T1rho in acute cerebral infarctions

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<u>Purpose</u>: T1rho is sensitive to changes in interaction between water and macromolecules, including dipolar interactions and chemical exchange. In the ischemic brain, it is likely that physicochemical conditions will slow down chemical exchange affecting T1rho¹, and T1rho may provide different contrast from T1 or T2. Clinical application of T1rho MRI for human brain is limited and there is little experience in the acute cerebral infarctions. In this study, we evaluated T1rho of acute cerebral infarctions comparing with DWI.

<u>Methods</u>: Seven patients with acute cerebral infarctions within 2 days of onset were enrolled. T1rho MRI was acquired by 3.0T clinical scanner with SENSE-Head-32ch coil (Achieva TX, Philips Healthcare, Best, The Netherlands). Each signal is read out by fast field echo sequence (TR = 3.9 ms, TE = 2.0 ms, flip angle = 35°) after the spin lock pulse of 500 Hz for the time of spin lock (TSL = 1, 10, and 80ms). The T1rho maps were obtained by fitting the equation for T1rho; S(TSL) = S(0) *exp(-TSL / T1rho). We evaluated values of the infarcted brain tissue on T1rho map compared with hyperintense area on DWI.

Results: There were 8 acute cerebral infarctions that showed hyperintensity on DWI. Seven of 8 lesions located in the deep white matter or deep gray matter and showed entirely prolonged T1rho compared with normal cerebral tissue. In 4 of 7 lesions, areas with prolonged T1rho included hyperintense areas on DWI (figure 1A, 1B). Mild elevation of T1rho was found around hyperintense areas on DWI (figure 1C).

Remaining 3 lesions could not be differentiated from background brain tissue on T1rho maps. One infarction located in the cortex showed variable T1rho values (**figure 2**). Their values were higher than, or equal to those of the normal tissue. The area with prolonged T1rho was much larger than the hyperintense area on DWI.

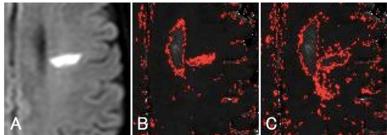


Figure 1. A) DWI depicted acute infarction in the deep white matter, B) T1rho map; red represents T1rho values between 95ms and 110ms. This area with abnormally high T1rho had a same distribution as hyperintensity on DWI. C) T1rho map between 85ms and 90ms showed mild prolongation just around the hyperintensity on DWI.

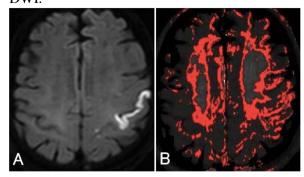


Figure 2. A) DWI demonstrated acute infarction in the cortex. B) T1rho map between 85ms and 100ms. Area with abnormally increased T1rho was more extensive than hyperintense area on DWI. However, normal T1rho value was also present in the hyperintense area on DWI.

Discussion: T1rho of brain was reported to increase immediately after the irreversible ischemia occurs². In this study, similarly, T1rho of the infarcted cerebral tissue was prolonged in the deep white matter and the deep gray matter. The areas with prolonged T1rho were slightly larger than the hyperintense areas on DWI and there were small mismatches between T1rho map and DWI. In the infarction of cerebral cortex, this mismatch was extensive. Area with prolonged T1rho was much larger than the hyperintense area on DWI. This normal appearance on DWI with prolonged T1rho may represent penumbra region. However, normal T1rho within hyperintensity on DWI cannot be interpreted. Three lesions could not be delineated on T1rho maps. This is because T1rho of background brain around acute infarctions was diffusely prolonged for chronic ischemia.

<u>Conclusion</u>: T1rho was prolonged in most of the acute infarctions. There were mismatches between T1rho map and DWI, and they may indicate penumbra regions or other pathological processes.

References:

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