

Case Report

Cortical Blindness after Motorcycle Accident: A Case Report

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A 47 year-old Thai man presented with acute bilateral blindness after suffering head trauma in a motorcycle accident. No neurological deficit was found. The visual acuity was light perception with poor light projection on both eyes. Other ocular examinations were normal except ecchymosis on both lower eyelids and mild subconjunctival hemorrhage on the left eye. Emergency computerized tomography showed normal appearance of optic nerves, optic canals and multiple contusional intracerebral hemorrhages at both occipital lobes. This case demonstrated a rare condition of permanent cortical blindness without other neurological deficits resulting from a head trauma.

Keywords: *Cortical blindness, Trauma*

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Acute bilateral blindness is a rare condition occurring after a head trauma. If the patient's globes are not ruptured, it may be caused by bilateral traumatic optic neuropathy, bilateral retrobulbar optic nerve compression, bilateral visual pathway injury or cortical blindness. The cortical blindness is a very serious condition, can happen following head trauma, coronary angiography, stroke and spine surgery, etc. Permanent cortical blindness arising from an automobile accident is usually associated with several neurological deficits. To the best of the authors' knowledge, cortical blindness without any neurological deficit resulting from a motorcycle accident has not been described before. Here the authors report the first case of cortical blindness without any neurological deficits in a patient with bilateral occipital lobe hemorrhage after a motorcycle accident.

Case Report

A 47 year-old Thai man suffered from head trauma with transiently impaired consciousness in a motorcycle accident on March 25, 2005. Upon initial evaluation, he complained of severe visual loss on both eyes. Physical examinations revealed unremarkable

results with no neurological deficit detected. There was only a shallow lacerated wound above the left eyebrow. Ocular examination showed visual acuity of light perception with poor light projection on both eyes. Both lower eyelids were edematous with ecchymosis presented. No proptosis or enophthalmos was found. Ocular movement was full in all directions of gaze. Intraocular pressure was 14 mmHg on the right eye and 10 mmHg on the left eye. The pupils were round and demonstrated equal diameter on both eyes. The pupillary reaction was normal and no RAPD was detected. Fundus examination did not reveal any abnormalities. Emergency computerized tomography of brain and orbit showed normal appearance of both optic nerves and contusional intracerebral hemorrhages at both occipital lobes, thin subdural hematoma at the right temporoparietooccipital lobe and tentorial cerebri as well as a mild degree of blowout fracture without bony displacement of the left orbit (Fig. 1, 2). The patient was treated by a medical team consisting of ophthalmologists, neurosurgeons and plastic surgeons. The team decided that the patient should best be treated conservatively. The patient, therefore, was given intravenous methylprednisolone in a 30 mg/kg loading dose, followed by 15 mg/kg at 2 hours after the loading dose and then 15mg/kg of methylprednisolone every 6 hour for 3 days. After completing the parenteral form of corticosteroid, the patient was treated with prednisolone

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Fig. 1 Multiple intracerebral hemorrhage at both occipital lobes (arrow)



Fig. 2 Tentorial hemorrhage (arrow)

60mg/day orally for 5 days then the oral dosage was tapering over the period of 2 weeks. The patient was hospitalized for 3 days with close neurological observation and the visual acuity improved to be counting finger at 1/2 foot on both eyes before being discharged from the hospital.

Discussion

Cerebral blindness or cortical blindness is defined as bilateral loss of vision with normal pupillary responses and no other ocular abnormalities⁽¹⁾. It is a rare syndrome of blindness, resulting from bilateral retrogeniculate lesions. Normal pupillary responses and optic nerve appearance distinguish cortical blindness

from total blindness caused by bilateral prechiasmal or chiasmal lesions. Frequently, the disorder may go undiagnosed or be confused with hysteria. Some patients with cortical blindness will deny their blindness (Anton syndrome). Patients with cortical injury sometimes perceive moving targets (Riddoch phenomenon) and may have unformed visual hallucination.

Cortical blindness is a syndrome with a wide variety of clinical presentation and causes, including cardiopulmonary arrest, cardiac surgery, cerebrovascular accidents, head trauma, hemorrhagic shock, central nervous system infection, epilepsy, cerebral or vertebral angiography, uremia, hypoglycemia, carbon monoxide poisoning and irradiation^(2,3). Trauma is among the causes of cortical blindness. The incidence of cortical blindness associated with head injury is approximately 0.4-0.6%^(1,4). The first recorded case may have been as early as 329 B.C., Alexander the Great in Cyropolis sustained a blow to the back of his head and neck that was followed by transient loss of vision and prolonged speech problems⁽⁵⁾. Post-traumatic cortical blindness may be transient or permanent. It is more common in children, who have a more labile vasculature than adults⁽⁶⁾. This vascular etiology was supported by an increased incidence of migraine in patients both preceding and subsequent to post-traumatic transient cortical blindness⁽⁴⁾. On the basis of clinical presentation, post-traumatic cortical blindness has been classified into juvenile, adolescent, and adult types⁽⁴⁾. Juvenile type is usually transient blindness following minor head trauma that may be associated with transient unconsciousness or mental disturbance⁽⁷⁻¹³⁾. The longest duration of blindness was 10 days⁽⁷⁾. Adult type usually has a delayed onset with longer duration. It is accompanied by many neurological deficits such as hemiparesis, alexia, quadriplegia, loss of consciousness, confusion and agitation. It usually represents those cases with major head trauma and a more guarded visual prognosis⁽¹⁴⁻¹⁹⁾. The utility of Visual Evoked Potentials (VEP) in cortical blindness is controversial because a wide variety of VEP abnormalities have been described and many studies have not shown any consistent ability for this procedure to predict recovery of sight^(9,20-23). Optokinetic nystagmus (OKN) may be a more objective means of recognizing cortical blindness. The absence of an OKN response helps to distinguish cortical blindness from nonorganic vision loss. In the cooperative patient, visual field testing may be the useful methods for follow up. Pattern of visual field defect may also delineate the pathogenesis of cortical blindness. The most common visual field

pattern was recovery of central vision with continued absence of any peripheral vision⁽²⁰⁾.

Cortical blindness rarely occurs after an automobile accident, as there were only 3 cases of permanent cortical blindness arising from head trauma after a car accident⁽¹⁷⁻¹⁹⁾. In previous reports, cortical blindness after a car accident always had several neurological deficits. The case reported here presented with cortical blindness after a motorcycle accident, but without other neurological deficits. This case demonstrated a very rare condition of cortical blindness without other neurological deficits resulting from a motorcycle accident.

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รายงานผู้ป่วยสูญเสียการมองเห็นทั้งสองตาภายหลังได้รับอุบัติเหตุรถจักรยานยนต์

สุธาสินี สีนะวัฒน์, ธนภัทร รัตนภากร, ยศอนันต์ ยศไพบูลย์

ชายไทยอายุ 47 ปี เกิดการสูญเสียการมองเห็นของตาทั้งสองข้างอย่างฉับพลันภายหลังได้รับการกระทบกระเทือนศีรษะจากอุบัติเหตุรถจักรยานยนต์ โดยปราศจากความผิดปกติของระบบประสาทอย่างอื่น ระดับการมองเห็นของตาทั้งสองข้างคือสามารถมองเห็นเพียงแสงไฟ โดยไม่สามารถบอกทิศทางของแสงไฟได้ การตรวจตาอย่างอื่นอยู่ในเกณฑ์ปกติ ยกเว้น มีเพียงรอยข้ำที่เปลือกตาด้านล่างของตาทั้งสองข้างและเลือดออกในชั้นเยื่อตาข้างซ้าย การตรวจด้วยเอกซเรย์คอมพิวเตอร์พบว่ามีเลือดออกในเนื้อสมองบริเวณด้านหลังทั้งสองด้าน เส้นประสาทตาและช่องกระดูกของเส้นประสาทตาทกติทั้งสองข้าง รายงานผู้ป่วยรายนี้เป็นรายงานที่แสดงภาวะตาบอดจากการกระทบกระเทือนทางสมองโดยปราศจากความผิดปกติอื่น ๆ ของระบบประสาทภายหลังอุบัติเหตุรถจักรยานยนต์ ซึ่งเป็นภาวะที่พบบ่อย
