Serial MR Analysis of Permanent and Transient Cerebral Ischemia in a Rat Model: High and Low b Value Diffusion – Weighted Imaging and Diffusion Tensor Imaging

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Introduction
It has been proposed that increased diffusion anisotropy in acute infarct indicates continued structural integrity and tissue salvageability (1). High b value DWI has been reported to improve the conspicuity of small infarct and hyperacute infarct in human (2) and it was reported that the diffusion of brain water showed biexponential decay when measured over a b factor range of up to 10000 s/mm² (3). We tried to determine the serial changes in fractional anisotropy (FA), ADC and DWI signal intensity (SI) at high and low b values during permanent and transient focal cerebral ischemia in a rat model and investigated their relationships.

Materials and Methods
Thirty male Sprague-Dawley rats were subjected to middle cerebral artery occlusion (MCAO) with the suture occlusion model. After MCAO, PWI, DTI, high and low b value DWI were performed at 15, 30, 45, 60 minutes in hyperacute permanent ischemia group (n=9), 1, 3, 5, 7, and 9 hour in acute permanent ischemia group (n=13), at 15 minute before reperfusion, 30 minute, 2.5 hour, and 24 hour after reperfusion in 30 minute transient ischemia group (n=8) on 3.0 T unit MR (Signa; GE Medical Systems, Milwaukee, WI.). All MR images were obtained with FOV = 8 X 8 cm, matrix = 64 X 128 and section thickness = 3 mm. Bolus-tracking PWI with spin-echo echo planar imaging (EPI) was performed with TR/TE = 2000/60 ms and 1 NEX. Imaging parameters for DTI were as follows: b = 700, 6 directions, TR/TE = 6000/78.9, and NEX = 32. For high b value, b = 3000 TR/TE= 6000/99.6, and NEX = 32 were used. Two same ROIs in the center of the ischemic lesion (caudoputamen) and contralateral normal brain at the level of anterior commissure were chosen to measure FA, ADC, and DWI SI on the corresponding permanent ischemia group (n=13), at 15 minute before reperfusion, 30 minute, 2.5 hour, and 24 hour after reperfusion in 30 minute transient ischemia groups. The ratios of DWI SI at both b factors increased with time. The ratio of b 3000 DWI SI was larger than b 1000 (P<0.000). The ratios of both ADCs showed initial decrease until 3 hour and then increasing tendency. The ratio of b 1000 ADC was smaller than b 3000 (P<0.000). At 3 hour after MCAO, the ADC ratios were lowest (b 3000: 0.66 ± 0.04, b 1000: 0.53 ± 0.04). The FAs were closest to normal between 3 hour and 5 hour after MCAO (3 hour: 1.06 ± 0.17, 5 hour: 0.93 ± 0.21). Figure 2. and 3. illustrate transient reversibility and secondary decay of FA, correlating with ADC change, in 30 minute transient ischemia group. There initially were complete reversals of the DWI hyperintensity and ADC hypointensity at both b values and FA hyperintensity at the left lateral caudoputamen area during the first 2.5 hours of reperfusion, followed by FA hypointensity (0.89±0.17) and the recurrences of the DWI hyperintensity (b 3000: 1.94±0.61, b 1000: 1.59±0.23) and ADC hypointensity (b 3000: 0.90±0.15, b 1000: 0.85±0.11) at 24 hours. In both permanent and transient ischemia groups, the lesion contrast ratio of b 3000 DWI was higher than b 1000. But the lesion contrast ratio of b 3000 ADC map was lower than b 1000. FA had no correlation with ADC (b=3000, 1000) and DWI SI (b=3000, 1000).

Conclusion
FA increase temporarily and then decrease sequentially in permanent ischemia. It might be related to the initial shift of water from extracellular space to intracellular space. In 30 minute transient ischemia, FA shows transient reversibility and secondary decay, correlating with ADC change. The benefit of high b value DWI in evaluating early ischemia has limitation because of poorer lesion contrast ratio of ADC. It might be related to biexponential decay of cerebral water diffusion.

References