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论文

常压氧疗对大鼠脑缺血再灌注后血脑屏障损伤的作用及机制

刘宝义1,张晓明2,郭晓笋2,胡维诚2,袁中瑞2

山东大学 1. 齐鲁医院呼吸内科,济南 250012;

2. 医学院病理生理学教研室,济南 250012

摘要:

目的 研究常压氧疗对大鼠脑缺血再灌注后血脑屏障损伤的作用及机制。方法 采用大鼠大脑中动脉线栓法制备脑缺血再灌注模 型。随机将30只SD雄性大鼠,分为模型对照组和常压氧疗治疗组各15只,缺血90min后再灌注24h。采用TTC染色、伊文思蓝法和 明胶酶谱技术,分别检测脑梗死体积、血脑屏障通透性及缺血脑组织基质金属蛋白酶-9(MMP-9)的活性,并进行神经功能评分。结 与对照组比较,常压氧疗治疗组大鼠的脑梗死体积、缺血脑组织伊文思蓝外渗率及MMP-9的活性显著降低(P<0.01),神经功 能缺陷也得到显著改善。结论 缺血期内的常压氧疗对大鼠脑缺血再灌注后血脑屏障的损伤具有保护作用,其作用机制可能是通过 抑制MMP-9的活性来实现的。

关键词: 再灌注损伤; 缺氧缺血, 脑; 血脑屏障; 明胶酶B; 大鼠

The effect and mechanism of normobaric hyperoxia on blood-brain barrier impairment following cerebral ischemia reperfusion in rats

LIU Bao-yi1, ZHANG Xiao-ming2, GUO Xiao-sun2, HU Wei-cheng2, YUAN Zhong-rui2

- 1. Respiratory Department, Qilu Hospital of Shandong University, Jinan 250012, China;
- 2. Department of Pathophysiology, School of Medicine, Shandong University, Jinan 250012, China

Abstract:

To investigate the effect of normobaric hyperoxia on blood brain barrier impairment following cerebral ischemia reperfusion in rats, and explore the mechanism. Methods 30 normal male SD rats were randomly divided into 2 groups: ① cerebral ischemia-reperfusion control group (n=15), and ② normobaric hyperoxia treatment group (n = 5). Cerebral ischemia-reperfusion rat models were established by the thread ligation of right middle cerebral artery . At 24 hrs after the reperfusion following 90min ischemia, TTC staining, Evans blue dye extravasation and Gelatin zymography were applied to quantify infarction volume, blood-brain barrier impairment and the activity of MMP-9, respectively. Neurological deficit scores were also evaluated. Results Compared with the control group, normobaric hyperoxia treatment significantly decreased Evans blue dye extravasation, the activity of MMP-9 and infarct volume (P < 0.01) at 24 hrs after the reperfusion. Correspondingly, neurological functions were improved. Conclusions Nnormobaric hyperoxia treatment during the period of focal cerebral ischemia demonstrates the protective effect on blood-brain barrier impairment in the rat model of cerebral ischemia-reperfusion, and the mechanism may be attributed to the suppression of MMP-9.

Keywords: Reperfusion injury: Hypoxia-ischemia, brain; Blood-brain barrier; Gelatinase B; Rats

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通讯作者: 袁中瑞 (1969-),女,博士,副教授,硕导,主要从事脑卒中的损伤、修复与防治的研究。 E-mail: zhongruiyuan@sdu.edu.cn

作者简介: 刘宝义(1970-), 男, 博士, 副主任医师, 主要从事机械通气及呼吸系统疾病的研究。

作者Email:

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