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Case Report

Isolated Neurogenic Traumatic Ptosis and Traumatic Optic Neuropathy as a Result of Blunt Trauma: A Rare Case Report

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Abstract

A 54-year-old male patient who had a laceration on the left lower lid laterally due to a branch hitting his left eye while riding a motorcycle presented to our clinic. Initially, the best-corrected visual acuity was found to be 10/10 in the right eye and 1/10 in the left eye. There were no pathological findings in the right eye. Direct pupillary light reflex was very weak in the left eye, there was no levator palpebrae superioris muscle function, and there was limitation of elevation. The fundus examination of the left eye was normal. The patient was consulted with the neurology clinic. The patient's radiological imaging was evaluated as normal by the neurologist. The patient was diagnosed with traumatic optic neuropathy, traumatic ptosis, and limitation of elevation due to damage to the upper branch of the oculomotor nerve caused by the trauma. The patient was started on 1mg/kg prednisolone therapy per day, and the plan was to taper the treatment by 5 mg every three days. Neuropraxia, which is defined as the temporary inability of the nerve to function without axonal loss, is usually a condition that improves within 6-8 weeks. Partial or complete loss of function in the optic nerve following direct or indirect trauma is called traumatic optic neuropathy (TON).

Keywords: Isolated Traumatic Neurogenic Ptosis; Neurogenic Ptosis; Oculomotor Nerve; Optic Neuropathy; Traumatic Optic Neuropathy

Introduction

Partial or complete loss of function in the optic nerve following direct or indirect trauma is defined as traumatic optic neuropathy (TON). Optic nerve damage is seen in approximately 0.5% to 5% of facial and head injuries [1]. Damage to the optic nerve following trauma with objects penetrating the orbit is classified as direct traumatic optic neuropathy, while damage to the optic nerve due to hemorrhage, edema, or concussion following non-penetrating trauma is classified as indirect traumatic optic neuropathy. Indirect traumatic optic neuropathy is more common than direct traumatic optic neuropathy and often occurs after indirect trauma, especially to the forehead. Injuries to the frontal and zygomatic bone areas increase the risk of optic nerve damage as the force of the trauma is concentrated in the optic foramen and the posterior orbital floor. Since the optic canal has no capacity for stretching, the optic nerve is more affected by edema that occurs in the intracanalicular area after trauma [2]. If visual loss is accompanied by an afferent pupillary defect and the appearance of the optic nerve head is normal during fundus examination, it suggests indirect traumatic optic neuropathy, while visual loss with afferent pupillary defect and hemorrhages in the optic nerve head suggest direct traumatic optic neuropathy. Due to atrophy in the retinal nerve fibers over time, pallor is seen in the optic nerve head on fundus examination about 3 weeks after the trauma. High-dose corticosteroid treatment, hyperbaric oxygen therapy and surgical optic nerve decompression can be performed in the treatment of traumatic optic neuropathy. Ptosis is defined as the coverage of more than 2 mm of the upper limbus in the primary position of the upper eyelid and narrowing of the palpebral fissure. Trauma can cause ptosis through various mechanisms. Mechanical ptosis can occur due to hematoma and edema in the eyelid, aponeurotic ptosis can occur due to the disruption of the relationship between the aponeurosis and the tarsus as a result of blunt trauma to the eyelid, muscular ptosis can occur due to direct damage to the levator palpebrae superioris muscle and Muller muscles, and trauma-induced ptosis can also occur due to damage to the motor branches of the oculomotor nerve that

innervate the levator palpebrae superioris muscle or the sympathetic fibers that innervate the Muller muscle. In this article, a case is presented of isolated neurogenic traumatic ptosis and elevation limitation due to damage to the superior branch of the oculomotor nerve and decreased visual acuity secondary to traumatic optic neuropathy. This study was performed with the principles outlined in Declaration of Helsinki.

Case Description

A 54-year-old male patient who had a collision with a branch hitting his left eye while riding a motorcycle and had a laceration on the lateral side of the left lower lid presented to our clinic with complaints of vision loss and drooping of the eyelid. Best-corrected visual acuity was 10/10 in the right eye and 1/10 in the left eye. No pathological findings were observed in the right eye. In the left eye, the direct pupillary light reflex was very weak, there was no levator palpebrae superioris muscle function, and there was limitation in elevation. The left eye fundus examination was normal. The patient was consulted with neurology, and his radiological imaging was reported as normal. It is considered that there is damage to the superior branch of the oculomotor nerve, which innervates the levator palpebrae superioris muscle and superior rectus muscles, with traumatic optic neuropathy in the patient. The patient was started on 1mg/kg prednisolone treatment, to be reduced by 5 mg every three days. At the first month of steroid treatment, the best-corrected visual acuity was determined as 10/10 in the right eye and 5/10 in the left eye. There was a relative afferent pupillary defect and 12 prism diopters of hypotropia in the left eye. Fundus examination was normal for both eyes. Visual evoked potential (VEP) test showed prolonged P-100 wave latency in the left eye. After 6 months post-trauma, the best-corrected visual acuity was found to be 10/10 in the right eye and 7/10 in the left eye and ptosis and elevation limitation of the left eye almost disappeared.

Discussion

Traumatic optic neuropathy is a significant cause of severe visual loss that occurs after blunt or penetrating head and facial trauma. Depending on the type of trauma, traumatic optic neuropathy is classified into two categories: direct and indirect. Direct traumatic optic neuropathy occurs when there is a disruption in the anatomical and functional integrity of the optic nerve after penetrating orbital injuries. Direct traumatic optic neuropathy is characterized by sudden, severe, and irreversible vision loss. Indirect traumatic optic neuropathy, which occurs after non-penetrating blunt trauma, preserves the anatomical integrity of the optic nerve, but damage to the nerve can occur due to the energy created by blunt trauma transmitted to the optic canal or sudden movement of the globe [3]. Although there is no widely accepted treatment protocol, the treatment of traumatic optic neuropathy can be classified as high-dose corticosteroid therapy, hyperbaric oxygen therapy, or surgical decompression. Spontaneous recovery cases of traumatic optic neuropathy have also been reported in the literature [4]. Surgical decompression is performed by removing bone fragments that directly contact the optic nerve or by decompressing the intracanalicular optic nerve by removing structures surrounding the optic nerve [5]. The oculomotor nerve gives off two branches during its course along the cavernous sinus, an upper and a lower branch, and passes through the superior orbital fissure into the orbit within the annular tendon of Zinn. The upper branch is responsible for the innervation of the levator palpebrae superioris muscle and superior rectus muscles, while the lower branch is responsible for the innervation of the medial rectus, inferior rectus, and inferior oblique muscles, as well as the parasympathetic fibers that go to the sphincter pupillae and ciliary muscles. In lower branch lesions, limitation of adduction and depression with accommodation defects and mydriasis are observed. Isolated neurogenic traumatic ptosis develops secondary to direct or indirect trauma to the upper eyelid area and can be self-limiting with conservative treatment or spontaneous recovery. The condition where the nerve cannot function temporarily due to damage, stretching, or distortion of axonal integrity without permanent damage to the neuron after trauma is defined as neuropraxia. There is no degeneration in the neuron in neuropraxia, and the Schwann sheath and axon are intact, but there is a temporary interruption in impulse transmission. Neuropraxia recovers in 6-8 weeks. In our case, the coexistence of elevation deficit with traumatic ptosis suggested damage to the upper division of the oculomotor nerve due to trauma. There were also signs of traumatic optic neuropathy including accommodative impairment, and mydriasis. The improvement over time in ptosis and elevation deficit supports the view that the neural damage in the upper division of the oculomotor nerve was not due to axonal degeneration but due to neuropraxia. Traumatic ptosis can also occur due to direct damage to the aponeurosis or levator palpebrae superioris muscle, or due to aponeurotic disinsertion or detachment. Unlike neurogenic ptosis, the recovery in cases of aponeurotic injury is slow [6]. Isolated neurogenic traumatic ptosis cases have been reported rarely in the literature, and there is no consensus on treatment protocols. Cases of isolated neurogenic ptosis related to paranasal sinusitis have also been reported in the literature. The isolated superior division oculomotor nerve palsy treated with sinusotomy was first mentioned by Stefanis L. and Przedborski S. in 1993. They observed ptosis and elevation restric-

tion in a patient with sphenoid sinusitis, which improved 8 weeks after surgical drainage of the sinus. They attributed the dramatic improvement seen after surgery to the relief of pressure on the superior division of the oculomotor nerve caused by sphenoid abscesses [7]. Kim and Shin reported a case of isolated neurogenic traumatic ptosis that developed after blunt trauma to the eyelid and resolved spontaneously within 4 weeks [8]. Similarly, McCulley., *et al*. reported three cases of isolated neurogenic traumatic ptosis that developed secondary to eyelid trauma and resolved spontaneously[9]. Shin., *et al*. reported a case of isolated neurogenic traumatic ptosis that developed after minor facial trauma and was treated with intravenous prednisolone 250mg/day for 2 days followed by oral prednisolone 15mg/day for 5 days, resulting in complete recovery in 7 weeks [10]. Li., *et al*. reported a case of head injury resulting from falling backwards and developing headache and ptosis, in which they detected a fracture in the upper part of the medial orbital wall on computed tomography, and reported that the fall caused a counter-blow, resulting in a fracture of the orbital bone without direct trauma to the periorbital region, making it the first case of isolated neurogenic traumatic ptosis without direct trauma to the periorbital region. The patient was managed conservatively and recovered spontaneously within 2 months [11]. Isolated neurogenic traumatic ptosis cases due to iatrogenic trauma from orbital surgery have been reported in the literature. Jung and Chi reported 3 cases of isolated neurogenic blepharoptosis successfully treated with high-dose oral steroids (1 mg/kg/day for 4 days and gradually tapered) after medial orbital wall reconstruction [12]. Song., *et al*. reported a case of isolated neurogenic traumatic ptosis following medial orbital wall reconstruction via transcaruncular approach due to periorbital trauma, and achieved recovery in 3 weeks with low-dose oral corticosteroid therapy (16 mg/day for 5 days) [6]. Satchi., *et al*. have reported 3 cases of secondary isolated neurogenic ptosis following trauma, and in 1 case, they have also reported elevation restriction due to the involvement of the superior rectus muscle. Spontaneous recovery has been observed in all 3 cases [13]. Jain., *et al*. have reported a case of a 10-year-old child with isolated superior division of oculomotor nerve palsy accompanied by ptosis, elevation restriction, and horizontal nystagmus following mild head trauma [14]. Upon reviewing the literature, this case presented is unique due to the combination of traumatic neurogenic ptosis, elevation restriction, and traumatic optic neuropathy.

Conclusion

This case report aims to bring to mind the ptosis, elevation limitation and vision problems that can be occured after blunt trauma and explained by neuropraxia in superior division of oculomotor nerve and traumatic optic neuropathy. Neuropraxia is defined as temporary loss of motor and sensory function due to damage, stretching or distortion of axonal integrity results in interruption of impulse transmission without degeneration in the neuron. Neuropraxia recovers in 6-8 weeks with conservative treatment or spontaneous recovery and traumatic optic neuropathy can be treated with high-dose corticosteroid therapy, hyperbaric oxygen therapy, surgical decompression and spontaneous recovery. In this article, a case is presented of isolated neurogenic traumatic ptosis and elevation limitation due to damage to the superior branch of the oculomotor nerve and visual disturbance caused by traumatic optic neuropathy. We treated our patient with 1mg/kg prednisolone therapy, to be reduced by 5 mg every three days and observed the mitigation of signs and symptoms gradually, almost fully recovery was achieved in six months.

Conflict of Interest Statement

The authors declare that there is no conflict of interest.

Approval of Ethics Committee

Written informed consent for the case to be published including case history and data was obtained from the patient for publication of this case report.

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