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论著

氯化锰对大鼠血-脑脊液屏障的损伤作用

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摘要 **目的** 探讨氯化锰对大鼠脑脉络丛组织的毒性损伤作用。**方法** 大鼠ip给予氯化锰6 mg(Mn)·kg^{-1}建立不同暴露时长（30 d, 90 d及90 d后无处理观察30 d）的氯化锰中毒动物模型，各时间点染毒结束后采集血清与脑脊液（CSF）样本，提取侧脑室的脉络丛组织。溴甲酚绿法和ELISA法检测血清白蛋白(SALB)与CSF白蛋白(CALB)水平，并计算CSF白蛋白指数。光镜与透射电镜下检测大鼠脉络丛组织、细胞及亚细胞结构的病理形态学改变。**结果** 氯化锰30 d组、氯化锰90 d组及氯化锰90 d+30 d恢复组CSF白蛋白、CSF白蛋白指数均明显高于其相应的对照组（P<0.05），并且氯化锰30 d组CSF白蛋白、CSF白蛋白指数低于氯化锰90 d组与氯化锰90 d+30 d恢复组（P<0.05），氯化锰90 d组CSF白蛋白、CSF白蛋白指数低于氯化锰90 d+30 d恢复组，但无统计学意义。光镜和电镜观察发现，氯化锰导致脉络丛上皮细胞形状不规则，微绒毛结构紊乱、缩短；胞浆内出现空泡、核质凝聚，线粒体结构破坏，细胞间连接部分断裂或消失等，随染氯化锰时间的延长有加重的趋势，并且脱离氯化锰接触30 d后仍表现为进行性加重。**结论** 氯化锰可以引起脉络丛组织病理形态学改变，具有时效性与不可逆性的特征；CSF白蛋白及CSF白蛋白指数与脉络丛的损伤程度呈正相关，可以作为判断血-脑脊液屏障损伤程度的参考指标。

关键词 氯化锰 血脑屏障 脉络丛 白蛋白类

分类号 R994.6

Injury of chloride manganese to blood cerebrospinal fluid barrier in rats

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Abstract

OBJECTIVE To investigate the effect of manganese chloride ($MnCl_2$) on rats′ choroid plexus (CP). **METHODS** The rats were ip given $MnCl_2$ 6 mg(Mn)·kg^{-1} for 30 d, 90 d or 90 d, followed by 30 d recovery. The samples of serum, cerebrospinal fluid (CSF) and choroid plexus of lateral ventricles were collected at each time point. Serum albumin and CSF albumin were determined by bromcresol green(BCG) and ELISA, respectively. Pathomorphological changes of choroid plexus cells were observed by the light microscopy and electron microscopy. **RESULTS** The levels of CSF albumin and CSF albumin index in $MnCl_2$ groups were obviously higher than those in corresponding control groups (P<0.05). The CSF albumin and CSF albumin index in $MnCl_2$ 30 d group was lower than those of other $MnCl_2$ groups (P<0.05), and those in $MnCl_2$ 90 d group were slightly lower than in $MnCl_2$ 90 d+30 d recovery group, but without any statistical difference. Light microscopy and transmission electron microscopy revealed that $MnCl_2$ exposure resulted in flattened and shrunken cell layers, cytoplasmic vacuolation, nuclei and chromosome condensation, mitochondrion destruction, shortening of microvilli, and partial disconnection in intracellular junctions between two adjacent epithelial cells. The structural alteration was mild after 30 d treated and moderate in $MnCl_2$ 90 d. Noticeably, the intracellular damage in $MnCl_2$ 90 d+30 d recovery group was even worse than that in $MnCl_2$ 90 d, suggesting a long lasting pathological damage even after $MnCl_2$ exposure be not ceased. **CONCLUSION** $MnCl_2$ can induce pathological changes of choroid plexus, with the characteristics of time-variation and irreversibility. The levels of CSF albumin and CSF albumin index correlate with the extent of injury, and are potentially of great value for assessing BCB impairment after manganese exposure.

Key words manganese blood brain barrier choroid plexus albumins

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