

# VISUAL RECOVERY FOLLOWING TREATMENT WITH VERY HIGH DOSE CORTICOSTEROID IN TRAUMATIC OPTIC NEUROPATHY

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## ABSTRACT

*Optic nerve compression is a true ophthalmic emergency. In addition to causes such as tumours, infection, mucocoeles and granulomas, the majority of cases are the result of orbitofacial or closed-head trauma. The appropriate management of such cases is controversial; with some authors favouring surgical decompression while others advocate medical treatment using very high-dose corticosteroids, or a combination of both. We report a case of traumatic optic neuropathy in which there was marked improvement in visual acuity following the administration of methylprednisolone.*

*Keywords: traumatic optic neuropathy, surgical decompression, methylprednisolone, corticosteroid therapy, visual prognosis*

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## INTRODUCTION

Indirect injury to the optic nerve may occur during orbitofacial or closed-head trauma, resulting in acute and marked visual loss. Severe loss is the outcome in these cases as a rule, although spontaneous recovery, improvement after surgical decompression, and recovery following corticosteroids treatment have been documented<sup>(1-3)</sup>. The management of traumatic optic neuropathy is controversial with no clear consensus on the modality and timing of treatment. We report a case of a patient who had marked visual improvement after being treated with very high dose corticosteroids.

## CASE REPORT

A 73-year-old Chinese female was admitted for a head injury. Following an accidental fall on the right side of her face, she lost consciousness for five minutes. Paramedics reported her to have had a nose bleed and coffee ground vomitus. She has a history of mild hypertension and a cataract removal in her right eye with intraocular lens implant six years previously.

Initial examination revealed a stable and alert patient. There was a right frontal haematoma with right periorbital swelling and bruising; her nose bleed had ceased. Her pupils were equal in size and were reactive to light. Skull X-ray did not reveal any fractures.

The patient complained of visual loss in her right eye the next day and was referred to the Eye Department. Her visual acuity was questionable perception of light in her right eye and

6/24 in the left. There was right afferent pupillary defect. Fundoscopy was unremarkable and the right optic disc appeared normal. There was no recordable visual field in the right eye on Goldmann perimetry. A diagnosis of acute traumatic optic neuropathy was made in view of the history and clinical findings.

She was then started on a course of very high dose intravenous methylprednisolone 1.5 gm as loading dose, followed by continuous 5.4mg/kg iv methylprednisolone over the next two days and replaced with oral prednisolone 40 mg daily for two weeks. An urgent computerised tomographic (CT) scan of the orbit revealed a right medial orbital wall fracture and a fracture of the floor of the right orbit. No bone fragments were seen to be impinging on the optic nerve. Her response to the treatment is shown in Table I.

Table I – Visual acuity in patient's right eye

Time	Visual Acuity in Right Eye
Day 1	Perception of light
Day 2	Counting finger at 1m
Day 3	Counting finger at 2m
Day 4	Counting finger at 2m
Day 15	6/18

The patient was discharged on day 4 and was seen again on day 15. Her visual acuity in the right eye had improved to 6/18 and her pupils were equal and reactive to light. Her right optic disc was pink and normal in appearance. Repeat Goldmann perimetry revealed a superior altitudinal field defect in the right eye. Colour vision was abnormal. Her findings remained unchanged thereafter.

## DISCUSSION

Traumatic optic neuropathy is characterised by visual loss which occurs following trauma to the orbitofacial region or head. In the absence of a penetrating wound, traumatic optic neuropathy is presumed to be the result of an indirect injury, where the force of impact is transmitted to the optic nerve and its vasculature through the soft tissue or bones.

The mechanisms of injury remain uncertain, and may include mechanical insults such as contusion necrosis, impingement of bones or fragments on the nerve, and haemorrhages in the optic

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nerve, dura and the vaginal sheaths. Oedema and ischaemia to the nerve due to injury to small nutrient vessels are other possible mechanisms<sup>(2,4)</sup>. Cellular injury mechanisms namely free radical damage, effects of arachidonic acid metabolites and bradykinins, disturbed calcium homeostasis and cell-mediated inflammation may have a role in the damage of the optic nerve subsequent to the force of impact<sup>(6)</sup>.

As illustrated in the above case, a diagnosis of traumatic optic neuropathy should always be considered in all cases of orbitocranial trauma with visual loss. Such patients should get a complete clinical ophthalmic evaluation once the head injury, respiratory and cardiovascular priorities have been attended to. Examination of visual acuity and pupillary reflexes is mandatory. Fundoscopy should be performed whenever possible. Neuroradiology, preferably high-resolution CT of the orbital apex and paranasal sinuses, would help to identify fractures, bony impingement and nerve sheath swelling or haematoma. Presently, the role of magnetic resonance imaging in traumatic optic neuropathy has yet to be defined. Visual fields, as seen in this case, can only be obtained when sufficient vision is present. It is probably more useful in documenting the return of visual function following injury. Once a clinical diagnosis of traumatic optic neuropathy is made, treatment should commence without delay.

No agreement exists as to the appropriate management of patients with traumatic optic neuropathy. Because of evidence suggesting the intracanalicular segment of optic nerve as the most frequent site of injury, surgical decompression has been advocated as the treatment traditionally<sup>(2,4-7)</sup>. It is believed that decompression will reduce the sequelae of the swelling of the optic nerve in the bony canal or haemorrhagic compression on the optic nerve and its vascular supply<sup>(2,8)</sup>. Fukado<sup>(9,10)</sup> and Fujitani<sup>(11)</sup> are strong advocates of surgery, be it through transcranial or extracranial approaches, and their patients had excellent outcome. However, because of the concerns about diagnostic and case selection criteria for treatment, the validity of their results were questioned by some<sup>(4,5)</sup>. Although some authors who advocate surgery felt that decompression should be done within the first week following injury<sup>(7)</sup>, Amrith et al<sup>(12)</sup> have reported a case of visual recovery following transthemoidal optic nerve decompression 13 days after initial trauma.

Over the past decade, evidence emerged that very high doses of corticosteroids when given within a few hours of injury, was able to reduce paralysis and severity of neurological deficits in patients with acute spinal cord injuries<sup>(4,13,14)</sup>. Hall and Braughler<sup>(14)</sup> have demonstrated that intravenous methylprednisolone in very high doses (30mg/kg) exhibited a separate and distinct pharmacologic effect than in doses usually encountered in clinical practice. Effects of very high dose corticosteroids in experimental model of spinal cord injuries include a decrease in lipid peroxidation products and better blood flow in the injured areas, and improved neurophysiologic recovery.

Based on the salutary effects of corticosteroids in the experimental models, Anderson et al<sup>(15)</sup> introduced the protocol of combined use of very high dose corticosteroids and surgery in the treatment of traumatic optic neuropathy. Since then, many other series have combined corticosteroids with surgery<sup>(5-7)</sup>, making it difficult to determine the efficacy of a particular treatment. Spoor et al<sup>(13)</sup> and Sieff<sup>(16)</sup> showed that 19 out of 22 eyes and 3 out of 6 eyes improved respectively with corticosteroids treatment. Wolin and Lanvin<sup>(17)</sup> reported 4 cases of traumatic optic neuropathy with total blindness had visual recovery following corticosteroids treatment.

In conclusion, until a prospective, multicentre, randomised

trial to establish a clear protocol for treatment of traumatic optic neuropathy is in sight, the appropriate management of traumatic optic neuropathy remains debatable. Nevertheless, the use of very high dose corticosteroids in the management of traumatic optic neuropathy is well accepted currently. We believe that very high dose of methylprednisolone may help restore vision in patients with traumatic optic neuropathy, as seen in the case under discussion. However, we do not imply that all optic nerve injuries be treated with corticosteroids alone. Rather, we agree with Steinsaper and Goldberg<sup>(4)</sup> in their recent excellent review of this topic that upon diagnosis of traumatic optic neuropathy, preferably within 8 hours of initial trauma, intravenous methylprednisolone 30mg/kg loading dose be given if there are no contraindications. This is followed by continuous 5.4mg/kg/hr iv methylprednisolone for 48 hours; and if vision improves, the patients be placed on oral prednisolone with taper over a week. If there is deterioration in vision, or no improvement after 48 hours despite iv methylprednisolone, or presence of suspicious neuroradiological findings, surgical decompression may be offered.

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