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Aerobic exercise activates myocardial FGF21/FGFR1/PI3K-AKT signaling pathway and inhibits cardiomyocyte apoptosis in post-myocardial infarction rats

Mingxiao Li,Zhenjun TIan
Institute of Sports and Exercise Biology， Shaanxi Normal University， Xi'an， Shaanxi 710119

Objective To investigate the effect of aerobic exercise on the expression of fibroblast growth factor 21 (FGF21) and cardiomyocyte apoptosis in Myocardial Infarction (MI) rats.
Methods male SD rats were randomly divided into three groups： the sham operation (S), sedentary MI group (MI) and MI with aerobic exercise group (ME). The MI model was established by ligation of the left anterior descending branch of the left coronary artery. ME group were trained four weeks after the operation. LVSP, LVEDP and ±dp/dtmax were used to evaluate cardiac function. H9C2 cardiomyocytes were stimulated by 400 μmol/L H_2O_2 for 4h to simulate myocardial apoptosis mode. AMPK agonist AICAR and FGF21 receptor inhibitor PD166866 were used to interfere with H9C2. Myocardial collagen volume fraction was calculated by Masson staining and myocardium FGF21, FGFR1, Bax, Bcl-2 and PI3K-AKT pathway by western blotting or RT-Qpcr. Cardiomyocytes apoptosis was evaluated by TUNEL.
Results Compared with S, the expression of FGF21, FGFR1, Bax, Bcl-2 and PI3K, AKT increased significantly in MI group, the apoptotic cardiomyocytes and collagen fibers increased significantly, but the cardiac function decreased. Compared to MI, myocardium FGF21, FGFR1 and PI3K, AKT were further increased in ME group, the Bax/Bcl-2 and the apoptotic cardiomyocytes decreased significantly. The percentage of collagen fibers decreased and the cardiac function was improved. Myocardium FGF21 was positively correlated with the inhibition of cardiomyocyte apoptosis and the improvement of cardiac function. Furthermore, the expression of Bax/Bcl-2, TNF-α/IL-10 and the apoptotic cardiomyocytes was significantly increased by PD166866, but PI3K-AKT pathway decreased significantly by PD166866. However, AICAR single intervention or PD166866 simultaneous intervention also can reverse this adverse effects.
Conclusions Exercise can increase myocardial FGF21/FGFR1 with MI. The one of the mechanisms is to activate PI3K-AKT pathway to inhibit cardiaomyocyte apoptosis and inflammatory. It indicates that FGF21/FGFR1/PI3K-AKT signaling pathway plays an important role in inhibiting myocardial apoptosis and improving cardiac function.