Curcumin attenuates inflammatory response in experimental model of chronic epilepsy

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Abstract

Evidence suggests that glial cells play a prominent role in inflammation in chronic epilepsy contributing to perpetuation of seizures and cognitive dysfunctions. The present study has been designed to evaluate the beneficial effects of curcumin, a polyphenol compound with pleiotropic properties, in chronic epilepsy. Kindled model of chronic epilepsy was induced by administering pentylenetetrazole (PTZ) at a dose of 40mg/kg, i.p, to rats every alternative day for 4 weeks. Curcumin was administrated to the animals at a dose of 100mg/kg, orally, half an hour before the treatment of PTZ. The effect of curcumin on PTZ-induced inflammation in chronic epilepsy was assessed in terms of astrocyte (GFAP) and microglia (Iba-1) activation markers and production of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and chemokines (MCP-1). PTZ-induced chronic epilepsy increased mRNA and protein expression of GFAP and Iba-1 in cortex and hippocampus suggesting activation of astrocytes and microglia. Immunohistochemical staining for GFAP and Iba-1 also showed higher number of astrocytes and microglia which were morphologically distinct. Concomitantly, there was an increased mRNA and protein expression for pro-inflammatory cytokines and chemokines in hippocampus and cortex. Administration of curcumin attenuated the activation of glial cells and suppressed PTZ-induced increased expression of GFAP and Iba-1 in both the regions of brain. Curcumin administration also suppressed the production of pro-inflammatory cytokines and chemokines. Based on the results we can conclude that curcumin attenuates activation of glial cells and hence suppresses hyperinflammatory condition in chronic epilepsy suggesting that curcumin could be considered as an adjuvant therapy in preventing recurrent seizures and cognitive dysfunctions in chronic epilepsy.