Embelin Abrogates Amyloid-β-Induced Neurotoxicity and Prevents Cognitive decline in Rats: Possible Role of Hippocampal Neurochemistry

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May 6, 2020

Abstract

Backgound: Extracellular deposits of amyloid-β (Aβ) in neuronal synapse have been considered as a major hallmark of Alzheimer's disease. Purpose: Here in we have investigated the neuroprotective potential of embelin Aβ-42 induced neurotoxicity in rats. Material and Methods: Amyloid β1-42 oligomer was infused (3nmol/3µl) intracerebroventrically twice on day-1to induced Alzheimer's type dementia in rats. Spatial and non- spatial memory was assessed at different time intervals and terminally biochemical, neurochemical and neuroinflammatory parameters were determined in rat hippocampal brain tissue. One week following Aβ1-42 infused rats were treated with different doses of embelin (2.5, 5 and 10mg/kg/p.o.) till 21st day. Results: Amyloid β1-42 infusion produced significant deterioration in learning and memory while hippocampal tissue showed elevation in AChE activity, oxidative stress, and pro-inflammatory cytokine levels (TNF-α, IL-β etc.) and disturbed pattern of GABA/glutamate levels in Aβ1-42 infused rats. On the other side, embelin significantly attenuated Aβ1-42 induced cognitive deficit & other biochemical changes in rats. Embelin treated rats showed improved learning and memory was able to reduce the burden of hippocampal oxidative stress and pro-inflammatory cytokines and also restored the GABA/glutamate balance in rats. Conclusion and Implication: The pro-cognitive effect of embelin may be due to its antioxidant and anti-inflammatory actions. The observed results indicate the therapeutic potential of embelin in cognitive disorders. Key words: Alzheimer's disease; Embelin; Amyloid-β· Neuroprotection; Dementia; Cognitive deficit.

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