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MTEP attenuated reactive gliosis and enhanced neuronal survival and functional recovery after hemorrhagic stroke

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Pathological metabotropic glutamate receptor 5 (mGluR5) signaling has been implicated in a number of neurodegenerative disorders. The expression of mGluR5 in reactive astrocytes and microglia has been shown to be increased in several acute and chronic neurodegenerative conditions but the functional relevance of mGluR5 inhibition and reactive gliosis and neuronal survival following intracerebral hemorrhage (ICH) have not been elucidated. In the present study, we aimed to investigate the 3-[(2-Methyl-4-thiazolyl) ethynyl]-pyridine (MTEP), a potent and selective mGluR5 antagonist effects on these aspects following stereotactic injection of collagenase type IV (0.0375 IU/1 µl of saline) into the right striatum inducing hemorrhagic stroke in mouse. Intraperitoneal (ip) injection of MTEP (3mg/kg) was administered once daily for three consecutive days starting at 2 h after collagenase infusion. We studied ICH induced cellular and molecular changes in acute (three days) and prolonged (28 days) cases and observed that ICH promoted hematoma volume and sensorimotor deficits. GFAP and pro-apoptotic proteins (Bax, Bcl-2 and active caspase-3) upregulation in ipsilateral white matter and inclined the number of GFAP+, Iba-1+ cells and declined NeuN+ cells in peri-hematoma areas of gray and white matter were recorded after ICH. FJC+ cells in ipsilateral gray and white matter were increased three days after ICH injury. MTEP mediated blockade of mGluR5 attenuated hematoma volume, reactive gliosis, neurodegeneration and pro-apoptotic proteins level subsequently enhanced neuronal survival and sensorimotor outcomes. Our study showed that mGluR5 inhibition reduced glial scarification and boosted neural circuit formation in the ipsilateral white matter. Taken together these observations indicated that inhibition of mGluR5 play a role in mediating neuroprotective and functional recovery after hemorrhagic stroke which required more study to uncover the underlying molecular mechanisms.

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