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The Effects of Periostin in a Rat Model of Isoproterenol-Mediated Cardiotoxicity

Authors : Mahmut Sozmen, Alparslan Kadir Devrim, Yonca Betil Kabak, Tuba Devrim

Abstract : Acute myocardial infarction is the leading cause of deaths in the worldwide. Mature cardiomyocytes do not have the ability to regenerate instead fibrous tissue proliferate and granulation tissue to fill out. Periostin is an extracellular matrix protein from fasciclin family and it plays an important role in the cell adhesion, migration, and growth of the organism. Periostin prevents apoptosis while stimulating cardiomyocytes. The main objective of this project is to investigate the effects of the recombinant murine periostin peptide administration for the cardiomyocyte regeneration in a rat model of acute myocardial infarction. The experiment was performed on 84 male rats (6 months old) in 4 group each contains 21 rats. Saline applied subcutaneously (1 ml/kg) two times with 24 hours intervals to the rats in control group (Group 1). Recombinant periostin peptide (1 µg/kg) dissolved in saline applied intraperitoneally in group 2 on 1, 3, 7, 14 and 21. days on same dates in group 4. Isoproterenol dissolved in saline applied intraperitoneally (85mg/kg/day) two times with 24 hours intervals to the groups 3 and 4. Rats in group 4 further received recombinant periostin peptide (1 µg/kg) dissolved in saline intraperitoneally starting one day after the final isoproterenol administration on days 1, 3, 7, 14 and 21. Following the final application of periostin rats continued to feed routinely with pelleted chow and water ad libitum for further seven days. At the end of 7th day rats sacrificed, blood and heart tissue samples collected for the immunohistochemical and biochemical analysis. Angiogenesis in response to tissue damage, is a highly dynamic process regulated by signals from the surrounding extracellular matrix and blood serum. In this project, VEGF, ANGPT, bFGF, TGFβ are the key factors that contribute to cardiomyocyte regeneration were investigated. Additionally, the relationship between mitosis and apoptosis (Bcl-2, Bax, PCNA, Ki-67, Phopho-Histone H3), cell cycle activators and inhibitors (Cyclin D1, D2, A2, Cdc2), the origin of regenerating cells (cKit and CD45) were examined. Present results revealed that periostin stimulated cardiomyocye cell-cycle re-entry in both normal and MCA damaged cardiomyocytes and increased angiogenesis. Thus, periostin contributes to cardiomyocyte regeneration during the healing period following myocardial infarction which provides a better understanding of its role of this mechanism, improving recovery rates and it is expected to contribute the lack of literature on this subject. Acknowledgement: This project was financially supported by Turkish Scientific Research Council- Agriculture, Forestry and Veterinary Research Support Group (TUBİTAK-TOVAG; Project No: 114O734), Ankara, TURKEY.

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