Serial Follow-Up in Traumatic Optic Neuropathy Using Scanning Laser Polarimetry and Visual Field Testing

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A 40-year-old male patient suffered from traumatic optic neuropathy in his right eye. Scanning laser polarimetry was arranged at 2 weeks, 9 weeks, 13 weeks, 24 weeks, and 34 weeks after the trauma. Manual or automated visual field testing was also arranged at 1 week, 5 weeks, 16 weeks, and 28 weeks correspondingly. The data revealed using scanning laser polarimetry (program GDx®, version 1.0.05; Laser Diagnostic Technologies, San Diego, Calif, USA) were nearly normal at 2 weeks after trauma, but lower visual field loss was revealed using visual field testing within 2 weeks after the trauma. The superior hump of the GDx deviated from normal at about 9 weeks and some GDx parameters (the Number, Superior/Nasal, Ellipse Modulation (Ellipse Mod.), Maximal Modulation (Max. Mod.), Symmetry, Superior Ratio (Super. Ratio)) became worse later in the series. We propose that visual field defects might be present before retinal nerve fiber layer loss. In this case, scanning laser polarimetry for evaluating the severity of traumatic optic neuropathy was limited especially within 2 weeks after the trauma. *(Chang Gung Med J 2005;28:581-6)*

Key words: traumatic optic neuropathy, optic nerve injury, visual field, scanning laser polarimetry, GDx.

Descending optic nerve degeneration leads to the retinal nerve fiber layer (RNFL) loss after traumatic optic neuropathy. Scanning laser polarimetry can measure the thickness of the peripapillary RNFL by assessing the changes in polarization due to the birefringent properties of the RNFL.[1] Some authors think that Temporal-Superior-Nasal-Inferior-Temporal (TSNIT) graph and parameters of Nerve Fiber Layer (NFL) Analysis table revealed by GDx are useful for early detection of traumatic optic neuropathy.[2,3] A good correlation between automated visual field testing parameters and peripapillary RNFL thickness as measured using scanning laser polarimetry was found in glaucoma patients.[4] Therefore, we attempted to observe a case of traumatic optic neuropathy with an 8-month serial follow-up using GDx and visual field testing (Goldmann and Octopus). Our aim was to observe the correspondences and differences between scanning laser polarimetry and visual field testing by time sequence in traumatic optic neuropathy.

CASE REPORT

A 40-year-old male patient suffered from head injury during a traffic accident. In his right eye, initial visual acuity was light perception only, and positive findings included periocular swelling, limited eye movement toward the temporal and inferior sides, congested conjunctiva, and relative afferent pupillary defect. The frontal process of the sphenoid bone was fractured over the right lateral orbital wall without optic nerve transection which was identified using image study. The left eye remained normal.

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Under the diagnosis of traumatic optic neuropathy, intravenous megadose steroid treatment was initiated. Six days after the trauma, visual acuity recovered to 20/30. Periocular swelling and conjunctival congestion decreased. Relative afferent pupillary defect was still detectable but it was more indistinct. The right visual field revealed a decrease in the perimetric sensitivity at the central area of visual field, absolute decline in the inferior visual field, and huge decline in the superior visual field within 2 weeks after the trauma, which became stabilized later in the series (Fig. 1). GDx data showed TSNIT graph and the parameters of NFL Analysis table nearly normal at 2 weeks after the trauma. The superior hump in the TSNIT graph deviated from normal at about 9 weeks after the trauma and some GDx parameters (Superior/Nasal, Ellipse Mod., Max. Mod., Symmetry, Super. Ratio) became worse later in the series (Fig. 2). The apparent progressive changes of some GDx parameters stopped on the 13th week after

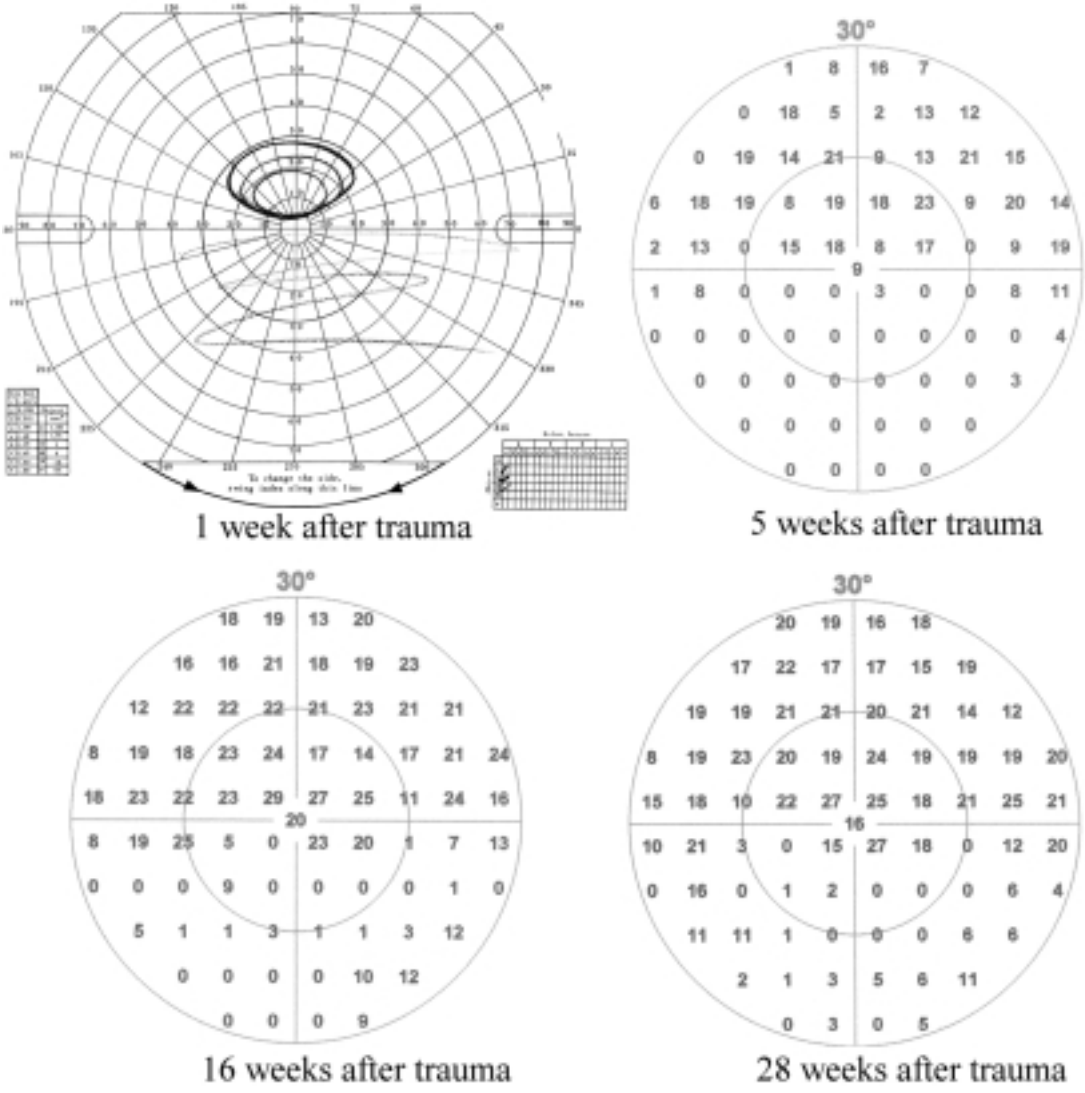

Fig. 1 Visual field defect in the right eye (traumatic eye) was revealed using Goldmann perimetry at 1 week after the trauma, and using Octopus perimetry (only showing the value of absolute sensitivity) at 5 weeks, 16 weeks, and 28 weeks after the trauma.

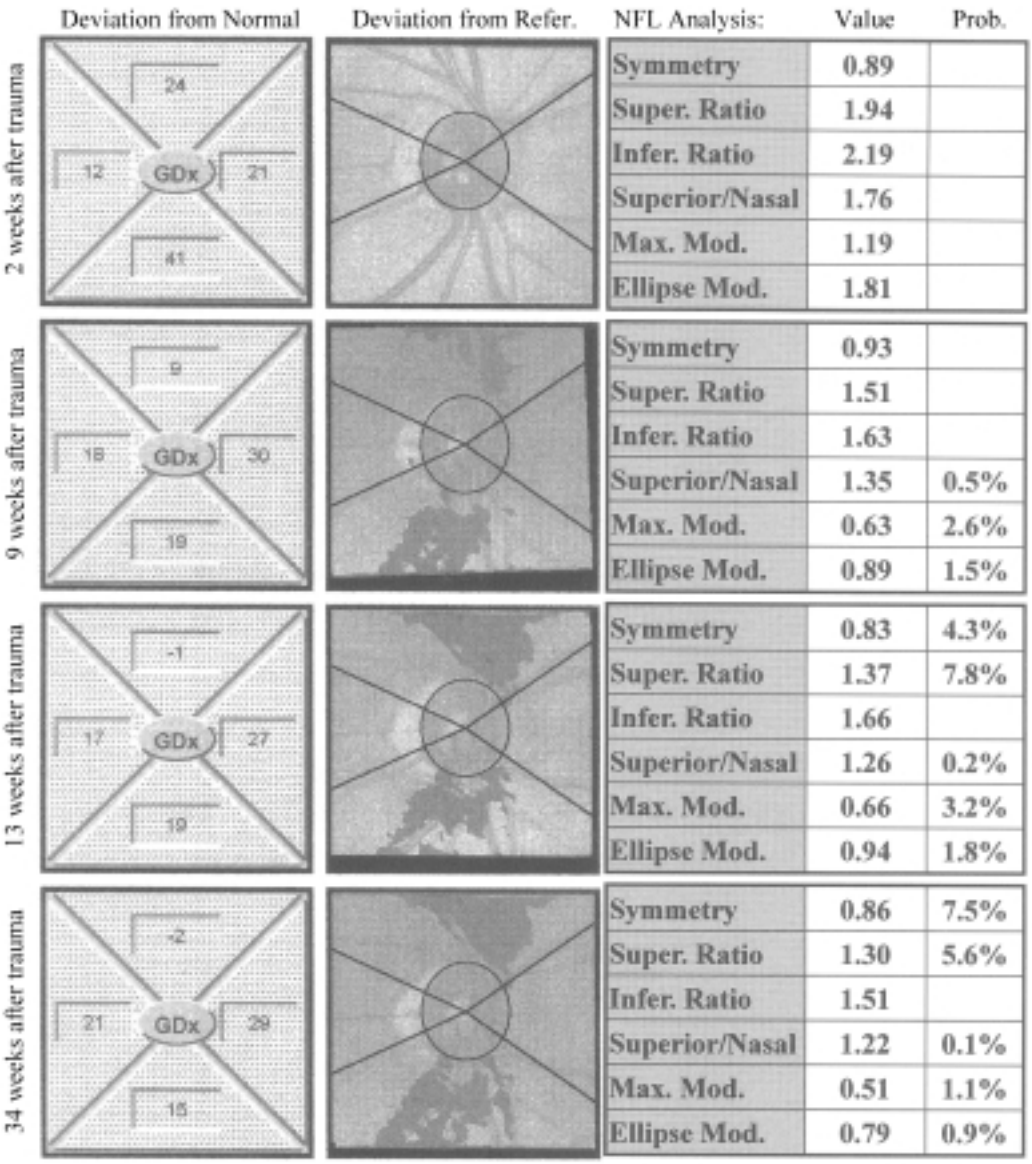

Fig. 2 Serial analysis using GDx integrated software at 2 weeks, 9 weeks, 13 weeks and 34 weeks after the trauma. Thickness map revealed progressive changes after 2 weeks and no more progression before 13 weeks after the trauma.

the trauma. From the NFL Analysis table, the most significant changes of GDx parameter were Superior/Nasal, followed by Ellipse Mod. and Max. Mod..

DISCUSSION

Traumatic optic neuropathy is a disorder of afferent visual function after trauma, characterized by loss of visual acuity, loss of color perception, visual field defect, or loss of contrast sensitivity in one or both eyes. Traumatic optic neuropathy may occur both directly and indirectly. Direct traumatic optic neuropathy involves actual disruption of anatomical structures along any of the paths of the optic nerve, from the scleral canal to the chiasm.

Indirect traumatic optic neuropathy can occur without disruption of anatomical structures, such as the optic nerve proper or the tissues around it, along its long course to the chiasm. Both primary mechanical shearing of the optic nerve and vasculature at the moment of impact as well as secondary insults including ongoing vasospasm and swelling of the optic nerve confined within the optic canal may be involved in the pathogenesis.[5] Retrograded retinal ganglion cell death and corresponding RNFL loss can occur a few weeks after optic nerve damage. In an experimental study of primates, significant descending degeneration of the retinal ganglion did not occur until about 3 weeks after optic nerve transection, with maximum loss at 6 weeks following the injury.[6] Red-free photographs of the fundus demonstrated detectable loss of the nerve fiber at 4 weeks with complete disappearance by 6 weeks in a patient with a gunshot injury to the intracranial optic nerve.[7] In our case, visual field defect was detected within 2 weeks of the injury. The value of GDx parameters in NFL Analysis table were found to be within normal limits about 2 weeks after the trauma. Progressive worsening occurred within 9 weeks of the injury and no more progression occurred between 9 to 13 weeks after the trauma, suggesting no further axon loss was detectable using GDx after that time. We compared the results of our patient with those of the case presented by Medeiros,[2] and we found visual field defects may be present earlier than RNFL loss in traumatic optic neuropathy. In addition, we also found the regressive curve for the time course of axonal loss ($y = 34.6 + 83.2 \times e^{-t/48.2}$, where y is the percentage of the initial retardation value, and t is the time from injury in days), proposed by Meier and their colleague,[3] may only be applied to acute severe retrobulbar optic neuropathy (Fig. 3). The case presented by Medeiros had segmental axonal loss and final best-corrected visual acuity (BCVA) 20/400.[2] Final BCVA of our case was 20/30. We propose that the axonal loss and final BCVA are influenced not only by the amount of time after the trauma, but also the location and severity of the optic nerve damage.

In conclusion, although our observation revealed that the scanning laser polarimetry is able to detect RNFL loss and that a good correspondence between the pattern of visual field loss and the pattern of RNFL loss can be found in cases of incomplete traumatic optic nerve injury, visual field defect may be presented earlier than RNFL loss. Scanning laser polarimetry for evaluating the severity of traumatic optic neuropathy is limited especially within 2 weeks after trauma. In addition, we should note the variations between functional change in visual field and anatomic changes of RNFL, and cannot interpret the result using a single GDx parameter in traumatic optic neuropathy.

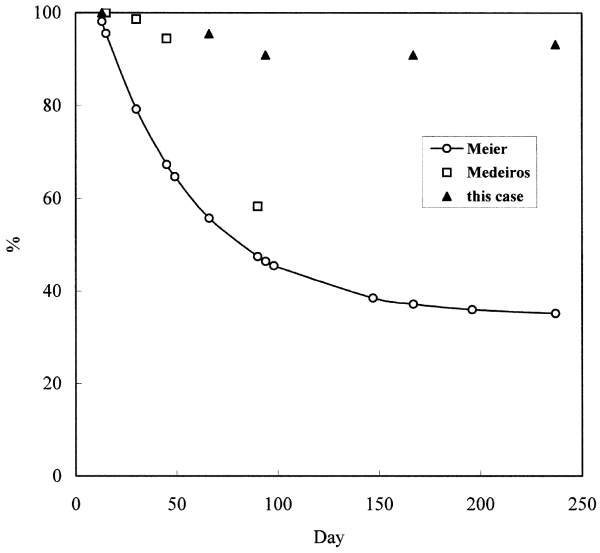

Fig. 3 Time course of axonal loss. Comparisons of our case and that presented by Medeiros with the regressive curve of axonal loss proposed by Meier et al.

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使用視網膜神經厚度分析儀及視野機對一外傷性視神經病變患者做序列的追蹤

郭明澤 賴盈州 鄧美琴

一位 40 歲的男性患者因車禍意外造成右眼外傷性視神經病變。他在受傷後的第 2、第 9、第 13、第 24、第 34 週接受視網膜神經厚度分析儀的檢查；此外，他在受傷後的第 1、第 5、第 16、第 28 週分別接受了手動及自動視野機的追蹤。視網膜神經厚度分析儀的檢查結果在受傷後的第 2 週幾乎完全正常，但視野檢查的結果卻顯示出下視野的嚴重缺損。在受傷後的第 9 週視網膜神經厚度分析儀的檢查結果上峰明顯偏離正常，而一些參數 (the Number, Superior/Nasal, Ellipse Mod., Max. Mod., Symmetry, Super. Ratio) 也隨著時間逐漸變地更差。我們由此推論，對於外傷性視神經病變而言，視野的缺損可能會比視神經厚度的減少出現的快。也因此，對於此類患者使用視網膜神經厚度分析儀來檢查視神經受傷程度時，受傷 2 週內判斷價值可能會受到限制。(長庚醫誌 2005;28:581-6)

關鍵字：外傷性視神經病變，視神經損傷，視野，視網膜神經厚度分析儀，GDx。

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