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I read the recent article by Mak et al[1] regarding the association of subcortical atrophy with cognitive impairment in Parkinson disease (PD), with great interest. The authors assessed cortical thickness and subcortical volumes in patients with PD without dementia and evaluated their associations with cognitive dysfunction. They found that patients with PD and mild cognitive impairment demonstrated reduced volumes of the thalamus and nucleus accumbens (NA) and significant associations for the NA and putamen with performances on the attention/working memory domains and NA and language domains.[1] The purpose of this communication is to comment on the phenomenon of NA atrophy in PD and its correlation with cognitive symptoms.

In the clinical study of Mak et al,[1] the total volume of the NA was significantly correlated with a range of cognitive variables, including overall scores of attention/working memory and language. They also found that a trend toward the associations between NA and global cognition and executive function performance on attention/working memory was associated with reduced volumes of the NA and putamen. Additionally, the NA was also significantly correlated with performance in the language domain. The authors suggested, as possible explanation for their findings, the previous evidence linking the NA to memory and learning processes.[1]

Although the involvement of subcortical deep gray matter and cortical thinning associated with mild PD remains poorly understood,[1] we currently know that atrophy of the human NA in PD, called Mavridis atrophy (MA), begins in early-stage PD and is correlated with psychiatric symptoms that occur in PD, mainly apathy and impulsive behavior.[2] Two recent clinical studies have also associated the phenomenon of MA with cognitive PD symptoms.[2-4] More specifically, O'Callaghan et al[3] found reduced learning rates in patients with PD without dementia relative to controls and that this learning impairment was directly related to gray matter loss in discrete frontostriatal regions, including the NA. Hanganu et al[4] found a significant decrease in the NA volume in patients with PD and mild cognitive impairment. Therefore, the study of Mak et al[1] is the third clinical study to confirm the MA association with cognitive symptoms in PD.

Finally, Mak et al[1] proposed that nuclei such as the NA may serve as potential biomarkers for PD–mild cognitive impairment. In agreement with their advice, previous authors have suggested that it is time to evaluate MA (as an imaging finding) as a risk factor for the expression of specific PD symptoms. The evaluation should emphasize those symptoms that we already know are related to MA, namely psychiatric and cognitive symptoms. It is also time to evaluate MA as a risk factor (prognostic factor) for the severity of the disease.[2] Further research is definitely needed, and PD seems ready to reveal some of its well-kept secrets.

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