中国病理生理杂志 2005 25 (8): 1587-1589 ISSN: 1000-4718 CN: 44-1187/R

论著

谷氨酰胺对脂多糖血症大鼠热休克蛋白70和肿瘤坏死因子-α表达的影响

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收稿日期 2004-11-5 修回日期 2004-5-30 网络版发布日期 2009-10-16 接受日期 2004-5-30

摘要 目的：观察谷氨酰胺对脂多糖血症大鼠热休克蛋白70和肿瘤坏死因子-α的影响。 方法: 将大鼠分为脂多糖组（LPS）、谷氨酰胺组（Gln）和对照组(C)。各组动物均在给予LPS前（对照组在给予生理盐水）和后2、4、6 h采血。用放射免疫法测定血浆肿瘤坏死因子-α（TNF-α）含量。于术后6 h处死，取肺、肝、肠组织，采用SABC方法，免疫组织化学染色进行HSP70检测，并HE染色，观察组织变化。 结果： 注射LPS后2 h，LPS组血浆TNF-α表达显著升高（P<0.01），Gln可显著抑制其升高（P<0.01）。而注射LPS后4、6 h，两组血浆TNF-α浓度没有明显差异。LPS组肺、肝、肠组织的HSP70灰度值分别为（107.94±10.96）、（120.04±5.73）和（123.31±14.81）。Gln组的HSP70灰度值显著降低，分别为（89.71±9.64）、（89.38±12.03）和（107.61±14.02）（均P<0.05）。病理学观察显示, Gln组肺、肝、肠组织损伤程度比LPS组轻。 结论： 谷氨酰胺可提高热休克蛋白70的表达，对脂多糖血症大鼠有防治作用。

关键词 谷氨酰胺； 脂多糖类； 热休克蛋白质70； 肿瘤坏死因子

分类号 R515.3

Effect of glutamine on heat shock protein-70 and tumor necrosis factor-α expession in endotoxemic rats

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Abstract

<FONT face=Verdana>AIM: To study the protective effect of glutamine (Gln) against endotoxemia by observing the effect of glutamine on heat shock proteins (HSPs) and tumor necrosis factor-α (TNF-α) in endotoxemic rats. METHODS: The rats were randomly divided into 3 groups, lipopolysaccharide group (LPS), glutamine-treated group (Gln) and control group (C). The blood was drawn from lateral tail vein for analysis of cytokine levels at 0, 2, 4 and 6 h post-lipopolysaccharide (LPS) challenge. TNF-α was measured by radioimmunity assay. Multiple tissues were harvested from the rats, and HSP70 was detected by immunohistochemistry. At the same time, lung, liver, and ileum tissue section were stained with hematoxylin and eosin. RESULTS: Gln treatment resulted in marked attenuation of TNF-α expression at 2 h post-LPS injection (P＜0.01). Gray gradients of HSP70 in lungs, liver and ileum tissue in group Gln were much lower than those of group LPS (P＜0.05), This suggested that HSP70 content in these tissues of group Gln was higher than that of group LPS. Tissue sample from lung, liver and ileum revealed significantly less evidence of endotoxin-induced tissue damage in Gln-treated animals. CONCLUSION: Gln can significantly enhance HSP70 expression in multiple tissues of endotoxin-treated rats. A single dose of intravenous Gln given concomitantly with an endotoxin injury can markedly reduce organ histological damage, and attenuate pro-inflammatory cytokine release. </FONT>

Key words Glutamine Lipopolysaccharides Heat shock proteins 70 Tumor necrosis factor

DOI: 1000-4718

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