Abstract

Parkinson’s disease is known to present and mostly persist as an asymmetrical movement disorder in most cases. The asymmetry is mainly described in motor features such as bradykinesia, rigidity and tremor in upper and lower limbs. Unilateral hypomimia however, has only been reported in 14 patients, all of whom showed right-sided hemihypomimia. In this case report we describe the symptoms of a 51-year-old man with predominant left-sided Parkinson’s disease in whom we discovered a left-sided hemihypomimia. We also briefly review the literature concerning hemihypomimia in Parkinson’s disease. We conclude that a larger case series needs to be studied to further elucidate the pathophysiology and clinical implications of this observation.

Key words: Parkinson’s disease; asymmetry; hypomimia; unilateral.

Case report

We report on a 51-year-old right-handed male, who was diagnosed with idiopathic Parkinson’s disease (PD) 3 years ago. The initial symptoms were clumsiness, subjective weakness and an intermittent resting tremor in the left upper limb, accompanied by occasional tripping. Medical history was unremarkable. Cranial nerve function was normal, except for left-sided hypomimia. The glabellar tap reflex was noted on the left side. Mild cogwheel rigidity was present in the left upper limb and moderate bradykinesia was present in both upper limbs. An intermittent resting tremor was noted in the left upper limb. Occasionally, resting tremor was also present in the right upper limb. Postural reflexes were conserved. Gait was normal with reduced left-sided arm swing. MR imaging of the brain did not reveal brainstem lesions or other abnormalities. 123I-FP-CIT SPECT showed severely reduced ligand binding in both putamina and slightly reduced ligand binding in the right caudate nucleus. Unified Parkinson’s Disease Rating Scale (UPDRS) motor score in ‘on’ phase is now 22/108 with Hoehn-and-Yahr stage 2. Over the 3 years of disease duration the motor symptoms remained quite stable. Recently, we noticed a predominance of the hypomimia for the left side of the face. The asymmetric hypomimia was especially prominent in spontaneous facial expressions (e.g. during spontaneous speech, see Figure 1) and less pronounced when given instructions for facial movement. We asked the patient to raise the eyebrows, close the eyes, smile and whistle. These voluntary facial movements were symmetric and were not indicative of paresis of facial muscles. Both upper and lower half of the left face exhibited hypomimia.

Discussion

We describe the first case of left-sided hemihypomimia (HH) in a right-handed PD patient. Fourteen PD patients with HH have previously been reported in the literature. In all of these patients the right side of the face was affected (1, 2). Right-handedness was observed in thirteen of these patients, while this information was not provided for the last case. We can therefore speculate that handedness is probably not influencing the side preference of hypomimia. Early onset age and short disease duration seem to be associated with HH, as in our patient (2). Probably, HH is not a rare feature, but merely often remains unnoticed. The subtle difference in symmetry of facial expressions was most obvious in our case during spontaneous speech. Therefore, it can easily be overlooked in clinical practice.

Various hypotheses concerning the asymmetry of motor symptoms in PD have been proposed. Genetic predisposition may confer a greater vulnerability of one substantia nigra compared to the contralateral one. Inborn variations in the number of nigral dopaminergic neurons could cause the side with a
Both upper and lower half of the left hemiface were involved in our patient, in contrary to the involvement of only the lower half in the previously reported patients. The bilateral corticobulbar innervation of the upper half of the face can therefore not longer serve as an explanation for the findings in our patient. We corroborate the observation of Zingler et al. that HH is especially prominent in spontaneous facial expressions and less pronounced when given instructions for facial movement. A possible explanation could be that the basal ganglia motor output is more directed towards the medial cortical motor areas, which are activated during internally guided movements. The supplementary medial motor cortex is hypoactive in PD, while the lateral motor areas are less affected. Therefore, an increased activity of the lateral motor cortex is often present, which can possibly explain the improved performance when external cues are given to guide movements (9).

Spontaneous facial expressions are internally guided movements and could be more vulnerable to hypomimia than voluntary ‘cued’ facial movements. Finally, we recommend being attentive for HH in PD patients. Larger case series need to be studied to further elucidate the pathophysiology and clinical implications of this observation.

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REFERENCES


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