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Irreversible Loss of Vision in a Child due to Occipital Infarction after Gastroenteritis

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Key Words

Occipital infarct · Gastroenteritis · Visual recovery

Abstract

A 2½-year-old girl developed a bilateral occipital infarct following severe gastroenteritis with bilateral vision of light perception. Evaluations for sickle cell anemia, hemolytic anemia and coagulopathies were negative. Cortical blindness is an uncommon but dramatic complication of gastroenteritis, hence the need of prompt hydration and other supportive measures to avoid irreversible visual loss or mental sequela.

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Introduction

Vision is well represented in the brain. It has been estimated that visual processing areas occupy more than half of the total surface of the primate neocortex [1]. Acute profound loss of vision from occipital infarct is a devastating event to the patient and family [2–10]. Despite significant medical progress over the past several decades, cortical blindness can still affect patients of various ages due to what is believed to be a relatively benign illness. We present a very young girl with cortical blindness following rotavirus gastroenteritis.

Case Report

This 2½-year-old girl was born at term after a normal pregnancy and uneventful labor. She had normal developmental milestones until she developed protracted afebrile diarrhea and vomiting for 2 days, necessitating the admission to a hospital and the insertion of an intravenous line, which was a difficult task in this dehydrated child. Her hematocrit was 15.7 with a normal electrolyte profile and positive rotavirus antigen. The girl required a blood transfusion for acute anemia during her admission to the hospital. The family history was positive for glucose-6-phosphate dehydrogenase (G6PD) deficiency. One day after hospitalization, the patient was noted to have lost her vision completely. MRI of the brain documented a bilateral occipital lobe infarction (fig. 1). She was started on steroid therapy and good hydration. A week later, her hematocrit level rose to 32. The patient continued to have light perception vision bilaterally. She made no eye contact. A fixation was absent and there was no blink response to threat. The pupillary light reflexes were normal. The fundus examination was normal and the neurological examination was nonfocal. Workup was negative for sickle cell anemia. Serum levels for antithrombin, protein S, protein C, pyruvate kinase (hemolytic anemia), G6PD, amino acids and thyroid function tests were within normal range. The working diagnosis was a hypotension-induced occipital infarct. The patient was placed on oral corticosteroid therapy for 2 months, without any change in visual acuity.

Discussion

It has been shown in a number of studies that the distribution of etiology of cerebral infarction among young patients differs from older patients. Furthermore, it has been shown that the long-term prognosis among young patients with cerebral infarction differs in terms of underlying risk factors. Occipital infarction is associated with younger age, female sex, and a prothrombotic state while extraoccipital infarction is more often associated with systemic hypertension and hypercholesterolemia [9].

Hypoxic-ischemic brain injuries are the commonest cause of cortical visual impairment in children. The resulting visual impairment relates to the age of the subject and both the severity and the duration of hypoxia [3]. Full-term infants commonly sustain injury at the watershed zones of the cerebral cortex, while premature infants have periventricular injury with little cortical damage [3]. Occipital infarction is often associated with an irreversible loss of visual perception [7]. However, unlike adults, children appear to have a better visual prognosis with a better chance for recovery. Plasticity (ability to reorganize and recover) of the child brain has been implicated in this visual recovery [3]. Bova et al. [10] described the visual recovery of a male child who had a bilateral occipital lobe infarction at the age of 2 years and 6 months, 10 days after a colon resection for Hirschsprung disease. In the acute phase, there was severe visual impairment without any visual response, but eventually, the patient recovered the ability to perceive movement, followed by the recovery of visual acuity and accompanied by a progressive reduction of the visual field defect. At 6 years and 8 months, visual recognition acuity was 20/20 in both eyes, with a persistence of the visual field defect in the upper hemifield, and he was also able to see overlapping figures. According to case reports and various animal models [11], processes related to cerebral plasticity may take years to develop after a brain lesion.

In our case, systemic hypotension (hypovolemic shock) from rotavirus gastroenteritis and dehydration [12] most likely led to a decrease in blood flow (oxygen and nutrients) to a critical level, jeopardizing the occipital lobe region in the watershed area. Hemolytic anemia,

triggered possibly by the virus infection, contributed also to the hypoxia theory herein, although we lacked laboratory documentation in our case. Watershed infarcts occur at the border zones between major cerebral arterial territories as a result of hypoperfusion. The occipital lobe is supplied most often by the posterior cerebral artery, a terminal branch of the basilar artery. Hence, occipital lobe infarcts are traditionally attributed to vertebrobasilar disease [13]. Occasionally, the occipital lobe is supplied uniquely by branches of the internal carotid disease in up to 11% of subjects [13].

Sickling crisis [5, 8] or thrombogenic risk factors (coagulation disorders) [4] can predispose children to cerebral infarcts, especially with a precipitating cause (surgical intervention, blood loss or viral illness like gastroenteritis). Management strategies include immediate hydration, correction of electrolyte imbalance, reversal of anemia, control of body temperature, oxygen administration (in case of hypoxia) and the administration of corticosteroid to decrease coexistent brain edemas from infarction. Although our patient did not show an improvement after 2 months, hope still exists that partial visual recovery will occur gradually years after the vascular event.

Disclosure Statement

The authors report no conflict of interests.

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Fig. 1. MRI of the brain upon admission: axial FLAIR image at the level of the occipital region showing symmetric high-signal intensity changes involving the cortical gray matter and the subcortical white matter of both occipital lobes. A prominent ventricular dilatation is also noted.