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Background:

We hypothesized the SDF-1 α /CXCR4 pathway is directly involved in the cardioprotective effect of ischemic-postconditioning (IPOC).

Method:

Isolated rat hearts were subjected to 30min of regional ischemia and 2hr of reperfusion. IPOC was induced by 6-cycles of 10sec-reperfusion/10sec-ischemia. AMD3100 was applied in IPOC-induced hearts. Infarct size, functional recovery and cardiac enzymes leakage were assessed. The phosphorylation of ERK1/2 and Akt was determined by Western blots.

Results:

IPOC reduced infarct size from 29.3 \pm 8.4% to 17.8 \pm 7.0% of the risk area (p=0.022 vs. CON). AMD3100 attenuated the infarct-reducing effect by IPOC (28.5 \pm 8.9%, p=0.045 vs. IPOC). LDH and CK were lower in the IPOC group (p=0.02 vs. CON and p=0.048 vs. CON) and reversed by AMD3100 (P=0.047 vs. IPOC and 0.042 vs. IPOC). ERK1/2 and Akt phosphorylation was increased by IPOC (226.1 \pm 71.8%; p=0.01 vs. CON and 296.4 \pm 93.1%; p=0.02 vs. CON) and blocked by AMD3100 (83.8 \pm 28.0%; p=0.02 vs. IPOC and 138.3 \pm 28.5%; p=0.009 vs. IPOC).

Conclusion:

The SDF-1 α /CXCR4 signaling is involved in the cardioprotection by IPOC and this pathway couples to the ERK1/2 and Akt pathways.

Figure 1

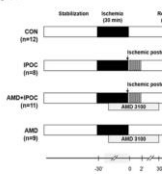


Figure 2

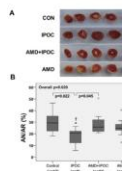


Figure 3

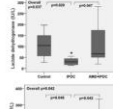


Figure 1

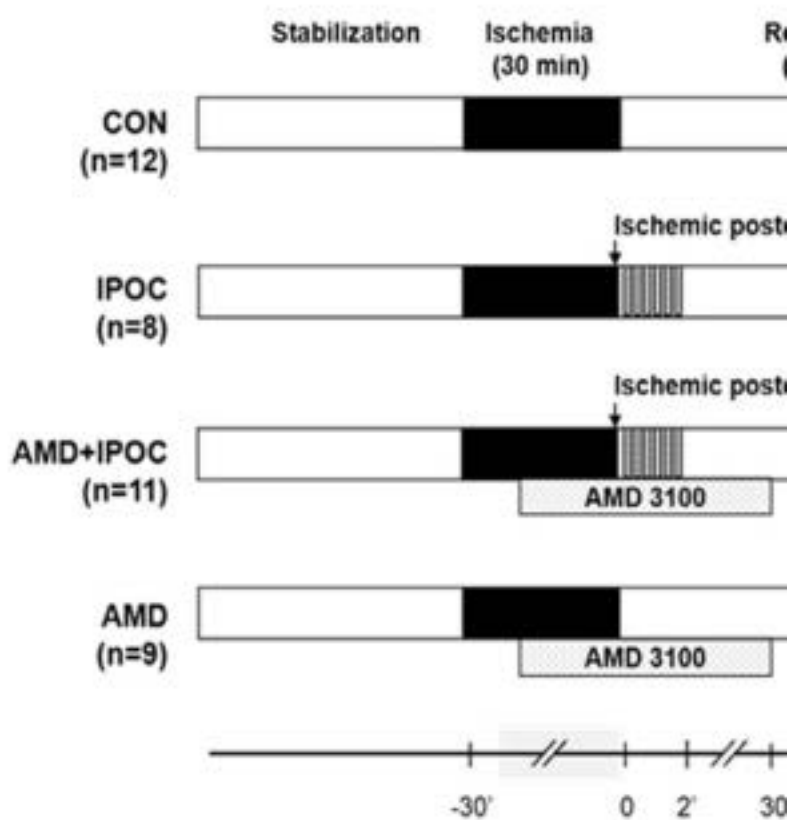


Figure 2

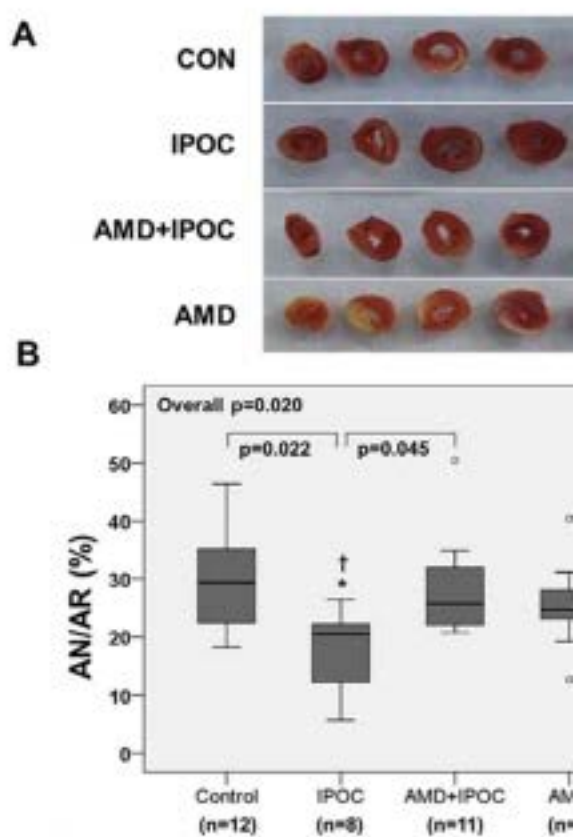


Figure 3

