

论著

重症及危重症甲型H1N1流感患者发病机制探讨

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摘要:

目的探讨重症及危重症甲型H1N1流感(甲流)患者的发病机制。方法分析某院收治的重症及危重症甲流患者外周血细胞在治疗前后的变化,同时通过流式细胞仪结合单克隆抗体动态观察危重症甲流患者淋巴细胞协同刺激分子与凋亡情况的变化。结果重症及危重症甲流患者治疗前白细胞数为 $(2.55\pm 0.87)\times 10^9/L$ ,淋巴细胞数为 $(1.02\pm 0.54)\times 10^9/L$ ,与治疗后白细胞数 $(6.95\pm 4.36)\times 10^9/L$ 及淋巴细胞数 $(2.13\pm 0.78)\times 10^9/L$ 比较,差异有统计学意义(分别 $t=3.28,P=0.01$ ;  $t=9.52,P=0.00$ );而治疗前红细胞数 $(4.10\pm 0.45)\times 10^{12}/L$ 与治疗后红细胞数 $(4.14\pm 0.39)\times 10^{12}/L$ 比较,差异无统计学意义( $t=0.35,P=0.73$ )。随着病情的好转,危重症甲流患者淋巴细胞数逐渐增多,同时其表面的协同刺激分子CD28、CD152表达降低,患者淋巴细胞的凋亡信号CD95也逐渐减低。结论甲流病毒诱导T淋巴细胞活化、增殖障碍及过度启动T淋巴细胞凋亡可能与淋巴细胞数及白细胞数量下降有关,这可能在重症及危重症甲流患者的发病机制中起非常重要的作用。

关键词: 流感 甲型H1N1流感 红细胞 白细胞 淋巴细胞 协同刺激分子 凋亡

Investigation on the pathogenesis of severe and critical H1N1 influenza A

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Abstract:

Objective To investigate the pathogenesis of severe and critical H1N1 influenza A. Methods Before and after medical treatment, changes in peripheral blood cells of patients with severe or critical H1N1 influenza A were analyzed, and changes in co-stimulatory molecules and apoptosis on lymphocytes of patients with severe or critical H1N1 influenza A were dynamically observed via flow cytometry and monoclonal antibodies. Results For patients with severe or critical H1N1 influenza A, the count of leukocytes and lymphocytes before treatment were  $(2.55\pm 0.87)\times 10^9/L$  and  $(1.02\pm 0.54)\times 10^9/L$ , respectively, after treatment were  $(6.95\pm 4.36)\times 10^9/L$  and  $(2.13\pm 0.78)\times 10^9/L$ , respectively, there was significant difference ( $t=3.28,P=0.01$ ;  $t=9.52,P=0.00$ ); count of erythrocyte before and after treatment were  $(4.10\pm 0.45)\times 10^{12}/L$  and  $(4.14\pm 0.39)\times 10^{12}/L$ , respectively, there was no statistical difference ( $t=0.35,P=0.73$ ). During the recovery phase, lymphocyte count increased gradually, the expression of co-stimulatory molecule CD28 and CD152, and the apoptosis signal CD95 of T lymphocytes decreased. Conclusion H1N1 influenza virus A can repress the activation and proliferation of T lymphocytes, and induce over-activation apoptosis of T lymphocytes, cause the decrease in lymphocyte and leukocyte count, which may play an important role in the pathogenesis of severe and critical H1N1 influenza A.

Keywords: influenza H1N1 influenza A erythrocyte leukocyte lymphocyte; co-stimulatory molecule apoptosis

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