

高血压大鼠足细胞的超微病变和podocalyxin的表达及其作用研究

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中文摘要:目的观察高血压大鼠足细胞的超微结构病变, 探讨足细胞蛋白podocalyxin (PCX) 在高血压大鼠肾组织中的表达及作用。方法随机将30只雄性SD大鼠以改进的“两肾一夹”方法建立高血压大鼠模型, 分为高血压组和对照组。分别于造模前和造模后1、5、10周检测两组大鼠的尿 β 2-微球蛋白 (β 2-MG)、血尿素氮 (BUN) 及血肌酐 (Scr) 水平; 免疫荧光方法观察PCX在肾组织的表达, 并用Image-pro plus 6.0软件, 以面积密度值进行分析; 光镜、电镜观察肾小球及足细胞超微结构改变。结果 (1) 术后2周起高血压组收缩压 (SBP) 较对照组显著升高 (137.2 ± 9.4 mmHg vs 92.3 ± 10.3 mmHg, $P < 0.01$), 并且逐渐升高至术后4周趋于平稳。(2) 术后5周起高血压组尿 β 2-MG显著高于对照组 [(110.28 ± 11.25) ng/L vs (56.68 ± 9.51) ng/L, $P < 0.01$], 并且有继续增高的趋势。两组BUN、Scr在整个实验过程中没有显著性差异。(3) 光镜、电镜观察均可见高血压组肾脏发生病理改变; 足细胞超微结构发生病变。(4) 免疫荧光可见高血压组PCX表达明显减少, 其面积密度值显著低于对照组 (0.204 ± 0.042 vs 0.296 ± 0.039 , $P < 0.01$), 且PCX表达与尿 β 2-MG呈显著负相关 ($r = -0.927$, $P < 0.01$)。结论PCX蛋白表达降低可能是参与高血压肾损害的机制之一, 其可能是导致肾小球电荷屏障受损、尿蛋白排泄增加、肾功能受损、肾脏病理及足细胞超微结构发生病变的基础之一。

中文关键词: [高血压肾损害](#) [足细胞](#) [超微病变](#) [Podocalyxin](#)

Podocalyxin Ultrastructure Pathological Changes of Podocyte and the Expression of Podocalyxin in the Kidney of Hypertensive Rats

Abstract: Objective To observe the ultrastructure pathological changes of podocyte and to investigate the expression and roles of the podocyte-associated molecule podocalyxin (PCX) in the kidney of 2-kidney-1-clip hypertensive rats. Methods Thirty male SD rats were randomly assigned into two groups: experimental group ($n=20$) and control group ($n=10$). Improved 2-kidney-1-clip hypertensive rats were used in the experiment. The urinary β 2-microglobulin (β 2-MG), blood urea nitrogen (BUN) and serum creatinine (Scr) were monitored at different stages. The expression of podocalyxin in the kidney was detected by using immunofluorescence, and analyzed by Image-pro plus 6.0 software. The pathological changes of the kidney and podocyte were observed under optical and electron microscope respectively. Results The systolic blood pressure 2 weeks after operation in experimental group was significantly higher than that in control group ($P < 0.01$). The urinary β 2-MG 5 weeks after operation in experimental group was significantly higher than in that control group ($P < 0.01$), but the BUN and Scr levels in experimental group had no significant difference compared with control group in the whole process. We could see pathological changes of the kidney under optical and electron microscope. The expression of PCX in experimental group was significantly lower than in control group ($P < 0.01$), and it was negatively correlated with the urinary β 2-MG ($r = -0.927$, $P < 0.01$). Conclusion The reduction of the expression of the podocyte-associated molecule podocalyxin may be one of the pathogenesises of hypertensive kidney lesion. It may lead to injury of charge barrier in glomerulus, the increase of the excretion of urinary β 2-MG, and impairment of renal function.

keywords: [Hypertensive renal disease](#) [Podocyte](#) [Ultrastructure pathological changes](#) [Podocalyxin](#)

