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Editorial

Treatment of Traumatic Optic Neuropathy (TON) with Megadose Methylprednisolone (MDMP)- Pros and Cons

Traumatic optic neuropathy (TON) is a condition in which there is severe impairment of vision due to acute injury to the optic nerve as a result of ocular trauma or head injury.

It is more common among males and working-age groups. There is an increase in the incidence of TON due to gradual urbanisation, industrialization, and road traffic accidents or assault[1]. The overall incidence of traumatic optic neuropathy (TON) is 0.07-2.5%[2]. Direct TON is frequently associated with severe vision loss and a lower chance of recovery compared to indirect TON.

The site of injury is most commonly the forehead and supra orbital ridge. The site of the lesion is the optic canalicular portion of the optic nerve.

Pathophysiology of TON:

Currently two basic mechanisms of TON are known. These are direct TON (DTON) and indirect TON (ITON). The former causes direct damage to the optic nerve fibre, and the latter causes damage to the optic nerve fibres when rotational or shearing force is transmitted to the frontal area in a closed head injury[3]. Indirect damage may be produced by oedema, haemorrhage, or disruption of microcirculation. Oedema and haemorrhage cause further damage by exerting a compressional effect on the optic nerve within the rigid optic canal.

Traumatic optic neuropathy (TON) is characterised by

i. Impairment of vision.
ii. Visual field defect (VFD).
iii. Colour vision defect.
iv. Relative afferent pupil defect (RAPD) is the most important finding in a compressive optic nerve lesion. Paradoxical dilatation of pupil when light is focused on the affected eye.
v. Fundus: Initially it appears normal.
vi. Eventually, optic atrophy may supervene after 4 to 6 weeks.

Pros: Treatment of TON with megadose steroid:

Several studies have suggested that about 25% to 50% of patients with isolated TON undergo some degree of spontaneous improvement without any treatment, and the rest of the patients require megadose steroid in order to have an increased likelihood of recovery. It is believable that even “no light perception of vision” is not a contraindication for considering treatment, as improvement of vision has been observed in some of these patients, not necessarily 6/6 vision[4].

Megadose steroid reduces oedema, inflammation, and swelling of the optic nerve and prevents secondary damage from the compression of the optic nerve within the rigid bony optic canal. It is presumed that megadose of steroid also prevent oxidative damage to the optic nerve and offer neuroprotection.

There are two regimes of megadose methyl prednisolone (MDMP):

i. Methylprednisolone 30 mg/kg loading dose I/V followed by a continuous infusion of 5.4 mg/kg/h for 24 hours.
ii. 2 gm methylprednisolone loading dose, followed by 0.4 g/h over 24 hours.

Cons: Treatment of TON with megadose steroid:

The initial concept and enthusiasm for the use of megadose steroid were derived from national acute spinal cord injury study (NASCIS) where megadose regimens have been shown to benefit traumatic cord lesions as evidenced by improved function[5]. This finding encourages ophthalmologists to use megadose steroid in the treatment of TON and has become a popular choice of treatment. Later on, it was observed that megadose of steroid is not specifically beneficial for TON.

The applicability of the successful result of NASCIS in the spinal cord injury to clinical management of TON remains uncertain because histologically and functionally, spinal cord and optic nerve are different.

On the other hand, the result of the CRASH study (Corticosteroid Randomization after Significant Head Injury) suggests that megadose steroid is associated with increased mortality when given in the context of head injury and exacerbates axonal loss[6]. This steroid therapy is said to be helpful to some extent in the case of isolated TON.

A review of several series reveals that 25 to 50% of patients with TON improve spontaneously. It is further corroborated by the International Optic Nerve Study on TON comparing observation alone, treatment with steroid, or optic nerve decompression. This study shows that vision improves by 03 Snellen lines in 57% untreated group, i.e., observation alone (Spontaneously improved), 52% steroid group, and 32% optical decompression[7].

No treatment has been shown to be convincingly effective. There is no standard of care for the treatment of TON and observation alone is reasonable as an expectation of spontaneous improvement.

Ultimately, even though ophthalmologists are in challenging circumstances and are on the horn of a dilema, they continue to advocate the use of conventional treatment with steroids rather than megadoses, viz., I/V methyl prednisolone (500 mg bd) for 3 days, followed by oral prednisolone (1 mg/kg/body weight) for 11 days, in the hope of improving vision by reducing oedema and inflammation of the optic nerve. Some opine that megadoses of steroids prevent oxidative damage to the optic nerve. Others are of the opinion that TON is almost always associated with craniofacial injury, and a megadose of steroids worsens the situation (CRASH study)[6]. Improvement of vision in TON is observed in some patients; these are not due to the effect of a steroid but rather considered to be spontaneous improvement (IONTS)[7].

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