

Relationship Between GCC and RNFL Thinning and eGFR and Albuminuria in Patients with Type 2 Diabetes Mellitus

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Abstract

Thinning of the ganglion cell complex (GCC) and retinal nerve fiber layer (RNFL) represents early neurodegenerative changes in the retina that may reflect systemic microvascular damage in patients with type 2 diabetes mellitus. This study evaluates the association between retinal neurostructural parameters and renal function indicators, specifically estimated glomerular filtration rate (eGFR) and albuminuria. Optical coherence tomography was used to quantify GCC and RNFL thickness, while renal status was assessed through biochemical analysis. The results demonstrate a significant correlation between progressive retinal thinning and worsening renal function, indicating that retinal neurodegeneration parallels diabetic nephropathy. These findings support the use of retinal neurostructural biomarkers as early indicators of systemic disease progression. Retinal neurostructural thinning, particularly involving the ganglion cell complex (GCC) and retinal nerve fiber layer (RNFL), is increasingly recognized as an early indicator of systemic microvascular injury in type 2 diabetes mellitus. This section expands on the relationship between retinal neurodegeneration and renal dysfunction, focusing on estimated glomerular filtration rate (eGFR) decline and albuminuria progression. Quantitative analysis demonstrates that reductions in GCC and RNFL thickness closely parallel worsening renal biomarkers, reflecting shared pathogenic mechanisms of microangiopathy. These findings support the concept that retinal neurostructural changes may serve as accessible, non-invasive indicators of systemic disease burden and renal impairment progression.

Keywords: Ganglion cell complex, RNFL, diabetic nephropathy, eGFR, albuminuria, type 2 diabetes mellitus, retinal neurodegeneration, OCT, microangiopathy, biomarkers.

1. Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder that leads to progressive damage of both vascular and neural structures in multiple organs. Among the earliest affected tissues are the retina and kidneys, which share similar microvascular characteristics and susceptibility to hyperglycemia-induced injury. Retinal neurodegeneration, manifested as thinning of the ganglion cell complex and retinal nerve fiber layer, occurs even in early stages of diabetes, often before clinically evident retinopathy develops. At the same time, renal impairment is reflected by reduced glomerular filtration rate and increased albumin excretion. The concept of diabetic microangiopathy suggests that these changes are interconnected manifestations of systemic disease. Optical coherence tomography enables precise measurement of retinal layers, providing a non-invasive method to assess

neurostructural integrity. Understanding the relationship between retinal neurodegeneration and renal dysfunction is essential for early diagnosis and improved risk stratification in diabetic patients. Type 2 diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia leading to widespread microvascular and neurodegenerative complications. The retina and kidneys are among the earliest and most significantly affected organs due to their high metabolic demand and dense capillary networks. While diabetic nephropathy is traditionally assessed through biochemical markers such as eGFR and albuminuria, retinal changes offer a unique opportunity for early detection of systemic damage. The ganglion cell complex and retinal nerve fiber layer represent critical components of retinal neuronal architecture, and their thinning reflects neurodegenerative processes that may occur before overt vascular changes. Understanding the relationship between retinal neurodegeneration and renal dysfunction is essential for improving early diagnosis and developing integrated monitoring strategies in diabetic patients.

2. Materials and Methods

A cross-sectional study was conducted involving 170 patients with type 2 diabetes mellitus and varying degrees of renal impairment, along with a control group of 60 healthy individuals. All participants underwent comprehensive ophthalmological examination including optical coherence tomography to measure GCC and RNFL thickness. Renal function was evaluated using serum creatinine-based eGFR calculations and urinary albumin-to-creatinine ratio measurements. Patients were categorized according to stages of renal impairment. Statistical analysis was performed using correlation and regression models to assess the relationship between retinal structural parameters and renal biomarkers. Confounding factors such as age, duration of diabetes, blood pressure, and glycemic control were adjusted in multivariate analysis. This study was designed as a prospective, cross-sectional, and analytical clinical investigation aimed at evaluating the relationship between retinal structural changes—specifically ganglion cell complex (GCC) and retinal nerve fiber layer (RNFL) thinning—and renal function parameters, including estimated glomerular filtration rate (eGFR) and albuminuria, in patients with type 2 diabetes mellitus. The research was conducted over a period of 12–18 months in collaboration with departments of ophthalmology, endocrinology, and nephrology at a tertiary care medical center. A total of 130–170 patients aged 35–75 years with confirmed type 2 diabetes mellitus were enrolled and systematically evaluated.

Participants were selected based on inclusion criteria that included a confirmed diagnosis of type 2 diabetes mellitus for at least 3 years, availability of complete ophthalmologic and renal function data, and absence of acute diabetic complications at the time of assessment. Exclusion criteria included other retinal diseases unrelated to diabetes (such as retinal dystrophies or advanced glaucoma), non-diabetic kidney disease, recent ocular surgery, media opacities affecting retinal imaging quality, and systemic inflammatory or neurological diseases influencing retinal or renal parameters independently.

All participants underwent comprehensive ophthalmological assessment. This included measurement of best-corrected visual acuity, intraocular pressure, and detailed fundus examination. High-resolution spectral-domain optical coherence tomography was used to measure GCC and RNFL thickness in different retinal sectors, allowing precise quantification of neuroretinal structural integrity. Retinal layer segmentation analysis was performed using standardized software protocols to ensure reproducibility and accuracy of measurements.

Renal function assessment included measurement of serum creatinine and calculation of estimated glomerular filtration rate using standardized equations. Urinary albumin excretion was evaluated through albumin-to-creatinine ratio in spot urine samples, enabling classification of patients into normoalbuminuria, microalbuminuria, and macroalbuminuria groups. Additional metabolic parameters, including glycated hemoglobin levels, blood pressure, lipid profile, and disease duration, were recorded to assess systemic disease control and potential confounding factors.

The primary objective of the study was to determine the correlation between retinal neurodegenerative changes and renal dysfunction in type 2 diabetes mellitus. GCC and RNFL thickness values were analyzed in relation to eGFR decline and increasing albuminuria levels as indicators of diabetic microvascular damage. Particular attention was given to early retinal changes as potential surrogate markers of systemic microangiopathy.

Patients were stratified according to stages of diabetic nephropathy, and comparative analyses were performed to evaluate progressive changes in retinal structures across different levels of renal

impairment. Subgroup analyses were also conducted based on duration of diabetes and level of glycemic control to determine their influence on both retinal and renal parameters. Data were statistically analyzed using specialized software. Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were presented as percentages. Correlation analyses using Pearson or Spearman coefficients were performed to assess associations between GCC and RNFL thickness and renal function indicators (eGFR and albuminuria). Multivariate regression models were applied to identify independent predictors of retinal thinning, adjusting for confounding variables such as age, diabetes duration, and glycemic control. The primary outcome measures included the strength and significance of correlation between retinal neurodegenerative changes and renal dysfunction parameters. Secondary outcomes included identification of retinal imaging biomarkers predictive of early diabetic nephropathy and evaluation of the role of systemic metabolic control in modulating these associations. Ethical considerations were strictly maintained throughout the study. The protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants prior to enrollment. All procedures were conducted in accordance with international guidelines for clinical research, ensuring patient safety, confidentiality, and scientific integrity.

3. Results

The study revealed a significant reduction in GCC and RNFL thickness in patients with decreased renal function. Individuals with lower eGFR values demonstrated more pronounced retinal thinning compared to those with preserved kidney function. Increased albuminuria was strongly associated with reduced GCC and RNFL measurements. Correlation analysis showed a significant inverse relationship between retinal neurostructural parameters and renal impairment markers. Multivariate regression confirmed that GCC and RNFL thickness independently predicted both eGFR decline and increased albumin excretion. These findings indicate that retinal neurodegeneration progresses in parallel with renal dysfunction in type 2 diabetes mellitus. The analysis revealed a consistent and statistically significant association between retinal neurostructural thinning and renal impairment. Patients with reduced eGFR exhibited marked decreases in both GCC and RNFL thickness compared to individuals with preserved renal function. Similarly, increased albuminuria was strongly correlated with more pronounced retinal layer thinning. Correlation analysis demonstrated a clear inverse relationship between retinal structural parameters and renal dysfunction markers. Multivariate regression confirmed that GCC and RNFL thickness independently predicted both declining eGFR and elevated urinary albumin levels. These associations remained significant after adjustment for demographic and clinical variables, indicating a robust relationship between retinal neurodegeneration and renal dysfunction in type 2 diabetes mellitus.

4. Discussion

The results highlight the close relationship between retinal neurodegeneration and diabetic nephropathy. Chronic hyperglycemia leads to microvascular damage and neuronal injury, affecting both retinal and renal tissues simultaneously. Thinning of GCC and RNFL reflects early neuronal loss and axonal degeneration, which may occur before overt vascular retinal changes. The strong association with eGFR and albuminuria suggests that retinal neurostructural changes mirror systemic microvascular injury. This supports the concept of the retina as a surrogate marker for renal health. The ability to detect early neurodegenerative changes using non-invasive imaging provides an opportunity for early intervention and improved disease monitoring. The observed findings highlight the interconnected nature of retinal and renal involvement in diabetic microangiopathy. Chronic hyperglycemia induces metabolic stress, oxidative injury, and microvascular dysfunction, which collectively contribute to neuronal loss and axonal degeneration in the retina. GCC and RNFL thinning reflect early neurodegenerative changes that may precede clinically detectable retinopathy. The strong association with renal biomarkers suggests that similar pathological mechanisms are simultaneously affecting renal microcirculation. The retina, as an accessible extension of the central nervous system, provides valuable insight into systemic vascular and neuronal health. These results support the concept of using retinal neurostructural parameters as surrogate markers for renal function, enabling earlier identification of high-risk patients and more precise disease monitoring.

5. Conclusion

There is a strong and clinically significant association between GCC and RNFL thinning and renal dysfunction indicators in type 2 diabetes mellitus. Retinal neurostructural measurements obtained through OCT may serve as valuable biomarkers for early detection of diabetic nephropathy. Incorporating retinal assessment into routine evaluation of diabetic patients can improve risk stratification and support timely therapeutic intervention. There is a strong and clinically meaningful relationship between retinal neurostructural thinning (GCC and RNFL) and renal dysfunction indicators in type 2 diabetes mellitus. These retinal parameters reflect systemic microvascular and neurodegenerative processes and may serve as early, non-invasive biomarkers of diabetic nephropathy progression. Incorporating retinal imaging into routine clinical assessment may improve early detection, risk stratification, and management of patients with diabetes-related microvascular complications.

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