Relationship between pain coping strategies with anxiety, depression and pain intensity in chronic back pain patients

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Introduction. Psychological factors play an important role in the treatment of diseases. The aim of this study was examine the relationship between pain coping strategies with anxiety, depression and pain intensity in patients with chronic back pain.

Materials and Methods. The research method was an ex-post facto. Sample size is consisted of 200 patients with chronic back pain (ages 20-60 yr) for more than two months, presenting to under evaluation clinics completed the Iranian adaptation to the coping strategy questionnaire, pain intensity scales, Beck Anxiety Inventory and Beck depression inventory second edition.

Results. The correlation coefficient between variables of study showed that some of the coping strategies were associated with severity of pain depression and anxiety. Some of coping strategy factors appears as the predictor of severe pain, depression and anxiety in a regression analysis as well; especially multi regression analysis revealed that more catastrophizing was associated with more severe pain, depression and anxiety. also, results indicated that not only catastrophizing in chronic pain was a significant and independent predictor of severe pain, but also, higher use of catastrophizing, even after controlling the severe pain, could predict depression and anxiety.

Conclusion. Maladaptive coping strategies are potentially modifiable and could be the target of treatment interventions to decrease depression, anxiety and therefore increase adaptability with chronic pain in patients.

Key words: pain coping strategies; pain intensity; chronic back pain

Reduction of chemical pain after intrathecal administration of vitamin K2 in the rats

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Introduction. Vitamin K2 is a derivative of vitamin K. Effect of vitamin K2 on the release and function of neurotransmitters remained unknown; but due to the effects of this substance in many neural processes, neurodegenerative diseases (e.g. Alzheimer, Parkinson, etc.) and inflammation, its influence on the mechanisms of neurotransmitter release and pain also can be involved. Therefore, this study examines the effect of intrathecal administration (i.t.) of vitamin K2 on the chemical pain.

Materials and methods. The adult male Wistar rats (250-300 g) initially were subjected to i.t. surgery as 8cm of PE-10 cannula was inserted into the spinal subarachnoid space. Groups were included: control, sham (salin+DMSO, i.t) and treatments consists of vitamin K2 (2, 10 and 20 µg/10µl, i.t). Five min after intrathecal administration, 0.05 ml of formalin %2/5 injected in the sub plantar region of the right hind paw and immediately rat's behavioral responses to pain were recorded for 60 min.

Results. Intrathecal administration of vitamin K2 (20-10-2µg/10µl) in the first and second phases of the formalin test showed a significant decrease in pain (P <0.001, P <0.01), the analgesic effect of dose (2µg/10µl) were higher than the others. The intermediate stage was also affected by vitamin and became longer but was not significant compared with the control.

Conclusion. Probably Vitamin K2 with indirect influence on the activity of NMDA receptors and inhibiting the release of mediators such as Brady kinin and SP, the enzyme inhibition of iNOS and reduction in the production of NO and its antioxidant property, reduces pain and inflammation. Also the effect of vitamin K2 in the blockage of sodium channels, possibly responsible for part of its analgesic function is in the spinal cord.

Keywords: Vitamin K2; Chemical pain; Intrathecal injection; Formalin test

The role of the ubiquitin proteasome system in Alzheimer's disease: Polymerization of human tau into filaments in the presence of peptides

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Microtubule-associated tau protein belongs to a family of intrinsically disordered proteins that promotes microtubule assembly and stability. Several studies have shown that pathogenesis of Alzheimer's disease is related to a decreased proteasome activity in cortex and hippocampus of the patient's brain. The deficiency in normal activities of ubiquitin proteasome system (UPS) and non-proteasomal proteases, causes partial degradation of neural misfolded proteins.

Based on the above studies, we were interested to investigate the effect of two peptides on recombinant human tau protein aggregation. Circular Dichroism data were shown that coincubation of tau protein in presence of these inducers, increased structural compaction of tau protein which was accompanied by significant secondary structural changes.

The results of tau fibril formation by ThT fluorescence and TEM assays were in agreement with far-UV CD assays. We suggest that structural and conformational characters of inducer are as important as charged distribution on anionic inducer molecules. To distinguish the mechanism of the effect of charge and structure of peptides on aggregation of tau protein further experiments would be required.

Keywords: Ubiquitin proteasome system; Misfolding; Aggregation; Tau protein; Alzheimer's disease