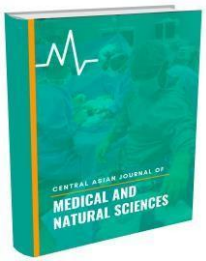

CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES
https://cajmns.centralasianstudies.org/index.php/CAJMNS
Volume: 07 Issue: 02 | April 2026 ISSN: 2660-4159

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Article

Association Between Serum Sirtuin-1 and Oxidative Stress Parameters in Type 2 Diabetic Patients

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Abstract: Samples were collected between October 2025 and January 2026. The study included 60 blood samples from patients diagnosed with Type 2 Diabetes Mellitus, including both males and females aged between 35 and 65 years. In addition, 30 blood samples were collected from apparently healthy individuals (males and females) aged between 35 and 55 years who served as the control group. All diabetic cases were clinically diagnosed by specialized physicians. The results showed a significant decrease in serum Sirtuin 1 (SIRT-1) levels in patients with type 2 diabetes mellitus (2.217±1.013 ng/mL) compared with the healthy control group (4.165±1.213 ng/mL) at a significance level of ($P \leq 0.01$). Conversely, a significant increase was observed in oxidative stress markers, including Malondialdehyde (MDA) and the inflammatory chemokine CCL2, among diabetic patients compared with the control group at a highly significant level ($P < 0.0001$). In addition, the level of Hemoglobin A1c (HbA1c) was markedly elevated in patients compared with healthy individuals. The effect of Body Mass Index (BMI) was also evaluated, and the results indicated a significant increase in BMI among most diabetic patients compared with the control group, suggesting that increased body mass index may contribute to a higher risk of developing type 2 diabetes. Correlation analysis revealed a very weak positive relationship between SIRT-1 and oxidative stress markers, including MDA ($r = 0.011$) and CCL2 ($r = 0.030$). Furthermore, Receiver Operating Characteristic Curve (ROC) curve analysis was performed to evaluate the discriminative performance of the studied biomarkers in distinguishing diabetic patients from healthy individuals. The results demonstrated that HbA1c and BMI showed perfect discriminative accuracy, while SIRT-1 and oxidative stress markers exhibited very good to excellent diagnostic performance. Overall, these findings suggest that alterations in the balance of inflammatory and metabolic biomarkers, particularly the decrease in SIRT-1 and the elevation of oxidative stress and inflammatory markers, may play an important role in the progression and severity of type 2 diabetes mellitus.

Keywords: T2DM, Sirtuin 1, Oxidative Stress.

Citation: Taha Z. T., Mohammad A. T., Kamil S. A. Association Between Serum Sirtuin-1 and Oxidative Stress Parameters in Type 2 Diabetic Patients. Central Asian Journal of Medical and Natural Science 2026, 7(2), 610-620.

Received: 14th Jan 2026
Revised: 16th Feb 2026
Accepted: 14th Mar 2026
Published: 20th Apr 2026

1. Introduction

One of the most common chronic illnesses in the world, type 2 diabetes mellitus (T2DM), is regarded as a serious lifestyle-related condition. According to the latest data from the NCD Risk Factor Collaboration (2022), approximately 828 million people are currently living with diabetes globally, with more than 95% of cases attributed to type 2 diabetes mellitus. Epidemiological projections indicate that the prevalence of diabetes will continue to increase in the coming decades. In the United States alone, it is estimated that the prevalence may reach approximately 10.8% by 2050, although this figure could be higher due to variations in current prevalence estimates. T2DM is a complex metabolic

disorder characterized by chronic hyperglycemia resulting from a progressive defect in insulin secretion and/or insulin resistance in peripheral tissues. The disease is also marked by varying degrees of pancreatic beta-cell dysfunction and insulin resistance among affected individuals [1]. There is a strong association between obesity and T2DM, involving multiple regulatory pathways, including those mediated by the central nervous system, which controls food intake and energy expenditure while integrating signals from peripheral organs and environmental factors. Notably, T2DM is no longer restricted to older adults. In recent years, the prevalence of the disease has increased significantly among younger individuals under the age of 40 [2]. It is crucial to rule out other types of diabetes, such as Type 1 Diabetes (T1DM), Latent Autoimmune Diabetes in Adults (LADA), and Maturity Onset Diabetes of the Young (MODY), when diagnosing type 2 diabetes in younger populations. Predicting any consequences and starting the right treatment approaches depend on an accurate diagnosis [3].

Sirtuins are a family of NAD+ (Nicotinamide Adenine Dinucleotide)-dependent deacylase enzymes that play essential roles in regulating numerous cellular processes [4]. Among the seven currently identified members of the sirtuin family, Sirtuin 1 (SIRT1) is considered one of the most important regulators, as it participates in cellular signal transduction, metabolic regulation, DNA repair, and the modulation of inflammatory responses. In addition, SIRT1 plays a critical role in the regulation of cellular senescence and organismal aging [5]. Despite the growing number of experimental studies highlighting the biological importance of SIRT1 in the human body, available data regarding its circulating levels in human serum remain limited. Notably, SIRT1 is directly involved in the intracellular signaling of growth hormone, promoting the secretion of Insulin-like Growth Factor 1 (IGF-1) through modulation of the JAK2/STAT signaling pathway. Furthermore, SIRT1 contributes to the regulation of growth hormone release in the central nervous system and plays an important role in growth plate chondrogenesis and longitudinal bone growth [6]. SIRT1 is considered a key molecular regulator of metabolic homeostasis. Increasing scientific evidence suggests that dysregulation of SIRT1 is closely associated with the pathophysiology of Type 2 Diabetes Mellitus. SIRT1 plays a crucial role in improving insulin sensitivity, regulating glucose and lipid metabolism, and maintaining pancreatic beta-cell function. Moreover, SIRT1 exerts antioxidant effects by activating free radical scavenging pathways and enhancing the expression of antioxidant enzymes through the regulation of FOXO transcription factors and PGC-1a [7]. In addition, SIRT1 suppresses inflammatory responses by inhibiting the activation of NF-kB and reducing the secretion of pro-inflammatory cytokines and chemokines. In the context of type 2 diabetes, chronic metabolic stress and persistent hyperglycemia can disrupt SIRT1 activity, thereby promoting oxidative stress and low-grade inflammation, which are major contributors to insulin resistance and the progression of diabetic complications [8], [9].

Oxidative stress and low-grade inflammation are considered major pathogenic mechanisms involved in the development and progression of Type 2 Diabetes Mellitus and its associated complications. Malondialdehyde (MDA) is one of the most important end products of lipid peroxidation and is widely used as a biomarker for assessing the level of oxidative stress in the body. Several studies have reported elevated MDA levels in patients with diabetes as a result of chronic hyperglycemia and increased generation of reactive oxygen species [10]. On the other hand, CCL2, also known as Monocyte Chemoattractant Protein-1 (MCP-1), is a key inflammatory chemokine that contributes to the recruitment of monocytes and the amplification of inflammatory responses associated with insulin resistance and metabolic dysregulation in type 2 diabetes [11]. The simultaneous elevation of both MDA and CCL2 reflects the close interaction between oxidative stress and inflammatory responses in the pathophysiology of type 2 diabetes, suggesting that these biomarkers may play significant roles in the progression and severity of the disease [12].

Objectives of the Study

This study aims to:

1. Evaluate the serum level of Sirtuin 1 (SIRT1) in patients with Type 2 Diabetes Mellitus and compare it with that of healthy individuals.
2. Assess the level of oxidative stress markers represented by Malondialdehyde (MDA) in diabetic patients compared with the healthy control group.
3. Measure the level of the inflammatory chemokine CCL2 and investigate its role in the inflammatory state associated with type 2 diabetes.
4. To assess the diagnostic performance of SIRT1, MDA, and CCL2 in differentiating patients with type 2 diabetes from healthy persons, as well as to ascertain the ideal cut-off value, sensitivity, and specificity, perform Receiver Operating Characteristic Curve (ROC) curve analysis.
5. Explore the combined predictive value of these biomarkers as potential supportive tools for the early assessment of metabolic and inflammatory alterations associated with type 2 diabetes.

2. Materials and Methods

2-1 Study Design and Participants

Between October 2025 and January 2026, a case-control design was used for this investigation. The study had 90 people in all, 60 of whom had been diagnosed with Type 2 Diabetes Mellitus, and 30 apparently healthy individuals who served as the control group. where they were diagnosed by Al-Shirqat General Hospital. The patients were recruited from the outpatient clinics of specialist physicians according to the diagnostic criteria established by the American Diabetes Association (ADA). The participants' ages ranged between 35 and 65 years. The control group was matched with the patient group in terms of age and gender, and individuals with a history of diabetes, cardiovascular diseases, chronic inflammatory disorders, kidney diseases, or malignancies were excluded.

2-2 Inclusion and Exclusion Criteria

Patients with a confirmed diagnosis of Type 2 Diabetes Mellitus were included in the study. The exclusion criteria comprised individuals diagnosed with Type 1 Diabetes, gestational diabetes, autoimmune diseases, acute infections, or chronic liver and kidney diseases. In addition, smokers and individuals who had used antioxidant supplements or anti-inflammatory medications within the three months preceding the study. were also excluded.

2-3 Sample Collection

After each participant fasted for eight to twelve hours, around five milliliters of venous blood were drawn. After letting the blood samples clot at room temperature, the serum was extracted by centrifuging them for ten minutes at 3000 rpm. After that, the serum samples were divided into aliquots and kept at -32°C till biochemical tests were carried out.

2-4 Biochemical Measurements

Serum levels of Sirtuin 1 (SIRT1) and the inflammatory chemokine CCL2 were measured using the Enzyme-Linked Immunosorbent Assay (ELISA) technique according to the manufacturer's instructions. The level of Malondialdehyde (MDA) was determined using the Thiobarbituric Acid Reactive Substances Assay (TBARS) method as an indicator of lipid peroxidation. The level of Hemoglobin A1c (HbA1c) was measured using a standard immunoassay method. The Body Mass Index (BMI) was calculated using the following equation: BMI = weight (kg) / height^2 (m^2).

2-5 Statistical Analysis

SPSS software (version XX, IBM Corporation, USA) was used to conduct statistical analyses. The information is displayed as mean ± standard deviation (SD). The two groups' differences were compared using independent-sample t-tests. Pearson correlation analysis was conducted to evaluate the associations between SIRT1 levels and other biochemical variables. Receiver operating characteristic (ROC) curve analysis was also performed to assess the diagnostic performance of SIRT1, MDA, and CCL2 in distinguishing patients with type 2 diabetes from healthy controls, including calculation of the area under the curve (AUC), sensitivity, specificity, and determination of the optimal cut-off value. A p-value < 0.01 was considered statistically significant.

3. Results

Analysis revealed that SIRT1 levels were significantly lower in patients with type 2 diabetes (2.217 ± 1.013) compared to the control group (4.165 ± 1.213; $p < 0.0001$), as shown in Figure 1 and Table 1, consistent with previous studies [13]. Similarly, malondialdehyde (MDA) levels were significantly elevated in patients (6.318 ± 1.772) relative to controls (3.117 ± 1.262; $p < 0.0001$), as illustrated in Figure 2 and Table 1, in agreement with prior findings [14]. Moreover, the inflammatory chemokine CCL2 was markedly higher in patients (220.928 ± 47.834) than in controls (92.218 ± 29.282; $p < 0.0001$), as shown in Figure 3 and Table 1, aligning with previous reports[15]. A significant difference was also observed in glycated hemoglobin (HbA1c) between the groups; controls exhibited the lowest mean (5.113 ± 0.405), while patients with type 2 diabetes had the highest levels (8.600 ± 1.193; $p < 0.0001$), as depicted in Figure 4 and Table 1, consistent with existing literature[16]. Body mass index (BMI) differed significantly between the groups ($p < 0.0001$), being lower in healthy individuals (23.487 ± 1.312) compared to patients with type 2 diabetes (31.450 ± 2.945), as shown in Figure 5 and Table 1, in line with previous studies [15]. Finally, a weak positive correlation was observed between SIRT1 levels and all biochemical variables among the patient group, as presented in Table 2.

Table 1. Shows the level of SIRT1 and the level of other biochemical variables in patients compared to the control group.

Groups / Parameters	Mean ± SD		P-value
	Control (n=30)	Patients (n=60)	
SIRT-1	4.165±1.213	2.217±1.013	< 0.0001*
MDA	3.117±1.262	6.318±1.772	< 0.0001*
CCL2	92.218±29.282	220.928±47.834	< 0.0001*
HbA1c	5.113±0.405	8.600±1.193	< 0.0001*
BMI	23.487±1.312	31.450±2.945	< 0.0001*

*** P ≤ 0.01**

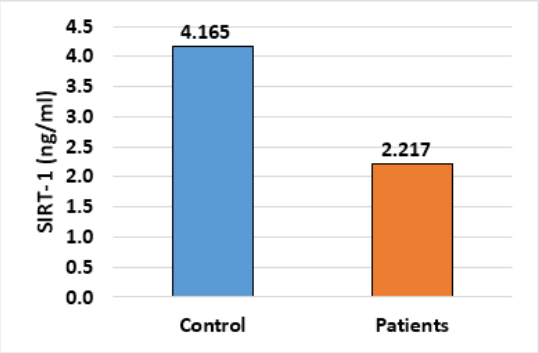

Figure 1. Shows the SIRT1 level in patients compared to the control group.

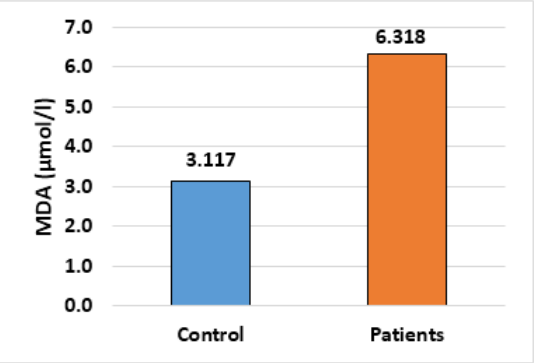

Figure 2. Shows the level of MDA in patients compared to the control group.

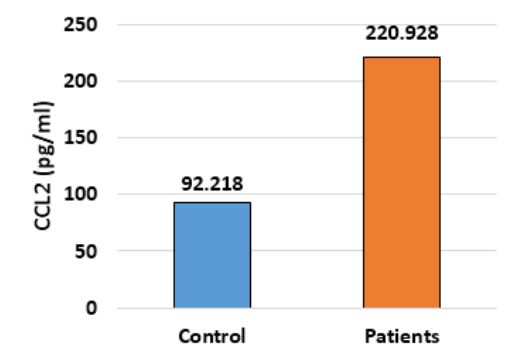

Figure 3. Shows the CCL2 level in patients compared to the control group.

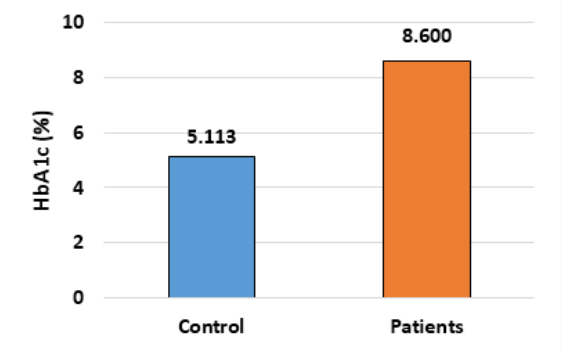

Figure 4. Shows the HbA1c level in patients compared to the control group.

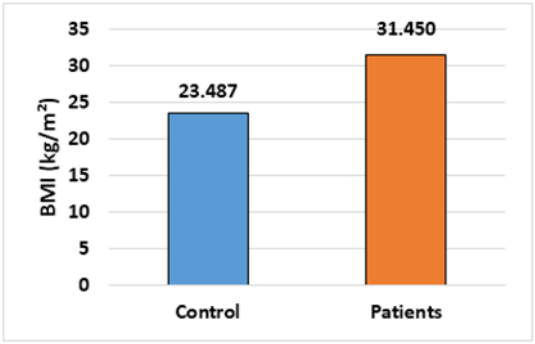

Figure 5. Shows the BMI level in patients compared to the control group.

Table 2. Shows the correlation between SIRT1 and all Parameters in the patient group.

		Patients			
		MDA	CCL2	HbA1c	BMI
SIRT-1	r	0.011	0.030	0.070	0.103
	p	0.931	0.822	0.597	0.432
	N	60	60	60	60

****. Correlation is significant at the 0.01 level (2-tailed).**

***. Correlation is significant at the 0.05 level (2-tailed).**

r. Pearson correlation

p. Sig. (2-tailed)

N. number of samples

3-1 Results of ROC analysis for SIRT1 and studied biochemical variables in patients and healthy individuals:

The data analysis results are shown in the following table:

Table 3. Shows the ROC analysis of SIRT1 and biochemical variables of patients and healthy individuals.

Parameters	Cut off	Sensitivity %	Specificity %	AUC	P-value
SIRT-1	≤2.93	85.00	90.00	0.901	<0.001
MDA	>4.27	88.33	86.67	0.934	<0.001
CCL2	>120.3	96.67	93.33	0.981	<0.001
HbA1c	>6.2	100.00	100.00	1.000	<0.001
BMI	>25.8	100.00	100.00	1.000	<0.001

Receiver operating characteristic (ROC) curve analysis, as presented in Table 3, demonstrated that SIRT-1 exhibited a high diagnostic performance in distinguishing diseased cases from healthy controls. The biomarker showed a sensitivity of 85.00% and a specificity of 90.00%, with an area under the curve (AUC) of .0.901, which was statistically significant ($P < 0.001$). Similarly, the other variables listed in Table 3, including SIRT-1, also demonstrated relatively high sensitivity, specificity, and AUC values. These findings suggest that these biomarkers may serve as effective preliminary diagnostic tools for identifying most pathological cases and differentiating them from normal conditions. Therefore, these markers could be considered reliable supportive indicators in the diagnosis of type 2 diabetes mellitus. The ROC curves for all evaluated variables are illustrated in the following figures:

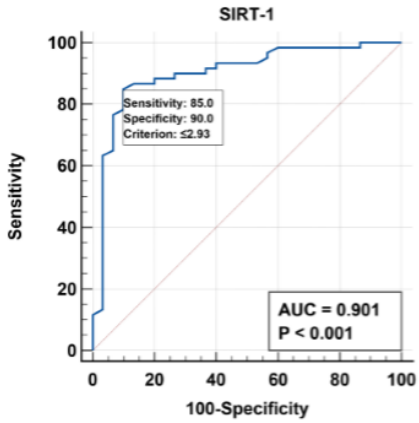

Figure 6. Represents the ROC curve for SIRT-1 in patients and healthy.

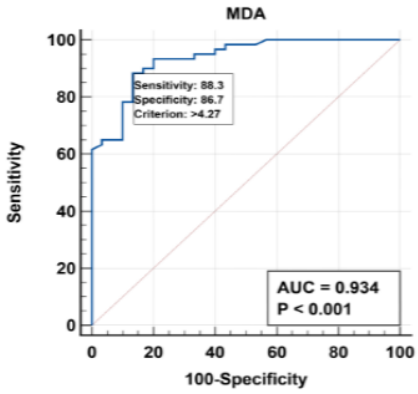

Figure 7. Represents the ROC curve for MDA in patients and healthy.

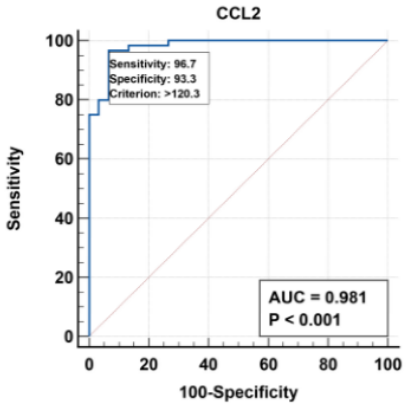

Figure 8. Represents the ROC curve for CCL2 in patients and healthy.

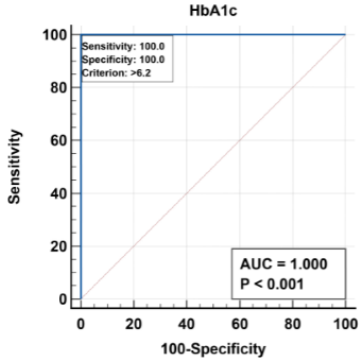

Figure 9. Represents the ROC curve for HbA1c in patients and healthy.

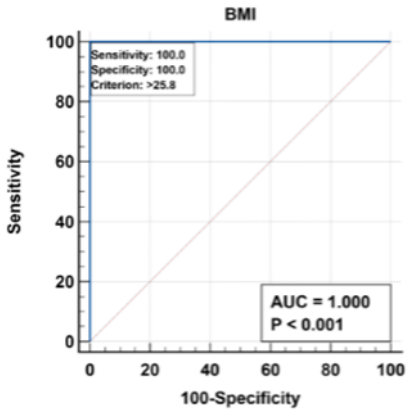

Figure 10. Represents the ROC curve for BMI in patients and healthy.

4. Discussion

By modifying insulin secretion, enhancing insulin sensitivity, and controlling circadian metabolic rhythms, Kitada et al. showed that SIRT1 had antidiabetic benefits [17]. In a similar vein, Yacoub et al. found that oxidative stress and long-term metabolic stress decrease SIRT1 expression [18]. The current investigation found that individuals with type 2 diabetes mellitus had significantly lower SIRT1 levels, which is consistent with these findings. Chronic hyperglycemia, which encourages oxidative stress and the production of reactive oxygen species (ROS), may be the cause of this decrease [19]. Furthermore, SIRT1 plays a protective role in pancreatic ß-cells by mitigating oxidative stress through inhibition of the NF-KB signaling pathway. Experimental studies in BESTO mice, which overexpress SIRT1 in B-cells, have shown that elevated SIRT1 levels improve glucose tolerance and enhance insulin secretion [20], [21]. In addition, SIRT1 overexpression increases ATP production by suppressing uncoupling protein-2 (UCP2) in B-cells [22], thereby promoting calcium-dependent insulin secretion [23]. Additionally, SIRT1 increases the transcription and activation of the transcription factors MafA and NeuroD, which are critical for sustaining insulin production and fostering in vivo ß-cell survival. Additionally, SIRT1 protects against several types of cellular stress by controlling insulin production through regulation of UCP2, FOXO1, and NAD metabolism [24]. Similarly, Calabrese et al. found that individuals with uncontrolled diabetes mellitus had considerably lower expression levels of both SIRT1 and SIRT3 [25]. Our findings also demonstrated a significant reduction in serum SIRT1 levels in patients with uncontrolled diabetes, who exhibited elevated fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) levels. SIRT1 plays a critical role in the regulation of glucose homeostasis. Under diabetic conditions, the activity of SIRT1 has been reported to decline in various tissues. SIRT1 is recognized as a key regulator of metabolic responses to caloric restriction in mammals [26]. In this context, Khweilid et al. reported that the significant increase in SIRT1 levels observed in a prediabetic group subjected to caloric restriction was closely associated with reduced serum glucose levels [27].

Patients with type 2 diabetes mellitus are often exposed to oxidative stress as a consequence of chronic hyperglycemia. This condition arises from increased production of reactive oxygen species (ROS) or free radicals. ROS can be generated through several mechanisms, comprising the pentose phosphate route, glycolysis, the polyol pathway, glucose auto-oxidation, and non-enzymatic protein glycation. Furthermore, decreased effectiveness of antioxidant defense mechanisms that shield cellular components from ROS-induced damage exacerbates oxidative stress [28]. Experimental studies and epidemiological analyses indicate that elevated ROS levels play a critical role in lipid peroxidation. Reactive oxygen species can induce oxidative injury by interacting with polyunsaturated double bonds present in membrane lipids, thereby initiating a chain of

oxidative reactions [29]. During the propagation phase of lipid peroxidation, ROS-mediated oxidation of lipids generates several secondary products, including malondialdehyde (MDA) [30]. Elevated MDA levels are widely considered a reliable biomarker of oxidative stress, reflecting increased free radical activity and lipid peroxidation in cellular membranes [31]. Although some studies have reported reduced CCL-2 levels in prediabetic individuals, our findings partially agree with previous reports demonstrating elevated CCL-2 levels in prediabetes as well as in type 1 and type 2 diabetes mellitus. Such discrepancies may be related to the lack of further subgroup classification based on body mass index (BMI) or obesity status in those studies [32], [33]. In contrast, a study conducted in Saudi Arabia reported increased CCL-2 levels in obese women and decreased levels in obese men, although the differences were not statistically significant, which differs from the findings of the present study and other previous reports [34]. Excess caloric intake and obesity have been shown to activate adipose tissue, particularly adipocyte-associated immune cells, leading to increased secretion of CCL-2 [35]. This chemokine functions as a major mediator in the inflammatory cascade linked to insulin resistance and metabolic syndrome by attracting monocytes, natural killer cells, and other inflammatory cells [36], [37].

5. Conclusion

The findings of the present study demonstrated significant alterations in serum Sirtuin 1 (SIRT1) levels in patients with type 2 diabetes mellitus (T2DM) compared with healthy individuals. These results suggest that SIRT1 may play an important role in the pathophysiology of T2DM through its involvement in oxidative stress and inflammatory signaling pathways. In addition, significantly elevated levels of the oxidative stress marker malondialdehyde (MDA) and the inflammatory chemokine CCL2 were observed in diabetic patients, indicating increased oxidative damage and inflammatory activity associated with the disease. Receiver operating characteristic (ROC) curve analysis further revealed that SIRT1, MDA, and CCL2 exhibited high diagnostic performance with considerable sensitivity and specificity, suggesting their potential utility as biomarkers for the diagnosis and monitoring of type 2 diabetes mellitus. Overall, these findings highlight the potential role of SIRT1 in modulating oxidative stress and inflammation in T2DM, supporting its possible application as a promising biomarker for disease progression and clinical evaluation.

Recommendations

1. Future studies involving larger and multicenter populations are recommended to further validate the role of SIRT1 as a diagnostic and prognostic biomarker in type 2 diabetes mellitus (T2DM).
2. Further research should focus on elucidating the molecular mechanisms linking SIRT1 with oxidative stress and inflammatory mediators, including MDA and CCL2, in diabetic patients.
3. Longitudinal studies are required to evaluate the therapeutic potential of targeting SIRT1 in preventing diabetic complications and improving disease management.

REFERENCES

[1] E. Młynarska, W. Czarnik, N. Dzieża, W. Jędraszak, G. Majchrowicz, F. Prusinowski *et al.*, "Type 2 diabetes mellitus new pathogenetic mechanisms, treatment and the most important complications," *International Journal of Molecular Sciences*, vol. 26, no. 3, p. 1094, 2025.

[2] GBD 2021 Diabetes Collaborators, "Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021," *The Lancet*, vol. 402, pp. 203–234, 2023.
[3] M. Strati, M. Moustaki, T. Psaltopoulou, A. Vryonidou, and S. A. Paschou, "Early onset type 2 diabetes mellitus: an update," *Endocrine*, vol. 85, pp. 965–978, 2024.
[4] A. Fedorczak, A. Lewiński, and R. Stawerska, "Sirtuin 1 serum concentration in healthy children: dependence on sex, age, stage of puberty, body weight and diet," *Frontiers in Endocrinology*, vol. 15, p. 1356612, 2024.
[5] M. C. Haigis and D. A. Sinclair, "Mammalian sirtuins: biological insights and disease relevance," *Annual Review of Pathology*, vol. 5, pp. 253–295, 2010.
[6] M. Kitada, Y. Ogura, I. Monno, and D. Koya, "Sirtuins and type 2 diabetes: role in inflammation, oxidative stress, and mitochondrial function," *Frontiers in Endocrinology*, vol. 10, p. 187, 2019.
[7] M. Kitada, Y. Ogura, and D. Koya, "The protective role of SIRT1 in diabetes and its complications," *Diabetes & Metabolism Journal*, vol. 40, no. 3, pp. 187–194, 2016.
[8] S. V. de Kreutzenberg *et al*., "SIRT1 and metabolic control: implications for insulin resistance," *Diabetes*, vol. 59, no. 8, pp. 1876–1885, 2010.
[9] A. Chalkiadaki and L. Guarente, "The multifaceted functions of sirtuins in cancer and metabolism," *Nature Reviews Cancer*, vol. 15, pp. 608–624, 2015.
[10] A. Ceriello and E. Motz, "Is oxidative stress the pathogenic mechanism underlying insulin resistance, diabetes, and cardiovascular disease?" *Arteriosclerosis, Thrombosis, and Vascular Biology*, vol. 24, no. 5, pp. 816–823, 2004.
[11] H. Kanda *et al*., "MCP-1 contributes to macrophage infiltration into adipose tissue and insulin resistance in obesity," *Journal of Clinical Investigation*, vol. 116, no. 6, pp. 1494–1505, 2006.
[12] C. Herder, E. Dalmas, M. Böni-Schnetzler, and M. Y. Donath, "Role of cytokines and chemokines in the pathogenesis of type 2 diabetes," *Endocrine Reviews*, vol. 38, no. 4, pp. 362–401, 2017.
[13] R. Rahimi, S. Ghanbari, M. Khazaei, and E. Niroman, "Comparison of sirtuin 1 level and related blood factors in diabetic and healthy subjects," *Pediatric Endocrinology Diabetes and Metabolism*, vol. 26, no. 1, pp. 17–21, 2020.
[14] R. M. Azeez, W. K. Al-Hashemi, T. R. Al-Auqbi, and J. Jebali, "Relation of MDA as oxidative stress marker with lipid profile in diabetic patients," *Al-Nahrain Journal of Science*, vol. 26, no. 3, pp. 12–17, 2023.
[15] M. M. Mir *et al*., "The role of pro-inflammatory chemokines CCL-1, CCL-2, CCL-4, and CCL-5 in the etiopathogenesis of type 2 diabetes mellitus," *Journal of Personalized Medicine*, vol. 14, p. 743, 2024.
[16] H. Selvaraj *et al*., "Assessment of serum sirtuin 1 levels in type 2 diabetes mellitus with coronary artery disease patients," *Romanian Journal of Medical Practice*, vol. 20, no. 3, p. 105, 2025.
[17] M. Kitada and D. Koya, "SIRT1 in type 2 diabetes: mechanisms and therapeutic potential," *Diabetes & Metabolism Journal*, vol. 37, pp. 315–325, 2013.
[18] R. Yacoub, K. Lee, and J. C. He, "The role of SIRT1 in diabetic kidney disease," *Frontiers in Endocrinology*, vol. 5, p. 166, 2014.
[19] H. W. Liu and S. J. Chang, "Moderate exercise suppresses NF-κB signaling and activates the SIRT1-AMPK-PGC1α axis to attenuate muscle loss in diabetic db/db mice," *Frontiers in Physiology*, vol. 9, p. 636, 2018.
[20] Y. Cao *et al*., "SIRT1 and insulin resistance," *Journal of Diabetes and Its Complications*, vol. 30, pp. 178–183, 2016.
[21] B. Schwer and E. Verdin, "Conserved metabolic regulatory functions of sirtuins," *Cell Metabolism*, vol. 7, pp. 104–112, 2008.
[22] K. Türkmen, A. Karagoz, and A. Kucuk, "Sirtuins as novel players in the pathogenesis of diabetes mellitus," *World Journal of Diabetes*, vol. 5, pp. 894–900, 2014.
[23] A. Chalkiadaki and L. Guarente, "Sirtuins mediate mammalian metabolic responses to nutrient availability," *Nature Reviews Endocrinology*, vol. 8, pp. 287–296, 2012.
[24] V. Calabrese *et al*., "Oxidative stress, glutathione status, sirtuin and cellular stress response in type 2 diabetes," *Biochimica et Biophysica Acta - Molecular Basis of Disease*, vol. 1822, pp. 729–736, 2012.
[25] D. Chen and L. Guarente, "SIR2: a potential target for calorie restriction mimetics," *Trends in Molecular Medicine*, vol. 13, pp. 64–71, 2007.
[26] E. A. Khowailed *et al*., "Is the effect of caloric restriction on type 2 diabetes mellitus in rats mediated via sirtuin-1?" *Medical Journal of Cairo University*, vol. 83, pp. 357–365, 2015.
[27] V. P. Jayasekharan *et al*., "Estimation of nitric oxide and malondialdehyde in serum and saliva of patients with oral lichen planus," *SRM Journal of Research in Dental Sciences*, vol. 5, no. 4, p. 230, 2014.

[28] M. W. A. Khan, K. Banga, and W. A. Khan, "Gluco-oxidation of proteins in the etiology of diabetic retinopathy," in *Diabetic Retinopathy*, InTech, 2012, pp. 31–52.
[29] M. Kumawat, "Antioxidant enzymes and lipid peroxidation in type 2 diabetes mellitus patients with and without nephropathy," *North American Journal of Medical Sciences*, vol. 5, no. 3, p. 213, 2013.
[30] N. Kurtul and E. Gökpınar, "Salivary lipid peroxidation and total sialic acid levels in smokers and smokeless tobacco users," *Mediators of Inflammation*, vol. 2012, 2012.
[31] Y. Yan *et al*., "Prevalence, awareness and control of type 2 diabetes mellitus and risk factors in Chinese elderly population," *BMC Public Health*, vol. 22, p. 1382, 2022.
[32] M. M. Mir *et al*., "Differential association of selected adipocytokines with the pathogenesis and progression of type 2 diabetes mellitus," *Journal of Personalized Medicine*, vol. 12, p. 735, 2022.
[33] S. Harakeh *et al*., "Chemokines and their association with body mass index among healthy Saudis," *Saudi Journal of Biological Sciences*, vol. 27, pp. 6–11, 2020.
[34] G. S. Hotamisligil, "Inflammation and metabolic disorders," *Nature*, vol. 444, pp. 860–867, 2006.
[35] C. A. Feghali and T. M. Wright, "Cytokines in acute and chronic inflammation," *Frontiers in Bioscience*, vol. 2, pp. d12–d26, 1997.
[36] S. Purohit *et al*., "Large-scale discovery and validation studies demonstrate significant reductions in circulating levels of IL-8, IL-1Ra, MCP-1, and MIP-1β in subjects with type 1 diabetes," *Journal of Clinical Endocrinology & Metabolism*, vol. 100, pp. E1179–E1187, 2015.
[37] S. Purohit *et al*., "Large-scale discovery and validation studies demonstrate significant reductions in circulating levels of IL-8, IL-1Ra, MCP-1, and MIP-1β in subjects with type 1 diabetes," *Journal of Clinical Endocrinology & Metabolism*, vol. 100, pp. E1179–E1187, 2015.