Alterations of cell-mediated immunity following cardiac operations: clinical implications and open questions

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Summary

Cardiac surgery with cardiopulmonary bypass (CPB) is known to induce an immune response whose nature has been increasingly elucidated during the recent decade. Clinically, patients usually show two to three of the four symptoms, which define the so-called systemic inflammatory response syndrome (SIRS). In addition, all parameters of the innate, nonspecific immune system, e.g., polymorphonuclear cells, elastase, and complement, are activated. This also applies to the proinflammatory mediators interleukin (IL)-1β, -6, and -8, and tumor necrosis factor (TNF)-α. Within the adaptive, specific immune system, a decrease of T lymphocytes and T helper (TH) cells is observed, whereas suppressor/cytotoxic T cells and B cells appear to be nearly unaffected.

Cytokine measurements provide more detailed information: IL-2 and IL-12, which are important for the activation of the type-1 TH-cell (TH1)-mediated immune response, are depressed following cardiac operation. In contrast, IL-10 and transforming growth factor-β essential to TH2-mediated humoral or anti-inflammatory immune response, are upregulated. In vivo tests, e.g., delayed type hypersensitivity skin reaction and tetanus antibody production, confirm the polarization of the adaptive immune response towards the TH2 pathway.

However, all these alterations usually do not result in clinical adverse events. Therefore, more information is needed about the immune response of patients at high preoperative risk or with serious perioperative complications to find out whether clinically relevant events are correlated to alterations of immune response. For this purpose, more readily available, standardized methods for immunologic monitoring appear highly desirable.

References


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