中国现代应用药学
Chinese Journal of Modern Applied Pharmacy
全国性科技期刊
全国中文核心期刊
中国科技论文统计源期刊

王刚,董晓巧,杜权,俞文华,张祖勇,朱强,车志豪,陈锋,王昊,陈军.氧化苦参碱抗大鼠脑外伤后神经细胞凋亡及机制研究[J].中国现代应用药学,2012,29(4):289-294

氧化苦参碱抗大鼠脑外伤后神经细胞凋亡及机制研究

Effect of Oxymatrine on Neuronal Cell Apoptosis and Its Mechanisms after Traumatic Brain Injury

投稿时间： 2011-09-20 最后修改时间： 2011-12-18

DOI：

中文关键词： 脑外伤 氧化苦参碱 细胞凋亡 自由基 炎症反应

英文关键词:traumatic brain injury oxymatrine apoptosis free radical inflammation

基金项目:浙江省中医药科技计划项目(2010ZA101)

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中文摘要:

目的 观察氧化苦参碱对大鼠脑外伤后神经细胞凋亡、氧化损伤和炎症反应的影响，探讨氧化苦参碱抗神经细胞凋亡的机制。方法 选用健康Wistar大鼠140只，♂，随机分成假手术组、脑外伤组、氧化苦参碱60 mg·kg^{-1}组和氧化苦参碱120 mg·kg^{-1}组，每组35只，利用改进的Feeney自由落体损伤装置制作脑外伤动物模型，分别于脑外伤模型形成后给予1 mL 0.9%氯化钠注射液及不同剂量氧化苦参碱(60和120 mg·kg^{-1})腹腔注射，每日1次，共5 d，在脑外伤后2，6，12 h，1，2，3 和5 d断头取血和获取脑组织，利用黄嘌呤氧化酶法检测血清超氧化物歧化酶活性，利用硫代巴比妥酸法测定血清丙二醛浓度，利用酶联免疫吸附试验测定血清白介素-1β、肿瘤坏死因子-α和白介素-6浓度，利用原位末端转移酶标记技术检测脑组织中凋亡神经细胞，同时进行统计分析。 结果 脑外伤后2，6，12 h，1，2，3和5 d，氧化苦参碱60 mg·kg^{-1}组大鼠凋亡神经细胞数量、血清超氧化物歧化酶活性及丙二醛、白介素-1β、肿瘤坏死因子-α和白介素-6浓度与脑外伤组比较差异没有统计学意义(P>0.05)；脑外伤后12 h，1，2，3和5 d，氧化苦参碱120 mg·kg^{-1}组大鼠凋亡神经细胞数量、血清超氧化物歧化酶活性及丙二醛、白介素-1β、肿瘤坏死因子-α和白介素-6浓度较脑外伤组显著减少(P<0.05)，脑外伤后2 h和6 h，氧化苦参碱120 mg·kg^{-1}组大鼠凋亡神经细胞数量、血清超氧化物歧化酶活性及丙二醛、白介素-1β、肿瘤坏死因子-α和白介素-6浓度与脑外伤组比较差异没有统计学意义(P>0.05)。结论 氧化苦参碱可能通过降低体内自由基和炎症反应，从而抑制脑外伤后神经细胞凋亡，达到神经保护作用。

英文摘要:

OBJECTIVE To investigate the effect of oxymatrine on neuronal cell apoptosis, oxidative injury and inflammatory reaction and furthermore discuss possible mechanism of oxymatrine suppressing neuronal cell apoptosis. METHODS All of 140 male Wistar rats were randomly divided into sham operation group, brain trauma group, as well as oxymatrine of 60 mg·kg^{-1} and 120 mg·kg^{-1} treatment group. The rat model of traumatic brain injury was induced by a modi?cation of Feeney's weight-drop model. Oxymatrine was given to rats in the oxymatrine of 60 mg·kg^{-1} and 120 mg·kg^{-1} treatment groups via intraperitoneal injection (60 and 120 mg·kg^{-1}) after trauma, and then once a day till day 5. The rats in the brain trauma and sham operation groups received an intraperitoneal administration of 1 mL of saline after trauma or procedures. Animals were sacrificed by decapitation at hour 2, 6 and 12, as well as day 1, 2, 3, and 5 after trauma. Brains were removed and blood was collected. Neuronal cell apoptosis was measured using terminal deoxynucleotidyl transferase biotin-dUTP nick end labeling; superoxide dismutase activity in serum, was determined by xanthine oxidase method; serum malondialdehyde level, was analyzed by thiobarbituric acid reactive substance assay; interleukin-1beta, tumor necrosis factor-beta, and interleukin-6 levels in serum, were measured by enzyme linked immunosorbent assay. Statistical analysis was performed. RESULTS At hour 2, 6 and 12, as well as day 1, 2, 3, and 5 after trauma, the differences between the number of apoptotic neuronal cell, superoxide dismutase activity in serum, as well as serum malondialdehyde, interleukin-1beta, tumor necrosis factor-beta and interleukin-6 levels in the oxymatrine of 60 mg·kg^{-1} treatment group and brain trauma group were not significant statistically (P>0.05). At hour 12, as well as day 1, 2, 3, and 5 after trauma, the number of apoptotic neuronal cell, superoxide dismutase activity in serum, as well as serum malondialdehyde, interleukin-1beta, tumor necrosis factor-beta and interleukin-6 levels in the oxymatrine of 120 mg·kg^{-1} treatment group were significantly lower than those in the brain trauma group (P<0.05), but at hour 2 and 6, these differences were not significant (P>0.05). CONLCUSION Oxymatrine may suppress neuronal cell apoptosis via inhibition of

free radical and inflammatory reactions after traumatic brain injury.