รายงานผู้ป่วย

ภาวะตาบอดที่แสดงอาการล่าช้าในผู้ป่วยภาวะสมองขาดเลือดจากหัวใจหยุดเต้น

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บทคัดย่อ

ภาวะตาบอดที่เกิดจากพยาธิสภาพของสมองส่วน Occipital lobe เกิดได้จากหลายสาเหตุ เช่น ภาวะสมองขาดเลือดเฉียบพลันจากโรคหลอดเลือดสมองตีบ ภาวะสมองขาดเลือดจากหัวใจหยุดเต้น หรือเกิดจากการได้รับสารพิษ โดยทั่วไปการเกิดภาวะตาบอดจากภาวะสมองขาดเลือดจากหัวใจหยุดเต้น ผู้ป่วยจะมีอาการทันทีหลังจากที่ผู้ป่วยกลับมารู้สึกตัว การเกิดอาการตาบอดหลังจากที่ผู้ป่วยกลับมารู้สึกตัวและมีการมองเห็นที่ปกติแล้วในระยะแรก มีรายงานน้อยมาก รายงานผู้ป่วยนี้ได้นำเสนอผู้ป่วยชายอายุ 33 ปีที่มีภาวะตาบอดเฉียบพลัน 5 วันหลังจากที่ผู้ป่วยเกิดภาวะหัวใจหยุดเต้น ดังนั้นจึงควรคิดถึงภาวะตาบอดที่แสดงอาการล่าช้าในผู้ป่วยที่มีภาวะหัวใจหยุดเต้น ถึงแม้ผู้ป่วยจะมีการมองเห็นที่ปกติในระยะแรก

คำสำคัญ: ตาบอด, สมองขาดเลือด, หัวใจหยุดเต้น

CASE REPORT

Delayed cortical blindness from hypoxic ischemic encephalopathy after cardiac arrest

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ABSTRACT

Cortical blindness (CB) is characterized by bilateral vision loss secondary to occipital lobe lesions. The most common causes of CB are bilateral occipital lobe infarction, hypoxemia, and toxic exposure. Complications of hypoxic-ischemic encephalopathy (HIE) usually appear immediately after cardiopulmonary resuscitation. Multifocal myoclonus is a recognized late complication of HIE; however, delayed occurrence of bilateral visual loss associated with HIE is rarely reported. We reported a case of delayed CB (5 days) after cardiac arrest in 33-year-old man. Physicians should be aware of this condition, even if patients have normal vision early after regaining consciousness.

KEYWORDS: cortical blindness, hypoxic-ischemic encephalopathy, cardiac arres

INTRODUCTION

Cortical blindness (CB) is characterized by bilateral loss of vision secondary to disruption of the geniculocalcarine visual pathways. Visual loss and absent optokinetic nystagmus in the presence of a normal fundoscopic examination and pupillary light reflex are the clinical hallmarks of CB[1]. The most common cause of CB is bilateral occipital lobe infarction. However, etiologies such as hypoxemia, posterior reversible encephalopathy syndrome, traumatic brain injury, tumors, meningoencephalitis, hypoglycemia, and exposure to toxins such as cyclosporine and tacrolimus have been reported.[1-3] Complications of hypoxic-ischemic encephalopathy (HIE) usually appear immediately after cardiopulmonary resuscitation (CPR).[4] Usually, a late complication of HIE is multifocal myoclonus; however, rarely, delayed occurrence of bilateral visual loss associated with HIE has been reported.[5] Here, we report such a case in a patient after CPR.

Case presentation

A 33-year-old man with no pre-existing medical conditions suddenly lost consciousness due to ventricular tachycardia caused by Brugada syndrome. After 16 minutes of CPR, spontaneous circulation was restored, and he regained consciousness. Immediate neurological examination, including vision, was normal. Five days after regaining consciousness, he reported acute bilateral vision loss on waking. The neurological examination revealed a normal level of consciousness, no motor weakness, and normal sensory perception. Unfortunately, he had developed memory loss and total bilateral loss of vision. A detailed ophthalmological examination revealed normal pupillary light reflexes and optic fundi. His visual acuity was limited to light perception. Psychogenic blindness was ruled out based on a visual threat test, which showed a lack of reflexive blinking. Laboratory investigations, including a complete blood count and blood chemistry tests, were normal. Brain magnetic resonance imaging (MRI) showed a symmetrical area of restricted diffusion involving the bilateral basal ganglia, medial temporal lobes, and occipital cortical sulci (figure1). His vision gradually improved, and he could achieve finger counting eight weeks after the onset.

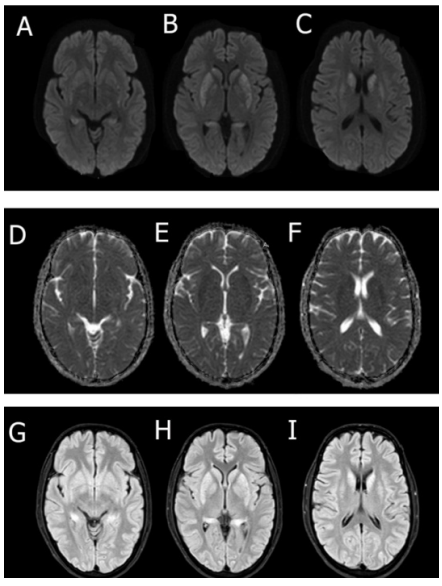

Figure1: MRI of the patient; (A, B, C) Axial diffusion-weighted imaging, (D, E, F) apparent diffusion coefficient, and (G, H, I) axial T2-weighted fluid-attenuated inversion recovery sequence shows the symmetrical area of restricted diffusion involving the bilateral basal ganglia, medial temporal lobe, and occipital cortical sulci.

DISCUSSION

HIE results from hypoxemia and hypoperfusion of the central nervous system. Usually, it affects the cerebral cortex, basal ganglia, hippocampus, and cerebellum, because the neurons in these regions have high metabolic demands.[4] The susceptible locations of the cerebral cortex are the border zones of the anterior, middle, and posterior cerebral artery territories. However, some case reports show that the occipital lobe may be vulnerable to HIE.[6-10] There are several possible explanations. For example, the primary visual cortex is supplied by the terminal branch of the posterior cerebral artery, and the granular cells in the occipital striate cortex may be less resistant to hypoxic injury. Interestingly, there have been reports of patients with a normal initial brain MRI who subsequently developed occipital lobe encephalomalacia during follow-up.[6,7]

To date, there have been few case reports of delayed CB from HIE.[6-10], and its pathophysiology remains unclear. A possible mechanism is secondary brain tissue injury due to a mismatch between oxygen demand and supply in vulnerable areas. Several mechanisms underlying the secondary injury have been proposed: first, endothelial dysfunction leading to further neuronal dysfunction. Second, impairment of vasomotor regulation and decreased nitric oxide production causing vasoconstriction. Finally, intravascular fluid leakage through an impaired blood-brain barrier with perivascular edema, resulting in increased cerebrovascular resistance. Other mechanisms, including increased free radical release, glutamate production, and intracellular calcium accumulation, have also been proposed.[11] The vision loss prognosis is mixed, ranging from no improvement to a return to normal visual acuity.[6-10] Our patient's vision had improved after eight weeks from onset.

SUMMARY

We present an unusual case of delayed CB associated with HIE to raise awareness of this rare and debilitating complication. The case shows that CB associated with HIE can develop a few days after the hypoxic event. Therefore, physicians should be aware of this condition and should perform a brain MRI to confirm the diagnosis.

Conflicts of Interest: None

Financial Support: None

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