

Case Report

Cortical Laminar Necrosis in an Infant with Severe Traumatic Brain Injury

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Cortical laminar necrosis appears as hyperintense lesions with a laminar pattern on T1 weighted magnetic resonance (MR) imaging, without signs of hemorrhage or calcification on T2 weighted MR imaging or computed tomography. It has been reported to be associated with hypoxia, metabolic disturbances, drugs, and infections. We present a 12 month-old male infant who suffered diffuse brain injuries following car accident and showed laminar necrosis of cortex.

Key Words : Cortical laminar necrosis · Hypoxia · Infant.

INTRODUCTION

Cortical laminar necrosis (CLN) is defined as the breaking down of a definite cell layer, especially in layers 3 and 5, in the cerebral cortex¹⁾, which seen as laminar high-signal on T1-weighted images, was described in Sawada et al.¹⁰⁾ and by Nabatame et al.⁷⁾, and was thought to indicate the presence of methemoglobin, but pathological study revealed no hemorrhage¹¹⁾.

High intensity cortical lesions are observed on T1-weighted magnetic resonance image (MRI) in brain infarcts^{7,10)}, which can be CLN, hemorrhagic infarcts or a combination of the two¹¹⁾. Though the imaging pattern of CLN in conventional imaging is the same for both entities, the pathophysiological mechanisms leading to its formation are entirely different. Infarction results from occlusion of intracranial artery whereas hypoxic insult causes selective neuronal loss affecting the most vulnerable regions of brain^{3,5)}. CLN represents to neuronal ischemia accompanied by gliosis and layered deposition of fat-laden macrophages¹¹⁾.

CLN has been identified in conditions with energy depletion and metabolic changes, including hypoxia, epilepsy, hypoglycemia, hyponatremia, and mitochondrial disorders⁹⁾.

Here, we report a 12 month-old male infant who suffered diffuse brain injuries following car accident and showed CLN.

CASE REPORT

An unconsciousness 12-month-old male infant was brought into the emergency room after car accident. On arrival, the patient's physical condition was unremarkable except for his neurological examination. Neurological examination revealed that pupils were equal (4 mm/4 mm) but not reactive to light and all extremities showed extension to nail bed pressure. Initial brain computed tomography (CT) showed severe brain swelling, intraventricular hemorrhage (IVH) and left parietal bone fracture (Fig. 1). His intracranial pressure (ICP) was expected to be critically high, so we performed external ventricular drainage on right Kocher's point. The initial pressure was 25 cm H₂O and serous, slightly bloody fluid was drained. During the supportive medical management in ICU, there was no event and the pa-

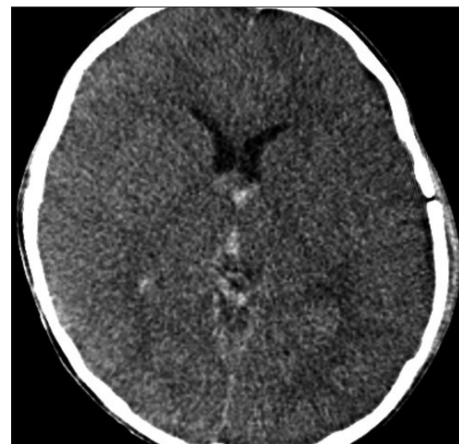


Fig. 1. Initial brain CT shows severe brain swelling, intraventricular hemorrhage and left parietal bone fracture. CT : computed tomography.

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tient's mental and neurological changes were not noticed. But his ICP gradually decreased and follow-up brain CT showed absorption of IVH. The drainage tube was withdrawn 11 days after the surgery. A brain MRI on hospital day 14 revealed diffuse linear hyperintense lesions in the cortex of bilateral cerebral hemispheres on T1-weighted, fluid attenuated inversion recovery (FLAIR) and diffusion-weighted images, which suggested CLN (Fig. 2). MR angiography of brain showed no abnormalities (Fig. 3). Despite persistent medical and rehabilitation management, his neurological deficits still persisted for 9 months. Last follow-up brain CT showed marked diffused brain atrophy in the bilateral cerebral hemispheres and ventricular dilatation (Fig. 4).

DISCUSSION

In the cerebral global hypoxic conditions, cerebral cortex is the most commonly affected region and the white matter is completely or relatively spared. This observation can be explained by the fact that the white matter is metabolically less active than cortex²⁾. The third of the six cortical layers is the most vulnerable, the fifth and sixth are somewhat less so. The second and fourth are more resistant^{1,11)}. The distribution of ischemic lesions of brain can also be explained by the selective vulnerability of different regions²⁾.

CLN appears as hyperintense lesions with a laminar pattern on T1 weighted MR imaging, without signs of hemorrhage or calcification on T2 weighted MR imaging or CT¹⁾. Pan-necrosis is found with death of neurons, glia and blood vessels. This may result in protein degradation and accumulation of lipids in macrophages⁶⁾. Although the mechanism of T1 shortening in cortical laminar necrosis remains unclear, high concentrations of proteins or other macromolecules enhance relaxivity by restricting the motion of water molecules, thus causing T1 shortening¹¹⁾. Lövblad et al.⁶⁾ have demonstrated that diffusion-weighted MR imaging can be useful in detecting the early signs of CLN also.

This signal change on MRI is commonly associated with CLN caused by different types of insults⁹⁾. Hypoxia causes selective destruction of limited number of neurons with preservation of astrocytes, microvessels and some of the neurons. In chronic stage, tissue shows volume loss and atrophy with no cavitations and gross brain structure is preserved⁵⁾.

CLN may remain for 1.5-2 years⁸⁾. Kashihara et al.⁴⁾ reported that CLN was observed in a patient with central nervous system lupus erythematosus for 5 years.

CONCLUSION

We have described an infant who suffered brain injuries fol-

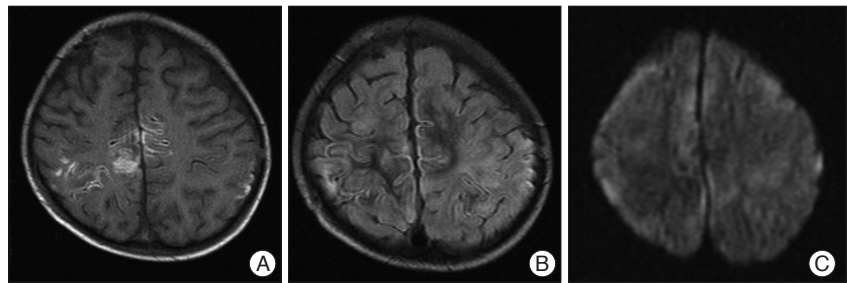


Fig. 2. T1-weighted (A), FLAIR (B) and diffusion-weighted (C) axial MRI on day 14 show linear high signal intensity selectively along the cortical regions of bilateral hemisphere. FLAIR : fluid attenuated inversion recovery, MRI : magnetic resonance image.

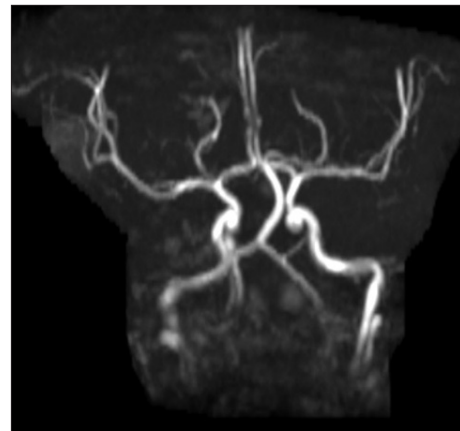


Fig. 3. MR angiography of the brain shows no abnormalities. MR : magnetic resonance.

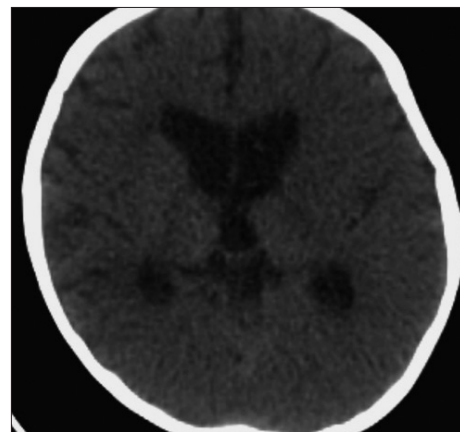


Fig. 4. Follow-up brain CT demonstrates diffused brain atrophy in the bilateral cerebral hemispheres and enlarged ventricle. CT : computed tomography.

lowing a car accident suggestive of hypoxic brain injury. It is possible that patients who display CLN acute phase after traumatic injury have more risk of developing permanent brain damage. But, further studies are needed to validate the clinical correlation of the CLN in subject with traumatic brain injury.

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