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Editorial

How to manage traumatic optic neuropathy?

In this issue of the *Taiwan Journal of Ophthalmology*, a review article on traumatic optic neuropathy by Dr Patrick Yu-Wai-Man[1] is included. The article reviews the clinical features and current evidence of treatment for traumatic optic neuropathy. The controversy surrounding the use of steroid in the management of traumatic optic neuropathy has been a matter of debate over a long period. Dr Patrick Yu-Wai-Man[1] has reviewed the background and rationale for the use of steroids and surgical decompression. Recent evidence of the benefit and disadvantage in each therapy has been described as well.

1. Treatment options for traumatic optic neuropathy

There are several options for the treatment of traumatic optic neuropathy, with steroid use and surgical decompression of the optic canal being the two main therapeutic modalities. Both of these modalities aim to decrease the optic nerve pressure within the optic canal because the main pathogenic mechanism hypothesizes that trauma-induced edema within the bony confines of the optic canal may further compromise the vascular supply of the optic nerve and cause secondary neuronal cell death.[1] Thus, decreasing optic nerve edema by steroid use or removing the bony confines of the optic canal by surgical procedure may help prevent secondary cell loss. In addition to these two treatment modalities, many doctors now adopt a conservative strategy such as clinical observation, because there is a high percentage of spontaneous recovery following traumatic optic neuropathy.[2]

2. Rationale for steroid treatment

As Dr Patrick Yu-Wai-Man[1] mentions in his review article, the use of steroid has become a popular choice for the treatment of traumatic optic neuropathy after the 1980s because of the successful use of steroids in the second National Acute Spinal Cord Injury Study (NASCIS II).[3] The NASCIS II was a multicenter, randomized, double-blind, prospective clinical trial conducted in 1990, and its results showed that patients receiving a megadose of steroid (initial dose, 30 mg/kg, then 5.4 mg/kg/h for 23 hours) within 8 hours of spinal injury may have better neurological functions and less residual sequelae. In the next trial (i.e., NASCIS III), the authors reported an even better outcome of motor function if the treatment lasts for 48 hours instead of 24 hours.[4] Traditional use of steroids (high dose, 1000 mg/d for 3 days) may reduce edema and anti-inflammation. When administered in megadoses, steroids are proposed to have an additional neuroprotective effect through the mechanism of reducing oxidative stress and decreasing reactive oxygen species.

3. Changing trend in steroid use

In the UK surveillance of traumatic optic neuropathy, Lee et al[5] found a total of 116 cases of traumatic optic neuropathy from 2004 to 2006. Only 41 (35%) of these 116 patients received steroid treatment, whereas the remaining 75 (65%) patients received clinical observation alone.[5] This implies that there is a trend toward more conservative treatment for traumatic optic neuropathy. This changing trend may result from the following evidence.

3.1. Applicability of the NASCIS II

Although administration of steroids in megadoses has been proved to have a neuroprotective effect on the acute spinal cord injury, it remains uncertain whether it has the same effect in optic nerve injury. The optic nerve is mainly composed of axons from retinal ganglion cells intermixed with glia, vessels, and connective tissues. By contrast, the spinal cord has gray matter and white matter and it includes motor neurons, interneurons, glia, and motor and sensory axons. They are different functionally and anatomically. Thus, extrapolating the successful outcome of NASCIS II to the treatment of traumatic optic neuropathy is not without concern.

In addition, the beneficial effect of megadose steroid is limited to patients receiving treatment within 8 hours. Patients who received treatment after 8 hours may have a worse outcome than the control group.[3] Most patients with traumatic optic neuropathy came for eye care later than 8 hours, because some patients may have concurrent craniofacial injury or even conscious disturbance at the initial stage. Thus, the applicability of NASCIS II results to clinical management of traumatic optic neuropathy remains uncertain.

3.2. Absence of large-scale clinical trial

It is difficult to recruit a large number of patients with traumatic optic neuropathy in a short period. Besides, the clinical condition is complex as some patients may suffer from concurrent craniofacial injury or conscious disturbance. It is difficult to assess visual

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function at the initial stage in traumatic optic neuropathy, and only approximate results are available most of the time. In addition, visual impairment may vary widely following an optic nerve injury. With all these limitations, to-date there is no large-scale, randomized, double-blind, prospective clinical trial for traumatic optic neuropathy. Only one small-scale, randomized, prospective clinical trial was found in the Cochrane report by Drs Patrick Yu-Wai-Man and Griffiths.[6] In this small trial, Dr Entezari et al[7] examined the effect of high-dose steroid (250 mg/6 h for 3 days, and oral steroid 1 mg/kg/d for 14 days) in 31 patients with traumatic optic neuropathy. There was no significant difference in visual outcome between patients who received steroid treatment and those who did not.[7]

3.3. Increased mortality in patients with combined significant head injury

The use of megadose steroid (initial dose, 2 g, followed by 0.4 g/h for 48 hours) was found to be associated with a higher mortality rate (21.1%), compared with the rate in the control group (17.9%), at 2 weeks following significant head injury in a large-scale clinical trial—Corticosteroid Randomization After Significant Head Injury.[8] This indicates that the megadose steroid use should be avoided in traumatic optic neuropathy patients with combined severe head injury.

3.4. High spontaneous recovery rate in traumatic optic neuropathy

A high spontaneous recovery rate is observed in patients with traumatic optic neuropathy.[2] This evidence supports conservative management of clinical observation to obviate potential complications arising from steroid use and surgical decompression.

4. How to manage traumatic optic neuropathy?

With no obvious advantage from any treatments,[1,2,9–12] clinical doctors may choose their favorable way of management. Dr Volpe and Dr Levin[12] presented an in-depth discussion of this specific issue in the *Journal of Neuro-Ophthalmology* in 2011. The current strategy to manage patients with traumatic optic neuropathy is suggested as follows.

Patients with traumatic optic neuropathy may choose to receive steroids, surgical decompression, or clinical observation. The benefit of surgical decompression for traumatic optic neuropathy remains obscure,[2,9] partly because visual outcome greatly varies depending on surgeon's experience and technique level. In each hospital, whether eye doctors choose to recommend surgical decompression or not mainly depends on if they have available expertise. Clinical observation is also a good choice because there is a high spontaneous recovery rate,[1,2] and it may avoid the potential side effects from the steroid and surgery. However, it seems hardly acceptable to do “clinical observation” alone as the treatment for traumatic visual loss, especially in the local health care culture here (Taiwan).

With regard to the use of steroid treatment, it is suggested that the possible benefits and side effects should be clearly explained to the patients. Clinical parameters, such as blood sugar and intraocular pressure, should be carefully monitored during the course of treatment. The dose of steroid treatment depends on the arrival time.

4.1. Patients arrive within 8 hours after trauma

If they arrive earlier than 8 hours after injury, megadose steroid may be instituted for the possible benefit of neuroprotection. If there is any sign of associated craniofacial injury, the neurosurgical department should be consulted first. There are several conditions unsuitable for the megadose use, for example, concurrent severe head injury, conscious disturbance, inability to assess visual function, or severe eyeball injury. For these complicated conditions, traditional high-dose steroid would be a better choice.

4.2. Patients arrive later than 8 hours after trauma

If the patient arrives later than 8 hours following injury, traditional high-dose steroid is suggested. High-dose steroid has the effect of reducing tissue edema and anti-inflammation, and most importantly, with a reasonable risk of drug-related side effects.

References

1. Yu-Wai-Man P. Traumatic optic neuropathy—clinical features and management Issues. *Taiwan J Ophthalmol*. 2015;5:3–8.
2. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. *Ophthalmology*. 1999;106:1268–1277.
3. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med*. 1990;322:1411.
4. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA*. 1997;277:1597–1604.
5. Lee V, Ford RL, Xing W, Bunce C, Foot B. Surveillance of traumatic optic neuropathy in the UK. *Eye (Lond)*. 2010;24:240–250.
6. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. *Cochrane Database Syst Rev*. 2013;6 [CD006032].
7. Entezari M, Rajavi Z, Sedighi N, Daftarian N, Sanagoo M. High-dose intravenous methylprednisolone in recent traumatic optic neuropathy; a randomized double-masked placebo-controlled clinical trial. *Graefes Arch Clin Exp Ophthalmol*. 2007;245:1267–1271.
8. Roberts I, Yates D, Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): randomised placebo-controlled trial. *Lancet*. 2004;364:1321–1328.
9. Joseph MP, Lessell S, Rizzo J, Momose KJ. Extracranial optic nerve decompression for traumatic optic neuropathy. *Arch Ophthalmol*. 1990;108:1091–1093.
10. Seiff SR. High dose corticosteroids for treatment of vision loss due to indirect injury to the optic nerve. *Ophthalmic Surg*. 1990;21:389–395.
11. Spoor TC, Hartel WC, Lensink DB, Wilkinson MJ. Treatment of traumatic optic neuropathy with corticosteroids. *Am J Ophthalmol*. 1990;110:665–669.
12. Volpe NJ, Levin LA. How should patients with indirect traumatic optic neuropathy be treated? *J Neuroophthalmol*. 2011;31:169–174.

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