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Case Report

Role of Visual Evoked Potential in Direct Traumatic Optic Neuropathy: A Case Report

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ABSTRACT
Traumatic optic neuropathy (TON) is a rare cause of visual impairment following blunt or sharp trauma and the diagnosis is established clinically. Lesions on the optic nerve may not always be visible in neuroimaging examinations. Studies on Visual Evoked Potential (VEP) in TON patients are still limited, despite being beneficial for objectively detecting optic nerve lesions. A 16-year-old male patient was referred to the neurology clinic with a sudden loss of vision in the left eye approximately 25 days after a head injury due to a motor vehicle accident. The patient had epidural bleeding and fractures in the orbital and facial bones, as revealed by a head CT scan. Neurological examination showed a visual acuity of 1/300 in the left eye, left midriasis, and a negative light reflex in the left eye. VEP examination of the left eye revealed prolonged P100 latency and a decrease in P100 amplitude (>50%), indicating a lesion in the left optic nerve. This visual impairment persisted for up to 6 months post-head trauma. TON is a vision-threatening disorder that should be considered in patients with ocular or head trauma and visual impairment. A decrease in amplitude ratio <50% and prolonged P100 latency >140 ms are associated with poor visual function recovery. VEP examination is an objective assessment of visual pathway integrity and serves as one modality for early TON diagnosis and a predictor of visual function prognosis in TON patients.

Keywords: diagnostic; prognostic; traumatic optic neuropathy; visual evoked potential

INTRODUCTION

Traumatic optic neuropathy (TON) is a condition characterized by severe vision impairment that can be caused by eye or head trauma. TON can be divided into two types, namely direct or indirect, based on whether there is direct exposure of the optic nerve to the trauma agent.[1] Patients with TON are typically young and experience severe and irreversible vision loss, significantly impacting their quality of life. The diagnosis of TON is generally established through a sudden loss of vision complaint after direct or indirect optic nerve trauma and objective findings through fundoscopy, such as abnormal

appearances of the optic disc and the presence of relative afferent pupillary defect (RAPD). Optic nerve lesions are rarely visible in neuroimaging examinations such as Computed Tomography (CT) scans or Magnetic Resonance Imaging (MRI) of the head. Visual Evoked Potential (VEP) studies can be used as one of the methods for confirming TON diagnosis because they can objectively assess optic nerve dysfunction. After confirming the diagnosis of TON, the following issues that need analysis include predicting the degree of visual function and the prognosis of visual function. Visual acuity is significantly reduced in the majority of patients. Electrophysiological studies have also been used to predict visual outcomes after eye injuries. VEP studies are believed to be a reliable method for obtaining information on whether visual function is still intact.[2,3] Studies on VEP examinations in TON are still rarely reported. Detection and management of TON are often delayed, exacerbating the visual outcomes for patients. This case report is compiled to investigate characteristic findings and the role of VEP in diagnosing and determining prognosis in TON cases.

CASE REPORT

A 16-year-old male patient was referred to the neurology clinic with a complaint of vision loss in the left eye for approximately 25 days. The patient reported a sudden loss of vision in the left eye after falling from a motorcycle accident. The patient could only perceive light in his left eye. The complaint of vision loss in the left eye was perceived as persistent and not improving. This complaint was accompanied by a disturbance in the movement of the left eye towards the center and upward. However, the patient could still glance to the left. No complaints of vision and eye movement disturbances were reported in the right eye. Other neurological complaints were denied. During the accident that occurred approximately 25 days prior, the patient was not wearing a helmet while riding and collided with another motorcycle, then fell on the left side. At that time, the patient experienced a loss of consciousness approximately 30 minutes after the incident. The patient

was immediately taken to the nearest hospital, where brain bleeding was found, and evacuation surgery was performed 22 days prior. The patient was referred for the management of facial bone fractures. Subsequently, the patient underwent post-reduction open surgery, excision, exploration, repositioning, and internal fixation approximately 18 days prior.

Upon initial general physical examination upon arrival at the Emergency Department, a post-craniotomy surgical scar was found on the left frontotemporoparietal region, bilateral eyelid hematomas, bilateral periorbital edema, and multiple excoriated wounds on the lateral eyelid and left nasal area. Neurological examination revealed the patient's vision in the left eye as 1/300, while the vision in the right eye was 6/18. Anisocoric pupils were observed, with a diameter of 3 mm in the right pupil and 5 mm in the left pupil. Direct and consensual light reflexes were negative in the left eye. Fundoscopy revealed papillary edema and retinal bleeding in the left eye. Examination of eye movement showed left oculomotor nerve paresis. No other focal neurological deficits were found.

A head CT scan performed at the initial incident revealed the presence of epidural haemorrhage in the left frontal region and fractures including Le Fort II fracture of the left maxilla, fractures in the left and right inferior orbital rim, left orbital floor, and right maxillary bone (Figure 1A-E). The CT scan images also indicated optic nerve avulsion, bleeding, and damage to the retroorbital tissue of the left eye (Figure 1F-I).

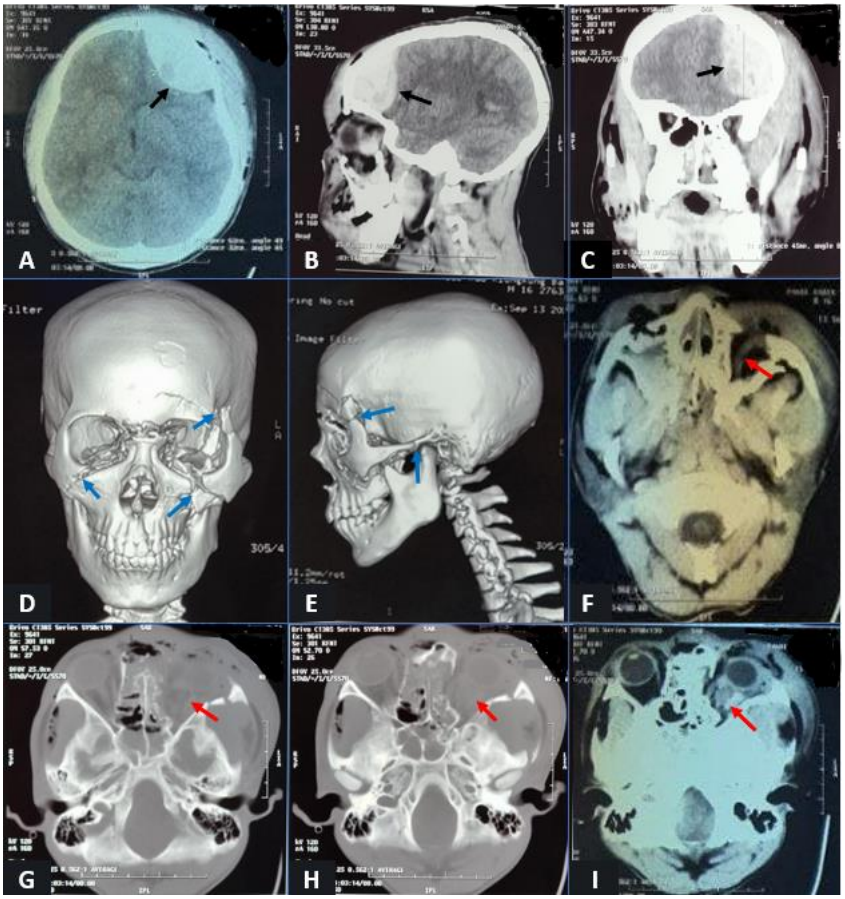

Figure 1. Head CT-scan with 3D reconstruction.
A,B,C) Epidural hemorrhage on left frontal region in axial, sagittal, and coronal view (black arrow); D,E) Multiple facial bone fractures: Le Fort II fracture of the left maxilla, fractures in the left and right inferior orbital rim, left orbital floor, and right maxillary bone (blue arrow); F,G,H,I) Optic nerve avulsion, bleeding, and retroorbital tissue damage of the left eye (red arrow)

Pattern VEP examination with full-field stimulation revealed a decrease in amplitude (1.29 µV) and prolonged latency duration at P100 (140 ms) in the left eye examination (Figure 2A). Amplitude and latency duration in the right eye examination were normal, measuring 6.9 µV and 112 ms, respectively (Figure 2B). The patient was diagnosed with direct traumatic optic neuropathy. The visual impairment persisted, with the pattern VEP examination showing consistent results after 6 months.

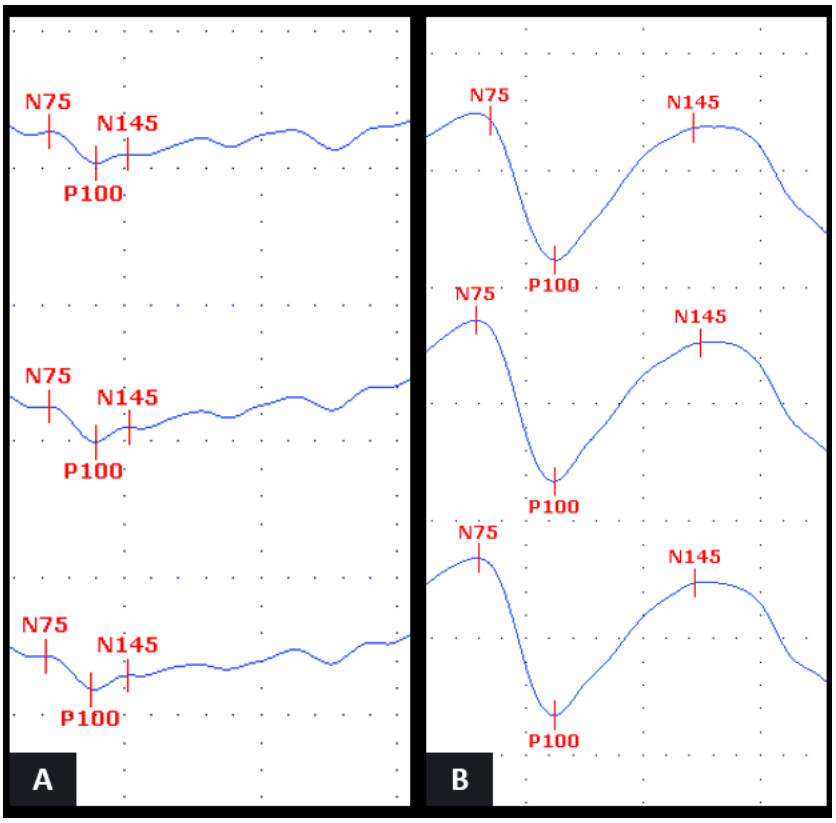

Figure 2. VEP result
A) VEP of the left eye showed decreased amplitude and prolonged latency duration; B) VEP of the right eye showed normal result

DISCUSSION

This case report serves as an example of cranial nerve injury (optic and oculomotor nerves) due to head trauma with impact on the ipsilateral facial area. Optic nerve injury resulting from trauma can lead to visual impairment, known as TON. The overall incidence of TON is reported to be 0.7-2.5%. The most common causes of TON in trauma cases are motor vehicle accidents (63%) and falls. TON is reported to occur in up to 80% of male patients with a median age of 31 years, while 21% of cases occur in individuals under the age of 18.[2] TON is divided into two categories: direct and indirect. Direct TON is often associated with severe vision loss and a lower chance of recovery compared to indirect traumatic optic neuropathy. Direct TON commonly occurs when bone fragments damage optic nerve fibers or when contusion or concussion causes anatomical disruption. Conversely, indirect TON frequently occurs when blunt head

injury or eye trauma is transmitted through the soft tissues of the ocular facial region and the framework to the optic nerve. This damages the integrity of the optic nerve and results in varying degrees of vision loss. Direct TON is more often linked with severe vision loss and a lower likelihood of recovery compared to indirect TON.[3,4] In this case, the patient experienced direct TON due to direct damage to the optic nerve caused by contusion and fractures of the orbital wall, as observed in the head CT scan images.

The diagnosis of TON can be established through clinical findings, including: 1) eye injury, 2) RAPD, 3) impaired visual acuity, 4) color vision impairment, and 5) visual field disturbances. Neuroimaging examinations should be performed in patients with head trauma or oculofacial injuries who exhibit symptoms of optic nerve damage. Head CT scan is the best and easily accessible imaging modality to detect optic canal fractures, orbital wall fractures, and orbital hemorrhage. Detecting and diagnosing optic neuropathy can be challenging, especially in uncooperative or unconscious patients. Visual Evoked Potentials are not necessary for diagnosing TON in most patients, but they can be beneficial in confirming the diagnosis in suspected cases under challenging conditions and determining the prognosis of visual function. Early diagnosis of optic nerve injury is crucial as it accelerates the administration of therapy. VEP holds diagnostic value for patients who cannot recall the time of nerve damage, unconscious patients, those with poor pupil response, and patients with bilateral TON. This is particularly evident in patients with severe head injuries where the altered state of consciousness makes early diagnosis challenging, leading to delayed management of TON.[2,3,5]

The VEP study provides crucial information about the functional integrity of the visual system. VEP is an electrophysiological signal generated in response to visual stimuli, extracted from electroencephalographic activity in the visual cortex recorded from electrodes on the scalp. Since the visual cortex is primarily activated by the central visual field, VEP relies on the functional integrity of central

vision throughout the entire visual pathway, including the eyes, retina, optic nerve, optic radiation, and the occipital cortex.[6] Knowledge about stimulation protocols, neuroanatomy, and VEP generators allows for the interpretation of the location of neurological abnormalities affecting VEP patterns. Several parameters are assessed to interpret VEP examination results, including P100 latency, interocular latency differences, and interocular amplitude differences. Abnormalities may be observed in the form of prolonged P100 latency, decreased P100 amplitude, absence of P100, or abnormal waveform shapes. P100 latency typically ranges between 114-117 ms. Prolonged P100 latency in one eye when the P100 latency in the other eye is normal almost always indicates optic nerve lesions. Relative P100 latency prolongation with significant interocular differences is also an indicator of optic nerve lesions. Lesions in the eyes usually result in decreased amplitude with relatively preserved latency.[7,8]

VEP examination in TON typically reveals prolonged P100 latency and decreased amplitude in the eye affected by trauma, along with an increase in the interocular amplitude difference ratio. One study reported an average interocular amplitude ratio of 0.29±0.023 and an average latency delay of 17.9±2.9 ms in TON patients.[9] A study in India reported significant differences between the abnormal and normal eyes in both P100 latency and amplitude duration. Researchers argued that VEP examinations not only provide additional diagnostic value not apparent in routine clinical and neuroimaging examinations, but also demonstrate high validity in tracking visual disabilities following head injuries.[10] The results of VEP examinations can also serve as predictors of visual function outcomes. Decreased visual acuity due to TON correlates with a reduction in amplitude ratio and latency ratio in Flash VEP. The lower the amplitude and the longer the latency in Flash VEP, the worse the visual acuity in the final outcome.[11,12] A case study suggests that VEP can serve as an indicator for aggressive therapy in TON. Patients with a better VEP response tend to have a higher rate of visual function recovery,

whereas the absence of VEP at the early stage almost always indicates no improvement. The study reports that patients with a decrease in VEP amplitude of less than 50% in the affected eye are likely to experience good recovery.[13–15] A decrease in amplitude in VEP reflects axonal damage and disruption of neuronal signaling in accordance with the pathophysiology of TON.[16] Another study reported that patients with TON and P100 latency prolongation less than 140 ms are associated with a better prognosis for visual function.[17] In this case, the patient experienced a decrease in amplitude ratio of less than 50% and prolonged P100 latency (140 ms) during the outpatient VEP examination. These results can serve as predictors of a poor prognosis for the visual function of this patient. Based on this case and other studies, VEP has diagnostic and prognostic value in TON.

CONCLUSION

Visual Evoked Potential examination is an objective assessment of the integrity of the visual pathway and serves as one of the modalities for early diagnosis of TON. VEP studies in TON reveal a decrease in amplitude and prolongation of P100 latency, consistent with the characteristics of optic nerve lesions. The results of VEP examination also act as predictors for visual function outcomes in patients with optic nerve injuries following head trauma.

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