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Hypochlorous acid damages neurovascular unit during the cerebral ischemia/reperfusion injury in rats

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Reactive oxygen species (ROS) is a main cause of cerebral ischemia/reperfusion injury, and hypochlorous acid (HOCl) is a member of ROS, but its pathogenicity remains unknown. In present study, we detected the production of HOCl in microglial cells, neurons, vascular endothelial cells and astrocytes respectively in a trans well co-cultured system and rats after cerebral ischemia-reperfusion injury with a novel fluorescent probe: HKOCl-3; and we used a specific inhibitor 4-ABAH to inhibit the production of HOCl, by suppressing it's the catalytic enzyme of MPO, in order to study the effect of HOCl on the integrity of neurovascular unit (NVU) in ischemic stroke. Our results showed that microglia was the main cell type expressing HOCl and its catalytic enzyme MPO in central nervous system, and microglia derived MPO would transfer into the adjacent neurons and vascular endothelia cells to catalyze the production of HOCl, which then induced neuronal deaths and destruction of blood-brain barrier by damaging mitochondria in cells, while the MPO inhibitor 4-ABAH could significantly alleviate the damage of NVU after cerebral ischemia-reperfusion in-vitro and in-vivo. Our results indicate that HOCl may play an important role in the pathogenesis of cerebral ischemia/reperfusion injury by damaging NVU

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