Protection mechanism of gadolinium trichloride against hemorrhagic brain injury

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We investigated the effect of gadolinium trichloride (Gd) on microglial polarization and neuronal injury after intracerebral hemorrhage (ICH). An *in vivo* mouse ICH model was prepared by intrastriatal microinjection of collagenase type VII. One day after ICH, the mRNA level of proinflammatory M1 microglial markers, such as inducible nitric oxide synthase (iNOS), increased. Anti-inflammatory M2 microglial markers arginase1 (M2a, c), Ym1 (M2a), and transforming growth factor-beta (M2c) increased 1 day after ICH, and chemokine CCL1 (M2b) increased after 3 days. Gd administration decreased these M1 and M2 markers. Arginase1 and iNOS protein levels also increased 3 days after ICH, and Gd decreased them because of the decrease of cell number due to apoptosis. Next, we investigated whether Gd had an anti-inflammatory effect in an ICH model. Three days after ICH, brain edema was formed, and the number of NeuN-positive cells, which indicates neuronal nuclei, decreased in the peripheral region inside the hematoma. Gd improved the edema, neuron loss, and behavioral abnormality, without affecting the hematoma size. Furthermore, Gd improved the mortality rate by ICH. Overall, Gd evoked M1 and M2 microglial apoptosis and had an acute protective effect after ICH.