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An Overview of Psoriasis Vulgaris

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ABSTRACT

Psoriasis is a chronic inflammatory skin disease with a genetic basis, characterized by impaired growth and differentiation of the epidermis, multiple biochemical, immunological, and vascular disorders. The aetiology of psoriasis is not known with certainty, thought to be triggered by an immune response due to environmental factors such as trauma, infection, or drugs in individuals who are genetically at risk of psoriasis. The pathogenesis of psoriasis begins with an inflammatory immune response that promotes the release of dendritic cells, T cells, keratinocytes, neutrophils, and cytokines.

1. Introduction

Psoriasis is a chronic inflammatory skin disease with a genetic basis, characterized by impaired growth and differentiation of the epidermis, multiple biochemical, immunological, and vascular disorders. [1,2] The aetiology of psoriasis is unknown; it is thought to be triggered by an immune response due to environmental factors such as trauma, infection, or drugs in individuals who are genetically at risk of psoriasis.[1] The pathogenesis of psoriasis begins with an inflammatory immune response that promotes the release of dendritic cells, T cells, keratinocytes, neutrophils, and cytokines. [2,3]

Psoriasis can affect all races and is found almost all over the world. The highest incidence rates are in Europe, Denmark with 2.9% cases and the Faeroe Islands with 2.8% cases. The incidence of psoriasis in Asians is low, i.e. 0.4% of cases.[1] Based on data from inpatient visits from the Department of Dermatology and Venereology, Dr Mohammad Hoesin Palembang from January 2012 to December 2017 found 57 new moderate to severe cases of psoriasis.

Psoriasis pathogenesis

The pathogenesis of psoriasis involves dynamic interactions between various cell types and cytokines in response to precipitating factors resulting in impaired skin immune homeostasis in genetically predisposed individuals.[4,5] The pathogenesis of psoriasis involves both innate and adaptive immune responses. Traditionally it is divided into two phases,

namely the initiation phase and the maintenance phase.

Several precipitating factors, including trauma, infection, and certain drugs, disrupt keratinocytes in the epidermis, which causes the release of self-deoxyribonucleic acid (self DNA) and self-ribonucleic acid (self RNA), which form complex bonds with antimicrobial peptide LL-37 (cathelicidin). The self DNA complex and LL-37 bind to toll-like receptor 9 (TLR9) on plasmacytoid dendritic cells (PDC) in the dermis. Plasmacytoid dendritic cells (PDC) secrete interferon type 1 (IFNα and), TNFα, IL-6, and IL-1β, which encourage myeloid dendritic cells (mDC) to migrate to nearby lymph nodes. Upon contact with latent nave T cells, mDCs secrete cytokines, such as TNFα, IL-12, and IL-23, which stimulate T cells to differentiate into mature Th1, Th17, and Th22 cells. LL-37 can also bind to self RNA. and directly activate mDC through TLR7 and TLR8, resulting in upregulation of TNFα and IL-6.

Studies in mice have shown that the TLR7/8 agonist imiquimod has been shown to induce psoriasiform skin inflammation. Inflammation is inhibited in mice lacking IL-23 or IL-17 receptors. This suggests a role for keratinocytes and IL-23/T17 in the pathogenesis of psoriasis. Upon returning to the skin, specific T cells release TNFα, IFNγ, IL-17A and F, IL-22, which stimulate keratinocytes for proliferation and differentiation. Erythema is associated with angiogenesis, dilatation and damage to blood vessels in the papillary dermis. Thickening of the skin is associated with acanthosis, and scaling is associated with accelerated proliferation and differentiation of keratinocytes.[6-8]

Proinflammatory cytokines increase the activation of existing mature T cells and close the loop of the chronic inflammatory cycle indicating a maintenance phase. In the maintenance phase, cytokine-secreting keratinocytes recruit neutrophils that enter the skin through the circulation and collect into Munro microabscesses in the epidermis. Macrophages are also recruited to the site of inflammation and produce TNF . Trigger factors such as trauma and infection cause the release of self-DNA and self RNA, which complexes with LL-37 and activates plasmacytoid dendritic cells (pDCs) and myeloid dendritic cells (mDCs), respectively. pDCs secrete type I interferon (IFN) and other cytokines, including TNFα, IL-6 and IL-1β, which promote the activation of mDCs. These antigen-presenting cells release proinflammatory cytokines that promote T cell-mediated inflammation and keratinocyte activation and proliferation. This promotes the recruitment and further activation of inflammatory cells such as neutrophils and macrophages, which contribute to the formation of inflammatory skin plaques and Th1 cytokines secreted by T cells.[9-10]

2. Conclusion

The pathogenesis of psoriasis involves dynamic interactions between various cell types and cytokines in response to precipitating factors resulting in impaired skin immune homeostasis in genetically predisposed individuals. The pathogenesis of psoriasis involves both innate and adaptive immune responses. Traditionally it is divided into two phases, namely the initiation phase and the maintenance phase.

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