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Review

Comprehensive Review of Psoriasis: Pathogenesis, Treatment, and Future Directions

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Abstract

Inflammation of the skin, often known as psoriasis, is a chronic skin condition that is brought on by the immune system. There are various factors that can contribute to its development, including genetics, the immune system, and the environment. Immunological dysregulation is primarily caused by the Th17 axis; however, genetics and the environment can also play a role in the development of this condition. Because the disease manifests itself in a variety of ways in various persons, it is essential to make use of standardised tools such as the PASI and the DLQI to arrive at an accurate diagnosis and determine the severity of the condition. There are a number of different methods that can be utilised to treat the illness. Some of these methods include topical therapies, phototherapy, systemic drugs, and biologics that target particular immune pathways. There are a number of severe health concerns that are associated with psoriasis, such as psoriatic arthritis and heart disease, both of which make going through life less enjoyable. As biomarkers and personalised medicine continue to advance, it is possible that treatments will become more therapeutically effective and less risky for each individual patient. It is vital to continue searching for novel drugs and molecular processes to improve the results for patients and to satisfy therapeutic needs that have not yet been satisfied.

Keywords: Psoriasis; Th17; PASI; DLQI; Quality of life

INTRODUCTION

About 2–3% of people in the globe have psoriasis, a long-lasting skin illness that is caused by the immune system. It has crimson plaques with silver-white scales on them (Parisi et al., 2013). People used to think that psoriasis was just a skin illness, but today we know that it is a systemic disease that can cause major problems

like psoriatic arthritis, heart disease, and metabolic syndrome (Greb et al., 2016). Genetic predisposition, environmental triggers, and immunological responses that aren't operating well, especially the IL-23/Th17 axis, all play a role in the pathogenesis (Lowes et al., 2014). Molecular biology has made targeted biologic medicines possible, which have changed the way diseases are treated. But there are still problems with how long these therapies last, how easy they are to get, and how safe they are in the long term. This overview talks about what we know so far about how psoriasis starts, the therapies that are available now, and innovative ways that focus on personalized therapy and transform the illness.

PATHOGENESIS OF PSORIASIS

Immune Dysregulation

Dendritic cells, T cells, and cytokines including TNF-α, IL-17, and IL-23 are important in the development of psoriasis because they make the innate and adaptive immune systems act in ways that are not normal. Things in the environment, such trauma or illness, can turn on plasmacytoid dendritic cells. This kind of stimulation makes them manufacture more type I interferon, which then gets myeloid dendritic cells going. After that, these cells let IL-12 and IL-23 out, which assist Th1 and Th17 cells proliferate and mature (Nestle et al., 2009). Th17 cells let out IL-17A, IL-17F, and IL-22. These substances make keratinocytes grow too quickly and keep inflammation going, which makes an inflammatory loop that keeps growing worse (Baliwag et al., 2015; Hawkes et al., 2017). IL-17 and IL-8 bring neutrophils to the site of infection, which helps to make Munro's microabscesses, a histological marker of the disease.

Genetic variables like HLA-Cw6 and changes in IL23R and TNFAIP3 also play a role in this immunological imbalance by changing how the body shows antigens and how cytokines talk to each other. Biologic medicines that are targeted are needed since psoriasis is a long-term condition that shows that the body's regulatory systems can't fix this pro-inflammatory environment.

Genetic Factors

Genetics plays a key role in psoriasis. Twin studies reveal that up to 66% of patients with the condition have a hereditary relationship (Lindqvist et al., 1978). The most relevant genetic relationship is the HLA-C06:02 allele in the PSORS1 locus on chromosome 6p21. It makes the disease start early and be very bad (Nair et al., 2006). Genome-wide association studies (GWAS) have found more than 60 places in the genome that make the disease more likely to happen. These include several genes that assist govern the immune system, such as IL12B, IL23R, TNFAIP3, TRAF3IP2, and TYK2. These genes are part of both the innate and adaptive immune systems (Tsoi et al., 2012). These genetic changes change how dendritic cells work, how Th17 cells develop, and how NF-κB signals, all of which make psoriatic lesions more inflamed.

Even though these results are interesting, the link between genetics and phenotype is not clear. This is because gene-environment interactions, epigenetic regulation, and non-coding RNAs all affect how diseases are and how effectively they react to treatment.

Environmental Triggers

People who are genetically prone to psoriasis can get it and make it worse because of environmental causes. Some things that can cause guttate psoriasis are infections (especially streptococcal pharyngitis), certain medications (like β-blockers, lithium, and antimalarials), stress, smoking, drinking, and being overweight (Griffiths & Barker, 2007; Takeshita et al., 2017). Antigens from streptococci may make T cells react with each other, which can make the skin swell up. Obesity and other metabolic problems can make systemic inflammation and disease worse by producing cytokines and adipokines (Setty et al., 2007). Also, ultraviolet light and the weather can influence how diseases work, and being in the sun often makes symptoms better.

These triggers probably work with immunological dysregulation and epigenetic pathways to affect when and how diseases start and get worse.

DIAGNOSIS AND CLINICAL FEATURES

Psoriasis makes red areas with silver scales on top of them that are quite clear. The scalp, elbows, knees, and lower back are the most common places it happens. If you have skin lesions, you can also have itching, changes to your nails (such pitting or onycholysis), and joint discomfort. PASI (Psoriasis Area and Severity Index), BSA (Body Surface Area), and DLQI (Dermatology Life Quality Index) are some of the methods that can be used to quantify how bad the condition is (Langley et al., 2005). Most of the time, a diagnosis is based on clinical indicators. However, in rare situations, histology can confirm it by exhibiting hyperkeratosis, parakeratosis, acanthosis, and Munro's microabscesses.

Different Kinds of Psoriasis

***Plaque Psoriasis (Psoriasis vulgaris)*:** This is the most prevalent kind, and it accounts for more than 80% of cases. It is characterized by stable, elevated plaques with silver scales.

***Guttate psoriasis*:** This is when small, drop-shaped lesions suddenly appear on the skin, generally following a streptococcal infection. It happens more often in kids and young adults.
Inverse psoriasis affects the areas between the fingers and toes and generates smooth, red spots that don't have any scaling.
***Pustular psoriasis*:** There are sterile pustules that can be localized (like on the palms and soles) or generalized and life-threatening (like the Von Zumbusch kind).
Erythrodermic psoriasis: It is an uncommon and severe type of the illness that causes the skin to turn red and peel off across a large region, affecting more than 90% of the body's surface area. It frequently means going to the hospital.
***Psoriatic arthritis*:** It is a kind of arthritis that causes inflammation. It can occur in up to 30% of patients with psoriasis. It makes the joints hurt, stiff, and swollen (Mease, 2015).

Criteria for diagnosis

People commonly find out they have psoriasis by looking at their skin, which has clear, red patches with silver scales. It often comes back over time and affects the elbows, scalp, and lower back (Griffiths & Barker, 2007). In certain rare cases, dermoscopy (which displays red spots and white scales) and a skin biopsy may aid confirm a diagnosis by exhibiting acanthosis, parakeratosis, neutrophilic microabscesses (Munro's abscesses), and elongation of rete ridges (Weedon, 2010). When there are difficulties with the joints, the CASPAR (Classification Criteria for Psoriatic Arthritis) criteria are applied. In rare situations, a skin biopsy and dermoscopy (which reveals red spots and white scales) can help confirm a diagnosis by showing thicker skin, skin cells that aren't normal, small groups of immune cells, and alterations in the structure of the skin.

When there are difficulties with the joints, the CASPAR (Classification Criteria for Psoriatic Arthritis) criteria are applied. These criteria need to show symptoms of joint inflammation and at least three of the following: present psoriasis, nail problems, swelling of fingers or toes, a negative rheumatoid factor, or new bone development near the joint observed on an X-ray.

Assessment of Severity

To find out how terrible psoriasis is, doctors generally use a combination of objective and patient-reported techniques. The Psoriasis Area and Severity Index (PASI) is the best test utilized in clinical studies. It gives a score of 0 to 72 for redness, thickness, and scaling in different parts of the body. The Body Surface Area (BSA) tells you how much of the skin is damaged as a percentage. More than 10% is bad. The Dermatology Life Quality Index (DLQI) looks at the effects on mental and social health. Scores above 10 show a big drop in quality of life (Mrowietz et al., 2011; Finlay & Khan, 1994).
In general, severity categories are characterized as:
Mild: PASI is 7 or less, BSA is 3% or less, and DLQI is 5 or less.
Moderate: PASI 7–12, BSA 3–10%, and DLQI 5–10
Severe: PASI is more than 12, BSA is more than 10%, or DLQI is more than 10, no matter what the PASI or BSA values are (European consensus criteria).

TREATMENT MODALITIES

Topical Therapies

Corticosteroids, vitamin D analogues, calcineurin inhibitors, coal tar, and keratolytics are some of the topical medicines that can help with mild to moderate psoriasis. People usually use topical corticosteroids because they halt cells from developing and lessen inflammation. Depending on where the lesion is and how bad it is, the medication's strength is changed (Menter et al., 2009). Calcipotriol and other vitamin D analogs affect how keratinocytes develop and change. When you combine corticosteroids with them, they frequently function better and have fewer negative effects (van de Kerkhof, 2001). Calcineurin inhibitors like tacrolimus and pimecrolimus are good for intertriginous and facial areas because they don't make steroids work (Rendon & Schäkel, 2019).

Other things, including salicylic acid and coal tar, aid by decreasing scaling and making it easier for active medications to enter into the skin. The location and severity of the lesion, as well as the patient's preferences and the risk of side effects such as skin thinning and irritation, all play a role in determining the best course of therapy.

Phototherapy

Phototherapy is a good second-line treatment for moderate to severe psoriasis, especially when topical treatments don't work. The most popular treatment is narrowband UVB (NB-UVB), which destroys T cells and reduces the levels of pro-inflammatory cytokines including IL-17 and IL-23 (Kollias & Baqer, 2005). It works well for treating plaque, guttate, and palmoplantar psoriasis when given 2 to 3 times a week.

Psoralen with UVA (PUVA) is a mix of oral or topical psoralen and UVA exposure that gets into DNA and induces cell death. But it has more long-term hazards, like skin cancer and photoaging (Parrish et al., 1974). Excimer laser (308 nm UVB) is a targeted therapy for localized lesions that works when other therapies don't.

Systemic Therapies

People with moderate to severe psoriasis that doesn't go better with topical treatments or phototherapy need systemic therapies. Methotrexate, which blocks folate, is still an important medicine since it inhibits cells from developing and weakens the immune system. It works well for psoriatic arthritis and plaque psoriasis (Flytström et al., 2008). Cyclosporine is a calcineurin inhibitor that swiftly prevents T-cell activation. However, using it for a long time can cause nephrotoxicity and elevated blood pressure (Franchi et al., 2001). Acitretin is a systemic retinoid that helps keratinocytes differentiate normally, although it might induce birth abnormalities. People typically combine it alongside phototherapy to make it function better (Ortonne et al., 2006).

It's important to pick the right medications for each patient and keep an eye on how hazardous they are to organs. They are also typically employed as a bridge between other treatments and biologics.

Biologic Therapies

Biologics function by blocking certain immune pathways that are critical for the growth of psoriasis. They work quite well and are safer than other options. TNF-α inhibitors, including etanercept and infliximab, were the first biologics to get the green light. Blocking TNF-α signaling is how they function to decrease inflammation (Leonardi et al., 2003). Ustekinumab and secukinumab are IL-12/23 inhibitors, and ixekizumab and secukinumab are IL-17 inhibitors. They block crucial proteins that are part of the Th17 pathway. This kind of treatment makes clinical responses better and remissions continue longer (Greb et al., 2016; Langley et al., 2014). Guselkumab and tildrakizumab are two new medications that specifically target IL-23. They look promising in terms of safety and effectiveness, which could change how we treat these illnesses.

Biologics have changed the way moderate to severe psoriasis is treated, but they need to be checked for infections and monitored all the time.

COMORBIDITIES AND QUALITY OF LIFE

Comorbid Conditions

There are a lot of health concerns that impact the whole body and are associated to psoriasis. These disorders have a huge effect on death and illness. There are a lot more cases of cardiovascular disorders, like hypertension, atherosclerosis, and myocardial infarction. This rise is likely due to chronic systemic inflammation and risk factors that are shared, such as metabolic syndrome and obesity (Gisondi et al., 2018). Up to 30% of people with psoriatic arthritis have joint damage that makes it hard for them to work (Gladman et al., 2005). Diabetes mellitus, inflammatory bowel disease, depression, and non-alcoholic fatty liver disease (NAFLD) are some more prevalent comorbidities. These signs and symptoms suggest that psoriasis is an inflammatory disease that affects more than simply the skin (Dowlatshahi et al., 2014).

Recognizing and treating comorbidities is critical for providing complete treatment to patients and getting better long-term results.

Effect on Quality of Life

Psoriasis makes life significantly worse since it generates noticeable skin lesions, lasts a long time, itches, and has a psychosocial burden that includes stigma, anxiety, and sadness (Rapp et al., 1999). The Dermatology Life Quality Index (DLQI) always demonstrates that a lot of patients have QoL difficulties that range from mild to severe. These issues are typically related to how bad the illness is, but they can also be altered by how effectively the person deals with it and how much social support they receive (Finlay & Khan, 1994). When your quality of life goes down, it affects your work and daily life. This illustrates how vital it is to have a thorough treatment plan that deals with both your physical and mental health.

FUTURE DIRECTIONS IN PSORIASIS RESEARCH

Novel Therapies

New treatments try to make things work better and be safer by going after new molecular targets and immunological pathways that are higher up in the immune system. Deucravacitinib and other Janus kinase (JAK) inhibitors look promising because they can affect a number of cytokine signals that are involved in the development of psoriasis and are easy to take by mouth (Papp et al., 2021). Researchers are also looking into medicines that stop IL-36 receptor signaling in pustular psoriasis. The proof reveals that different varieties of the disease are getting different kinds of treatment (Bachelez et al., 2021). Researchers are also looking at modifying the microbiota and editing genes to deal with genetic predisposition and environmental variables. These could help you stay in remission for a long time.

Biomarkers

Researchers are looking into biomarkers to help with diagnosis, keep an eye on disease activity, predict how well treatments will work, and learn more about how diseases start. A lot of IL-17A, IL-22, and TNF-α in the blood means that the condition is worse and that Th17 cells are causing inflammation (Zaba et al., 2009). C-reactive protein (CRP) and vascular endothelial growth factor (VEGF) are other markers of systemic inflammation that are associated to other health issues (Helliwell et al., 2017). New research has shown that microRNAs like miR-203 and miR-146a might be useful non-invasive markers since they help skin cells proliferate and control the immune system.

Doctors could determine the appropriate treatment for each patient and start it early if they had reliable biomarkers.

Personalized Medicine

Personalized therapy for psoriasis employs genetic, immunological, and clinical data to tailor medicines to each person, making them work better and less likely to produce side effects. Pharmacogenomic markers like HLA-C06:02 can assist clinicians identify the optimum medication by showing how well a person will respond to biologics like ustekinumab (Hawkes et al., 2017). Molecular profiling and biomarker discoveries have come a long way, making it easier to categorize individuals by disease subtype and risk of comorbidity. The outcome backs up precision management methods (Nograles et al., 2010).

CONCLUSION

Psoriasis is a difficult disease that affects the immune system. There are numerous things that might cause it, such as genetics, immune system disorders, and elements in the environment. Researchers have learned more about how it operates, which has led to a wide range of treatments, from topical medicines to biologics, that have improved patient outcomes. But the necessity for thorough treatment is even more critical because of other health problems and how they affect quality of life. New biomarkers and personalized medicine procedures promise to make it easier to choose the right treatment and manage diseases. This is the start of a new era in precision dermatology.

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