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Case Report

Temporal Protrusion of the Optic Disc Implicated in Central Visual Field Abnormalities in High Myopia: Two Case Reports

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High myopia · Focal lamina cribrosa defect · Optic disc · Visual field

Abstract
Introduction: Severe central visual field defects are frequently observed in highly myopic eyes. This report details 2 cases of central visual field defects in individuals with high myopia, characterized by an unusual temporal protrusion of the optic disc, a feature not previously documented. ***Case Presentation:*** Two patients, a 54-year-old man and a 65-year-old woman, were diagnosed with high myopia in their left eyes, displaying an outward protrusion of the optic disc toward the macula. Swept-source optical coherence tomography revealed a focal lamina cribrosa defect at the temporal edge of the protruding optic disc, corresponding to the papillomacular bundle area of retinal nerve fibers, which exhibited thinning around the focal lamina cribrosa defect. Visual field examination indicated a central visual field defect in the affected eyes, which pattern corresponded to the papillomacular bundle responsible area. ***Conclusion:*** The emergence of a temporal protrusion in the optic disc may lead to a focal lamina cribrosa defect, resulting in a central visual field defect in highly myopic eyes. This distinctive optic disc feature may constitute a critical risk factor for a central visual field defect. Hence, optic disc protrusion in high myopia warrants attention, necessitating careful ophthalmic examinations for central visual field defects.

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Introduction

Myopia is a global health concern, with recent prevalence rates of 90% in East Asia and 50% in the USA and Europe, indicating a continued increase [1–3]. High myopia, defined as an axial length exceeding 26.5 mm or a myopic refractive error below −8 diopters, involves progressive eyeball elongation [4], with its prevalence rising [3]. It is associated with severe complications, such as retinal detachment, macular schisis, macular hole, chorioretinal atrophy, choroidal neovascularization, and intrachoroidal cavitation [5, 6]. Additionally, an association with glaucoma, including primary open-angle and normal-tension glaucoma, has been reported [7]. Despite myopia's increased prevalence, the role of morphological features in high myopia in contributing to susceptibility to glaucomatous visual field (VF) loss remains unclear. Diverse optic disc shapes in high myopia present challenges in glaucoma diagnosis. Glaucomatous VF defects in high myopia may exhibit specific patterns, such as central vision defects [8]. Recent studies link structural abnormalities in the optic nerve head, including intrachoroidal cavitation, acquired optic nerve pit, peripapillary pit, scleral ridge, and focal lamina cribrosa defect (fLCD), to VF defects [9–15]. Studies in eyes with and without myopia emphasize a strong relationship between fLCD and glaucoma [11, 12, 14, 15]. For instance, Sawada et al. [12], using OCT, investigated LCD characteristics in myopic eyes with glaucoma, concluding that fLCD in myopic eyes may evolve into larger defects as glaucoma develops. They suggested myopia might influence glaucomatous VF defects through an increased number of LC defects at the temporal periphery of the optic disc. Tatham et al. [15], analyzing fundus structures in glaucoma eyes using OCT, frequently identified fLCDs in eyes with localized retinal nerve fiber layer (RNFL) defects. However, the relationship between optic disc morphology and fLCD development in eyes remains unclear. The study aimed to present the clinical features of two high myopia patients with unique optic disc morphology: protruding toward the macula, experiencing severe central VF defects, and developing an fLCD at the protruding optic disc site.

Case Presentation

Case 1

A 54-year-old man with progressive glaucoma and a central VF defect in the left eye reported a gradual decline in visual acuity and VF loss over a decade. Having undergone cataract surgery in both eyes, he had no other medical history or medication use. In the left eye, visual acuity was 20/25, intraocular pressure was 16 mm Hg, axial length was 30.03 mm, and refractive error was −2.75 D. Fundoscopic examination revealed a tigroid fundus with thinning of the temporal rim of the optic disc rim and significant peripapillary atrophy (Fig. 1a, b). Remarkably, the optic disc's temporal side protruded toward the macula (Fig. 1b; arrowheads). The 10-2 VF test showed a mean deviation value of −1.90 dB, with defects corresponding to the papillomacular bundle (PMB) of the RNFL (Fig. 1c). The HF30-2 VF test indicated an enlarged blind spot due to peripapillary atrophy, with no VF defect (Fig. 1d). Swept-source optical coherence tomography illustrated a horizontal section across the protruding optic disc edge toward the fovea (Fig. 1e). A closer view (Fig. 1f) disclosed a fLCD at the temporal side, recognized by three OCT specialists (T.N., T.Y., and T.I.). The fLCD had a width of 154 μm, and the thinnest prelaminar tissue on the LC measured 67 μm. While the OCT image depicted normal retinal morphology around the macula, the RNFL thickness between the macula and fLCD appeared reduced (Fig. 1f).

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Fig. 1. **a** Fundus photography illustrates a temporal protrusion of the optic disc. **b** Higher magnification of the optic disc. White arrowheads show a temporally protruding optic disc. **c** Humphrey perimetry 10–2 analyzer shows a VF defect in the corresponding area of the PMB. **d** Humphrey perimetry 30–2 analyzer shows the enlargement of the blind spot. **e**, **f** Swept-source optical coherence tomography (OCT) across the temporally protruding site of the optic disc. The white line shows an anterior surface of lamina cribrosa, and the yellow dotted line shows the area of the focal lamina cribrosa defect (**f**). **f** Horizontal OCT image of the macula. LC, lamina cribrosa; LCD, lamina cribrosa defect; PT, prelaminar tissue.

Fig. 2. a Fundus photography illustrates a temporal protrusion of the optic disc. **b** Higher magnification of the optic disc. White arrowheads show a temporally protruding optic disc. **c** Humphrey perimetry 10–2 analyzer shows a serious central VF defect. **d** Goldmann perimetry examination shows the enlargement of the blind spot and a central VF defect. **e**, **f** Swept-source optical coherence tomography across the temporally protruding site of the optic disc. The white line shows an anterior surface of lamina cribrosa (**f**). The yellow dotted line shows a large hyporeflective area in the lamina cribrosa, which was diagnosed as a focal lamina cribrosa defect (**f**). LC, lamina cribrosa; LCD, lamina cribrosa defect; PT, prelaminar tissue; Sc, sclera.

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Case 2

A 64-year-old woman presented with progressive glaucoma and a central VF defect in the left eye, reporting gradual VF loss and diminished visual acuity. Having undergone cataract surgery, she had no other medical, present, or past illnesses and used no systemic medications. On examination, her left eye displayed a best-corrected visual acuity of 20/40, intraocular pressure of 13 mm Hg, axial length of 32.39 mm, and refractive error of +0.75 D. Anterior segment analysis revealed aphakia. Fundoscopic examination showed thinning of the temporal optic disc rim, significant peripapillary atrophy, and a tigroid fundus (Fig. 2a). Similar to Case 1, the temporal side of the optic disc protruded toward the macula (Fig. 2b; arrowheads). The 10-2 Humphrey VF test revealed a severe central VF abnormality with a mean deviation value of −15.56 dB, encompassing the central VF area, including the PMB region (Fig. 2c). Goldmann perimetry showed an enlarged blind spot due to extensive peripapillary atrophy, confirming the central VF defect observed in the HF10-2 VF test (Fig. 2d). Swept-source optical coherence tomography illustrated a horizontal section across the protruding optic disc edge toward the fovea (Fig. 2e). A closer view (Fig. 2f) revealed a highly reflective anterior surface of the LC on the optic disc (white dot line). A wide hyporeflective space (534 μm) at the temporal edge was diagnosed as a large fLCD by three OCT specialists (T.N., T.Y., and T.I.) (Fig. 2f, yellow dot line). The LC was fully detached from the sclera, and the thinnest prelaminar tissue on the LC was 55 μm. Horizontal OCT images across the macula depicted preserved retinal structures; however, the thinning of the RNFL and roughness of the retina were evident due to myopic diffuse chorioretinal atrophy (Fig. 2e). The visual impairment in this case might be attributed not only to nerve damage due to LCD but also to the presence of chorioretinal atrophy.

Discussion

Myopia, particularly high myopia, is a recognized risk factor for glaucomatous VF disturbances [7]. However, the causes of this association remain unclear. We previously investigated VF defects in highly myopic eyes (myopic refractive error >−8 D or AL ≥26.5 mm), reporting significant defects in 13.2% of selected eyes, with 73.8% showing progression during follow-up [8]. The relationship between glaucoma and high myopia raises questions, as the sites of the VF defects did not correspond to those found in typical glaucomatous eyes. In typical glaucomatous eyes, the VF defect initially manifests in the nasal area, known as the Bjerrum scotoma or the nasal step, while defects in the central sector are typically absent until the late stage of glaucoma [16]. However, in highly myopic eyes, VF defects often originate from the central sites [8], which contrasts with typical glaucomatous eyes. The exact pathogenesis of central VF defects in highly myopic eyes remains less understood. We reported 2 cases of high myopia with unusual features: stretched optic discs protruding toward the macula and central VF defects. Additionally, fLCD at the temporal edge of the optic disc, corresponding to the PMB, may implicate the development of a central VF defect in highly myopic eyes.

LC deformation is a key factor in glaucoma pathogenesis [17, 18]. Berkowska et al. [19] noted impaired ocular blood flow associated with LC deformation in glaucoma patients, influencing the thinning of the RNFL and VF disorders. Some studies showed the implications of fLCD and myopia. Miki et al. [11] observed fLCD in 42% of high myopia patients with glaucoma, suggesting fLCD's role in VF defects and its progression. Sawada et al. [12] associated optic disc tilting with fLCD and glaucomatous VF defects in moderate myopia. They also suggested that the optic disc tilt increased mechanical stress on the LC and increased the number of LC defects [12]. In highly myopic eyes, extreme elongation induces chronic optic disc stretch, resulting in varied morphology [20]. OCT studies demonstrate fLCD predominantly at the optic disc's temporal

periphery, similar to some cases [12, 21, 22]. Temporal protrusion, in part due to highly myopic eyes, may facilitate fLCD development due to concentrated mechanical stress. Considering that the RNFL between the optic disc and macula is referred to as the PMB and is responsible for central vision [23], it is reasonable to assume that the fLCD between the optic disc and macula implicates central VF abnormalities. Furthermore, the prelaminar tissue's thickness in our cases was notably thinner compared to healthy individuals, potentially resulting from RNFL thinning secondary to the development of fLCD, affecting the central VF [24, 25]. The large, unusual morphology of the optic disc protruding toward the macula may be a crucial risk factor for fLCD and central VF disturbance in highly myopic eyes. We are the first to report the temporal optic disc protrusion's role in the development of central VF abnormalities and fLCD in high myopia eyes. Considering the potential involvement of the PMB and its strong influence on central visual function, there is an increased likelihood of central VF disturbance in such eyes. However, the characteristics of fLCD in high myopia eyes remain unclear, necessitating large-scale and long-term prospective studies. However, the simultaneous presence of multiple myopic changes, such as chorioretinal atrophy, macular hole, myopic tractional maculopathy, and myopic choroidal neovascularization, is frequently observed in highly myopic eyes, requiring careful observation. In these cases, it is challenging to determine the extent to which either of them is involved in the VF abnormalities. It is also unclear whether intraocular pressure-lowering therapy would be effective for maintaining the central VF in such a case. Longitudinal follow-up of a large number of eyes is required to answer these questions. The CARE Checklist has been completed by the authors for this case report and is attached as supplementary material (for all online suppl. material, see https://doi.org/10.1159/000538440).

In conclusion, this case report provides evidence for the importance of the protrusion of the optic disc toward the macula. Although rare in the high myopia eyes, attention should be paid to the morphological feature of the optic disc in the high myopia eyes. If this optic disc morphology is observed in the high myopia eyes, a careful ophthalmic examination should be performed for the presence of an fLCD and central VF defect.

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Statement of Ethics

This study adheres to national guidelines, and ethical approval is not required. Written informed consent was obtained from the patients for the publication of their medical case details and any accompanying images.

Conflict of Interest Statement

The authors confirm that there is no conflict of interest.

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Author Contributions

T.N. and T.Y. authored the paper and collected clinical data. T.N., T.Y., T.I., and K.O.H. reviewed and interpreted the paper's clinical data. T.Y. conducted clinical revisions and supervised data interpretation. All authors have read and approved the manuscript.

Data Availability Statement

All data generated or analyzed during this study is included in this article. For additional inquiries, please contact the corresponding author.

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