

Clinical Image

Cortical Laminar Necrosis: Imaging Features and Etiological Orientation

Nourrelhouda Bahlouli, MD^{*}; Fatima Chait, MD; Nazik Allali, PhD; Siham El Haddad, PhD; Latifa Chat, PhD
Pediatric Teaching Hospital, Department of Radiology,
Mohammed V University, Rabat, Morocco

***Corresponding author: Nourrelhouda Bahlouli**

Pediatric Teaching Hospital, Department of Radiology,
Mohammed V University, Rabat, Morocco.
Email: nourrelhoudabahlouli14@gmail.com

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Cortical laminar necrosis corresponds to neuronal ischemic remodeling with gliosis and accumulation of lipid-rich macrophages secondary to various etiologies: hypoxia, prolonged arterial hypotension, hypoglycemia, status epilepticus or arrest. Diffusion and T2 (FLAIR) show cortical and basal ganglia hypersignal from the first 24 hours. T1 shows cerebral edema during the first two weeks and a characteristic spontaneous cortical and basal ganglia hypersignal delayed and transient (3rd week). On T2*, the cortical lesions do not show any asignal, which makes it possible to differentiate them from a hemorrhagic transformation. Cortical contrast can be observed from the first weeks and for several months (up to 9 months in case of focal ischemia) and translates a rupture of the blood-brain barrier. In CT, hypodense and hyperdense gyriform density abnormalities are seen depending on the time.

Differential diagnoses can be made with: Sturge-Weber-Krabbe syndrome, encephalitis, PRES, arteriovenous malformation.

Cerebral cortical laminar necrosis might be another clue for predicting the poor prognosis in an individual with neuronal aggression.

