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Berberine chloride can ameliorate the spatial memory impairment and increase the expression of interleukin-1beta and inducible nitric oxide synthase in the rat model of Alzheimer's disease

Feiqi Zhu and Caiyun Qian*

Address: The neurology department of the first affiliated hospital, Sun Yet-Sen University, Guangzhou, Guangdong province, 510080, PR China

Email: Feiqi Zhu - zfqzsu2004@yahoo.com.cn; Caiyun Qian* - qiancyzsu@yahoo.com.cn

* Corresponding author

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Abstract

Background: Berberine is the major alkaloidal component of *Rhizoma coptidis*, and has multiple pharmacological effects including inhibiting acetylcholinesterase, reducing cholesterol and glucose, lowering mortality in patients with chronic congestive heart failure and anti-inflammation etc. Thus berberine is a promising drug for diabetes, hyperlipemia, coronary artery disease and ischemic stroke etc. The present study was carried out to investigate the effect of berberine chloride on the spatial memory, inflammation factors interleukin-1 beta (IL-1beta) and inducible nitric oxide synthase (iNOS) expression in the rat model of Alzheimer's disease (AD) which was established by injecting Abeta (1–40) (5 microgram) into the rats hippocampuses bilaterally.

Results: The rats were given berberine chloride (50 mg/kg) by intragastric administration once daily for 14 days. The spatial memory was assayed by Morris water maze test, IL-1beta and iNOS in the hippocampus were assayed by immunohistochemistry and real time polymerase chain reaction (PCR). Intragastric administration of berberine significantly ameliorated the spatial memory impairment and increased the expression of IL-1beta, iNOS in the rat model of AD.

Conclusion: Berberine might be beneficial to AD by intragastric administration though it might exaggerate the inflammation reaction.

Background

AD is the most prominent dementia in senile population affecting approximately 5% of the over 65-year old populations, but the cause of AD remains largely unknown. In the pathogenesis of AD, the inflammation mechanism is seemed to play an important role [1]. The senile plaque is the hallmark of AD. The core of the senile plaque is the deposition of β-amyloid (Aβ) and the activated microglia and astroglia are around the senile plaque. In these glias, numerous inflammation factors including IL-1β, interleukin -6(IL-6), tumor necrosis factor-α (TNF-α) and iNOS etc, are overexpressed. These inflammation factors have been seemed to be neurotoxic. At the same time, some epidemiological studies demonstrated that long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) could prominently delay the onset of AD [2-5]. The current drugs for AD treatment including cholinesterase inhibitors (donepezil, rivastigmine and galanthamin) and N-methyl-D-aspartate (NMDA) receptor antagonist (memantine) which were approved by Food and Drug Administration of USA (FDA) are symptomatic treatment, but these drugs usually can not delay the devel-