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Glatiramer is Beneficial for Ischemic Hearts

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Ischemia is a condition in which blood flow is reduced, as opposed to hypoxia, which is characterized by a reduction in oxygen supply. Nonetheless, in clinical settings where ischemia and hypoxia coexist, the terms ischemia and hypoxia are used interchangeably. The institution of reperfusion, on the other hand, contributes to the enhancement of ischemia-induced myocardial injury, and the phrase ischemia reperfusion-induced myocardial injury is used to represent the harm caused by ischemia and reperfusion. Multiple mechanisms have been proposed to explain its positive effects, including a reduction in the release of inflammatory cytokines.

The closure of the left coronary artery in rats has been frequently used to induce ischemia in rats, and it represents an in vivo model of ischemia-reperfusion injury that is faithful to ischemia in humans. As a result, the current study was conducted to look into the cardioprotective effects of various Glatiramer dosages in a rat in vivo model of ischemia-reperfusion injury.

Given the importance of AKT12, GSK-3$13, TNF-"14, and Nrf2, a transcriptional factor that controls antioxidant activities in ischemia-reperfusion injury in rats, the current study looked into the role of AKT, GSK-3$, TNF-" and Nrf2 in Glatiramer-mediated beneficial effects in ischemia-reperfusion injury in rats.

The Study was carried out in the department of Cardiology, Shanxi Cardiovascular, China. Male Wistar Albino rats were used in the study with the approval of Shanxi hospital's approval committee. The left anterior descending coronary artery (LAD) was located and ligated for 10 min with a 5.0 silk suture to induce ischemia. The ligation was then removed, and blood flow was resumed in order to cause reperfusion damage. Rats were sacrificed after 120 min of reperfusion to obtain blood samples and hearts for further analysis. There were five groups and each group included 10 animals. On the other hand, non-ischemic groups were kept for 150 min and rats were sacrificed to collect blood and hearts.

Effect of Glatiramer on ischemia-reperfusion-induced cardiac injury: When the left coronary artery was ligated, there was a considerable increase in myocardial injury, which was measured by assessing the levels of cardiac injury-specific biomarkers. Treatment with Glatiramer (0.5, 1.0, and 2.0 mg kg^{-1}), on the other hand, significantly reduced the ischemia-reperfusion-induced rise in cTnT and CK-MB release in a dose-dependent manner. In Glatiramer-treated mice with doses of 0.5, 1.0, and 2.0 mg kg^{-1}, the levels of cTnT were reduced to 1746, 900, and 319 pg mL^{-1} while the levels of CK-MB were reduced to 1285, 870, and 672.5 U L^{-1}.

The injection of Glatiramer in three doses had cardioprotective effects and dramatically reduced the release of cTnT and CK-MB from the heart into the bloodstream in response to ischemia and

reperfusion injury in this investigation. In the ischemia reperfusion injury model in rats, it illustrates the cardioprotective effects of Glatiramer in a dose-dependent manner. Glatiramer is an immunomodulatory that has been used to treat remitting multiple sclerosis (RRMS). Apart from that, it has been shown to reduce CK-MB levels to 1285, 870, and 672.5 U L^{-1} in Glatiramer-treated animals with doses of 0.5, 1.0, and 2.0 mg kg^{-1} in ischemia reperfusion induced cerebral injury in non-diabetic and diabetic rats, as well as improve hypo perfusion induced cognitive dysfunction.

The other researchers were also unsuccessful in identifying the molecular mechanism behind Glatiramer-mediated cardio protection, which requires activation of the AKT-GSK-3$ signalling pathway. This research will aid in the discovery of Glatiramer cardioprotective properties in ischemic hearts.

JOURNAL REFERENCE

Zhang, Z., X. Ma, G. Yang and L. Zhang, 2022. Cardioprotective effects of glatiramer against ischemia-reperfusion injury in coronary artery ligation model in rats through activation of AKT-GSK-3$-TNF-"-Nrf2 signalling pathway. Int. J. Pharmacol., 18: 79-86.

KEYWORDS

Ischemia, hypoxia, myocardial injury, inflammatory cytokines, Glatiramer-mediated cardio protection