Case Report

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Post Traumatic Transient Cortical Blindness

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Abstract

Transient cortical blindness is a rare complication of mild head trauma, but it always creates fear in the patient and their family. However, it usually has a benign outcome.

Post traumatic Transient Cortical Blindness is defined as temporary and complete visual loss associated with normal pupillary reflexes and fundoscopic examination with return of visual function without any long term neurological complications following minor head trauma. Since the number of reported cases are very less, transient blindness after head injury has been mostly attributed to hysteria if brain imaging and fundoscopy examination is normal.

Post traumatic Transient Cortical Blindness was first reported by Lemiere [in 1918]but its pathophysiology is still unknown. We report one such case of transient blindness.

Keywords: Head Injury; Transient Blindness; Cortical Blindness; Brain Cortex; Post Traumatic Blindness.

Case Presentation

A 19-year-old female patient was brought to emergency department with history of head injury after having a fall at home. She had history of loss of consciousness for around 10 minutes and 4 episodes of vomiting. On regaining of consciousness, she complained of complete vision loss in right eye and blurring of vision in left eye. She was conscious and completely coherent during her evaluation in Emergency Department. On examination of eyes; pupillary reflex was normal in both eyes. There was no perception of light in right eye with decreased light perception in left. Finger counting was possible at a distance of 30 cm only for left eye. Fundoscopic examination of both eyes were normal. Optokinetic nystagmus was absent in both eyes. CT scan of brain and orbit showed normal study. Ophthalmology consultation was given and perimetry was advised. Perimetry was suggestive of post chiasmal visual defect. Patient was admitted under neurosurgeon for neurological observation. On day 3, patient stated gradual improvement in vision and on day 4, her vision improved gradually from light perception, finger counting and finally to 20/20 vision in both eyes. At one month follow up, neither neurologic nor ophthalmologic sequelae had developed.

Discussion

Cortical Blindness is defined as complete or incomplete visual loss due to injury to occipital cortex of brain. It can be caused due to various reasons including stroke, meningitis, uremia, cardiac surgery, hemorrhagic shock, cardiac arrest and carbon monoxide poisoning. Head trauma, meningitis and cardiac arrest are more common causes of cortical blindness in pediatric age group while cerebrovascular accidents predominate in adults [2].

Bodian described (Post traumatic Transient Cortical Blindness [PTTCB] as a distinct phenomenon following head injury in 1964 [3] but there has been prolonged debate concerning the pathophysiology of

PTTCB. PTTCB is associated with symptoms like headache, confusion, irritability, vomiting which are quite similar to classic migraine symptoms [4,5]. This has led researchers to believe it to be due post-trauma cerebral vascular response. The vascular mechanism explains the observed pupillary and extra-ocular abnormalities seen in PTTCB and also explains sudden, sometimes intermediate transient visual loss . The vascular response to trauma also explains increased incidence of PTTCB in pediatric age group who have a more labile vasculature as compared to adults [3].

Lindenberg and Spatz described "Border Zone "hypothesis which postulates that in transient cerebral hypotensive episodes, the areas which are affected most are the border zone regions between the distribution of three major cerebral arteries [6]. This makes occipital regions most susceptible to hypoxia leading to visual impairment. Occipital region is also highly susceptible to head trauma both by direct and countercoup mechanism which can cause momentary displacement of brain matter leading to sudden traction of cerebral vasculature inducing vasospasm. This causes sudden and immediate symptoms. The decreased cortical blood flow cause reactive hyperemia leading to local cerebral edema and ischemia which might explain the intermediate onset of symptoms [7].

Frequent association of cerebral concussion with head trauma had led Pickles to suggest localized occipital contusion causing focal cortical edema and ischemia as an alternative explanation to PTTCB [8]. Meyer et al suggested a local percussive blow may induce a transient neuronal paralysis without causing a generalized concussion syndrome [9]. This "local percussive paralysis " of visual cortex can explain the rapid onset of symptoms in PTTCB while unable to explain intermediate onset in some patients. Patients with PTTCB demonstrates occipital slowing of alpha waves on EEG supporting cerebral origin of PTTCB. However, brain CT scan after head injury have not demonstrated tissue oedema in patients of PTTCB.

Differential diagnoses in cases of visual loss after head injury includes hysterical blindness, commotio retinae, cerebral contusion, intra-cranial hematoma, and optic nerve or chiasmal injury. In patients with post-traumatic vision loss, priority is given to assessment of vital functions in the emergency room unless the trauma is minimal and the visual loss is isolated. A complete neurological evaluation along with basic ophthalmic evaluation should be performed systematically in the emergency room. The presence of a neuro-imaging abnormality should be ruled out with brain CT without contrast being the best modality of imaging. Both axial and coronal orbital views in addition to routine brain CT must be included in patients with complain of visual loss to rule out any intraocular injury or foreign body. Objective tests such as Optokinetic Stimulation of nystagmus (OKN) can help in evaluating cortical blindness in hysteric, mentally retarded or semi-obtunded patients. A patient with hysterical blindness will blink or withdraw in response to threatening gestures along with presence of optokinetic nystagmus [10].

PTTCB requires no treatment. Patients with suspected PTTCB must kept for in-patient neurological observation. Spontaneous resolution of symptoms occurs with majority of them within hours while some prolonged syndromes have also been reported [11].

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