MOLECULAR NEURODEGENERATION

MEETING ABSTRACT Open Access

Gastrodin improves learning behavior in a rat model of Alzheimer's disease induced by intra-hippocampal Aβ 1-40 injection

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Background

Gastrodin extracted from the rhizome of *Gastrodia elata* Blume, a Chinese herbal medicine, has long been used for treating vertigo, general paralysis, epilepsy, tetanus, stroke and dementia. Although several reports have shown that gastrodin has neuroprotective effects to rescue lead-induced synaptic plasticity deficits in rat hippocampus, and hippocampal cell damage in cellular model of Alzheimer's disease induced by Aβ25-35, its effect on behaviors of rat model of Alzheimer's disease induced by intra-hippocampal Aβ injection is not studied yet.

Methods

Forty-one male adult Sprague–Dawley rats were randomly divided into groups: normal (n=10), sham operated (n=7, intra-hippocampal saline injection), saline (n=8, intra-hippocampal Aβ injection and then ig saline), gastrodin (n=8, intra-hippocampal Aβ injection and then ig gastrodin), huperzine A (n=8, intra-hippocampal Aβ injection and then ig huperzine A). One week after intra-hippocampal Aβ1-40 injection (5 μg in 1 μl PBS, bilaterally), gastrodin (200 mg/kg), huperzine A (300 μg/kg) or saline were administrated by ig for 27 days (*q.d.*). At end of gastrodin or huperzine A treatment, the 5-day Morris water maze test was performed to observe the learning and memory function of 5 groups of animals.

Results

Two-way ANOVA (repeated measures) was used to compare the difference of escape latency in place navigation trials among testing days or groups, and showed both differences among 5 test days and 5 groups were very significant (P<0.001). Bonferroni posttests indicated that the difference between normal and sham operated was not significant (P>0.05), but the latency in saline group was significantly longer than normal or sham operated groups (P<0.05 or P<0.01), suggesting establishment of rat model. Compared to other groups, the latency in gastrodin group at 2nd test day was significantly shorter than saline group (P<0.05), indicating learning improvement of rat model. However, the latency in huperzine A group was observed to be longer than normal group (P<0.01). In spatial probe trial after 5-day place navigation trials, difference of numbers to cross the assumed platform among 5 groups was not significant (One-way ANOVA, P>0.05).

Conclusion

Gastrodin may have therapeutic effect to improve learning behavior in rat model of Alzheimer's disease induced by intra-hippocampal Aβ1-40 injection by mechanism different from huperzine A.

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