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论著

γ干扰素治疗对日本血吸虫病肝纤维化小鼠转化生长因子β1及其受体的影响

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摘要

目的 γ干扰素 (IFNγ)治疗日本血吸虫病肝纤维化小鼠 ,观察小鼠肝组织转化生长因子β1(TGFβ1)及其I、II型跨膜受体(TβRI、TβRII)表达的变化,同时动态观察日本血吸虫病小鼠肝纤维化形成过程中TGFβ1、TβRI及TβRII的变化趋势。 方法 日本血吸虫尾蚴感染BALB/c小鼠,16周后随机分为模型组、吡喹酮治疗组和吡喹酮联合IFNγ治疗组,治疗8周。于感染后8、12、16周和疗程结束后进行肝组织病理学检查;免疫组织化学法检测TGFβ1、TβRI及TβRII表达部位;逆转录 聚合酶链反应(RTPCR)检测小鼠肝组织TGFβ1、TβRI及TβRII转录水平。 结果 TGFβ1、TβRI和TβRII在小鼠肝细胞、窦周细胞中均有表达,随感染时间的延长,表达增强,特别在虫卵肉芽肿周围;IFNγ治疗后,虫卵肉芽肿减少,TGFβ1、TβRI及TβRII表达较治疗前降低;TGFβ1mRNA在感染后12周表达开始上调,模型组达高峰(P<0.05),经吡喹酮联合IFN γ治疗后降至正常水平;TβRIImRNA在感染后8、16周表达下调(P<0.05),疗程结束后恢复正常;TβRImRNA表达水平在发病和治疗过程中均无明显变化。 结论 TGFβ1表达上调和TβRIImRNA表达下调促进肝纤维化形成,IFNγ抑制TGFβ1的分泌的机制可能是通过下调TGF

关键词 日本血吸虫病 肝纤维化 转化生长因子 受体 治疗

分类号

Influence of InterferonγTreatment on Expression of TGF-β1 and Its Receptors in Liver Fibrosis of Mice with Schistosomiasis japonica

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Abstract

Objective To investigate the effect of interferon-γ(IFN-γ)on the expression of TGF-β1 and its two membrane receptors - TGF-β receptor I(TβRI), TGF-β receptor II(TβRII), and observe the expression of TGF-β1, TβRI and TβRII during the development of liver fibrosis in BALB/c mice infected by Schistosoma japonicum. Methods BALB/c mice, aged 6-8 weeks, were infected with cercariae of S. japonicum. The infected mice were divided randomly into three groups 16 week after infection:model group,praziquantel group and praziquantel combined with IFN-γ group. Liver specimen were obtained at 8, 12, 16 week and at the end of treatment. Pathological examination, immunohistochemistry and RT-PCR were used to evaluate the pathological change, the expression of TGFβ1, TβRI and TβRII and the mRNA level respectively. Results The expression of TGF-β1, TβRI, and TβRII can be detected in infected mice, while the expression around egg granulomas enhanced along with the progress of the disease. With the therapy of IFN-γ, the reduction of egg granulomas, and of the expression of TGF-β1, TβRI and TβRII was observed. From the transcription level, it was found that TGF-β1 mRNA increased at 12 week and peaked at model group, then decreased to the normal level after treatment with IFN-γ combined with praziquantel. The level of TβRII mRNA reduced at 8 and 16 week and returned to normal at the end of treatment. More interestingly, TβRI mRNA remained at the normal level on the whole course both in the development of fibrogenesis and the period of treatment. Conclusion The up regulation of TGFβ1 and down regulation of TβRII mRNA may induce liver fibrogenesis and IFN-γ might suppress TGFβ1 to reverse fibrosis. The mechanism of the suppression is mediated by down regulation of expression of its two receptors at protein level but not by influencing the mRNA expression.

Key words Schistosomiasis japonica Liver fibrosis Transforming growth factor-β1 Receptor Therapy

扩展功能

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