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Research Article

Comparison Of Serum Levels of Interleukin-18 And Asprosin in Type 2 Dm with And Without Retinopathy

Dr Hossam Khurshid Al Rahman Khan[1], Dr Bharti Nigam[2], Dr Farhat Siddiqui[3]
[1]*Resident (JR -3), Department of Ophthalmology, Era's Lucknow Medical College and Hospital, Lucknow*
[2]*Head Of Department, Department of Ophthalmology, Era's Lucknow Medical College and Hospital, Lucknow.*
[3]*Assistant Professor, Department of Ophthalmology, Era's Lucknow Medical College and Hospital, Lucknow.*

Abstract

Introduction: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder associated with long-term complications such as diabetic retinopathy (DR), a leading cause of preventable blindness. Chronic hyperglycemia induces inflammatory and vascular changes in the retina. Recent studies suggest Interleukin-18 (IL-18), a pro-inflammatory cytokine, and asprosin, a glucogenic adipokine, may be involved in the pathogenesis of DR. This study aimed to compare the serum levels of IL-18 and asprosin in T2DM patients with and without DR and to evaluate their association with DR severity. ***Material and Methods***: A cross-sectional observational study was conducted involving T2DM patients attending a tertiary care hospital. Participants were divided into two groups: those with diabetic retinopathy (DR group) and those without retinopathy (non-DR group). Serum levels of IL-18 and the asprosin were measured using enzyme-linked immunosorbent assay (ELISA) kits. The severity of DR was classified using standard ETDRS guidelines. Statistical analyses were performed using SPSS to assess the differences and correlations among groups. ***Results***: Serum IL-18 and asprosin levels were significantly elevated in the DR group compared to the non-DR group ($p<0.05$). A positive correlation was observed between the levels of both biomarkers and the severity of diabetic retinopathy, particularly in cases of proliferative DR. These findings suggest a strong association between elevated IL-18 and asprosin levels and retinal microvascular damage in T2DM. ***Conclusion***: This study demonstrates that elevated serum IL-18 and asprosin levels are associated with the presence and progression of diabetic retinopathy in T2DM patients. These biomarkers may serve as potential indicators for early detection, disease monitoring, and risk stratification in diabetic retinopathy.

Keywords: Type 2 Diabetes Mellitus, Diabetic Retinopathy, Interleukin-18, Asprosin, Biomarkers

Introduction:

Type 2 Diabetes Mellitus (T2DM) is a chronic, multifactorial metabolic disorder characterized by persistent hyperglycemia resulting primarily from insulin resistance in peripheral tissues, impaired insulin secretion by pancreatic β-cells, and enhanced hepatic glucose output. It is among the most pressing global health issues due to its rising prevalence driven by aging populations, sedentary lifestyles, increasing rates of obesity, and urbanization. T2DM imposes a heavy burden on healthcare systems and society by predisposing individuals to numerous long-term complications. These include macrovascular diseases such as coronary artery disease, stroke, and peripheral artery disease, as well as microvascular complications like nephropathy, neuropathy, and retinopathy. Diabetic retinopathy (DR), in particular, is a leading cause of avoidable visual impairment and blindness among working-age adults globally [1,2].

The development of diabetic retinopathy is primarily attributed to chronic hyperglycemia-induced damage to the retinal microvasculature. The pathogenic process is multifaceted, involving both metabolic and hemodynamic abnormalities that disrupt the delicate balance of the retinal neurovascular unit. Biochemical pathways activated by prolonged hyperglycemia include the polyol and hexosamine pathways, activation of protein kinase C, and accumulation of advanced glycation end-products (AGEs). These processes result in oxidative stress, mitochondrial dysfunction, inflammation, and eventual loss of pericytes and endothelial cells, leading to a compromised blood-retinal barrier (BRB). DR typically progresses from non-proliferative stages (NPDR), marked by microaneurysms, retinal hemorrhages, and venous abnormalities, to proliferative diabetic retinopathy (PDR), which is distinguished by neovascularization and fibrotic tissue growth that can cause vitreous hemorrhage and retinal detachment. Despite technological advancements in therapeutic options—such as laser photocoagulation, intravitreal anti-VEGF injections, and corticosteroids—early diagnosis and risk stratification remain vital for reducing visual impairment associated with DR [3–6].

Recent findings underscored the role of chronic inflammation in the pathogenesis of both T2DM and its vascular complications, including DR. Inflammatory mediators such as cytokines and chemokines contribute significantly to endothelial dysfunction, increased vascular permeability, and pathological neovascularization in the retina. Among these, Interleukin-18 (IL-18), a pro-inflammatory cytokine belonging to the IL-1 family, has gained attention for its pivotal role in immune responses and inflammation. IL-18 is produced by activated macrophages, dendritic cells, and epithelial cells and is activated through cleavage by caspase-1. It stimulates the release of interferon-gamma (IFN-γ), promotes Th1-type immune responses, and enhances endothelial adhesion molecule expression. Elevated IL-18 levels have been linked to metabolic syndrome, insulin resistance, and T2DM. Notably, increased concentrations of IL-18 have been detected in the vitreous humor of patients with diabetic retinopathy, particularly those with PDR, suggesting its involvement in retinal inflammation and neovascular pathology [7–9].

Address for Correspondence: Dr Hossam Khurshid Al Rahman Khan, Resident (JR -3), Department Of Ophthalmology, Era's Lucknow Medical College and Hospital, Lucknow

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Parallel to IL-18, asprosin is a novel glucogenic protein hormone that has attracted considerable interest in metabolic disease research. Asprosin is the C-terminal cleavage product of profibrillin, encoded by the FBN1 gene, and is primarily secreted by white adipose tissue. It functions by binding to the OLFR734 receptor on hepatocytes and activating the cAMP-PKA pathway, thereby stimulating hepatic glucose release during fasting states. Besides regulating glucose metabolism, asprosin has been implicated in the pathogenesis of obesity, insulin resistance, and chronic inflammation. Elevated circulating levels of asprosin have been observed in patients with T2DM and metabolic syndrome, showing strong correlations with insulin resistance indices such as HOMA-IR. Mechanistically, asprosin affects β-cell function, induces adipose tissue inflammation, and compromises vascular endothelial function. Although limited, emerging data suggest that asprosin may also play a role in diabetic microvascular complications such as DR, given its pro-inflammatory and glucoregulatory properties [10–12].

Given the combined influence of IL-18 and asprosin on inflammation and glucose metabolism, their potential roles in the pathophysiology of diabetic retinopathy warrant further investigation. Measuring and comparing the serum levels of these biomarkers in T2DM patients with and without DR could provide valuable insights into their diagnostic and prognostic significance. Identifying such biomarkers may facilitate a better understanding of the molecular distinctions between uncomplicated T2DM and those cases progressing to microvascular complications like DR. Furthermore, IL-18 and asprosin may hold potential as tools for disease monitoring or prediction of therapeutic response. The current study is thus designed to assess the serum concentrations of IL-18 and asprosin among T2DM patients with and without diabetic retinopathy. By analyzing the differences in these biomarkers and exploring their correlation with the severity of DR, the study aims to enhance our understanding of their possible utility in early detection, risk assessment, and development of personalized treatment strategies for diabetic retinopathy in the broader context of diabetes management [13,14].

The aim of the study was to compare the serum levels of Interleukin-18 (IL-18) and asprosin in subjects with diabetic retinopathy and those without. The objectives were to estimate and compare the serum concentrations of IL-18 and asprosin in diabetic patients with and without retinopathy, and to further analyze the correlation between the severity of diabetic retinopathy and the levels of these two biomarkers.

Materials and Methods

This prospective observational study was conducted at the Department of Ophthalmology, Department of Medicine, and the Department of Biotechnology, Era's Lucknow medical college & hospital, Lucknow from 2022-2025. Ethical approval has been obtained from the Ethical Approval Committee of Era's Lucknow medical college & hospital, Lucknow.

Study Population

The study included 100 patients diagnosed with Type 2 Diabetes Mellitus aged between 30 to 70 years, attending the outpatient or inpatient departments of Era's Lucknow Medical College & Hospital. Patients were divided into two groups: 50 patients with diabetic retinopathy and 50 without, confirmed through detailed fundoscopic examination and OCT. Exclusion criteria included other retinal diseases, inflammatory disorders, and systemic infections to eliminate potential confounders.

Data Analysis

Statistical analysis was conducted using SPSS software version 21. Quantitative data were expressed as mean ± standard deviation and compared using Student's t-test or ANOVA where applicable. Categorical variables were analyzed using the chi-square test. Pearson's correlation coefficient was used to evaluate the relationship between serum IL-18 and asprosin levels and DR severity. A p-value of <0.05 was considered statistically significant for all tests performed

Results

This collaborative study by the Departments of Ophthalmology, Medicine, and Biochemistry at Era's Lucknow Medical College & Hospital (ELMC\&H), Lucknow, aimed to compare serum levels of IL-18 and asprosin in patients with and without diabetic retinopathy and to assess the relationship between these biomarkers and the severity of retinopathy. Among the 122 participants, the majority (54.1%) were between 41–60 years of age, followed by 40.98% aged above 60 years, while only 4.92% were in the 18–40 years age group. Gender distribution showed a male predominance, with 57.3% males and 42.6% females included in the study.

Table 1: HBA1C value between Diabetic Retinopathy and Non Diabetic Retinopathy

Group (N)	Mean ± SD	p-value
No Diabetic Retinopathy (61)	6.56 ± 1.23	
Diabetic Retinopathy (61)	8.20 ± 1.42	<0.0001

The mean HbA1c level was significantly higher in patients with diabetic retinopathy (8.20 ± 1.42) compared to those without (6.56 ± 1.23), with a p-value < 0.0001, indicating a strong association between poor glycemic control and the presence of diabetic retinopathy.

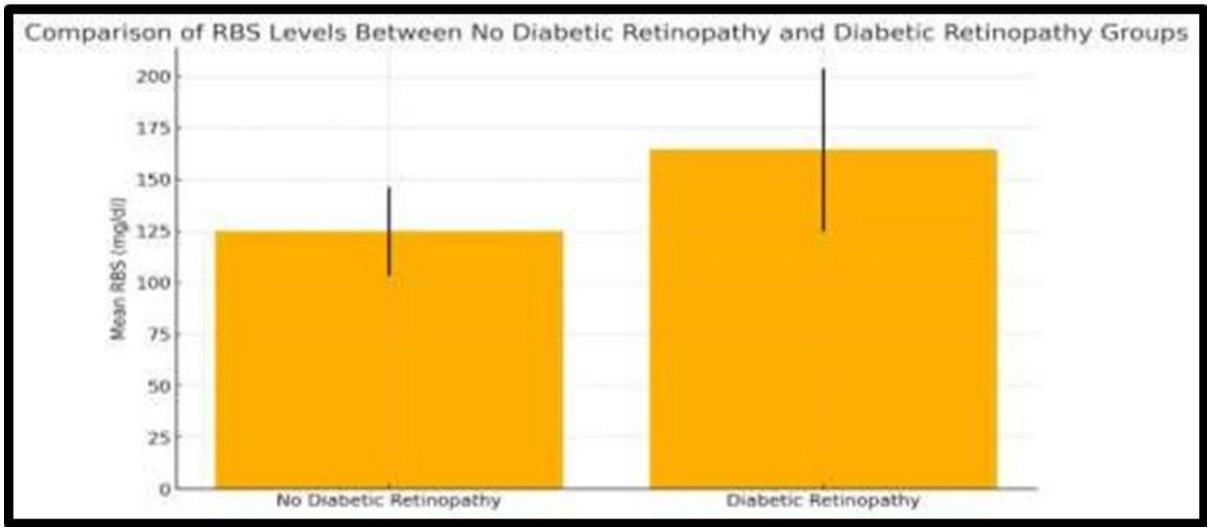

Figure 1: Comparison of RBS Levels between No Diabetic Retinopathy and Diabetic Retinopathy Groups

The mean RBS level was significantly higher in diabetic retinopathy patients (164.20 ± 39.36) compared to those without retinopathy (124.64 ± 21.43), with a p-value < 0.0001, indicating a strong link between elevated blood sugar and diabetic retinopathy.

Table 2: Comparison between Years of T2DM in No Diabetic Retinopathy and Diabetic Retinopathy Groups

Group (N)	Mean ± SD	p-value
No Diabetic Retinopathy (61)	6.95 ± 4.39	0.0002
Diabetic Retinopathy (61)	9.82 3.55	

The mean duration of T2DM was significantly longer in patients with diabetic retinopathy (9.82 ± 3.55 years) than in those without (6.95 ± 4.39 years), with a p-value of 0.0002, indicating that longer diabetes duration is strongly associated with retinopathy development.

Table 3: Comparison of Serum IL-18 Levels between No Diabetic Retinopathy and Diabetic Retinopathy Groups

Group (N)	Mean ± SD	p-value
No Diabetic Retinopathy (61)	658.73 ± 275.56	
Diabetic Retinopathy (61)	760.95 ± 238.35	0.03

Serum IL-18 levels were significantly higher in diabetic retinopathy patients (760.95 ± 238.35 pg/ml) compared to those without retinopathy (658.73 ± 275.56 pg/ml), with a p-value of 0.03, suggesting its potential role as a biomarker in diabetic retinopathy.

Table 4: Comparison of Serum Asprosin Levels between No Diabetic Retinopathy and Diabetic Retinopathy Groups

Group (N)	Mean ± SD	p-value
No Diabetic Retinopathy (61)	3.44 ± 2.09	
Diabetic Retinopathy (61)	3.29 2.07	0.004

Serum asprosin levels were slightly higher in diabetic retinopathy patients (3.66 ± 2.17 ng/ml) compared to those without retinopathy (3.57 ± 2.03 ng/ml), with a statistically significant p-value of 0.004, suggesting its potential utility as a biomarker in distinguishing diabetic retinopathy presence.

Table 5: Comparison of Serum IL-18 levels across different Retinopathy Grades

Marker	No DR (pg/ml)	Mild NPDR (pg/ml)	Moderate NPDR (pg/ml)	Severe NPDR (pg/ml)	PDR (pg/ml)	p-value
Serum IL-18	658.73 ± 275.56	789.16 ± 231.09	642.62 ± 211.88	824.05 ± 275.42	840.70 ± 194.84	0.04

Serum IL-18 levels showed a significant upward trend with increasing severity of diabetic retinopathy, rising from 658.73 ± 275.56 pg/ml in the no DR group to 840.70 ± 194.84 pg/ml in PDR, with a p-value of 0.04, suggesting IL-18 may serve as a marker of retinopathy progression.

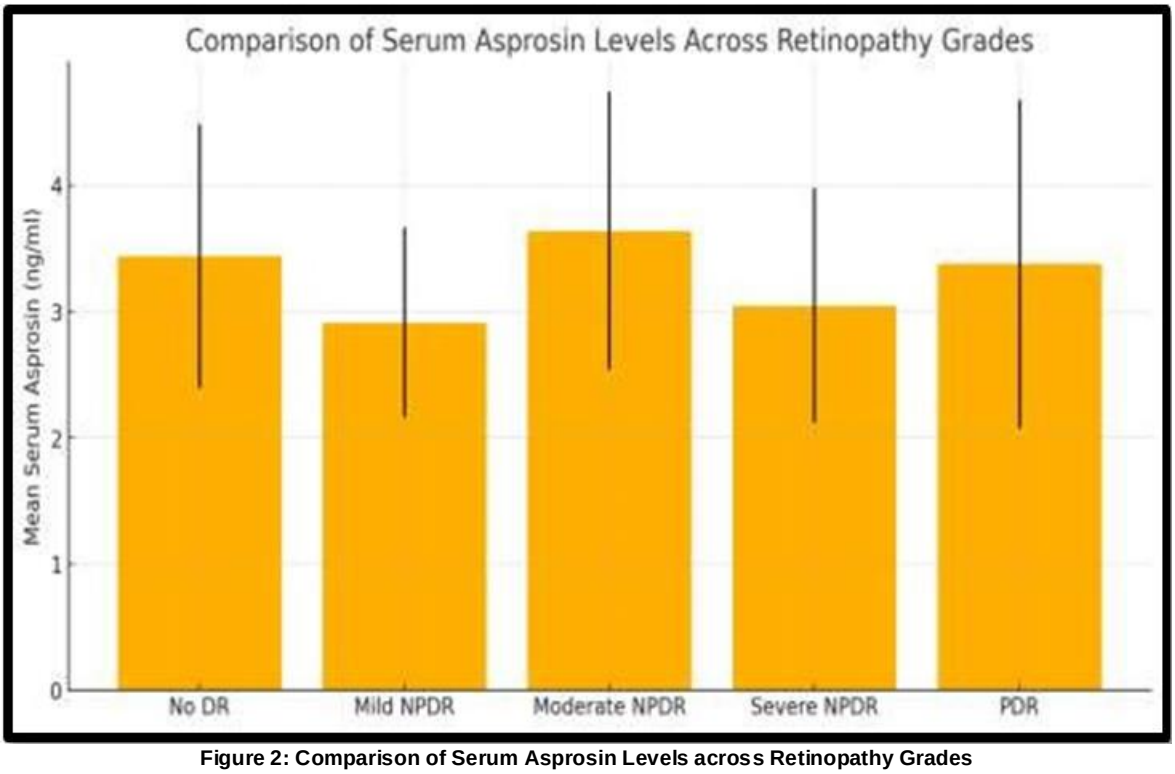

Figure 2: Comparison of Serum Asprosin Levels across Retinopathy Grades

Serum asprosin levels varied across diabetic retinopathy stages, with a significant p-value of 0.04, showing slight fluctuations but an overall association with disease severity, suggesting its potential as a biomarker for retinopathy progression.

In the analysis of serum IL-18 levels across different grades of diabetic retinopathy, half of the study participants (50%) had no clinical signs of retinopathy, representing the largest group. Among those with retinopathy, 16.39% had moderate NPDR, while severe NPDR and proliferative diabetic retinopathy (PDR) were each observed in 11.48% of patients. Mild NPDR was the least represented, comprising 10.66%

of the cohort. This distribution suggests that a considerable proportion of the population was in the early or preclinical stages of retinopathy, which is relevant when evaluating inflammatory markers like IL-18 across disease progression.

Table 6: Analysis of Serum IL-18 Levels Across Retinopathy Severity Grades

Retinopathy Grade	Number of Patients	Percentage (%)	Mean ± SD
No DR	61	50	658.73 ± 275.56
Mild NPDR	13	10.66	789.16 ± 231.09
Moderate NPDR	20	16.39	642.62 ± 211.88
Severe NPDR	14	11.48	824.05 ± 275.42
PDR	14	11.48	840.70 ± 194.84

Serum IL-18 levels progressively increased with retinopathy severity, peaking in the PDR group (840.70 ± 194.84 pg/ml), while the No DR group had the lowest levels (658.73 ± 275.56 pg/ml), indicating IL-18's potential role in monitoring diabetic retinopathy progression.
In the distribution of patients based on retinopathy grade and serum asprosin levels, half of the 122 individuals (50%) exhibited no signs of diabetic retinopathy. Among those with retinopathy, 10.66% had mild NPDR, 16.39% had moderate NPDR, and both severe NPDR and proliferative diabetic retinopathy (PDR) were each present in 11.48% of patients. This distribution offers a baseline overview of the prevalence of various stages of retinopathy in the study population and serves as a reference for interpreting serum asprosin concentrations in relation to disease severity.

Table 7: Analysis of Serum Asprosin Levels Across Retinopathy Severity Grades

Retinopathy Grade	Number of Patients	Percentage (%)	Mean ± SD
No DR	61	50	3.44 ± 2.09
Mild NPDR	13	10.66	2.91 ± 1.51
Moderate NPDR	20	16.39	3.64 ± 2.20
Severe NPDR	14	11.48	3.05 ± 1.86
PDR	14	11.48	3.38 Â± 2.60

Serum asprosin levels varied across retinopathy grades, peaking in moderate NPDR (3.64 ± 2.20 ng/ml) and lowest in mild NPDR (2.91 ± 1.51 ng/ml), showing no consistent trend but indicating possible stage-specific fluctuations in asprosin during diabetic retinopathy progression

Discussion

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by impaired insulin action and diminished insulin secretion. It poses a major global health concern, primarily due to lifestyle changes, sedentary behavior, unhealthy diets, and genetic susceptibility. The global burden of T2DM is rapidly increasing, especially in developing countries experiencing urbanization and dietary transitions. This condition leads to severe complications affecting both small and large blood vessels. Microvascular issues like diabetic retinopathy (DR), nephropathy, and neuropathy are common, along with macrovascular problems such as heart disease and stroke, all contributing to increased disability and premature death [15].
In 2019, 9.3% of the global population (463 million people) had diabetes, a number projected to reach 700 million by 2045. Urban areas and high-income nations show a higher prevalence, and about half of those affected remain undiagnosed, complicating disease management. Moreover, 374 million people had impaired glucose tolerance in 2019, indicating a high future diabetes risk. India, notably, had 77 million diabetes cases in 2019, expected to exceed 134 million by 2045, with over half undiagnosed, highlighting the urgency for early detection and intervention [16].
Epidemiological studies such as those by Saha SK, et. al; 2019 emphasize the influence of age, gender, lifestyle, and genetics on T2DM risk. In Nigeria, T2DM prevalence increased with age, with women more affected. In Bangladesh, specific mitochondrial DNA variants were linked to increased risk in males, illustrating both demographic and genetic factors in disease development. In our own cohort of 122 patients, 54.1% were aged 41–60 and 40.98% over 60, mirroring these trends. The male predominance (57.3%) further supports gender-linked vulnerability noted in previous studies [17].
Glycemic control remains central to DR management. Rahman MO, et. al; 2025 found that while HbA1c was not statistically linked to DR severity in their sample, insulin use and macular edema were significant predictors. Conversely, study showed that DR can still develop even when HbA1c is below 7%, due to glucose variability and other factors. Our data confirmed a strong association between HbA1c and DR severity (mean HbA1c: 8.20 ± 1.42 in DR vs. 6.56 ± 1.23 in non-DR, $p < 0.0001$), emphasizing tight glycemic control [18].
Kant D, et. al; 2022, linked poor glycemic markers to advanced DR, and study highlighted rural residence, hypertension, and prolonged diabetes duration as predictors of DR [19]. Our study also found significantly higher mean RBS in DR patients (164.20 ± 39.36) compared to those without DR (124.64 ± 21.43, $p < 0.0001$). Similarly, longer diabetes duration was evident in DR patients (9.82 ± 3.55 years vs. 6.95 ± 4.39 years, $p = 0.0002$), aligning with findings from Olafsdottir E, et. al; 2016, who showed duration and glycemic control are pivotal in DR development and progression [20].
Inflammatory cytokine IL-18 has gained attention for its role in DR. Jiang Y., et. al; 2023, confirmed higher IL-18 levels in DR patients across four countries (SMD = 3.41), while study emphasized early inflammatory changes like leukocyte adhesion and pyroptosis. Our study echoed these findings, with DR patients showing higher IL-18 (760.95 ± 238.35 pg/ml) compared to non-DR (658.73 ± 275.56 pg/ml, $p = 0.03$), suggesting IL-18 as a biomarker for inflammation-driven retinal damage [21].
Asprosin, a recently discovered fasting-induced glucogenic hormone, is also emerging as a potential marker. In this study found significantly elevated asprosin in DR patients, correlated with glycemic and insulin resistance markers. Goodarzi G, et. al; 2021 linked high asprosin levels to nephropathy and inflammation in T2DM. Our study also found a statistically significant difference in asprosin levels between DR and non-DR patients (3.29 ± 2.07 ng/ml vs. 3.44 ± 2.09 ng/ml, $p = 0.004$), despite the modest absolute difference [22].
Kovacs K, et. al; 2015, identified systemic cytokines like IL-8, IL-6, and VEGF-A as contributors to DR and diabetic macular edema, with stepwise increases in cytokine levels as disease severity progressed. Our findings showed IL-18 levels increased from no DR to PDR, with highest values in severe NPDR and PDR (840.70 ± 194.84 pg/ml), supporting IL-18's relevance in advanced disease [23].
Atta SE, et. al; 2024, reported that visfatin levels and lipid abnormalities were associated with DR severity, while Zhang H, et. al; 2020, showed asprosin's involvement in diabetic kidney disease. Similarly, our asprosin analysis showed fluctuating levels, peaking in moderate NPDR (3.64 ± 2.20 ng/ml) and decreasing slightly in advanced stages, indicating a non-linear expression pattern potentially linked to complex regulatory mechanisms [24,25].
Our findings support existing literature that inflammation and metabolic dysfunction—reflected by markers such as IL-18 and asprosin—play significant roles in DR pathogenesis. These biomarkers offer promising avenues for early detection, monitoring, and risk stratification in T2DM-related complications.

Conclusion

The results indicated that both serum IL-18 and asprosin are significantly associated with diabetic retinopathy (DR) in patients with type 2 diabetes mellitus. IL-18 levels were consistently elevated in DR patients and increased progressively with retinopathy severity, highlighting its potential as a biomarker for early detection and disease monitoring. Asprosin levels also differed significantly between DR and non-DR groups but showed a more variable trend across severity stages, suggesting a stage-dependent role. These findings emphasize the contribution of chronic inflammation to diabetic complications and support IL-18 as a promising target for early prediction and intervention.

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