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Asymmetric and Upper-Body Parkinsonism in Patients with Idiopathic Normal-Pressure Hydrocephalus

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Dear Editor,

Kang et al.[1] have illustrated asymmetric and upper-body parkinsonism signs in detail via follow-up clinical assessments [before and after cerebrospinal fluid-tap test (CSFTT)] in a large number of patients with idiopathic normal-pressure hydrocephalus (INPH). I would like to make some comments in order to add new perspectives to aid the understanding of that report.

An important aspect of this study is whether the selection criteria of the patients were sufficient to distinguish patients with only normal-pressure hydrocephalus (NPH). For example, the co-occurrence of NPH and other degenerative disorders has been emphasized in recent reports.[2] Although a few reports have mentioned NPH patients with atypical, marked upper-body and asymmetric parkinsonism manifestations,[3,4] the landmark knowledge emphasizes lower-body parkinsonism in NPH. I therefore consider that drawing conclusions about such devastating results requires the possibility of the co-occurrence of other parkinsonian disorders to be excluded. The results of pathological studies may substantially contribute to such discussions. On the other hand, the dramatic improvement in most parkinsonian signs of the patients supports the presence of underlying NPH hydrocephalus. Nonetheless, I wonder if the authors have data on whether or not levodopa responsiveness was absent in these patients.

Another important point may be that the authors reported a significant improvement in global tremor score after CSFTT. However, the specific type of tremor was not reported, which might be useful in further discussions on reversible disturbances of possible responsible pathways. For example, while parkinsonian tremor is known to be associated with disturbances in the basal ganglia loop, disturbance in the cerebellar feedforward control of voluntary movements has instead been attributed to the occurrence of cerebellar tremor.[5] Hence, I think that the specific tremor subtypes need to be clarified.

Bradykinesia is reportedly the most common condition to improve after CSF drainage.[6] Some authors argue that specific symptoms in NPH revert to normal function with reduction of direct shearing forces. As also emphasized in the report, a probable explanation for the motor function recovery in INPH could be the reversible suppression of frontal periventricular corticobasal ganglia-thalamo-cortical circuits.[1] It has also been suggested that postsynaptic D2 receptors in the putamen can be damaged by hydrocephalus, and that a pressure load on the midbrain and the floor of the third ventricle may result in parkinsonian symptoms in patients with severe NPH.[7] However, conflicting with this hypothesis, the patients in Kang et al.[1] did not have severe NPH–their score on the INPH grading scale was 5.7±1.9 (mean±SD). Remarkably, the study found no significant differences in white-matter lesions and frontal horn diameter between the hemispheres ipsilateral and contralateral to the dominant motor symptoms. I consider that additional evaluations of these parameters before and after CSFTT might contribute substantially in understanding the underlying mechanisms of asymmetrical manifestations in NPH and the differing recovery of distinct parkinsonism

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symptoms after CSFTT.

In conclusion, I think that this is a very important study illustrating asymmetric and upper-body parkinsonism in a considerable number of patients with NPH. However, re-evaluation of some of the points mentioned in this letter may provide a more comprehensive understanding of this interesting report. Future studies focusing on these atypical manifestations of NPH using detailed neuroimaging techniques are needed to clarify the arguments concerning NPH pathophysiology and the mechanisms of other parkinsonian signs in NPH.

Conflicts of Interest

The author has no financial conflicts of interest.

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