Journal of Research in Medical and Dental Science
2021, Volume 9, Issue 11, Page No: 80-85

Available Online at: www.jrmds.in
eISSN No.2347-2367: pISSN No.2347-2545

Retinal Nerve Fiber Layer Thickness in Nonarteritic Anterior Ischemic Optic Neuropathy in Diabetic Patients: From the Acute to Resolving Phases

Moutei Hassan[1*], Bennis Ahmed[1], Chraibi Fouad[1], Abdellaoui Meriem[1], Idriss Andaloussi Benatiya[1], Belahsen Mohammed Faouzi[2]

[1]Department of Ophthalmology, Hassan II University Hospital of Fez, Morocco

[2]Department Chief of Neurology, Hassan II University Hospital of Fez, Morocco

ABSTRACT

Purpose: To describe changes in RNFLp thickness over time in diabetic patients with Nonarteritic anterior ischemic optic neuropathy (NOIA-NA).

Materials and methods: This is a prospective observational study, conducted in the ophthalmology department at the University Hospital of Fez, between January 2018 and January 2020. Diabetic patients diagnosed with NOIA-NA underwent, at the time of diagnosis, at three and six months after the initial consultation, a complete ophthalmological examination, including assessment of best corrected visual acuity, a visual field (SITA 24-2 program, Humphrey-Zeiss Instruments), and papillary optical coherence tomography (TOPCON3D-OCT-Maestro).

Results: We included 80 eyes of 80 diabetic patients with NOIA-NA. At the baseline evaluation, the average best corrected visual acuity was 0.50 ± 0.28 logMAR, while after six months of follow-up, it improved to 0.401 ± 0.33 logMAR with a significant difference between the two visits. The average peripapillary nerve fibre layer thickness (RNFLp) in the affected eye was 161.91 ± 14.96 µm. This represents an average increase in RNFLp thickness of 70.75% compared to the RNFLp thickness of control eyes. The average percentage loss of RNFLp compared to control eyes was 32.6% (37.5 µm) at three months and 37.7% (42.3 µm) at six months, At the six-month visit, the percentage thinning of RNFLp for the superior, inferior, nasal, and temporal quadrants compared to the control eye was 44% (53.1 µm), 34% (48.4 µm), 21% (17.7 µm), and 31% (21.3 µm), respectively.

In the acute phase, there was no correlation between initial average RNFLp thickness and VA or initial mean deviation. However, RNFLp thickness was correlated with mean deviation at 3 months and 6 months (-0.646, p<0.001; 0.610, p<0.001, respectively). Similarly, RNFLp thickness was correlated with average visual acuity at 3 months and 6 months (0.556, p<0.001; -0.395, p<0.001; -0.533, p<0.001, respectively). Using regression analysis, it was found that for each micrometre of mean RNFL thickness lost, there was a 2.1 dB decrease in DM at 3 months and a 1.8 dB decrease at 6 months. Similarly, there was a decrease of one line of VA for every 6.2 µm of mean RNFLp thickness lost at 3 months and for every 2.2 µm at 6 months after onset.

Conclusion: The present study confirmed the capability of OCT for the diagnosis and monitoring of RNFLp changes after NOIA-NA in diabetic patients. The characteristics of papilledema as shown by OCT at baseline have limited prognostic value: Initial RNFLp thickness was not correlated with VA and DM. However, the significant correlation between RNFLp changes and visual field deficits and VA in the chronic phase represents an important structure-function relationship and underscores the importance of OCT in assessing the functional and structural course of eyes with NOIA-NA in diabetic patients.

Key words: Nonarteritic anterior ischemic optic neuropathy, Diabetes, Optical coherence tomography

HOW TO CITE THIS ARTICLE: Moutei Hassan, Bennis Ahmed, Chraibi Fouad, Abdellaoui Meriem, Idriss Andaloussi Benatiya, Belahsen Mohammed Faouzi, Retinal Nerve Fiber Layer Thickness in Nonarteritic Anterior Ischemic Optic Neuropathy in Diabetic Patients: From the Acute to Resolving Phases, J Res Med Dent Sci, 2021, 9(11): 80-85

Corresponding author: Moutei Hassan
E-mail ✉ :mouteihassan@yahoo.fr
Received: 09/08/2021
Accepted: 27/10/2021

INTRODUCTION

The emergence of diabetes and its complications is a worrying public health problem. In Morocco, complications from diabetes are major causes of blindness. In addition to diabetic retinopathy (RD) and diabetic macular edema, diabetes is an important risk factor for the development of non-arteritic anterior

ischemic optic neuropathy (NAON-NA). It can cause irreversible loss of vision and is one of the acquired vascular complications affecting the quality of life of diabetic patients.

The pathophysiology has been the subject of much controversy and is still debated. It is the result of circulatory insufficiency in the optic nerve head (ONH). However, the exact mechanism of the vasculopathy remains unknown [1].

Patients usually present with an acute, unilateral, painless visual decline that may progress over a few days. There is a relative afferent pupillary deficit homolateral to the decrease in visual acuity, indicating optic nerve damage. On the affected side of the eye, there is sectorial or diffuse papilledema, often accompanied by flaming peripapillary haemorrhages. Papilledema is the most obvious and important initial diagnostic sign of NOIA-NA. Papilledema disappears within a few weeks and is usually followed by papillary pallor.

Recent technologies for optic nerve exploration have attracted the interest of many researchers, as these diagnostic modalities can easily provide us with information about NOI-NA at any stage of the disease, including optical coherence tomography, which may be a useful tool in Neuro-ophthalmology, although its applications in this field have been more limited to date. Of the many reports that have studied patients with NOIA-NA, few have analyzed tomographic changes in RNFLp prospectively in patients with diabetes.

The current study analyzed tomographic changes in RNFLp in diabetic patients with NOIA-NA over 6 months. It was possible to determine the changes in RNFLp, compare them to control eyes, and investigate possible correlations with visual acuity and DM.

MATERIALS AND METHODS

This is a prospective observational study, conducted in the ophthalmology department at the University Hospital of Fez, between January 2018 and January 2020. The study adhered to the principles of the Declaration of Helsinki and was approved by the local ethics committee. Informed consent was obtained from all patients prior to inclusion.

The diagnosis of NOIA-NA was based on acute, painless visual impairment, papilledema in the fundus, and visual field deficit related to optic nerve damage. The diagnosis of diabetes in this study was based on available biological test results showing abnormal fasting blood glucose (>1.26 g/dl) and/or elevated glycated haemoglobin levels (>6.5%) and reports from the patient's endocrinologist.

However, we excluded:

- All patients with a previous diagnosis of Horton's disease or with evidence suggesting temporal arteritis, such as a history of scalp tenderness, a sedimentation rate ≥ 40 mm / h, or a positive C-reactive protein.
- Patients followed for glaucoma and patients with TNO coloboma or TNO drusen.
- All patients with vitreous or retinal pathologies that may affect central vision or cause visual field defects (e.g., vitreous haemorrhage), vitreous organization, retinal detachment, hereditary vitreoretinal or retinochoroidal diseases).

All patients underwent a complete ophthalmologic examination, including: measurement of best corrected visual acuity (BCVA), Goldmann applanation tonometry, and careful slit lamp examination (model BQ 900; Haag-Streit, Bern, Switzerland) of the anterior and posterior segments.

Patients benefited from the visual field with standard automated perimeter, SITA 24-2 program (Humphrey Field Analyser II, Humphrey-Zeiss Instruments, San Leandro, CA) and papillary optical coherence tomography (TOPCON3D-OCT-Maestro, Tokyo, Japan). The following procedures were used. We performed a circular scan of the papilla with a diameter of 3.46 mm cantered on the papilla. RNFLp thickness was measured around the entire papilla comprising four 90° quadrants. The scan was performed three times, and the clearest image with the strongest signal was selected. The RNFLp thickness at the four quadrants and the overall mean RNFLp thickness were automatically analyzed using the analysis software provided with the system.

Patients were monitored at 3 months and 6 months. At each follow-up visit, visual acuity measurement, papillary OCT, and CV were performed.

Changes in visual acuity, visual field, and RNFLp were assessed at 3 months and 6 months after the initial visit. CVAM was converted to log minimum angle of resolution (logMAR). RNFLp measurements in affected eyes were compared to those of a control group. Because NOIA-NA patients have optic discs at risk [2,3] and because optic disc size is a factor that can influence RNFLp analysis [4], we used unaffected eyes of the same patients as controls. Patients who had a previous episode of NOIA-NA in the other eye were excluded from the analysis.

All statistical analyses were performed using SPSS statistical software (version 18.0; SPSS, Inc., Chicago, IL, USA). Student's t-test was used to compare quantitative variables between affected and control eyes. Pearson correlation was performed to assess the linear correlation between variables (RNFLp, mean visual field deviation, and visual acuity). Statistical significance was defined as $P < 0.05$.

RESULTS

We included 80 eyes of 80 diabetic patients with NOIA-NA satisfying our inclusion criteria. (Mean age was 60.78 ± 6.88 years with extremes ranging from 40 to 75 years, sex ratio F/H= 1.28.) During the follow-up period, 75 patients had at least three months of follow-up and 71 patients were followed for six months. At the initial consultation, the best mean corrected visual acuity was 0.50 ± 0.28 logMAR, while after six months of follow-up,

it improved to 0.401 ± 0.33 logMAR with a significant difference between the two consultations. The mean change in visual acuity from the initial visit to the last visit was 0.11 logMAR (Table 1).

The visual field abnormalities at initial presentation were mainly altitudinal deficits that involved primarily the inferior hemifield in 50% of cases, followed by diffuse deficits in 38.8% of cases and superior altitudinal deficits in 11.3% of cases. The mean deviation varied from -20.45 to -9.01 dB with a mean ± standard deviation of -14.23 ± 2.47 db. The mean deviation in the inferior hemifield was -8.95 ± 3.18 dB greater than the mean deviation in the superior hemifield -5.28 ± 4.09 db. A worsening of 2 decibels (dB) or more was found in 15.49% of the cases, an improvement of 2 dB or more in 26.76% of the cases, and it was stable in 57.74% at the last control (Table 1).

Table 1: The evolution of the VA, the mean deviation at the initial consultation, after 3 months and after 6 months.

	Initial visit	After 3 months	After 6 months	Pa	Pb
MAVC (LogMAR)	0.50 ± 0.28	0.403 ± 0.29	0.401 ± 0.33	0.001	0.767
mean deviation (MD)	-14.23 ± 2.47	-14.60 ± 3.04	-14.90 ± 3.74	0.05	0.372
MD in Superior	-5.28 ± 4.09	-5.09 ± 4.45	-5.17 ± 4.33	0.347	0.24
MD in inferior	-8.95 ± 3.18	-9.50 ± 3.52	-9.06 ± 2.84	0.047	0.113
Visual field deficit					
IAD	40(50%)	39(48.8%)	29(36.3%)		
SAD	9(11.3%)	9(11.3%)	7(8.8%)		
Diffuse deficit	31(38.8%)	27(33.8%)	35(43.8%)		
CVMA: Best corrected visual acuity; MD: Mean deviation					
IAD: Inferior altitudinal deficit					
SAD: Superior altitudinal deficit					
Pa: Comparison between the results of the initial consultation and after 3 months					
Pb: Comparison between the results of the consultation after 3 months and after 6 months					

OCT examination revealed that the mean RNFLp thickness was 161.91 ± 14.96 µm in the affected eyes and 94.82 ± 8.72 µm in the control eyes. This represents an average increase in RNFLp thickness of 70.75% compared to the RNFLp thickness in control eyes (Table 2). At controls, the mean RNFLp thickness was 75.79 ± 12.44 µm at three months and 74.77 ± 13.10 µm at six months. The mean percentage of RNFLp loss compared to control eyes was 32.6% (37.5 µm) at three months and 37.7% (42.3 µm) at six months, ranging from a low of 15% to a high of 72%. At the six-month visit, the percentage thinning of the RNFLp for the superior, inferior, nasal, and temporal quadrants compared to the control eye was 44% (53.1 µm), 34% (48.4 µm), 21% (17.7 µm), and 31% (21.3 µm), respectively. Of note, the decrease in RNFLp thickness in the superior quadrant was statistically higher than that in the temporal and nasal quadrants (P 0.01 and P 0.001, respectively).

Table 2: The evolution of RNFLp at the initial visit, after 3 months, and after 6 months in the affected eyes.

	Initial visit	After 3 months	After 6 months	Pa	Pb
RNFLp:					
Average RNFLp	161.91 ± 14.96	75.79 ± 12.44	74.77 ± 13.10	<0.001	0.62
Superior Quadrant	197.89 ± 18.49	80.01 ± 15.99	78.77 ± 16.15	<0.001	0.03
Inferior Quadrant	199.08 ± 17.99	90.93 ± 15.18	90.11 ± 17.61	<0.001	0.292
Nasal Quadrant	135 ± 16.68	68.81 ± 17.29	67.49 ± 17.02	<0.001	0.355
Temporal Quadrant	114.91 ± 14.05	63.40 ± 13.87	62.72 ± 14.45	0.002	0.476
RNFLp: the papillary fiber layer					
Pa: Comparison between the results of the initial consultation and after 3 months					
Pb: Comparison between the results of the consultation after 3 months and after 6 months					

In the affected eye, there was no correlation between initial mean RNFLp thickness and VA or initial mean deviation. However, RNFLp thickness was correlated with mean deviation at 3 months and 6 months (0.646,

p<0.001; 0.610, p<0.001, respectively). Similarly, RNFLp thickness correlated with mean visual acuity at 3 months and 6 months (-0.395, p<0.001; -0.533, p<0.001, respectively) (Table 3). Using regression analysis, it was found that for every micrometre of mean RNFLp thickness lost, there was a 2.1 dB decrease in DM at 3 months and a 1.8 dB decrease at 6 months. Similarly, there was a decrease of one line of VA for each 6.2 µm loss of mean RNFLp thickness at 3 months and each 2.2 µm loss at 6 months after onset.

Table 3: The correlation between RNFLp and VA (logMAR) and the mean deviation at different consultations.

	RNFLp		
	Initial visit	**After 3 months**	**After 6 months**
Visual acuity (LogMAR)	0.556	-0.395**	-0.533**
MD (decibels)	-0.694	0.646**	0.610**
RNFLp: The peripapillary fiber layer			
P*<0.05			
P**<0.001			

DISCUSSION

Diabetes is expanding significantly worldwide. In Morocco, complications of diabetes represent major causes of blindness. In addition to diabetic retinopathy and diabetic macular edema, diabetes is an important risk factor for the development of non-arteritic anterior ischemic optic neuropathy. NOIA-NA can cause irreversible vision loss and is one of the acquired vascular complications affecting the quality of life of diabetic patients. This emphasizes the need to take the issue seriously. The prevalence remains high in poorer populations and developing countries, due mainly to a lack of resources. It usually occurs in patients over 50 years of age, with an estimated annual incidence of 2.3 to 10.3 per 100,000 people in the United States [5]. Recent technologies for optic nerve exploration have attracted the interest of many investigators, as these diagnostic modalities can easily provide us with information about NOI-NA at any stage of the disease-in particular, optical coherence tomography, which can be a useful tool in Neuro-ophthalmology, although its applications in this field have been more limited until now. Hence, our study aimed to describe changes in RNFLp thickness over time in diabetic patients with NOIA-NA.

The mean age and gender distribution of patients included in this study were similar to those of other studies published in the literature [6]. The information on visual acuity at the first visit showed that 57.6% of the patients had an initial visual acuity better than 0.5 logMAR. This result is comparable to the results of the case-control study by Sharma et al., in which 63.3% of diabetic patients had a VA greater than 0.5 logMAR [7]. After the initial assessment, the VA was stable in 53.8% of cases at the last control. This finding was described in the optic nerve sheath decompression study, in which the VA was stable in 44.9% of patients [8].

In NOIA-NA, visual field damage is an important clinical criterion for the diagnosis and determination of the extent of visual loss. In brief, eyes may show a variety of visual field deficits related to the type of optic nerve injury. Previous studies have described the patterns of visual field deficits in NOIA-NA. Inferior hemifield deficits were the most common, ranging from 36% to 52% [9]. Therefore, our results are consistent with the different patterns of visual field deficits observed at the initial visit in diabetic patients with NOIA-NA. Fifty percent of the patients showed an inferior altitudinal deficit, in which the mean deviation in the inferior hemifield was more impaired than the mean deviation in the superior hemifield. It is noteworthy that the visual field at 3 months was stable in 68% of cases, improved in 16% of cases, and worsened in 16% of cases, whereas the corresponding figures in the Cullen series were 77%, 15%, and 7.5%, respectively [10]. However, no significant change in mean deviation was observed between the 3-month and 6-month visits (P=0.372). From these results, we deduce that eyes with NOIA-NA in diabetic patients may show a change in visual function up to about 3 months from the initial visit, but not thereafter.

In this study, we found that OCT was able to detect papilledema in the acute stage of the pathology. The mean thickness of the RNFLp was 161.91 ± 14.96 µm in the affected eyes. The RNFLp in the acute phase had almost doubled in thickness compared to control eyes. The mean RNFLp thickness value of 161.91 ± 14.96 µm was similar to the values found by Savini et al. [11] and Contreras et al. [12]. Progressively, papilledema had regressed to be followed later by pallor and papillary atrophy. At the three-month visit, the mean RNFLp had decreased by 32.6% compared to control eyes. Thereafter, the loss of RNFLp stabilized with an additional decrease of only 5.1% at the 6-month visit. This percentage loss of RNFLp was similar to that after an episode of NOIA-NA in non-diabetic patients [12]. However, it was greater than this after an episode of optic neuritis, which seems logical given that NOIA-NA is generally a much more aggressive pathology compared to the favourable visual prognosis of optic neuritis [13]. After the resolution of papilledema, the loss of RNFLp was less pronounced in the nasal quadrant. However, the superior quadrant showed a higher percentage of RNFLp loss. This was consistent with the high rate of visual field deficits in the inferior hemifield (36.3% of patients at the

end of follow-up). Deleón-Ortega et al. evaluated patients with good visual acuity after NOIA-NA, performing OCT and CV once the papilledema resolved. They objectified a statistically significant decrease in mean RNFLp thickness in 15 patients with altitudinal deficits compared to normal control eyes (74.8 vs. 109.5 m, P 0.001). All quadrants except the temporal quadrant showed statistically significant thinning compared to normal eyes. The authors suggested that this might be due to the good VA of all patients [14]. Indeed, in our study, among the patients who showed poor VA and severe CV deficits, the RNFLp thickness of the temporal quadrant was almost 40% less than that of the control eyes. Thus, it seems plausible that temporal quadrant assignment is a good clinical indicator of central vision.

Optical coherence tomography has been widely used in glaucoma patients to diagnose and monitor RNFLp loss. RNFLp loss has been shown to correlate with DM in patients with ocular hypertonia and glaucoma [15]. One study found that a 9.3 µm loss of global RNFLp results in a 5 dB decrease in CV DM [16]. In our study, the mean RNFLp thickness was correlated with DM from the third month. We found that DM decreased by 2.16 dB for each micrometre lost in RNFLp thickness. Two recent studies also found a correlation between RNFLp thinning and DM [14,17]. In contrast, the relationship between RNFLp thickness and DM from the third month onwards may not be detected earlier due to the learning effect of the patients. Most of the individuals who presented with this NOIA-NA were elderly individuals who had never had a CV before. Thus, this artifact may prevent the detection of a relationship between RNFLp thickness and VA.

Recent studies have sought to characterize the relationship between RNFLp and visual loss in other optic nerve disorders. Trip et al. found that after an episode of optic neuritis, RNFLp decreased by 27% compared to the other eye [13]. Fisher et al. reported that after optic neuritis, a 4 µm decrease in RNFLp led to a one-line decrease in VA [18]. Our study also demonstrated that RNFLp thickness in the affected eye was significantly correlated with VA from the third month onward, with a one-line decrease in VA for every 7 µm loss in mean RNFLp thickness after the acute phase. Compared to the correlations between VA and RNFLp described for optic neuritis mentioned above [13,19], this represents a greater functional impairment for a given level of RNFLp loss. However, there is no clear explanation for the existence of this significant functional impairment in patients with NOIA-NA as compared to patients with optic neuritis. This may be due to the greater damage to temporal fibers in NOIA-NA, particularly the papillomacular bundle that carries visual signals for the central CV. This hypothesis also explains why, in Vigabatrin toxicity, the VA remains normal despite a large loss of RNFLp, as the temporal quadrant is rarely affected. These results support a possible correlation between mean temporal RNFLp thickness and final visual acuity.

We believe that our data provide an approach to understanding the changes in RNFLp in diabetic patients with NOIA-NA in the acute and chronic phases. However, some limitations of our study are worth mentioning. The presence of papilledema with superimposed peripapillary haemorrhages may produce a blocking artifact that affects the quantification of the tomographic signal in the sample region. In addition, severe visual impairment may limit fixation and, in turn, degrade image and acquisition quality.

CONCLUSION

The present study confirmed the capability of OCT for the diagnosis and monitoring of RNFLp changes after NOIA-NA in diabetic patients. The characteristics of papilledema as shown by OCT at baseline have limited prognostic value: Initial RNFLp thickness was not correlated with VA and DM. However, the significant correlation between RNFLp changes and visual field deficits and VA in the chronic phase represents an important structure-function relationship and underscores the importance of OCT in assessing the functional and structural course of eyes with NOIA-NA in diabetic patients.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no relationship with this article.

REFERENCES

1. Arnold AC. Pathogenesis of nonarteritic anterior ischemic optic neuropathy. J Neuroophthalmol 2003; 23:157–63.
2. Beck RW, Savino PJ, Repka MX, et al. Optic disc structure in anterior ischemic optic neuropathy. Ophthalmology 1984; 91:1334–7.
3. Burde RM. Optic disk risk factors for nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol 1993; 116:759–64.
4. Savini G, Zanini M, Carelli V, et al. Correlation between retinal nerve fibre layer thickness and optic nerve head size: an optical coherence tomography study. Br J Ophthalmol 2005; 89:489–92.
5. Johnson LN, Arnold AC. Incidence of nonarteritic and arteritic anterior ischemic optic neuropathy. Population-based study in the state of Missouri and Los Angeles County, California. J Neuroophthalmol 1994; 14:38–44.
6. Hayreh SS, Zimmerman MB. Nonarteritic anterior ischemic optic neuropathy: Clinical characteristics in diabetic patients versus nondiabetic patients. Ophthalmology 2008; 115:1818–25.
7. Sharma S, Kwan S, Fallano KA, et al. Comparison of Visual outcomes of nonarteritic anterior ischemic optic neuropathy in patients with and without diabetes mellitus. Ophthalmology 2017; 124:450–5.

8. Ischemic Optic Neuropathy Decompression Trial Study Group. Characteristics of patients with nonarteritic anterior ischemic optic neuropathy eligible for the ischemic optic neuropathy decompression trial. Arch ophthalmol 1996; 114:1366-74.
9. Hayreh SS, Zimmerman B. Visual field abnormalities in nonarteritic anterior ischemic optic neuropathy: their pattern and prevalence at initial examination. Arch Ophthalmol 2005; 123:1554–62.
10. Cullen JF, Chung SHR. Non-arteritic anterior ischaemic optic neuropathy (NA-AION): outcome for visual acuity and visual field defects, the Singapore scene 2. Singapore Med J 2012; 53:88–90.
11. Savini G, Bellusci C, Carbonelli M, et al. Detection and quantification of retinal nerve fiber layer thickness in optic disc edema using stratus OCT. Arch Ophthalmol 2006; 124:1111–7.
12. Contreras I, Noval S, Rebolleda G, et al. Follow-up of nonarteritic anterior ischemic optic neuropathy with optical coherence tomography. Ophthalmology 2007; 114:2338-2344.
13. Trip SA, Schlottmann PG, Jones SJ, et al. Retinal nerve fiber layer axonal loss and visual dysfunction in optic neuritis. Ann Neurol 2005; 58:383–91.
14. Deleón-Ortega J, Carroll KE, Arthur SN, et al. Correlations between retinal nerve fiber layer and visual field in eyes with nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol 2007; 143:288–94.
15. Zangwill LM, Williams J, Berry CC, et al. A comparison of optical coherence tomography and retinal nerve fiber layer photography for detection of nerve fiber layer damage in glaucoma. Ophthalmology 2000; 107:1309–15.
16. Kanamori A, Nakamura M, Escano MFT, et al. Evaluation of the glaucomatous damage on retinal nerve fiber layer thickness measured by optical coherence tomography. Am J Ophthalmol 2003; 135:513–20.
17. Danesh-Meyer HV, Carroll SC, Ku JYF, et al. Correlation of retinal nerve fiber layer measured by scanning laser polarimeter to visual field in ischemic optic neuropathy. Arch Ophthalmol 2006; 124:1720–1726.
18. Fisher JB, Jacobs DA, Markowitz CE, et al. Relation of visual function to retinal nerve fiber layer thickness in multiple sclerosis. Ophthalmology 2006; 113:324–32.
19. Noval S, Contreras I, Rebolleda G, et al. Optical coherence tomography versus automated perimetry for follow-up of optic neuritis. Acta Ophthalmol Scand 2006; 84:790–794.