Case report

Transient post-traumatic cortical blindness due to bilateral occipital lobe infarcts in a multiply-injured patient: A case report

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ABSTRACT

Cortical blindness as a sequela of trauma has been reported in the literature but its pathophysiology remains unknown. We report a case of transient post-traumatic cortical blindness as a result of bilateral occipital lobe infarcts in a multiply injured patient after a 6-storey fall from height. We discuss the possible aetiologies for our patient’s condition and reviewed the relevant literature. An awareness of this condition and their causes is important and should be followed with the appropriate imaging and management.

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1. Introduction

Cortical blindness is defined as bilateral complete visual loss with normal pupillary responses and no other ocular abnormalities [1]. There have been many causes of cortical blindness reported in the medical literature. These include hypoxia/ischaemia (stroke, cardiac arrest), infections (meningitis, encephalitis), trauma, haemorrhagic shock, metabolic disturbances (uremia, hypoglycaemia), drugs/toxins (carbon monoxide poisoning) and post-procedural (vertebral angiography, cardiac surgery) [1,11].

The most common cause of cortical blindness is bilateral occipital lobe infarctions in the vascular territory of the posterior cerebral arteries (PCA) [2]. The most frequent finding in patients with PCA infarction is a hemianopia. Bilateral infarctions may be simultaneous and are seen in hypertensive crisis, cerebral hypoperfusion, embolism to the basilar artery or trans-ventricular herniation [2].

Transient cortical blindness has been described in the literature following head injury, often in the occipital region, and mostly in children [1,11]. Blindness may be transient or permanent and may not always manifest at the time of injury [2].

We report a case of transient post-traumatic cortical blindness secondary to bilateral occipital lobe infarcts after a 6-storey fall.

2. Case report

A 23-year-old woman was brought to the emergency department after a 6-storey fall from height. She arrived in the emergency department (ED) 25 min after the accident and was noted to have a Glasgow Coma Scale (GCS) of 15. Her parameters upon arrival were as follows: heart rate 118 beats per minute, blood pressure 90/65 mmHg, respiratory rate 23 breaths per minute and oxygen saturation 97% on room air. She sustained bilateral open calcaneal fractures, right proximal tibia and fibula fractures (Fig. 1), comminuted bilateral sacral fractures (Fig. 2), left undisplaced acetabular fracture and bilateral pulmonary contusions. There was no external evidence of head injury. In view of the trauma mechanism, a computed tomography (CT) of the brain was done soon after her arrival and was normal. She subsequently became hypotensive in the trauma bay, with a systolic blood pressure of 82 mmHg, and was resuscitated with 500mls of crystalloids and 2 l of red blood cell transfusion. She remained persistently tachycardic with the systolic blood pressure ranging between 83 and 124 mmHg.

Two hours after her arrival in the ED, she complained of complete bilateral total loss of vision. Apart from a left-sided subconjunctival haemorrhage already present since arrival, there were no other signs of global trauma. Pupillary reflexes were normal and fundoscopy showed no obvious abnormality. She underwent external fixation of her tibia fracture later the same day and then transferred to the intensive care unit (ICU) for further resuscitation and stabilization.

A magnetic resonance imaging (MRI) of her brain obtained the next day showed bilateral PCA-territorial acute infarcts in the occipital lobes with extensive cortical haemorrhagic conversion
There were also scattered small foci of bilateral PCA & middle cerebral artery (MCA) border-zone acute infarcts. Magnetic resonance angiography (MRA) of the major intracranial vessels showed normal configuration and calibre of both the anterior and posterior circulation with no flow limiting stenosis. A CT Circle of Willis and carotid angiogram done to rule out trauma to the major blood vessels showed no abnormality. Transthoracic echocardiography showed an ejection fraction of 65% and an intact inter-atrial and inter-ventricular septum. There was also no intra-cardiac vegetation seen. She did not require any inotropic or ventilatory support during her entire hospital stay.

The patient’s vision spontaneously improved over the course of her inpatient stay and she was able to perceive light on post-injury day 1. She reported to be able to see objects on post-injury day 3. Visual acuity (VA) tested using a hand-held Snellen chart over the course of the subsequent days showed a VA of 6/21, 6/12 and 6/6 on post-injury day 4, 7 and 9, respectively. A repeat CT head done on post-injury day 11 showed stability of the size and extent of the previously noted bilateral PCA infarcts (Fig. 4) and the patient’s visual acuity remained at 6/6.

Her hospital stay was complicated by rhabdomyolysis. She was eventually discharged after 33 days.

### 3. Discussion

Our patient’s uncommon presentation posed to us a challenging diagnostic dilemma.

Cortical blindness is a consequence of dysfunction or destruction of Brodmann area 17 in both occipital lobes. The degree of visual loss can vary from subtle visual field defects to complete visual loss. Trauma has been known to result in cortical blindness but the exact pathophysiology remains unknown and remains a matter of continued debate [1,13].

Injuries to the posterior parts of both occipital poles and compression and obliteration of both cerebral arteries or injury to the vertebro-basilar circulation may result in cortical blindness [13,5]. However, this was ruled out of the differential list by the CT Circle of Willis and carotid angiogram.
Fat embolism was a considered aetiology for our patient’s occipital infarction, given the history of significant trauma, her comminuted sacral and proximal tibia fractures and normal-appearing intracranial vertebral arteries and lack of features of direct brain trauma on MRI. Although a known complication of fractures of the pelvis and long bones, fat embolism syndrome (FES) typically manifests as an acute respiratory insufficiency, cerebral dysfunction and a petechial rash occurring 24–48 h after injury [8]. In addition, most reports of fat embolism and subsequent FES resulted from some form of skeletal manipulation [9].

Gurd’s criteria are commonly used for the diagnosis of FES [3]. While isolated cerebral fat embolism syndrome have been reported in the literature, they frequently present as an acute confusional state or an altered level of consciousness [8]. Seizures and focal neurological deficits have also been described. Two theories have been postulated for the pathogenesis of fat embolism, the mechanical and biochemical theories, to account for the genesis of fat droplets in the systemic circulation [8]. The mechanical theory postulates that long bone trauma disrupts fat cells in the marrow of fractured bones. The fat droplets released enter torn veins near the fracture site which are then transported to the pulmonary vascular bed where they are trapped in the lung capillaries [6]. Fat particles may pass through the pulmonary filter if they are small enough and reach the systemic circulation causing embolization in locations like the brain, kidney, retina or skin. The biochemical theory postulates that the degradation of emulsified fat, released during trauma or during the breakdown of fat, results in the production of toxic intermediaries that may occur instead or in addition to the mechanical theory as described above. Animal studies have demonstrated the hydrolysed fat may directly affect the lung pneumocytes, resulting in the adult respiratory distress syndrome. A chemical event occurring at the site of the fracture can also result in the release of mediators that affect the solubility of lipids, causing coalescence and eventual embolization. Normal chylomicrons may then coalesce and form fat globules 10–40 μm in diameter, which may occlude lung capillaries. Toxic intermediaries offer an alternative explanation for FES in non-traumatic conditions [6].

A literature search found only one report of cortical blindness as the initial presenting symptom of fat embolism syndrome after an external fixation [12]. That patient eventually developed respiratory failure and deteriorating mental status and subsequently succumbed from cardiopulmonary failure. There is no previous report of cortical blindness as the only manifestation of fat embolism in the literature. Our patient’s visual loss began 2 h after her arrival to hospital and before any orthopaedic procedure was done. She did not develop any respiratory or skin manifestations of FES throughout her entire length of stay, and thus did not fulfill Gurd’s criteria. Echocardiography did not reveal any septal defects or any echogenic material within the cardiac chambers to account for an embolic cause for her occipital infarction. The hypothesis of fat embolism for the cause of her blindness was based on the close temporal association of her injury and the onset of symptoms and the hyperintense signal on the T2 weighted images of the MRI brain. Furthermore, her visual loss spontaneously resolved. This is characteristic of the neurologic dysfunction in fat embolism, which is known to be transient and fully reversible in most cases.

Another theory propounded for the cause of her occipital infarction was the initial hypotension she had on arrival. This “border-zone” hypothesis described by Lindenberg and Spatz [11,7], postulates that in transient cerebral hypotension, the watershed areas between the distribution of the three major cerebral vessels suffer the most. The occipital cortical regions are amongst the most susceptible to post-traumatic vasospasm and the first to suffer from it [10]. This vasospasm has been postulated as the pathophysiology behind the transient post-traumatic cortical blindness seen after head injury. This entity has been well documented in the literature, especially in the paediatric population [11,2,4]. Our patient was initially in haemorrhagic shock from her pelvic and lower limb fractures and with resuscitation, her systolic blood pressure was maintained between systolic 80 to 100 mmHg during the period prior to the onset of her symptoms (to allow for permissive hypotension). Although her GCS was maintained at 15 throughout this time, the hypotension may have contributed to the cause of the occipital infarction.

Other theories we considered for the aetiologic cause included cerebral arterial spasm induced by sudden traction on the base of the brain from her fall was the initial event leading to her visual disturbance [4]. However this remains a postulation and we do not have any of definitively proving it.

4. Conclusion

Cortical blindness may occur after trauma. However, most cases regardless of aetiology, are reversible and have no long term sequelae. In the setting of polytrauma, a careful ophthalmologic and neurologic examination of the trauma patient, together with a high index of suspicion, is necessary for the diagnosis of this condition. Heightened awareness of the causes should be followed with appropriate imaging and management.

References


Fig. 4. Interval CT head on post-injury day 11.


