[gw22-e0660]

THE RECOMBINANT 53-KDA PROTEIN OF TRICHINELLA SPIRALIS ATTENUATE CARDIAL DYSFUNCTION ON POLYMICROBAL SEPSIS IN MICE

Xu Jia[1], Cairui Bin[1], Xiong Yan[1], Ye Zi[1], Zhan Wei[1], Xu-Chu Hu[21]First Affiliated Hospital of Zhongshan University, Guangzhou, China; [2]Zhongshan University, Zhongshan Medical College, Guangzhou, China

10.1136/heartjnl-2011-300867.60

Aims In present study, we aimed to investigate the contribution of recombinant Trichinella spiralis 53-KDa protein on myocardial injury and dysfunction during polymicrobal sepsis in BALB/c mice.

Methods BALB/c mice were treated subcutaneously with 50 μg rTsP53 three times at an interval of two weeks. Sepsis was induced by cecal ligation puncture. Mortality, macroscopic and microscopic changes were evaluated. The ability of rTsP53 to reduce pro-inflammatory NF-Kappa B signalling pathway and expression of inflammatory cytokines were investigated in primary cardiomycocytes, as well as in the mouse myocardium in vivo. The ventricular function was evaluated in mice by a conductance pressure-volume catheter. To determine immune response provoked by rTsP53, we measured specific IgG1 and IgG2a values against rTsP53 in sera of mice.

Results RTsP53 reduced significantly the total mortality of septic mice and the macroscopic and microscopic changes. IgG1 was the predominant specific antibody detected in the sera of immunised mice, but not IgG2a, indicating a highly polarised Th2-type immune response triggered by rTsP53. In vitro and in vivo, rTsP53 attenuated cardiac activation of NF-KappaB, expression of inflammatory cytokines (TNFα, IL-6 and IL-1β), and improved a state of myocardial dysfunction, characterised by increased ejection fraction and end-systolic elastance.

Conclusion RTsP53 is a potential protective agent for sepsis to cause a Th2 bias immune response, decrease mortality and inflammatory cytokines, and to improve mycocardial contractility.