“Interaction of progenitor endothelial cells and NK lymphocytes in blood of patients as the sign and reason the late low survival rate after EVAR”

A.V. Svetlikov¹,², A.I. Ermakov², L.B. Gaikovaya², V.S. Gurevich¹,²,³, P.A. Galkin¹, L.E. Ishpulaeva¹,², G.G. Hubulava⁴

Background. The long-term outcome of EVAR are not satisfactory. In 10 years the mortality heads for 80%. It is assumed that immune and autoimmune reactions can play a role in the development and progression of abdominal aortic aneurysms (AAA). The aim of this study was to assess the content of circulating natural killer cells (NK) in the pre- and postoperative period of endovascular aneurysm repair (EVAR) of AAA and its comparison with the level of circulating endothelial cells (CEC).

Materials and methods. NK and CEC were counted by flow cytometry in blood samples of patients before EVAR, within 2 weeks and in 6 months after the operation. Markers (CD16 + CD56 +) and (CD146 + CD45 -), respectively, were used to identify NK and CEC.

Results. CEC levels in patients with AAA are significantly increased in comparison with healthy donors (22.1 ± 2.9 cells /μl and 3.0 ± 0.5 cells /μl, respectively). In the early postoperative period (Pic.1,3), a slight insignificant decrease in CEC levels was observed. However in the remote postoperative period a statistically significant decrease in the level of the CEC was detected (p < 0.05) (Pic.1,4).

The level of NK in early postoperative period (Pic.2,5) was significantly decreased compared to the preoperative level (212.9 ± 23.6 cells /μl and 326.5 ± 30.3 cells /μl, respectively, p < 0.05) and increase in the long-term postoperative period (Pic.2,6).

A negative correlation was found between NK and CEC after EVAR (r = -0.440, p < 0.02)(Pic.7).

Conclusions. The obtained data confirm participation of innate immunity in the reparative process after endovascular correction of AAA. It causes the systemic chronic inflammatory reaction. That explains unsatisfactory long-term outcome after EVAR and point to new therapeutic targets in this disease.

2018