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## 基础研究

兔急性后腔静脉血栓形成中内皮细胞VEGF 与bFGF 表达的变化

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摘要:

目的:观察血管内皮生长因子(VEGF)和碱性成纤维细胞生长因子(bFGF)在兔急性后腔静脉血栓 形成后内皮细胞中的表达变化及溶栓抗凝治疗对其的影响。方法: 60只健康成年新西兰白兔采用缩窄 法制作急性后腔静脉血栓模型后,随机均分为模型组和治疗组。术后第2天造影检查证实模型均构建 成功后,治疗组行溶栓抗凝治疗,模型组给予等体积的生理盐水。两组分别于用药后第1,4,7天, 随机各取10只动物,用免疫组化法检测受累血管内皮细胞VEGF和bFGF的表达。同期以10只正常新西 兰兔作为对照。结果:VEGF与bFGF在正常兔血管内皮细胞中均呈少量表达。两个实验组中,受累血 管内皮细胞的VEGF表达较正常对照组明显升高(均P<0.05),并在观察时间内呈进行性升高,但治 疗组升高的程度在各时间点上均明显大于模型组(均P<0.05); bFGF的变化趋势与VEGF基本一致, 只是在给药后第4天后未见继续升高。结论: 急性血栓形成后,内皮细胞VEGF与bFGF表达增高可能是 机体的内源性保护作用,溶栓抗凝治疗能促进该作用的发挥。

关键词: 静脉血栓形成: 血管内皮生长因子类: 成纤维细胞生长因子2: 兔

Alterations of VEGF and bFGF expression in endothelial cells of rabbits with acute caudal vena cava thrombosis

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Abstract:

Objective: To observe the expression alterations of vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) in the endothelial cells of rabbits with thrombosis of posterior vena cava (PVC) and the effects exerted by thrombolytic and anticoagulant therapy. Methods: Sixty healthy adult New Zealand white rabbits were equally randomized into model group and treatment group after the establishment of PVC thrombosis model with the coarctation procedure. After the confirmation of model establishment by contrast examination on the second postoperative day, rabbits in treatment group received thrombolytic and anticoagulant therapy while those in model group were given normal saline of the same volume. At the 1, 4 and 7 d after dosing administration, 10 rabbits at each time point were randomly selected from each group, and the expressions of VEGF and bFGF in the endothelial cells of the involved PVC were detected by immunohistochemical staining. Meanwhile, 10 normal New Zealand rabbits served as control. Results: There was only a small amount of VEGF and bFGF expression in the endothelial cells of normal rabbits. In the two experimental groups, the VEGF expressions in the endothelial cells of the involved PVC were significantly increased versus control (all P<0.05), and the increases were progressively greater during the observation period, but the increasing amplitude at each time point in treatment group was significantly greater than that of model group (all P<0.05); the alteration pattern of bFGF was generally similar to that of VEGF, but it reached the peak at 4 d after administration and did not continue to rise. Conclusion: The increased VEGF and bFGF expressions in the endothelial cells may be an endogenous protective action following acute venous thrombosis, and thrombolytic and anticoagulant therapy can promote this action.

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