measuring the concentration of the chosen inflammatory markers of patients with Type 2 Diabetes Mellitus (T2DM) and their comparison with healthy non-diabetic people. The goal was to identify the presence of systemic inflammatory activity (measured by particular biochemical indicators) that had a significant difference in diabetic patients and the association of biochemical indicators with glycemic control and other specific clinical features.

A non-probability consecutive sampling method was used to enroll 110 participants in the study. Two groups were used as the study population. The former was a population of 55 previously diagnosed Type 2 Diabetes Mellitus patients and the latter a population of 55 seemingly healthy age- and gender-matched controls having no known history of diabetes or chronic inflammatory disease. The sample group was identified based on the number of qualified individuals at the time of the study and the possibility of carrying out the necessary biochemical and immunological research in the laboratory of the institution.

The diabetic group participants were eligible in case they were aged between 30 and 65 years old, had a confirmed diagnosis of Type 2 Diabetes Mellitus, at least one year, and had given written informed consent to take part in the study. The healthy control group was within the same age bracket and had never been diagnosed with diabetes mellitus, neither had acute nor chronic systemic inflammatory disease, and nor had any history suggestive of major metabolic or immunological disorders. Trying to make both groups similar regarding age and sex distribution in an attempt to minimise the confounding variation.

Patients were not eligible who were known to have Type 1 Diabetes Mellitus, gestational diabetes, acute infections, fever, autoimmune diseases, chronic inflammatory diseases, malignancy, chronic liver disease, chronic kidney disease, recent trauma or surgery, and corticosteroids, immunosuppressive therapy, or anti-inflammatory drugs. These exclusion criteria were used in order to reduce the effect of other clinical conditions that may interfere with the interpretation of biochemical results by acting independently to change the levels of inflammatory markers.

Following the written informed consent, a comprehensive clinical evaluation was conducted on all the participants. A structured proforma that was intended to be used in the study was used to collect information. The demographic and clinical information were based on age, gender, years of having diabetes, history of current treatment, body weight, height, and body mass index, with comorbid conditions as needed. The standard procedures were used to record anthropometric measures. A calibrated digital weighing scale was used to determine body weight in kilograms and a stadiometer to determine height in meters. The Body Mass Index (BMI) was determined as a ratio of the weight in kilograms to the square of the height in meters.

To examine in laboratories, each individual had his or her venous fasting blood of about 5 mL collected under aseptic conditions following an 8-10 hour fast. The samples of blood were collected and examined in the hospital clinical laboratory under controlled conditions. The estimation of fasting blood glucose (FBG), glycated hemoglobin (HbA1c), and the chosen inflammatory markers, such as C-reactive protein (CRP), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-alpha), were done with the obtained samples.

Blood glucose levels were determined by glucose oxidase-peroxidase method and HbA1c was done via a standardized automated laboratory analyzer. The estimation of serum CRP was done through immunoturbidimetric assay, and the serum IL-6 and TNF-alpha concentrations through commercially available enzyme-linked immunosorbent assay (ELISA) kit, as per the instructions provided by the manufacturers. All the assays were carried out using the laboratory quality control procedures so that the results obtained were uniform and dependable.

To analyze it, the poor glycemic control was set at 7.0% and above HbA1c and good glycemic control was set at below 7.0. Comparison of the serum levels of inflammatory markers between the patients with Type 2 Diabetes Mellitus and healthy controls was the main study outcome. Secondary outcomes were the evaluation of the correlation between inflammatory biomarkers and HbA1c, Body Mass Index, and length of diabetes, to investigate whether the deteriorating metabolic control correlated with the growing systemic inflammation.

The research was done following the ethical review committee of the University of Lahore Teaching Hospital in which ethical approval was obtained. Before they were enrolled, all the participants were informed of the nature and purpose of the study. All the subjects gave informed consent in writing, and their participation was voluntary. The privacy of personal and clinical data was assured during the research process, and all operations were conducted following the established standards of research in relation to human subjects.

Statistical Package of Social Sciences (SPSS) version 26.0 was used to enter and analyze all the data collected. Quantitative variables, which included age, BMI, fasting blood glucose, HbA1c, CRP, IL-6 and TNF- alpha were given out in mean ± standard deviation, whereas categorical variables including gender were given out as frequencies and percentages. Comparison of the mean values between diabetic patients and controls was done using an independent sample t-test. Where necessary, Chi-square test was used to compare categorical variables. The Pearson correlation analysis was done to establish the relationship between inflammatory markers and HbA1c, BMI, and length of diabetes. A p-value below 0.05 was taken to be statistically significant during the study.

RESULTS

The total number of participants involved in the study was 110: 55 patients with Type 2 Diabetes Mellitus (T2DM) and 55 seemingly healthy controls. The baseline clinical and demographic data of the study participants are given in Table 1. Average age of patients in diabetic cohort was 53.18 years of age and average age of control cohort was 51.74 years of age and the difference between the two groups did not significantly differ ($p = 0.361$). On the same note, there was no significant difference between the gender distribution in the diabetic and the control groups with 31 (56.4) and 24 (43.6) males and females, respectively ($p = 0.699$). But the mean Body Mass Index (BMI) of the patients with T2DM (29.42 ± 3.51 kg/m 2) was much greater compared to the controls (24.96 ± 2.87 kg/m 2), which suggests that the adiposity burden in diabetic patients was much greater ($p < 0.001$) (Table 1).

As presented in Table 2, the biochemical comparisons between diabetic patients and controls demonstrate highly statistically significant differences in the state of glycemic control and inflammatory markers. The mean fasting blood glucose level was significantly higher in the diabetic group (171.36 ± 34.28 mg/dL) than in the control group (91.45 ± 10.72 mg/dL, $p < 0.001$). The mean HbA1c concentration was also significantly higher in diabetic patients (8.61 ± 1.47%) than in controls (5.21 ± 0.52%) ($p < 0.001$), suggesting poor glycemic control in the diabetic group.

As for the inflammatory markers, the diabetic group had significantly higher serum concentrations of the three markers examined. The mean CRP concentration in the group of T2DM patients was 7.18 ± 2.34 mg/L but markedly lower in the control groups (2.41 ± 1.08 mg/L, $p < 0.001$). Similarly, the mean IL-6 level was markedly elevated in diabetics (10.24 ± 3.66 pg/mL) compared with the control group (3.92 ± 1.47 pg/mL) ($p < 0.001$). The same pattern was noted for TNF-α, with a mean value of 11.88 ± 4.12 pg/mL among diabetics, and 4.83 ± 1.91 pg/mL in the controls ($p < 0.001$). This clearly demonstrates that the inflammatory status of patients with Type 2 Diabetes Mellitus was significantly increased compared to healthy controls (Table 2). This general trend is in

agreement with past observational and review studies that have reported an association between T2DM and increased CRP, IL-6 and TNF-α concentrations.

We conducted additional analysis to determine if the level of inflammatory markers was related to glycemic control in patients with diabetes. Pearson correlation analysis revealed a moderate to high positive relationship between HbA1c and inflammatory markers investigated (Table 3). The correlation between HbA1c and CRP was the strongest ($r = 0.64$, $p < 0.001$), suggesting that as HbA1c increased, the systemic inflammatory load also increased. Similarly, IL-6 was positively correlated with HbA1c ($r = 0.59$, $p < 0.001$), and TNF-α was also found to have a positive correlation ($r = 0.56$, $p < 0.001$).

BMI also demonstrated a positive correlation with inflammatory markers. CRP showed a positive association with BMI ($r = 0.48$, $p < 0.001$), that obesity and adiposity may lead to low-grade inflammation in diabetics. Similarly, there were positive correlations between BMI and IL-6 ($r = 0.42$, $p = 0.002$) and between BMI and TNF-α ($r = 0.39$, $p = 0.004$). We also found a weak but significant association between duration of diabetes and inflammatory biomarker levels, which suggests that duration of diabetes may play a role in modulating the inflammatory response (Table 3). This association is consistent with published reports that demonstrate inflammatory burden is associated with adiposity and decreasing metabolic control in patients with T2DM.

In addition, to better assess the influence of glycemic control on inflammatory status, diabetic subjects were also divided into two groups according to their HbA1c level: well-controlled diabetics (HbA1c <7.0%) and poorly controlled diabetics (HbA1c ≥7.0%). Of the 55 diabetics, 14 patients (25.5%) were well-controlled and 41 patients (74.5%) were poorly controlled. There were significant increases in the levels of inflammatory markers in poorly controlled diabetics over those with better glycemic control. Diabetic patients with poor glycemic control had a mean CRP level of 7.84 ± 2.18 mg/L, compared to 5.24 ± 1.63 mg/L in the better-controlled diabetics ($p < 0.001$). IL-6 levels were also significantly greater in poorly controlled patients (10.98 ± 3.44 pg/mL) than in better-controlled diabetics (7.89 ± 2.15 pg/mL) ($p = 0.002$), and TNF-α levels were also significantly higher (12.67 ± 4.06 pg

These results suggest that the inflammatory state in T2DM is not only more severe than in non-diabetics but also worsens with poor glycemic control. This may imply that suboptimal control of diabetes may not only lead to elevated systemic inflammatory status, but may also promote diabetes complications (Table 4). Recent reviews also support the notion that inflammatory dysregulation is physiologically linked to hyperglycemia and diabetic complications.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Variable	T2DM Patients (n = 55)	Controls (n = 55)	p-value
Age (years)	53.18 ± 8.41	51.74 ± 7.96	0.361
Male, n (%)	31 (56.4%)	29 (52.7%)	0.699
Female, n (%)	24 (43.6%)	26 (47.3%)	0.699
BMI (kg/m²)	29.42 ± 3.51	24.96 ± 2.87	<0.001
Fasting Blood Glucose (mg/dL)	171.36 ± 34.28	91.45 ± 10.72	<0.001
HbA1c (%)	8.61 ± 1.47	5.21 ± 0.52	<0.001

Table 2: Comparison of Glycemic and Inflammatory Markers Between T2DM Patients and Controls

Variable	T2DM Patients (n = 55)	Controls (n = 55)	p-value
Fasting Blood Glucose (mg/dL)	171.36 ± 34.28	91.45 ± 10.72	<0.001
HbA1c (%)	8.61 ± 1.47	5.21 ± 0.52	<0.001
CRP (mg/L)	7.18 ± 2.34	2.41 ± 1.08	<0.001
IL-6 (pg/mL)	10.24 ± 3.66	3.92 ± 1.47	<0.001
TNF-α (pg/mL)	11.88 ± 4.12	4.83 ± 1.91	<0.001

Table 3: Correlation of Inflammatory Markers with HbA1c, BMI, and Duration of Diabetes in T2DM Patients

Variable	CRP (r)	p-value	IL-6 (r)	p-value	TNF-α (r)	p-value
HbA1c (%)	0.64	<0.001	0.59	<0.001	0.56	<0.001
BMI (kg/m²)	0.48	<0.001	0.42	0.002	0.39	0.004
Duration of Diabetes (years)	0.33	0.014	0.29	0.029	0.31	0.021

Table 4: Comparison of Inflammatory Marker Levels According to Glycemic Control in T2DM Patients

Variable	Good Glycemic Control (HbA1c <7.0%) (n = 14)	Poor Glycemic Control (HbA1c ≥7.0%) (n = 41)	p-value
CRP (mg/L)	5.24 ± 1.63	7.84 ± 2.18	<0.001
IL-6 (pg/mL)	7.89 ± 2.15	10.98 ± 3.44	0.002
TNF-α (pg/mL)	9.56 ± 2.72	12.67 ± 4.06	0.006

DISCUSSION

The current cross-sectional research was designed to assess the magnitude of CRP, IL-6, and TNF-α levels in patients with Type 2 Diabetes Mellitus (T2DM) and to find out their connection to the glycemic condition and the chosen clinical features[1]. The study results revealed that diabetic individuals were considerably more likely to have high levels of all the three inflammatory markers than the healthy patients. Moreover, the levels of inflammatory markers were found to have a positive correlation with the HbA1c, Body Mass Index (BMI) and the length of diabetes, indicating that the systemic inflammation is increased with the aggravation of metabolic dysregulation and with the chronicity of the illness[2].

The Type 2 Diabetes Mellitus is now being regarded as not only a disease of glucose metabolism, but as a persistent inflammatory-metabolic disease[3]. In the modern world, recent statistics indicate that the burden of diabetes is increasing at a fast rate, especially in low- and middle-income environments, which further predetermines the fact that the inflammatory aspect of T2DM is even more clinically important to understand. There were about 589 million adults with diabetes in the world in 2024, and over 90% of them had Type 2 Diabetes Mellitus, which highlights the magnitude of the issue and the necessity to find other mechanisms of diabetes other than hyperglycemia[4].

Among the significant findings of this study was the fact that, Body Mass Index of diabetic patients was significantly higher as compared to that of the controls[5]. This observation is clinically relevant as the excess adiposity, particularly the visceral fat mass, is tightly linked with the chronic low-grade inflammation. The adipose tissue of obese and insulin-resistant patients is an active endocrine gland that releases a number of pro-inflammatory mediators such as IL-6 and TNF-α. These inflammatory molecules disrupt the insulin receptor signalling pathways and play a role in progressive insulin resistance, endothelial dysfunction and glucose inability to be utilized[6]. Thus, it is possible that the high BMI in the

diabetic population in the current study was a significant contributor of the inflammatory burden in the patients[7].

This study found that the serum CRP was significantly high among diabetic patients than among controls[8]. CRP is an acute-phase reactant that is commonly used and a recognized measure of systemic inflammation. High levels of CRP among T2DM patients indicate that a state of inflammation exists, and indicates that chronic metabolic stress in diabetes prompts inflammatory signaling pathways, which are beyond glucose dysregulation. The large increase in CRP in the diabetic group of the study is in line with the pathophysiological principle that the hepatic acute phase reactions are stimulated in the patients with insulin resistance and persistent hyperglycemia[9]. It has clinical significance as several studies have also linked CRP with the occurrence of endothelial dysfunction, cardiovascular risk, and advancement of diabetic complications. Our results of a high positive relationship between CRP and HbA1c also confirm the idea that inflammatory activation is enhanced by poor glycemic control[10].

In like manner, Interleukin-6 (IL-6) was very high in diabetic patients than in controls[11]. IL-6 is a multitasking cytokine that interacts in the immune activation, the hepatic production of acute-phase proteins, and metabolic control. The high levels of IL-6 in diabetic patients indicate the condition of persistent immune-metabolic stimulation, potentially leading to β-cell stress, aggravation of insulin resistance, and inflammation of the vascular walls. Another important observation in the current research is that the level of IL-6 was positively correlated with the level of HbA1c, which implied that the inflammatory state was more likely to be expressed by patients with worse glycemic control[12]. This connection is biologically plausible, in that hyperglycemia in itself favors oxidative stress, the formation of end-products of glycated tissues, and the secretion of cytokines, which thus maintains a persistent chronic inflammatory milieu. Recent publications still uphold IL-6 as one of the key bridges between adiposity and insulin resistance on the one hand, and T2DM-related inflammatory load on the other[13].

Another inflammatory factor that was considered in this study, Tumor Necrosis Factor-alpha (TNF-α), was also observed to be significantly high in diabetic patients[14]. One of the most significant pro-inflammatory cytokines that have a role to play in insulin resistance is TNF-α. It is also associated with metabolic dysfunction because of impaired insulin receptor signaling, lowered glucose transporter activity, stimulated lipolysis, and increased oxidative stress. The drastically larger levels of TNF-α in diabetic group demonstrate that the inflammatory signaling is actively engaged in the pathogenesis of T2DM in this group of people[15]. Moreover, the TNF-α level and HbA1c were positively related in the present study, indicating that chronic hyperglycemia might cause inflammatory dysregulation with time[16].

The most significant conclusion of the current research was probably the obvious correlation between glycemic control and the levels of inflammatory markers[17]. Patients with diabetes with poor glycemic control (HbA1c ≥7.0) exhibited considerably elevated levels of CRP, IL-6, and TNF-α than patients who had comparatively good glycemic control. This finding goes a long way in upholding the principle that uncontrolled diabetes has a higher inflammatory load. This is clinically very pertinent since chronic inflammatory stimulation can lead to the pathogenesis and worsening of microvascular and macrovascular complications[18]. It is possible that patients with chronic and high levels of inflammatory markers are at higher risk of nephropathy, neuropathy, retinopathy, atherosclerosis, and cardiovascular occurrence[19].

The supportive results of the positive correlation between inflammatory markers and the period of diabetes in the given study contribute to the progressive character of inflammatory-metabolic damage in T2DM[20]. The increased period of the disease could be due to the increased exposure to hyperglycemia, oxidative stress, endothelial damage, and metabolic imbalance, which can then result in cumulative inflammatory activation. It implies that inflammation in diabetes is not merely incidental laboratory results but it may be a long-term biological event that increases with the advancement of the disease[1].

Clinically, the results of this study have implications[2]. Type 2 Diabetes Mellitus routine management is mostly concerned with the control of blood glucose, HbA1c, dietary changes, and pharmacological treatment. Nevertheless, the findings of the current research indicate that the inflammatory markers that could be used to further understand the biological severity of the disease include CRP, IL-6, and TNF-alpha. Such indicators can be used to define the patients with a greater inflammatory load that can be at higher risk of complications, despite the fact that they are not yet visibly manifested in clinical practice[3]. Hence, inflammatory profiling could be useful as a supplemental instrument in the risk evaluation and disease follow-up[4].

The local application of this study is also evident in the Pakistani clinical environment where the prevalence of diabetes is high and patients have poor metabolic controls, obesity, and late disease diagnoses[5]. In these environments, the consideration of inflammatory biomarkers could enhance prompt risk identification and assist clinicians to better address diabetic patients using an integrative metabolic-inflammatory strategy to deal with patients instead of glycemic indices[6].

Although significant results were obtained, the current research has some limitations[7]. Since it is a cross-sectional study, it is not able to develop a temporal or causal relationship between inflammation and diabetes progression. The research was carried out in one center and this can be considered to be a limitation to the extrapolation of the results to the wider groups of people. Moreover, only three inflammatory markers were measured, and it is possible that the other biomarkers like IL-1β, MCP-1, adiponectin, leptin, and oxidative stress parameters could give a more detailed picture of the inflammatory-metabolic profile of the diabetic patients[8]. It is suggested that additional research on the prognostic and treatment implications of inflammatory biomarkers in Type 2 Diabetes Mellitus needs to be conducted in future with larger sample sizes and longitudinal follow-up of the subjects[9].

All in all, the results of this research indicate strongly in favor of the idea that Type 2 Diabetes Mellitus is closely related to a high inflammatory burden, which grows in proportion to poor glycemic control, high BMI, and length of the disease[10]. These findings support the emerging knowledge that inflammation is not simply related to diabetes, but that it plays a profound role in the pathophysiology and clinical outcomes of the condition[11].

CONCLUSION

Finally, this paper has shown that Type 2 Diabetes Mellitus patients showed a great increase of the serum levels of CRP, IL-6, and TNF-a relative to healthy controls suggesting that there was a high level of systemic inflammatory condition. The research also established that these inflammatory indicators had positive relationships with HbA1c, Body Mass Index, and length of diabetes and thus, deteriorating glycemic control and metabolic burden have a strong connection with augmented inflammation. These results indicate that Type 2 Diabetes Mellitus must not be perceived as an illness of glucose metabolism but a disorder of continuous inflammatory stimulation. The high inflammatory state of diabetic patients can be the cause of the long-term vascular and metabolic consequences and it can consequently play a significant prognostic and therapeutic role. Inflammatory biomarkers like CRP, IL-6, and TNF- 0 could be of further clinical use in the identification of high-risk diabetic individuals as well as the information about the overall biological cost of the disease. Integration of inflammatory assessment into the diabetes research, and, when possible, clinical evaluation can help in the earlier risk stratification and more detailed management of patients. More extensive and longitudinal research is encouraged to find whether such inflammatory markers could be used as predictors of not only the severity of the disease but also as a means of therapeutic intervention of Type 2 Diabetes Mellitus in the long term.

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