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介入导入Ad-p53基因联合超声辐照治疗兔VX2肝癌

Interventional targeting administration of Ad-p53 combined with ultrasound irradiation in rabbit models of hepatic VX2 tumors

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中文摘要:

目的:探讨介入导向下联合超声辐照对兔VX2肝癌模型Ad-p53转染效率及该基因对VEGF、MMP2的影响。方法:青紫蓝兔42只,直视下手术,将VX2肿瘤细胞种植于肝左叶,以超声检测肿瘤生长情况。根据第14天超声检测结果,将建模成功的30只动物随机分为3组,每组各10只,分别给予介入导向肝动脉灌注Ad-p53(Ad-p53组)及介入导向联合超声辐照Ad-p53给药(Ad-p53+US组)或等量生理盐水(对照组)。治疗后第4天超声检测肿瘤大小,之后处死动物,以ELISA法检测血清中VEGF含量,免疫组化法检测瘤组织内p53、MMP2、VEGF的表达水平,蛋白质印迹法检测Ad-p53不同给药方法对野生型p53表达水平的影响。结果:治疗前3组动物瘤体直径差异无统计学意义,治疗后各组瘤体均有增加,但Ad-p53+US组瘤体相对较小。治疗后第4天,野生型p53水平在Ad-p53组明显高于对照组,而Ad-p53+US组p53的表达水平明显高于Ad-p53组。Ad-p53+US组血清中VEGF含量显著降低。Ad-p53组和Ad-p53+US组肿瘤组织中VEGF及MMP2表达均减低,但以Ad-p53+US组更为明显。结论:Ad-p53基因对肝癌生长具有抑制作用。Ad-p53经载瘤动脉介入给药联合超声辐照的治疗作用优于单纯Ad-p53灌注。

英文摘要:

Objective: To observe the effect of interventional targeting administration of Ad-p53 combined with ultrasound irradiation in rabbit models of hepatic VX2 tumors, as well as its impact on VEGF and MMP2. **Methods:** Forty-two Chinchilla rabbits were collected and VX2 cancer cells were injected into the left lobe of liver on the observation in rabbits. The growth of cancer was monitored by ultrasound. Thirty rabbit models were successfully made and divided into 3 groups (each *n*=10) randomly. Fourteen days after transplantation of cancer cells, Ad-p53 was administrated through hepatic artery (in Ad-p53 group) or combined with ultrasound wave irradiation (in Ad-p53+US group), while the same amount of saline was given for rabbits in control group. Three days later, the tumor size was observed with ultrasound, and then all rabbits were sacrificed, the serum VEGF level was measured by ELISA, the hepatic tissue expression of p53, MMP2 and VEGF were detected respectively by immunohistochemistry, and expression level of wild type p53 was measured using Western blot. **Results:** No difference of tumor size was found between 3 groups before therapy. All tumor sizes increased, but the tumors in Ad-p53+US group were relatively smaller. The efficiency of Ad-p53 transfection was improved in Ad-p53 group compared with control group, which was the highest in Ad-p53+US group. Furthermore, the serum VEGF level decreased in Ad-p53+US group, so did the expression of MMP2 and VEGF in Ad-p53 group and Ad-p53+US group, more obviously in Ad-p53+US group. **Conclusion:** Ad-p53 can suppress the growth of hepatic VX2 tumors in rabbit models. The therapeutic efficacy of Ad-p53 can be improved by interventional targeting administration combining with ultrasound irradiation.

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