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Intravenous dl-3-n-butylphthalide enhances hemodynamics and vascular plasticity but also improves learning and memory deficits and neuronal degeneration early after chronic cerebral hypoperfusion in rat

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Background: Chronic cerebral hypoperfusion (CCH) induces cognitive impairment, even though there was a compensative mechanism attempting to maintain optimal cerebral blood flow (CBF) to the brain. Preclinical studies and clinical trial showed that dl-3-n-butylphthalide (dl-NBP) is effective for cognitive deficits induced by vascular origin. However, it remains unclear whether it has the therapeutic effective on cerebral hemodynamics early after CCH.

Aim: The aim of this study was to investigate the effect of intravenous dl-NBP on the cerebral hemodynamics and cognitive deficits early after CCH induced by modified permanent occlusion of common carotid arteries (2VO) method model in rats.

Methods: Rats were treated with intravenous dl-NBP (5 mg/kg) daily for 14 days starting from the first day after permanent bilateral common carotid artery occlusion. Magnetic resonance imaging (MRI) techniques, immunochemistry, and Morris water maze were employed to the study.

Results: The CBF of the cortex and hippocampus dramatically decreased after bilateral common carotid artery occlusion (BCCAO), and returned to the pre-occlusion level from two to four weeks after BCCAO in dl-NBP-treated rats; in contrast, it remained lower level up to four weeks after BCCAO in vehicle-treated rats. At two and four weeks after BCCAO, dl-NBP-treated rats exhibited significantly reduced escape latency in the Morris water maze task. Meanwhile, at two weeks after BCCAO, the drugs increased the number of nestin-expressing stem cells, astrocytes and microvessels in the CA1 and CA3 subfields of the hippocampus and parietal association cortex, inhibited neuron apoptosis occurred in the cortex and hippocampus.

Conclusions: The results indicated that promoting neurogenesis and angiogenesis, inhibiting neuronal apoptosis might contribute to the improvement of intravenous dl-NBP on cerebral hypoperfusion and hypoperfusion-induced cognitive deficits. Therefore, intravenous dl-NBP has therapeutic potential for the treatment of improving cognitive or dementia caused by decrease of cerebral blood flow.

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