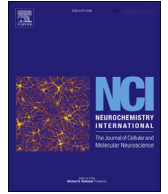




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An insight into the vision impairment following traumatic brain injury

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ABSTRACT

Traumatic brain injury (TBI) is one of the major cause of morbidity and mortality and it affects more than 1.7 million Americans each year. Depending on its location and severity, TBI leads to structural and functional damage in several parts of the brain such as cranial nerves, optic nerve tract or other circuitry involved in vision, and occipital lobe. As a result, the function associated with vision processing and perception are significantly affected and cause blurred vision, double vision, decreased peripheral vision and blindness. In this mini-review, we will focus the recent progress made to understand the pathology and underlying cellular/molecular mechanisms involved in the impairment of the integrity of visual systems following TBI.

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1. Introduction

Traumatic brain injury (TBI) is one of the major causes of morbidity and mortality in the world and is multifactorial in nature (Feigin et al., 2013; Hyder et al., 2007; Rondina et al., 2005). Primary damage can happen by the mechanical forces at the moment of the injury but the secondary pathology results from the occurrence of multiple cellular, neurochemical and metabolic factors. Contact mechanisms of primary damage result either from an object striking the head or from contact between brain and skull, while acceleration/deceleration mechanisms of TBI result from an unrestricted head movement that leads to shear, tensile and compressive strains resulting in widespread damage to axons and blood

vessels (Goodrich et al., 2007; Taber et al., 2006). TBI can be classified as focal, and diffuse (Harmon et al., 2013; Management of Concussion/m, 2009; Menon et al., 2010). The focal TBI results from the impact of the brain against the cranium which leads to contusions on the brain and subdural hemorrhage (Taber et al., 2006). The standard neuroimaging such as CT or MRI is usually employed to diagnose the focal injury (Chalela et al., 2007). On the other hand, the primary diffuse injury is an ongoing process consisting of hypoxic brain damage, brain swelling, vascular injury, and axonal injury which is the most common characteristic feature of diffusion injury (Taber et al., 2006). The most common locations for diffuse injury at the junction of gray matter-white matter, internal capsule, upper brainstem, and corpus callosum. MRI is reportedly more sensitive than CT in detecting the diffuse injury (Chalela et al., 2007; Lee and Newberg, 2005; Moreau et al., 2013). The severity of TBI depends on not only on the pre-injury condition but also

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secondary mechanisms such as cell death, inflammation, edema, neurogenesis impairment and axonal damage associated with TBI (Cernak and Noble-Haeusslein, 2010; Faden, 2002).

Despite the fact that TBI affects a significant part of the brain, vision impairment following TBI has not been well studied. Clinical studies suggest that TBI-patients have been suffering from various vision-related issues including photophobia, double vision, blurred vision, loss of vision, palsy, optic nerve abnormalities and visual processing problems (McCann and Seiff, 1994; Sarkies, 2004; Warner and Lessell, 1995; Warner and Eggenberger, 2010). A substantial number of studies are aimed to understand the alteration in the integrity of the visual system that can provide the critical information of vision related issues and helps screen and monitor the recovery of patients with TBI. Thus in this mini-review, we highlight the visual function deficits which are common in mild and moderate TBI along with molecular mechanisms associated with optic neuropathy and the vision deficits following TBI.

2. Traumatic optic neuropathy

Traumatic optic neuropathy can result from a direct and indirect injury (Levin, 2004; Sarkies, 2004; Steinsapir and Goldberg, 2011; Warner and Eggenberger, 2010). Direct injury results from the penetrating injury, however, the indirect injury results from the transmission of the forces from the distant site to the optic nerve which includes optic nerve head, intraorbital, intercanalicular, or intracranial portion. Both the direct and indirect traumatic event affects the optic nerve and causes functional impairment of vision (Steinsapir and Goldberg, 2011). Although, the optic nerve injury is the most common event after TBI but the diagnosis of optic nerve injuries in acutely injured patients are sometimes challenging for the clinicians. Using several imaging techniques and ophthalmoscopic studies the direct optic nerve injury can be classified in the following manner.

- Optic nerve avulsion:** It is characterized by the absence of optic disc and a sign of hemorrhage and mostly occurs following several orbital trauma. More specifically, the optic nerve is damaged at the lamina cribrosa due to the rotation of the globe and results in an increase in intraocular pressure. This can be diagnosed by optical imaging and ultrasound imaging (Sawhney et al., 2003; Schumann et al., 2013; Ventura et al., 2014).
- Optic nerve transection:** It adversely affects sensitivity towards the light that can be evidenced by visual evoked potentials. This kind of damage results from midfacial trauma and orbital fracture (Levkovitch-Verbin, 2004; Magharious et al., 2011; Ventura et al., 2014).
- Optic nerve sheath hemorrhage:** It is identified as an expansion of nerve sheath with proptosis that ultimately leads to the hematoma. Detection of nerve sheath hemorrhage is challenging to clinicians (Budenz et al., 1994; Leeuw et al., 2015; Ventura et al., 2014).
- Orbital hemorrhage:** Typically this kind of damage is associated with proptosis and ophthalmoplegia and results in an increase in intraocular pressure (Brooks and Finkelstein, 1984; Krohel and Wright, 1979; Ventura et al., 2014).
- Orbital emphysema:** A hair-line fracture in the orbital wall causes an accumulation of air in the orbit that ultimately leads to proptosis and compression of the eye and nerve (Caesar et al., 2003; Gauguet et al., 2008; Ventura et al., 2014).

During the traumatic optic neuropathy from the indirect injury, the impact of head injury is transmitted to the optic nerve that may ultimately lead to blindness. In general, the injury on the forehead but not in the temporal region is responsible for blindness along

with a loss of consciousness (Anderson et al., 1982; Atkins et al., 2008; Walsh, 1966). The histopathological analysis suggests that there was a significant hemorrhage in the optic nerve sheath and the nerve interstitium associated with shearing lesions and ischaemic necrosis of the intercanalicular and intracranial segments of the nerve (Crompton, 1970). The anterior indirect traumatic optic damage results from the separation of the optic nerve on the globe due to the rotation of the globe after trauma (Keane and Baloh, 1992). Impairment in the retinal blood circulation another critical factor that contributes to the optic neuropathy and is associated with axonal injury. The posterior indirect injury results in several vision defects including decreased color vision and field defects due to the either frontal or midfacial blow and can be diagnosed by fundoscopy along with the ophthalmic examination such as decreased acuity (Atkins et al., 2008). However, if the injury is severe, it can cause the loss of consciousness in 40–72% of patients (Blyth and Bazarian, 2010). Interestingly, the intracanalicular portion of the optic nerve is the most susceptible to posterior indirect optic damage which will be followed by shearing and ischemia that will cause vision loss as a long term effect (Atkins et al., 2008; Blyth and Bazarian, 2010). Moreover, a fracture within the canal is also considered as an indirect optic neuropathy and be diagnosed using CT imaging. The diagnosis of posterior optic neuropathy varies depending on how long after optic nerve injury patients are studied (Atkins et al., 2008; Blyth and Bazarian, 2010; Ventura et al., 2014).

Increased intracranial pressure (ICP) is associated with worse outcomes following TBI. Studies have confirmed that ICP is correlated with optic nerve sheath diameter (ONSD) on ultrasound (Blyth and Bazarian, 2010). Alteration in the ONSD is closely associated with mortality in patients with severe traumatic brain injury. The use of clinical markers to predict ICP is desirable as a first line measure to assist in decision making as to whether invasive monitoring is required. Correlations between ICP and ONSD using CT and Magnetic resonance imaging (MRI) have been observed in adult populations (Young et al., 2016). The ultrasonographic measurement of the ONSD is known to be an accurate monitor of elevated ICP (Blyth and Bazarian, 2010). However, it is yet unknown whether fluctuations in ICP result in direct changes in ONSD. In patients who have sustained a TBI, ultrasonography of the ONSD is an accurate, simple, and rapid measurement for detecting elevated ICP as well as immediate changes in ICP. Therefore, it might be a useful tool to monitor ICP, especially in conditions in which invasive ICP monitoring is not available, such as at trauma scenes (Maissan et al., 2015). Another interesting study using linear regression shows that ONSD was independently associated with increased ICP during initial hours. A total of 220 patients were included in the analysis. Overall, the cohort had a mean age of 35 years and 171 of 220 were male. The median admission GCS was 6 (Sekhon et al., 2014). The correlation between ONSD and mortality was further supported by another study where the enlargement of ONSD on initial CT scan has been found to be associated with increased mortality after severe TBI. This could offer the possibility to detect patients with raised ICP requiring urgent therapeutic interventions and/or invasive intracranial monitoring to guide the treatment. More specifically it is suggested that ONSD measured on the initial brain CT scan is independently associated with ICU mortality rate particularly when less than 7.3 mm which is the most common in severe TBI patients (Legrand et al., 2013).

3. Optic chiasm and related pathways

Severe head injury mostly results in traumatic chiasmal syndrome and the prevalence of this syndrome is about 3 in 326 patients. This syndrome is associated with a skull fracture and cranial

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