

# Postictal Aphasia with Transient Sulcal Hyperintensity on MRI

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A 38-year-old man had postictal global aphasia after generalized tonic-clonic seizure, but recovered within 1 day. There was a focal increase in the signal intensity of the left frontoparietal sulci on fluid-attenuated inversion recovery magnetic resonance (MR) sequence 6 hours after the seizure, which disappeared 1 month later. The transient seizure-induced MR hyperintensity was possibly caused by ictal or postictal hyperemia and breakdown of the blood-brain barrier. This report aims to increase awareness of this condition and avoid unnecessary management. [*J Chin Med Assoc* 2006;69(10): 499–502]

**Key Words:** electroencephalography, fluid-attenuated inversion recovery, magnetic resonance imaging, postictal aphasia, seizure

## Introduction

It is not unusual to have transient aphasia after a seizure attack (postictal aphasia), which is thought to be caused by postictal inactivation of any of the 3 language areas (basal temporal, Wernicke's, or Broca's language area), via a mechanism similar to that of Todd's paralysis.<sup>1</sup> Postictal transient magnetic resonance (MR) enhancement in cortical regions has been reported in cases of complex partial seizures, which is consistent with ictal or postictal hyperemia and breakdown of the blood-brain barrier.<sup>2</sup>

We present a case of reversible and focal increase in signal intensity on fluid-attenuated inversion recovery (FLAIR) MR sequence over the left frontoparietal sulci, during postictal global aphasia after generalized tonic-clonic seizure. The pathogenesis is discussed.

## Case Report

A 38-year-old right-handed man was admitted due to generalized tonic-clonic seizure that lasted for 3 minutes. He was delivered spontaneously at full term via vagina without birth asphyxia. His developmental

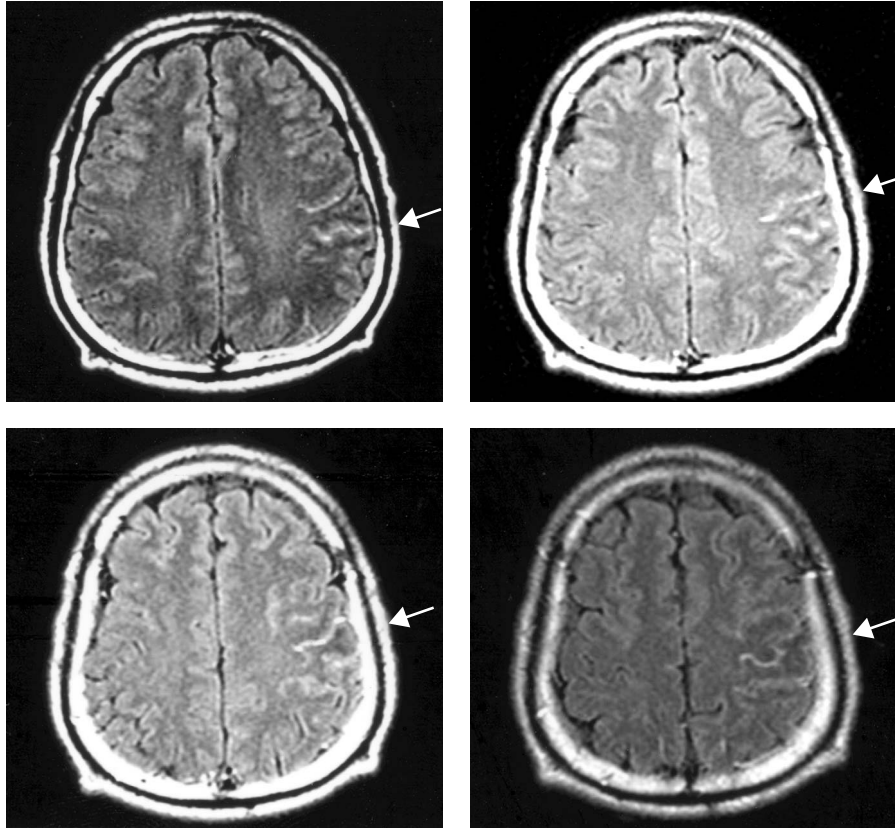
history was normal. He had never had epilepsy, major head trauma or central nervous system infection.

After the seizure, the patient had postictal confusion for 4 hours. After regaining consciousness, he was found to have global aphasia with language difficulty in comprehension, fluency and repetition. No focal weakness was noted. Brain computed tomography (CT) was normal. Brain MR imaging was done 6 hours after the seizure. The FLAIR image revealed focal hyperintensity over the left central and peri-Rolandic sulci (Figure 1). Electroencephalography (EEG) in the 8<sup>th</sup> hour post-seizure showed a sharp wave over the left frontocentral regions (F3, C3), maximal at frontal and intermittent slow activities at 2–3 Hz over the left frontotemporal region (Figure 2). Blood routine, serum biochemistry and cerebrospinal fluid (CSF) studies, including herpes simplex virus titer (HSV IgM, HSV IgG, HSV2 IgG) were normal. Four-vessel cerebral angiography performed 3 days after the seizure was also normal.

The patient's aphasia gradually recovered within 24 hours. One month later, brain MR imaging was repeated, which showed that the previous left parietal cortical abnormality had disappeared completely (Figure 3).

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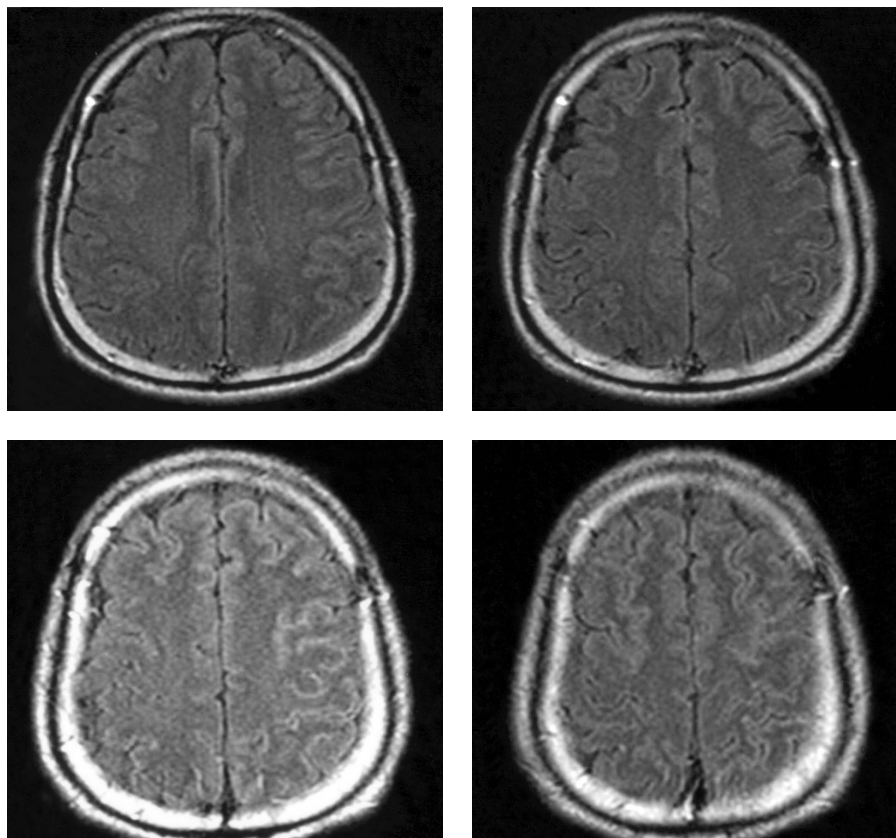
E-mail: epilepsy2000@gmail.com • Received: November 30, 2005 • Accepted: August 7, 2006



**Figure 1.** Fluid-attenuated inversion recovery magnetic resonance sequence shows focal hyperintensity over the left central and peri-Rolandic sulci (arrows).



**Figure 2.** Electroencephalography shows a medium to high voltage sharp wave over the left frontocentral region (F3, C3), higher at frontal (arrow) and also high-voltage intermittent slow activities at 2–3 Hz lasting for 1 second over the left frontotemporal region (arrowhead).



**Figure 3.** Fluid-attenuated inversion recovery magnetic resonance sequence 1 month after the seizure is normal.

## Discussion

Our case is fully consistent with the diagnosis of postictal aphasia with transient MR hyperintensity. Penfield<sup>3</sup> reported an epileptic patient during surgery with focal, postictal hyperperfusion, documented by angiography.<sup>4</sup> Epileptic seizures associated with increased cerebral blood flow and metabolism were disclosed in both experimental studies in animals and clinical studies in humans.<sup>5-7</sup> Horowitz et al<sup>2</sup> reported a case of complex partial seizure-induced transient MR hyperintensity in FLAIR sequence, possibly caused by ictal or postictal hyperemia and breakdown of the blood-brain barrier.

The FLAIR technique is widely applied in contemporary workup for epilepsy surgery.<sup>8</sup> Signal changes on T2-weighted images associated with some forms of cortical lesions are usually evident on standard imaging. However, T2-weighted images may be degraded by CSF, obscuring signal change in superficial structures. Use of the FLAIR sequence may overcome these problems, enhancing the visualization of signal change associated with small superficial lesions.<sup>9</sup> This may explain why the lesion (left parietal superficial cortex) in our case was enhanced in the FLAIR sequence only.

Postictal aphasia is thought to be caused by postictal inactivation of any of the 3 language areas (basal temporal, Wernicke's, or Broca's language area), via a mechanism similar to that of Todd's paralysis.<sup>1</sup> Jackson<sup>10</sup> hypothesized that cortical deactivation secondary to neuronal exhaustion was the most likely mechanism underlying these deficits. However, Gowers<sup>11</sup> argued that they are caused by cortical inhibition.

Several studies have evaluated the lateralizing significance of ictal and postictal speech manifestations. All of them showed postictal language deficits to be more frequent and more specific for dominant temporal lobe epilepsy than for ictal speech manifestation.<sup>12-15</sup> However, without invasive monitoring to better define the epileptogenic zones and their proximity to language centers, or resection to prove the suspected location of the epileptogenic zone, the findings of ictal or postictal speech manifestation could still be caused by seizure activity spreading from a remote focus outside the speech area. This may explain why the epileptiform discharge in our case was over the left frontocentral region on EEG, which was distant from the parietal region on MR imaging.

Based on the postictal speech deficit, MR and EEG findings, we presumed that our patient suffered from

a secondary generalized partial seizure with an epileptogenic zone over the dominant (left) hemisphere. His postictal aphasia was caused by seizure activity spreading simultaneously from the left frontocentral region to Broca's area in the left frontal region and Wernicke's area in the left parietal region. To conclude, postictal transient MR hyperintensity may be found occasionally and should not be erroneously attributed to leptomeningoencephalitis, infarction, or tumor.

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