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POSSIBLE INVOLVEMENT OF POLY (ADP-RIBOSE) POLYMERASE (PARP) IN THE CARDIOPROTECTIVE POTENTIAL OF ISCHEMIC POSTCONDITIONING IN RAT HEART

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Objective The present study has been designed to investigate the possible role of Poly (ADP-ribose) polymerase (PARP) and protein kinase C-delta (PKC-δ) in myocardial ischemic postconditioning (IPOST).

Methods Hundred male wistar rats were divided into 8 groups randomly: ischemic /reperfusion in diabetic rat (D+IR), ischemic postconditioning in diabetic rat (D+IPOST), ischemic/reperfusion in drug-treated diabetic rat (D+IR+PJ34), ischemic postconditioning in drug-treated diabetic rat (D+IPOST+PJ34) and their respective corresponding groups in the normal rats (C+IR, C+IPOST, C+IR+PJ34 and C+IPOST+PJ34). Diabetic rats were induced by streptozotocin (STZ) injection (55 mg/kg). Ischemia and reperfusion were induced by occlusion and reperfusion (I/R) of the left anterior descending artery (LAD). All the rats were subjected to ischemia for 30 min followed by reperfusion for 180 min. And three episodes of occlusion and reperfusion (10 s each) at the early beginning of reperfusion were induced to make IPOST. Myocardial infarct size was assessed macroscopically using triphenyltetrazolium chloride (TTC) staining and the number of apoptotic cells was determined using terminal deoxynucleotide nick-end labelling (TUNEL) method. We examined the expression of PARP and PKC-δ by the means of western blotting.

Results After eight weeks of STZ treatment, the rats in group D+IR have a larger infarct size (56.00±3.17%) and apoptosis index (42.51±2.69%) than that in the corresponding control group (C+IR) (44.00±2.62%, 33.12±2.74%). Ischemic postconditioning can efficiently decrease the reperfusion injury in the group C+IPOST, while failing to do so in the DM rats. Treated with PJ34, a specific PARP antagonist, DM rats can partially restore the cardioprotection. Western blot shows a positive correlation between PARP/PKC-δ and reperfusion injury.

Conclusion This study demonstrated that PARP is an important protein, which participates in the cardioprotection mediated by ischemic postconditioning. DM rats have worse results than the nondiabetic heart in the condition of ischemic-reperfusion. Ischemic postconditioning, which is efficient to limit the infact size and reperfusion injury in the NDM heart, diminished in the DM heart possibly due to impaired down-regulation of PARP and PKC-δ.