

Predictive value of visual evoked potentials, relative afferent pupillary defect, and orbital fractures in patients with traumatic optic neuropathy

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Background: The purpose of this study was to determine the predictive value of flash visual-evoked potentials (VEP), relative afferent pupillary defect, and presence of orbital fractures in patients with traumatic optic neuropathy.

Methods: A prospective study was conducted in 15 patients with indirect traumatic optic neuropathy. All patients underwent a thorough ophthalmic examination. Initial visual acuity, final visual acuity, and relative afferent pupillary defect were determined, and visual acuity was converted into logMAR units. We performed flash VEP and an orbital computed tomography scan in all patients.

Results: There was a good correlation between relative afferent pupillary defect and final visual acuity ($r = -0.83$), and better initial visual acuity could predict better final visual acuity ($r = 0.92$). According to findings from flash VEP parameters, there was a relationship between final visual acuity and amplitude ratio of the wave ($r = 0.59$) and latency ratio of the wave ($r = -0.61$). Neither primary visual acuity nor final visual acuity was related to the presence of orbital fractures in the orbital CT scan.

Conclusion: Patients with traumatic optic neuropathy often present with severe vision loss. Flash VEP, poor initial visual acuity, and higher grade of relative afferent pupillary defect could predict final visual acuity in these patients. Presence of orbital fracture was not a predictive factor for primary visual acuity or final visual acuity.

Keywords: visual acuity, flash VEP, RAPD, orbital fracture, CT scan

Introduction

Traumatic optic neuropathy is a form of optic neuropathy divided into direct and indirect types, depending on whether the optic nerve is damaged due to contact with the agent of trauma or not. Patients usually suffer from some type of craniofacial trauma, but in rare cases they also have orbital or eye injury.^{1,2} Indirect traumatic optic neuropathy is the most common form of traumatic optic neuropathy, and is a clinical diagnosis based on evidence of optic nerve dysfunction in patients who have had craniofacial trauma.³

Patients with traumatic optic neuropathy are generally young and have severe and irreversible loss of vision. Generally, the ocular examination is normal in the early stages except for presence of relative afferent pupillary defect (RAPD). In these cases, performing an orbital computed tomography (CT) scan is necessary to

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detect acute orbital hemorrhages, bony fractures, and also fractures of the optic canal.¹⁻³ The mode of visual loss onset is an important factor in diagnosis of optic neuropathies. Visual loss is rapid after traumatic optic neuropathies, but the pattern of visual loss is gradual after compressive and toxic optic neuropathies.^{1,4}

Once the diagnosis of traumatic optic neuropathy is made, the main issue is predicting the degree of visual function and also prognosis of vision. In most patients, visual acuity is severely reduced and the remaining visual function is of significant value in the affected side. Electrophysiological studies have been used to predict visual outcome after ocular injuries. A variety of methods such as the visual-evoked potential (VEP), flash electroretinography (ERG), and pattern ERG have been used for this purpose.^{1-3,5} VEP measures the cortical activity of the visual system in response to flash or pattern stimulus, and it can be abnormal in any type of optic neuropathy. Visual recovery may be impossible when VEP results are not recordable and, according to some results in unilateral cases of traumatic optic neuropathy, a flash VEP amplitude ratio (affected side to normal side) > 0.5 appears predictive of a favorable visual outcome.⁶

In this study, we measured the quantified parameters of VEP to assess optic nerve dysfunction, in an attempt to find a predictive tool for visual outcome in patients with traumatic optic neuropathy. We also evaluated the predictive value of the RAPD before any treatment in our patients. For traumatic optic neuropathy, there has been controversy about treating patients with high-dose corticosteroids. However, in most studies, it has been demonstrated that corticosteroid therapy does not have any clear benefit for traumatic optic neuropathy.⁷⁻¹¹

Methods

A prospective study was conducted in 15 patients with indirect traumatic optic neuropathy treated at the emergency ward in Farabi Eye Hospital between January 2008 and March 2009. Only patients with acute onset and unilateral ocular injury were included in the study population. Diagnosis of traumatic optic neuropathy was made in the presence of decreased visual acuity after a history of head trauma associated with a relative afferent pupillary defect and after exclusion of other possible diagnoses. The diagnosis in every case was confirmed by an ophthalmologist (S.A.T.) who was expert in that field. The RAPD was graded by the same ophthalmologist (S.A.T.), as a conventional

rule from 1+ to 4+, supposing 4+ as no change in pupillary size. We estimated the best-corrected visual acuity for distance in the affected eye and then converted this into log of the minimum angle of resolution (logMAR) units to provide a numeric scale of visual acuity for the purposes of statistical analysis.^{12,13}

An orbital CT scan was performed for all of the patients to detect any orbital fracture and flash VEP to detect amplitude reduction and latency in the optic nerve. The contralateral eyes were used as the normal group. Patients with ocular injuries other than traumatic optic neuropathy, such as open globe injuries, vitreous hemorrhage, traumatic cataract, choroidal rupture, and pathological intraocular conditions not involving the optic nerve were excluded from the study. A motility examination was performed for all patients to rule out cranial nerve VII palsy or internal ophthalmoplegia.

After obtaining informed consent and a complete initial ophthalmic and systemic evaluation, intravenous methylprednisolone was started in all patients within 12 hours of admission at a dose of 1 g/day in four divided doses for 3 days. Patients were discharged on the fourth day, and were asked to attend follow-up at 2 weeks, and 1 and 3 months later.

Flash visual-evoked potentials

For each patient we conducted the flash VEP according to the clinical guidelines developed by the American Neurophysiology Society.¹⁴ Visual flashes were delivered by light-emitting diode goggles placed over the eyes. Electrodes were placed in the mid occipital, right, and left occipital regions, and at the midline vertex position based on the locations of the left or right earlobe and ground position (any scalp position). Filter setting was performed at a low frequency of 1 Hz, a high frequency of 250 Hz, and notch filter (60 Hz) on-off. The study included 200 flashes for each eye with a stimulus duration of 250 milliseconds, a rate of 1.9 Hz, and a sensitivity up to 100 μ V. Overall averages were measured for each of the four channels of each recording. Waves were labeled I through VI and were described in each channel within the first 250 milliseconds if each of them was measurable. Latencies and amplitudes of each wave were calculated.

For this population, we measured the averages of the aforementioned regions in the eye affected by traumatic optic neuropathy and in the otherwise normal or control eye in each case. The wave on the traumatic optic neuropathy

side that presented at approximately 100 milliseconds after the stimulus and had the greatest amplitude in one of these locations (corresponding to P100 in pattern-reversal VEP studies) was identified. This was typically the second positive wave (P2). The corresponding waves in the same location on the control side were then identified. The amplitudes of these waves were calculated. Flash VEPs were performed within 48 hours after primary injury and all were interpreted by one ophthalmologist (M.M.) who was expert in electrophysiological tests. We considered the ratio of amplitude and latency of the traumatic optic neuropathy eye to the normal eye.

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences (v 13; SPSS Inc, Chicago, IL). The Chi-squared test was used for quantitative and qualitative data. *P* values < 0.05 were considered statistically significant. Pearson's coefficients were calculated for finding statistically significant relationships.

Results

Table 1 enumerates the details of the patients. The average age of the patients was 18.2 (range 9–33) years and the median age was 16 years. The gender ratio of males to females was 4:1. The right eye was more frequently involved (ratio 9:6). Accidents involving cars and machinery were the most common cause of trauma (seven cases). The mean duration of admission and initiation of treatment after trauma was 2.2 (1–7) days. Evidence of orbital wall fractures was found on the orbital CT scan in seven patients (46%) in whom neither primary visual acuity nor final visual acuity was related to the presence of orbital fracture. At the time of presentation, five patients had no light perception. We performed correlation analysis to determine any possible relationships. There was a good correlation between RAPD and initial visual acuity ($r = -0.91$). Moreover, there was a good correlation between RAPD and final visual acuity ($r = -0.83$), and better initial visual acuity could predict better final visual acuity ($r = 0.92$). Only one patient with no initial light perception had improvement in visual acuity. In this patient, initial flash VEP was not recordable, but 3 months after treatment, visual acuity increased to hand motion and flash VEP showed a severe reduction of amplitude and marked latency.

According to the findings from flash VEP parameters, the amount of primary visual acuity loss due to traumatic optic neuropathy was correlated with reduced amplitude ratio in the P2 wave ($r = 0.63$) and the latency ratio of flash VEP waves ($r = -0.68$). Moreover, there was a relationship between final visual acuity and the amplitude ratio of the wave ($r = 0.59$) and its latency ratio ($r = -0.61$). In other words, the lower the flash VEP amplitude and the longer the latency in traumatic optic neuropathy, the worse the visual acuity.

Discussion

Hippocrates first described traumatic optic nerve damage after craniofacial injury.¹⁵ Indirect traumatic optic neuropathy starts as optic nerve dysfunction, without direct disruption of anatomic structures or surrounding associated tissues. Traumatic optic neuropathy is seen in 0.5%–5.0% of patients with closed head trauma.¹⁶

The possibility of traumatic optic neuropathy should be considered in all cases of cranial or maxillofacial trauma, although it is generally an uncommon condition. Indirect injury to the optic nerve following head trauma occurs as a result of shearing of nerve fibers, interruption of blood supply, or secondary edema and hemorrhage following shearing or transection of the optic nerve vessels.^{16–19}

In our study, the mean age was 18.2 years, and 75% of patients were male, which is consistent with other studies showing that traumatic optic neuropathy is more common in young male patients.^{20,21} Due to the difficulty in neuro-ophthalmological testing of visual pathway function, VEP studies are believed to be reliable methods of obtaining valuable information as to whether visual function is intact or not.

Visual recovery may be impossible when VEP results are not recordable.⁶ The orbital CT scan along with VEP is important for the immediate identification of optic nerve trauma.²¹ In our study, there was no relationship between the presence of orbital fracture and severity of visual loss. In a study reported by Lessell,¹⁸ 17 of 33 cases showed craniofacial fracture on imaging, and seven had a fracture intersecting the optic canal, but neither the presence of the fracture nor the location was related to the severity of the optic neuropathy. In another study, it was proposed that posterior rather than anterior orbital fracture on CT imaging had a worse prognosis for visual outcome, but the presence of a blowin or blowout fracture on the CT scan did not significantly predict the visual prognosis.¹³

Table 1 Summary of patient characteristics

Subject	Age	Gender	Eye	Nature of trauma	Time of presentation	Primary snellen visual acuity	RAPD	CT scan findings	Flash VEP	Snellen visual acuity 3 months after treatment	Follow-up duration
1	33 y	F	OS	Trauma with scissors	1 day	NLP	4+	Medial orbital wall fracture	Severe delay in P2	NLP	3 months
2	18 y	M	OD	Motor accident	7 days	NLP	4+	Zygomatic fracture	Reduction in amplitude and severe delay in P2	NLP	3 months
3	12 y	M	OS	Trauma with belt	3 days	HM	3+	No fracture	Reduction in amplitude and delay in P2	FC at 1.5 m	3 months
4	19 y	F	OD	Fall from height	1 day	NLP	4+	No fracture	Reduction in amplitude and delay in P2	NLP	3 months
5	11 y	M	OD	Bullet	2 days	NLP	4+	No fracture	Absence of flash VEP response	HM	3 months
6	15 y	M	OS	Fall from bike	1 day	HM	3+	Orbital hemorrhage	Reduction in amplitude and severe delay in P2	FC at 2 m	3 months
7	15 y	M	OS	Trauma with metallic screw	1 day	HM	3+	Medial orbital wall fracture	Reduction in amplitude and delay in P2	HM	3 months
8	23 y	M	OD	Car accident	2 days	20/200	2+	Medial orbital wall fracture	Severe delay in P2	20/200	3 months
9	32 y	M	OS	Fall from height	1 day	HM	3+	No fracture	Severe delay in P2	20/400	3 months
10	9 y	M	OD	Car accident	3 days	20/160	2+	Medial orbital wall fracture	Severe delay in P2	20/50	3 months
11	10 y	F	OS	Trauma with pipe	1 day	20/200	3+	Medial orbital wall fracture	Reduction in amplitude and severe delay in P2	20/50	3 months
12	30 y	M	OD	Car accident	7 days	20/200	3+	No fracture	Reduction in amplitude in P2	20/30	3 months
13	17 y	M	OD	Motor accident	2 days	NLP	4+	Orbital floor fracture	Absence of Flash VEP response	NLP	3 months
14	16 y	M	OD	Car accident	1 day	HM	3+	No fracture	Reduction in amplitude and severe delay in P2	HM	3 months
15	14 y	M	OD	Car accident	1 day	20/400	2+	No fracture	Severe delay in P2	20/50	3 months

Abbreviations: y, years; CT, computed tomography; VEP, visual-evoked potentials; HM, hand motion; NLP, no light perception.

However, all of our patients with orbital fracture underwent surgical orbital repair, and in the case of orbital hemorrhage we performed canthotomy, although these surgical procedures could be confounding factors in terms of the visual recovery in cases associated with orbital fracture or orbital hemorrhage.

In our study, we found that there might be an association between the amount of amplitude reduction, latency in flash VEP, and poor visual acuity, and the role of latency in this correlation was probably more prominent. These parameters may express the function of the optic nerve. Flash VEP may have a role in anticipating visual outcome after direct ocular injury, but is of limited value in predicting neurological outcome after head trauma.^{21,23–26}

Another prognostic tool is the greater degree of RAPD in patients with traumatic optic neuropathy. Patients who have worse RAPD are likely to have a worse visual prognosis.²⁷ In our study, there was a relationship between primary and final visual acuity and RAPD, so it would be better to quantify RAPD. However, in a study of indirect optic nerve injury in two-wheeler riders in northeast India, the authors mentioned that only flash VEP was related to final visual acuity and neither RAPD nor initial visual acuity was related to final visual acuity. The reason for these conflicting findings could be the different inclusion criteria, given that most of their cases were recruited over a varying period of time following trauma, as mentioned by the authors.¹⁹

It is documented in the literature that the chance of spontaneous improvement is 25%–55%.^{28,29} According to other studies, such as the International Optic Nerve Trauma Study, there is no evidence for treating patients with traumatic optic neuropathy using corticosteroids. However, we treated all of our patients with intravenous methylprednisolone, despite the lack of definite efficacy of this treatment strategy in traumatic optic neuropathy, because the same patients were also participating in another larger study at our center for determining the role of corticosteroids in the treatment of traumatic optic neuropathy, and this might also have been a confounding factor.^{8–11,30,31}

In conclusion, patients with traumatic optic neuropathy often present with severe visual loss. Flash VEP, poor initial visual acuity, and higher grade of RAPD could predict final visual acuity in these patients. Presence of orbital fracture was not a predictive factor for either primary visual acuity or final visual acuity.

Disclosure

The authors report no conflicts of interest in this work.

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