Arterial spin labelling MRI at 1.5T does not differentiate parkinsonism-predominant multiple system atrophy from Parkinson disease

Because the progression of multiple system atrophy (MSA) is rapid without any effective disease-modifying therapy, an early and accurate diagnosis is important. However, sometimes it is hard to make a secure diagnosis, especially in early cases. For MSA-P, when a patient presents with mild parkinsonism without overt ataxia or autonomic dysfunctions, a diagnosis cannot be made with certainty, and the major differential diagnosis includes Parkinson disease (PD). Demonstration of hypometabolism in the putamen, brainstem, and cerebellum by FDG-PET can help the diagnosis of MSA. However, FDG-PET is limited by its high cost, restricted availability, and exposure to radioactive agents.

Arterial spin labeling (ASL) MRI provides a non-invasive measure of cerebral blood flow (CBF) without using exogenous contrast or radioactive tracers. Because CBF is generally tightly coupled to cerebral metabolism, it has been suggested that CBF assessed by ASL MRI can be used as a proxy for cerebral metabolism. ASL MRI has in fact been used in several studies on neurodegenerative disorders including PD as a substitute for FDG-PET. The aim of this study was to examine whether MSA-P can be distinguished from PD using ASL MRI at 1.5T.

Forty-two patients with PD and 26 patients with probable MSA-P were included in this study. The diagnosis of MSA was made based on the consensus criteria. All the patients were in the early stage of disease and followed for a mean duration of 2.3±1.2 years after ALS MRI without change in the diagnosis. A 1.5-T MR imaging unit with a 16-channel head coil was used to acquire the ASL MRIs. The details of the technique have been published elsewhere. Regional CBF (rCBF) was compared between PD and MSA-P in the cerebellum, right and left putamen, and right and left caudate using region-of-interest (ROI) analysis.

There was no significant difference in sex distribution, age at disease onset, age and disease duration at MRI between PD and MSA-P (Table 1). On conventional MRI, out of 26 patients with MSA-P, putaminal atrophy was demonstrated in 11 patients; putaminal hyperintense rim in 9, posterolateral putaminal hypointensities in 4, hot cross bun sign in 3, and pontocerebellar atrophy in 11. Comparison of the ASL MRI findings between the groups showed no significant rCBF differences in any ROI between PD and MSA-P.

One may argue that the lack of a difference in this study might have resulted because the patients with MSA-P were at a very early stage of the disease such that there were no detectable changes in the striatum and cerebellum that had developed yet. However, changes in metabolism in the striatum and cerebellum have repeatedly been reported in patients with shorter disease durations compared to our patients in studies using FDG-PET. Furthermore, putaminal abnormalities in susceptibility weighted imaging may have been missed by ASL MRI due to limitations in its spatial resolution.

| Table 1. Clinical characteristics of the patients |
|--------------------------------------------------|-----------------|-----------------|-----------------|
| PD (n=42) | MSA-P (n=26) | p-value |
| M:F | 13:29 | 11:15 | NS |
| Age at onset | 63.1±8.3 | 63.0±8.0 | NS |
| Age at MRI | 66.5±8.0 | 66.0±7.6 | NS |
| Duration (yr) at MRI | 3.4±2.9 | 3.0±1.8 | NS |
| Follow-up (yr) after MRI | 2.4±1.1 | 2.3±1.3 | NS |

Values are expressed as mean ± SD.
PD, Parkinson disease; MSA-P, parkinsonism-dominant multiple system atrophy; NS, non significant.
Values between groups were compared using t-test or chi-square test as appropriate. The level of statistical significance was set at two-tailed p <0.05.
imaging and diffusion weighted imaging, which indicate structural changes, have been reported in association with FDG-PET abnormalities in patients with early MSA.\(^5\) Thus, it is more likely that ASL MRI at 1.5T cannot detect the change in metabolism in the striatum and cerebellum in MSA-P. It probably is because the change in regional metabolism in the striatum and cerebellum is not parallel with the rCBF change. Alternatively, it is also likely that ASL MRI at 1.5T cannot detect the change in rCBF itself due to its inherent limitations. In this regard, ASL MRI at higher field strengths may provide a better assessment. Indeed, CBF changes in other neurodegenerative disorders including PD, corticobasal syndrome, ataxia, and AD have been reported using ASL MRI at 3T.\(^1\)

In conclusion, this study shows that the rCBF in the striatum and cerebellum measured by ASL MRI at 1.5T cannot differentiate between MSA-P and PD. Further studies with ASL MRI at higher field strengths are needed to examine the possible role of ASL MRI in the diagnosis of MSA.

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