Neurological Sciences (2019) 40:1089–1091
https://doi.org/10.1007/s10072-018-3676-8

LETTER TO THE EDITOR

Early- and late-phase ^{18}F-FP-CIT PET images in vascular parkinsonism due to midbrain infarct

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Received: 11 October 2018 / Accepted: 3 December 2018 / Published online: 7 December 2018

Dear Editor,

The term vascular parkinsonism (VP) has remained debated because the definition is unclear and the mechanisms seem to be heterogeneous [1]. It is suggested that the damage to nigrostriatal and subcortico-cortical pathways may lead to parkinsonism [2]. Dopamine transporter imagings in VP seem to be inconsistent. We introduce a VP patient showing the abnormal dopaminergic activity of the ipsilateral striatum after focal midbrain infarction, which was rarely reported.

A 71-year-old, right-handed woman with a previous history of hypertension visited our clinic for both hand tremor and bradykinesia. She reported that the right hand tremor occurred 4 years ago, and the left hand tremor and bradykinesia, 1 year ago. She denied acute onset of those symptoms. She told us that her hand tremor occurred while writing, using a spoon, and the rest. Constipation occurred 1 year ago, but there were no other non-motor symptoms such as REM sleep behavior disorder (RBD) and urinary problem. There was no family history of movement disorders and no history of neuroleptics, head trauma, or cerebrovascular disease. Neurological examination showed a masked face, resting tremor, rigidity, and bradykinesia, and decreased arm swing on the right when walking. The tremor, rigidity, and bradykinesia were more severe in the right limb than in the left limb. She did not present postural instability and gait abnormalities including short step gait, festination, freezing, and a wide-based gait. She was stage 2 on the Hoehn and Yahr scale, and The Unified Parkinson's Disease Rating Scale (UPDRS) score was 29 (part I, 3; II, 7; III, 19; IV, 0). The Korean version of the Mini-Mental State Examination (K-MMSE) was 21 points (education, 6 years) and the Korean version of the Montreal Cognitive Assessment (MoCA-K) was 17 points. Beck's Depression Inventory (BDI) was 31 points. The Korean Version of Sniffin' Sticks test (KVSS) showed hyposmia (the odor threshold test, 3.5 points; the odor discrimination test, 8 points; the odor identification test, 9 points).

A brain MRI showed an old lacunar infarct in the left midbrain. [18F] N-(3-fluoropropyl)-2b-carbon ethoxy-3b-(4-iodophenyl) nortropane (FP-CIT) PET images were obtained 10 and 180 min after injection of ^{18}FFP-CIT (5 mCi; 185 MBq) as in a previous study [3]. The early phase FP-CIT images were reported to provide complementary FDG-like information to the dopamine transporter (DAT) images of the late phase [3]. The early phase images showed an asymmetrically reduced perfusion at the left caudate and putamen, and the late phase images (routine delay images) showed reduced DAT activity at the left caudate and putamen (Fig. 1). Her myocardial ^{123}I-metaiodibenzylguanidine (MIBG) scintigraphy was normal.

The patient received a total of 300 mg of levodopa and 75 mg of carbidopa for 3 weeks, but she complained no response to the medication and asked for a request to go to another hospital.

The concept of VP has been commonly used but also criticized since the first description of Critchley in 1929 [4] because pathogenesis and pathology are unclear. There are poor clinicopathologic data and no specific pattern of brain lesions in VP. Focal strategic infarct can cause VP, which has been rarely reported. Most previous reported cases showed acute onset and unilateral symptom and signs [1, 2, 5, 6]. Only one case had unilateral parkinsonism without stroke-like episode [7]. In our case, it was a silent infarct and bilateral, asymmetric manifestations of

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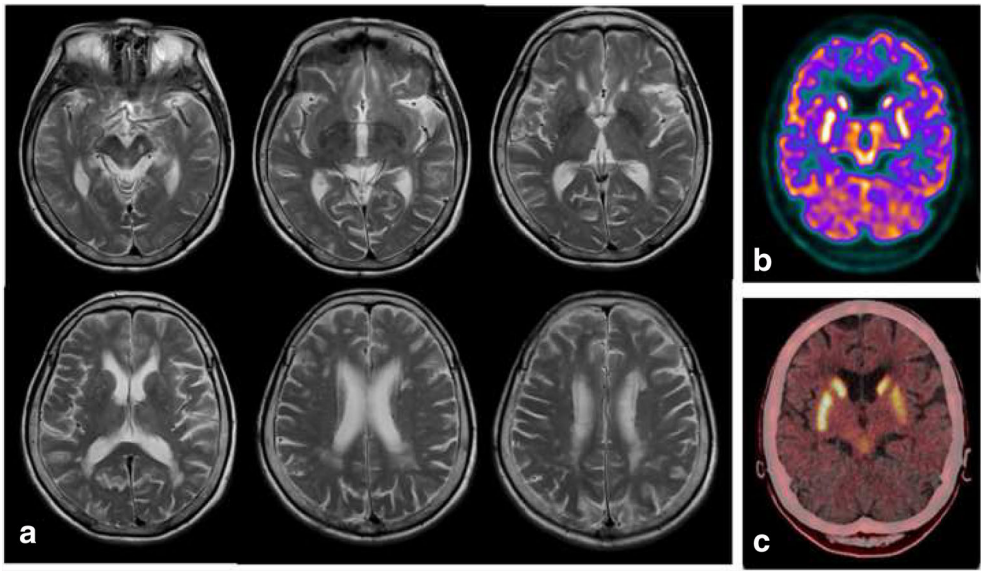

Fig. 1 **a** Brain MRI shows focal old infarction in the left substantia nigra. **b** Early phase image (i.e., early perfusion dominant image, first 10-min image) of ^{18}F-FP-CIT PET shows asymmetrically reduced perfusion at the left caudate and posterior putamen. **c** Late phase image (i.e., routine delay image, first 180-min image) shows marked reduction of presynaptic dopaminergic uptake in the left caudate and posterior putamen

parkinsonism that can be explained by additional bilateral subcortical white matter lesions or collateral projection from the substantia nigra to the striatum [8].

In clinical practice, VP should be differentiated from the idiopathic Parkinson's disease (IPD), because the treatment and prognosis are different [9]. Searching for non-motor symptoms may be helpful for the differentiation, but our patient had hyposmia, depressive mood, and constipation, and denied RBD. Studying brain perfusion was reported to be useful in the differential diagnosis of parkinsonism [3]. The perfusion of the striatum is known to be bilaterally increased in IPD [3], but the perfusion of the putamen was decreased ipsilateral to the SN lesion in our patient, which was consistent with DAT findings. A similar imaging finding has been previously published, although the parkinsonism was a sudden onset [10]. The regional cerebral metabolic rate of glucose and the dopaminergic activity was measured using [^{18}F]-fluorodeoxyglucose and [^{18}F]-L-6-fluorodopa, respectively [10]. In that case, FDG uptake was reduced in the putamen, frontal cortex, and cerebellum. In addition to VP, similar DAT findings (reduced DAT activity at the striatum ipsilateral to the SN lesion) have also been reported in post-infective parkinsonism [11].

Cardiac MIBG scintigraphy is also useful for the differential diagnosis and it shows normal or slightly decreased uptake in VP compared with healthy people [12], which is consistent with our case.

In conclusion, we report a rare case of vascular parkinsonism due to SN lesion. The DAT findings were decreased activity of the ipsilateral striatum that may be due to anterograde degeneration of the nigrostriatal pathway [6]. Our findings support the idea that damage to nigrostriatal pathways may lead to parkinsonism [2].

Our observation has some limitations. Because hyposmia, depressive mood, and constipation are known to be prodromal symptoms in IPD, we could not completely exclude an additional possibility that this may be a case of early IPD with superimposed nigral ischemic damage. There was some lack of a suitable follow-up and the objective evaluation of drug response for the differential diagnosis.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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