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白首乌C₂₁甾体苷诱导肝癌细胞凋亡的作用及其机制

王冬艳:张洪泉:李心

扬州大学 医药研究所, 江苏 扬州 225001

摘要:

研究白首乌 C_{21} 甾体苷对肝癌实体瘤细胞的凋亡作用及其机制。建立肝癌实体型(Heps)小鼠移植性肿瘤模型,随机分为模型组和 C_{21} 甾体苷各用药组,连续灌胃,10 d后脱颈椎处死小鼠,进行抑瘤率计算,对肿瘤组织进行电镜下 观察,采用免疫荧光(AO/EB)测定肿瘤细胞凋亡,免疫组化染色检测*bcl-2*基因的表达。C₂₁甾体苷(10,20和40

mg·kg⁻¹)对小鼠移植性肝癌Heps有抑制作用,3个剂量组的抑瘤率分别为34.79%,47.08%和50.23%。C₂₁甾 体苷(10,20和40 mg kg $^{-1}$)可增加肿瘤细胞的凋亡,电镜下可见凋亡的形态学改变,并出现凋亡小体;免疫组化结果显示,bcl-2基因的表达与模型组比较明显降低(p<0.01),但不同于凋亡结果的是高剂量组的阳性面积表达比 中剂量略高。降低高表达的bcl-2基因从而促进肝癌细胞的凋亡,可能是C₂₁甾体苷抗肝癌的机制之一。

关键词: 甾体苷; 肝癌; 细胞凋亡; bcl-2基因

Apoptosis induced by the C₂₁ sterols in Baishouwu and its mechanism of action in hepatoma

WANG Dong-yan; ZHANG Hong-quan; LI Xin

Abstract:

This study is to investigate the effect of the C₂₁ sterols on inducing apoptosis of hepatocellular cancer cells and its potential mechanism. The transplanted model of hepatoma substantiality (Heps) was established in mice, and the mice were divided into four groups: negative controls group and C21 sterols groups (10, 20, 40 mg·kg⁻¹), treated with drugs separately once a day for 9 days. Then the mice were sacrificed, the tumor growth inhibition rate (IR) was calculated and tumor tissue samples were taken and examined under electron microscope. The tumor cells were harvested and cell viability or apoptosis was analyzed by acridine orange and ethidium bromide (AO/EB) stain. B-cell lymphoma/leukemia-2 gene (bcl-2) in tumor cells was inspected by immunohistochemistry. After treatment with C21 sterols (10, 20,

40 mg·kg $^{-1}$), inhibitory effect on the transplanted Heps was observed. The IR was 34.79%, 47.08% and 50.23%, respectively. Apoptosis induced by the C $_{21}$ sterols was observed, low growth density and some apoptotic cells were observed in tumor under the electron microscope. The expression of bcl-2 gene on tumor cells decreased in the C₂₁ sterols groups, but the percentage of positive area is higher in 40

mg·kg⁻¹ group than that in 20 mg·kg⁻¹ group, which differed from apoptosis results. Inhibiting the excessive expression of bcl-2 gene to promote apoptosis may be one of anti-tumor mechanisms for the C₂₁ sterols in Baishouwu.

Keywords: hepatoma apoptosis bcl-2 gene sterols

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作者简介:

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