عنوان مقاله:

Nimodipine promotes neurite outgrowth and protects against neurotoxicity in PC12 cells

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خلاصه مقاله:

Objective(s): Nimodipine is an L-type voltage-dependent calcium channel (VDCC) antagonist. However, the actions of nimodipine except calcium blocking are poorly understood. This study aimed to investigate the effect of nimodipine on neurite outgrowth and neuroprotection in vitro. Materials and Methods: After PC12 cells were treated with different concentrations of nimodipine, neurite outgrowth was estimated using the ImageJ software. Neuroprotective effects of nimodipine against H2O2 and calcium ionophore-induced neurotoxicity were investigated using (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay. In addition, the activation of extracellular signal-regulated kinase (ERK) and cyclic AMP-response element-binding protein (CREB) pathway was investigated for clarifying the action mechanism of nimodipine. Results: Nimodipine treatment at doses of higher than 10 µM induced neurite outgrowth in the cells. Additionally, VDCC knockdown by siRNA significantly suppressed the nimodipine-induced neurite outgrowth in PC12 cells, suggesting that the drug promotes neurite outgrowth by binding to VDCC. H2O2 and calcium ionophore induce oxidative and calcium stress in PC12 cells. Nimodipine exhibited neuroprotective effects against H2O2- and calcium ionophore-induced neurotoxicity by increasing the mRNA expression levels of neurotrophic factors, calcium-binding proteins, and antioxidants that are transcribed by CREB activation. Conclusion: This is the first report that nimodipine induces neurite outgrowth and exerts its neuroprotective activity through the .ERK/CREB signaling pathway in PC12 cells

کلمات کلیدی:

calcium channels, MAP Kinase Signaling System, Neuronal outgrowth, Neuroprotection, Nimodipine