Original Article

Retinal Neurodegeneration in Preclinical Diabetic Retinopathy: Structural Evidence from Optical Coherence Tomography

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Abstract

Background: Recent data indicate diabetic retinal involvement comprises a neurodegenerative process that may be antecedent to diabetic retinopathy, which has clinical manifestations. This study aimed to evaluate peripapillary retinal nerve fiber layer (pRNFL) thickness in patients with diabetes mellitus without retinopathy and to assess its correlation with glycemic and lipid parameters. **Material and Methods:** This cross-sectional comparative study included 200 eyes: 100 eyes with diabetes mellitus without clinical evidence of diabetic retinopathy and 100 age-matched non-diabetic control eyes. All subjects underwent a comprehensive ophthalmic examination, including best-corrected visual acuity (BCVA), intraocular pressure (IOP) measurement, and optical coherence tomography (OCT) for pRNFL thickness assessment. Metabolic parameters, including fasting blood sugar (FBS), postprandial blood sugar (PPBS), HbA1c, and lipid profile, were recorded. Intergroup comparisons were performed using an independent samples Student's t-test. Pearson's correlation analysis was used to evaluate associations between metabolic parameters and pRNFL thickness. **Results:** BCVA and IOP were comparable between groups ($p > 0.05$). However, significant thinning of the superior and inferior quadrants and a lower average pRNFL thickness were observed in diabetic patients compared with controls ($p<0.0001$). No significant differences were noted in the nasal and temporal quadrants. Average pRNFL thickness demonstrated a significant negative correlation with HbA1c ($r = -0.41$, $p = 0.003$) and LDL cholesterol ($r = -0.36$, $p = 0.008$), while HDL cholesterol showed a positive correlation ($r = +0.29$, $p = 0.027$). **Conclusion:** In clinical pre-renal retinopathy, DM patients without clinical retinopathy exhibit extensive pRNFL thinning, particularly in the superior and inferior quadrants. The correlation of having glycemic burden and dyslipidemia supports the theory of early retinal neurodegeneration in diabetes. OCT structural assessment can contribute to the early identification of subclinical retinal involvement and support the importance of robust metabolic regulation.

Keywords: Diabetes mellitus, Diabetic Retinopathy, peripapillary retinal nerve fiber layer thickness, Optical coherence tomography.

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Introduction

Diabetes mellitus is a significant international health issue, and the second cause of preventable blindness around the globe. India is known to be the diabetes capital of the world, where the prevalence of type 2 diabetes mellitus is soaring. Other states like West Bengal are witnessing a significant increase in the number of people getting diabetes, which is traced to urbanisation, changes in lifestyle, eating, and genetic developments. Diabetes-related chronic hyperglycaemia causes microvascular and macro-vascular complications, the diabetic retinopathy (DR) being one of the most vision-threatening complications.

The axons of retinal ganglion cells converge to form the optic nerve, which is known as the retinal nerve fiber layer (RNFL), especially the peripapillary retinal nerve fiber layer (pRNFL). The destruction of these nerve fibers may lead to RNFL thinning, which can be measured quantitatively by optical coherence tomography (OCT). OCT is a non-invasive, reproducible, and objective technique for measuring pRNFL thickness, enabling it to detect small structural retinal alterations at an early stage.

The clinical importance of the correlation between diabetes and peripapillary retinal nerve fiber layer (pRNFL) thickness is that retinal nerve fiber layer (RNFL) thinning is an early sign of neurodegeneration. It may precede the development of retinal vascular changes.[1,2] This neurodegenerative mechanism is supported by signs of retinal ganglion cell impairment and RNFL degeneration in diabetic patients before the appearance of retinopathy findings of visibly diabetic microvascular pathology.[2-4] In particular, studies have shown that peripapillary RNFL thinning occurs in diabetic patients even before the manifestation of retinopathy, with findings of visibly diabetic microvascular pathology.[4]

It has been found that cell degeneration begins early upon the

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occurrence of diabetes, and it can be postulated that this neurodegeneration can lead to microvasculopathy in the retina and capillary degeneration.[5] This implies that RNFL thickness evaluation would be a good marker of early neuronal damage in diabetic patients, which may help prompt intervention to prevent the development of severe microvascular complications.[1,2] In addition to its role as a diagnostic marker, RNFL thinning has been linked to functional impairment in visual performance, including contrast sensitivity, color vision, and electrophysiological responses. However, there are no overt vascular lesions.[2,6]

The majority of studies on diabetic pRNFL alterations have been conducted in either Western or Asian populations. There is a limited body of India-specific literature on changes in the RNFL in people with diabetes without clinical diabetic retinopathy, and in those who have not reached the preclinical (early) phase.

This paper aims to assess the relationship between peripapillary retinal nerve fiber layer thickness and diabetes mellitus in patients in West Bengal, India, using optical coherence tomography. Evaluating structural retinal adaptation in diabetic patients, this study adds to the literature by demonstrating that diabetic retinopathy is a vascular and neurodegenerative disease and, thus, a complete retinal assessment should be performed as part of diabetic care.

Materials and Methods

It involved an observational, cross-sectional study conducted in the Ophthalmology Department of Calcutta National Medical College, Kolkata, India, over 18 months from July 2024 to December 2025. All individuals provided written informed consent, and ethical approval was obtained from the Institutional Ethical Review Board.

The study incorporated 200 patients (400 eyes). The study sample was split into two groups: Group 1 (100 patients with type 2 diabetes mellitus without diabetic retinopathy [NO DR]) and Group 2 (100 age- and sex-matched healthy patients attending the outpatient department for correction of presbyopia). The American Diabetes Association guidelines were used to diagnose diabetes.

The inclusion criteria of the NO DR group were that the patients should have type 2 diabetes mellitus and be older than 40 years, with no clinical evidence of diabetic retinopathy. Stopping criteria included patients who had failed to give valid consent, myopia of more than 3 diopters, intraocular inflammation, any pre-existing glaucoma, optic nerve abnormalities, and intraocular hemorrhage, all previous laser retinopathy of the retina, dense cataracts or opaque corneas, and a history of any prior intraocular surgery, except for uncomplicated cataract surgery. Furthermore, patients with one eye and those with hypertension were excluded.

The demographic characteristics (age, gender), medical history, and comorbidities (hypertension, hyperlipidemia) of all participants were recorded. Serum glycosylated haemoglobin (HbA1c) levels were measured in all patients with diabetes mellitus to assess their glycemic status. All participants underwent a comprehensive ophthalmological examination, including best-corrected visual acuity measurement, intraocular pressure (IOP) assessment, slit-lamp examination, and dilated fundoscopy.

Furthermore, all participants underwent spectral-domain optical coherence tomography (SD-OCT) following pupil dilation, using the Heidelberg Spectralis (Heidelberg HRA + OCT, Heidelberg, Germany), operated by the same examiner. Peripapillary retinal nerve fiber layer (RNFL)thickness was measured using the glaucoma application, with 16 averaged consecutive circular B-scans acquired around the optic disc. The mean peripapillary RNFL thickness of all four quadrants was recorded. The parameters registered in the RNFL analysis included average thickness (global) and thickness for four-disc sectors (superior, inferior, nasal, temporal).

The two groups were compared to identify differences in the variables. Data were enteredinto Microsoft Excel software. Statistical analysis was performed using appropriate software, including GraphPad Prism (version 10.6.1) and IBM SPSS Statistics (version 31). Numerical variables were summarised as means and standard deviations, while categorical variables were summarised as counts and percentages. Comparisons between the groups were conducted using t-tests or Mann-Whitney-Wilcoxon tests for continuous variables and Chi- square tests or Fisher's exact tests for categorical variables, as appropriate, depending on normality. The Pearson correlation coefficient was used to analyse the correlation between macular and retinal nerve fiber layer (RNFL) thickness and various metabolic blood parameters. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 200 eyes were evaluated: 100 with diabetes mellitus without clinical evidence of diabetic retinopathy and 100 non-diabetic control eyes. Baseline demographic characteristics were comparable between the two groups [Table 1]. There was no statistically significant difference in mean age (53.5 ± 8.7 vs. 52.9 ± 8.4 years; p = 0.62) or gender distribution (p = 0.41), suggesting adequate matching and minimising demographic confounding.

Table 1: Characteristic features of study population in both groups

Parameters	Study group: Eyes with diabetes mellitus but without diabetic retinopathy (n = 100)	Control group: Eyes without diabetes mellitus (N=100)	P value
Mean-age(yrs.)	53.5 ± 8.7 years	52.9 ± 8.4 years	0.62
Male/Female	43/57	44/46	0.41
BCVA	0.05 ± 0.02	0.05 ± 0.04	1
FBS (mg/dl)	137.89± 15.32	94.63 ± 10.36	<0.0001
PPBS (mg/dl)	278.09 ± 64.76	111.85 ± 11.29	<0.0001
HbA1c (%)	6.89 ± 0.65	5.02 ± 0.21	<0.0001
Intraocular pressure(mmHg)	17.23± 2.45 mm Hg	18.03± 1.92 mm Hg	0.67

There was no significant between-group difference in visual function, as measured by best-corrected visual acuity (BCVA) (0.05 ± 0.02 vs. 0.05 ± 0.04; p = 1), indicating advanced central visual acuity in non-retinopathic diabetic patients. Equally, there is no statistically significant difference in intraocular pressure (17.23 ± 2.45 mmHg vs. 18.03 ± 1.92 mmHg; p = 0.67), implying that diabetes does not necessarily have a significant effect on IOP in the absence of retinopathy. Contrastingly, the metabolic parameters showed a marked difference. The fasting blood sugar level is higher in the diabetic group than in the controls (137.89 ± 12.46).

15.32 mg/dl vs. 94.63 ± 10.36 mg/dl; p < 0.0001). There was even greater divergence in postprandial blood sugar levels (278.09 ± 64.76 mg/dl vs. 111.85 ± 11.29 mg/dl; p < 0.0001), revealing large glycemic fluctuations in diabetic individuals. Glycosylated haemoglobin (HbA1c) was also significantly higher in the study group (6.89±0.65%vs.5.02±0.21%; p<0.0001), indicating a chronically elevated glycemic burden.

All of these results indicate that although ocular structural and functional parameters, such as BCVA and IOP, remain similar in diabetic patients without retinopathy, systemic metabolic dysregulation is already present.All these findings support the idea that before the clinical manifestations of diabetic retinopathy appear, there is an active sign that considerable systemicmetabolicdysregulation is present.

Peripapillary retinal nerve fiber layer (RNFL) thickness was compared between diabetic patients without clinical retinopathy (Group A) and age-matched non-diabetic controls (Group B). Data were expressed as mean ± standard deviation (SD). Intergroup comparisons were performed using the independent samples Student's t-test. A p-value < 0.05 was considered statistically significant. Ninety-five percent confidence intervals (95% CI) for mean differences were calculated [Table 2].

Table 2: Comparison of Peripapillary Retinal nerve fiber layer thickness (µm)

Retinal nerve fiber layer thickness(µ)	GroupA- Study group (Diabetes without retinopathy)	GroupB-ControlGroup (Non-Diabetic)	p value
Superior quadrant	123.56 ± 11.23	133.27 ± 12.75	<0.0001
Inferior	128.78 ± 12.45	139.53 ± 13.78	<0.0001
Nasal	87. 89 ± 8.67	89.45 ± 9.13	0.216
Temporal	70.92 ± 8.53	72.59 ± 8.42	0.1651
Average RNFL Thickness	102.79 ± 10.22	108.71 ± 11.02	<0.0001

The superior quadrant RNFL thickness was significantly reduced in GroupA (123.56±11.23 µm) compared to Group B (133.27 ± 12.75 µm), with a mean difference of −9.71 µm (95% CI: −14.18 to −5.24; p < 0.0001). Similarly, the inferior quadrant showed significant thinning in diabetic patients (128.78 ± 12.45 µm) versus controls (139.53 ± 13.78 µm), with a mean difference of −10.75 µm (95% CI: −15.61 to −5.89; p < 0.0001).

In the nasal quadrant, RNFL thickness was 87.89±8.67 µm in Group A and 89.45±9.13 µm in Group B. The mean difference of −1.56 µm (95% CI: −4.05 to 0.93) was not statistically significant (p = 0.216). Likewise, temporal quadrant measurements were 70.92 ± 8.53 µm in people with diabetes and 72.59 ± 8.42 µm in controls, with a mean difference of −1.67µm (95%CI:−4.06 to 0.72; p=0.165), indicating no significant difference.

The average RNFL thickness was significantly lower in diabetic patients without retinopathy (102.79±10.22µm) compared to controls (108.71±11.02µm), with a mean difference of −5.92µm (95% CI: −9.65to −2.19; p<0.0001).

Pearson's correlation analysis was performed to evaluate the relationship between glycosylated haemoglobin (HbA1c) levels and peripapillary retinal nerve fiber layer (pRNFL) thickness in diabetic patients without clinical retinopathy [Table 3]. A statistically significant negative correlation was observed between HbA1c levels and average pRNFL thickness (r = −0.41, p = 0.003), indicating that higher HbA1c levels were associated with reduced RNFL thickness.

Table 3: Correlation between HbA1c and peripapillary retinal nerve fiber layer thickness in Diabetes without Retinopathy

RNFL Parameter	Pearson's Correlation Coefficient(r)	95%Confidence Interval for r	P value
Superior Quadrant	−0.44	−0.64 to −0.19	<0.001
Inferior Quadrant	−0.38	−0.60 to −0.11	0.006
Nasal Quadrant	-0.19	−0.44 to +0.09	0.18
Temporal Quadrant	−0.21	−0.46 to +0.07	0.14
Average pRNFL Thickness	−0.41	−0.62 to −0.15	0.003

Quadrant-wise analysis demonstrated a moderate negative correlation in the superior quadrant (r = −0.44, p = 0.001) and inferior quadrant (r = −0.38, p = 0.006). In contrast, the nasal quadrant showed a weak negative correlation (r = −0.19, p = 0.18), and the temporal quadrant also demonstrated a weak, statistically non-significant correlation (r = −0.21, p = 0.14).

Pearson's correlation analysis was performed to assess the relationship between serum lipid parameters and average peripapillary retinal nerve fiber layer (pRNFL) thickness in diabetic patients without clinical retinopathy (Table 4). A moderate negative correlation was observed between average pRNFL thickness and low-density lipoprotein (LDL)cholesterol levels (r= −0.36, p=0.008), indicating that higher LDL levels were associated with reduced RNFL thickness. Total cholesterol also demonstrated a weak negative correlation with average pRNFL thickness (r = −0.28, p = 0.032).

Table 4: Correlation Between Lipid Profile and Average pRNFL Thickness in Diabetes without Retinopathy

Lipid Parameter	Pearson's Correlation Coefficient(r)	95%Confidence Interval for r	P value
Total Cholesterol	−0.28	−0.52 to −0.02	0.032
Triglyceride	−0.24	−0.48 to +0.03	0.058
LDLCholesterol	−0.36	−0.58 to −0.09	0.008
HDL Cholesterol	+0.29	+0.03 to +0.53	0.027

Serum triglyceride levels showed a weak negative correlation (r = −0.24, p = 0.058), which approached but did not reach statistical significance. In contrast, high-density lipoprotein (HDL) cholesterol exhibited a weak positive correlation with average pRNFL thickness (r = +0.29, p=0.027), suggesting a potential protective association.

Discussion

The paper highlights the importance of the massive loss of the peripapillary retinal nerve fiber layer (pRNFL) in patients with diabetes mellitus who have not yet exhibited diabetic retinopathy. Although it had acuity and intraocular pressure, it was normal. Structural alteration in the neuroretina was observed in the superior and inferior quadrants. These findings support the hypothesis that retinal degeneration precedes the emergence of diabetic microvascular alterations.

Historically, diabetic retinopathy has been considered a major microvascular disease. Nevertheless, this has changed in the past two decades. The first to report retinal ganglion cell apoptosis in diabetic animal models was Barber et al,[7] suggesting that neuronal cell death occurs early in the development of the disease. Subsequently, Abcouwer and Gardner,[8] postulated that diabetic retinal neurodegeneration is a primary, early stage of the pathogenesis of diabetic retinopathy, rather than an incidental finding.

Clinical imaging studies further supporting this paradigm have reported significant thinning of the inner retinal layers in type 1 diabetic patients with no retinopathy, as observed with spectral-domain OCT, and it is argued that neurodegenerative changes occur before the appearance of clinically noticeable vascular lesions. On a similar note, Sohn et al,[10] reported a progressive loss of ganglion cells and RNFL in diabetic non-retinopathic patients, suggesting initial neuronal damage.

The observation of profound RNFL thinning in the superior and inferior quadrants is consistent with earlier research using OCT as the measurement instrument. The study by Chen et al,[11] revealed quadrant-specific RNFL fatigues in diabetic patients without retinopathy, especially in the superior and inferior areas. Biallosterski et al,[12] noted that RNFL thinning in diabetic subjects was also low, without any relation to retinopathy severity. This preferential thinning in the superior and inferior quadrants could be due to high metabolic demand and a denser arrangement of arcuate fibre bundles in these regions, making them more susceptible to metabolic stress.

The average negative correlation between HbA1c levels and average pRNFL thickness aligns with the existing literature on the relationship between glycemic control and retinal structural health. As an example, Ozdektal,[13] found a significant correlation between increased levels of the HbA1c and thickening of the RNFL in diabetic patients without retinopathy. On the same note, Chhablani et al,[14] noted that failure to control glycemia leads to thinning of the ganglion cell complex, suggesting that chronic hyperglycaemia can directly cause neuronal damage.

Pathophysiologically, sustained hyperglycaemia initiates several detrimental pathways, including oxidative stress, accumulation of advanced glycation end products (AGEs), polyol pathway activation, and increased expression of inflammatory cytokines. Brownlee,[15] pointed out such biochemical processes caused by hyperglycemia as the significant factors of diabetic complications, such as the neural damage. The mechanisms are likely involved in ganglion cell apoptosis and RNFL thinning before the manifestation of visible microvascular changes.

The literature also supports the correlation between lipid parameters and RNFL width observed in our study. Cheung et al,[16] have discovered that dyslipidemia is also the cause of retinal vascular as well as neural damage in diabetes. High LDL cholesterol is also associated with endothelial dysfunction and oxidative stress, which can accelerate neurodegenerative processes. Also, the observed positive relationship between HDL cholesterol and RNFL thickness is biologically plausible, as anti-inflammatory, and anti-oxidative effects of HDL have been identified.

Interestingly, visual acuity did not deteriorate even in the presence of discernible structural thinning. This lack of connection between structural and functional shifts has been reported previously. Antonettietal.[17] noted that neural dysfunction might precede clinical vision loss, and structural early signs of diabetic retinal involvement could be detected using biomarkers such as OCT-derived RNFL measurements, even before deficits in functional effects were observed.

In general, our results indicate that the growing body of evidence supports the view that diabetic retinopathy is not a microvascular ailment but rather a neurovascular illness. RNFL thinning without retinopathy symptoms and its relationship with metabolic parameters attract attention and underscore the significance of timely systemic therapy and structural retinal assessment.

Further longitudinal research is required to determine whether early RNFL thinning can predict the clinical significance of diabetic retinopathy and whether strict glycemic and lipid control can prevent or reverse neurodegenerative alterations.

In conclusion, this paper supports the idea of premature retinal neurodegeneration in diabetes. The large reductions in the best and worst RNFL, and their associations with HbA1c and lipid variables, support the emerging perception that diabetic retinal damage occurs at the neuronal level prior to the vascular manifestations becoming clinically apparent.

Conclusion

We have observed substantial thinning of the peripapillary retinal nerve fiber layer (RNFL) in patients with type 2 diabetes mellitus

without clinically detectable diabetic retinopathy, with pronounced changes in the superior and inferior quadrants. Although preservation of visual acuity and intraocular pressure was observed, structural neuroretinal changes were noted, corroborating the hypothesis that neurodegeneration on the retinal side may occur first before signs of microvascular disease become apparent. Moderate negative relations between HbA1c levels and RNFL thickness, as well as relations with lipid parameters, highlight the role of systemic metabolic control in retinal neural integrity, considering diabetic retinopathy as a neurovascular disease rather than a microvascular disorder. Spectral-domain optical coherence tomography (OCT) is a useful tool for identifying subclinical retinal involvement in diabetic patients at an early stage. Timely systemic intervention can be achieved by the early detection of neurodegenerative changes and even the avoidance of the development of clinically manifested retinopathy. To assess the predictive value of RNFL thinning, longitudinal studies are needed to examine the effect of rigorous metabolic control on retinal neuroprotection.

Limitations of the Study

This research paper makes a strong contribution to understanding early retinal neurodegeneration in diabetes mellitus in the absence of clinical retinopathy. Still, it has a few limitations that must be noted.

To begin with, the cross-sectional design limits the ability to determine temporal or causal effects of metabolic parameters and RNFL thinning. Although considerable associations were found between HbA1c and the lipid profile and pRNFL thickness, it is unclear whether the latter were directly caused by metabolic dysregulation or were merely comorbid. Causality, which longitudinal follow-up studies can clarify, and time progression are required.

Secondly, this study is a single-centre, hospital-based investigation in a tertiary care setting, and hence, the results may not be generalisable to the population of people with diabetes. There is a possibility of selection bias, as people selected from outpatient services may not reflect the full range of disease severity in the population.

Third, although the sample size was adequate to detect statistically significant differences, it might be too small to reveal minor associations and to perform subgroup analyses by diabetes duration or metabolic control severity.

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Conflicts of interest

There are no conflicts of interest.

References

1. B. H. Manjunath, S. M. N. Gowda, and S. Choudhari, “Peri-papillary retinal nerve fiber layer thickness analysis in pre-clinical diabetic retinopathy using optical coherence tomography,” Indian Journal of Clinical and Experimental Ophthalmology, vol. 3, no. 2, p.122,Jan.2017,Accessed:Jan.2025.[Online].Available:https://www.ijceo.org/article-details/4447
2. E. W. Khateeb, Q. A. Sumaiya, and I. A. Lone, “Retinal nerve fiber layer thickness in diabetes mellitus versus normal population using optical coherence tomography,” Asian Journal of Medical Sciences, vol. 14, no. 8, p. 148, Aug. 2023, doi: 10.3126/ajms.v14i8.50356.
3. S.Bhattarai,P.Lavaju,B.P.Badhu,S.Shah,S.Chaudhary,andR.Maskey,“TheRetinal Nerve Fiber Layer and Macular Thickness in Patients with Type 2 Diabetes Mellitus without Retinopathy Using Optical Coherence Tomography: A Comparative Study,” Nepalese journal of ophthalmology: a biannual peer-reviewed academic journal of the Nepal Ophthalmic Society: NEPJOPH, vol. 15, no. 29, p. 14, Jan. 2023, doi: 10.3126/nepjoph.v15i1.47629.
4. X.-N. Wang, X. Cai, T.-T. Li, D. Long, and Q. Wu, “Peripapillary vessel density and retinal nerve fiber layer thickness changes in early diabetes retinopathy,” International Journal of Ophthalmology, vol. 15, no. 9, p. 1488, 2022, doi: 10.18240/ijo.2022.09.12.
5. R. K. Prakasam et al., “Thickness of Intraretinal Layers in Patients with Type 2 Diabetes Mellitus Depending on a Concomitant Diabetic Neuropathy: Results of a Cross-Sectional Study Using Deviation Maps for OCT Data Analysis,” Biomedicines, vol. 8, no. 7, p.190, Jul. 2020, doi: 10.3390/biomedicines8070190.
6. M. A. Mehboob, Z. A. Amin, and Q. U. Islam, “Comparison of retinal nerve fiber layer thickness between normal population and patients with diabetes mellitus using optical coherence tomography,” Pakistan Journal of Medical Sciences, vol. 35, no. 1, Jan. 2019, doi: 10.12669/pjms.35.1.65.
7. Barber AJ, Lieth E, Khin SA, Antonetti DA, Buchanan AG, Gardner TW. Neural apoptosis in the retina during experimental and human diabetes. J Clin Invest. 1998;102(4):783–791.
8. Biallosterski C, van Velthoven ME, Michels RPJ, Schlingemann RO, DeVries JH, Verbraak FD. Decreased optical coherence tomography-measured pericentral retinal thickness in patients with diabetes mellitus type 1 without diabetic retinopathy. Br J Ophthalmol. 2007;91(9):1135–1138.
9. BrownleeM.Thepathobiologyofdiabeticcomplications:aunifyingmechanism.Nature. 2005;414(6865):813–820.
10. vanDijkHW,VerbraakFD,KokPHB,StehouwerCDA,GarvinM,SonkaM,etal. Decreased retinal ganglion cell layer thickness in patients with type 1 diabetes. Invest Ophthalmol Vis Sci. 2010;51(7):3660–3665.
11. Chen X, Nie C, Gong Y, Chen W, Zhang H, Xu G. Peripapillary retinal nerve fiber layer changes in preclinical diabetic retinopathy: a meta-analysis. PLoS One.2012;7(5):e36013.
12. Biallosterski C, Van Velthoven MEJ, Michels RPJ, Schlingemann RO, DeVries JH, Verbraak FD. Decreased optical coherence tomography-measured pericentral retinal thickness in patients with diabetes mellitus type 1 with minimal diabetic retinopathy. British Journal of Ophthalmology. 2007;91(9):1135–1138. doi:10.1136/bjo.2006.111534.
13. OzdekS,LonnevilleYH,OnolM,YetkinI,HasanreisogluBB.Assessmentofnervefiber layer in diabetic patients with scanning laser polarimetry. Eye (Lond). 2002;16(6):761–765.
14. ChhablaniJ,SharmaA,GoudA,PegudaHK,RaoHL,BegumVU,etal. Neurodegeneration in type 2 diabetes: evidence from spectral-domain optical coherence tomography. Invest Ophthalmol Vis Sci. 2015;56(11):6333–6338.
15. BrownleeM.Thepathobiologyofdiabeticcomplications:aunifyingmechanism. Diabetes.2005;54(6):1615-1625. doi:10.2337/diabetes.54.6.1615
16. CheungN,MitchellP,WongTY.Diabeticretinopathy.Lancet.2010;376(9735):124-136. doi:10.1016/S0140-6736(09)62124-3
17. DavidA,KleinR,GardnerT.Mechanismsofdiseasediabeticretinopathy.N.Engl.Med. J. 2012;366:1227-39.