Count: 433
Abstract ID: 51
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Vitamin K2 can exert a protective effect against amyloid beta peptide induced PC12 cell toxicity

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Background and Aim : Alzheimer's disease (AD) is a major cause of amnesia and behavioral injuries in old people that characterized by a progressive loss in cognitive function. This study has been questioned the protective effect of vitamin K2 against toxicity and oxidative damage induced by Aβ in PC12 cells as an appropriate model of Alzheimer's cell damage.

Methods : PC12 cells were pretreated with vitamin K2 (5, 10, 20, 50 µM) for 24h and then exposed to Aβ (25 µM) for 48h. At the end, the cell survival, intracellular reactive oxygen species (ROS) production were assessed by analysis of cell viability, ROS generation and apoptosis in PC12 cells was measured by Flow cytometery after PI staining.

Results : Vitamin K2 (5, 10, 20, 50 µM) could decrease A? (25 µM) toxicity and showed significant difference compared to the A? group (*P<0.05). After exposure of cells to A? (25 µM) for 48 h, Vitamin K2 (5, 10, 20, 50 µM) decreased ROS. Cell apoptosis was significantly increased to 14.3% after treatment with A? (25 ?M) compared to control (3.7%). After pretreatment with vitamin K2 (20, 50 ?M); however, apoptosis was significantly reduced to (4.2%, 7.6%).

Conclusion : Our study revealed that Vitamin K2 may exhibit protective effect on the apoptosis induced by Aβ in PC12 cells, possibly by reducing oxidative reagents. Thus, vitamin K2 could be considered for more investigation as a potential treatment in AD patients.