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论著

日本血吸虫感染小鼠肝脏IL-2、TNF-α的表达及注射该因子后对肝纤维化的影响

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摘要

目的探讨小鼠感染日本血吸虫后不同时期肝脏白细胞介素2(IL-2)和肿瘤坏死因子(TNF-α)的表达水平及注射该因子后对肝纤维化的干预。方法小鼠感染血吸虫尾蚴后分3组,每组16只,其中2组于感染6wk后分别隔日注射(ip)IL-2和TNF-α连续4wk,另设未感染正常鼠为对照组,采用ABC免疫组织化学技术,利用多媒体病理图文定量分析,动态观察相关因子活性。结果感染未处理组小鼠肝脏中IL-2和TNF-α含量随感染时间(8、11、14、18wk)延长而缓慢下降,而感染6wk后经腹腔注射IL-2或TNF-α组小鼠则随着相应因子的补充而显著上升,末次注射后1～8wk,肝内IL-2或TNF-α水平明显高于感染组和正常对照组(P<0.01),肝组织肉芽肿炎症反应及纤维化程度较对照组减轻。结论小鼠6wk后(成虫排卵后)给予外源性IL-2或TNF-α注射,能诱导相应细胞因子表达增强,并有减轻肝脏炎症和肝纤维化的表现。

关键词 日本血吸虫 IL-2 TNF-α 肝纤维化

分类号

Expression of IL-2 and TNF-a in the Liver and the Effect of Injection of These Cytokines on Liver Fibrosis in Mice Infected with Schistosoma japonicum

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Abstract

Objective To detect the expression of IL-2 and TNF-α in the liver at different period postinfection of Schis-tosoma japonicum and their effect on liver fibrosis after supplementary injection of these cytokines. Methods Mice were infected with schistosome cercariae and divided into 3 groups. Two groups were injected (ip) every other day with IL-2 and TNF-α respectively for consecutive 4 wk. The third group and an uninfected group of normal mice were regarded as control. The ABC immunohistochemistry and pathologic image multimedia quantification system were applied to detect activity of IL-2 and TNF-α. Results The level of IL-2 and TNF-α in the liver in infected but untreated group slowly decreased (from 8, 11, 14 to 18 wk). The supplementary injection of the cytokines at 6 wk postinfection in the two groups increased the cytokines significantly, the level of IL-2 or TNF-αwas higher at 1 - 8 wk after the last injection than that of both infected and uninfected control groups (P<0.01). The granulomatous inflammation and fibrosis in the livers of the two groups were slighter than that of the control. Conclusion At the 6th wk postinfection with egg deposition, exogenous supplementation with TNF-αor IL-2 induces enhanced expression of the two kinds of cytokines, corresponding to a diminished degree of the liver granulomatous inflammation and fibrosis.

Key words Schistosoma japonicum IL-2 TNF-α liver fibrosis

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