Streszczenie w języku angielskim

Cytokines play an important role in the immunopathology of viral infections. A rapid and well-coordinated immune system response is the first line of defence in a viral infection. However, a disturbed, over-activated immune response can be counterproductive, causing damage to the organism. The 'cytokine storm' phenomenon is a pathological response of the immune system characterised by rapid proliferation and hyperactivation of T lymphocytes, macrophages, and NK cells, as well as overproduction of pro-inflammatory cytokines and chemical mediators released by the body's immune and non-immune responses. A growing number of studies suggests that the Th17 response plays an important role in the pathogenesis of COVID-19-induced pneumonia. The immune response is exacerbated by releasing cytokines such as IL-17 and GM-CSF, promoting neutrophil migration and attenuating the Treg response. Suppression of the excessive immune response by timely administration of GCS in the early stages of the 'cytokine storm' effectively prevents acute respiratory failure, protecting patients' vital functions.

The aim of this study was analysis of the effect of treatment on the course of the 'cytokine storm' in patients with COVID-19, with emphasis on immunomodulatory treatment, and evaluation of selected subpopulations of lymphocytes T and cytokine profile released during SARS-CoV-2 infection and their correlation with the severity of the disease.

The study showed that CCL5/RANTES, GM-CSF, IL-4, IL-6, IL-10 and CXCL10/IP-10 levels were significantly elevated in COVID-19 patients compared to healthy volunteers. Furthermore, on admission, patients with severe disease had significantly higher levels of IL-10 and CXCL10/IP-10 than patients with moderate disease. The administration of antiviral treatment resulted in a significant decrease in IL-6, IL-10, IFN-alpha and CXCL10/IP-10, while the administration of the immunomodulatory treatment contributed to a significant decrease in IL-10, IFN-alpha, CXCL10/IP-10 and B7-H3, as well as an increase in IL-22 and IL-1 beta. Whereas the combination of antiviral and immunomodulatory treatment resulted in a significant decrease in levels of IL-17F, IL-10, IFN-alpha, CXCL10/IP-10 and B7-H3, and an increase in IL-17A and IL-1 beta. COVID-19 patients also had a significantly higher proportion of IL-17A-producing CD4+ and CD8+ T cells and IL-22-producing CD4+ T cells.

The following conclusions were drawn from the study:

1. The administration of antiviral and/or immunomodulatory treatment resulted in a significant reduction in pro-inflammatory cytokine expression and an increase in absolute T-cell count.
2. SARS-CoV-2 infection causes increased cytokine expression in hospitalised COVID-19 patients, as well as lymphopenia, in particular a decrease in CD4+ and CD8+ T-cell counts.
3. Despite reduced CD4+ and CD8+ T-cell counts, both subgroups showed over-activation and increased expression of IL-17A and IL-22, thus targeting Th17 response may alleviate the inflammatory response in patients with severe disease.