Growth Factors in Psoriasis and Mycosis Fungoides

Thesis

Submitted for the partial fulfillment of MD degree in Dermatology and andrology

Submitted by

Mohamed Hassan Mohamed

(MSc)

Supervisors

Prof Dr. Mohamed Ibrahim EL-Zorkany

Professor of Dermatology and andrology

(Faculty of Medicine (Girls

Al-Azhar University

Prof Dr. FatenAbd EL-WadodAbd EL-Kawey

Professor of Dermatology and andrology

(Faculty of Medicine (Girls

Al-Azhar University

Prof Dr. Nayera Hassan Moftah

Professor of Dermatology and andrology

(Faculty of Medicine (Girls

Al-Azhar University

Prof Dr. OlfatGamil Shaker

Professor of Biochemistry

Faculty of Medicine

Cairo University

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Psoriasis is a common genetically determined inflammatory and proliferative disease of the skin and joints, It affect ٠.٣ – ٢.٥ % of the general population,The most common form of psoriasis is plaque psoriasis, Although psoriasis is characterized by proliferation of the epidermis, the immune system has a prominent role in development of this disease.

There is parakeratosis associated with focal orthokeratosis and Munro micro abscesses formation. In addition, there is near absence of granular layer, spongiform pustules in the malpighian layer, hyperplasia with elongation of rete ridges and small epidermal thinning. Dilated tortuous capillary blood vessels almost touch the under surface of the thin suprapapillary epidermis, as well as extravasatederthrocytes. Of all the listed features, only the spongiform pustules of Kogoj and Munro micro abscesses are truly diagnostic of psoriasis.

Aetiopathogenesis of psoriasis

I- Genetic

The genetic determinants of psoriasis have been long studied. At least three lines of evidence support the role of hereditary transmission in the development of psoriasis **(Anne and William, ٢٠٠٤)**. First, there is a strong familial association of the disease approximately one-third of patients with psoriasis have a history of other family members with the disease. Secondly, as previously noted, there is a strong association between psoriasis and HLA markers, particularly in patients with early-onset disease. A number of HLA class I and II antigens have shown a positive association with psoriasis, including HLA-B ١٣, -B ١٧, -B٣٩, -B٥٧, -Cw٦, -Cw٧, -DR٤, and –DR٧ **(Schon and Boehncke, ٢٠٠٥),** through up till now the strongest genetic association for psoriasis is with HLA gene cluster on chromosome ٦p٢١, and there is a linkage with the HLA-Cw٦ allele, Thirdly, the analysis of ten pairs has revealed ٧٢% concordance for psoriasis among pairs of monozygotic (genetically identical) twins, but only ٢٢% concordance among dizygotic twins.

The other genes (on different chromosomes other than chromosome ٦) have been studied. **Enerback et al., (١٩٩٩)** revealed an association of psoriasis with a region on chromosome ١١q, . Other loci identified to be associated with psoriasis include: locus on chromosome ٢p (١٢-١٣) **(Veal et al., ٢٠٠١),** loci on chromosomes

٨q٢٤.١١ and ٢٠p١٣ **(Trembath et al., ١٩٩٧),** locus on chromosome ٣q٢١ **(Enlund et al., ١٩٩٩),** locus on chromosome ١p, locus on chromosome ١٩p١٣ **(Lee et al., ٢٠٠٠),** locus on chromosome ١٤q and a suggested locus on chromosome ٢١q.

II- IMMUNOLOGICAL ROLE IN PATHOGENESIS OF PSORIASIS:

Psoriasis is genetically programmed disease of dysregulated inflammation, which is driven and maintained by multiple components of immune system. The pathologic collaboration between innate immunity (mediated by antigen-presenting cells and natural killer T-lymphocytes) and acquired immunity (mediated by T-lymphocytes); results in the production of cytokines, chemokines and growth factors. These contribute to the inflammatory infiltrate seen in psoriatic plaques.Hyperplasia and altered differentiation of epidermal keratinocytes (KCs) are classic features of psoriatic lesions. It is recognized that epidermal hyperplasia is a reaction to the activation of the immune system in focal skin regions stimulated by cytokines released from both T-lymphocytes and keratinocytes