Endoplasmic reticulum stress pathway involvement in local and remote myocardial ischemic conditioning.

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Résumé en anglais	Remote ischemic perconditioning (RIPer) and local ischemic postconditioning (IPost) are promising methods to decrease ischemia-reperfusion injury. We tested whether these two methods were effective in reducing infarct size through activation of endoplasmic reticulum (ER) stress response, a potential survival pathway. Rats exposed to myocardial ischemia-reperfusion were allocated to one of six groups: control, no intervention at myocardial reperfusion; IPost, three cycles of 10-s coronary artery occlusion followed by 10-s reperfusion applied at the onset of myocardial reperfusion; RIPer, 10-min limb ischemia followed by 10-min reperfusion initiated during coronary artery occlusion; control + 4-PBA, injection of ER stress inhibitor 4-phenylbutyrate (4-PBA) 1 h before coronary occlusion; IPost + 4-PBA; and RIPer + 4-PBA. Infarct size was significantly reduced in IPost and RIPer groups (33.32% ± 3.65% and 21.86% ± 3.98%, respectively) compared with the control group (54.86% ± 6.01%, $P < 0.05$). Western blot analysis of GRP78 (glucose-regulated protein) level and cleaved activating transcription factor 6, two ER stress markers, demonstrated an enhancement of ER stress response in IPost group but not in RIPer group at 15-min reperfusion. Furthermore, 4-PBA abolished cardioprotection induced by IPost (infarct size 53.75 ± 3.49 vs. 33.32 ± 3.65%, $P < 0.05$) but not by RIPer (28.80 ± 10.45% vs. 21.86 ± 3.98%, not statistically significant). GRP78 and cleaved activating transcription factor 6 levels were no longer increased in IPost group after 4-PBA. These findings point to a role for ER stress response in cardioprotection against reperfusion injury in IPost but not RIPer, suggesting differences in cardioprotective mechanisms between local and remote conditioning.
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