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Case Report

Transient cortical blindness following peripheral vascular trauma: A case report

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ABSTRACT

Cortical blindness is characterized by unilateral or bilateral vision loss despite an intact pupillary reflex, full extraocular movements, and normal fundoscopic examination. Common causes include stroke, cardiac emboli, head trauma or rarely, a hypoxic-ischemic event which results to decreased perfusion to the occipital lobes supplied by the posterior cerebral artery. Imaging with computed tomography is usually diagnostic documenting stroke or embolization as well as ensuring an intact cerebral circulation. Prognosis largely depends on the etiology as most reports document an irreversible condition or at least the patient is left with some residual visual symptoms.

We present a case of a 25-year-old male who underwent brachial artery repair with reverse saphenous vein graft interposition after sustaining a right upper arm laceration associated with massive hemorrhage and shock due to delayed consult. He presented with profound bilateral loss of vision 12 h after surgery characterized as right homonymous hemianopsia. Computed tomography of the brain demonstrated ischemic infarcts in the occipital lobes. Close observation was instituted, and his symptom resolved spontaneously within a week. This case highlights the importance of considering atypical causes of perioperative vision loss as early recognition and timely diagnosis are essential to improve patient outcomes. To our knowledge, this is the first report of transient cortical blindness after peripheral vascular trauma.

Introduction

Perioperative vision loss is a rare occurrence most frequently found after spine and cardiac surgery with reported incidence of 0.2 % and 0.09 %, respectively [1]. These surgeries commonly share features including massive blood loss, hemodynamic instability, embolic episodes, and significant inflammation which may predispose patients resulting to a lesser-known condition such as cortical blindness. Cortical blindness is defined as bilateral visual loss with normal pupillary reflexes and no other ocular abnormalities as the optic tracts and radiations are unaffected. This is due to damage to the geniculocalcarine visual pathway manifesting in several ways, which can be transient and self-limiting. Reported causes include stroke, cardiac emboli, metabolic imbalance, or head trauma [2]. Unfortunately, the literature on this topic is limited due to its rarity, consisting mostly of individual case reports and series.

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Herein we report a patient who presented with transient cortical blindness after undergoing brachial artery reconstruction for trauma associated with hemorrhagic shock. To our knowledge, this is the first report of such phenomenon in peripheral vascular trauma, highlighting the need to recognize and understand its pathophysiology in the current practice of vascular surgery.

Case presentation

A 25-year-old male came into the emergency department with a laceration at the right upper arm associated with significant prolonged bleeding as he arrived 5 h after injury. He had a Glasgow Coma Scale of 15 with no airway compromise, however, was hypotensive (80/50 mmHg), tachycardic (140 bpm), with a respiratory rate of 26 and clinically pale. Pulsatile bleeding was noted on the involved extremity with pain, muscle weakness and faint pulses. The patient was then immediately brought to the operating room for wound exploration after resuscitation. Past medical history was unremarkable with no known comorbidities. Intraoperatively, there was a transected brachial artery for which a reverse saphenous vein interposition graft was placed as vascular reconstruction (Fig. 1A, B, C). Twelve hours after the procedure, he complained of sudden and profound loss of vision bilaterally. Ocular examination confirmed bilateral vision loss with preservation of corneal and pupillary reflexes while extraocular muscle movements, intraocular pressure, and funduscopic examination were all normal. There were no neurological deficits, and his cerebellar and cognitive functions were intact. Electrocardiogram and echocardiography did not reveal any underlying cardiac pathology or source of emboli. Computed Tomography (CT) of the brain revealed bilateral occipital lobe hypodensities confirming the diagnosis of acute posterior circulation ischemia (Fig. 2A, B, C). Further neuro-ophthalmologic examination with Automated Visual Field (AVF) testing showed right homonymous hemianopsia for which close observation was done (Fig. 3). The patient thereafter noted a gradual resolution of the visual field defect and was discharged after 6 days with a Visual Acuity Score of 20/30. He currently continues to have no other visual or neurologic symptoms at 1 year of follow-up with the same visual acuity score although a repeat imaging with a cranial MRI (Magnetic Resonance Imaging) is planned.

Discussion

The Roman philosopher and politician Seneca (4 BCE to 65 CE) described a case of his wife's maid (Harpaste) who suddenly became blind and kept asking to change her quarters because she found her room to be “too dark” [3]. This was probably the first ever description of not perceiving one's own blindness in the absence of cognitive impairment or psychiatric illness. In 1595, French writer Michel de Montaigne mentioned a similar case in his book *Les Essais*, however, it was Austrian neuropsychiatrist Gabriel Anton and French neurologist Joseph Francois Babinski who eponymized visual anosognosia in 1914 [4–6]. Due to their lack of awareness of being blind, patients would often result into confabulation and denial. Cortical blindness is the result of decreased perfusion to the occipital (visual) cortex via the posterior cerebral artery commonly either from hypoperfusion or embolism. There is paucity of epidemiological data, however, there are reports that among stroke patients, incidence can range from 20 to 57 % [7].

In trauma and non-ocular surgery, cortical blindness is either the result of direct injury to the visual cortex which are often permanent or because of hypoxia-ischemia which can be transient. The latter frequently occur in children, known as post-traumatic transient cortical blindness, first described by Bodian in 1964 with an incidence of 0.4–0.6 % [8]. Children often present with hysteria, characterized as agitation and uncooperative state, because of their sudden inability to see and verbalized their condition. This phenomenon is believed to be caused by vasospasm or edema in the occipital cortex as they have a more labile vasculature than adults. Other etiologies have been previously mentioned, however, in the absence of significant head trauma, ischemia from profound

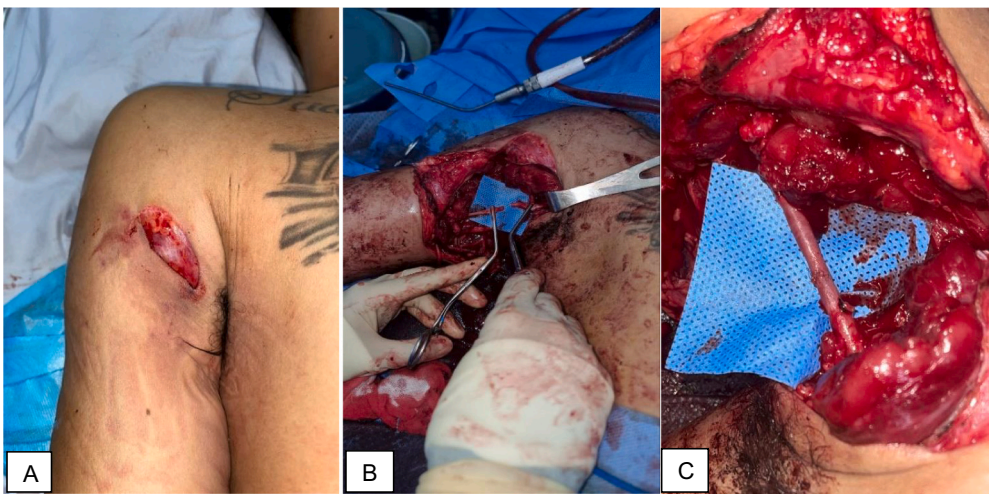

Fig. 1. Intraoperative findings demonstrating site of injury (A) brachial artery transection (B) and reverse saphenous vein graft interposition as vascular reconstruction (C).

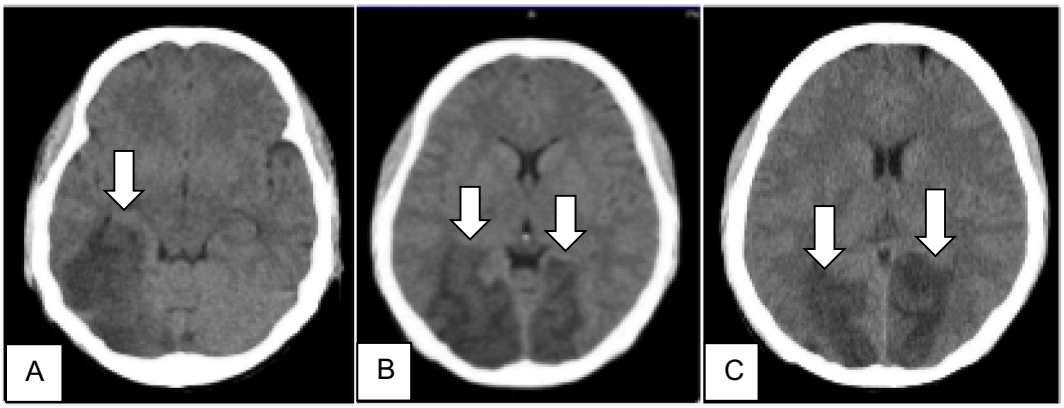

Fig. 2. Cranial CT scan showing bilateral occipital lobe infarct leading to cortical blindness (A, B, C, white arrow).

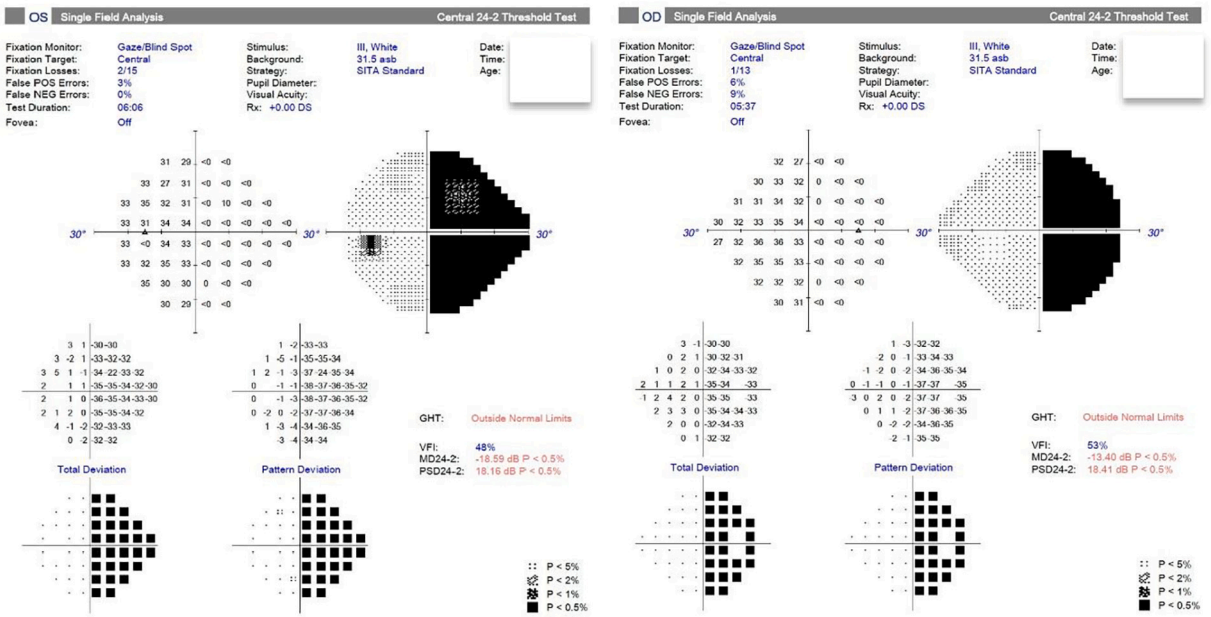

Fig. 3. Automated Visual Field demonstrating bilateral right superior and inferior visual field defects (homonymous hemianopsia).

hypotension is an atypical but plausible explanation in our case.

Visual disturbance can manifest in various ways such as sudden blindness, visual neglect, agnosia, or perceptual disorders. The posterior cerebral artery arises from the basilar artery which is formed by the right and left vertebral arteries with its terminal branches supplying the occipital lobes. They are considered watershed areas making them prone to hypoxic damage from ischemia. When one side is affected, the patient presents with contralateral homonymous hemianopsia, however, if both sides suffer ischemic insult, the patient may have peripheral vision loss or complete blindness. A complete neurologic, ophthalmologic, and cardiac evaluation are essential. Nonetheless, delayed presentation of symptoms has been reported. Limaye et al. described a case of cortical blindness that occurred days after an initial anoxic event [9].

Diagnosis is made by clinical examination and radiographic evaluation with CT scan being the initial diagnostic imaging of choice to document stroke or embolization. Angiography is occasionally performed to ensure that the cerebral circulation is intact. Conversely, cortical blindness can occur as complication of diagnostic procedures using contrast including coronary, cerebral, and vertebral angiography [10]. While MRI is superior to CT in demonstrating stroke, it may not always be readily available and can be costly. Although the CT scan findings and visual field examination may seem discordant in this case, a likely explanation would be a transient cerebral vasospasm exacerbated by hemorrhagic shock and hypoperfusion. Furthermore, there was a delay in the visual field testing of several days after the imaging was performed.

Ischemic optic neuropathy is a similarly rare disease occurrence in trauma which can also present with sudden blindness, however, CT scan findings are normal for these patients. Asensio et al., described such patient with penetrating thoracoabdominal trauma

associated with profound shock [11]. Unfortunately, vision never recovered, and patient remained blind even after 3 years from injury. This condition has been documented among burn patients as well. Medina et al., reported a series of 3 patients with >50 % burn areas associated with hypotension, massive resuscitation, and sepsis with all patients having a dismal recovery of vision [12].

The optimal management of cortical blindness remains limited and unclear albeit with good prognosis. The use of corticosteroids, antiplatelets and adequate hydration have been postulated however, recent literature has provided no evidence for any specific therapeutic intervention other than close monitoring as the condition is generally reversible and resolves within a week. Visual training and rehabilitation with restitution therapy may help recover visual field defects [13].

Conclusion

In conclusion, cortical blindness in peripheral vascular trauma is an unusual complication. Prolonged hypotension from massive blood loss leading to hypoperfusion of the occipital cortex can explain such sequelae. Although it usually resolves spontaneously, increased awareness of its potential to occur may aid in prompt diagnosis, improvement of outcomes and monitoring for long-term complications.

CRediT authorship contribution statement

Dante G. Ang: Writing – review & editing, Data curation. **Siegfredo R. Paloyo:** Writing – review & editing, Conceptualization. **Ferri P. David-Paloyo:** Writing – review & editing, Supervision. **Mayou Martin T. Tampo:** Writing – original draft, Data curation. **Emmanuel T. Limpin:** Writing – original draft, Data curation. **Eduardo C. Ayuste:** Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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