|| Print ISSN: 2589-7837 || Online ISSN: 2581-3935 ||
International Journal of Medical Science and Diagnosis Research (IJMSDR)
Available Online at www.ijmsdr.com
PubMed (National Library of Medicine ID: 101738824)
Volume 3, Issue 4; April: 2019; Page No. 15-23
Original Research Article
crossref DOI: https://dx.doi.org/10.32553/IJMSDR/v3i4.4

TO STUDY THE EFFECT OF INTRAVITREAL TRIAMCINOLONE ACETONIDE IN CASES OF NON ARTERITIC ANTERIOR ISCHEMIC OPTIC NEUROPATHY.

Dr. J. L GOYAL[1], Dr. Narendra Meel[2], Dr. Ritu Arora[3], Dr. Prateeksha Sharma[4]

[1,3]Director Professor, [2]Junior Resident, Senior Resident[4]

Guru Nanak Eye Centre[1,2,3,4]

Conflicts of Interest: Nil

Corresponding author: Dr. Prateeksha Sharma

Abstract:

Purpose: To study the effect of intravitreal Triamcinolone Acetonide in cases of Non Arteritic Anterior Ischemic Optic Neuropathy (NAION).

Method: Prospective interventional study included fifteen patients with unilateral acute NAION who received 2 mg of intravitreal triamcinolone acetonide and were followed up for 12 weeks.

Result: Disc edema was replaced by pallor within six weeks. Visual Acuity improved from log MAR 1.48 to log MAR 0.97 at 12 weeks along with significant improvement in latency and amplitude of pattern visual evoked potentials. Visual fields, colour vision, contrast sensitivity showed incomplete recovery. Pattern Electroretinogram showed reduction of N95 amplitude (4.183 to 3.377uV) along Retinal nerve fiber layer loss (RNFL) (135.13 to 64.8 um).Visual acuity at 12 weeks showed significant correlation only to the amplitude of pattern electroretinogram.

Conclusion: Since acute NAION is associated with significant ganglion cell loss. Study recommend complete evaluation for visual function in all cases of NAION in affected and unaffected eye which includes, visual acuity, colour vision, contrast senstivity, visual fields, electrophysiology and Optical Coherence tomography RNFL, Ganglion cell complex and Macular volume. It signifies the use of these parameters in neuroprotective trials to monitor the efficacy of therapies aimed at nerve protection. It also emphasise the role of IVTA in treatment of acute NAION presenting as early as 2 weeks.

Keywords: NAION, Pattern ERG, Triamcinolone Acetonide, Intravitreal

Introduction

Non arteritic anterior ischemic optic neuropathy (NAION) is the most common cause of optic nerve ischemic disease in middle aged and older populations. It is characterized by sudden, painless, monocular visual loss associated with pallid swelling of the optic disc. NAION is caused by occlusion of the short posterior ciliary artery resulting in partial or total infarction of the optic nerve head [1,2]. Objectively, electrophysiology reveals an impairment of Visual evoked potentials (VEP) with reduction in P100 amplitude along with Pattern electroretinogram (PERG) showing an affected N95 component in cases of NAION [3-5]. Thinning of retinal nerve fiber layer (RNFL) thickness due to ganglion cell loss is seen on optical coherence tomography (OCT) in these cases [6-8].

Various treatment modalities have been tried, management of NAION remains controversial [9]. Single Intravitreal triamcinolone acetonide injection (IVTA) has been tried to improve visual outcomes in patients of NAION [10-14]. It has been suggested that a rapid reduction of optic disc edema by IVTA could prevent further tissue

damage by stopping the vicious cycle of ischemia, edema, and compartment syndrome occurring in these cases [12] however, studies on the efficacy of IVTA show conflicting results [10,12,15]. Evaluation of electrophysiological parameters as well as anatomical changes in RNFL and ganglion cell complex (GCC) in patients of NAION undergoing treatment with IVTA has rarely been studied previously. Thus, a comprehensive longitudinal study to evaluate effectiveness of IVTA in causing visual recovery with special focus on electrophysiological and anatomical changes in acute cases of NAION is needed and has been studied by means of this study.

Method

Ethics approval for this study was obtained from Maulana Azad Medical College and associate Hospital Ethics Committee.

It was a prospective interventional study conducted from September 2016 to march 2017 at Guru Nanak Eye Centre associated with Maulana Azad Medical College a tertiary care hospital in New Delhi, India, including a total of 15 patients age 35 to 70 years presenting within first two weeks of unilateral acute NAION.After taking relevant history, each patient underwent a comprehensive systemic and ophthalmological examination including complete general physical examination, visual acuity using Snellen's chart (converted to log MAR values for ease of statistical analysis), fundus evaluation, Slit lamp examination, colour vision testing using Ishihara color plates, contrast sensitivity testing using Pelli-Robson chart, visual field analysis using Humphrey field analyzer (Swedish Interactive Threshold Algorithm SITA central 30-2 program), Pattern VEP was done on Medelec Synergy System using the guidelines of International Society or Clinical Electrophysiology of Vision (ISCEV) [16]. Pateint was seated in dark room at 1 meter from monitor (17 inch screen). Surface disc silver chloride electrodes were used. The ground electrode was placed at the forehead in the midline, the active electrode placed at 5 cms above the inion and the reference electrode was placed at the vertex. A reversing checkboard pattern with a check size of 16 at a frequency of 1 Hz was used. 64 pattern reversals were taken for each eye. Procedure was performed monocularly.PERG was performed on Medelec Synergy System on the guidelines of ISCEV [17].Ground electrode, a Silver Chloride surface electrode was placed on the forehead. Before placement of electrode, the area was thoroughly cleaned with a cleaning agent & a conductive paste was used to ensure good electrical connection. It helped in decreasing impedance & rejecting artefacts. Reference electrode a silver chloride surface electrode was placed on the outer canthus of the eye being examined. Active electrode which is a Gold foil electrode was placed in the inferior fornix of the eye just touching the inferior limbus. It was stabilized by tethering it to the cheek of the patient. Impedance was set at <4KΩ.A black and white reversing checkerboard was used . The mean of the width and the height of the stimulus field was 15º with a check size of 0.8º. Photopic luminance level for the white areas was greater than 80cd/m^2. The mean luminance of the stimulus screen was constant during checkerboard reversals. The contrast between black and white squares was 100%. PERG was recorded in dim room light as background illumination. A reversal rate of 2 rev/s (1 Hz) was used for the recording. AC-coupled amplifiers with input impedance of 10 MX were used for amplification. The frequency response of bandpass amplifiers was from 1–100 Hz. The analysis period (sweep time) for the standard PERGs was 200 ms. The PERG was recorded with the patient seated in a dark room at a distance of 1m from the screen, wearing their appropriate optical correction. Pupils were not dilated. It preserves accommodation and thus retinal image quality. Cornea was anaesthetized with Proparacaine eye drops & gold foil electrodes were placed. Ground & reference electrodes were placed on the forehead & ipsilateral canthus respectively. Uniocular recordings were obtained. The patient was asked to fixate at a fixation mark (Red Square) in the centre of the screen. Blinking was allowed After every 10 secs. A minimum of 150 artefact-free sweeps were collected and averaged for reporting. P50 amplitude was measured from the trough of N35 to the peak of P50. P50 latency was measured from stimulus onset to the peak of P50.

N95 amplitude is measured from the peak of P50 to the trough of N95.Ganglion cell complex thickness, Global loss volume (GLV) and focal loss volume (FLV) evaluation using Spectral domain OCT (Optovue, Heidelberg Engineering, Heidelberg, Germany RT 100 version 5.1). Patients were followed up for a period of at least 12 weeks with periodic evaluation. The unaffected eye of cases was also evaluated for all the parameters. After complete evaluation 2mg intravitreal injection of preservative free Triamcinolone Acetonide (IVTA) was given under aseptic conditions along with topical antibiotics. Thirty eyes of age and sex matched controls were also evaluated to get baseline values.

Exclusion criteria included all the patients who were unable to cooperate for OCT or VEP testing, who had other causes of vision loss in the clinically affected eye (including amblyopia, any retinal/macular pathology, cataract or glaucoma) and who were suffering from neurological disorders like multiple sclerosis, mental retardation, cortical visual impairment which could also be responsible for optic disc edema and visual impairment.

Informed consent was obtained from all individual participants included in the study.

The data was analyzed using Statistical Package for the Social Sciences (SPSS) version 17.0.

Results:

The mean age of 15 patients enrolled in the study was 54.47 years ±7.43 ranging from 43 to 67 years. Out of the 15 patients, 8 (53.3%) were male while 7 (46.7%) were females.Mean best corrected visual acuity BCVA in the affected eyes at presentation was log MAR 1.48 ± 0.35 which significantly ($p<0.001$) improved to log MAR 0.97 ± 0.47 at 3 months (table 1). While mean BCVA in the fellow eyes at presentation was 0.21 ± 0.17 which remained same after 12 weeks. On Pattern VEP, mean P100 latency was 112.27± 4.59 (range 105 to 121 ms) and mean P100 amplitude was 1.77 ± 0.70 (range 1.0 to 3.8 µV) in the affected eyes as compared to mean latency of 103.8 ± 5.21 (range 100 to 122 ms) and mean amplitude of 8.05 ±_1.48 (range 6.4 to13.7µV) in the fellow eyes at presentation ($p=0.000$). Latency (table 2) improved to 102.73 ± 3.49 ($p=0.001$) which was within the normal range while amplitude (table 3) improved to 2.57 ± 0.61 ($p=0.001$) in the affected eyes at 12 weeks. On PERG, mean N95 latency (table 4) was 97.53 ± 4.15 (range 95 to 123 ms) and mean N95 amplitude (table 5) was 4.18 ± 0.53 (range 3.6 to 5.1 µV) in the affected eyes as compared to mean latency of 96.2 ±_2.08 ($p = 0.26$) and mean amplitude of 6.22 ± 1.015 (range 4.8 to 7.7 µV) in the fellow eyes at presentation ($p=0.006$). Latency improved to 95.80 ± 3.73 ($p=0.14$) while amplitude improved to 3.37 ± 0.37 ($p=0.006$) in the affected eyes at 12 weeks. The difference of N95 latency between the affected and the unaffected eyes at presentation and at 12 weeks was statistically not significant ($p=0.267$ and $p=0.367$ respectively) but for amplitude it was statistically significant ($p=0.006$ and 0.006 respectively).

On OCT evaluation for ganglion cell layer thickness, mean GCC Average Thickness (table 6)in the affected eyes reduced significantly from 89.96 ±13.79 µm to 64.68±5.93 µm at 12 weeks ($p=0.001$). Reduction in mean GCC average thickness in the fellow eyes from 92.10±7.72 µm to 87.46 ± 6.40 µm at 12 weeks was noted. Reduction in GCC thickness in the affected eyes when compared to the fellow eyes was statistically significant at 12 weeks ($p=0.000$).

Mean overall RNFL (µm) (table 7) in the affected eyes reduced significantly from 135.13±29.64 (range 95 to 202 µm) to 64.8±4.70 µm (range 57 to 70 µm) over 12 weeks ($p=0.001$). Mean overall RNFL in the unaffected eyes changed from 104.8±13 to 100.2 ±12.2. Mean overall RNFL in the affected eyes as compared to the unaffected eyes was significantly lower at 12 weeks ($p>0.001$).

Mean focal loss volume FLV (%) (table 8) in the affected eyes increased significantly from 5.92 ± 3.08% to 21.72±5.89% over 12 weeks ($p=0.001$). Mean FLV in the fellow eyes changed from 2.34±1.38% to 3.39±1.67% ($p=0.02$). Mean FLV (%) in the affected eyes as compared to the fellow eyes was significantly higher at 12 weeks ($p=0.000$).

Mean global Loss Volume GLV (%) (table 9) in the affected eyes increased significantly from 14.79±7.39 % to 34.07±6.43% over 12 weeks (p=0.001). Mean GLV in the unaffected eyes changed from 4.11±2.79 % to 5.23±2.99% (p=0.03). Mean GLV (%) in the affected eyes as compared to the unaffected eyes was significantly higher at 12 weeks (p>0.001).

Table 1: Mean Best Visual Acuity (BCVA) logMAR

BCVA logMAR		**At presentation**	**1 week**	**4weeks**	**12 weeks**
Affected Eye	Mean	1.48	1.44	1.08	0.97
	SD	0.35	0.40	0.46	0.47
	p-value (v/s presentation)		0.102	**0.001**	**0.001**
Unaffected Eye	Mean	0.212	0.212	0.212	0.212
	SD	0.17	0.17	0.17	0.17
p-value (Affected v/s Unaffected Eye)		**0.00**	**0.00**	**0.00**	**0.00**

Table 2: Pattern VEP Mean P100 Wave Latency (ms)

P100 LATENCY (ms)		**At presentation**	**4 WEEKS**	**12 WEEKS**
Affected Eye	Mean	112.27	104.67	102.73
	SD	4.90	5.59	3.49
	p-value (v/s presentation)		**0.005**	**0.001**
Unaffected Eye	Mean	103.8	105.27	105.73
	SD	5.35	4.92	3.92
p-value (Affected v/s Unaffected Eye)		**0.000**	0.838	0.089

Table 3: Pattern VEP Mean P100 Wave Amplitude (µV)

P100 AMPLITUDE (µV)		**At presentation**	**4 WEEKS**	**12 WEEKS**
Affected Eye	Mean	1.77	2.14	2.57
	SD	0.70	0.73	0.67
	p-value (v/s presentation)		0.001	0.001
Unaffected Eye	Mean	8.05	8.82	8.57
	SD	1.48	1.99	1.99
p-value (Affected v/s Unaffected Eye)		0.000	0.000	0.000

Table 4: PERG Mean N95 Latency (ms)

N95 Latency(ms)		**At presentation**	**4 Weeks**	**12Weeks**
Affected Eye	Mean	97.53	96	95.8
	SD	4.15	4.10	3.73
	p-value(v/s presentation)	-	0.347	0.139
Unaffected eye	Mean	96.2	95.92	96.10
	SD	2.08	1.24	1.43
p-value (Affected eye v/s Unaffected eye)		0.267	0.838	0.367

Table 5: PERG Mean N95 Amplitude (µV)

N95 AMPLITUDE (µV)		At presentation	4 Weeks	12 Weeks
Affected Eye	Mean	4.183	3.546	3.436
	SD	0.445	0.344	0.212
	p-value (v/s presentation)		0.0006	0.0006
Unaffected eye	Mean	6.22	6.206	6.38
	SD	1.015	1.113	0.880
p-value (Affected eye v/s Unaffected eye)		0.0006	0.0006	0.005

Table 6: GCC: Average Thickness (µm)

AVERAGE THICKNESS (µm)		At presentation	4 Weeks	12Weeks
Affected Eye	Mean	89.96	75.71	64.68
	SD	13.79	8.47	5.93
	p-value (v/s presentation)	-	**0.001**	**0.001**
Normal Eye	Mean	92.10	89.05	87.46
	SD	7.72	6.18	6.40
p-value (Affected v/s Normal Eye)		0.567	**0.000**	**0.000**

Table 7: Overall Mean RNFL (µm)

OVERALL RNFL (µm)		At presentation	4 WEEKS	12 WEEKS
Affected Eye	Mean	135.13	86.33	64.8
	SD	29.64	9.01	4.70
	p-value (v/s presentation)	-	**0.001**	**0.001**
Normal Eye	Mean	104.8	102.46	100.2
	SD	13.84	12.79	12.20
p-value (Affected v/s Normal Eye)		**0.001**	**0.000**	**0.000**

Table 8: GCC: Focal Loss Volume (FLV) (%)

FLV (%)		At presentation	4 Weeks	12Weeks
Affected Eye	Mean	5.92	14.45	21.72
	SD	3.08	3.52	5.89
	p-value (v/s presentation)	-	**0.001**	**0.001**
Normal Eye	Mean	2.34	2.89	3.39
	SD	1.39	1.68	1.67
p-value (Affected v/s Normal Eye)		**0.000**	**0.000**	**0.000**

Table 9: GCC: Global Loss Volume (GLV) (%)

GLV (%)		At presentation	4 Weeks	12Weeks
Affected Eye	Mean	14.95	25.04	34.07
	SD	7.65	6.25	6.43
	p-value (v/s presentation)	-	**0.001**	**0.001**
Normal Eye	Mean	4.11	4.93	5.23
	SD	2.79	2.99	2.99
p-value (Affected v/s Normal Eye)		**0.000**	**0.000**	**0.000**

DISCUSSION

This study is an eye hospital based, single center, prospective interventional study.

Fifteen patients who presented with first episode of acute unilateral NAION within first two weeks of symptoms onset with mean age of 54.47±7.43years (35-70 years) were the subjects of our study. Each patient received 2mg of I.V.T.A. and follow up evaluation was done at 1^{st}, 4^{th} and 12^{th} week. The results of the affected eyes of patients were compared with the unaffected eyes of the same patient.The demographic profile of patients in our study was similar to those conducted by Hayreh et al and Hayreh (2007) et al [18,19].

In our study, the mean duration of presentation was 10.13±4.03 days (range 5 to 14 days) from onset of symptoms. NAION was diagnosed in all 15 patients. Common presenting symptom amongst the patients was sudden loss of vision (100% patients). On examination all patients had relative afferent pupillary defect (RAPD), disc edema and hyperemic disc with flame shaped peripapillary haemorrhages in the affected eye. Unaffected eyes of all patients had normal fundus findings. In our study, visual acuity was evaluated using Snellen's chart and the values were converted to Log MAR (Logarithm of minimum angle of resolution). Mean BCVA in the affected eyes significantly improved after 3 months .Jonas at el. showed the improvement at the end of 5 months after injection IVTA while study done by Kaderli et al reported no improvement in visual acquity after follow up of 9 months [10,12] .Our study showed increased latency and decreased amplitude of P100 wave of pattern VEP in the acute phase of NAION as has been shown by Atilla et al. 2006, Jana´ky at el in 2006 and by Parisi at el in 2008 [3,4,20].No significant changes were seen in pattern ERG N95 latency in the affected eyes over 12 weeks but mean N95 amplitude reduced significantly figure [1-4]. Studies conducted by Froehlich et al, Atilla et al and Jana'ky et al. (2006) reported significant difference between affected and non-affected eyes for mean N95 amplitude. Although many studies show the same results as ours but there is lack of prospective interventional studies with long follow up like ours.[3-5].Injury to the optic nerve is followed by retrograde degeneration of the retinal ganglion cells. There is neuronal (retinal ganglion cell) loss in the macula that occurs with RNFL axonal degeneration. Holder et al. concluded that the N95 component of PERG is a contrast related component generated in relation to the retinal ganglion cells while P50 is partially generated distal to the ganglion cells [20].

In present study RNFL reduced significantly in the affected eyes at 12 weeks (p=0.001). Various studies reported significant difference between affected and non-affected eyes for mean RNFL [22- 24]. No similar prospective study was found in the literature to correlate the RNFL thickness in post IVTA in acute NAION.

This decrease in RNFL thickness from an initial baseline swelling in the RNFL, probably consisted of two components i.e. resolution of inflammation and oedema initially, followed by loss of tissue due to inflammation mediated axonal death and retrograde degeneration leading to axonal loss visualised as thinning of RNFL and GCC on OCT. No significant loss of RNFL

thickness was found in the unaffected eyes at 12 weeks.

In our study a significant GCC thinning occurred at 4 weeks of follow-up (p=0.001). Similar to other studies [8, 23- 25] we observed serial GCC thinning from presentation to 12 weeks that likely indicates permanent neuronal loss. GCC thinning occurs several weeks before RFNL thinning in NAION eyes. The most plausible explanation is axonal swelling, which masks the detection of RNFL loss after NAION.

Tan et al (2009) were the first to develop FLV and GLV as novel macular parameters for assessment of GCC in glaucomatous eyes. Evaluation of GCC is of considerable importance as a significant loss to Retinal Ganglion Cell (RGC) population can occur prior to detectable visual field deficits in glaucoma and structural loss can precede detectable functional loss by up to 5 years. FLV and GLV are the quantitative measurements of the amount of change in GCC volume [26]. Other studies have found GLV and FLV as sensitive measures of detecting ganglion cell loss in glaucoma[27,28].

To the best of our knowledge, there is no longitudinal study that has evaluated GCL in NAION using FLV and GLV on NAION. As a pilot study, we found a significantly higher mean FLV and mean GLV in affected eyes in NAION as compared to the fellow eyes. This signified that the FLV and the GLV can be important tools in measuring ganglion cell loss in patients of NAION.

Also, there was a statistically significant change in mean FLV and mean GLV in the fellow eyes at 12 weeks signifying ganglion cell loss with possible subclinical inflammation occurring in the fellow eyes as well. Thus, FLV and GLV can be used as effective tools for evaluating ganglion cell loss in NAION.

Limitations of our study include small sample size,small follow up and lack of control group.One patient out of 15 developed high intraocular pressure after 2 weeks of IVTA but was controlled on topical antiglaucoma drugs.

Conclusion:

We recommend that cases of NAION should be comprehensively evaluated for visual functions (visual acuity, color vision, contrast sensitivity, visual fields), electrophysiology (Pattern VEP and Pattern ERG) and structural abnormalities (OCT RNFL, GCC and Macular volume) both in the normal and the affected eyes.

Longitudinal follow up should be done for at least 12 weeks to evaluate these changes. NAION cases are associated with retinal ganglion cell loss which can be assessed anatomically by OCT using GCC, FLV and GLV parameters and electro physiologically by decrease in amplitude of N95 wave of PERG. We recommend the use of these parameters in any neuroprotection trials to monitor the efficacy of therapies aimed to protect optic nerve in cases of NAION. Study also recommends the use of triamcinolone acetonide in cases of acute NAION.

Acknowledgement:

The authors would like to thank the staff at Guru Nanak Eye Centre and the Medical Records Department for their support in the execution of this study.

Financial support and sponsorship: Nil.

Compliance with Ethical Standards: Informed consent was obtained from all individual participants included in the study.

REFERENCES

1. Miller NR, Arnold AC. Current concepts in the diagnosis, pathogenesis and management of nonarteritic anterior ischaemic optic neuropathy. Eye (Lond). 2015 Jan; 29(1):65-79.
2. Olver JM, Spalton DJ, McCartney AC. Micro vascular study of the retro laminar optic nerve in man: the possible significance in anterior ischaemic optic neuropathy. Eye (Lond). 1990; 4 (Pt 1):7-24.
3. Janáky M, Fülöp Z, Pálffy A, Benedek K, Benedek G. Electrophysiological findings in patients with nonarteritic anterior ischemic optic neuropathy. Clin Neurophysiol. 2006 May; 117(5):1158-66.
4. Atilla H, Tekeli O, Ornek K, Batioglu F, Elhan AH, Eryilmaz T. Pattern

electroretinography and visual evoked potentials in optic nerve diseases. J Clin Neurosci. 2006 Jan; 13(1):55-9.

5. Froehlich J, Kaufman DI. Use of pattern electroretinography to differentiate acute optic neuritis from acute anterior ischemic optic neuropathy. Electroencephalogr Clin Neurophysiol. 1994 Nov; 92(6):480-6.
6. Bellusci C, Savini G, Carbonelli M, Carelli V, Sadun AA, Barboni P. Retinal nerve fibre layer thickness in nonarteritic anterior ischemic optic neuropathy: OCT characterization of the acute and resolving phases. Graefes Arch Clin Exp Ophthalmol. 2008 May; 246(5):641-7.
7. Gonul S, Koktekir BE, Bakbak B, Gedik S. Comparison of the ganglion cell complex and retinal nerve fibre layer measurements using Fourier domain optical coherence tomography to detect ganglion cell loss in non-arteritic anterior ischaemic optic neuropathy. Br J Ophthalmol. 2013 Aug; 97(8):1045-50.
8. Larrea BA, Iztueta MG, Indart LM, Alday NM. Early axonal damage detection by ganglion cell complex analysis with optical coherence tomography in nonarteritic anterior ischaemic optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2014 Nov; 252(11):1839-46.
9. Atkins EJ, Bruce BB, Newman NJ, Biousse V. Treatment of nonarteritic anterior ischemic optic neuropathy. Surv Ophthalmol. 2010 Jan-Feb; 55(1):47-63.
10. Jonas JB, Spandau UH, Harder B, Sauder G. Intravitreal triamcinolone acetonide for treatment of acute nonarteritic anterior ischemic optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2007 May; 245(5):749-50.
11. Sohn BJ, Chun BY, Kwon JY. The effect of an intravitreal triamcinolone acetonide injection for acute nonarteritic anterior ischemic optic neuropathy. Korean J Ophthalmol. 2009 Mar; 23(1):59-61.
12. Kaderli B, Avci R, Yucel A, Guler K, Gelisken O. Intravitreal triamcinolone improves recovery of visual acuity in nonarteritic anterior ischemic optic neuropathy. J Neuroophthalmol. 2007 Sep; 27(3):164-8.
13. Hayerh SS. Intravitreal triamcinolone acetonide for treatment of acute nonarteritic anterior ischemic optic neuropathy.J neroopthalmol; 2008:77-8.
14. Yaman A, Selver OB, Saatci AO, Soylev M. Intravitreal triamcinolone acetonide injection for acute non-arteritic anterior ischemic optic neuropathy.Clin Exp Optom. 2008 Nov; 91(6):561-4.
15. Radoi C, Garcia T, Brugniart C, Ducasse A, Arndt C.Intravitreal triamcinolone injections in non-arteritic anterior ischemic optic neuropathy.Graefes Arch Clin Exp Ophthalmol. 2014 Feb; 252(2):339-45.
16. Odom JV,Bach M,Brigell M,Holder GE,McCulloch DLL,Mizota A,Tormene AP (2016) ISCEV standard for clinical visual evoked potentials –(2016 update).Doc Ophthalmol 133(1):1-9.
17. McCulloch DL,Marmor MF,Brigell MG,Hamilton R,Holder GE,Tzekov R,Bach M (2015) ISCEV standard for full-field clinical electroretinography (2015 update).Doc Ophthalmol 130:1-12.
18. Hayreh SS, Joos KM, Podhajsky PA, Long CR. Systemic diseases associated with nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol 1994;118:76680.
19. Hayreh SS, Zimmerman MB. Incipient nonarteritic anterior ischemic optic neuropathy. Ophthalmology. 2007 Sep 30; 114(9):1763-72.
20. Parisi V, Gallinaro G, Ziccardi L, Coppola G. Electrophysiological assessment of visual function in patients with non-arteritic ischemic optic neuropathy. European journal of neurology. 2008 Aug 1; 15(8):839-45.
21. Holder GE. The significance of abnormal pattern electroretinography in anterior visual pathway dysfunction. Br J Ophthalmol. 1987; 71:166–71.
22. Bellusci C, Savini G, Carbonelli M, Carelli V, Sadun AA, Barboni P. Retinal nerve fibre layer thickness in nonarteritic anterior ischemic optic neuropathy: OCT characterization of the acute and resolving

phases. Graefes Arch Clin Exp Ophthalmol. 2008; 246:641-7.

23. Han M, Zhao C, Han QH, Xie S, Li Y. Change of retinal nerve layer thickness in non-arteritic anterior ischemic optic neuropathy revealed by Fourier domain optical coherence tomography. Current Eye Research. 2016 Aug 2; 41(8):1076-81.

24. Keller J, Oakley JD, Russakoff DB, Andorrà-Inglés M, Villoslada P, Sánchez-Dalmau BF. Changes in macular layers in the early course of non-arteritic ischemic optic neuropathy. Graefe's Archive for Clinical and Experimental Ophthalmology. 2016 Mar 1; 254(3):561-7.

25. De Dompablo E, García-Montesinos J, Muñoz-Negrete FJ, Rebolleda G. Ganglion cell analysis at acute episode of nonarteritic anterior ischemic optic neuropathy to predict irreversible damage. A prospective study. Graefe's Archive for Clinical and Experimental Ophthalmology. 2016 Sep 1; 254(9):1793-800.

26. Tan O, Chopra V, Lu AT, Schuman JS, Ishikawa H, Wollstein G et al. Detection of macular ganglion cell loss in glaucoma by Fourier-domain optical coherence tomography. Ophthalmology. 2009 Dec; 116(12):2305-14.e1-2.

27. Kim NR, Lee ES, Seong GJ, Kim JH, An HG, Kim CY. Structure-function relationship and diagnostic value of macular ganglion cell complex measurement using Fourier-domain OCT in glaucoma. Invest Ophthalmol Vis Sci. 2010 Sep; 51(9):4646-51.

28. Rao HL, Babu JG, Addepalli UK, Senthil S, Garudadri CS. Retinal nerve fibre layer and macular inner retina measurements by spectral domain optical coherence tomography in Indian eyes with early glaucoma. Eye (Lond). 2012 Jan; 26(1):133-9.