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Regarding ''Perfusion MR Imaging Using a 3D Pulsed Continuous Arterial Spin-Labeling Method for Acute Cerebral Infarction Classified as Branch Atheromatous Disease Involving the Lenticulostriate Artery Territory''

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Regarding "Perfusion MR Imaging Using a 3D Pulsed Continuous Arterial Spin-Labeling Method for Acute Cerebral Infarction Classified as Branch Atheromatous Disease Involving the Lenticulostriate Artery Territory"

e read the article on the use of 3D pulsed continuous arterial spin-labeling (ASL) MR imaging for acute infarction associated with presumed branch atheromatous disease (BAD) by Shinohara et al¹ with great interest. The authors suggested that the baseline NIHSS score was significantly correlated with the asymmetry index of CBF in the contralateral cerebellar hemisphere (crossed cerebellar diaschisis, AI_{CCD}) (r = 0.515) and DWI lesion volume (r = 0.664), whereas it was not with the asymmetry index of CBF in the affected area (DWI lesion). The point of this study would be that even acute cerebral infarction by presumed BAD can show crossed cerebellar diaschisis on ASL MR imaging, which is correlated with the degree of neurologic severity. Their observations will draw the interest of the American Journal of Neuroradiology readers. As the authors pointed out, however, the clinical implication of the so-called BAD-type DWI lesions, which is usually determined by their location and size, is that they are strongly associated with early neurologic deterioration (END). A recent study with 587 patients showed that BAD-type DWI lesions and relevant artery stenosis can predict END, whereas the baseline NIHSS score cannot.² Thus, we already know that larger or longer DWI lesions may be a red flag for END, particularly when they are associated with relevant artery stenosis. In this circumstance, it would be better to provide firm evidence for obtaining ASL MR imaging by assessing its utility for the prediction of END. We also would like to comment on the methods in this study. First, the correlation between the $\mathrm{AI}_{\mathrm{CCD}}$ and the baseline DWI lesion volume should be tested because it is expected that baseline DWI lesion volumes correlate well with baseline NIHSS scores, and larger DWI lesions have CCD more frequently. Thus, AI_{CCD} and

baseline DWI lesion volume may have collinearity. If that is the case, the authors should conduct multivariable regression analysis to determine which one is independently associated with baseline NIHSS score. Second, it seems that the baseline NIHSS scores of 23 patients are not normally distributed (based on the Shapiro-Wilk test). Thus, nonparametric linear regression and Spearman rank correlation are rather appropriate. Third, the prevalence of END and clinical outcomes (such as discharge NIHSS scores or mRS at 3 months) should be compared between the patients with higher AI_{CCD} and those with lower value to confirm the clinical implication of obtaining ASL MR imaging in these patients. The authors only showed that the AI_{CCD} detected by ASL MR imaging is correlated with the baseline NIHSS score in this study. Finally, if they obtained ASL MR imaging in patients with non-BAD acute infarction, it would be better to include them for further analysis to determine whether they are different from those with BADtype acute infarction in terms of prediction of END or baseline neurologic severity.

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