MicroRNA 103 inhibitor as a potential promising therapeutic target for myocardial infarction

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Myocardial infarction (MI) is myocardial cell death due to severe and prolonged ischemia produced from atherosclerosis-related coronary artery disease. MI triggers a cascade of events and reparative phases end with myocardial cell necrosis. MicroRNA (miR) is non-coding single stranded RNA that regulates protein expression. miR-103 is used to regulate expression of Fas-associated death domain (FADD) which decreases necroptosis of ischemic myocardium. The study aims to investigate the modulatory effect of up-regulating mRNAs translation processes of myocardial infarction induced with Isoprenaline HCL 100 mg/kg (ISO) by injecting miR-103 inhibitor. Eighteen mice (15-25 gm) were allocated into three groups; Group A (control) received normal saline, Group B received ISO and Group C received ISO and miR-103 inhibitor. Mice were sacrificed by cervical dislocation under urethane anesthesia. Blood and hearts samples were collected for biochemical analysis of miR103, FADD, receptor interacting protein kinase (RIPK), nuclear factor-kB (NF-kB), tumor necrosis factor-α (TNF-α), interleukine-6 (IL-6),Troponin-I and creatine kinase-MB (CK-MB). In addition, hearts were used for histopathological examination. Results showed that administration of miR-103 antagomir leads to increase in FADD protein levels in group C compared to A and B. While miR-103, RIPK, NF-kB, TNF-α and IL-6 showed high levels of expression in group B that is attenuated in group C. Troponin-I and CK-MB also supported the previous results. Histopathological test showed normal histological structure in groups A and C while focal degeneration in myocardium in B. Accordingly, these results indicate a promising suppression of MI manifestations upon inhibition of miR-103.

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