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Case Report

Delayed Diagnosis of Glaucoma in a Patient with Myelinated Nerves

Erkan Ünsal, Kadir Eltutar, İlkay Kılıç Müftüoğlu, Yıldız Acar, Osman Kızılay

Abstract

This study aims to present a glaucoma case with delayed diagnosis because of myelinated retinal nerve fiber layer. On ophthalmological examination of a 58-year-old female patient with noted decrease in vision, corrected visual acuities were 0.9 in the right eye and 0.4 in the left eye, and intraocular pressures were 24 and 23 mmHg, respectively. In a slit-lamp examination, grade 1 nuclear sclerosis was observed in both the eyes. The iridocorneal angles were Grade 3 in both the eyes. On dilated fundus examination, the optical disc was bilaterally surrounded with myelinated nerve fibers, and the right one was larger than the left one. Cup/disc ratio was found to be 0.9 on the right and 0.5 on the left. On standardized Humphrey visual field test peripheral advanced deep, concentric visual field constriction and enlargement of the blind spot, which was prominent on the right side, were detected. Moreover, the patient was diagnosed with primary open-angle glaucoma. The appropriate medical treatment was initiated. In this study, we mentioned that myelinated nerve fibers can be associated with glaucoma. In patients with myelinated nerve fibers, the evaluation of the optical disk should be more detailed, and in these patients, visual field assessment should be more carefully performed.

Keywords: Myelinated nerve fibers, retina, glaucoma

Introduction

Myelination in the optic nerve starts in the 7th month of intrauterine life from chiasm and reaches lamina cribrosa at birth and is completed in the first 3 months after birth. Myelination normally ends at lamina cribrosa (1).

In this case report, it was aimed to present a glaucoma case whose complaint of decreased vision was thought to be due to myelinated nerve fibers and thus, received a late diagnosis.

Case Report

It was identified from her history that a 58-year-old woman who was admitted to our hospital with the increasing complaint of decreased vision in the right eye was being followed due to a myelinated nerve fiber at another hospital. In the ophthalmological examination, the best corrected visual acuity was 0.4 (Snellen) in the right eye and 0.9 in the left eye. Intraocular pressure (IOP) of the patient was 24 mmHg in the right eye and 23 mmHg in the left, whereas the central corneal thicknesses were 532 and 526 µm, respectively. In the examination of the iridocorneal angle, the angle was at the level of stage 3 (Schaffer) in both eyes. In biomicroscopical examination of anterior segment, no pathology other than bilateral grade 1 nuclear sclerosis was detected. In dilated fundus examination, there was a myelinated nerve fiber, larger on the right, surrounding the optical disk and the cup/disk ratio was 0.9 on the right and 0.5 on the left (Figures 1, 2). In a standard 30-2 Humphrey threshold test, advanced expansion in the blind spot, which was more apparent on the right, and deep peripheral concentric visual field constriction were found. The analysis of retinal nerve fiber layer (RNFL) thickness was attempted in an optical coherence tomography (OCT) test, but it could not be evaluated clearly because the optic nerve boundaries, particularly in the right eye, could not be determined accurately. For the analysis of the optic nerve head through optical coherence tomography, the ring was placed manually and the peripapillary area was scanned, and RNFL thickness measurements were made. The retinal nerve fiber layer thickness was detected within the normal (average 123.83 µm) borders in the measurements. Primary open-angle glaucoma diagnosis was made with these findings and topical latanoprost drops therapy was initiated. In the 6-month follow-up period of the patient, the IOP remained within normal limits and no progression was detected in the visual field. A written informed consent was obtained from the patient.

Discussion

Myelinated nerve fibers were reported at a rate of 0.98% in the community, and 33% of the cases were found to be associated with the optic nerve (2). The pathogenesis of myelinated nerve fibers

Clinic of Eye, İstanbul Training and Research Hospital, İstanbul, Türkiye

Address for Correspondence:
Erkan Ünsal
E-mail: erkanunsal@gmail.com

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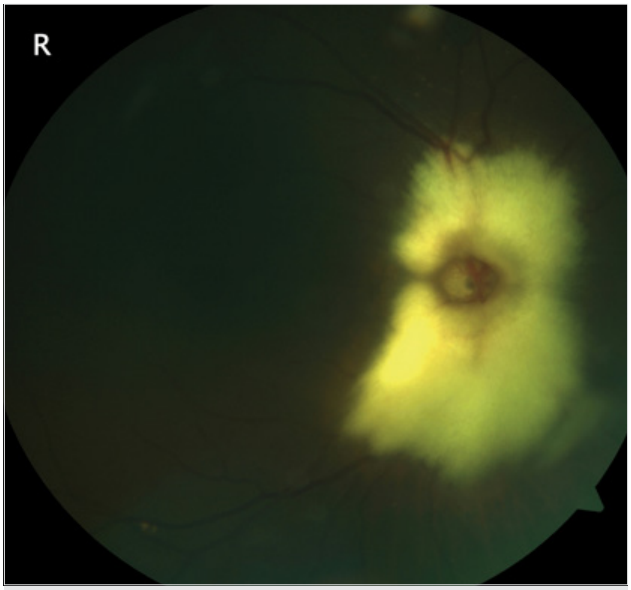

Figure 1. Fundus photograph of the right eye. It can be seen that as the cup/disk ratio increased, myelinated nerve fibers exist around the optic disk

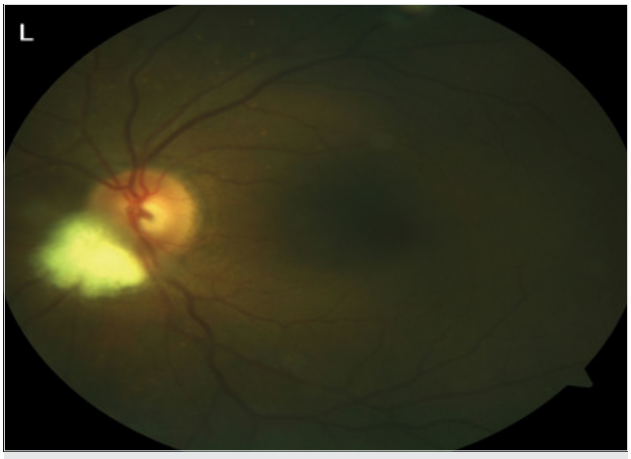

Figure 2. Fundus photograph of the left eye. Optic disk in the lower nasal, where myelinated nerve fibers can be seen. The cup/disc ratio is increased

is speculative. However, it is thought that in lamina cribrosa, a defect allows oligodendrocytes to enter the retina and then myelin occurs here.

In some cases, the myelination progresses to the optical disk and/or the retinal surface creates yellow-white colored retinal lesions with feathery-looking edges, which are seen on the edge of the optical disk most commonly, but can rarely be monitored in the periphery in the form of isolated foci. When myelinated fibers in the retina are located in the peripapillary, they may be confused with papilledema (3). In our case, myelinated nerve fibers were also in the peripapillary area.

Visual field defects are associated with the density of myelination rather than the anatomical location of myelinated fibers. Functional losses rarely develop even in very intense myelination but they may cause small scotomas. Visual field defects defined to be dependent on myelination are often in the form of an irregular enlargement in a blind spot. In the presence of isolated myelination, arcuate defects isolated relative or absolute scotomas can, in rare cases, be encountered (4).

Glaucoma is a progressive and chronic optic neuropathy characterized by the damage of the ganglion cells in the optic nerve head (5). Primary open-angle glaucoma (POAG), which is also accepted as chronic progressive anterior optic neuropathy, is the most common type of glaucoma. It is a clinical condition characterized by pitting-atrophy in the optical disk due to a high IOP and the presence of specific defects in the visual field of patients a with large anterior chamber angle (6). The risk of POAG gradually increases with the IOP level, and the disease, which is often bilateral, is chronic and progressive. In the beginning, it is often insidious and if the disease does not cause a subjective symptom for a long time, it instead results in absolute glaucoma with visual loss in the final phases (6). In our case, the IOP was 24 mmHg in the right eye and 23 mmHg in the left eye, and the anterior chamber angle was detected as open.

Growth and deepening of the disk cupping, vertical cupping, growth in the cup/disk ratio, the pores of the lamina cribrosa becoming apparent, neuroretinal rim thinning, nasalization of the vessels, the disk hemorrhages, and peripapillary atrophy can be sorted among the glaucomatous optical disk changes (6). Growth and deepening in the eye cup, increases in the cup/disk ratio, neuroretinal rim thinning, and nasalization in the blood vessels were also detected in the right eye of our patient. However, peripapillary atrophy could not be detected because the parapapillary area was covered by the myelinated nerve fibers. Our patient was diagnosed with POAG with these findings. Although glaucomatous optic disk cupping was very significant, it was observed that it could be overlooked in a careless examination because of the lack of color contrast due to the peripapillary myelinated nerve fibers.

Situations that cause changes in the optic nerve head and that can be confused with glaucoma include optic disk coloboma, morning glory disk anomaly, peripapillary staphyloma, megalopapilla, 'blank optical disk' associated with the optic pit, and papillorenal syndrome are among the excavated optic disk anomalies. Among these, myelinated nerve fibers can also make the diagnosis of glaucoma difficult.

Nowadays, the follow-up of glaucoma patients through OCT has almost entered into routine practice thanks to rapidly developing technology. However, because the boundaries of the optic nerve head in these cases could not be evaluated clearly, a reliable optic nerve head (ONH) examination cannot be made. We also performed RNFL and ONH examinations by placing the ring manually in ONH for the screening of the peripapillary region in our patient. In addition to the application challenges, a lack of sufficient data in the literature about how myelinated nerve fibers will affect the RNFL thickness make it difficult to make an accurate comment.

In the literature, Nourinia et al. (7) found that the RNFL thickness measured by OCT of a patient who has myelinated nerve fibers around the optic nerve increased significantly and was hyper-reflective. Sowka and Nadeau (8) found that myelinated nerve fibers became atrophied in a glaucoma patient who had myelinated nerve fibers around the optic nerve and who they followed for 8 years. In our case, RNFL thickness was observed to be within normal limits. However, the detection of a normal RNFL thickness can be explained by that the RNFL was too thick before the development of glaucoma and it just fell down to normal values after getting thinner.

Conclusion

We believe that myelinated nerve fibers can be seen with glaucoma, and it is necessary to assess the optic disk in more detail in patients with myelinated nerve fibers. Furthermore, the evaluation of the visual field should be made more carefully in these patients.

Informed Consent: Informed consent was obtained from patients who participated in this study.

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References

1. Kayıkçıoğlu Ö, Mayalı H. Miyelinli Sinir Lifleri Nedeniyle Geç Tanınan Yırtıklı Retina Dekolmanı. Retina-Vitreus 2007; 15: 63-6.
2. Straatsma BR, Foos RY, Heckenlively JR, Taylor GN. Myelinated retinal nerve fibers. Am J Ophthalmol. 1981:91; 25-38. [CrossRef]
3. Özer A: Yapısal optik sinir anomalileri. İçinde Aydın P: Görme Alanı El kitabı. Aksu Kitabevi, İstanbul. 2005. p. 171-2.
4. Kansky JJ: Clinical Ophthalmology, 4. Baskı. Oxford, London. 1999; 605-6.
5. Shields MB. The Textbook of Glaucoma. Baltimore, Maryland, Williams and Wilkins. 1992: 500-10.
6. Özcan AA. Glokomda sınıflandırma ve klinik. Kısım 12, Bölüm 37. Editör: Pınar Aydın O'Dwyer. Temel Göz Hastalıkları. 2. Baskı. Ankara. Ayrıntı Basımevi. 2011. s. 477-88.
7. Nourinia R, Behdad B, Montahaei T. Optical coherence tomography findings in a patient with myelinated retinal nerve fiber layer. J Ophthalmic Vis Res 2013; 8: 280-1.
8. Sowka JW, Nadeau MJ. Regression of myelinated retinal nerve fibers in a glaucomatous eye. Optom Vis Sci 2013; 90: e218-20. [CrossRef]