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Cortical Laminar Necrosis Caused by Critically Increased Intracranial Pressure in an Infant

—Case Report—

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Abstract

A 3-month-old boy presented with critically elevated intracranial pressure (ICP) due to bilateral subdural hematomas, which resulted in diffuse cortical laminar necrosis, manifesting as a 1-week history of appetite loss, fever, and intermittent seizure. Initial computed tomography revealed bilateral subdural fluid collections. Burr hole drainage was carried out to control the ICP. T_1-weighted magnetic resonance imaging on day 26 revealed diffuse linear hyperintense lesions, which suggested cortical laminar necrosis. This is an extremely unusual case of cortical laminar necrosis caused by elevated ICP due to subdural hematoma in an infant.

Key words: cortical laminar necrosis, magnetic resonance imaging, diffusion-weighted imaging, subdural hematoma

Introduction

Cortical laminar necrosis is defined as the breaking down of a definite cell layer, especially in layers 3 and 5, in the cerebral cortex, which typically occurs after temporary cardiac arrest and hypoglycemia.[14,15] Cortical laminar necrosis is a classic entity in adulthood related to conditions of energy depletions, but is little known in children and infants.[11] Here we present unique imaging findings of a 3-month-old boy, who suffered critically elevated intracranial pressure (ICP) due to bilateral subdural hematomas, resulting in diffuse cortical laminar necrosis.

Case Report

A 3-month-old boy presented with a 1-week history of appetite loss, fever, and intermittent seizure at our hospital. His past medical history was unremarkable. No obvious trauma scars were found on the outer body surface. His head circumference was 48 cm (exceeding normal limits by 2 standard deviations) and the anterior fontanel was bulging, which implied increased ICP. Computed tomography (CT) revealed bilateral huge subdural fluid collections, with densities slightly higher than that of the cerebrospinal fluid, and no obvious abnormality in the cerebral hemispheres (Fig. 1A). Diffusion-weighted magnetic resonance (MR) imaging showed diffuse hyperintense lesions in the right frontal and parietal lobes, as well as the bilateral occipital lobes (Fig. 1B). His ICP was expected to be critically high, so burr hole drainage of the subdural fluid collections was carried out.

The initial pressure was 30 cmH_2O and serous, slightly bloody fluid was drained. Drainage tubes were inserted for continuous drainage. His ICP gradually decreased and normalized within 3 days, and the drainage tubes were withdrawn 7 days after the surgery. The anterior fontanel continued to be concaved after removal of the drainage tubes, but CT on day 14 showed residual subdural hematomas with marked diffuse brain atrophy in the bilateral

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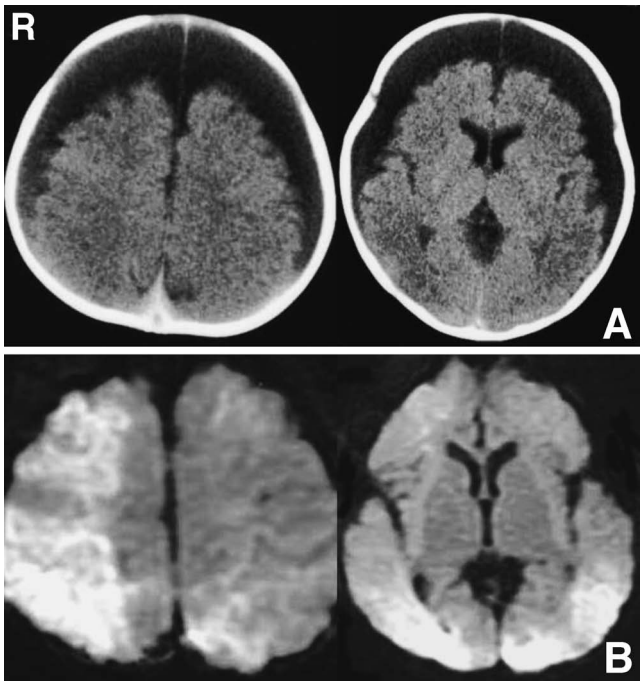

Fig. 1 **A: Computed tomography scans showing bilateral subdural fluid collections with densities slightly higher than that of cerebrospinal fluid. B: Diffusion-weighted magnetic resonance images revealing diffuse hyperintense lesions in the right frontal and parietal lobes, and the bilateral occipital lobes.**

cerebral hemispheres. No evidence of hemorrhagic change was detected in the cerebral hemispheres (Fig. 2). T_1- and T_2-weighted MR imaging on day 26 revealed diffuse linear hyperintense lesions in the bilateral cerebral hemispheres, which suggested cortical laminar necrosis (Fig. 3).

Discussion

The present case of a 3-month-old boy with critically elevated ICP due to bilateral subdural hematomas is

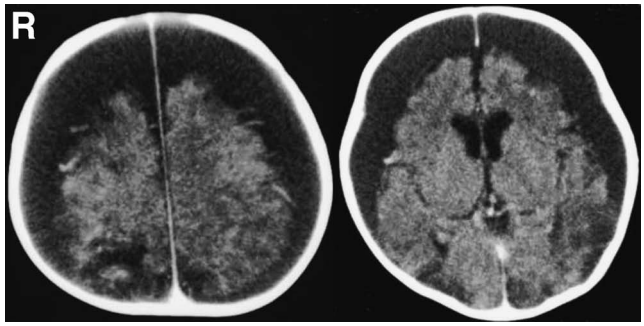

Fig. 2 **Computed tomography scans on day 14 demonstrating residual subdural hematomas with marked diffuse brain atrophy in the bilateral cerebral hemispheres, but no evidence of hemorrhagic change in the cerebral hemispheres.**

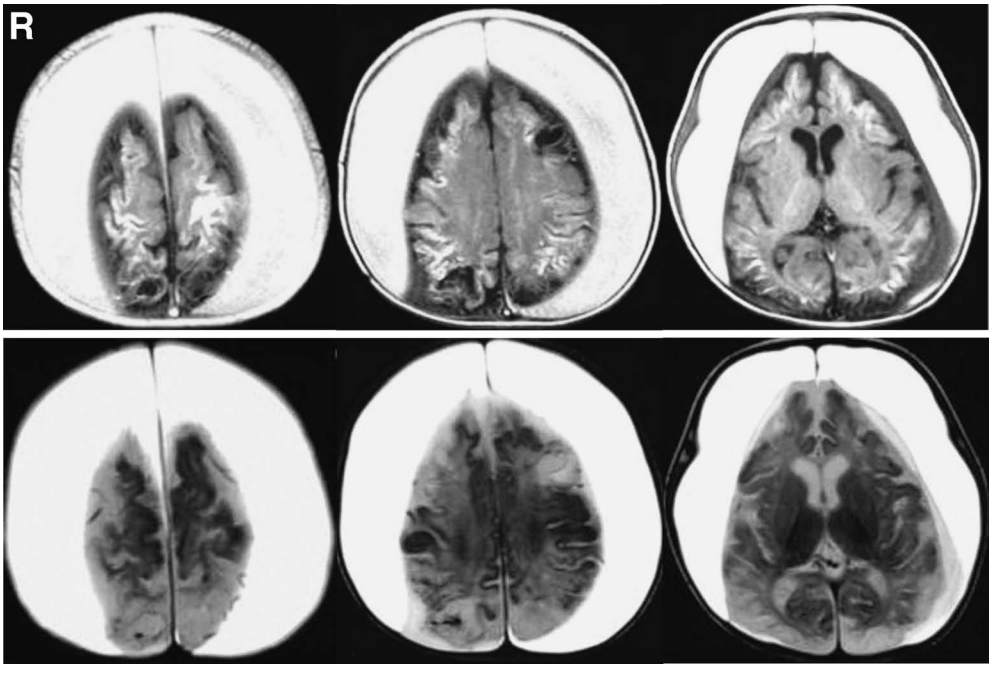

Fig. 3 **T_1- (*upper row*) and T_2-weighted (*lower row*) magnetic resonance images on day 26 revealing diffuse linear hyperintense lesions in the bilateral cerebral hemispheres, suggesting cortical laminar necrosis.**

important for two aspects: the etiology of diffuse cortical laminar necrosis originating in critically elevated ICP, and the occurrence in an infant.

Cortical laminar necrosis presents as a band of necrosis in the cortex at histological examination, and is a specific pattern of ischemic injury to the neurons in the cortex.[1,2,7,16,17] Within the cortex, the third layer is the most vulnerable, followed by the fifth and sixth, with the second and fourth being relatively more resistant to ischemic necrosis from hypoperfusion.[5]

Cortical laminar necrosis appears as hyperintense lesions with a laminar pattern on T_1-weighted MR imaging, without signs of hemorrhage or calcification on T_2*-weighted MR imaging or CT.[4,6,9,12,13] Histological examination shows neuronal damage and reactive tissue change with reactive gliosis and fat-laden macrophages resulting in protein degradation. High concentrations of proteins or other macromolecules enhance reactivity by restricting the motion of water molecules, and thus cause T_1 shortening.[4,6,9,12,13] In our case, T_1-weighted MR imaging showed linear hyperintensity (Fig. 3), and CT detected no hemorrhagic changes (Fig. 2), consistent with the characteristics of cortical laminar necrosis.

Cortical laminar necrosis results from a global rather than a focal ischemic insult to the cerebrum and implies either a profound loss of cerebral blood supply or hypoxia despite adequate perfusion, most commonly due to cardiac or respiratory arrest. The sequelae are often devastating and severe neurological deficits, including severe memory loss, personality changes, persistent vegetative state, or death.[8] Other causes include hypoxia resulting from pulmonary disease, carbon monoxide poisoning, hypoglycemia, overdose, toxins, strangulation, near drawing, status epilepticus, electrocution,[8] central nervous system lupus erythematosus,[10] immunosuppressive therapy,[3] and brain infarcts.[12,13,16] In the present case, ICP was elevated up to 30 cmH_2O (normal range 2.0–8.2 cmH_2O for infants) due to bilateral subdural hematomas, which resulted in critical decrease in cerebral perfusion pressure and eventually led to severe hypoperfusion in the cerebral hemispheres. Surgical intervention successfully controlled the patient's ICP but the chronic hypoperfusion, persisting for 1 week since the onset of the symptoms, resulted in diffuse cortical laminar necrosis. Cortical laminar necrosis is a classic entity in adulthood related to the conditions of energy depletions,[11] so the present pediatric presentation is very unusual.

Recently there are two studies have demonstrated that diffusion-weighted MR imaging can be useful in detecting the early signs of cortical laminar necrosis.[14,15] Diffuse hyperintense lesions were also detected by our initial MR imaging studies, which resulted in the diagnosis of cortical laminar necrosis. Early diffusion-weighted MR imaging can help to identify the extent of lesions in patients with cortical laminar necrosis.

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