Magnetic resonance imaging of degeneration induced experimentally in central nervous system (CNS) of rats

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This study was performed to examine the effects of the degeneration of central nervous system (CNS) in rats on magnetic resonance imaging (MRI) under a high magnetic field of 7.05 T. The animal model for brain injuries such as the accumulation of lipid peroxides, the neuronal loss and edema was employed for this purpose. First, for the accumulation of the lipid peroxide, ferrous-citrate was microinjected to the unilateral region of caudate putamen, and second, for the neuronal loss of the brain, a neurotoxic substance, malonate, which inhibits intracellular respiratory chain and results in apoptotic cell death, was microinjected to the unilateral region of caudate putamen, and third, in the case of the edema, animals were challenged with brain injury by applying a liquid-nitrogen-cold probe to one side of the cerebral hemisphere for 15 s after removing the bony skull.

In the model of the accumulation of lipid peroxides, all MR images taken under conditions that T1-weighted imaging (T1 WI), T2-weighted imaging (T2 WI) and proton density weighted imaging (PWI) gave no difference in signal intensity between the ipsilatelal and contralatelal regions, although the lipid peroxides were accumulated 1.8 times in the ipsilateral region without neuronal loss over the contralateral region, indicating that the accmulation of lipid peroxides in the brain did not influence the MR signal intensity. In malonate-injected rats, the significant strong MR signal were observed under the T2 WI condition in the ipsilateral region compared with the contralateral region, whereas histological examination revealed that the neuronal cell loss occurred in the wide area of ipsilateral caudate putamen. Furthermore, in cold-induced edema model, it was demonstrated that the MR signal intensity under the T2 WI condition significantly increased in the probe-contact area of cerebral hemisphere and the administration of the paramagnetic contrast agent, gadolinium-diethylene triamine pentaacetic acid (Gd-DTPA), which was impermeable to blood-brain barrier, induced the increase of the MR signal intensity under the T2 WI condition. This meant that the dysfunction of blood-brain barrier occurred in cold-induced edema of rat brain and Gd-DTPA accumulated in this region, resulting in the contrast enhancement in MR image.

MRI observations of brain injuries in laboratory animals suggested that the neuronal loss, edema and dysfunction of blood-brain barrier but not the accumulation of lipid peroxides affected MR images under the high magnetic field T2 WI condition.