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Case report

Bilateral parkinsonism in a patient with infarcts involving the unilateral basal ganglia

Shiroh Miura [a,*], Masayuki Ochi [a], Hirofumi Ochi [a], Michiya Igase [a], Naoto Kawaguchi [b], Masao Miyagawa [b], Yusuke Uchiyama [c], Yasumasa Ohyagi [a]

[a] Department of Neurology and Geriatric Medicine, Ehime University Graduate School of Medicine, Ehime, Japan
[b] Department of Radiology, Ehime University Graduate School of Medicine, Ehime, Japan
[c] Department of Radiology, Kurume University School of Medicine, Fukuoka, Japan

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ABSTRACT

We describe a 61-year-old woman with bilateral parkinsonism caused by unilateral infarction limited to the territory of the lenticulostriate arteries. Although dopamine transporter imaging with single-photon emission computed tomography (DaTSPECT) demonstrated reduced putaminal tracer binding concordant with the size and location of the vascular lesion, the specific binding ratio was within the normal range. Five months after onset, the patient was free from parkinsonism without the use of any antiparkinsonian agents. When patients show bilateral parkinsonism, it is important to consider infarction of the lenticulostriate arteries. Additionally, DaTSPECT might be useful for predicting the prognosis of parkinsonism caused by infarction.

1. Introduction

The acute or subacute post-stroke subtype of vascular parkinsonism is typically characterized by hemiparkinsonism related to the location of the acute or subacute ischemic or hemorrhagic lesions [1]. It is important to distinguish vascular parkinsonism from Parkinson's disease when choosing therapeutics; however, it is not always easy to make a correct diagnosis. Herein, we present a patient with bilateral parkinsonism caused by unilateral cerebral infarction, whose illness was first suspected to be Parkinson's disease.

2. Case report

A 61-year-old right-handed woman who had a history of falling twice in the previous month developed muscle weakness in her left extremities after eating lunch. Eight hours later, she consulted a hospital. She had no other significant medical history. Her systolic blood pressure was 200 mmHg, and she had a mask-like face and decreased spontaneity. She had moderate bradykinesia, rigidity in the neck and four limbs with postural retention disorder, brachybasia, and bilateral resting tremor with right dominancy in the lower extremities. There was no muscle weakness or sensory disturbance and her deep tendon reflexes were normal, without pathological reflexes. Repeated brain computed tomography (CT) revealed no abnormalities. Treatment with amlodipine and valsartan was initiated. Because her illness was suspected to be Parkinson's disease, she was referred to our hospital 2 days later. On her first visit to our hospital, her neurological symptoms were the same as in the first hospital, with an additional resting tremor in her right thumb and improvement of her postural retention disorder. Iodine-123 meta-iodobenzylguanidine (^{123}I-MIBG) myocardial scintigraphy revealed a normal heart to mediastinum (H/M) ratio. The washout rate with background and decay correction was also within the normal range. She was taking no additional medication because her symptoms had ameliorated. Six weeks later, brain magnetic resonance imaging (MRI) revealed abnormal signals in the right putamen, caudate nucleus, and external segment of the globus pallidus (Fig. 1). Dopamine transporter imaging with single-photon emission computed tomography (DaTSPECT) demonstrated reduced putaminal tracer binding concordant with the size and location of the vascular lesion (Fig. 2). However, the specific binding ratio was within the normal range (right 5.76, left 7.07; normal values for 61 years of age: 5.39–10.79). Upon treatment with cilostazol (200 mg/day), her symptoms gradually improved. Six weeks

* Corresponding author at: Department of Neurology and Geriatric Medicine, Ehime University Graduate School of Medicine, Shitsukawa, Toon, Ehime 791-0295, Japan.
E-mail address: shiroh46@m.ehime-u.ac.jp (S. Miura).

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later, her only symptoms were bilateral resting tremor with right dominancy in the lower extremities, and her clinical symptoms fully recovered after a further 2 months.

3. Discussion

We were able to pursue the clinical course of a patient with cerebral infarction involving the striatum and external segment of the globus pallidus, who presented with bilateral parkinsonism.

There have been several reports describing parkinsonism caused by infarction in the territory of the lenticulostriate arteries [2–4]. Although the majority of cases showed unilateral parkinsonism, some cases showed bilateral parkinsonism as a result of unilateral striatal infarcts [2–4]. Only two published studies have investigated DaTSPECT findings in such patients [3,4]. One study described two patients with bilateral parkinsonism confirmed at the time of follow up, 3 months after stroke, who showed bilateral pathological DaTSPECT with the classical pattern of idiopathic Parkinson's disease [3]. The symptoms of both patients improved with levodopa (600 and 440 mg/day, respectively). The authors eventually diagnosed the patients' parkinsonism as idiopathic Parkinson's disease. The other such study described a patient with bilateral parkinsonism confirmed at 11 months after stroke, who had a unilateral decrease of ligand uptake following the limits of the vascular lesion, which was different than the classical pattern seen in idiopathic Parkinson's disease [4]. A negative levodopa (450 mg/day) response was observed in this patient, and the authors speculated that the disturbance in dopaminergic transmission caused by the infarction was not able to be compensated by levodopa [4]. However, the authors did not suggest any possible reasons as to why bilateral parkinsonism may be induced by a unilateral vascular lesion. We propose two possibilities for our patient. The first is that she was in the prodromal stage of Parkinson's disease, and the parkinsonism appeared with the deterioration

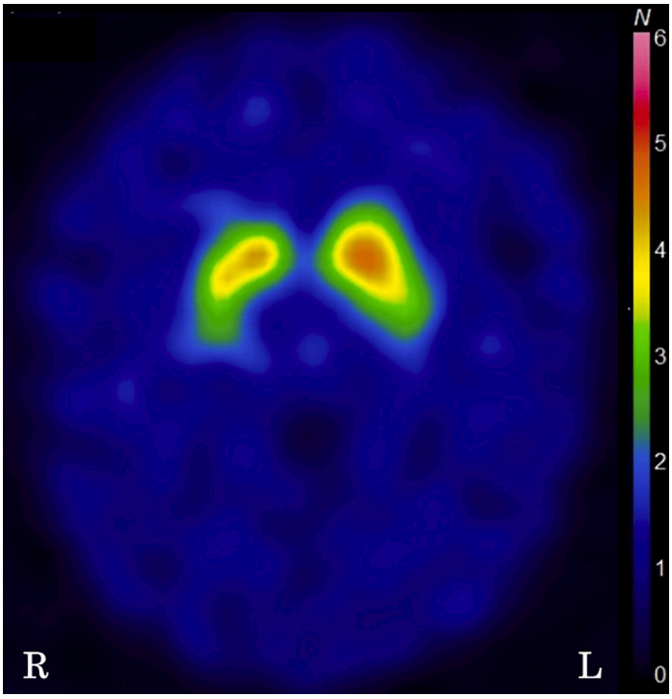

Fig. 2. Dopamine transporter imaging with single-photon emission computed tomography (DaTSPECT).
DaTSPECT demonstrated reduced right putaminal tracer binding compared with the left putamen, which was concordant with the size and location of the vascular lesion. Bilateral specific binding ratios were within the normal range (right 5.76, left 7.07; normal values for 61 years of age: 5.39–10.79).

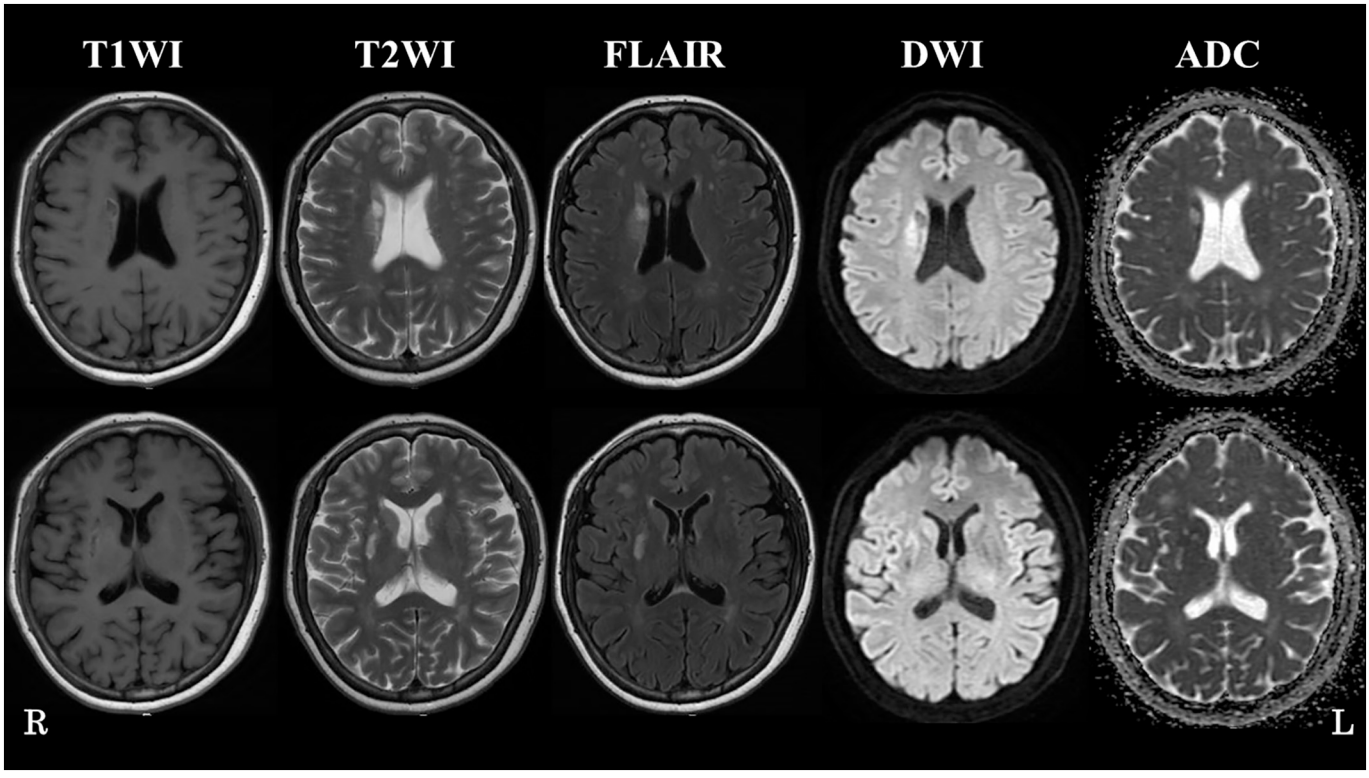

Fig. 1. Brain magnetic resonance imaging (MRI).
High signal intensity was observed in the territory of the left lenticulostriate arteries on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences. The lesions showed high signal intensity with partial low intensity on diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) map. The lesions showed low or iso signal intensity on T1-weighted sequences. These images suggest a subacute phase of infarction.

of her general condition. The second possibility is that she had a loss of microstructural integrity within the periventricular frontal regions that was not visible on conventional MRI, and that this was related to the presence of mild parkinsonian symptoms [5].

We present the first case of parkinsonism caused by cerebral infarction with symptoms that fully recovered without antiparkinsonian agents. The reversible course of the patient's parkinsonism may reflect insignificant reduction of neuronal integrity in the striatum resulting from ischemia [6], which is consistent with the DaTSPECT findings of the patient. Therefore, DaTSPECT may be useful for predicting the prognosis of parkinsonism caused by infarction.

In conclusion, it is important to be aware that infarction of the lenticulostriate arteries can be the cause of acute/subacute onset of bilateral parkinsonism. DaTSPECT may be useful to predict a patient's prognosis.

Informed consent

Obtained.

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Declaration of Competing Interest

None.

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