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μ-CALPAIN REGULATE CARDIOMYOCYTES APOPTOSIS VIA APOPTOSIS INDUCING FACTOR AND BID IN RAT HEART ISCHAEMIA/REPERFUSION INJURY

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¹Song Zhao-Feng, ²Chen Dong-Yu, ¹Du Bo, ¹Wang Li-Zhen, ¹Wang Fan, ¹Qi Xin-Yan, ¹Zhou Qing-Hua, ¹Song Zhao-Feng. ¹Department of Cardiology, Taian Central Hospital, 271000 Taian, Shandong Province, China; ²Department of Rheumatology, Taian Central Hospital, 271000 Taian, Shandong Province, China

Objectives Heart I/R may exacerbate myocardial injury by the release of massive of reactive oxygen and nitrogen species. Calpain has been proposed to play a role in the pathogenesis of heart I/R injury. However, the underlying mechanisms are still not thoroughly understood. Here we investigated the effect of μ-Calpain on heart I/R injury and elucidated the underlying mechanisms.

Methods Studies were performed with I/R rats' hearts. MDL-281703, a μ-Calpain inhibitor, was used to inhibit the activate of μ-Calpain. The cardiac function and cells apoptosis were detected. The activation of μ-Calpain, Bid and Apoptosis-inducing factor (AIF) were evaluated during the I/R protocol.

Results During the I/R protocol, the 76 kDa size of fragment, which was active fragment of μ-Calpain, significantly increased. In the same time, the expression of AIF in nuclear and Bid also increased. Administration of MDL-281703 decreased cells apoptosis from 36±3.9% to 25±7.1% ($p<0.05$) and simultaneously improved the cardiac function. Administration of MDL-281703 also reduced the expression of Bid and attenuated mitochondrial-nuclear translocation of AIF.

Conclusions Our results suggested that μ-Calpain played a role of cells apoptosis in rats heart I/R injury. In this process, μ-Calpain regulate cardiomyocytes apoptosis via both caspase-dependent pathway (Bid) and non-caspase dependent pathway (AIF).