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PROTECTIVE EFFECTS OF METFORMIN ON THE PROCESS OF ATHEROSCLEROSIS WITH AGING IN THE RAT HEART

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OBJECTIVE

For cardiovascular protection, many pharmacological agents provide clues as to where the protective effects about insulin resistance and metabolic syndrome associated with atherosclerosis remain the focus of interest. Although developed as an antidiabetic drug, *Metformin* is still up to date with effective research showing its positive contribution to many anti-inflammatory processes.

With this laborious study on the elderly rat heart, we hope that the protective effect of metformin in the process of atherosclerosis will make an effective contribution to larger-scale studies, particularly vascular pathologies.

METHODS

Wistar-Albino male rats were separated into four groups: young mice (< 12months-old), young mice & metformin, old mice (24months-old), old mice& metformin. Metformin was supplemented into drinking water at concentration of 100mg/kg for 8 weeks. The apical half of left ventricle was excised. The effects on cardiac tissue cells in aging rat was evaluated by expression analysis of proliferation(*Ki67*), oxidative stress(Superoxide Dismutase 1, *SOD1*) and inflammation (Transforming growth factor beta(*TGF-β*), Tumor necrosis factor - α (*TNF-α*), Interleukin-1β (*IL-1β*), Inducible nitric oxide synthase(*i-NOS*) markers. The expression of pericyte marker, *PDGFR-β* (Platelet derived growth factor receptor- β), was also estimated in tissue.

RESULTS

When compared to young tissue, expression of TGF-β, TNF-α and IL-1β were increased in tissue of aged tissue at rate of 1.89, 1.27 and 1.09-fold, respectively. Oral consumption of metformin by aged rat attenuated the expression of these inflammatory markers to levels of 1.41 (p=0.044), 1.03 (p>0.05) and 0.97-fold (p>0.05), respectively.

The SOD1 expression was increased in metformin groups. In young tissue, SOD1 expression was improved 1,89-fold (p=0.021) after metformin. Anti-oxidative capacity was slightly decreased in aged tissue (0.84-fold) when compared to young. SOD1-expression in old mice treated with metformin was improved to 2.67-fold (p=0.015). Cell proliferation in aged group with metformin was substantially improved (3.26-fold, p=0.007) compared to young animals. The i-NOS expression was also decreased after metformin treatment in aged animals, while no effect could be observed in young animals in both treated and non-treated groups. PDGFR-β expression level in young tissue was decreased slightly to 0.89-fold, while PDGFR-β expression was increased in aged tissue from 1.54-fold (p=0.056) to 1.78-fold (p=0.043).

CONCLUSIONS

Metformin supports pericytes and enhanced regeneration capacity, which deteriorated with aging. Metformin treatment strengthened antioxidant capacity and attenuated inflammation indicators in the aged group. Metformin may have a preventive effect on the process of atherosclerosis.

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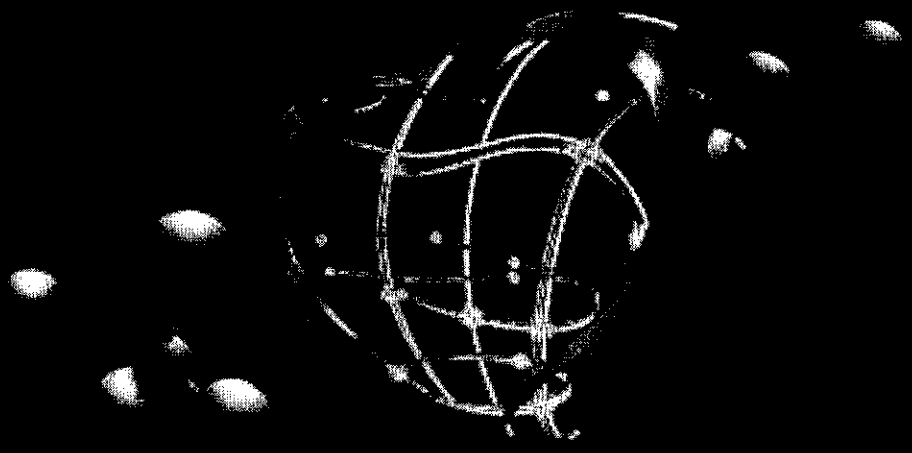
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Dear Dr. Özgür Barış,

It is my disticinct pleasure to inform you that you won one month scholarship at the "Hearth Center of Ruhr University in Bochum" by Evaluation of the Abstracts and Award Committee with the presentation of your abstract on "Protective Effects of Metformin on the Process of Atherosclerosis with Aging in the Rat Heart" at the 16th International Congress of Update in Cardiology and Cardiovascular Surgery which was held between 30 October-1 November 2020.

I cordially congratulate you for your scientific efforts and I am sure that your collabaration with UCCVS Congresses will continue even more strongly after completing your one month stay in Hearth Center of Ruhr University in Bochum.

Prof. Oztekin Oto, MD, FESC, FACC

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