European Journal of Ophthalmology / Vol. 16 no. 6, 2006 / pp. 847-850

Long-term outcome after conservative treatment of indirect traumatic optic neuropathy

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PURPOSE. *To report the long-term outcome of patients with indirect traumatic optic neuropathy (TON) which showed useful vision for a short period after trauma.*
METHODS. *A cohort of 12 TON patients treated with steroids megadose immediately after trauma was followed every 6 months for an overall period of 5 years. Other than a full neuro-ophthalmologic examination, each visit included quantitative Goldmann perimetry and pattern reversal visual evoked potentials. The results of each examination were compared with the visual function at baseline. The main outcome measures were visual acuity and visual field. Data were analyzed using the Wilcoxon signed-rank test. A p value of less than 0.05 was considered statistically significant.*
RESULTS. *All patients showed a stable visual function 5 years after optic nerve trauma. There was no difference in visual acuity levels (p=0.65) and no visual field surface area between the visit at baseline and the last follow-up. However, a significant improvement in visual field extension (p=0.036) was observed after perimetry evaluation.*
CONCLUSIONS. *This cohort of patients clearly demonstrates that the residual visual function found in the short term after TON is maintained for at least 5 years. These findings add further important clinical information for patients with TON. Furthermore, these data may be helpful to better quantify morbidity related to optic nerve trauma and its permanent sequelae. (Eur J Ophthalmol 2006; 16: 847-50)*

KEY WORDS. *Traumatic optic neuropathy, Long-term visual outcome, Steroids megadose, Visual field comparison*

Accepted: June 26, 2006

INTRODUCTION

Indirect damage to the optic nerve is the most common form of traumatic optic neuropathy (TON), which is a rare but often devastating complication of closed head trauma. Patients with TON are generally young and their visual outcome is light perception (LP) or no light perception (NLP) in 50% of cases; this makes TON a significant cause of severe permanent visual loss (1, 2). At present, there is no proved form of treatment for this disease and there are different opinions about how to manage with it (2-4). For these reasons, TON is a challenging condition for physicians, who find many difficulties in the clinical management of patients with such a problematic neuropathy.

Once TON diagnosis is made, three frequently asked questions are as follows: “Will I regain my sight?” “Is there any treatment to improve my sight?” “How long will my residual visual function (if any) last?”

This latter question regards TON patients who show a residual visual function after trauma. In 70% of cases visual acuity is severely reduced; however, the visual function left is important in the affected side. As a matter of fact, residual visual field may be very useful in these people’s future. For this reason, patients with TON are strongly concerned about the possibil-

This work was presented at the ARVO meeting; May 2005; Fort Lauderdale, FL

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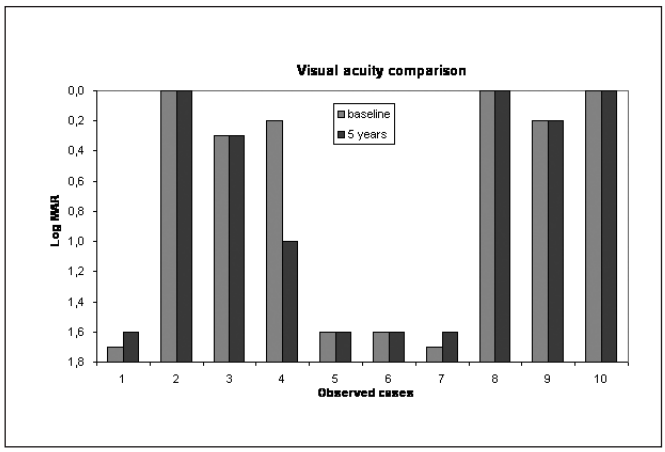

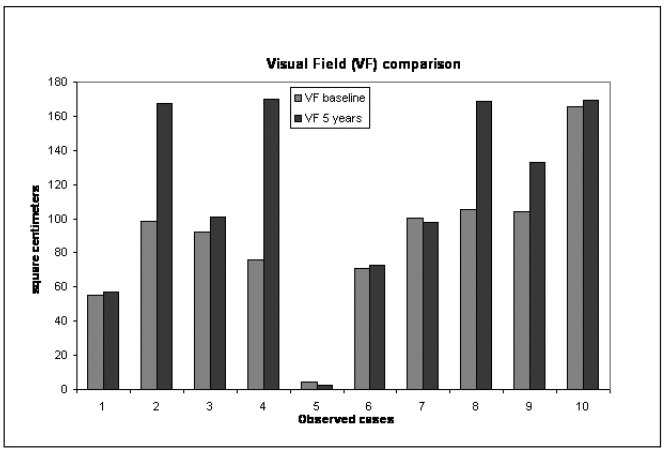

Fig. 1 - *Visual function (VF) comparison.*

ity of losing the preserved sight in the long term.

In a 5-year follow-up study, Mariak et al concluded that high-dose steroid therapy of TON may fail to protect the optic nerve permanently (5). According to the authors, unfavorable long-term results of conservative treatment in patients with TON are possible in clinical practice, despite satisfactory immediate effects.

After more than 10 years of experience in the main trauma center of our region, our clinical impression contrasts with what was observed in Mariak et al study (5): the visual function maintained in the short term proves generally stable and preserved for several years after trauma. So far, no prospective studies revealed if the maintained or recovered vision after TON is stable in a long-term follow-up. On this basis, we performed a 5-year noncomparative prospective study on a cohort of patients treated with steroids megadose immediately after trauma who preserved useful vision after an initial damage of the optic nerve.

TABLE I - PATIENT DEMOGRAPHICS

No. of patients	10
Age, yr, mean (SD)	42 (11)
Male, n (%)	6 (60)
Right side, n (%)	6 (60)
Injury type, n (%)	
Vehicle/bicycle accident	6 (60)
Domestic fall	2 (20)
Sports trauma	2 (20)
Baseline BCVA, n (%)	
HM	2 (20)
CF	2 (20)
20/40	1 (10)
20/32	2 (20)
20/20	3 (30)
Baseline VF area (cm^2), n (%)	
0–60	2 (20)
60–120	7 (70)
120–180	1 (10)
Median	95

VF Normal surface area = 165 to 172 cm^2
BCVA = Best-corrected visual acuity; HM = Hand motion
CF = Counting fingers; VF = Visual field

METHODS

This study followed a cohort of non-randomized patients with TON who underwent the same treatment within 24 hours from trauma and consisting of steroids megadose according to NASCIS III protocol (6). The cohort of patients examined arise from a large group of TON cases whose characteristics have been previously reported in a retrospective multicentric study (4). At the end of that study, we followed a cohort of 12 patients for 5 years, in order to obtain significant data. The baseline evaluation for the current cases examined was the follow-up visit 3 months after trauma. Patients were scheduled at 3, 6, and 12 months and then at yearly intervals.

The study protocol at baseline and during each follow-up visit included a full neuro-ophthalmologic examination performed by the same neuro-ophthalmologist (A.C.). Best-corrected visual acuity (BCVA) was measured using the Ferris-Bailey charts (Lighthouse Low Vision Products, Long Island City, NY). The pa-

tients who had a BCVA lower than 20/200 were tested by count fingers (CF), hand motion (HM), LP, or NLP. For statistical purposes, Snellen fractions were converted to logMAR equivalents. A 0.1 change in the logMAR visual acuity score represented a one-line change in visual acuity. Visual acuity measured by CF, HM, LP, and NLP was assigned logMAR values of 1.6, 1.7, 1.8, and 1.9, respectively. A variation in visual acuity was considered significant when showing a difference of at least 0.1 logMAR between baseline and the last follow-up visit.

Visual field examination was performed using Goldmann perimetry. Each visual field was later digitized on a personal computer by using a high resolution scanner (Epson Perfection 1660 photo).

We made a comparison between each patient's visual perceived area at baseline and 5 years later using the AutoCAD 2000 software program (Autodesk Inc., San Rafael, CA). We drew a line around the perceived visual space and calculated the surface. As regards central scotomas, their areas were also measured for each patient and subtracted from the overall area of the entire visual field left. A variation in visual field extension was considered significant when the surface area after 5 years showed a 10% difference if compared with the baseline one.

Pattern reversal VEP (PRVEP) were performed according to the standardized ISCEV clinical protocol (7). To increase the sensitivity of PRVEP to a unilateral disease, an interocular amplitude and peak analysis was performed for each patient.

The Wilcoxon signed-rank test was used to compare logMAR and visual field values at baseline and 5 years later. The analyses were made using STATA Statistical Software.

RESULTS

The patients had an average age of 42 years (range 31–53); no difference was found in sex distribution. Ten (83%) of 12 patients completed at least a 5-year follow-up. Two cases were excluded from the study: one for a unilateral glaucoma secondary to traumatic angle recession and the other one for a retinal detachment 2 years after TON. In this latter case, there was no difference in visual function between the baseline and the 2-year follow-up visit. Demographic characteristics of the patients of this study are reported in Table I.

Visual loss was moderate in most eyes, with an average baseline BCVA of 20/40 in the study group (range: LP–20/20). Visual field was severely compromised in all but one case at baseline, with no typical pattern of damage. The average visual field extension was 95 cm^2 at baseline (range: 4–105, normal value between 165 and 172). This means that in nearly all patients the visual field tested immediately after trauma was halved if compared with normal ones. The interocular comparison of PRVEP reveals moderate to severe damage of the optic nerve. Figure 1 displays the visual function of the study patients at baseline and at the last follow-up visit. All patients showed a stable visual function 5 years after optic nerve trauma with no difference in visual acuity levels ($p=0.65$) and no visual field surface area. Indeed, a significant improvement in visual field extension ($p=0.036$) was observed when the perimetry of the last follow-up visit was compared with the baseline one.

DISCUSSION

The cohort of patients clearly shows that residual visual function documented 3 months after TON is identical 5 years later. Despite the limited number of cases, these data reflect our clinical impressions in following such patients, and are in accordance with what has been reported in many experimental optic nerve crush models (8-11). In such models, graded crush of the optic nerve in adult rats leads to retinal ganglion cells (RGCs) loss by primary or secondary degeneration (11, 12). Under experimental conditions, even mild insults cause a relatively massive loss of neurons and extensive secondary degeneration, which stabilize between 1 and 2 months after the primary damage. For these reasons, it is not surprising that visual function observed in our patients 3 months after trauma comes out to be exactly the same 5 years later. We suppose that 3 months after TON all degenerative processes triggered by trauma (such as apoptosis, secondary and tertiary axonal degeneration) are extinguished; thus, there is no reason for further deterioration of the residual visual function in the long term.

At the beginning of the present study, we considered the visual field besides visual acuity to be a significant outcome measure. This has been suggested

by our clinical experience on TON patients, as visual acuity alone is not a satisfactory parameter to evaluate their residual visual function. Once again, our decision is in accordance with what is observed in experimental studies where graded crush of the optic nerve in adult rats reveals a different pattern of functional impairment of RGCs with visual acuity preserved up to a severe axonal loss (11). Therefore, RGCs loss, after inducing an optic nerve lesion, could be patchy in nature and leave groups of undamaged axons and, as a consequence, an intact area of peripheral visual field.

An interesting feature observed in our study is a significant improvement in visual field extension revealed by the comparison of the perimetry at the last follow-up visit and at baseline one ($p=0.036$). We are unable to give an univocal interpretation of these data. As all patients underwent the same therapy in order to prevent a treatment bias in our results, we can only suppose that steroids megadose played a role against the lipoperoxidation of some axons in the acute phase of the disease; it is possible that these borderline axons regained their function after our baseline examination (i.e., 3 months after trauma), thus giving a slight increase in the visual field area. The improvement observed in visual field extension after TON needs confirmation in further studies.

Based on the present study, the spared function of the optic nerve observed 3 months after trauma is identical to the one observed 5 years later. Furthermore, the visual status was exactly the same after comparison between the 7 days post steroids visit and the 5-year examination; thus, in TON cases, the sooner, the better. These findings may be used by physicians as important clinical information and may be helpful to better quantify the morbidity related to optic nerve trauma and its permanent sequelae.

ACKNOWLEDGEMENTS

The authors thank Anna Maria Luvoni for technical assistance in calculating the patients' visual field area with the AutoCAD 2000 software program (Autodesk Inc., San Rafael, CA) and Antonella Piazza for assistance in reviewing the English version of this paper.

The authors have no proprietary interest.

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