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TREATMENT WITH A NEUTRALISING ANTI-MURINE INTERLEUKIN-17 ANTIBODY AFTER THE ONSET OF COXSACKIEVIRUS B3-INDUCED VIRAL MYOCARDITIS REDUCES MYOCARDIUM INFLAMMATION

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Background Recently, some studies indicated that interleukin (IL)-17, known as a T cell (Th17)-derived proinflammatory cytokine, is the major mediator of tissue inflammation in inflammatory and autoimmune diseases. Viral myocarditis (VMC) is a T cell-mediated autoimmune disease, but the role for IL-17 in VMC is not well defined.

Results Using IL-17 monoclonal antibody (IL-17 mAb)-treated VMC mice, we tested the pathogenic role of IL-17 in the development of VMC. VMC mice were treated with monoclonal rat anti-murine IL-17 antibody (anti-IL-17) or rat IgG_{2A} isotype control or phosphate-buffered solution 3 days after Coxsackievirus B3 (CVB3) injection. Normal mice without any manipulation were taken as normal control. The survival rates of mice were monitored and heart pathology was examined histologically. IL-17, IL-6, and TNF α mRNA of the myocardium were assessed by semi-quantitative RT-PCR. Systemic IL-17, IL-6, and TNF α level were measured by enzyme-linked immunosorbent assay, and local myocardium IL-17 expression was analysed using immunohistochemical staining. Flow cytometric analysis was used to evaluate the frequencies of Th17 subsets in CD4⁺T cells. Results showed that neutralisation of IL-17 with anti-IL-17 can ameliorate clinical symptoms, defer disease course, decrease serum IL-17 level, without declining the IL-17, IL-6 and TNF α mRNA transcript level and serum IL-6, TNF α level. The differentiation and proliferation of the Th17 cells were unchanged.

Conclusions Our data suggest that IL-17 is crucially involved in the pathogenesis of murine VMC, IL-17 inhibition might ameliorate the myocardium inflammation after the onset of VMC.