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SCIENTIFIC LETTER

Immune Mechanism of Hand Foot and Mouth Disease Sepsis

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To the Editor: The pathogenesis of sepsis has not been very clear. In the past, it was believed that sepsis mainly results from tissue and organ damage caused by excessive inflammation. However, more and more studies have found that the pathophysiological process of sepsis is very complex, including inflammation, immune and coagulation dysfunction, and involves various changes in cell function, metabolism and microcirculation [1]. Sepsis is a highly heterogeneous syndrome that can lead to death due to systemic inflammatory response syndrome. The activation and regulation of immune system plays a key role in the occurrence, development and prognosis of sepsis [2]. In recent years, some scholars have discussed the immune mechanism of sepsis from the perspective of system theory, and put forward the theoretical viewpoint of "compensation/decompensation" of innate immunity [3]. By selecting a group of EV71 cases, the authors divided them into severe (cured) group (25 cases), critical (death) group (23 cases) and control (healthy) group (30 cases). The immune system group was divided into lymphocyte subsets and lymphocyte neutrophil system groups according to the grades. CD3 + T (%) and CD19 + B (%) were used as the observation indicators of lymphocyte subsets. The results of the control group, the severe group and the critical group were 68.50±3.36 and 14.48±3.08, 50.65±3.85 and 33.72 ±3.27, 45.91±4.35and 79.31±2.93, respectively. L (%) and N (%) were used as the observation indexes in the lymphocyte neutrophil system group. The test results of the control group, the severe group and the critical group were 50.30 ±2.55 and 36.42±3.03, 31.25±2.62 and 61.94±2.97, 15.43 ±2.76 and 79.31±2.93, respectively. White blood cells (WBC) ($\times 10^9$/L), C-reactive protein (CRP) (mg/L), glucose (GLU) (mmol/L) and platelets (PLT) ($\times 10^9$/L) were used as the observation indicators of other system groups: the control group (healthy), the severe group (cured) and the critical group (death): 6.24±1.32, 1.13±3.58, 5.21±0.33 and 215.2 ±19.29; 10.80±1.38, 6.42±0.35, 6.42±0.35 and 335.1 ±20.35; 18.68±1.64, 16.86±5.15, 7.52±0.41 and 395.6 ±21.36 respectively. The analysis of variance of the above test results showed that there were significant differences among the groups with different diseases in different systems ($P < 0.01$). The authors hold that the cellular immune function of patients with sepsis of Hand, Foot and Mouth disease (HFMD) is defective [4], and humoral immune enhancement is a compensation. The results of this study show that the immune mechanism of sepsis in Hand, Foot and Mouth disease, at all levels of the blood system, follows the rule of systemic equilibrium compensation, and the response of each level, from small to large, leads to intrinsic immune compensation/decompensation and a larger systemic compensation inflammatory response [5].

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Compliance with Ethical Standards

Conflict of Interest None.

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References

1. Yao Y-Y, Zhang Y-M. A new understanding of the pathogenesis of sepsis. J Grad Med Sci. 2017;30:678–83.
2. Qiu Y, Tu G-w, Ju M-j, Yang C, Luo Z. The immune system regulation in sepsis: from innate to adaptive. Curr Protein Pept Sci. 2019;20:799–816.
3. Yao L-L, Xiao Y, Tan H-Q. Rational analysis of pathogenesis of systemic inflammatory response syndrome. J Med Philos. 2016;37:79–81.
4. Namath A, Patterson AJ. Genetic polymorphisms in sepsis. Crit Care Clin. 2009;25:835–6.
5. Da-Rin HE. Introduction to philosophy of medical science and technology [M]. Beijing: Higher Education Press; 2005. p. 175–91.