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MEETING ABSTRACT Open Access

The role of AMPKα2 in cardiomyocytes anoxia/reoxygenation injury mediated by Cl^-

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Background

During anoxia/reoxygenation (A/R) injury, intracellular chloride ion concentration ($[Cl^-]_i$) homeostasis may play a role in maintaining the normal physiological function of cardiomyocytes. The cells protection was induced by Cl^--free when they were subjected to A/R injury, and we have found involvement of AMP-activated protein kinase-α2(AMPK-α2) in A/R injury. In the current study we investigated the mechanism of Cl^--free induced protection agaist A/R injury in H9c2 cells.

Methods

AMPKα2 shRNA recombinant plasmid was contructed by using pSuper.Retro vector. The H9c2 cells were randomly divided into five groups: (1) Control group; (2) A/R group; (3) removal of extracellular Cl^-+A/R group (Cl^--free A/R group); (4) pSuper+ Cl^--free A/R group; (5) pS-AMPKα2+ Cl^--free A/R group. The AMPKα2 protein expression was detected by western blotting. The activity of LDH was determined by auto-biochemistry analysator. Cells viability was analyzed by MTT, MDA, SOD and GSH-Px activity in H9c2 were detected by kits. The level of intracellular ROS, the percentage of apoptosis and the mitochondria membrane potential were measured by flow cytometry.

Results

AMPKα2 shRNA recombinant plasmid was contructed successfully. A/R injury obviously decreased H9c2 cell viability, activity of SOD and GSH-Px. Cl^--free A/R group has been shown to produce a protective effect against A/R injury by increasing antioxidant enzyme. Once knockdowning the level of AMPKα2, the protective effect against A/R injury mediated by Cl^--subsitution apparently disappeared. Its cell viability, activities of SOD and GSH-Px, the mitochondria membrane potential were decreased while LDH activity and the level of ROS and apoptosis were remarkably increased in H9c2 cells compared with Cl^--free A/R group ($p<0.01$). There was no significant difference between Cl^--free A/R group and pSuper+ Cl^--free A/R group.

Conclusion

AMPKα2 participated in the protective effect against A/R injury produced by administration with Cl^--free, and shAMPKα2 could abolish the protective effect. The mechanism underlying Cl^--free agaist A/R injury is mainly that of low $[Cl^-]_i$ attenuate oxidative stress by AMPKα2.

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