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Liver-protecting and fibrosis-resisting effect of Ganxianning on rats with spleen deficiency and stagnation of Liver-qi

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Abstract

AIM: To study the liver-protecting and fibrosis-resisting effect of Ganxianning (GXN) and its mechanism.

METHODS: Model of carbon tetrachloride hepatic injury fibrosis rats was reproduced. In the experiment there were six groups, the treatment groups with GXN's large, moderate and small dose (GXNb, GXNm and GXNs), the treatment group with colchicine, the blank model group and normal control group. The course of treatment was 30 d, then the rats were killed with their blood and liver tested.

RESULTS: In treatment groups, alanine aminotransferase was lower than that in the model group ($P < 0.01$), and albumin (Alb) higher than that in the model ($P < 0.01$). Hydroxylproline and red cell membrane C3B receptor garland in GXNb's and GXNm's groups were lower and circulation complex (CIC) was slightly higher. Fibrinogen (Fb) in both colchicine and model groups was higher than that in normal group and the difference was significant ($P < 0.05$, $P < 0.01$). Compared with model group, acid-α-naphthyl acetate esterase (ANAE) increased in GXNb's and GXNm's groups ($P < 0.05$, $P < 0.01$). Under light and electron microscopes, level of hepatic fibrosis of GXN groups was much lower than that of the model group, $P < 0.01$, and their difference was very significant. In GXNm's group, liver cell was normal on the whole and its chromatin was more than the model group and its nucleolus was evident.

CONCLUSION: GXN has rather good functions of protecting liver and resisting fibrosis, and these functions are related to the increase of ANAE and C3B, decrease of CIC and Fb, and improvement of body immunity function.

Key words: Ganxianning; Liver fibrosis/drug therapy; Colchicum; Microscopy, electron; Deficiency, spleen; Stagnation, Liver-qi; Rats

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