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PSORIASIS. ETIOLOGY, PATHOGENESIS, CLINICAL FORMS

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Psoriasis is a chronic inflammatory skin disease with a strong genetic predisposition and autoimmune pathogenic traits. The worldwide prevalence is about 2%, but varies according to regions. It shows a lower prevalence in Asian and some African populations, and up to 11% in Caucasian and Scandinavian populations. The World Health Organization recognized psoriasis as a serious non-communicable disease and highlighted the distress related to misdiagnosis, inadequate treatment and stigmatisation of this disease.

Psoriasis presents in multiple forms such as plaque, flexural, guttate, pustular or erythrodermic. The most common form is plaque psoriasis, which presents as well-demarcated salmon pink plaques with silvery-white scale, typically in a symmetrical distribution and affecting the extensor surfaces (especially elbows and knees), trunk and scalp. Flexural psoriasis presents without much scaling and may affect the axillae, sub-mammary and genital areas. Guttate psoriasis causes an acute symmetrical eruption of drop-like papules/plaques mainly involving the trunk and limbs, that is classically but not always preceded by streptococcal infection. Patients with guttate psoriasis may later develop plaque psoriasis. In rare cases of severe uncontrolled disease, psoriasis causes a widespread erythematous rash (erythroderma) that is life-threatening due to potential complications including hypothermia, risk of infection, acute kidney injury and high-output cardiac failure.

Etiology and pathogenesis. The precise etiology of psoriasis still remains unknown but a few are believed to be responsible. First of all genetic factor - mode of inheritance is uncertain but is thought to be polygenic. HLA system provides a potential genetic marker. Different groups of psoriatic population showed significant association with HLA Cw6, B13, B16, and B27.

The next factor is connected with epidermis. The epidermis and dermis usually contain increased numbers of several different types of immune cells, including activated T cells, activated antigen-presenting cells (APCs), Langerhans cells, other dendritic cells and macrophages), neutrophils, and hyperproliferating keratinocytes.

And finally activation of APCs, keratinocytes or dermal cells results in induction of antigen presentation, cytokine release, and enhanced T-cell activation plus lymphokine release. Lymphokines, in turn, produce inflammation and hyperproliferation of epidermal cells.

Environmental triggers have been known to exacerbate psoriasis such as obesity, stress, beta-blockers, smoking and lithium.

The symptoms of psoriasis can sometimes go through cycles, flaring for a few weeks or months followed by periods when they subside or go into remission. Mild psoriasis can often be successfully treated with creams or ointments, while moderate and severe psoriasis may require pills, injections, or light treatments. Managing common triggers, such as stress and skin injuries, can also help keep the symptoms under control.

Having psoriasis carries the risk of getting other serious conditions, including psoriatic arthritis (a chronic form of arthritis that causes pain, swelling, and stiffness of the joints and places where tendons and ligaments attach to bones); cardiovascular events, such heart attacks and strokes; mental health problems, such as low self-esteem, anxiety, and depression. People with psoriasis may also be more likely to get certain cancers, Crohn's disease, diabetes, metabolic syndrome, obesity, osteoporosis, uveitis (inflammation of the middle of the eye), liver disease, and kidney disease.

Proper treating psoriasis based on the deep knowledge about etiology and pathogenesis can help improve symptoms as well as lower the risk of developing psoriasis comorbidities and significantly improve the quality of patient's life.