Review Article

A Brief Epidemiologic Review of Psoriasis

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Abstract

Psoriasis is an intricate, long-lasting, immune-mediated inflammatory skin condition that has a genetic origin. The body is affected to varying degrees; it can affect almost the entire body or just a few isolated red, scaly plaques. Psoriatic lesions may remain unchanged for years, growing, decreasing, and regressing over time. As the patient ages, the condition could develop worse or "wax and wane" in intensity. Indeed, examples of primary skin lesions are pustules, macules, papules, and plaques. Psoriasis, however, tends to affect more than just the skin and nails; it can cause inflammatory arthritis and inflammatory bowel disease. Additionally, patients' symptoms may vary greatly from one another. Skin appearances can manifest at any age and can be either monomorphic or polymorphic. Furthermore, numerous emotionally charged, stressful physiological and psychological events, systemic infections, and environmental factors are associated with the beginning and worsening of the condition. The underlying patho-mechanisms involve complex interactions between the innate and adaptive immune systems. T cells interact with dendritic cells, macrophages, and keratinocytes, which their secreted cytokines can mediate. Biologics targeting tumor necrosis factor, interleukin IL-23, and IL-17 have been developed and approved for treating psoriasis in the past decade. These biologics have dramatically changed the treatment and management of psoriasis.

Keywords: Decompression Sickness (DCS), diagnosis, epidemiology, etiology, High-Frequency Ultrasound (HFUS), Human Immunodeficiency Virus (HIV), psoriasis, Psoriasis Area and Severity Index (PASI) score

Introduction

The immune system's chronic inflammatory skin illness psoriasis has a connection to several morbidities, including psoriatic arthropathy, hepatic, mental, and cardiovascular. In 2014, psoriasis was acknowledged by the WHO (World Health Organization) as a notable noncommunicable illness and noted the misery that a misdiagnosis might cause, inadequate treatment, and the stigma associated with the condition.[1] Psoriasis caused 5.6 million disability-adjusted life years (DALYs) across all age groups in 2016, the Global Burden of Disease Study states. This is at least three times more than the number of DALYs caused by inflammatory bowel disease.[2] No single instrument has been established for psoriasis, and none of the severity scores now employed for the condition match all of the validation requirements for the perfect assessment tool, according to multiple published systematic reviews.[3] Currently, in order to satisfy all standards, it is advised to combine two or more scores. Therefore, continuous study is needed to both produce newer and better scores and validate those that already exist. Like other medical fields like psychiatry and rheumatology, consensus on the pertinent outcome measures that are widely acknowledged requires an international collaborative approach.[4] Psoriasis is systematically characterized by alterations in cytokine production, vascular expansion, leukocyte infiltration, and abnormal keratinocyte differentiation as well as hyperproliferation in the skin.[5] The beginning and exacerbation of the illness are linked to the number of emotional, stressful, physiological, and psychological events, as well as systemic infections and environmental factors.[5] The objective of this review is to describe the epidemiologic factors associated with psoriasis.

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Epidemiology

Prevalence and incidence

Because most research was done in wealthier nations like the US, Europe, and Australia, psoriasis showed the highest prevalence and incidence in these regions.[4,6] It is estimated that two to three percent of the world's population suffers from psoriasis worldwide.[7] The relationship between genetics and environment has a substantial correlation with the disease's incidence and prevalence. Psoriasis occurs less frequently in children than in adults.

Age of onset

Although the beginning age of psoriasis can occur at any age, a bimodal distribution is typical. Seventy-five percent of cases begin before the age of forty, peaking between the ages of twenty and thirty. According to the study, the disease's frequency in adults ranges from 0.27% to 11.4%, depending on a number of variables such as age, gender, area, ethnicity, genetics, and environment. Morphological differences in psoriasis between males and females have not been documented so far. Some observations seem to suggest that a moderate to severe extent of involvement is more frequent in men than in women with respect to the distribution of clinical variants. A remarkable gender-related feature regards palmoplantar pustulosis that more commonly affects women, with a female/male ratio of 9:1.[6] Palmoplantar pustulosis has a special predilection for female smokers. A retrospective evaluation of 102 patients with adult-onset generalized pustular psoriasis reported a female-to-male ratio of 2:1.[7,8] Variables such as genetic predisposition, exposure to environmental antigens, and climate may account for the variation observed in epidemiological studies.[9] Psoriasis symptoms often become better in the summer and get worse in the winter for a large number of people.[10] Nineteen percent of Iraqis have psoriasis, according to 2009 research.[11]

Genetic basis

Although the idea of a hereditary component to psoriasis has existed for more than a century, the true establishment of the genetic component in psoriasis dates back to Lomholt's seminal epidemiologic study in 1963. Among the 30,000 people living in the Faroe Islands, he evaluated almost 11,000 people, including psoriatic sufferers and their unaffected relatives. The incidence of psoriasis was much greater among first- and second-degree relatives of individuals with psoriasis, which led him to conclude that the condition had a clear hereditary foundation. It was not possible for Lomholt to identify a particular inheritance pattern.[12] Lomholt's findings were corroborated by additional research conducted in Sweden and later in Germany. Hellgren released comprehensive statistics demonstrating the frequency of first-degree relatives having a 7.8% prevalence of psoriasis, compared to 1.97% in the general population and 3.14% in matched controls.[12] Genes that contribute to the etiology of psoriasis may be corrected or their effects lessened by the use of gene therapy. Genes such as interleukins, interferons, and JAK/STAT, for instance, that are implicated in inflammatory pathways may be suitable targets. Certain genes that are overexpressed in psoriatic lesions could have their expression decreased by using methods such as RNA interference (RNAi). Psoriasis has been linked to changes in DNA methylation patterns, which may be the target of a therapeutic intervention. One important epigenetic process that modifies gene expression without affecting DNA sequence is DNA methylation.[13] Utilizing a nonsynonymous nucleotide substitution in the IL23R gene,[14,15] certain IL23R gene haplotypes are linked to the sickness[14,15], along with additional autoimmune conditions. New psoriasis susceptibility loci in the chromosomes 1q21, 3q21, 4q32–35, 16q12, and 17q25 have been identified by genomic scanning. Recent mapping of two areas on chromosome 17q revealed a 6-mega-base pair split, suggesting separate linkage factors. The genes NAT9 and SLC9A3R1 are found in the first region, while RAPTOR is found in the second.[16] T cell proliferation, immunological synapse formation, and signal transduction are all facilitated by SLC9A3R1 and NAT9. T cell development and function are regulated by RAPTOR. By using these genes as an example, we can forecast that changes to regulatory genes—even ones that are not yet known—may promote T cell proliferation and the manifestation of inflammation.

Etiology

Extrinsic and intrinsic variables both play important roles in the complex disease known as psoriasis. It is thought that genetic susceptibility has a significant role, particularly in those whose disease manifests early (under 40 years).[17,18] Environmental and behavioral factors may also be present. Small-scale, localized trauma [19], stress [20], drugs [21], infections [22], alcohol consumption and smoking[23], and weight gain[24] are all known to either aggravate or cause psoriasis. The effects of climate change, especially exposure to sunlight, may exacerbate or even cause psoriasis.[25] Genetics is one of the most important aspects. Hereditary factors are significant because over 40% of individuals with psoriasis or psoriatic arthritis have a family history of the condition.[26] The common IL-12 and IL-23 receptor components are encoded at loci associated with psoriasis risk.[27,28] It appears that several receptor polymorphisms either protect against or predispose to psoriasis.[28,29] Psoriasis and the IL12B gene, which codes for the p40 component of IL-12 and IL-23, are closely related [30], as is the p19 component of IL-23 and IL-39, which is encoded by the IL23A gene.[27] Pustular psoriasis seems to be genetically distinct, involving many susceptibility genes, despite a paucity of data. Pustular psoriasis appears to be genetically unique, involving many susceptibility genes, despite the paucity of available data

(IL36RN, AP1S3 in Europeans, and CARD14 in other ethnicities).[17,31]

Pathogenesis

Up until the early 1980s, psoriasis was thought to be a condition of epidermal keratinocyte proliferation, with cutaneous inflammatory infiltration coming later. Nonetheless, an abundance of evidence indicates that the cell-mediated adaptive immune response plays a major role in psoriasis treatment.[32] It is now believed that psoriasis is not a T-helper (Th-1-mediated) issue but rather an inflammatory disease mediated by Th-1 and Th-17.[33,34] Reduced regulatory T cell (Treg) inhibitory function in psoriatic lesions may cause other effector cells to behave uncontrollably.[34] Hence, rather than being the result of a single group of T cells, it is more accurate to consider psoriasis as an effect of complex interactions between multiple T cell subsets. These are the primary immunological reactions that are thought to occur in psoriasis.[35] In the skin, antigenic cues activate innate immune cells such as plasmacytoid dendritic cells (pDCs) and others. Innate immunity cells produce proinflammatory cytokines, such as IFN-α, which stimulate and move myeloid dendritic cells (mDCs) in the skin. mDCs release cytokines such as IL-23 that induce, attract, and differentiate T cells. Recruited T cells release cytokines, including IL-17A, which stimulates the development of keratinocytes and the generation of proinflammatory cytokines. Cytokines, which are produced by immune cells and keratinocytes, take part in positive feedback loops that sustain the inflammatory process.[33] IFN-α is crucial to the first stages of the pathogenesis of psoriasis because it activates dendritic cells (DCs), which in turn generate a significant amount of IFN-α and stimulate the maturation and activation of mDCs.[36,37] Activated mDCs, macrophages, and keratinocytes will also generate TNF. Such TNF overexpression will cause the inflammation to become chronic [38] and induce the production of IL-12, IL-23, and other inflammatory cytokines by the immune cells. Additionally, it will promote the skin's invasion of neutrophils and monocytes from peripheral blood, with DCs and KCs further activating them.[39] Figure 1 shows the activation of mDC by INF-γ and TNF-a.

Because it sustains the chronic inflammatory state associated with psoriasis, interleukin-23 (IL-23) is a master cytokine regulator. It triggers the production of the cytokine IL-17A as well as other interleukins, including IL-12, IL-6, and IL-20, that maintain and intensify the activation of KCs by a number of innate and adaptive immune cells.[40,41] Conversely, IL-17-A is the primary cytokine involved in drawing neutrophils to the infection site; neutrophils are thought to be the source of AMPs (IL-37), IL-17A, and many reactive oxygen species that aid in the further activation of pDCs.[42] An autoantigen on the skin is captured by some activated mDCs, which then go to a local lymph node where they are recognized by T0 cells that are naive to the immune response.[34,43] The native T0 cell will develop into several Th cell subtypes upon identification. TNF, IL-12, IL-23, and IL-6 overexpression causes the naive T0 cell to mostly develop into Th-17, Th-22, and Th-1 cells.[34,44] Such activated T-lymphocytes will leave the lymph node to the skin; cytotoxic T cells (Tc) will be concentrated in the epidermis, while T helper (Th) in the dermis.[36,45] Where the lymphocytes identify the antigen once more, and Th-17 will multiply and cause inflammation when IL-23 and IL-12 are present. Where the lymphocytes identify the antigen once more, and Th-17 will multiply and cause inflammation when IL-23 and IL-12 are present. The pathophysiology that leads to the establishment of psoriasis plaque is summarized in Figure 2.

Figure 3 illustrates six prevalent clinical forms of psoriasis, as follows:

Plaque psoriasis

Affected by around 80% of psoriasis patients, this is the most common kind. It manifests as elevated, red areas coated in a layer of scale or dead skin cells, that is, silvery white in color. Common locations for these patches include the lower back, knees, elbows, and scalp.

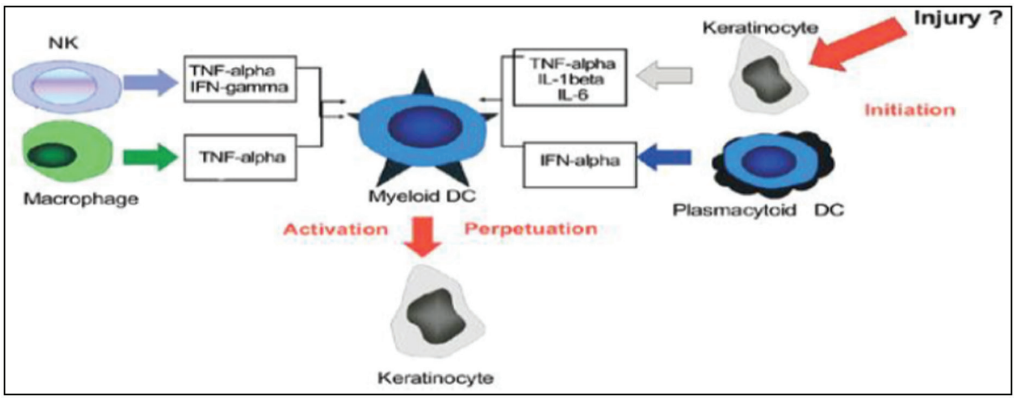

Figure 1: The activation of mDC by INF-γ and TNF-a[40]

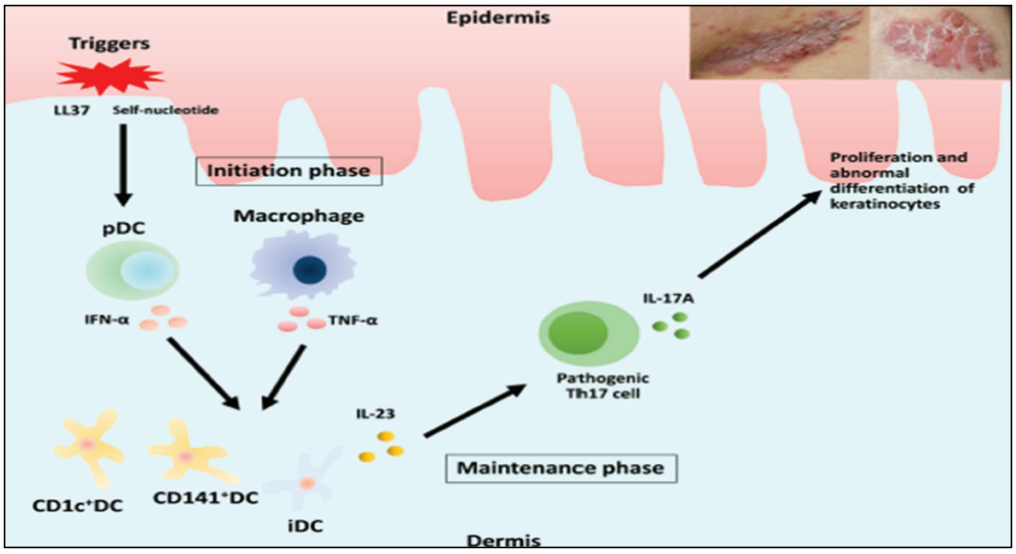

Figure 2: Summary of the major steps of pathogenesis giving rise to psoriasis plaque[36]

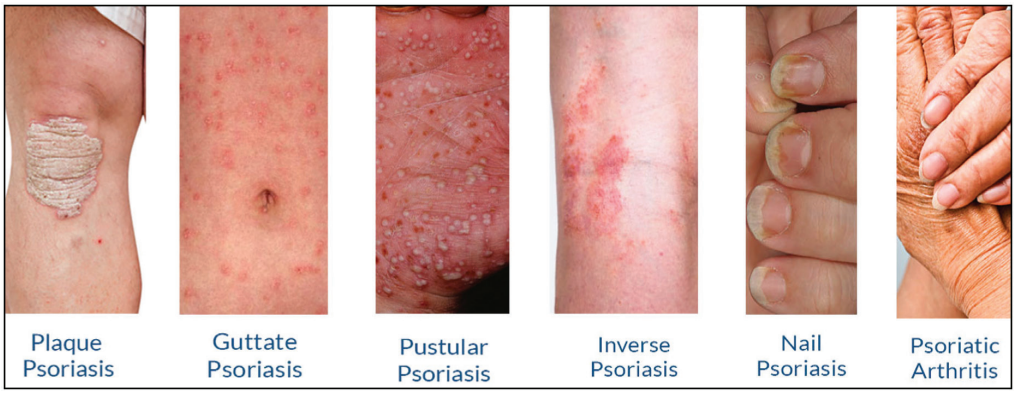

Figure 3: The image depicts six types of psoriasis: Plaque, guttate, pustular, inverse, nail, and arthritis[45]

Guttate psoriasis

This kind, which makes up 8% of cases of psoriasis, is distinguished by tiny, dot-shaped lesions. A strep infection can cause guttate psoriasis, which typically begins in infancy or early adulthood.

Pustular psoriasis

Usually affecting adults, this condition is typified by red skin encircled by white blisters of noninfectious pus. It might affect the majority of the body or just specific parts of it, such as the hands and feet.

Inverse psoriasis

This type manifests as extremely red lesions in areas where the body folds, including the crotch, under the arm, or behind the knee. It could seem glossy and silky.

Nail psoriasis

Psoriasis can cause pitting, abnormal development of the nails, and discoloration of the fingernails and toenails. Nails with psoriasis may become loose and come away from the nail bed.

Psoriatic arthritis

Psoriasis is not just a skin ailment; it can also lead to joint inflammation, which in turn causes psoriatic arthritis. This kind affects the lower back and the joints at the tips of the fingers and toes.

Risk factors

About 6.7 million people in the US suffer from psoriasis, a persistent inflammatory skin disease. While the exact cause of psoriasis remains unknown, several risk factors and triggers may provide insight into possible pathways leading to the condition. Risk factors and psoriasis triggers overlap significantly; in fact, in predisposed individuals, perceived risk factors may act as triggers for the illness to develop. We have outlined the main elements that influence the development and course of psoriasis in this review. It may be helpful to inform patients about these factors and how they might impact the course of their psoriasis while learning how to treat this chronic condition.[16]

The pathophysiology of psoriasis is strongly impacted by heredity. One important susceptibility locus for psoriasis

is psoriasis susceptibility 1 (PSORS1), which is located on chromosome 6p21 within a roughly 220 kb area of the major histocompatibility complex.[46] The susceptibility allele in PSORS1 is HLA-Cw6.[47] It is linked to severe, unstable, and early onset diseases.[47,48] Being overweight is a long-term, low-grade inflammatory condition that can aggravate or cause psoriasis.[49,50] It is also believed that smoking raises the chance of developing psoriasis, probably via the same mechanism.[51] The frequency of psoriasis was positively correlated with the quantity or duration of smoking. The correlation between alcohol use and psoriasis is not as well established as the correlation between psoriasis and smoking or obesity. Psoriasis patients who are overweight or obese may benefit from weight loss using a low-energy diet. Psoriasis symptoms have been found to lessen in intensity in correlation with a drop in body weight. Because of their potential to lower inflammation and aid in the treatment of psoriasis, a variety of nutrients, including omega-3 fatty acids, antioxidants (such as vitamins A, C, and E, carotenoids, flavonoids, and selenium), and vitamin D supplements, are advised. Elevated leptin levels, often associated with obesity, have been connected to inflammation and the severity of psoriasis. The inflammatory properties of leptin may exacerbate psoriatic lesions. However, ghrelin has anti-inflammatory properties and may delay the onset of psoriasis. The precise processes and interactions between these hormones and psoriasis are still being investigated.[51,52] Although psoriasis patients tend to consume more alcohol, there is not enough evidence to classify alcohol as a risk factor.[52] Human skin has been greatly damaged by rising air pollution levels throughout time. A variety of air contaminants, including UV radiation, polycyclic aromatic hydrocarbons, volatile organic compounds, oxides, particulate matter, ozone, and heavy metals, can produce oxidative stress in the skin.[53] One of the atmospheric contaminants is cadmium, which contributes to the causes of psoriasis. Individuals with severe psoriasis exhibited higher blood cadmium levels than the general population.[54] Apart from its typical skin lesions, psoriasis is a persistent systemic inflammatory condition that is, linked to other comorbidities. The most common comorbidity with psoriasis is metabolic syndrome (MetS), which also increases the risk of cardiovascular disease, which is the leading cause of mortality for psoriasis patients. While the precise etiology of these two conditions remains unclear, the underlying pathophysiology of MetS and psoriasis appears to entail shared inflammatory pathways and genetic predispositions. In both conditions, dysregulation of the IL-23/Th-17 immunological signaling system plays a pivotal role in increasing susceptibility to metabolic and cardiovascular disorders in both psoriasis-affected and non-psoriasis-affected individuals. Therefore, biological psoriasis treatments that block these signals may be able to lower the risk of atherosclerosis and other cardiometabolic disorders as well as the inflammatory load associated with psoriasis. According to recent imaging studies, improving skin lesions is linked to improved vascular inflammation, supporting the theory that biological agents have therapeutic effects beyond the skin and may help prevent cardiovascular disease. "Drug-related psoriasis" refers to psoriasis that develops and gets worse after using specific medications. It is often challenging to determine the underlying drug-induced causes of psoriasis in clinical settings. This is because different medications have different half-lives between when they should be given and the onset of psoriatic skin lesions.[55] Plaque, palmoplantar, nail, scalp, pustular, and erythrodermic psoriasis are the various manifestations of drug-induced psoriasis.[56]

Infection

There is ample evidence linking streptococcal infection to psoriasis.[57] Psoriasis, of which guttate psoriasis is the most prevalent variety, originates from an infection with streptococci. These are self-limiting symptoms, but if the streptococcal infection recurs, they might not go away. Therefore, tonsillectomy might be a good course of action for those with resistant psoriasis who also have tonsillitis episodes.[58] While guttate psoriasis is linked to a prior Streptococcus pyogenes infection, guttate psoriasis cannot be caused by a specific serotype. According to research, a COVID-19 infection may possibly result in or restart psoriasis. The virus's capacity to inflame the body may have an adverse effect on psoriasis that was previously under control or may set off a hereditary predisposition to the illness. Individuals with psoriasis may be somewhat more likely to get COVID-19. This may be because immunosuppressive treatments for psoriasis frequently make patients more prone to infections. Indeed, the probability of contracting COVID-19 was higher in individuals with psoriasis than in the general population. This increased risk was determined to be 33%, depending on the number of individuals with psoriasis who also got the virus.[57]

Psychogenic Stress

One well-known systemic psoriasis-triggering mechanism is psychogenic stress. It has been linked to both the disease's early onset and exacerbations of preexisting psoriasis.[59]

Human immunodeficiency virus (HIV)

There is evidence that HIV infection exacerbates psoriasis.[60] Immunosuppression can result from atopic diathesis. Furthermore, a considerable number of patients with psoriatic arthritis also have HIV infection while receiving therapy for refractory cutaneous conditions.[61]

Severity index

The reason behind the creation of the Psoriasis Area and Severity Index (PASI) was that the proportion of body

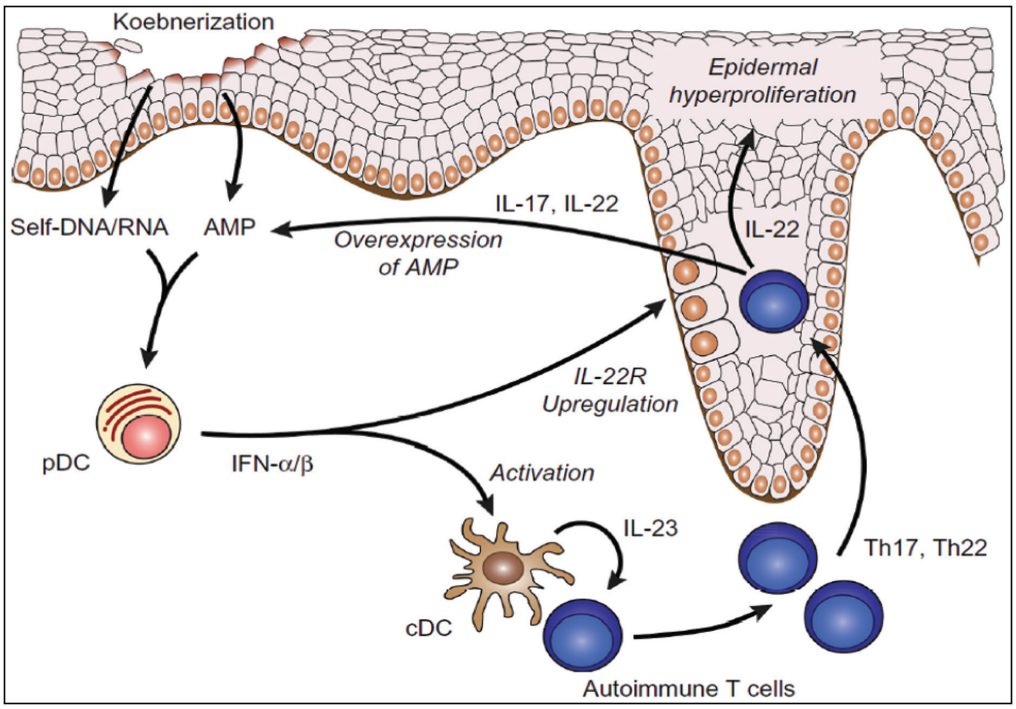

Figure 4: Psoriasis pathogenesis: Following skin injury, known as koebnerization, antimicrobial peptides (AMPs) such as LL-37 generated by keratinocytes or deposited by infiltrating neutrophils form complexes with self-nucleic acids (DNA and RNA) released by dying cells[67]

surface area affected did not precisely represent the degree of erythema, induration, and scaling associated with individual lesions.[62] The psoriasis area severity index (PASI), which considers other characteristics associated with the condition, is a more widely used tool for assessment since it provides a solid indication of the disease's severity based on the afflicted body area. The PASI scores are divided into three primary groups for classification scenarios.

A. Mild

Not only did it have negligible or no effect on quality, but 65% of all psoriatic patients had an afflicted body surface area.

B. Moderate

A substantial reduction in body surface area and up to 25% of all psoriatic patients had a significant influence on their quality of life.

C. Sever

Significantly impairing quality of life in addition to affecting more than 10% of all psoriatic patients with referred body surface area affected. In general, a PASI score of more than twenty percent is considered severe, a score of ten to twenty percent is considered moderate, and a score of less than ten percent is considered mild [63], as a moderate type of psoriasis.[63]

Pathophysiology

The distinctive scale or flakes of the lesions are caused by the hyperproliferation and aberrant differentiation of epidermal keratinocytes with a weakly adherent stratum corneum in psoriasis.[64]

T-lymphocytes made up the majority of the infiltrating lymphocytes, and the dermal layer's endothelial vascular alterations included angiogenesis, dilatation, and the development of high endothelial venules (HEVs).[65] In addition, there are more leukocytes infiltrating the cell (inflammation).[66] According to our present understanding of the molecular etiology of psoriasis, the interplay between innate and acquired immunity is crucial. Certain DCs in the dermis and epidermis get activated when the disease initially manifests. Among their numerous functions, these cells produce the messenger molecules IL-23 and tumor necrosis factor (TNF-α), which stimulate the development of Th1 and Th17 cells. These T cells release mediators involved in the vascular and epidermal changes linked to psoriasis.[64] T-lesion formation and persistence appear to be primarily driven by lymphocytes and the cytokines and chemokines they produce; however, neutrophils, natural killer cells, and endothelial cells might also be crucial. Intercellular adhesion molecules and other selections are cooperated with by this cytokine (ICAM), as shown in Figure 4.[67]

Autoimmunity

There are clear autoimmune-related pathway mechanisms for psoriasis. A deeper comprehension of the function autoantigen-specific T cells play in the onset, carbonification, and overall progression of the illness will be possible thanks to this significant field of study. One of the two T-cell

autoantigens in psoriasis that has received much research is IL-37. Based on a study of individuals with moderate to severe plaque psoriasis, two-thirds of the patients had CD4+ and CD8+ T-lymphocytes specific to IL-37. IFN-α is produced by T cells that are specific to IL-37, and IL-17, IL-21, and IL-22 are also produced by CD4+ T cells. Lesional skin or blood contains T-lymphocytes that are specific to IL-37 and are associated with disease activity.[68] IL-37-activated CD8+ T cells participate in autoantigen detection, epiderma tropism, and further Th17 cytokine release. It was found that an autoreactive CD8+ T cell TCR recognizes the melanocytic protein ADAMTSL5, which is an autoantigen restricted to HLA-C*06:02. This discovery confirms that target cells for the immune system are melanocytes., However, it does not rule out other biological targets.[69] Two other potential autoantigens are lipid antigens generated by phospholipase A2 (PLA2) group IVD (PLA2G4D) and keratin 17 obtained from hair follicles.[70,71] It is interesting to note that CD8+ T cell proliferation is limited to those with the HLA-Cw*0602 genotype when exposed to keratin 17.[72]

Diagnosis and method of evaluation

Evaluation of the distribution of psoriasis lesions and clinical indicators is necessary for the diagnosis of psoriasis vulgaris since these symptoms are quite evident and therefore relatively simple to evaluate.[73] A key tool in the diagnosis and treatment of psoriasis is dermoscopy. It is a noninvasive diagnostic instrument that makes it possible to examine skin lesions more thoroughly than it is possible to see with the unaided eye. This is the way that dermoscopy is applied to psoriasis. In particular, HFUS is useful for assessing subclinical characteristics and tracking therapy responses when it comes to assessing and managing psoriasis. If the presentation is abnormal, a skin sample may need to be examined histologically.[74] Additionally, plausible biomarkers that are suitably sensitive and specific can be found by utilizing flow cytometry, proteomics, and molecular signaling approaches.[75] Serum C-reactive protein (CRP), fibrinogen, and ESR are examples of inflammatory indicators that can be used to measure how inflammatory psoriasis is.[76] A variety of tools are available for the clinical evaluation of psoriasis. Certain assessment instruments can be used to determine the severity of a skin illness. The PGA (Psoriasis Global Assessment) is a rating system that ranges from 0 to 5 for scales, induration (thickening or elevation of the skin), and erythema (redness of the skin).[77] Body Surface Area, or BSA, calculates the percentage of the body covered in psoriasis (one palm is estimated to cover 1% of the body). The BSA serves as the multiplier for the PASI, a rating system that ranges from 0 to 72 for scaling, erythema, and induration. Severe psoriasis appears to be best assessed using the PGA and PASI scores; higher scores are indicative of more severe illnesses.[78] The absolute PASI score is often used to measure severity, but the percentage response rate is used to define the response to psoriasis treatment.[79] Thus far, in psoriasis studies, the most commonly used method for evaluating the quality of life of patients with skin conditions is the DLQI. There are ten questions in this assessment, encompassing six domains (feelings and symptoms, leisure, daily activities, school and work, personal relationships, and bother with psoriasis treatment). The choices that would be made in order to respond to the questions range from 0 (not at all influenced) to 3 (very much affected). This adds up to a total range of 0–30, where lower values indicate a higher quality of life.[80] The Salford Psoriasis Index, or SPI, is a tool used to evaluate the psychosocial effects and severity of skin lesions. It assesses the severity of the psoriasis, the psychological effects, and the length of previous psoriasis therapies.[81] Further study of the underlying pathophysiology of psoriasis may provide additional targets for therapy.[81,82]

Conclusion

Psoriasis is a complex disease characterized by heightened innate immune responses in patients with a genetic predisposition. This is due to a network of cells producing cytokines that cause chronic inflammation. Immunology, molecular biology, and genetics advancements have led to targeted immune therapy, with further research potentially providing additional therapeutic objectives.[83,84]

Ethical Approval

Not Applicable

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Conflicts of interest

The authors declare no conflict of interest.

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