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四逆散加味抗大鼠肝纤维化作用机制

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中文摘要:目的: 探讨四逆散加味对肝纤维化的防治作用机制。 方法: 80只Wister大鼠随机分为8组:正常对照组、病理模型组、四逆散组、四逆散加味高、中、低剂量组、四逆散预防组。除正常对照组外,其余各组均采用猪血清ip诱发肝纤维化,0.5 mL/只,2次/周,连续10周,5周后即可形成肝纤维化。预防组于造模同时给药(以四逆散加味7 g·kg^{-1}),各治疗组于造模第6周给药,连续4周。四逆散组4 g·kg^{-1},四逆散加味高、中、低剂量组(14,7,3.5 g·kg^{-1})。采用酸性水解法检测肝组织羟脯氨酸(HYP)含量;放射免疫法(RIA)检测肝组织白介素-1(IL-1)和肿瘤坏死因子-α(TNF-α)含量;原位杂交、免疫组化法分别检测肝组织转化生长因子-β_1 (TGF-β_1)和α-平滑肌肌动蛋白(α-SMA)mRNA与蛋白表达。 结果: 与模型组比较,四逆散加味中剂量组大鼠肝组织羟脯氨酸(478.32±42.35) *vs* (327.09±39.41) μg·L^{-1},P<0.01; IL-1(0.58±0.89) *vs* (0.35±0.47) μg·L^{-1}(P<0.05); TNF-α(4.82±0.49) *vs* (4.21±0.45) μg·L^{-1}(P<0.05),含量均显著降低, TGF-β_1 mRNA(9.92±2.57) *vs* (5.27±1.39),(P<0.01)和α-SMA mRNA(12.87±0.39) *vs* (6.33±0.72), (P<0.01)及其蛋白表达显著减弱(均P<0.01)。 结论: 四逆散加味有明显的抗肝纤维化作用,其机制可能与抑制TGF-β_1和α-SMA基因表达有关。

中文关键词:四逆散 肝纤维化 转化生长因子-β_1 α-平滑肌肌动蛋白

Effects and Mechanism of Supplemental Sini San on Hepatic Fibrosis in Rats

Abstract:Objective: To investigate the protective effects and its mechanism of Supplemental Sini San(SNS) on hepatic fibrosis in rats. **Method:** The immunohepatic fibrosis model was induced by intraperitoneal injection of porcine serum. Eighty Wister rats were divided into eight groups:normal control,model control,Sini San Jiawei(SNSJW) high-dose,mediate-dose and low-dose,prevention and SNS group. Except the normal control groups,all the other groups were injected with pig's serum without inactivation into abdominal cavity, twice per week and 0.5 mL each time,persisting for 10 weeks. The prevention groups were orally given with the medicines(SNSJW 7 g·kg^{-1},10 weeks)at the same time models wasduplicated, the other treatment groups were dealed with just as the prevention groups 6 weeks later, lasted for 4 weeks. The dose was following:SNS group 4 g·kg^{-1}, SNSJW high-dose group 14 g·kg^{-1}, mediate-dose group 7 g·kg^{-1},low-dose group 3.5 g·kg^{-1}. The changes of hepatic fibrosis were detected. The content of liver tissue interleukin-1(IL-1) and tumor necrosis factor-alpha (TNF-α)were tested by RIA. In situ hybridization assay and immunohistochemical S-P method were used to detect the expression of transforming growth factor-β_1(TGF-β_1) mRNA and α-smoth muscle actin(α-SMA) mRNA and there protein in hepatic fibrosis tissues. **Result:** Compared with model group in SNSJW mediate-dose group, the content of HYP, IL-1,TNF-α in liver tissue was depressed significantly, the expression of TGF-β_1 and α-SMA genes was significantly lower in SNSJW mediate-dose group. **Conclusion:** Supplemental SNS has an action on hepatic fibrosis in rats. The mechanism is related with its inhibiting the expression of TGF-β_1 and α-SMA genes in rats liver.

keywords: Sini San liver fibrosis transforming growth factor-β_1 α-smooth muscle actin

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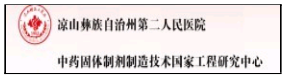


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