

VISUAL OUTCOME OF TRAUMATIC OPTIC NEUROPATHY IN PATIENTS TREATED WITH INTRAVENOUS MEGADOSE OF STEROIDS

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Abstract- Although uncommon, traumatic optic neuropathy (TON) is an important cause of visual loss. Different therapeutic approaches including different dosages of steroids, surgical decompression of optic canal and observation alone have been suggested but there has been no conclusive evidence to establish a standard approach to this devastating cause of visual loss. To determine the effectiveness of intravenous (IV) steroids in the treatment of these patients, the medical records of patients with TON, including one bilateral case, treated with IV steroids were reviewed. Twenty-eight patients (22 males, 6 females) with mean age of 24.1 (11 to 41 years) were enrolled. All patients had received 30 mg/kg loading dose of methylprednisolone succinate followed by 5.4 mg/kg/ hour for 48 hours. Visual acuity (VA) was improved by ≥ 1 line in 8 eyes (28.6%) immediately after treatment and in 10 eyes (37%) after 3 months; however, most of them (6 and 8, respectively) were in the range of initial VA of no light perception to hand motion. After adjustment for the baseline VA, these improvements in visual acuities were not considered significant. Neither different orbital fractures, nor various extraocular muscle palsies had any significant effect on the prognosis of ultimate VA. Regarding the natural course of TON, this investigation showed that IV megadose steroids had no clear benefit on the visual outcome of patients with TON.

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Key words: Traumatic optic neuropathy, megadose steroid, extraocular muscle palsy, orbital fracture

INTRODUCTION

Traumatic loss of vision, along with deficits in visual field, color perception and an afferent pupillary defect is called traumatic optic neuropathy (TON). Although uncommon, TON which occurs mostly after blunt trauma is an important cause of visual loss (1). It can take two major forms; direct injuries are caused by projectiles or sharp penetrating

objects that enter the orbit, while indirect injuries are caused by concussive forces that are transmitted to the optic nerve as a result of orbitofacial or cranial trauma (1). This impact may generate a shock wave which can lead to optic nerve avulsion or posterior indirect traumatic optic neuropathy (2).

While the diagnosis of indirect TON is made through a careful history taking and examination, its appropriate management has been far from being well defined. Different approaches including different dosages of steroids (60 mg to 7 g/day), surgical decompression of optic canal (via intracranial, transethmoidal, endonasal, sub labial or other techniques) (3-7), and observation alone (8), have been suggested but there has been no conclusive evidence to establish a standard approach to this

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devastating cause of visual loss. Even the investigation of the optimal treatment through the comparison of these different approaches has been far beyond the scopes of the International Optic Nerve Trauma Study (9). Regarding the controversial findings in the literature, and as the first report from Iran, we reviewed the medical records of 28 patients who had TON and received intravenous (IV) steroids at Farabi Eye Hospital to find out the results of such treatment in these patients.

MATERIALS AND METHODS

The study design was existing data analysis. We included patients with indirect optic nerve injuries in otherwise healthy individuals. All enrolled cases had a complete eye examination including best corrected visual acuity (VA) measurement, slit lamp exam, IOP measurement, relative afferent pupillary defect control, ocular motility and fundus examination on admission, immediately post-treatment and at least three months later and had axial and coronal CT scans of orbit accordingly. Patients who lacked any of above mentioned criteria were excluded.

Visual acuity was the main outcome measure of the study, which was measured by Snellen chart on admission, immediately after treatment and 3 months later. Snellen fractions of best corrected VA of the patients were converted to log MAR equivalents. A 0.1 change in the log MAR visual acuity score represents a one line change in VA. Visual acuity recorded as less than 1.5 m counting fingers (CF) hand motion (HM), light perception (LP), and no light perception (NLP) were arbitrarily assigned log MAR values of 1.6, 1.7, 1.8 and 1.9, respectively, for the purpose of analysis. A one line change in VA (equivalent to 0.1 of log MAR) was statistically considered significant.

On admission, which was between 3 hours to 1 week after trauma, all patients received intravenous (IV) methylprednisolone succinate, 30 mg/kg as loading dose (diluted in 500 ml of 5% glucose solution) and then 5.4 mg/kg/hour (diluted in 250 ml 5% glucose solution) was infused within 6 hours for the next 48 hours. Then the patients were placed on oral prednisolone (50 mg/day) in a tapering fashion for two weeks. For patients with optic canal fracture

who remained NLP despite IV steroids, optic canal decompression was offered, although none of them yielded to this surgical remedy.

RESULTS

The data of 28 patients were analyzed. Mean age of the patients was 24.1 years (11 to 41) and 22 were male and 6 were female. Vehicle accidents (including bicycle and motorbike) were the main causes of trauma (71.4%). Majority of patients had poor VA (NLP to HM, 23 cases) at first examination (Table 1). Associated extraocular palsies and orbital fractures are shown in tables 2 and 3.

Visual acuity improved by one line in 8 eyes (28.6%) immediately after treatment and in 10 eyes (37%) after 3 months; however, most of them (6 and 8 cases, respectively) were in the range of initial VA of NLP to HM (Table 4). After adjustment for the baseline VA and by Fisher's exact test, this improvement in VA was not considered significant (Table 5).

Table 1. Demographic and clinical characteristics*

| Characteristic | Total (n= 28) |
|---------------------------|---------------|
| Age | 24.10 (8.33)† |
| Sex | |
| Male | 22 (78.6) |
| Female | 6 (21.4) |
| Eye | |
| Right | 11 (39.3) |
| Left | 16 (57.1) |
| Both | 1 (3.9) |
| Injury type | |
| Vehicle/ bicycle accident | 20 (71.4) |
| Assault | 5 (17.9) |
| Fall | 3 (10.7) |
| Baseline visual acuity | |
| NLP, LP, HM | 16 (57.1) |
| < 20/ 200 to CF | 7 (23.0) |
| < 20/ 40 to ≥ 20/ 200 | 4 (14.3) |
| ≥ 20/ 40 | 1 (3.6) |

Abbreviations: NLP, no light perception; SD, standard deviation; CF, counting fingers; LP, light perception; HM, hand motion.

*Data are given as number 9percent) unless specified otherwise.

†Mean (SD).

Table 2. Frequency of different paresis

| Paresis | Number (percent) |
|-----------------------------|------------------|
| Ptosis | 7 (25) |
| Pupil palsy | 10 (35.7) |
| 3rd nerve palsy | 4 (14.3) |
| 6 th nerve palsy | 2 (7.1) |
| Medial rectus palsy | 2 (7.1) |
| Total ophthalmoplegia | 6 (21.4) |

Table 3. Frequency of different orbit fractures

| Type of fracture | Number (percent) |
|------------------------------------|------------------|
| Orbit fx (total) | 20 (71.4) |
| No fracture | 8 (28.6) |
| Optic canal fx (total) | 8 (28.6) |
| Ethmoid fx (total) | 14 (50.0) |
| Sphenoid fx (total) | 12 (42.9) |
| Maxillary fx (total) | 2 (2.1) |
| Zygomatic fx (total) | 2 (7.1) |
| Tripod fx (total) | 3 (10.7) |
| Eth + Sph fx | 6 (21.4) |
| Eth + Sph + Optic canal fx | 3 (10.7) |
| Eth fx (alone) | 2 (7.1) |
| Optic canal fx (alone) | 2 (7.1) |
| Max fx (alone) | 1 (3.6) |
| Zyg fx (alone) | 1 (3.6) |
| Optic canal + Trp fx | 1 (3.6) |
| Zyg + Optic canal fx | 1 (3.6) |
| Eth + Sph + Max fx | 1 (3.6) |
| Eth + Sph + Trp fx | 1 (3.6) |
| Eth + Sph + Trp + sella turcica fx | 1 (3.6) |
| Total | 28 (100) |

Abbreviations: fx, fracture; Eth, ethmoid; Sph, sphenoid; Max, maxillary; Zyg, zygomatic; Trp, Tripod.

Table 4. Frequency of different visual acuities in assessment times

| VA | First visit (n=28) | Immediately post-treatment (n=28) | After 3 months (n=25) |
|-------|-----------------------|---|-----------------------------|
| NLP | 16 | 14 | 10 |
| LP | 0 | 0 | 1 |
| HM | 7 | 5 | 6 |
| CF | 4 | 7 | 3 |
| ≥ 0.1 | 1 | 2 | 5 |

Abbreviation: VA, visual acuity; NLP, no light perception; LP, light perception; HM, hand motion; CF, counting fingers.

Different extraocular palsies from ptosis to total ophthalmoplegia were noted (Table 2) which did not have significant effect on the visual outcome in the affected patients (Table 5). This was also the case when type of injury and ultimate visual acuity were considered (Table 5). Twenty patients had one to multiple orbital fractures (Table 3). Since orbital fractures are of most prognostic importance, overall visual outcome of this group of patients was compared with no fracture group and it failed to prove to be significant (Fisher's exact test).

DISCUSSION

Hippocrates might have been the first to identify the phenomenon of acute and delayed vision loss after injuries placed to and slightly above the brow (2). While there is little controversy on the macroscopic mechanism of trauma to the optic nerve, including the deceleration theory (1), multiple hypotheses have been proposed at microscopic level of damage to the optic nerve, including contusion necrosis, nerve fiber tears and nerve infarction secondary to closed space edema, hemorrhage, thrombosis, vasospasm, impingement by bone spicules, and shearing of dural vessels in the optic canal (10).

Treatment of TON has been a topic of controversy and there has not yet been any answer to this question (11-16). Even after 2 years, the International Optic Nerve Trauma Study (TIONTS) failed to recruit enough eligible patients to conduct a clinical trial to compare the results of steroid only arm with surgery plus megadose steroid arm (9). Therefore, it was transformed into an observational study and ultimately found no clear benefit for either corticosteroid or optic canal decompression approach. The idea of the use of megadose steroids is extrapolated from traumatic spinal cord injury studies (2). Although the exact mechanism of its action is not clear yet, it seems that the main mechanism by which corticosteroids are thought to block neuronal death in the setting of trauma is through inhibition of free radicals and not through activation of glucocorticoid receptors (10).

Table 5. Percentage of patients with a line improvement in visual acuity according to different baseline characteristics

| | Immediately post treatment (n=28) | | After 3 months (n=27) | |
|----------------------------------|-----------------------------------|---------|-----------------------|---------|
| | Frequency | P value | Frequency | P value |
| Baseline VA | | | | |
| NLP, LP, HM | N= 23 | | N= 23 | |
| ≥1 line improvement | 6 (26.1%) | 0.66* | 8 (34.7%) | 0.613* |
| CF or better | N= 5 | | N= 4 | |
| ≥ 1 line improvement | 2 (40.0%) | | 2 (50.0%) | |
| Concurrent orbit fracture | | | | |
| Without fracture | N= 8 | | N= 8 | |
| ≥1 line improvement | 3 (37.5%) | 0.651* | 4 (50.0%) | 0.415* |
| With fracture | N= 20 | | N= 19 | |
| ≥ 1 line improvement | 5 (25.0%) | | 6 (31.6%) | |
| Injury type | | | | |
| Vehicle/bicycle accident | N= 20 | | N= 19 | |
| ≥ 1 line improvement | 5 (25.0%) | | 7 (21.0%) | |
| Assault | N= 5 | 0.813* | N= 5 | 0.954* |
| ≥ 1 line improvement | 2 (40.0%) | | 2 (40.0%) | |
| Fall | N=3 | | N= 3 | |
| ≥ 1 line improvement | 1 (33.3%) | | 1 (33.3%) | |
| Concurrent nerve paresis | | | | |
| Without nerve paresis | N=12 | | N= 11 | |
| ≥ 1 line improvement | 4 (33.3%) | 0.691* | 4 (36.3%) | 0.953* |
| With nerve paresis | N= 16 | | N= 16 | |
| ≥ 1 line improvement | 4 (25%) | | 6 (37.5%) | |

Abbreviations: VA, visual acuity; NLP, no light perception; LP, light perception; HM, hand motion; CF, counting fingers.

* Fisher exact test.

Indeed, glucocorticoid activity is less important in the purported mechanism than the ability of these agents in high doses to scavenge free radicals and prevent lipid peroxidation, perhaps a final pathway in white matter injury. They may also enhance blood flow (2). Despite these theoretical benefits, our treatment results are in concert with those of TIONTS (9), reminding the evidence of spontaneous recoveries reported in the literature (8, 9, 17).

Vehicle accidents were the main cause of TON in our study, while assaults had the second highest incidence rate (Table 1), a finding similar to a report from Glasgow (18).

Similar to the most reports (9, 19, 20), our findings indicate that patients with poor initial visual acuities (NLP to HM) have poorer visual prognosis. Although our study had limited number of patients, but 71.4% of them had at least one orbital fracture

(Table 3) with different degrees of extraocular nerve palsies (Table 2) but in contrast to the conclusion of some reports (21), we failed to show significance of these findings and their effect on final visual results (Table 5).

Despite the fact that our study suffers from limited number of eligible patients, the assumption of minimum change in visual angle as the criteria for statistically significant improvement in VA instead of doubling of visual angle (0.1 log MAR vs 0.3 log MAR change) and delay in institution of treatment in some patients, comparing our results with natural course of TON shows that intravenous steroids have no significant effect on the visual outcome of patients with TON. It also indicates that concurrent orbito-facial fractures, extraocular nerve palsies and type of injury have no relevance to the final visual results.

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