CORRESPONDENCE

Retinal nerve fiber layer loss in pigment dispersion syndrome

Dear Editor,

We read with interest Mocan and associates' article describing polarimetric findings in patients with pigment dispersion syndrome (PDS) (Eur J Ophthalmol 2003; 13: 377-82). The authors comment that retinal nerve fiber layer (RNFL) loss, as documented by scanning laser polarimetry (GDx-NFA), is present to some extent in eyes with PDS. We wish to share our polarimetric findings in a similar group of patients. We imaged 12 eyes of 12 patients with PDS using the scanning laser polarimeter with variable corneal compensation (GDx-VCC; Laser Diagnostic Technologies, San Diego, CA).

PDS was diagnosed by the presence of Krukenberg spindles, mid-peripheral, slit-like, radial iris transillumination defects, and trabecular pigmentation. All included eyes with PDS had intraocular pressure (IOP) < 21 mmHg, wide open anterior chamber angles, normal achromatic automated perimetry (AAP), and were on no anti-glaucoma medications. We compared the GDx-VCC parameters of the PDS patients with those of 9 age, sex and race-matched control subjects. In contrast to the results reported by Mocan et al, we found no statistically significant differences between the two groups with respect to the average RNFL thickness, superior and inferior average RNFL thickness, superior and inferior maximum RNFL thickness, ellipse average thickness and superior integral (p > 0.05, by both Student's t-test and Wilcoxon rank sum test). We further analyzed the GDx RNFL parameters with the normative database. The GDx-VCC™ printout shows TSNIT (RNFL) parameters, and a deviation map for each scanned eye, and compares the information with a normative database, flagging parameters by colors based on the probability of normality. None of the GDx-VCC parameters were flagged in the PDS eyes upon comparison with the normative database.

In their article, Mocan et al posit that their PDS subjects group may have had more active anterior chamber pigment dispersion and thus, a more severe trabecular obstruction. They also acknowledge that RNFL loss may not be seen in PDS subjects with less or even arrested pigment dispersion. The authors may consider repeating their measurements with the GDx-VCC, as correcting for corneal compensation on a sub-

ject-specific individual basis improves the ability to discriminate between healthy and glaucomatous eyes when compared to GDx-NFA with fixed corneal compensation (1).

Association between PDS and retinal abnormalities have been reported, including retinal detachment (2) and lattice degeneration (3). Although there have not been any reports of RNFL loss, retinal pigment epithelial dysfunction has been described in patients with PDS (4, 5). Clearly, there are still many unanswered questions. We feel that further investigation is necessary to determine whether retinal changes are intrinsically associated with PDS.

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Author reply

We appreciate Dr. Ritch and associates' interest in our article, and thank them for sharing their polarimetric findings in pigment dispersion syndrome (PDS) using GDx-VCC. In our study (1), employing scanning laser polarimetry (NFA GDx version), we showed retinal nerve fiber layer (RNFL) loss in eyes with PDS compared to the healthy subjects. All cases in our study underwent macular scanning to rule out the effect of corneal polarization. Nevertheless, we agree with Dr. Ritch and associates that it would be of further interest to repeat the measurements with the GDx-VCC to determine the exact retardation measurements in these patients. Despite that, we doubt using GDx-VCC would change our results significantly, since the difference in thickness between the groups and the number of the subjects in our study groups were high enough for parametrical statistical analysis.

In contrast with our results, Dr. Ritch and associates found no statistical differences in their study between the PDS patients and the controls and the normative database of the instrument. The difference between the two studies might originate from high inter-individual variability, overlap in GDx parameters between the glaucomatous and healthy subjects and ethnic differences. As mentioned before in our study, our PDS subject group might have had more active anterior chamber pigment dispersion and more severe trabecular obstruction and degeneration. There-

fore, it might be possible that these subjects could have developed spontaneous or exercise related intermittent high IOP, undetected during routine ophthalmologic examination, yet leading to RNFL loss. On the other hand, some patients with PDS might have less or arrested pigment dispersion and, consequently, RNFL loss might not be present. We agree with Dr. Ritch and associates that there are many unanswered questions about PDS and pigmentary glaucoma and further studies with large number of patients are essential to evaluate both the morphological and the functional changes in these subjects.

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