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PI3K/AKT/ENOS/HSP70 MEDIATES ATORVASTATIN POST-CONDITIONING AGAINST MYOCARDIAL ISCHAEMIA-REPERFUSION INJURY IN TYPE 2 DIABETIC RATS

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Objectives group
Methods group

Results Compared with I/R group, different dosage (0.1, 0.5, 1.0 or 2.0 mg/kg) of atorvastatin intervention significantly decreased IS ($p<0.01$, each dosage), cTnT ($p<0.01$, each dosage) and Flameng score ($p<0.05$, each dosage), and increased myocardial expression of phosphorylated AKt ($p<0.05$, each dosage), phosphorylated eNOS ($p<0.05$, each dosage) and HSP70 ($p<0.05$, each dosage), with a dose-dependent manner. As compared to I/R group, LY294002 intervention (PI3KI group, combined intervention of atorvastatin and LY294002) abolished atorvastatin-afforded cardio protection, with no significant differences in IS ($p>0.05$), cTnT ($p>0.05$), Flameng score ($p>0.05$) as well as myocardial expression of phosphorylated AKt ($p>0.05$), phosphorylated eNOS ($p>0.05$) and HSP70 ($p>0.05$) between groups.

Conclusions Our data suggests that activation of the upstream PI3K-Akt-eNOS pathway and up regulation of the downstream protein HSP70 contribute to atorvastatin post conditioning cardio-protection in type 2 diabetic rat.