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Study on the role of active oxygen in the pathogenesis of respiratory distress syndrome in dogs

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Abstract Twenty-five mongrel dogs were randomized into 3 groups; the animals of group Ⅰ were traumatized with bone marrow extract, those of group Ⅱ were similarily traumatized and then treated with anisodamine, and those of group Ⅲ received saline only and served as control. The specimens of arterial and venous blood were collected and bronchoalveolar lavage fluid (BALF) and lung homogenate were obtained after the animal was killed. The activity of superoxide dismutase (SOD) and the concentration of lipid peroxides (LPO) were determined. Meanwhile, the clinical manifestations, blood gas analysis, chest radiography, and pathological examinations were performed or observed. It was found there were following findings: (1) In group Ⅰ, SOD activity and LPO level in the lung homogenate and BALF were markedly elevated immediately after injury, which suggests that there had been a rapid production of active oxygen. As a result, various degrees of lung damages were pricipitated. (2) In group Ⅱ, an increase of SOD activity and LPO level in the blood, lung homogenate and BALF: The elevation was more marked than that in group Ⅲ but less marked than that in group Ⅰ, which indicates that there was a relatively of less amount of active oxygen production by the interference anisodamine. (3) In group Ⅲ, no significant changes of SOD activity and LPO livel were found.

Our findings suggest that active oxygen is likely to play a very important role in the pathogenesis of acute lung damages in respiratory distress syndrome.

Key words oxygen; superoxide dismutase; lipid peroxides; dogs

多腺体自身免疫综合征Ⅱ型一例报告

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患者 女,52 岁,1990 年 9 月入院。21 年前曾患甲亢,行甲状腺次全切除术,次年复发经放射性碘治愈。18 个月以前,感皮肤紧束,四肢无力,经血清学及皮肤活检等检查,诊为皮肌炎,强的松 30mg/d 治疗 2 个月,症状缓解后减至小剂量维持。12 个月前现多饮、多尿、消瘦,空腹血糖>11.1mmol/L,诊为Ⅰ型糖尿病(NIDDM),间断服 $D_{860}1\sim3g/d$,血糖较稳。近一月,感极度疲乏、易感冒。家族性阴性。体检:血压 20/13kPa,体重 40kg,皮肤粘膜无色素沉着,无突眼,甲状腺未及,心肺无异常发现,肌力正常,阴腋毛稀少。辅助检查:心肌酶谱、TT_3、TT_4、TSH、血生化均正常,血脂略高,血沉 35mm/h,FBG 9.0mmol/L,晚餐前血糖 17mmol/L,尿酮体±,4 次尿糖 −、卄、卅、卅,RF(-),Tg-Ab 及 Tm-Ab(-),胰岛素释放试验:各点分别为:79.6 pmol/L、148.5 pmol/L、185.9 pmol/L、178.6 pmol/L、165.7 pmol/L。24h 尿蛋白 0.6g、糖 48.3mmol/L,肝胆胰脾肾及肾上腺 B 超未见异常。蝶鞍片正常。入院诊断:NIDDM、甲亢;甲状腺次全切除术后及放疗后,皮肌炎。

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给予强的松 15mg/晨,优降糖 2.5mg/早、晚,降糖灵 75mg/d,血糖在 2 周后缓降至正常范围,尿糖转阴。半月后,多次在午夜 2～5 时出现心悸、大汗、头晕,两次测得血糖为 3.0mmol/L、5.6mmol/L,随即停用全部降糖药,半月后仍有上述症状,但程度略减轻,在排除饮食和药物因素后,连续测定夜间 23 时、1 时、3 时、7 时血糖,生长激素、胰岛素、糖皮质激素,胰高糖素水平,结果:血糖 1.44～3.6mmol/L, 血浆皮质醇分别为:66.21nmol/L、38.63nmol/L、33.12nmol/L、41.4nmol/L,余激素水平正常。皮质醇节律测定呈低水平曲线,晨 8 时无峰值。尿 17-OH 及 17-KS 明显低于正常。因缺药,未行 ACTH 兴奋试验。依检查及临床表现,诊断:多腺体自身免疫综合征Ⅰ型。经调整强的松剂量与用法,中、晚餐前服 D_{860}0.5g,患者白天高血糖得到控制,夜间低血糖现象消失,症状改善出院。

讨论

多腺体自身免疫综合征(PGAS)是指患一种内分泌腺功能减退的病人伴有另一种或多种其它内分泌靶腺特异性或/和非器官特异性自身免疫性疾病。其中甲状腺、甲状旁腺、肾上腺、胰岛及性腺最易受累。根据受累器官不同常分Ⅰ、Ⅱ、Ⅲ型。本例根据患者有甲亢、皮肌炎和肾上腺皮质功能减退,诊为 PGAS Ⅱ型。较特殊的是通过寻找低血糖原因而发现了肾上腺皮质功能减退,而此时尚未出现皮肤色素沉着。低血糖是慢性肾上腺皮质功能减退常见的症状,临床上常因表现不典型而被忽略,本例低血糖突出,在于患者同时存在 NIDDM,按常规剂量投入了降糖药,使低血糖表现明显化。

PGAS 是一种自身免疫性内分泌疾病,从大量的研究已得到证实[2][3][4][5]。由于各腺体病变出现可前可后,表现变化不一,诊断十分困难,提高对本病的认识非常重要。治疗上无固定模式,具体病人具体对待。若患者抗体滴度高可考虑用免疫抑制剂、血浆置换[1]。

关键词 内分泌疾病;肾上腺皮质疾病

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