JAMA Ophthalmology Clinical Challenge

Delayed Vision Loss Following Cardiopulmonary Resuscitation

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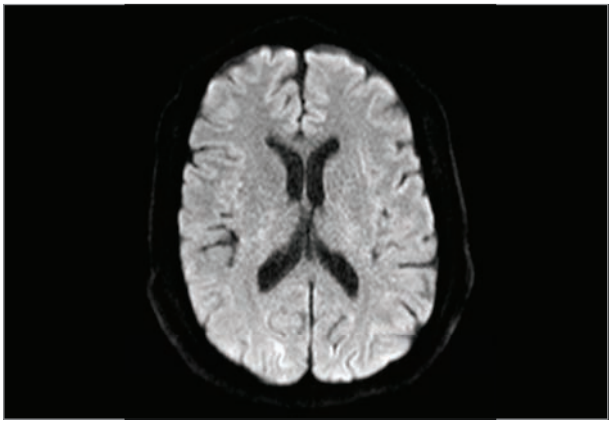

Figure 1. Diffusion-weighted imaging axial magnetic resonance imaging brain 3 days after symptom onset with very subtle evidence of areas of diffusion restriction in the bilateral occipital lobes. There was no posterior cortical atrophy or other abnormality.

A man in his early 50s was admitted to the hospital after acute hypoxemic respiratory failure and cardiac arrest following aspiration, requiring 3 minutes of cardiopulmonary resuscitation. The patient was intubated on the scene and remained intubated due to lactic acidosis and elevated liver function test results. After these abnormalities normalized 3 days later, he was extubated and breathing comfortably on room air with normal vital signs, mild right-sided weakness, and no other known complications. One day after extubation, he noticed blurry vision peripherally and difficulty focusing at near. Ophthalmology was consulted. On evaluation, he had near visual acuity (VA) of 20/25 OU, no afferent pupillary defect, and full but slow confrontational visual fields (VF) on bedside examination. Anterior segment and dilated fundus examination results were normal. Results from computed tomography (CT) head scan without contrast and magnetic resonance imaging (MRI) brain scan with and without contrast were unremarkable. Two days later, the patient noted worsening vision and was found to have a VA of 20/40 in each eye with homonymous right-inferior quadrantanopia. A code stroke was called with repeat CT scan/CT angiography and MRI brain both with and without contrast that remained unremarkable, as did repeat serum electrolytes and metabolic panels (**Figure 1**). The following day, the patient noted further vision loss and was found to have bilateral light perception vision. There was no pupillary defect, blink to threat, optokinetic reflex response, or intraocular pathology explaining the substantial vision loss including fundus abnormalities. Repeat CT scan/CT angiography demonstrated no abnormalities.

WHAT WOULD YOU DO NEXT?

- **A.** Repeat brain MRI
- **B.** Obtain an electroretinogram
- **C.** Consult psychiatry
- **D.** Observation

CME Quiz at jamacmelookup.com

Diagnosis

Delayed cortical vision loss

What to Do Next

D. Observation

Discussion

Cortical blindness occurs secondary to damage to the geniculocalcarine visual pathways and has been described in the setting of cerebrovascular disease, extensive surgeries particularly cardiac procedures, hypoxic-ischemic insults, and encephalopathy.[1,2] Visual loss may present as a decrease in VA and/or VF loss, which is often incomplete.[1] The remainder of the ophthalmic examination is generally unremarkable given the intact anterior visual pathway, with normal pupillary light reflexes, anterior segment, and optic nerve on fundus examination.[3] Other testing, such as electroretinogram, should not be affected by isolated cortical vision loss. Visual evoked potential may have diagnostic utility, though it has not been extensively studied in this setting.[4] Visual evoked potential was not available in this case.

There are several reported cases of cortical blindness after cardiac arrest with subsequent prolonged resuscitation and/or intubation efforts. Several mechanisms of injury to the postgeniculate visual pathways have been proposed, including lactic acid ac-

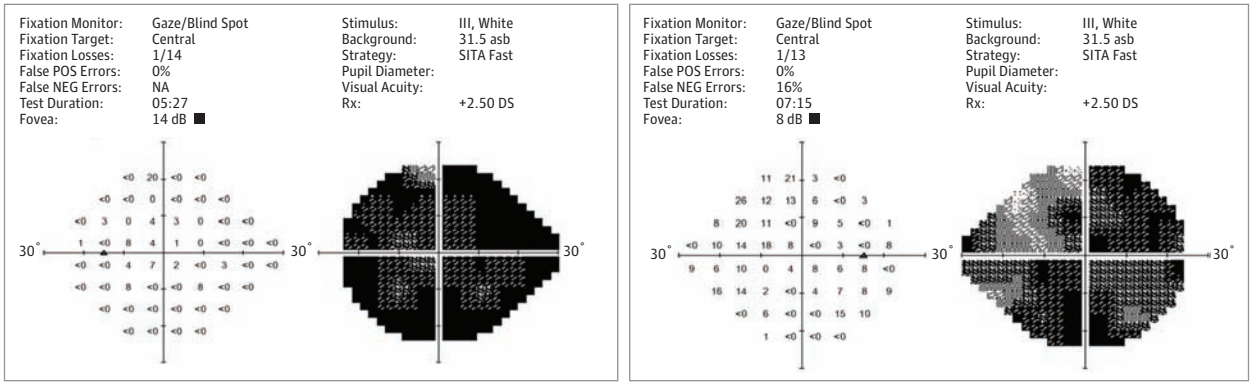

Figure 2. Humphrey visual-field (VF) testing performed at follow-up visit 5 months after cardiac arrest, demonstrating generalized depression of the right and left eyes with reduced foveal thresholds. Fixation loss is likely low due to preserved central vision. dB indicates decibel; NA, not applicable; Rx, prescription; SITA, Swedish interactive threshold algorithm.

cumulation, oxidative stress, and glutamate-induced glial and neuronal injury.[2,3] Brain MRI following presentation of cortical blindness is often unremarkable or demonstrates only subtle changes, such as mild diffusion restriction detected in the occipital lobes, easily overlooked if appropriate clinical history is not provided.[3,5,6] In this case, the patient's latest MRI was reviewed by neuroradiology following clinical diagnosis and areas of diffusion restriction in the bilateral occipital lobes and bilateral perirolandic regions were noted. Delayed MRI (eg, 6 months after injury) may demonstrate cortical volume loss corresponding to the primary visual cortex.[5,6] However, multiple repeat MRI scans in the acute setting may have little additional diagnostic utility. Results of tests for bilateral functional vision loss, such as optokinetic reflex, drum response, and abnormal finger touching, should be negative.

While cortical vision loss is generally reported as rapid onset following hypoxic-ischemic injuries, delayed-onset cortical vision loss, occurring days to weeks following the initial insult, has been reported following cardiopulmonary resuscitation.[1-3,7] These few reported cases have described a relatively rapid improvement in visual recovery, on the order of weeks, suggesting a reversible etiology and overall good prognosis, possibly secondary to improvement of cortical changes, such as axonal reconstitution or resolution of edema indiscernible on imaging.[1,3] This case demonstrates the importance of maintaining suspicion for delayed cortical vision loss following a hypoxic-ischemic insult and not to presume functional origin despite the atypical course and unremarkable ophthalmic examination and imaging.

Patient Outcome

At the time of discharge, the limited reports on similar presentations were discussed with the patient, as well as the potential for visual improvement. During most recent neuro-ophthalmology clinic follow-up visit 5 months after initial injury, the patient noted improvement in vision but with persistent flashing lights and formed visual hallucinations of animals and people suggestive of Charles Bonnet syndrome.[8] His VA had improved to 20/25 OD and 20/20 OS. Humphrey VF testing demonstrated diffuse VF loss in both eyes (**Figure 2**). The patient noted significant subjective improvement beyond that suggested by VF testing. He was also referred for low-vision evaluation and rehabilitation to aid in performing activities of daily living and navigating.

ARTICLE INFORMATION

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