



THE EFFECTS OF PRECONDITIONED MESENCHYMAL STEM CELLS ON GASTRIC ULCER REGENERATION IN MICE

Zhunosova M.S.

National Center for Biotechnology, Nur-Sultan, Kazakhstan

zhunosova@biocenter.kz

Keywords: mesenchymal stem cells, preconditioning, cytokines, gastric ulcer, regeneration

Introduction: During the last decade, preconditioned mesenchymal stem cells (MSCs) have gained much attention in the field of cell therapy due to their capacity to differentiate into different cell types and to promote immunosuppressive effects in multiple diseases. However, there is little information, however, about the therapeutic effects of preconditioned MSCs in gastrointestinal disorders. In the present study, we examined whether transplantation of preconditioned MSCs could improve gastric ulcer healing.

Methods: MSCs were isolated from compact bone of C57BL/6 mice. MSCs were cultured in α -MEM containing 10% FBS and 1% antibiotics. Preconditioning of MSCs was performed with IFN- γ and TNF- α for 24 h. Mice were divided into 3 groups: group 1(PBS), group 2(MSCs) and group 3 (preconditioned MSCs). Induction of gastric ulcer was made with alcohol-acetic acid solution. Each stomach was isolated for macroscopical and histological analysis. The levels of vascular endothelial growth factor (VEGF) and prostaglandin E2(PGE2) in stomach ulcer tissue were measured with ELISA-kits.

Results: Our results showed that transplantation of preconditioned MSCs markedly improved the histopathology of the gastric tissue in comparison to MSCs and PBS-treated groups. Also the result of ulcer index was significantly decreased in preconditioned MSC-treated group at day 3 and 5 of the study comparing with the PBS-treated group. ELISA data showed that preconditioned MSCs restored the levels of PGE2 to the normal levels and significantly increased the VEGF levels.

Conclusion: Thus, our results showed that transplantation of preconditioned MSC significantly improved gastric ulcer healing in mice, possibly through the inhibition of inflammation and induction of angiogenesis in the gastric mucosa via the secretion of VEGF and PGE2.