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EFFECT OF SGLT1 INHIBITION IN HEART ISCHEMIC REPERFUSION INJURY

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Prevalence of cardiac ischemia is rapidly increasing day by day in the world. Among all the cardiovascular pathologies, ischemic heart disease has been shown to be the most prevalent in the 21st century. Restoration of blood supply to the myocardium during ischemia reperfusion leads to the cell damage. These events include arrhythmias, vascular damage and no-reflow, myocardial functional stunning, cell swelling and apoptosis. Here our main objective was to investigate the effect of phlorizin, SGLT1 inhibitor, in cobalt chloride ($CoCl_2$) induced hypoxia on myocardial apoptosis. For these three different models i.e. *ex-vivo* I/R model on Langendorff apparatus were taken. $CoCl_2$ induced hypoxia in SGLT1 overexpressed H9C2 cells and SGLT1 knocked down neonatal cardiomyocytes by siRNA. We found that among all the three models there were significant decrease in oxidative stress parameters in I/R as well as in I/R+PZ group. Gene expression studies also have shown more apoptotic effect in both I/R and I/R+PZ group. Rhodamine 123, Hoechst 33342 and annexin V/PI staining studies also have shown more depolarization in cell membranes in hypoxia as well as hypoxia + phlorizin groups among all the three models along with pyknotic and condensed nuclei. No recovery has been seen after phlorizin treatment. Flow cytometry studies has shown more cells in early and late apoptotic phase in hypoxia group as well as phlorizin treated hypoxia group. More apoptosis has been seen in phlorizin treated cardiomyocytes which were knocked down by SGLT1 siRNA. The blunted effect of phlorizin has been seen in I/R injury. SGLT1 inhibition has any effect on other glucose transporters have to be seen. In conclusion, phlorizin doesn't show any preventive effect in this study and inhibiting SGLT1 or knocking out SGLT1 is not a preventive approach in ischemia reperfusion injury.

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