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谷氨酰胺对肝移植大鼠肠黏膜NF-κB、ICAM-1、TN

导航/NAVIGATE
本期目录/Table of Contents
下一篇/Next Article
上一篇/Previous Article

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Title: Effect of glutamine on expression of NF-κB, ICAM-1 and TNF-α in intestinal mucosa after liver transplantation in rats

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关键词: 谷氨酰胺; 肝移植; 核转录因子-κB; 细胞间黏附分子-1; 肿瘤坏死因子-α

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摘要: 目的　研究谷氨酰胺（Glutamine, Gln）对原位肝移植(orthotopic liver transplantation, OLT)大鼠肠黏膜内核转录因子-κB（NF-κB）、细胞间黏附分子-1（ICAM-1）、肿瘤坏死因子-α（TNF-α）表达的影响。　方法　选择健康雄性Wistar大鼠70只，按随机数字表法分为对照组(*n*=10)、原位肝移植组(OLT组，*n*=30)和原位肝移植+Gln组（EEN组，*n*=30）；对照组只分离肝十二指肠韧带，OLT组和EEN组按改良的两袖套法进行原位肝移植。EEN组受体在术前3 d、术后3 h开始给予肠内营养混悬液能全力+谷氨酰胺灌胃，OLT组及对照组受体仅给予肠内营养混悬液。对照组分离肝十二指肠韧带12 h后，OLT组和EEN组肝移植后12、24、72 h分别取回肠肠壁组织免疫组化测定肠组织NF-κB与ICAM-1的表达，荧光定量PCR(fluorescence quantitive-polymerase chain reaction, FQ-PCR)测定肠黏膜TNF-α mRNA表达、HE染色观察回肠组织的病理改变，测定微绒毛长度。　结果　与对照组比较OLT组和EEN组肝移植后12、24、72 h，NF-κB、ICAM-1、TNF-α mRNA表达明显升高，肠黏膜损害明显加重，差异有统计学意义（*P*<0.01）；而肝移植后12、24、72 h EEN组与OLT组比较，NF-κB、ICAM-1、TNF-α mRNA表达明显下降，肠黏膜损害明显减轻，差异有统计学意义（*P*<0.01）。　结论　大鼠原位肝移植可引起肠组织中NF-κB活化，ICAM-1、TNF-α表达上调导致肠黏膜屏障损伤，Gln能抑制肝移植大鼠肠黏膜NF-κB活性，减少ICAM-1、TNF-α的表达而起到肠黏膜保护作用。

Abstract: Objective To investigate the effect of glutamine (Gln) on the expression of nuclear factor kappa B (NF-κB), intercellular adhesion molecule-1 (ICAM-1) and tumor necrosis factor α (TNF-α) in intestinal mucosa after orthotopic liver transplantation (OLT) in rats. Methods Seventy healthy male Wistar rats were divided randomly into three groups including a normal control group (control group, *n*=10), an orthotopic liver transplantation group (OLT group, *n*=30) and an early enteral nutrition group (EEN group, *n*=30). Only hepatoduodenal ligament dissection was performed in the control group, and OLT was performed by modified two-cuff method in the OLT group and EEN group. For the EEN group, the recipients were supplied with Nutrison Fiber 125 mL/（kg • d) plus Gln 0.3 g/（kg • d) for 3 d by gastric perfusion before surgery, and Nutrison Fiber plus Gln was administered again until the animal was killed at 3 h after OLT. For the OLT group and control group, the same volume of the Nutrison Fiber was supplied by gastric perfusion at the same time. The expression of NF-κB and ICAM-1 in the ileal mucosa were determined by immunohisto chemistry, the mRNA level of TNF-α in the ileal mucosa was detected with fluorescence quantitative-polymerase chain reaction (FQ-PCR), and the ultrastructural changes of the ileal mucosa were observed with transmission electron microscopy (TEM) at 12 h after hepatoduodenal ligament dissection in the control group and at 12, 24 and 72 h after OLT in the OLT group and EEN group. Results Compared with the control group, the levels of NF-κB, ICAM-1 and TNF-α mRNA significantly increased and the mucosal lesion significantly aggravated in the OLT group and EEN group at 12, 24 and 72 h after OLT (*P*<0.01). Compared with the OLT group, the levels of NF-κB, ICAM-1 and TNF-α mRNA were significantly decreased and the mucosal lesion was significantly relieved in the EEN group at 12, 24 and 72 h after OLT (*P*<0.01). Conclusion OLT can activate NF-κB in intestinal mucosa, and up-regulate the expression of ICAM-1 and TNF-α to induce the injury of intestinal mucosal barrier. Gln can protect the intestinal mucosal barrier through suppressing the activation of NF-κB and reducing the expression of ICAM-1 and TNF-α after OLT.

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