SUMMARY

The natural course of psoriasis is characterized by long-term persistence of skin lesions and predilection for relapse in the same area despite effective treatment. It is caused by the inherence of tissue-resident memory cells (TRM) in apparently healthy skin, which are able to initiate an inflammatory cascade and induce relapse of the disease. These cells are characterized by high resistance to damaging factors and apoptosis, which determines their longevity.

The aim of the study was to determine the location and amount of TRM markers in psoriatic plaques in 32 patients compared to 10 healthy volunteers, and to analyze the relationship between the expression of TRM markers and the duration and severity of the disease. The number of TRM markers in psoriatic lesions was also assessed before, during and after 12 weeks of systemic therapy with methotrexate (MTX) or secukinumab or ixekizumab (anti-IL-17) or adalimumab (ADA), as well as topical therapy with calcipotriol and betamethasone dipropionate (Cal/BD) – a total of 23 patients. For this purpose, the amount and localization of TRM markers (CD4, CD8, CD103, CD69, CD49, CXCR6) and the tissue expression of cytokines (IL-17A, IL-22) in the lesional psoriatic skin were assessed using immunohistochemistry. Biopsy samples were taken from the same area before treatment and after 4 and 12 weeks of treatment. The obtained results were subjected to statistical analysis, which allowed the following conclusions to be drawn:

1. patients diagnosed with plaque psoriasis have an increased amount of TRM markers in the epidermis and dermis compared to the control group,
2. there is a clear positive relationship between the expression of TRM markers in patients with plaque psoriasis and the duration of the disease,
3. no relationship was found between the number of TRM markers and the intensity of skin lesions (assessed according to the PASI, BSA, DLQI scales),
4. a decrease in the expression of TRM markers in psoriatic lesions was demonstrated over time with each systemic therapy (anti-IL-17, ADA, MTX), and the fastest reduction in the expression of TRM markers concerned anti-IL-17 therapy in the 4th week of treatment,
5. during the 12-week observation, general therapy (anti-IL-17, ADA, MTX) reduces the expression of TRM markers mainly in the dermis of the lesions, not in the epidermis,
6. during the 12-week observation, topical Cal/BD therapy reduces the expression of TRM markers mainly in the epidermis, but does not significantly affect their expression in the dermis,

7. decrease in TRM markers was observed only in the 12th week of topical therapy with Cal/BD.