eNeurologicalSci 13 (2018) 33–34

Contents lists available at ScienceDirect

eNeurologicalSci

journal homepage: www.elsevier.com/locate/ensci

Letter to the editor

Delayed cortical blindness in hypoxic-ischemic encephalopathy

Dear Editor,

Cortical blindness (CB) is known to be caused by damage to the geniculocalcarine visual pathways [1]. CB is most commonly associated with cerebrovascular diseases, but it can also occur as a complication of cardiac surgery or cerebral angiography [1]. Here, we report an extremely rare case of delayed-onset CB after resuscitation from cardiac arrest.

A 42-year-old man with no significant medical history experienced out-of-hospital cardiac arrest of unclear etiology. He regained spontaneous circulation after undergoing cardiopulmonary resuscitation (CPR) for 2 min. The intervals between the last normal/first abnormal time and the initiation of CPR were 134 and 14 min, respectively. After undergoing therapeutic hypothermia, the patient showed no neurological deficit and brain magnetic resonance imaging (MRI) showed no significant findings (Fig. 1-A). Transthoracic echocardiography and 24-hour holter monitoring showed no evidence of cardiac abnormalities.

Four days after regaining consciousness, the patient presented with acute-onset decreased visual acuity in both eyes, as well as a myoclonic jerk of the left arm. During the ophthalmological examination, he had prompt pupillary reflexes to light, normal ocular movements, and normal fundoscopic findings. His optical coherence tomography results were normal; nonetheless, his best-corrected visual acuity measured using a Snellen chart was 0.02/0.02 in decimals and he could not perceive shape or color. Psychosomatic blindness was ruled out by the absence of reflexive eye blinking to a visual threat. A follow-up brain MRI showed subtle T2 high signal intensities along the bilateral occipital cortices (Fig. 1-B). The results of the Humphrey test showed near-complete blindness in both eyes (Fig. 1-C). The posterior alpha rhythm was absent on the electroencephalogram. Twenty-four days after regaining consciousness, his visual acuity was significantly improved with the appearance of metamorphopsia.

Hypoxic-ischemic encephalopathy (HIE) usually affects the neocortex, deep cerebral gray nuclei, cerebellum, and hippocampi [2]; however, a recent case series reported that the occipital lobe can be an isolated target of HIE [3]. Although the primary visual cortex is generally not considered to be vulnerable to HIE, it has several features that could make it susceptible to this condition. For example, the blood supply of the visual cortex arises from the terminal branches of the posterior cerebral artery, and the granular cells in the occipital striate cortex are reported to be less resistant to hypoxic injury [4].

CB is a rare, but well-known manifestation of HIE in the acute phase of disease. To our knowledge, there have been only a few case reports on delayed-onset CB caused by HIE [5–7]. The pathophysiology of delayed-onset CB is poorly understood; however, possible mechanisms include cumulative oxidative stress and glutamate excitotoxicity leading to neuronal cell damage, as well as cortical laminar necrosis affecting layers 3 and 4 of the striate cortex [5,6]. The present case highlights that CB can manifest days to weeks after the hypoxic event in HIE patients who have no immediate neurological deficits. A high degree of clinical suspicion is needed to make the right diagnosis.

https://doi.org/10.1016/j.ensci.2018.11.020
Received 23 October 2018; Accepted 17 November 2018
Available online 19 November 2018

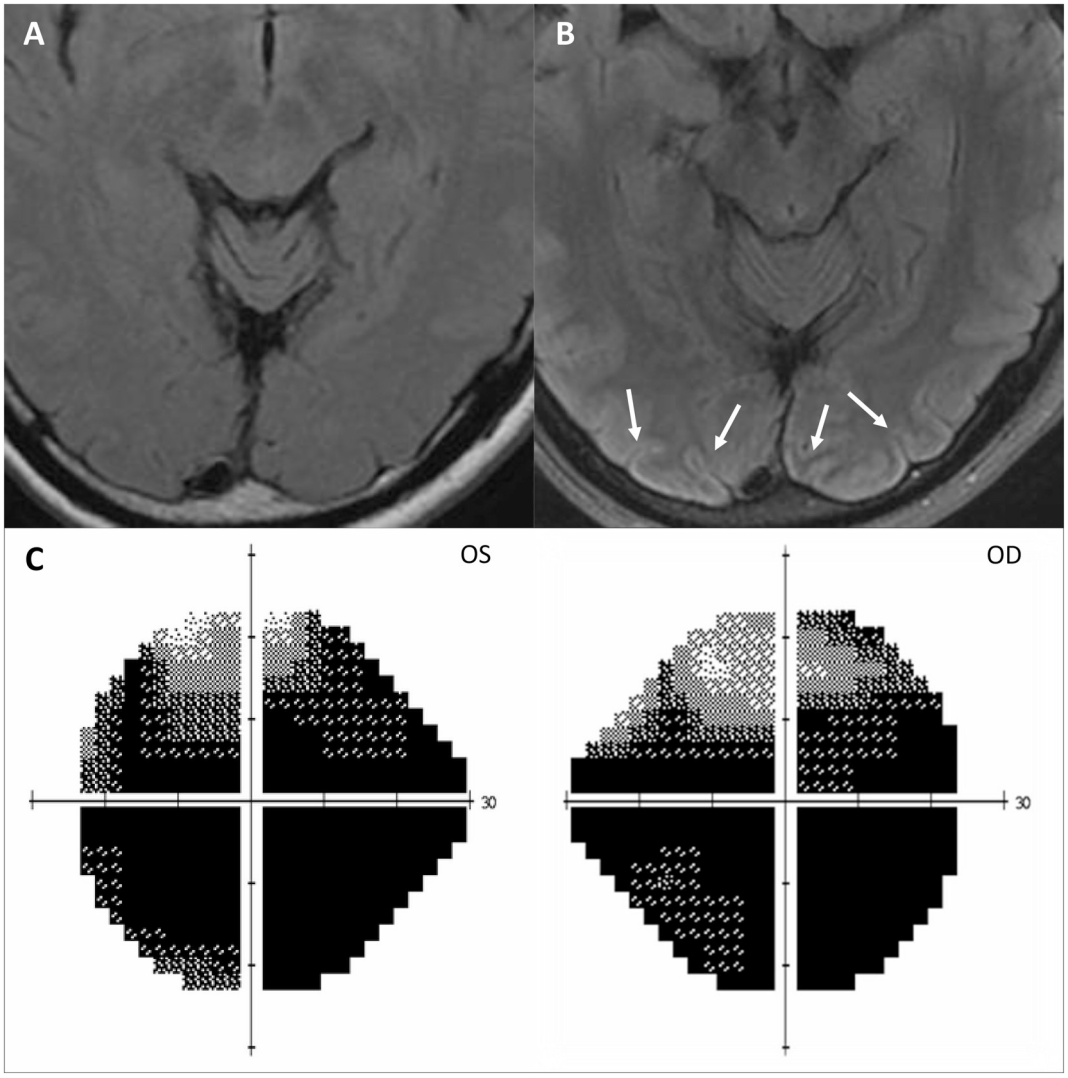

Fig. 1. Brain magnetic resonance imaging. (A) Initial fluid-attenuated inversion recovery (FLAIR) image shows no significant signal abnormalities, (B) Follow-up FLAIR image shows subtle T2 high signal intensities along the bilateral occipital cortices (white arrows), (C) The results of Humphrey visual field test demonstrate near complete blindness in the bilateral eyes.

Conflicts of interest

The authors have no financial conflicts of interest.

Funding

This work was supported by INHA UNIVERSITY HOSPITAL Research Grant.

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