Psoriasis, an autoimmune disease promoted by defective innate immunity

Helgi Valdimarsson
Landspitali University Hospital, Iceland

It is now generally accepted that psoriasis is a T-lymphocyte mediated inflammatory disease. Early data supporting this concept, published about 25 years ago, were initially subjected to vigorous and healthy scepticism and discussed as current controversy as late as 2000. However, this paradigm is now generally accepted and management with agents that selectively block certain T-lymphocyte sub-populations is now the treatment of choice for patients with moderate to severe psoriasis. The nature of the antigens involved, remains to be fully established but some recent findings have strongly implicated streptococcal components. Genetic analyses have identified a number of susceptibility alleles that may predispose to psoriasis, including the HLA-Cw6 allele that is carried by about 60% of the patients, compared with only about 15% of population controls. Many other susceptibility alleles with lower penetrance have been identified and some may be associated with defects in innate immunity. Although the innate immune system does not directly involve antigen specific immune mechanisms, it closely interacts in a variety of ways with adaptive immunity. Thus, it is well established that defective innate immunity predisposes to antigen specific autoimmune diseases. HLA-Cw6 positive psoriasis patients respond abnormally to short keratin peptides that share sequences with streptococcal M protein and vice versa. Furthermore, improvement of psoriasis after tonsillectomy correlates closely with decrease in the frequency of circulating CD8 T lymphocytes that recognize such peptides. It is therefore proposed that HLA-Cw6 positive psoriasis patients have molecular mimicry based autoimmunity. Whether other mechanisms operate in HLA-Cw6 negative psoriasis patients remains to be elucidated.

helgiv@landspitali.is

Innate immunity of keratinocytes in rosacea & perioral dermatitis

Irina Khamaganova1, N N Potekaev1,2,3, O L Novozhilova1, O A Svitich1 and Ly Gankovskaya1

1Pirogov Russian National Research Medical University, Russia
2Moscow Health Department, Russia
3Moscow Scientific & Practical Centre for Dermatology & Cosmetology, Russia

Rosacea is a common inflammatory facial skin disease, characterized by erythema, telangiectasia, papules and pustules. Perioral dermatitis is a common inflammatory facial skin disorder as well. A typical perioral dermatitis presentation involves the eruption of papules and pustules that may recur over weeks to months, occasionally with fine scales. The differential diagnosis includes rosacea besides other diseases. The dysregulation of the innate immune system may have a role in promoting the clinical features of rosacea & perioral dermatitis. The objective of this study was the complex analysis of innate immunity of the keratinocytes in patients with rosacea & perioral dermatitis. Materials & methods: 35 patients with rosacea & 5 patients with perioral dermatitis were examined. Total RNA was isolated from keratinocytes RNA was combined with random and reverse primers for the target genes for cDNA synthesis. The real-time PCR was performed for quantitative analysis. The median of the expression of TLR2 was significantly reduced both in the affected skin to 3856 and in healthy skin in rosacea patients to 2627 in comparison with 34191 in patients with perioral dermatitis. The median of the expression of hBD1 was significantly raised both in the affected skin to 28318 and in healthy skin in rosacea patients to 24732 in rosacea patients in comparison with 7415. The median of the expression of hBD2 was significantly reduced both in the affected skin to 2267 and in healthy skin in rosacea patients to 1990 in comparison with 5553. The investigation showed statistically significant difference of the indicators of innate immunity in patients with rosacea & perioral dermatitis.