

Postictal Todd's Paralysis Associated with Focal Cerebral Hypoperfusion on Magnetic Resonance Perfusion Studies

Hussam A. Yacoub, DO, MS, Nathan Fenstermacher, PA, and John Castaldo, MD

Department of Medicine, Division of Neurology, Lehigh Valley Health Network, Allentown, PA

Abstract

Background—The exact underlying physiology of postictal motor deficits, known as Todd's paralysis, is not well understood and its vascular perfusion physiology is not well studied. Reversible postictal perfusion abnormalities have been sparsely described in the literature.

Methods—We report abnormal brain magnetic resonance perfusion maps in a 9-year-old boy who presented with postictal left hemiparesis. This case correlates postictal hemispheric cerebral hypoperfusion with clinical evidence of Todd's paralysis.

Conclusions—Our case provides an insight into the potential pathophysiology mechanism underlying Todd's paralysis and the practicality of magnetic resonance perfusion studies in localizing an epileptogenic zone in the postictal patient.

Keywords

focal cerebral hypoperfusion; magnetic resonance; postictal Todd's paralysis

Case Report

A right-handed, 9-year-old boy with juvenile-onset diabetes was brought to our pediatric emergency department by his grandmother for the acute onset of stroke-like symptoms. The boy had awoken that day with newly discovered left hemiparesis unassociated with confusion, dysarthria, or aphasia. Immediate finger stick revealed a glucose level of 78 mg/dL, and the left hemiparesis did not improve with sugar ingestion. A code stroke was called for emergency neurological intervention with the potential use of thrombolytics.

Further history revealed that the boy had three spells prior to this presentation that were clinically suspicious for complex partial seizures. All events were witnessed by family and described as word-salad aphasia followed by confusion lasting for approximately 2 minutes. He was evaluated by a pediatric neurologist, and the diagnosis of seizures was entertained, but no antiepileptic medications were prescribed. An electroencephalogram (EEG) revealed left hemispheric slowing but no epileptiform activity. After 2 weeks, during follow-up, 48-hour video EEG monitoring and magnetic resonance imaging (MRI) of the brain were normal. The past medical his-

tory included insulin-dependent juvenile-onset diabetes mellitus and attention deficit disorder. Medications used included insulin, methylphenidate, and risperidone.

On initial evaluation, the patient was afebrile with a blood pressure of 112/84, pulse of 114 beat per minute, and 100% oxygen saturation on room air. He was awake, alert, and oriented to name, age, and place. He had the mentation, intellect, and judgment of a nine-year-old child. Pupils were equal and reactive to light and extraocular muscles were intact. Funduscopic and visual field examination were unremarkable. No facial weakness was appreciated. Motor examination was normal except for a mild left arm and leg hemiparesis rated at 5/5. Muscle bulk and tone were normal as were the sensory, coordination, and gait examinations.

Laboratory studies revealed a WBC of 4,400/ μ L, platelets 302,000/ μ L, sodium 142 mEq/L, potassium 4.7 mEq/L, glucose 105 mg/dL, and serum creatinine 0.5 mg/dL.

Magnetic resonance imaging (MRI) of the brain showed no areas of restricted diffusion. There was evidence of mild cortical edema in the right frontal region involving the middle and anterior cerebral arteries vascular terri-

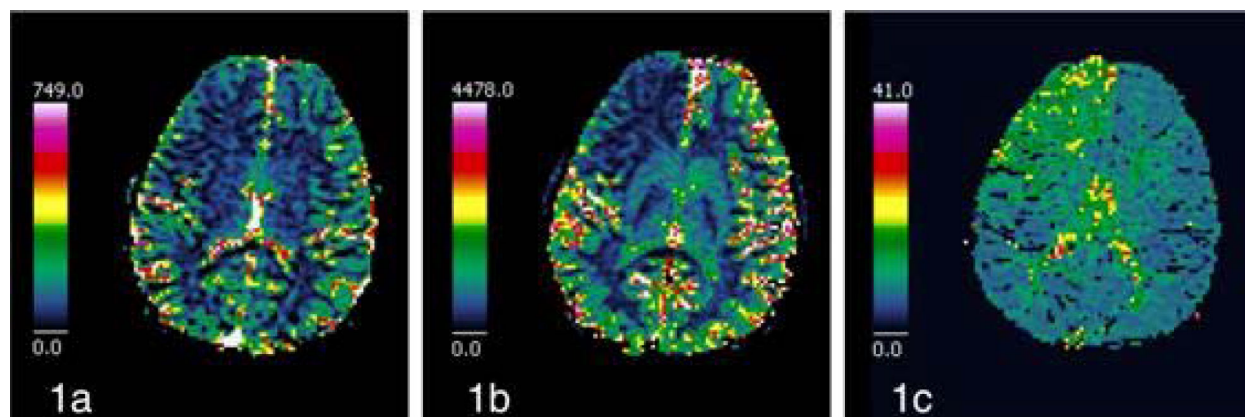


Figure 1. Magnetic resonance perfusion maps demonstrated decreased relative cerebral blood volume (a) and flow (b) with a corresponding increase in the mean transient time (c) in the right frontal region involving the middle and anterior cerebral arteries vascular territory.

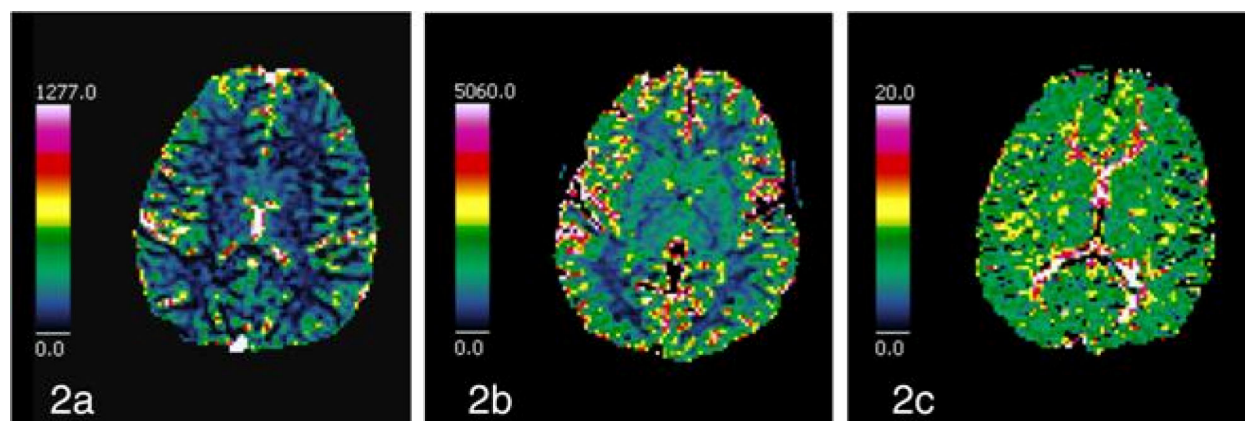


Figure 2. Magnetic resonance perfusion maps obtained 24 hours after the initial study demonstrated symmetrical relative cerebral blood volume (a) and flow (b), and mean transient time (c) in the right frontal region involving the middle and anterior cerebral arteries vascular territory.

tory (images not shown). The conventional images demonstrated a very subtle T1 hypointensity and T2 hyperintensity in the right frontal cortex but no abnormal enhancement. A magnetic resonance angiogram (MRA) of the head showed diminution in the caliber of the distal right internal carotid artery and decreased flow in the distal branches of the right middle and anterior cerebral arteries (images not shown), but an immediate follow-up percutaneous 4-vessel cerebral arteriography was unremarkable. Magnetic resonance perfusion (MRP) maps demonstrated abnormally decreased regional cerebral blood volume (rCBV) and flow (rCBF) with a corresponding increase in the mean transient time (MTT) in the right frontal lobe involving the middle and anterior cerebral arteries vascular territory (Fig. 1).

An EEG showed focal delta slowing over the right frontal region. The patient's left hemiparesis improved to normal within 2 hours of presentation. A follow-up MRP completed approximately 24 hours from the initial presentation showed total resolution of the abnormal perfusion maps (Fig. 2) and susceptibility in the right frontal sulci.

The patient was started on levetiracetam and discharged home in a stable condition.

DISCUSSION

Postictal motor deficits, known as Todd's paralysis, have been described as early as the 1800s, [1] but its exact underlying pathophysiology is still not well understood

and vascular perfusion physiology is not well studied. Reversible postictal perfusion abnormalities have been sparsely described in the literature. Most case reports of reversible MRI findings cite increase in T2-weighted signals, increased rCBV, decreased apparent diffusion coefficient (ADC) mapping, coupled with hyperperfusion in the postictal state. Hassan et al. [2] reported two cases of postictal paresis associated with computed tomography perfusion evidence of increased cerebral blood flow and volume and decreased mean transient time in the vascular territory corresponding to patient's symptoms. These changes are believed to be in response to hypermetabolic neuronal activity from seizure discharges and can be differentiated from ischemia by the finding of increased perfusion. [3–5] Hypoperfusion has rarely been reported and when seen has occurred later in the postictal phase and after an initial period of hyperperfusion. [6,7]

Mathews et al. [8] described a similar case of an adult patient who presented with aphasia and right-sided hemiparesis with abnormal computed tomography perfusion studies, revealing a dramatic reduction in rCBF and rCBV but relative symmetry of MTT. A follow-up MRP study revealed normalization of cerebral blood flow. Leonhardt et al. [6] assessed regional, dynamic, interictal, and postictal MRP changes in patients with temporal lobe epilepsy and documented relative hyperperfusion in the interictal period followed by hypoperfusion postictally. The study, however, did not assess changes in the MTT.

The clinical presentation of our patient and the MRA findings of decreased flow in the right anterior circulation were consistent with evolving ischemia/oligemia of the right frontal lobe. A congenital chronic arteriopathy with oligemia of the frontal lobe was initially suspected. The subsequent completely normal cerebral arteriogram coupled with the reversal to normal of the follow-up MRP study supported the diagnosis of a seizure with secondary reversible brain perfusion abnormalities.

To our knowledge, our case is the first reported with postictal reversible MRP maps involving cerebral blood volume, cerebral blood flow, and mean transient time, which could be misinterpreted for acute evolving ischemic infarction.

MTT is a sensitive indicator of acute cerebral ischemia secondary to large vessel stenosis or occlusion. Our case

is distinctive because a marked increase in the mean transient time did not correlate with a large vessel stenosis. We therefore advise caution when interpreting MRP parameters when the clinical presentation is most consistent with a seizure and a postictal state. A decrease in cerebral blood flow and volume, along with an increase in MTT, is a pattern that typically denotes cerebral infarct rather than a metabolic disturbance or a seizure. Careful interpretation and correlation of this data with the clinical presentation, EEG findings, previous history, and lack of vascular risk factors may avoid unnecessarily revascularization procedures in the postictal patient. Our case further provides an insight of the potential pathophysiology mechanism underlying Todd's paralysis and the practicality of MRP studies in localizing an epileptogenic zone in the postictal patient.

References

1. Todd RB. On the pathology and treatment of convulsive disease. *London Med Gazette* 1849;8:661–671. 724–729, 766–772, 815–822, 837–846.
2. Hassan AE, Cu SR, Rodriguez GJ, Qureshi AI. Regional cerebral hyperperfusion associated with postictal paresis. *J Vasc Interv Neurol* 2012;5:40–42.
3. Yaffe K, Ferriero D, Barkovich J, Rowley H. Reversible MRI abnormalities following seizures. *Neurology* 1995;45:104–108.<http://www.neurology.org/content/45/1/104.short>
4. Cartagena A, Young G, Lee d, Mirsattari S. 10.1212/WNL.78.1.MeetingAbstracts.P03.116*Neurology* 2012;78(Meeting Abstracts 1):P03–116.
5. Flacke S, Wullner U, Keller E, Hamzei F, Urbach H. Reversible changes in echo planar perfusion and diffusion weighted MRI in status epilepticus. *Neuroradiology* 2000;42:92–95.<http://link.springer.com/content/pdf/10.1007%2Fs002340050021.pdf#page-1>
6. Leonhardt G, Greiff A, Weber J, Ludwig T, Wiedemayer H, Forsting M, Hufnagel. Brain perfusion following single seizures. *Epilepsia* 2005;46:1943–1949.<http://onlinelibrary.wiley.com/doi/10.1111/j.1528-1167.2005.00336.x/full>
7. Szabo K, Poepel A, Pohlmann-Eden B, Hirsch J, Back T, Sedlaczek O, Hennerici M, Gass A. Diffusion-weighted and perfusion MRI demonstrates parenchymal changes in complex partial status epilepticus. *Brain* 2005;128:1369–1376.<http://brain.oxfordjournals.org/content/128/6/1369.short>
8. Mathews MS, Smith WS, Wintermark M, Dillon W, Binder D. Local cortical hypoperfusion imaged with CT perfusion during postictal Todd's paresis. *Neuroradiol* 2008;50:397–401.http://www.bestcenteroc.com/files/5813/0800/5076/Todd_paralysis_case_report.pdf