DOI:10.16016/j.1000-5404.1994.04.010

Studies of the functional and morphological changes of gastric mucosa after portoazygos disconnection in cirrhotic portal hypertension in rats

Liu Jikui, Cai Jingxiu, Wang Aochuan, Yang Hengwen (Department of Hepatobiliary Surgery, Southwest Hospital, Third Military Medical College, Chongqing, 630038)

Abstract The functional and morphological changes of the gastric mucosa after portoazygos disconnection in cirrhotic portal hypertension were observed in rats. It was found that the gastric mucosal blood flow (GMBF) and the level of glycoprotein (GP) and prostaglandins (PGE_2 and $6-keto-PGF_{1\alpha}$) of the gastric corpus and antrum were significantly lower in portal hypertension than in the control. There were desquamation of the epithelial cells of the gastric mucosa, edema and thickening of the submucosal layer and narrowing of the capillaries in cirrhotic portal hypertension. After the disconnection of the portoazygos junction, the above findings became more prominant. The epithelium of the gastric mucosa was obviously ischemic and hypoxic. These findings suggest that in cirrhotic protal hypertension, the defensive capacity of the gastric mucosa is weakened to result in the pathological changes of the gastric mucosa. The portoazygos disconnection further weakens the defensive capacity of the gastric mucosa, which might be one of the causes of high rate of hemorrhage after disconnection.

Key words gastric mucosa; liver cirrhosis; hypertension portal; portoazygos disconnection

慢性呼吸衰竭带3蛋白及酸碱调节的研究

The study of band 3 protein and acid-base regulation in chronic respiratory failure

杨晓静 毛宝龄 钱桂生 (第三军医大学新桥医院呼吸中心) 630038

慢性呼吸衰竭是临床常见危重并发症,常与酸碱失衡和电解质紊乱共存,是致死的重要原因之一。有关细胞内酸碱变化及其调节机制,尤其是带3蛋白阴离子转运功能在酸碱调节中作用的研究才刚刚起步,国内外报道不多。

本研究分为实验研究与临床研究两部分,通过对慢性呼吸衰竭病人及带压缺氧、缺氧伴高二氧化碳大鼠观察:从红细胞膜带3蛋白结构、含量和阴离子转运功能,红细胞内外血气和电解质及肾带3蛋白的作用和意义。结果发现:(1)直接向放动物的舱内灌入低氧或低氧伴高二氧化碳的混合气体,可在短时间内使大鼠发展成典型的肺动脉高压和右心室肥厚模型;(2)肺心病Ⅰ、Ⅱ型呼吸衰竭病人及常压缺氧和缺氧伴高二氧化碳大鼠红细胞带3蛋白阴离子转运区域——TrC55肽段各氨基酸残基数与对照组无显著差别($P>0.05$);(3)肺心病Ⅱ型呼吸衰竭病人带3蛋白相对含量明显低于Ⅰ型和对照组($P<0.01$)。常压缺氧大鼠带3蛋白相对含量显著地低于对照组($P<0.05$),缺氧伴高二氧化碳大鼠带3蛋白相对含量非常显著地低于对照组($P<0.01$);(4)肺心病Ⅱ型呼吸衰竭病人及常压缺氧和缺氧伴高二氧化碳大鼠红细胞膜对Cl^-通透系数(P_{Cl})均明显低于对照组($P<0.01$)。动态观察Ⅱ型呼吸衰竭病人的P_{Cl},发现P_{Cl}在急性期明显降低,随着病情好转逐趋正常,各期间差别非常显著($P<0.01$);(5)带3蛋白阴离子转动特异性抑制剂4,4′—二硫氰基—2,2′二碳基芪(DIDS)对人和大鼠各组的P_{Cl}影响程度一致,在DIDS终浓度为0.5 μmol/L时,各组P_{Cl}下降比值无显著差别($P>0.05$);(6)速尿、双氢克尿塞和安定对肺心病Ⅰ、Ⅱ型呼吸衰竭病人和正常健康人的P_{Cl}均产生明显抑制,其50%抑制的药物浓度分别为2×10^{-4} mol/L、5×10^{-3} mol/L、2×10^{-3} mol/L,去除药物成份后,P_{Cl}可恢复正常;(7)肺心病Ⅱ型呼吸衰竭组的pH_i、$[HCO_3^-]_i$均明显高于对照组和Ⅰ型呼吸衰竭组($P<0.01$)。带压缺氧大鼠的红细胞内外pH差值(pH_{e-i})和红细胞内pH下降比值($pH_{\frac{e-i}{e}}$)显著地低于对照组($P<0.05$);常压缺氧伴高二氧化碳大鼠的pH_{e-i}和$pH_{\frac{e-i}{e}}$非常显著地低于对照组和常压缺氧组($P<0.01$);(8)慢性缺氧伴高二氧化碳大鼠及CO_2潴留2 d和5 d大鼠的肾皮质、髓质带3蛋白mRNA相对含量均明显高于其对照组($P<0.01$)。

以上结果表明:(1)慢性常压缺氧和缺氧伴高二氧化碳肺动脉高压模型类似于平原肺心病发病情况,可供实验研究用;(2)肺心病Ⅱ型呼吸衰竭病人及慢性常压缺氧和缺氧伴高二氧化碳大鼠红细胞膜带3蛋白阴离子转动位点结构可能无异常,但阴离子交换功能降低,HCO_3^-/Cl交换受限,阻止红细胞内HCO_3向细胞外转移,使红细胞内呈偏碱状态;(3)肺水病Ⅱ型呼吸衰竭病人带3蛋白阴离子转动功能降低是是可逆的。因此,及时纠正缺氧,也许有助于阴离子转动功能的恢复;(4)速尿、双氢克尿塞、安定可快速、可逆地抑制带3蛋白阴离子转动功能,使HCO_3/Cl交换受限,减少CO_2排出。因此,肺功能不显著,特别是肺心病Ⅱ型呼吸衰竭病人,应慎用此类药物;(5)慢性高碳酸血症大鼠肾带3蛋白mRNA表达水平明显增加,此种适应性改变也许在酸碱调节中起重要作用。

关键词 呼吸功能不全;缺氧;二氧化碳;区带3蛋白质;阴离子;酸碱平衡

中图法分类号 R563.8

?1994-2016 China Academic Journal Electronic Publishing House. All rights reserved. http://www.cnki.net