Signal Change of the Substantia Nigra on Diffusion-Weighted Imaging Following Striatal Infarction

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Abstract

Diffusion-weighted imaging can depict secondary signal changes of the substantia nigra in patients with ipsilateral striatal infarction. We report four patients who demonstrated obvious signal changes of the substantia nigra in the subacute phase of stroke. Embolic stroke was diagnosed in all of the cases, and none of the patients presented clinical deterioration in their course. Embolic mechanism might be more closely related to the secondary change of the substantia nigra than thrombosis. The relationship between secondary nigral degeneration and stroke etiology or between the nigral lesions and recanalization of the middle cerebral artery remains unclear.

Key words: embolism, secondary degeneration, substantia nigra, striatal infarction, diffusion-weighted imaging

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Introduction

Secondary degeneration of the substantia nigra and the corticospinal tract is demonstrated as a hyperintensity lesion on diffusion-weighted imaging (DWI) during the subacute phase of ipsilateral striatal infarction (1-3). Four patients with typical DWI findings, including two who underwent follow-up magnetic resonance imaging (MRI) in the chronic phase, are reported.

Case Reports

Case 1: A 42-year-old woman was admitted due to abruptly developed consciousness disturbance and mild left-sided hemiparesis. DWI showed early ischemic change of the right striatum. T2-weighted imaging (T2WI) and DWI on the 10th hospital day showed a hyperintense area in the right substantia nigra and cerebral peduncle with a decreased apparent diffusion coefficient (ADC) (Fig. 1A-1D). The abnormal intensity decreased after 3 months (Figs. 1E, 1F).

Case 2: A 71-year-old woman was admitted with severe consciousness disturbance and complete left-sided paralysis. A hyperintensity lesion was seen in the right striatum and deep white matter in the right frontal lobe on DWI. Follow-up MRI on the 10th day showed a new hyperintensity lesion in the right substantia nigra both on DWI and T2WI, accompanied with a decreased ADC (Fig. 2).

Case 3: A 71-year-old man experienced progressive left-sided hemiparesis. On admission, DWI revealed a faint hyperintensity lesion in the right striatum and insular cortex. Eleven days after onset, a new hyperintensity lesion was seen in the ipsilateral substantia nigra in addition to the index infarction (Figs. 3A, 3B). After 5 months, the abnormal intensity of the substantia nigra became normal intensity, while the adjacent cerebral peduncle showed a vague intensity change (Figs. 3C, 3D).

Case 4: A 59-year-old woman with right-sided hemiparesis was admitted. DWI demonstrated hyperintensity lesions in the left striatum and left frontal cortex and subcortex. After 6 days, a faint hyperintensity lesion was seen in the ipsilateral substantia nigra on DWI (Fig. 4). All of the patients demonstrated occlusion of the middle cerebral artery (MCA) in the lesion side on magnetic resonance angiography on admission, and demonstrated recanalization...
Figure 1. Diffusion-weighted imaging (DWI) on the 10th day after stroke onset in Case 1 (A). A hyperintense lesion is seen in the right substantia nigra and cerebral peduncle. Apparent diffusion coefficient map in color spectrum (B) reveals decreased ADC in the same lesion. T2-weighted imaging (T2WI) also shows hyperintensity in the same lesion (C), in addition to hyperintensity in the right putamen and globus pallidus (D). On follow-up MRI after one month, intensity abnormality is not seen in the midbrain on DWI (E) or T2WI (F).

Figure 2. DWI on the 10th day after stroke onset in case 2 (A) demonstrates a hyperintense lesion in the pars reticulate of the right substantia nigra with decreased ADC (B). On T2WI on the same day, an ischemic lesion in the right putamen demonstrates hemorrhagic transformation (C), and the right substantia nigra shows mild hyperintensity.
Figure 3. DWI on the 11th day after stroke onset in case 3 reveals a faint hyperintense lesion in the pars reticulate of the right substantia nigra (A); a hyperintensity region is seen in the right striatum and insular cortex on FLAIR image on the same day (B). Five months later, the intensity change has diminished on DWI (C), while a mild hyperintensity lesion is seen on the ipsilateral cerebral peduncle on FLAIR image (D).

Figure 4. DWI 6 days after onset in case 4, shows a faint high intensity signal in the left substantia nigra and cerebral peduncle (A). The left striatum has hemorrhagic infarction seen on T2WI (B).

zation on follow-up. Cardioembolic stroke was diagnosed in cases 1 and 4; an artery-to-artery embolism was suspected in case 3. Although brain embolism was suspected in case 2, no embolic source was detected. None of the patients showed progression or fluctuation of symptoms during their admission.

Discussion

Secondary degeneration of the substantia nigra is often seen over 1-4 weeks following ipsilateral striatum lesions, in particular, putaminal (2) or external capsular (4) lesions. In previous reports, the nigral change was mainly explained by a transsynaptic mechanism: the loss of an inhibitory $\gamma$-
aminobutyric acidergic (GABAergic) output from the striatum to the substantia nigra results in excessive excitation, causing the neuronal damage in the substantia nigra (1, 2).

In previous reports, a unilateral striatal lesion due to embolic stroke (5), arterial dissection (3), or external capsular hemorrhage (4) caused secondary degeneration of the substantia nigra. However, these authors did not clearly discuss the mechanism of striatal infarction, including embolism or thrombosis. We speculate that sudden, destructive damage of the striatum might be related to the mechanism, because a sudden cessation of GABAergic output would cause stronger excessive excitation of the substantia nigra rather than gradual cessation. Further reports of such cases are necessary to verify this hypothesis.

All of four index cases showed recanalization of the MCA in the lesion side. However, we suppose that recanalization of the MCA is not always necessary for the secondary degeneration of the substantia nigra, because it is not directly related to the injury of the striatum. Further study is needed to investigate the relationship between the recanalization of MCA and nigral degeneration.

A well-known corticospinal tract change in the chronic phase, Wallerian degeneration, is another possibility. Wallerian degeneration is secondary antegrade degeneration of distal axons and myelin sheaths after injury of the neuronal cell body and/or its proximal axon (6), of which mechanism fundamentally differs from nigral degeneration. DWI demonstrates the early changes of on-going Wallerian degeneration (3, 6-8). Such a signal change of the corticospinal tract adjacent to the substantia nigra may contribute to the DWI abnormality. An MRI system with higher resolution may anatomically distinguish the secondary degeneration of substantia nigra and Wallarian degeneration of the corticospinal tract.

MRI provides vast clinical information not only in the hyperacute phase, but also during the subacute phase of ischemic stroke. Secondary intensity change in the substantia nigra following ipsilateral striatal infarction on DWI is considered to be the swelling of astrocytes and neurons (3). However, it might be misdiagnosed as a recurrent stroke and might influence clinicians to initiate unnecessary antithrombotic agents. Clinicians need accurate knowledge to correctly interpret these findings.

The relationship between the secondary nigral degeneration and stroke etiology or between the nigral lesions and recanalization of the MCA corresponding to the ischemic lesion remains unclear. Further clinical research using DWI with a large population would clarify these points.

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References


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