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LETTER TO THE EDITOR

Is nucleus accumbens atrophy correlated with cognitive symptoms of Parkinson's disease?

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Sir,

I read the recent article of Hanganu *et al.* (2014) entitled 'Mild cognitive impairment is linked with faster rate of cortical thinning in patients with Parkinson's disease longitudinally' with great interest and I would like to comment on some of their important findings.

Hanganu *et al.* (2014) investigated the longitudinal changes of the cortical and subcortical grey matter in patients with Parkinson's disease, at the early stages of the illness, with and without mild cognitive impairment. They found a higher rate of cortical thinning in the temporal, occipital, parietal and supplementary motor area, in patients with Parkinson's disease with mild cognitive impairment compared with both cognitively stable patients and healthy controls. Subcortically, a significant decrease in the volume of the amygdala and nucleus accumbens was observed in the group with Parkinson's disease with mild cognitive impairment versus both Parkinson's disease without mild cognitive impairment and healthy controls. Yet, only these two structures showed a significant decrease of grey matter over time in Parkinson's disease with mild cognitive impairment versus the Parkinson's disease group without such impairment (Hanganu *et al.*, 2014).

Their results indicate that the early presence of mild cognitive impairment in patients with Parkinson's disease is associated with a faster rate of grey matter thinning in various cortical regions as well as a significant diminishment of limbic subcortical structures. Their results also indicate that dopamine depletion in the ventral striatum of patients with Parkinson's disease with mild cognitive impairment might be significantly greater than in those with Parkinson's disease without such impairment. They interestingly mentioned that 'dopamine depletion in Parkinson's disease progresses from the dorsal striatum to the ventral striatum, and in the early stage of the disease the dorsal striatum is severely depleted, whereas the ventral striatum is relatively intact' (Hanganu *et al.*, 2014).

Nucleus accumbens atrophy in Parkinson's disease was first described a few years ago and was initially proposed to be associated with the neuropsychiatric symptoms of the disease (Mavridis *et al.*, 2011). Neuronal loss due to dopaminergic degeneration is probably the major cause of this atrophy. Functional nucleus accumbens changes such as decreased dopamine, neuronal dysfunction and changes in its synaptic plasticity are expected to accompany its atrophy. Degeneration of limbic areas could be easily considered as a pathological consequence of this atrophy (Mavridis, 2014).

The possible clinical consequences of nucleus accumbens atrophy were recently suggested to include both neuropsychiatric (limbic dysfunction symptoms, depression, learning impairment, apathy, anxiety, anhedonia, bradyphrenia) and motor (hypokinesia, akinesia) symptoms of Parkinson's disease (Mavridis, 2014). I consider the study of Hanganu *et al.* (2014) as particularly important because it proves the correlation of a specific symptom of Parkinson's disease, namely cognitive impairment, with nucleus accumbens atrophy. It also confirms the phenomenon of this atrophy.

Four years after the discovery of nucleus accumbens atrophy in Parkinson's disease (Mavridis' atrophy), it is certain that we still have a lot to learn about its role in the expression of the disease. Thanks to research efforts like that of Hanganu *et al.* (2014), the clarification of this role has begun and we have started having answers to questions like 'Is nucleus accumbens atrophy correlated with cognitive symptoms of Parkinson's disease?'. The answer is 'Yes'.

References

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