## Visual outcome following mega dose corticosteroid therapy in indirect traumatic optic neuropathy

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**Abstract: Purpose**: To evaluate the visual outcome following mega dose corticosteroid therapy in indirect traumatic optic neuropathy (ITON). **Methods**: In the retrospective study, the medical records of 18 patients who were treated with intravenous mega dose Methylprednisolon due to ITON were evaluated. Mega dose Methylprednisolon was administered to all patients during the first 3 days after trauma. The main outcome measures were visual acuity at 3 months after treatment (final visual outcome). **Results**: Visual acuity at presentation for 18 patients ranged from no light perception to 1.85 log mar .The mean age of patients was 28.27 $\pm$ 7.73 years old. No visual improvement was noted in patients with initial visual acuity of no light perception. There was no statistically significant difference between the initial and final visual outcome (*P* =0.08) in patients with initial visual acuity of 2.3 log mar or better. **Conclusion**: Our results demonstrate that the mega dose corticosteroid therapy has no significant effect on visual outcome of ITON.

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### 1. Introduction

Traumatic optic neuropathy leads to significant visual acuity and visual field loss with relative afferent papillary defect in an otherwise clear optical media and normal retina. It can be either due to direct or indirect trauma. Indirect traumatic optic neuropathy (ITON) is usually caused by transmitted shock wave of blunt trauma to the ipsilateral forehead. Diagnosis is made by clinical exam and history of trauma (Sadeghi-Tari, 2005; Noble and Mc Fadzean, 1987; Davidson, 1938; Glaser, 1999; Steinsapir, 1999).

There is no accepted treatment modality, though; Intravenous steroids, surgical decompression and just observation are among the advocated treatment modalities (Wolin and Lavin, 1990; Joseph, 1990; Lubben, 2001).

The idea of mega dose Methylprednisolon in treatment of traumatic optic neuropathy arises from its partial effectiveness in spinal cord injury, but the similar effect in traumatic optic neuropathy was controversial (Steinsapir, 1999; Steinsapir and Goldberg, 1998). Therefore, in this study we evaluate the visual outcome following mega dose corticosteroid therapy in ITON.

### 2. Material and Methods

In this retrospective study, the medical records of 18 patients who were treated with intravenous mega dose Methylprednisolon due to ITON in Nikookari Eye Hospital from 2006 through 2011 were evaluated. The diagnosis of ITON was based on significant visual loss with relative afferent papillary defect, normal slit lamp and fundus examination and history of trauma. All patients had normal orbital computed tomography.

The informed consent was obtained from all patients. All patients treated with intravenous mega dose Methylprednisolon during the first 3 days after trauma (30mg/kg bullous dose and 15mg/kg every 6 hours for 3 days). The main outcome measures were visual acuity immediately and at 3 months after treatment.

For statistical analysis, count finger was assigned equvalent to 1.85 log mar and hand motion was assigned equivalent to 2.30 log mar. Patients with no light perception and light perception were excluded from analysis as no light perception and light perception are not real visual acuitis<sup>10</sup>. The data were analyzed with the SPSS statistical package (version16; SPSS, Inc, Chicago, IL). Analysis was conducted by Wilcox on signed ranks test. P value less than 0.05 was considered significant.

### 3. Results

The mean age of patients was  $28.27\pm7.73$  (range of 20 to 50 years old). 13 patients were male and 5 patients were female. Visual acuity at presentation for 18 patients ranged from no light perception to 1.85 log mar (Table 1).

There were 4 (22%) patients with initial visual acuity of light perception or no light perception. No visual

improvement was noted in patients with initial visual acuity of no light perception or light perception.

Table.1.	Visual	acuity	ofp	oatients

Visual acuity	First visit	Immediately post treatment	After 3 months
NLP*	2	2	2
LP**	2	2	2
2.3 log mar	6	3	1
1.85 log mar	8	11	10
>1 log mar	0	0	3

\* No light perception; \*\* Light perception

There were 14 (78%) patients with initial visual acuity of hand motion or better. The mean initial visual acuity of patients was  $1.97\pm0.21\log$  mar. The mean visual acuity immediately after treatment was $1.85\pm0.29 \log$  mar. The improvement of visual acuity after treatment was not significant in these patients (*p*=0.1). Visual acuity improved in just 3 of 14 patients (22%) at 3 months after treatment. The mean visual acuity of patients at 3 month was  $1.57\pm0.54 \log$  mar. There was no statistically significant improvement of the initial visual acuity at 3 months after treatment (p=0.08).

Neither life threatening complications due to mega dose corticosteroid therapy nor worsening of vision was encountered in our series.

### 4. Discussions and conclusion

Inhibition of lipid peroxidation at the site of injury and suppresses the breakdown of membrane is the main effect of high dose corticosteroid therapy in spinal cord injury(Braughler, 1987). Also, high doses of methylprednisolone markedly enhance the flow of blood in injured spinal cords(Young and Flamm, 1982). The second cranial nerve is not a true nerve but a fiber tract of the brain. Also retina is a specialized structure of the brain adapted for purpose of vision (Steinsapir, 1999) Therefore, it seems that the mentioned result may be true in traumatic optic neuropathy.

Our results demonstrate that mega dose corticosteroid therapy had no significant effect on visual outcome of ITON. Also, the International Optic Nerve Trauma Study failed to show a clear benefit from corticosteroid therapy or optic nerve decompression in traumatic neuropathy(Levin, 1999). Sadeghi et al failed to show significant improvement of visual acuity by high dose corticosteroid therapy (Sadeghi-Tari, 2005). Steinspair et al believe that clinical evidence does not support using of high dose corticosteroid for traumatic optic neuropathy and it should be abandoned till there are new and compelling data (Steinsapir and Goldberg, 2011).

Furthermore, there is no animal model of ITON demonstrating a benefit for corticosteroid therapy at

any dose (Steinsapir, 2006). However, some studies show improvement of vision after corticostoid therapy (Kountakis, 2000).

Some reports found spontaneous recovery of visual acuity in traumatic optic neuropathy ranging from 20% to 38% of patients (Wolin and Lavin, 1990; Seiff, 1990). In our study, visual acuity improved in 3 of 14 patients (21%) at 3 months after treatment. Therefore, these improvements may be related to this phenomenon.

There are several animal studies that show high dose corticosteroid may be toxic to injured optic nerve. They found that the animals treated by saline retained greater numbers of axons comparing with those treated with corticosteroid (Steinsapir, 2000). However, there was no worsening of vision in our study.

In conclusion, we failed to show any significant improvement in visual acuity after mega dose corticosteroid therapy in ITON.

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