Nerve fiber layer findings by scanning laser polarimetry in central retinal vein occlusion after panretinal photocoagulation

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Abstract

This report is of a patient with ischemic central retinal vein occlusion who underwent panretinal photocoagulation. Serial measurements of the retinal nerve fiber layer were performed using scanning laser polarimetry, which showed increased thickness of the peripapillary region as early as 2 weeks after the panretinal photocoagulation.

Key words: Laser coagulation, Retinal vein occlusion

Introduction

The retinal nerve fiber analyzer (NFA) performs scanning laser polarimetry and is used to assess the thickness of the peripapillary nerve fiber by measuring the retardation of the laser beam as it double-passes the birefringent retinal nerve fiber layer (RNFL). Structures such as axonal cell membranes, microtubules, neurofilaments, and mitochondria all contribute to birefringence. This provides a quantitative measurement of nerve fiber thickness consistent with areas of axonal loss, that can then be used for comparative analyses. The NFA has been primarily used to detect early glaucoma and monitor disease progression. Recently, there have been reports of NFA findings in central retinal artery occlusion and non-arteritic anterior ischemic optic neuropathy showing depression of the RNFL within weeks of the occurrence of visual loss. This report is of a patient with ischemic central retinal vein occlusion (CRVO) in whom NFA showed an increase of RNFL thickness as early as 2 weeks after panretinal photocoagulation (PRP).

Case report

A 50-year-old Asian male presented to the Department of Ophthalmology and Visual Sciences at the Prince of Wales Hospital with decreased vision in the left eye of 5 days duration. His vision was 20/200 in the left eye and 20/10 in the right eye. He was found to have a relative afferent pupillary defect in the left eye. Fundal examination of the left eye revealed a classic ischemic CRVO. The ischemic nature was later confirmed by electroretinography. Prophylactic PRP was performed 2 weeks after presentation with no intraoperative complications. Serial nerve fiber thickness measurements using scanning laser polarimetry were done at presentation and at monthly intervals for 3 months. After 3 months, the vision in his left eye stabilized at 10/200 after 2 sessions of PRP, with no clinical evidence of retinal ischemia.

Figure 1 shows a compiled illustrative printout of the 4 serial extended analytical scans performed on this patient. The average value of the 4 quadrants were -18.75 (baseline), -16 (1 month), -2.25 (2 months), and 10.75 (3 months). The superior quadrant showed the most evident increase, from -38 at baseline to 27 at 3 months. This is best illustrated by...
the tomographical estimate with decreasing blue coloration in subsequent scans (Figure 1, column 1).

Symmetry values corresponded to the average 1500 thickest pixels in the superior quadrant over the inferior quadrant. In this patient, poor values (1.76) were noted at 3 months. Maximum modulation and ellipse modulation both indicated the difference between the thickest and thinnest layers in the RNFL. Normalization of values was seen at 3 months. The RNFL image at the bottom of the printout showed initial attenuation of retardation, which improved on later scans.

Discusssion

Since the introduction of scanning laser polarimetry, its use has mainly been to predict early glaucoma, assess the visual field damage, and monitor progression of the disease. The RNFL findings for central retinal artery occlusion have recently been described by Foroozan et al who showed diffuse depression of retardation of the RNFL. Although acute insult to the neuoretina should theoretically cause depression of the RNFL thickness, other researchers have reported paradoxical increases in the RNFL thickness in traumatic optic neuropathy and acute optic neuropathy.

Medeiros et al reported a paradoxical increase in RNFL thickness in a patient who had traumatic optic neuropathy after 1 month of observation. These researchers attributed this increase to an overall diffuse loss of axonal fibers, which resulted in normal values. Another investigation from Meier et al also highlighted this paradoxical change, which was attributed to axonal swelling in patients with acute optic neuropathy.

Ischemic CRVO occurs when the site of occlusion is closer to the lamina cribrosa, resulting in fewer venous tributaries available for collateral circulation. Consequently, extreme retinal ischemia with nerve fiber layer and ganglion cell...
damage occurs. This should translate into thinning of the RNFL but we observed the opposite. This patient showed an increase in RNFL thickness in the peripapillary region after PRP as early as 2 weeks post-procedure, as seen by nerve fiber layer scans, retardation images, and numerical values obtained by NFA/GDx software. Although these calculated values are arbitrary, the results were compared with normal Asian population values. The exact interpretation of these values will require further investigation.

Banks et al, in their study of atrophic and edematous optic nerve heads, concluded that optic nerve head and RNFL edema was not associated with increased birefringence elements. Histopathologic and ultrastructural observations did not show increased neurofilaments or microtubules. Ozdek et al questioned the effect of birefringence in their study. They speculated that the polarization compensator for other structures in the eye, mainly the cornea and lens, exceeded the capacity of the compensator.

The paradoxical increase in RNFL in this patient cannot be explained by retinal edema alone. Hsu and Chung showed that retinal edema did not increase laser retardation in their study, in which they measured RNFL thickness in patients with diabetes undergoing PRP.

These authors do not believe the observations for this patient are due to inter-test variation. The most likely cause of the increase in RNFL thickness is the presence of blood (present in all layers of the retina in CRVO) that can affect readings of the NFA, and that subsequent resolution of hemorrhage led to an improvement of values.

The authors hypothesize that PRP caused an even diffuse loss of axonal fibers, leading to decreased oxygen and metabolic demands. This may have contributed to the recovery of the RNFL, which was not damaged, or minimally affected, by the initial insult, averaging out the values and giving a ‘simulated’ improved numerical result.

In summary, this report shows a paradoxical increase in RNFL thickness in a patient with ischemic CRVO after PRP. Further studies with longer longitudinal follow-up, with a larger cohort, and histopathologic correlation may prove useful in determining the clinical significance of these observations.

References