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Case Reports & Case Series

Cortical visual impairment as an initial clinical manifestation of post-traumatic brain injury: A case report and review of literature☆

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ABSTRACT

Background: Although unilateral epidural hematoma (EDH) has been well documented, its presentation with subdural hematoma (SDH) post-traumatic brain injury, especially on the same side and accompanied with cortical visual impairment (CVI) as an initial clinical manifestation, is a rare occurrence, thus determining the outcome.

Case description: We describe a unique case of acute post-traumatic CVI due to EDH accompanied with SDH at the right occipital lobe caused due to bone fractures and cerebral contusion that was identified at Dr. Hasan Sadikin Bandung Hospital. EDH accompanied with SDH was evacuated and the dura mater tear was repaired. The ophthalmologic *examination revealed* normal visual acuity recovery postoperatively.

Conclusion: We discuss the pathological mechanisms of acute post-traumatic CVI with an emphasis on the importance of lesions located along the visual tract.

1. Introduction

Traumatic brain injury (TBI) can manifest with various symptoms that depend on the location of the brain that has been damaged [1]. Impaired motor function and sensation, altered emotion and behavior, and impaired cognitive function are some of the common or typical clinical manifestations of TBI. According to Ponsford et al., the most common symptoms that occur after TBI are headaches, fatigue, feeling drowsy, tending to sleep more, difficulties to concentrate, feeling foggy, visual difficulties, and greater sensitivity to noise [2,3]. The occurrence of simultaneous unilateral epidural hematoma (EDH) accompanied with subdural hematoma (SDH) on the same side, even more with cortical visual impairment (CVI) as an initial clinical manifestation, is a rare case. The location of the lesions along the visual tract without any other neurological abnormality is important. We describe a unique case of acute post-traumatic CVI caused by EDH accompanied with SDH at the right occipital lobe; the evacuation of the lesions resulted in improvement and reversal of visual acuity. Several reports have described about hemianopia as the presenting symptom of EDH [1,2]; however, transient bilateral cortical blindness as a presenting EDH symptom has been very rarely reported [4]. Several previous hypotheses have been suggested to explain this phenomenon, but the exact mechanisms responsible for the development of post-traumatic CVI after TBI are still unknown. Herein, we report our case of a post-traumatic CVI caused by EDH and SDH, along with a literature review.

2. Case report

A 24-year-old male was admitted to the emergency unit with a primary complaint of blurry vision that was worsening into CVI or blindness after falling from height. His medical history did not reveal any visual problem before the accident. Neurological examination was unremarkable, except for his blurry vision. Ocular movement was good in all directions, and the pupils were reactive to light. Fundoscopy examinations were also within normal limits. The patient was later diagnosed with CVI or cortical blindness by our ophthalmologist. Computed tomography (CT) scan of the head revealed a right occipital fracture with depression and interlocking that was apparent in the bone windows; there were also right-sided occipital EDH and SDH with a significant mass effect (Fig. 1). An emergency surgery involving right-sided occipital craniectomy was planned and performed. A large EDH and SDH, on the same side, were evacuated, leaving a dural tear. As the dura was lax and pulsatile, the bone defect was replaced with titanium mesh (Fig. 2); the evacuation led to reversal of blindness. The

☆ Authors' contributions: AF, IMB, ANS and MZ had examined, treated, observed, and followed up the patient. AF and IMB performed the operation on the patient. All authors participated in writing the manuscript. All authors has read and approved of the final manuscript.

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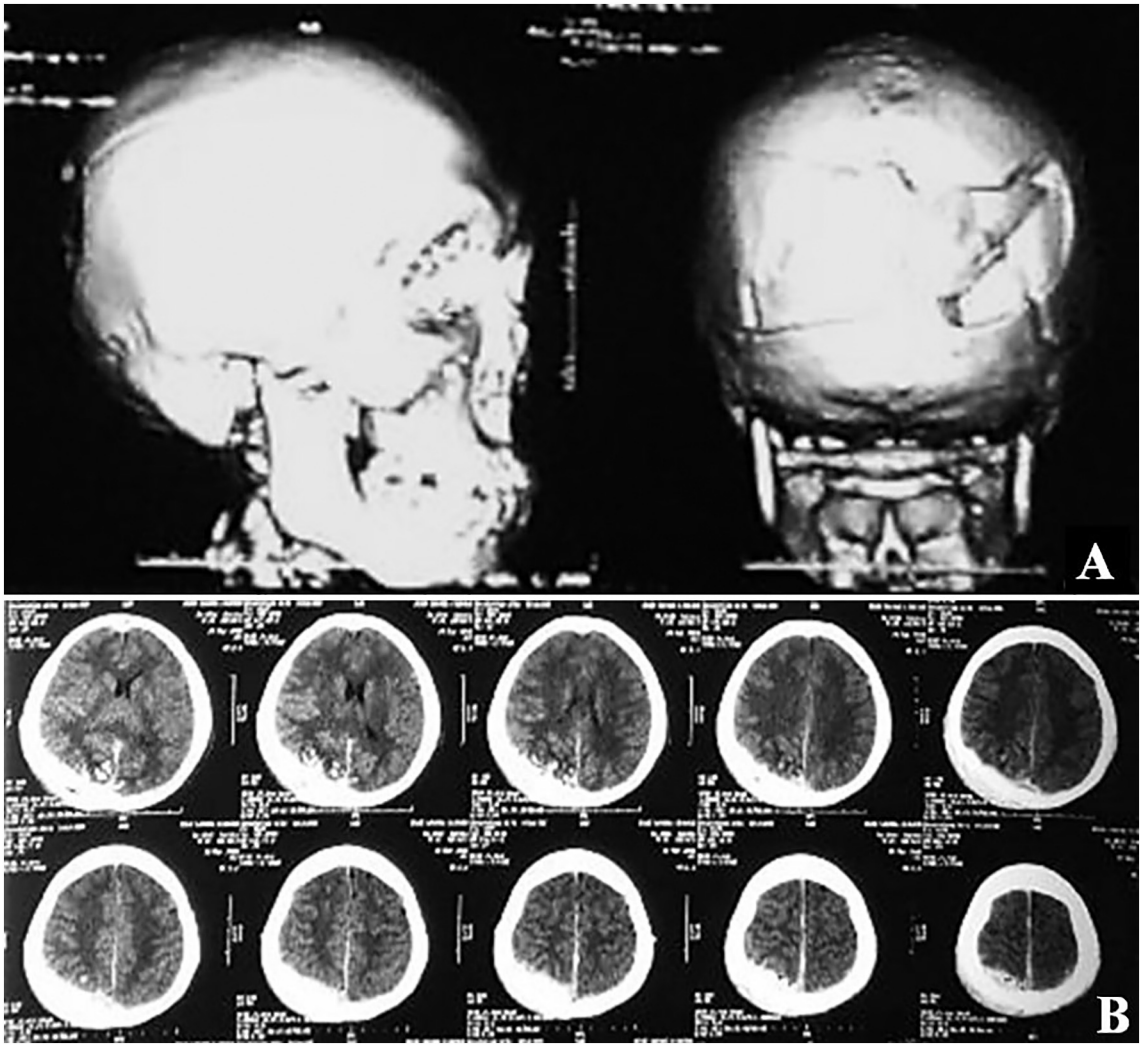

Fig. 1. Pre-operative head X-ray imaging showing depressed fracture at region right occipital (A). Pre-operative head CT scan showing unilateral right occipital hyperdense, suggestive of both EDH and SDH (B).

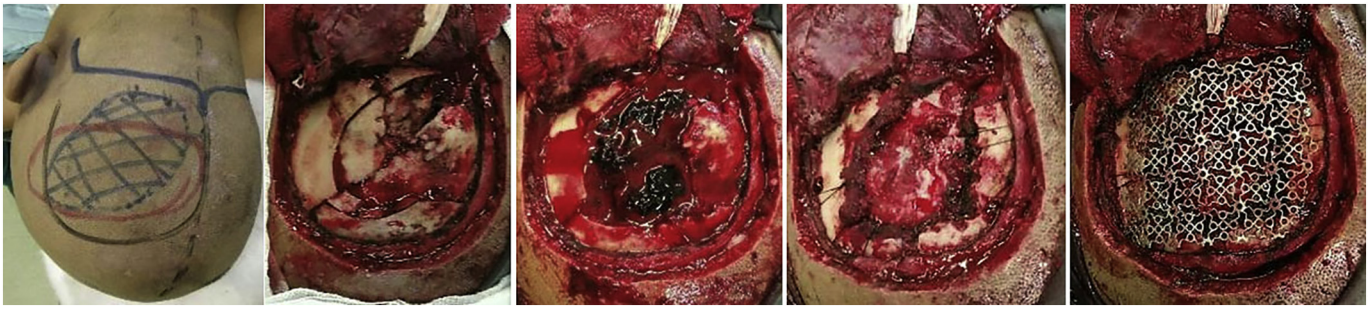

Fig. 2. Intra operative finding of depressed fracture, fragmented and interlocking sized 8 × 6 cm with both EDH and SDH, performed craniectomy evacuation following duroplasty and cranioplasty with titanium mesh.

ophthalmology *examination revealed* normal visual acuity recovery postoperatively.

3. Discussion

As mentioned earlier, even mild TBI can cause physical, cognitive, and psychosocial symptoms. It can also affect vision. The most common visual symptoms associated with mild TBI or concussion include blurring or loss of vision, double vision, photophobia, vestibular symptoms appearing in visually crowded environments, restricted peripheral vision, greater sensitivity to light, color vision deficit, and difficulty in focusing, reading, and tracking objects. The presence of such symptoms often leads to challenges for the patient at school or in the workplace, implying that special attention should be drawn [1]. Our patient came with the primary complaint of blurry vision with a history of head trauma a day before admission. The patient was later diagnosed with CVI by the Department of Ophthalmology. CVI or cortical blindness is a visual dysfunction caused due to a damage or malfunction of the retrochiasmatic visual pathways in the absence of damage to the anterior visual pathways or any other major ocular disease [5]. Bilateral visual

loss is secondary to disruption of the visual pathways posterior to the lateral geniculate nuclei [6,7]. CVI may be transient caused due to seizures, trauma, hypertensive encephalopathy, or toxins [6]. The most common cause of CVI is infarction on the bilateral occipital lobe that receives vascularity from the posterior cerebral arteries [7]. Although CVI is considered as a rare trauma complication [6,8], it is still worth to spotlight. According to our observation, this case is the first report of CVI associated with unilateral EDH and SDH after injury to the occipital lobe.

CVI develops due to dysfunction or destruction of Brodmann area 17 in both occipital lobes [7]. The exact pathophysiology of CVI following head trauma is uncertain [7], but there are three primary hypotheses. I) It is believed to be a post-traumatic vasospasm in primarily the pregeniculate area [8,9]. According to the "border-zone" hypothesis proposed by Linderberg and Spatz, the occipital cortex is one of the most susceptible regions to transient cerebral hypotension [7]. II) The most common cause of CVI in the occipital region is focal cortical edema and ischemia following the contusion. Post-traumatic cerebral edema can cause uncal herniation that compresses the ipsilateral posterior cerebral artery, resulting in unilateral or bilateral occipital lobe infarction. Intracranial lesion, EDH or SDH with mass effect, and subarachnoid hemorrhage with vasospasm and cerebral infarction may also produce visual defect [10]. III) Post-traumatic cerebral venous thrombosis (CVT), as described by Asadollahi et al., a rare case of visual disturbance after head trauma that revealed in MR venography as cerebral venous sinus thrombosis (CVST) in the left transverse sinus [11]. In our case, the ischemia and cortical edema were worsened by the presence of SDH within the area that further compresses the cortex. This might explain the improvement of the symptom following surgery.

The visual symptoms after TBI do not always manifest at the time of injury. They may persist or may be transient [7]. The degree of impairment also varies. Pursuits and saccades are the symptoms that are frequently affected by neurological events [12]. In general, the visual dysfunction after TBI can be classified into afferent, efferent and higher order dysfunctions. Table 1 describes further about the classification [3,13,14]. Regarding our patient's primary complaint of blurry vision, it might have resulted from the changes in refractive error or structural components of the eye, the cortical changes along the primary visual pathway, or an anomaly along the oculomotor pathway. Structural damage may be evident and include altered integrity of the tear film, cornea, crystalline lens, vitreous and retina. Damage along the primary visual pathway in the calcarine cortex results in loss of vision or decreased vision clarity, which may or may not be amenable to changes in refractive correction.

The required emergency assessment of visual function in the emergency room must include external eye inspection, visual acuity, confrontation visual fields, pupils, extra ocular movement inspection and funduscopic examination [10]. In our case, blindness was the only deficit that led to an emergency situation requiring an ophthalmologist since the patient was alert and had no other complaint except visual impairment. These findings are critical to exclude the differential diagnoses; as stated earlier, the cause of blurry vision in this patient varies. The visual acuity was decreased as expected. The loss of vision in CVI is bilateral, symmetric and may be severe to the level of light- or no-light-perception [6]. We can exclude structural damage in this patient because we could not detect any abnormalities from the refractive media. In CVI, pupil examinations are normal, which is in contrast to bilateral optic neuropathies [10], supporting the fact that the anomaly does not stem from the optic nerve or the oculomotor nerve. There was no disturbance in ocular movement, which further confirms that the oculomotor nerve was unaffected. The funduscopic examination was normal, suggesting that there was no pupil abnormality. We can provisionally ensure that the problem is possibly nonorganic.

CVT is a common disease in medical practice, potentially fatal but treatable and sometimes is overlooked. CVT after head trauma is not common. Dalgic et al., reported a case of CVST following minor head trauma. They concluded that post traumatic CVST is usually accompanied by depressed skull fracture, epidural or subdural hematoma [15]. The frequency of the appearance of CVT symptoms including headache (70%), followed by papilledema (28.3%), diplopia (13.5%) and visual deficit (13.2%) [16]; patients usually only have severe headache and diplopia if intracranial pressure is very high, the sixth cranial nerve compression. In the fundus papilledema is observed, with temporary visual impairment, which may be permanent [17]. A correct clinical history and imaging studies allow us to make an accurate diagnosis and timely managing this complication.

Based on the CT scan result of our patient, we confirm that the culprit was the intracranial hemorrhages occurring on the visual cortices. The mechanism involved in our case may be the direct compression of the calcarine cortices by the hemorrhages. It became much clearer when the patient regained his vision after decompressive surgery in those areas. Furthermore, there may be additional findings; lesion in one occipital lobe will cause a direct compression of the left transverse sinus by EDH, skull fracture crossing the sinus and heterogeneous distribution of hyperdense areas in the sinus. These findings suggest the possibility of thrombosis formation. The possibility that focal venous congestion in left occipital lobe may contribute to right homonymous hemianopsia; in our case, the occipital lobe was affected unilaterally due to EDH and SDH, but somehow both resulted in CVI, creating a very rare clinical situation.

It is ultimately important to locate the abnormality and treat it thoroughly. In this patient, the definitive treatment was surgical decompression of the SDH. After the surgery, some supportive measures can be applied. A combination of surgery and corticosteroids has been proposed. Corticosteroids are used because they can decrease the production of prostaglandin endoperoxides and thromboxane by decreasing the rate of protein synthesis and also inhibit the release of arachidonic acid from phospholipids. Treatment with methylprednisolone (initial loading dose 1 g IV, followed by 250 mg IV every 6 h for 72 h) has been proven to be better than dexamethasone. However, the patient should not receive a megadose of corticosteroids, as it would exacerbate ischemic-induced hippocampal excitotoxicity. Even in the absence of ischemia, glucocorticoids can induce apoptosis in the hippocampus and retinal ganglion cells [18].

4. Conclusions

CVI as an initial clinical manifestation of post-TBI in a conscious patient was suggested for further evaluation as an occipital fracture and hemorrhages were observed.

Consent

Informed consent was obtained from the patients for publication of this case report and any accompanying images. The patient's family was present at the time.

Authors' contributions

AF and IMB performed the operation. All authors participated in writing the manuscript. All authors has read and approved of the final manuscript.

Declaration of Competing Interest

The authors declare that they have no competing interests.

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