SUMMARY

Most elderly people aged over 60 suffer from coronary artery disease (CAD), which can become very likely cause of death. Coronary artery bypass grafting (CABG) represents the standard way of treatment for CAD. CABG surgery is traditionally performed on an arrested heart with the use of cardiopulmonary bypass. The heart is usually accessed through a median sternotomy. Although this kind of surgery strongly stimulates both humoral and cell-mediated immunity, inflammatory response is self-terminating and only a small percentage of patients develop serious postoperative complications, such as organ failure, wound dehiscence, and sepsis.

This work focuses on regulatory mechanisms controlling inflammatory response in cardiac surgical patients. It is a retrospective study that follows changes of humoral and cell-mediated immunity induced by cardiac surgery in two different groups of patients. Both groups were operated with the same surgical approach, when identical devices and types of equipment were used; the only difference was the content of 500 mg of methylprednisolone (MP) in priming solution of cardiopulmonary bypass.

Blood samples were collected before surgery, after weaning from cardiopulmonary bypass, at the end of surgery, on the 1st, 3rd, and 7th postoperative day. Changes in number of leukocytes and percentages of myeloid populations (monocytes and granulocytes) were statistically evaluated. Expression of cell-surface molecules in these populations was measured using flow cytometry. Analyzed molecules included scavenger receptor for haptoglobin-hemoglobin (CD163), high affinity receptor Ia for Fc fragment IgG (FcγRI), Fas receptor (Fas), and Toll-like receptor 2 and 4 (TLR2 and TLR4). Concentration of interleukins IL-10 and IL-6 was quantified by ELISA.

The surgery with cardiopulmonary bypass was followed by an increase in number of leukocytes, of which granulocytes represented prevailing population.

A large quantity of anti-inflammatory molecules IL-10 and CD163 was produced during surgery, after surgery, and on the 1st postoperative day. Moreover, methylprednisolone stimulated over-production of IL-10 and CD163. Patients with organ failure showed very high values of CD163, but the highest values were measured in septic patients.

A shedding of membrane receptors TLR2 and TLR4, which signaling is connected to proinflammatory response, was another indication that mostly anti-inflammatory mechanisms took part during surgery. Expression of these receptors was increased in postoperative period, similarly to IL-6, which serum level was more elevated in patients without MP than in patients with MP.

Surprisingly, methylprednisolone did not affect FcγRI expression on granulocytes. On the contrary, methylprednisolone induced expression of Fas.

Methylprednisolone impacts regulatory mechanisms of the inflammatory response of cardiac surgical patients. In spite of this fact, methylprednisolone is unlikely to decrease or increase frequency of postoperative complications.