

论著

前列地尔对脑死亡大鼠肝脏功能的保护及血清TNF- α 和内皮素-1表达的影响

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摘要:

目的: 研究前列地尔脂微球载体剂对脑死亡状态下SD大鼠肝脏功能和形态的影响。方法: 选取SD大鼠24只, 随机分为4组, 即脑死亡组(B组): 应用改进的缓慢间断颅内加压法建立脑死亡模型; 假手术组(C组): 除不进行硬脑膜外导管加压外, 其余处理同脑死亡组; 前列地尔干预1, 2组(L1, L2): 建立脑死亡模型, L1, L2组分别于SD大鼠确认脑死亡后, 持续泵入前列地尔脂微球载体剂20, 40 ng/(kg·min) 至实验结束。抽取血标本检测丙氨酸氨基转移酶(ALT)、天门冬氨酸氨基转移酶(AST)、肿瘤坏死因子- α (TNF- α)、内皮素-1(ET-1) 水平。于脑死亡后6 h取材, 切取肝脏组织标本包埋固定, 切片, HE染色观察肝细胞损伤情况。结果: 在脑死亡时, B, L1和L2组ALT, AST, ET-1, TNF- α 水平与C组比较, 差异有统计学意义 ($P<0.05$), L1和L2组低于B组, 差异有统计学意义 ($P<0.05$), L2组与L1组比较, 差异无统计学意义 ($P>0.05$)。结论: SD大鼠脑死亡状态下肝脏可发生损伤性改变。前列地尔脂微球载体剂能减轻脑死亡状态下大鼠肝脏的损伤。

关键词: 前列地尔 脑死亡 大鼠 肝脏 内皮素

Effect of alprostadil on hepatic injury of brain-dead rats and on serum TNF- α and endothelin-1 expression

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Abstract:

Objective To study the effect of alprostadil lipid microballoons (lipo PGE1) on the function and morphous of livers from brain-dead rats. Methods Twenty-four SD rats were randomly assigned into 4 groups: a control group (Group C), a brain-dead group (Group B) and 2 lipo PGE1 protection groups (Group L1 and Group L2). Brain-dead models were established in Group B, L1 and L2. There was no inflation of Fogarty balloon in Group C, while other operations were the same as Group B. Lipo PGE1 [20 ng/(kg·min) and 40 ng/(kg·min)] was injected via the femoral vein in Group L1 and Group L2 immediately after the establishment of the brain-dead model. The serum levels of alanine aminotransferase (ALT), aspartate amino transferase (AST), endothelin (ET)-1, and tumor necrosis factor (TNF)- α were detected by radioimmunological analyzer. Liver tissues were observed by HE staining 6 h after the brain death. Results At the time of brain death, the level of ALT, AST, ET-1, and TNF- α in Group B, L1 and L2 was significantly different compared with that in Group C. That in Group L1 and L2 was significantly lower than in Group B ($P<0.05$). There was no significant difference between Group L1 and L2 ($P>0.05$). Conclusion Brain death can cause damage to the liver of rats. Lipo PGE1 can relieve the injury of brain death donors. The protective mechanism of Lipo PGE1 is to decrease the release of serum inflammatory mediators.

Keywords: alprostadil; brain death; rat; liver; endothelin

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