Variability of diffuse plaques and amyloid angiopathy in Parkinson's disease with mild cognitive impairment

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We appreciate Professor Jellinger's correspondence [2] regarding our study [1] and his findings also showing heterogeneity of Lewy body and Alzheimer pathology in an additional eight cases of PD-MCI (four amnestic, four non-amnestic) [2]. Professor Jellinger questioned our lack of data on diffuse (amyloid) plaques and cerebral amyloid angiopathy (CAA), and so those data are presented.

As seen in the Table 1, five cases had 3+ diffuse amyloid plaque density in the cortex. Caudate-putamen amyloid plaque density ranged from 0 to 2+. While we did not measure CAA in the meninges, we did in multiple

cortical regions (frontal, temporal, parietal, and occipital) using a 0–3 scale that was then totaled (maximum possible score = 12). Four cases (3 amnestic, 1 non-amnestic) had CAA score of 0, then one case each had scores of 1 (amnestic), 3 (non-amnestic), 4 (non-amnestic), and 5 (non-amnestic). All the cases with CAA score ≥ 1 had a diffuse amyloid plaque density of 3+.

We therefore agree with Professor Jellinger that further data are needed, with multivariate analysis, to determine if diffuse amyloid plaque load and CAA contribute to cognitive impairment in PD.

Table 1 Demographics and neuropathologic findings

Age	Gender	PD duration (years)	MCI type	Unified Lewy body stage	Neuritic plaques	Braak AD stage	Amyloid plaques, cortex	Amyloid plaques, caudate putamen	CAA total score
74	Male	25	N-A	Brainstem-Limbic	0	III	0	0	0
77	Male	14	A	Brainstem	0	III	0	0	0
79	Female	17	A	Neocortical	Moderate	IV	3	2	0
81	Male	11	A	Brainstem	0	II	0	0	0
85	Male	13	A	Brainstem-Limbic	Moderate	IV	3	2	1
88	Female	2	N-A	Neocortical	Sparse	II	3	1	3
89	Male	7	N-A	Brainstem-Limbic	Moderate	III	3	0	5
89	Male	2	N-A	Brainstem	Moderate	III	3	0	4

N-A non-amnestic MCI, A amnestic

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