中国病理生理杂志 2007 23 (3): 500-504 ISSN: 1000-4718 CN: 44-1187/R

论述

肥大心肌细胞缺氧复氧损伤特点及干预能量代谢的作用

冯兵,徐静,刘伟,杨晓,何作云,杨惠标

第三军医大学新桥医院肾内科, 重庆 400037

收稿日期 2005-7-15 修回日期 2005-11-1 网络版发布日期 2008-8-3 接受日期 2005-11-1

摘要 目的：探讨肥大心肌细胞缺氧复氧损伤与能量代谢途径转换的关系。 方法: 应用血管紧张素Ⅱ (AngⅡ, 0.1 μmol/L)+去甲肾上腺素（NE 1 μmol/L）诱导培养大鼠心肌细胞肥大，以同位素液闪计数法测定葡萄糖有氧氧化率（GOR）、葡萄糖酵解率（GLR）和脂肪酸有氧氧化率（FOR），TUNEL法测定细胞凋亡。 结果: (1)常氧培养时，肥大心肌细胞的葡萄糖氧化率（GOR）、糖酵解率（GLR）均高于正常心肌细胞，而脂肪酸氧化率（FOR）低于正常心肌细胞，但与细胞凋亡率一样，与正常心肌细胞比较无显著差异。(2)肥大心肌细胞缺氧6 h后细胞凋亡率显著高于缺氧前，复氧后不仅细胞凋亡更显著高于缺氧前，还出现了细胞坏死。(3)正常心肌细胞和肥大心肌细胞缺氧后GLR无明显变化，GOR、FOR均低于缺氧前，缺氧6 h时出现显著差异，但肥大心肌细胞GOR在缺氧2 h时即显著低于缺氧前。缺氧复氧后，正常心肌细胞和肥大心肌细胞GOR均低于对应单纯缺氧时间组，而GLR和FOR轻度高于对应单纯缺氧2 h组，在肥大心肌细胞均显著高于对应单纯缺氧时间组，并随缺氧时间延长更明显。(4)二氯乙酸（DCA）和曲美他嗪（TMZ）预处理后的肥大心肌细胞缺氧复氧后的GOR显著高于对应的无干预肥大心肌细胞组，GLR、FOR和细胞凋亡率均显著低于无干预肥大心肌细胞组。 结论: 肥大心肌细胞更易于受到缺氧复氧刺激的损伤，活化糖代谢可部分抑制这种损伤。

关键词 心肌肥大 缺氧 细胞凋亡 能量代谢

分类号 R363

The characteristics of hypertrophic cardiomyocyte injury and the effect of intervention on energy metabolism after different time of hypoxia/reoxygenation

FENG Bing, XU Jing, LIU Wei, YANG Xiao, HE Zuo-yun, YANG Hui-biao

Department of Kidney Disease, Xinqiao Hospital, Third Military Medical University, Chongqing 400037, China

Abstract

<FONT face=Verdana>AIM:To investigate the characteristics of pathological injury and its relationship with the transformation of energy metabolism of hypertrophic cardiomyocytes after hypoxia-reoxygenation.
METHODS:Cultured rat cardiomyocytes were induced to be hypertrophy by angiotensin Ⅱ (Ang Ⅱ) and norepinephrine (NE). Glucose oxidation rate (GOR), glucolysis rate (GLR) and fatty acid oxidation rate (FOR) were determined by liquid scintillation counting, and cell apoptosis was detected by TUNEL.
RESULTS:(1) Compared with the normal cardiomyocytes (NC), the GOR and GLR were slightly higher and the FOR was slightly lower in the group of hypertrophic cardiac cells (HC) than that in the group of normal cardiomyocytes cultured under the normal oxygen partial pressure. The apoptosis rate had no difference between the two groups. (2) The apoptosis rate of hypertrophic cardiomyocytes after hypoxia was significantly higher than that of hypertrophic cardiomyocytes in normal culture. It was higher and moreover, some necrosis cardiomyocytes appeared after reoxygenation. (3) GOR and FOR in both group (NC and HC) were slightly lower in a time-dependent manner after hypoxia than that in each group in normal culture condition. GLR had no difference in both group. The GOR was more lower in both NC and HC group when reoxygenation than that at the point of hypoxia for 2 hours, but the GLR and FOR were significantly higher in HC than that in NC when reoxygenation. (4) The GOR was significantly higher and the GLR and FOR were significantly lower in the hypertrophic cardiomyocytes group (HC) with dichloroacetate (DCA, 1 000 μmol/L) or trimetazidine (TMZ, 1 μmol/L) treated respectively than that in the responded hypertrophic cardiomyocytes after stimulation by hypoxia-reoxygenation. In the meanwhile, the apoptosis rate also was markedly lower in the treated hypertrophic

cardiomyocytes group.
CONCLUSION:The transformation of energetic metabolism pathway plays an important role in the pathogenesis (mainly the apoptosis) of the hypertrophic cardiomyocytes after hypoxia-reoxygenation. </FONT>

Key words Myocardial hypertrophy Anoxia Apoptosis Energy metabolism

DOI: 1000-4718

通讯作者