

Delayed Visual Recovery from Optic Nerve Injury Following a Procedure of Orbital Wall Reconstruction

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Abstract

The acute onset of the vision loss by optic nerve injury following orbital wall reconstruction, has been reported in 0.5% - 5.0% of the cases. Visual impairment can be recovered within an early period after injury. Delayed visual recovery from optic nerve injury during a procedure of orbital wall reconstruction has not been reported. We report a case of delayed recovery from optic nerve injury which occurred following orbital wall reconstruction. A 78-year-old man underwent orbital wall reconstruction for medial wall fracture and resulting enophthalmos in the right eye, one week after a traffic accident. Immediately after surgery, postoperative visual acuity in the right eye decreased to light perception, and relative afferent pupillary defect (RAPD) was detected. In spite of mega-dose steroid treatment, the visual acuity did not improve. However, 8 months after surgery, visual acuity began to recover to 0.1, and the degree of RAPD decreased. Twelve months after surgery, visual acuity in the right eye was 0.4, and pupillary light reflex was normal. Our report suggests that patients with optic neuropathy by surgery or trauma require long-term follow-up, regardless of early response to mega-dose steroid treatment.

Keywords

Visual Loss, Delayed Visual Recovery, Optic Nerve Injury, Mega-Dose Steroid

1. Introduction

Complications after orbital wall reconstruction include visual loss, persistent diplopia, enophthalmos, implant displacement, etc. Among these, visual loss is a rare and serious condition. Its occurrence results from orbital compartment

syndrome, direct intraoperative injury to the optic nerve from surgical manipulation, bony fragment, orbital implants, or inferior retinal arteriolar occlusion [1]. Recovery from visual impairment usually recovers within an early period after injury. However, delayed visual recovery from optic nerve injury following a procedure of orbital wall reconstruction has not been reported. We report a case of delayed recovery from optic nerve injury which occurred following orbital wall reconstruction.

2. Case Report

A 78-year-old man was admitted complaining of right periorbital swelling after a road traffic accident. He had been diagnosed with herpes simplex keratitis in the left eye a long time ago. At the initial examination, corrected visual acuity was 0.8 in the right eye, and 0.08 in the left eye, and his pupillary light reflex was normal. Slit lamp examination showed normal findings in the right eye and diffuse corneal opacity with neovascularization in the left eye. Orbital CT demonstrated a large medial orbital wall fracture on the right side (Figure 1). Hertel exophthalmometer revealed enophthalmos of the right eye. Orbital wall reconstruction was performed 1 week after injury. However, postoperative visual acuity in the right eye immediately decreased to light perception, and relative afferent pupillary defect (RAPD) was detected. Under the diagnosis of postoperative optic neuropathy, mega-dose steroid treatment was administered as follows: intravenous methylprednisolone 5 mg/kg per 6 hours for 3 days, followed by oral prednisolone 1 mg/kg for 11 days, 0.4 mg/kg for 1 day, and 0.2 mg/kg for 3 days. Despite steroid treatment, visual acuity of the right eye showed no improvement.

For visual rehabilitation, we performed penetrating keratoplasty in his left eye



Figure 1. Orbital CT showed a large fracture of the medial orbital wall on the right side.

one month after verteporfin photodynamic therapy [2]. One month after keratoplasty, corrected visual acuity in the left eye recovered to 0.3. Eight months after orbital wall reconstruction, corrected visual acuity in the right eye began to recover to 0.1, and RAPD decreased from (3+) to (1+). Twelve months after orbital wall reconstruction, corrected visual acuity in the right eye was 0.4, and pupillary light reflex was normal. Visual field examination showed peripheral field defects, and the visual evoked potential test was normal (Figure 2(a), Figure 2(b)).

3. Discussion

Visual acuity loss following orbital wall reconstruction surgery is an uncommon condition. Its occurrence results from orbital compartment syndrome, or direct intraoperative injury to the optic nerve from surgical manipulation, bony fragment, orbital implants, or inferior retinal arteriolar occlusion. The management of indirect optic nerve injury remains controversial. Either surgical decompression or mega dose corticosteroid is used for managing indirect optic nerve injury. However, no consensus exists regarding the definitive treatment.

In general, visual acuity after optic nerve injury shows improvement within an early time after mega-dose steroid treatment. If vision does not return, it is assumed that the optic nerve has been irreversibly injured. Sabri *et al.* [3] reported on treatment of 11 patients who had traumatic superior orbital fissure and orbital apex with mega-dose corticosteroid. All patients showed visual improvement within the first 24 hours of treatment. Matsuzaki *et al.* [4] reported a retrospective study on treatment of traumatic optic neuropathy. Visual improvement was observed in 58.8% of 22 patients treated with mega-dose therapy. In contrast, Mahapatra [5] reported 2 cases of delayed recovery from indirect optic nerve

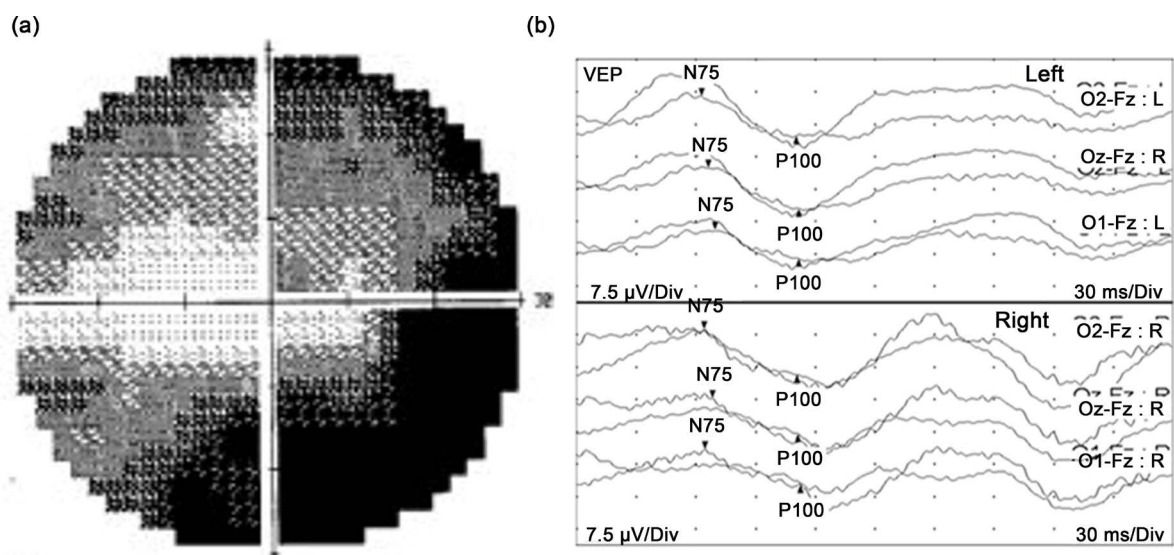


Figure 2. Visual field and evoked potential images in the right eye at 12 months after surgery. (a) Visual field examination shows periphery visual field defects; (b) Visual evoked potential test shows normal amplitude and latency of P100.

injury at 8 and 12 weeks after head trauma. In our case, visual loss by optic nerve injury developed following orbital wall reconstruction, but did not improve despite mega-dose steroid therapy. However, vision recovered from 8 months after orbital wall reconstruction surgery. This is the first report of delayed visual recovery of optic nerve injury during orbital wall reconstruction. In conclusion, based on this experience, patients with optic neuropathy by surgery or trauma require long-term follow-up, regardless of early response to mega-dose steroid treatment.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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