INTRODUCTION

Postictal neurologic deficit is a well-known complication mimicking the manifestation of a stroke. We present a case of a patient with clinical evidence of Todd’s paralysis correlating with reversible postictal parenchymal changes on perfusion CT and magnetic resonance (MR) imaging. In this case, perfusion CT and MR imaging were helpful in the differential diagnosis of stroke-mimicking conditions.

CASE REPORT

A 72-year-old male was presented with sudden-onset right extremity weakness after clonic seizure. He had a history of nasopharyngeal cancer and his overall health condition was poor. He was alert and was able to follow commands. At initial neurologic examination, motor grade was III in the extremity of the right limb.

He underwent brain CT angiography (CTA), perfusion CT and magnetic resonance (MR) imaging including diffusion-weighted image (DWI) immediately after admission. On MR imaging (3T system, Achieva; Philips Medical Systems, Best, the Netherlands), an axial DWI showed subtle cortical hyperintensity in the left parietal lobe with restricted diffusion involving mainly the cortical areas of the left parietal lobe with sparing white matter. Mean transition time (MTT), a sensitive indicator of acute cerebral ischemia, was relatively symmetric in
Regional Cortical Hyperperfusion on Perfusion CT during Postictal Motor Deficit

configuration (Fig. 1C-E). CTA source images demonstrated slightly abundant vessels in the corresponding region with normal-appearing major intracranial arteries (Fig. 1F, G). Such findings suggested that acute neurologic deficit in this patient was unlikely to have been secondary to a vascular occlusive event. An electroencephalography was performed on the next day, and it demonstrated a diffuse left-hemispheric dysfunction without a definite epileptiform discharge.

However, a follow-up MR imaging on the same day revealed the disappearance of the preexisting diffusion abnormality (Fig. 1H). In addition, perfusion CT demonstrated normalization of CBF and CBV in the left parietal lobe (Fig. 1I, J).

The patient had no further seizures, and symptoms of the right hemiparesis gradually improved from grade III to IV over the next several days. Then, he was transferred to the medical department in order to treat acute gangrenous cholecystitis; no further neuroimaging was performed.

DISCUSSION

It is estimated that between 5 to 30% of cases identified as “brain attacks” are, in fact, due to stroke-mimicking conditions (3, 4). Stroke mimics include diagnosis such as complex migraine, infectious condition, metabolic disorder, intracranial tumor, epilepsy, Todd’s paralysis and psychiatric illness, such as conversion disorder (3, 4). Despite the fact that Todd’s paralysis is a well-known entity for postictal motor deficit, the physiologic explanation for it has not been established yet (2). Many mechanisms may account for the postictal state, including neurotransmitter depletion, neuronal desensitization, altered local cerebral blood flow and various forms of active inhibition, for which the utility of various imaging modalities have been investigated in

![Fig. 1. A 72-year-old male with acute onset aphasia and right extremity weakness.](image)

A. Axial DWI shows subtle cortical hyperintensity in the left parietal lobe.
B. ADC reveals mild diffusion restriction.
C-E. Perfusion CT images demonstrate increased CBF (C) and CBV (D) in the left parietal cortices, but relatively normal configuration of MTT (E).
F, G. Axial CTA source images demonstrates mild increased vascular pattern.
H. Follow-up DWI on the next day shows no diffusion abnormality.
I, J. Follow-up perfusion CT at one week shows complete normalization of the preexisting hyperperfusion on CBF (I) and CBV (J) maps.

Note.—ADC = apparent diffusion coefficient, CBF = cerebral blood flow, CBV = cerebral blood volume, CTA = CT angiography, DWI = diffusion-weighted image, MTT = mean transition time
the previous studies (2, 3, 5-8). However, the results of the previous studies regarding postictal perfusion findings are still controversial. Recently, Mathews et al. (2) demonstrated postictal regional hypoperfusion on perfusion CT caused by postictal exhaustion or inhibition, whereas Masterson et al. (3) reported regional hyperperfusion with postictal neurologic deficit due to the spread of a residual ictal discharge.

In this case, our patient's clinical and neuroimaging findings were consistent with those of the postictal state. We observed regional cortical hyperperfusion showing an increase in CBF and CBV with a relative preservation of MTT on the perfusion CT. According to previous studies, it is generally accepted that an increase in CBF and CBV is found in the seizure-onset zone as well as in the cortical areas affected by the spread of ictal discharges during the course of seizure, and the presence of a cortical hyperperfusion can be a valid indicator of ongoing seizure activity (3, 7, 9, 10). Further, there was increased vascularity in the corresponding region similar to the previous study with MR angiography (10). In addition, subtle cortical hyperperfusion with lower ADC value on DWI showing normalization in the follow-up can be explained as a reversible excitotoxic brain injury mediated by prolonged seizure activity. The neuronal seizure activity increases the release of glutamate from the presynaptic terminals of neuronal axons, and excessive glutamate crosses the synaptic cleft in order to bind to the N-methyl-D-aspartate (NMDA) and non-NMDA receptors. This mechanism causes cytotoxic edema in neurons and adjacent glial cells, leading to apoptosis or selective neuronal necrosis. In this process, astrocytic response to excessive glutamate release plays an important role in tissue repair by dampening its excitotoxic effects. Therefore, cytotoxic edema in the acute phase of reactive astrocytosis is presumed to be responsible for reversible signal intensity abnormalities (6, 8).

In summary, our case provides a temporal and spatial correlation of regional cortical hyperperfusion in patients with postictal motor deficits. The transient nature of this clinical deficit correlates with the reversibility of cortical perfusion and diffusion abnormalities, and such findings can be helpful in diagnosing stroke-mimicking conditions.

REFERENCES


발작 후 운동 결손 환자에서 관찰된 관류 영상에서의 국소적 피질 과다관류 : 증례 보고

백혜진

발작 후 신경학적 결손은 뇌졸중으로 오인할 수 있는 잘 알려진 합병증이다. 저자는 토드씨 마비의 임상적 근거를 가진 환자에서 발견된 관류 컴퓨터단층촬영과 뇌 자기공명영상상의 발작 후 뇌실질의 변화가 경과 관찰 중 가역적인 변화를 보인 증례를 경험하였다. 본 증례를 통해 저자는 관류 컴퓨터단층촬영과 뇌 자기공명영상이 뇌졸중과 유사한 임상질환을 감별하는 데 도움이 될 수 있음을 보고하고자 한다.

인제대학교 의과대학 해운대백병원 영상의학과