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

Mai A. Zaafan, Amr M. Abdelhamid, Ayman Selim, Amr Mohamed Alaa, Bana Ammar, Shahd Yehia, Al Hasnaa Abdel Tawwab, Asmaa Esmail, Heba Abdelhakim and **Yara Hamdy**
October University for Modern Sciences and Arts (MSA), Egypt

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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