

Metabolic Alterations in Patients with Levodopa-Treated Parkinson Disease by *In Vivo* ^1H MR Spectroscopy

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Purpose: To evaluate alterations of observable metabolite ratios between the cerebral lesion and the contralateral region related to the clinical symptomatic side in levodopa-treated Parkinsons disease (PD) and to investigate a correlation between age in patients and metabolite ratios of the lesion in PD.

Materials and Method: Patients with levodopa-treated PD (n = 54) and age-matched normal controls (n = 15) underwent MRS examinations using a stimulated echo acquisition mode (STEAM) pulse sequence that provided 2x2x2 cm³ volume of interest in the selected regions of substantia nigra (SN) and putamen-globus pallidus (PG). To evaluate dependence of metabolite ratios on age, we divided into two groups (i.e., younger and older age). We quantitatively measured N-acetylaspartate (NAA), creatine (Cr), choline-containing compounds (Cho), inositols (Ins), and the sum (Glx) of glutamate and GABA levels and obtained proton metabolite ratios relative to Cr using a Marquart algorithm.

Results: Compared with the contralateral region, a significant neuronal laterality of NAA/Cr ratio in the lesion of SN related to the clinical symptomatic side was established ($P = 0.01$), but was not established in the lesion of PG ($P = 0.24$). Also, Cho/Cr ratio tended toward significance in the lesion of SN ($P = 0.07$) and was statistically significant in the lesion of PG ($P = 0.01$). Compared with that in the younger age group, NAA/Cr ratio in the older age was decreased in the lesion of SN ($P = 0.02$), while NAA/Cr ratio was not statistically significant in the lesion of PG ($P = 0.21$).

Conclusion: Significant metabolic alterations of NAA/Cr and Cho/Cr ratios could be closely related with functional changes of neuropathological processes in SN and PG of levodopa treated PD and could be a valuable finding for evaluation of the PD. A trend of NAA/Cr reduction, being statistically significant in older patients, could be indicative of more pronounced neuronal damage in the SN of the progressive PD.