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Febrile Convulsion in Childhood**

M.P. Menezes, T. Nowland and E. Onikul

AJNR Am J Neuroradiol 2013, 34 (4) E43-E44

doi: <https://doi.org/10.3174/ajnr.A3527>

<http://www.ajnr.org/content/34/4/E43>

This information is current as
of July 29, 2025.

Diffusion-Weighted Imaging Changes Caused by Acute Hypoglycemia and Prolonged Febrile Convulsion in Childhood

We read with interest the article by Jokhura et al¹ on diffusion-weighted imaging in acute hypoglycemia in adults. Although there is now an increasing amount of literature on the MR imaging changes and outcome in adults with hypoglycemia, there is a paucity of literature that details imaging abnormality with acute hypoglycemia in childhood beyond the neonatal period. We describe the early progression of DWI abnormalities in a child with acute hypoglycemia and prolonged febrile seizure, and we document the poor outcome that is associated with diffuse hemispheric white matter DWI change.

A 4-year-old girl with congenital adrenal hyperplasia presented after a prolonged right focal seizure. She had been previously well with normal intelligence and had previous episodes of hypoglycemia with illness, but none that resulted in a seizure or encephalopathy. She had a fever and vomiting in the 24 hours before presentation. The seizure commenced with right arm

twitching that secondarily generalized, despite intramuscular hydrocortisone administered by the mother. The blood glucose level was measured with a hand-held glucometer while the patient was being transported by ambulance and showed a reading of “low.” She was given intramuscular glucagon and had a repeat blood glucose level of 12.5 mmol/L and a temperature of 40.5°C 25 minutes after seizure onset. The seizure lasted for 45 minutes and was terminated with a midazolam infusion and intravenous phenytoin. Subsequent investigations including extensive biochemical and serologic investigations were normal.

The first MR imaging was performed at 28 hours after admission. At that time, she had a Glasgow Coma Scale score of 8, intact brain stem reflexes, brisk reflexes, upgoing plantars, and no decorticate or decerebrate posturing. The MR imaging (Fig 1A, –B) showed diffusion restriction in the cortex with sparing of the temporal and most of the occipital lobes, with corresponding T2 hyperintensity. There was also diffusion restriction in the left hippocampus.

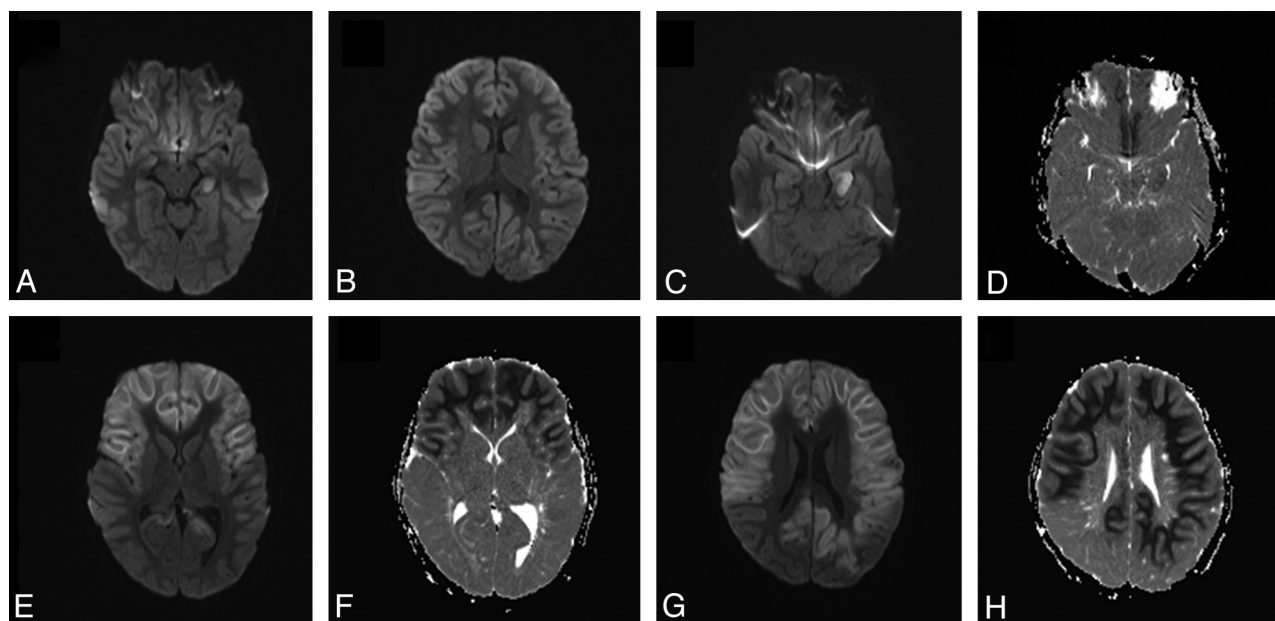


FIG 1. MR imaging changes at 28 hours (A, B) and 50 hours (C–H) after admission. At 28 hours, there is restricted diffusion in the left medial temporal lobe (A) and in the frontal, parietal, and parts of the occipital cortex (B). At 50 hours, there is prominent hippocampal (C), frontal, and parietal cortical and subcortical white matter (E, G) diffusion restriction with corresponding changes on the ADC map (D, F, H).

The MR imaging was repeated at 50 hours after admission because of right focal face seizures and deterioration in responsiveness. This showed prominent diffusion restriction and ADC map changes involving both the cortex and subcortical white matter of the frontal and parietal lobes bilaterally, and the left hippocampus, with prominent corresponding T2 hyperintensity (Fig 1C–H). She improved slowly, and, on discharge 48 days after admission, she was significantly disabled with limited functional use of her upper limbs, nonambulatory, nonverbal, and fed through a nasogastric tube.

There were some differences in the early DWI in our child when compared with imaging at presentation in adults, as reported by Jokhura et al. Imaging at 28 hours in our patient showed diffusion restriction only in the cortex, though there was prominent subcortical white matter involvement on DWI at 50 hours. The basal ganglia, internal capsule, centrum semiovale, and corpus callosum remained uninvolved. The diffusion restriction primarily involved the frontal and parietal lobes. This predominantly cortical involvement and predilection for the frontal and parietal cortex has been previously reported in adults and differs from the predominantly occipital involvement reported with neonatal hypoglycemia.^{2,3} As reported in adults, diffuse hemispheric cortical and white matter change was predictive of a poor outcome in our patient.^{1,2} The acute diffusion restriction seen in the left hippocampus is consistent with hippocampal edema after a prolonged febrile convulsion. Prolonged febrile convulsion in childhood is associated with unilateral diffusion restriction in the hippocampus contralateral to the focal seizure and increase in T2 relaxation time on MR imaging performed early after the convulsion, with resolution on follow-up imaging, indicating that these changes probably are caused by transient hippocampal edema.^{4,5}

Prolonged ictal activity causes increased glucose requirements

in the neuronal and glial cells, which, in combination with the glucose deprivation, causes severe energy failure, resulting in cytotoxic edema and restricted diffusion on DWI imaging. It is likely that the combination of hypoglycemia and prolonged seizure in our patient caused severe cytotoxic edema and neuronal injury in the hemispheric neuronal cells and was responsible for severe injury and poor outcome in this case. Diffuse hemispheric white matter change on DWI should raise the possibility of acute hypoglycemia in those with unexplained encephalopathy. In conclusion, our case demonstrates the early DWI changes after acute hypoglycemia in childhood encephalopathy. The combination of acute hypoglycemia and prolonged seizures lead to a poor long-term outcome.

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M.P. Menezes

Institute for Neuroscience and Muscle Research

T. Nowland

E. Onikul

Department of Radiology

The Children's Hospital at Westmead

Sydney, New South Wales, Australia