



Letter to the Editor

The role of remote ischemic preconditioning beyond myocardial infarction size reduction



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Dear Editor,

We have read an experimental study entitled “Remote ischemic preconditioning attenuates adverse cardiac remodeling and preserves left ventricular function in a rat model of reperfused myocardial infarction” written by Pilz and his colleagues [1]. They demonstrated that anti-remodeling and anti-inflammatory effects of RIPerc are through activating Neuregulin-1 (NRG-1)/ErbB3 signaling pathway. Moreover, upregulation of NRG-1 is associated with a significant decrease in influx of CD68+ macrophages and subsequently the expression of inflammatory cytokines and matrix metalloproteinases, all of which played a pivotal role in the remodeling of the reperfused myocardium.

In addition, Wei et al. [2] found that remote preconditioning (PostC) after myocardial infarction (MI) significantly reduced macrophage infiltration and inflammation levels in rats. The biggest difference between RIPerc and PostC is that RIPerc is performed earlier [3]. Recently, an interesting randomized controlled trial demonstrated that the combination of remote ischemic conditioning (RIC) and PostC significantly reduced the incidence of new congestive heart failure in patients with ST-elevation myocardial infarction (STEMI), indicating that it may be related to the attenuation of adverse cardiac remodeling and preservation of left ventricular function [4]. Taken together, the

combination of RIC and PostC has shown cardioprotective effect on STEMI patients, especially on cardiac function. However, the mechanism remains to be further studied.

First of all, the mechanism of RIPerc affecting the expression of NRG-1 and ErbBs remains unclear. Second, the time and mode of RIPerc performed will also affect the results. In general, strengthening the understanding of RIC signal transduction mechanism and exploring its best mode will bring more benefits to clinical patients!

Declaration of Competing Interest

None.

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