Preferred Involvement of the Basal Ganglia After Lenticulostriate Infarction as a Possible Indicator of Different Gray and White Matter Vulnerability

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**Background and Purpose**—Symptomatic progression is frequently observed in lacunar infarcts. The exact mechanisms of this phenomenon have not yet been clarified.

**Summary of Cases**—We report 2 patients with lenticulostriate artery infarcts that presented with skip lesions that were restricted to gray matter. One of the patients subsequently developed symptomatic deterioration; the other experienced no further neurological events.

**Conclusions**—A possible mechanism of differential vulnerability to ischemia of gray and white matter is considered. White matter may have a longer therapeutic time window for neuroprotective treatment than gray matter. 


**Key Words:** cerebral ischemia ■ lacunar infarction ■ MRI ■ white matter

We report 2 patients with lenticulostriate artery (LSA) infarcts that presented with skip lesions that involved areas of gray matter separated by the unaffected white matter of the internal capsule. One of the patients subsequently developed motor function deterioration; the other experienced no further neurological events. Based on these findings, a possible mechanism that could explain the progression of symptoms is proposed.

To our knowledge, this is the first report focusing on the morphological characteristics of such skip lesions visualized by MRI. All images were obtained with a 1.5-T MRI (Gyroscan Intera; Philips Medical Systems), using thin-section DWI with a 3-mm slice thickness and no inter-slice gaps. This imaging method may have led to the more accurate characterization of these cases.1 Fiber tracking was performed by using the continuous tracking method available on Philips Research Integrated Development Environment (PRIDE) software.

**Patient 1**
A 65-year-old, right-handed man with a history of diabetes mellitus and coronary artery disease was admitted to our department because of right-sided weakness. On the morning of admission, the patient had come to the hospital for a regularly scheduled internal medical consultation. At 9:45 AM, he suddenly developed weakness in his right arm and leg while in the hospital cafeteria. The patient was referred to a neurologist at 11:05 AM, while the symptoms were resolving. His blood pressure was 154/80 mm Hg, and he had a regular pulse of 60 beats per minute. On neurological examination, power in his right face, arm, and leg was slightly reduced; there were no sensory changes. The patient was alert and had no cortical signs. The blood glucose level was 274 mg/dL. The initial MRI, done 5 hours after onset, showed DWI restriction in the left LSA territory (Figure 1). Intravenous plasma expander, ozagrel sodium (antithromboxan A2 inhibitor), and edaravone (radical scavenger) were started. Complete resolution of the symptoms occurred over the next hour. However, the next morning, the patient again developed right face, arm, and leg weakness, which progressed to a dense right hemiplegia on the third day. A second MRI, done on the fourth day, showed enlargement of the lesions, which now included the internal capsule. Magnetic resonance angiography showed a mild wall irregularity of the left LSA territory (Figure 1). Intravenous plasma expander, ozagrel sodium (antithromboxan A2 inhibitor), and edaravone (radical scavenger) were started. Complete resolution of the symptoms occurred over the next hour. However, the next morning, the patient again developed right face, arm, and leg weakness, which progressed to a dense right hemiplegia on the third day. A second MRI, done on the fourth day, showed enlargement of the lesions, which now included the internal capsule. Magnetic resonance angiography showed a mild wall irregularity of the left middle cerebral artery. There was no evidence of artery-to-artery or heart-to-artery emboli on further examinations, which included carotid sonography, echocardiography, and a Holter ECG.

**Patient 2**
A 64-year-old, left-handed woman with a history of hypertension and hypercholesterolemia developed left hand clum-
At about 5:30 AM, the symptom resolved in 30 minutes. At about 7:00 AM, she developed left arm and leg weakness with profound dysarthria. These symptoms resolved as she was waiting for the ambulance. The neurological examination done at 7:30 AM in the emergency room was normal, but, at 8:50 AM, the patient’s symptoms returned, and she developed a dense left hemiplegia involving her face, arm, and leg; there were no cortical signs. The patient’s blood pressure was 158/101 mm Hg with a regular pulse of 69 beats per minute. Intravenous argatroban and edaravone were started, and the symptoms completely resolved at about noon. The initial MRI, done 80 hours after onset, showed DWI restriction in the right LSA territory (Figure 2). There was no evidence to suggest artery-to-artery or heart-to-artery emboli on further examinations. A second MRI, done on the 13th day, showed no enlargement of the lesions. The patient developed no further neurological events. A third MRI, done 5 months after onset, showed high signal intensities on T2-weighted imaging in the same areas of DWI restriction seen on the initial MRI.

**Discussion**

Two cases that had similar imaging characteristics with skip lesions involving only gray matter of the LSA territory have been presented. One of the patients subsequently developed severe motor deficits; the other experienced no further neurological events.

A high frequency of progressive motor deficits has been reported in lacunar infarcts; however, the mechanisms of deterioration remain unclear. Terai et al reported a case with enlargement of the LSA infarct and neurological deterioration; they assumed that clot propagation resulted in the obstruction of the adjacent branches of a large perforating artery.

However, the characteristic appearance of the lesions in our patients suggests another possible explanation, namely selective vulnerability of the lesions.
vulnerability. When these patients were first seen, we wondered why such skip lesions occurred in the same vascular territory. Previous studies have shown that gray matter is more vulnerable to acute ischemia than white matter, both in animals and humans. This finding also applies to LSA territory infarcts; thus, when gray and white matter within the same arterial territory is exposed to acute ischemia, gray matter is more vulnerable to the ischemia than white matter.

Other possible explanations include: a microembolic genesis involving different LSAs for the gray and white matter tissue; different level of tissue oxygen consumption of the basal ganglia (gray matter) and the internal capsule (white matter). Further investigations involving a large number of patients using perfusion imaging techniques, such as MR perfusion imaging or nuclear medicine techniques, may be needed to determine whether gray and white matter within the LSA territory actually have a different infarct threshold.

Of note, our standard stroke protocol involves using thin-section DWI with a 3-mm slice thickness and no inter-slice gaps; this may have led to more accurate characterization of these cases. Compared with the DWI commonly used for stroke imaging, this imaging method has approximately twice the spatial resolution in the craniocaudal direction. The caudate lesions in our cases may have been so small that they might have been missed on conventional DWI. Coronal views are also thought to be advantageous in depicting such LSA infarcts.

The possibility that white matter bundles have a higher resistance to ischemia raises important clinical therapeutic issues. If white matter does indeed have a high threshold for infarct than gray matter, white matter may have a longer therapeutic time window for adequate neuroprotective treatment for acute stroke than gray matter. Thus, white matter protection should receive more attention in neuroprotection trials.

Disclosures

None.

References


Figure 2. MRI findings of patient 2, 80 hours (A–E) and 5 months after onset (F–H). A–D, DWI restriction is shown in the right LSA territory: one is located in the putamen and the other is in the caudate (white arrow). D, On coronally reconstructed DWIs, the superimposed corticospinal tracts (CSTs) depicted on tractography (purple lines) are not involved by the lesions. E, The ADC-map shows no obvious signal changes, however the ADC values are moderately decreased in the putamen (0.72). F–H, T2-weighted imaging shows high signal intensities in the same areas as the high signal intensity lesions of the first DWI (white arrow).
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