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Psoriasis: A Dillema

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Abstract
Research on psoriasis pathogenesis has largely increased knowledge on skin biology in general. In the past 15 years, breakthroughs in the understanding of the pathogenesis of psoriasis have been translated into targeted and highly effective therapies providing fundamental insights into the pathogenesis of chronic inflammatory diseases with a dominant IL-23/Th17 axis. This review discusses the mechanisms involved in the initiation and development of the disease, as well as the therapeutic options that have arisen from the dissection of the inflammatory psoriatic pathways. Our discussion begins by addressing the inflammatory pathways and key cell types initiating and perpetuating psoriatic inflammation. Next, we describe the role of genetics, associated epigenetic mechanisms, and the interaction of the skin flora in the pathophysiology of psoriasis. Finally, we include a comprehensive review of well-established.

Keywords: psoriasis, inflammation, chronic skin disease

Introduction
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 [1]. It shows a lower prevalence in Asian and some African populations, and up to 11% in Caucasian and Scandinavian populations [2-5].
The dermatologic manifestations of psoriasis are varied; psoriasis vulgaris is also called plaque-type psoriasis, and is the most prevalent type. The terms psoriasis and psoriasis vulgaris are used interchangeably in the scientific literature; nonetheless, there are important distinctions among the different clinical subtypes (See Figure 1).

Psoriasis Vulgaris
About 90% of psoriasis cases correspond to chronic plaque-type psoriasis. The classical clinical manifestations are sharply demarcated, erythematous, pruritic plaques covered in silvery scales. The plaques can coalesce and cover large areas of skin. Common locations include the trunk, the extensor surfaces of the limbs, and the scalp [6, 7].

Inverse Psoriasis
Also called flexural psoriasis, inverse psoriasis affects intertriginous locations, and is characterized clinically by slightly erosive erythematous plaques and patches.

Guttate Psoriasis
Guttate psoriasis is a variant with an acute onset of small erythematous plaques. It usually affects children or adolescents, and is often triggered by group-A streptococcal infections of tonsils. About one-third of patients with guttate psoriasis will develop plaque psoriasis throughout their adult life [8, 9].

Pustular psoriasis
Pustular psoriasis is characterized by multiple, coalescing sterile pustules. Pustular psoriasis can be localized or generalized. Two distinct localized phenotypes have been described: psoriasis pustulosa palmoplantaris (PPP) and acrodermatitis continua of Hallopeau. Both of them affect the hands and feet; PPP is restricted to the palms and soles, and ACS is more distally located at the tips of fingers and toes, and affects the nail apparatus. Generalized pustular psoriasis presents with an acute and rapidly progressive course characterized by diffuse redness and subcorneal pustules, and is often accompanied by systemic symptoms [10].

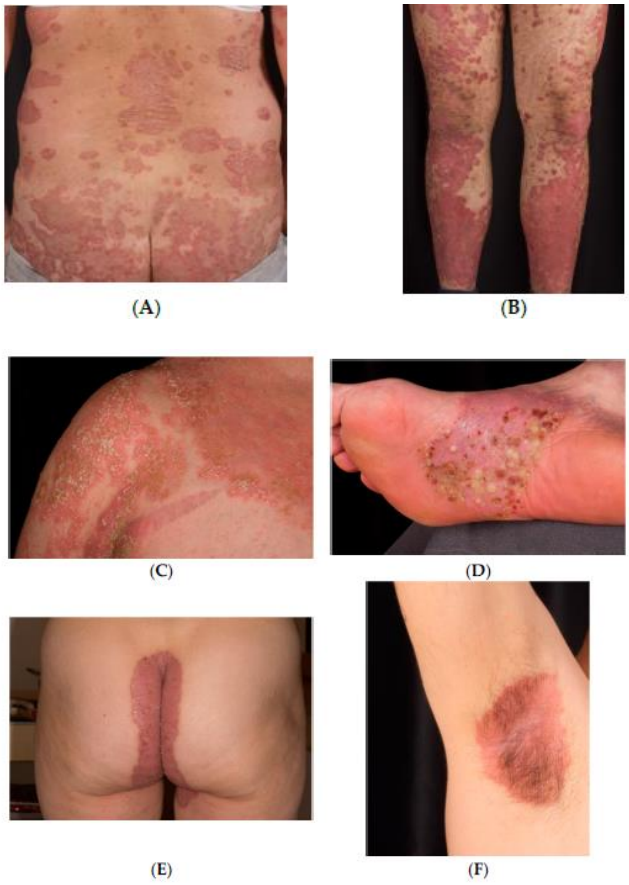

Fig 1: Clinical manifestations of psoriasis. (A, B) Psoriasis vulgaris presents with erythematous scaly plaques on the trunk and extensor surfaces of the limbs. (C) Generalized pustular psoriasis. (D) Pustular psoriasis localized to the soles of the feet. This variant typically affects the palms of the hands as well; hence, psoriasis pustulosa palmoplantaris. (E, F) Inverse psoriasis affects the folds of the skin (i.e., axillary, intergluteal, inframammary, and genital involvement).

Erythrodermic psoriasis

Acute condition in which over 90% of the total body surface is erythematous and inflamed. Erythroderma can develop on any kind of psoriasis type, and requires emergency treatment (Figure 2).

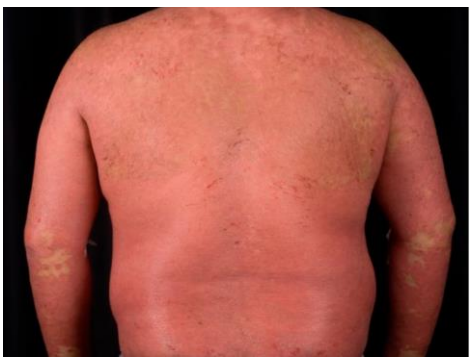

Fig 2: Requires emergency treatment

Comorbidities in Psoriasis

Psoriasis typically affects the skin, but may also affect the joints, and has been associated with a number of diseases. Inflammation is not limited to the psoriatic skin, and has been shown to affect different organ systems. Thus, it has been postulated that psoriasis is a systemic entity rather than a solely dermatological disease.

When compared to control subjects, psoriasis patients exhibit increased hyperlipidemia, hypertension, coronary artery disease, type 2 diabetes, and increased body mass index. The metabolic syndrome, which comprises the aforementioned conditions in a single patient, was two times more frequent in psoriasis patients [11, 12]. Coronary plaques are also twice as common in psoriasis patients when compared to control subjects [13]. Several large studies have shown a higher prevalence of diabetes and cardiovascular disease correlating with the severity of psoriasis [14–18].

There are divided opinions regarding the contribution of psoriasis as an independent cardiovascular risk factor [19, 20]; however, the collective evidence supports that psoriasis independently increases risk for myocardial infarction, stroke, and death due to cardiovascular disease (CVD) [21-28]. In addition, the risk was found to apply also to patients with mild psoriasis to a lower extent [21, 27]. Vascular inflammation assessed via 18F-fluorodeoxyglucose positron emission tomography computed tomography (18F-FDG PET/CT) found psoriasis duration to be a negative predicting factor.

It was suggested that the cumulative effects of low-grade chronic inflammation might accelerate vascular disease development [29]. In a study by Metha *et al*., systemic and vascular inflammation in six patients with moderate to severe psoriasis was quantified by FDG-PET/CT. Inflammation foci were registered as expected in the skin, joints, and tendons. In addition, FDG uptake in the liver and aorta revealed subclinical systemic inflammation [30]. Furthermore, standardized uptake values were reduced in the liver, spleen, and aorta following treatment with ustekinumab {Kim, 2018 #359}. A new biomarker to assess CVD risk in psoriasis patients was proposed by nuclear magnetic resonance spectroscopy [31].

The signal originating from glycan N-acetylglucosamine residues called Glyc A in psoriasis patients was associated with psoriasis severity and subclinical CVD, and was shown to be reduced in response to the effective treatment of psoriasis.

Psoriatic inflammation of the joints results in psoriatic arthritis (PsA). The skin manifestations generally precede PsA, which shares the inflammatory chronicity of psoriasis and requires systemic therapies due to a potential destructive progression. Psoriatic arthritis develops in up to 40% of psoriasis patients [32-38]; around 15% of psoriasis patients are thought to have undiagnosed 1475 PsA [39]. It presents clinically with dactylitis and enteritis in oligoarticular or polyarticular patterns.

The polyarticular variant is frequently associated with nail involvement [40]. Nails are specialized dermal appendages that can also be affected by psoriatic inflammation. Nail psoriasis is reported to affect more than half of psoriasis patients, and can present as the only psoriasis manifestation in 5–10% of patients [41]. The clinical presentation of nail psoriasis depends on the structure affected by the inflammatory process. Nail matrix involvement presents as pitting, leukonychia, and onychodystrophy, whereas inflammation of the nail bed presents as oil-drop discoloration, splinter hemorrhages, and onycholysis (Figure 3) [42]. Psoriatic nail involvement is associated with joint involvement, and up to 80% of patients with PsA have nail manifestations [43, 44].

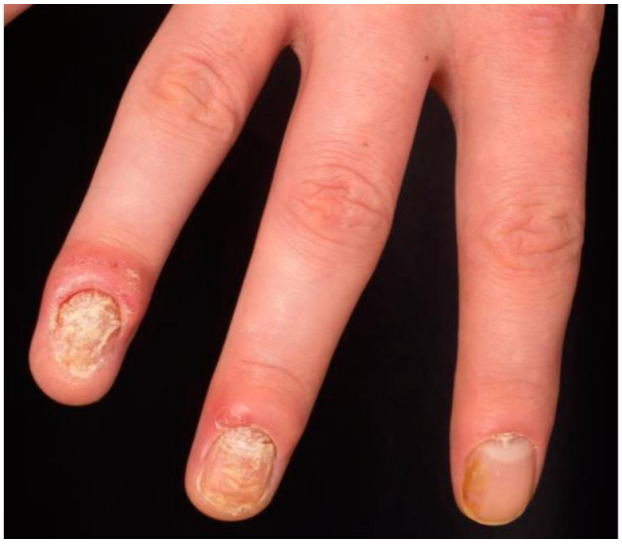

Fig 3: Psoriatic Nails

Pathogenesis

The hallmark of psoriasis is sustained inflammation that leads to uncontrolled keratinocyte proliferation and dysfunctional differentiation. The histology of the psoriatic plaque shows acanthosis (epidermal hyperplasia), which overlies inflammatory infiltrates composed of dermal dendritic cells, macrophages, T cells, and neutrophils in histological investigations.

Neovascularization is also a prominent feature. The inflammatory pathways active in plaque psoriasis and the rest of the clinical variants overlap, but also display discrete differences that account for the different phenotype and treatment outcomes.

Psoriasis homeopathic treatments

Some of the most commonly touted homeopathic treatments for psoriasis include those listed below. There's no scientific evidence that any of them are effective at treating psoriasis or its symptoms.

Sepia

Sepia is used by some people who practice homeopathy for widespread psoriasis and dry skin. However, there's no scientific evidence that it's an effective treatment.

Arsenicum album

Anecdotal evidence suggests that arsenicum benefits people with dry, scaly skin made worse by itching and better by applying heat. There's no scientific evidence that it helps with psoriasis.

It's also arsenic-based, so it can be dangerous if it contains more of the active ingredient than stated.

Graphites

Graphites are used in homeopathy for people with long-term skin disorders and leathery, cracked skin. There's only anecdotal evidence that it can help psoriasis symptoms.

Sulfur

There's anecdotal evidence that sulfur reduces skin lesions and itching. Although using sulfur alone as a homeopathic treatment is unproven, it may be mixed with proven psoriasis treatments, such as coal tar or salicylic acid.

Petroleum

Anecdotally, petroleum helps people whose physical problems are made worse by stress. Ingesting petroleum, even in small amounts, can be very dangerous. But petroleum jelly, such as Vaseline, can help seal moisture into your skin and reduce itching, flaking, and irritation.

Calcarea carbonica

Calcarea carbonica, which is made from shells, is used in homeopathy to treat many illnesses, particularly in people who are often cold and get tired easily.

Research shows that people with psoriasis have low levels of calcium in their blood, but there's only anecdotal evidence supporting the use of calcarea carbonica for this condition.

Staphysagria

An animal study has suggested that staphysagria may be anti-inflammatory, but there's only anecdotal evidence of it being effective for people with psoriasis. It's mostly used in homeopathy for scalp psoriasis.

Mercurius solubilis

Mercurius solubilis is a type of mercury, which is toxic to ingest or put on your skin. High exposures can even cause kidney failure, respiratory issues, and death. There's no scientific evidence that mercurius solubilis is a safe or effective treatment for psoriasis.

Rhus toxicodendron

Rhus toxicodendron is poison ivy. There's mixed evidence that it helps with arthritis and, therefore, psoriatic arthritis. However, there's only anecdotal evidence that it can help with other symptoms of psoriasis, under the theory "like cures like."

Mezereum

Mezereum is a flowering shrub used in homeopathy for thick, crusty plaques. It's poisonous to humans when ingested or put on the skin. There's no scientific evidence that mezereum is a safe or effective treatment for psoriasis.

Potential side effects and precautions

Research hasn't found much evidence for homeopathic medicine's effectiveness for any health condition. There's also not much research on the safety of homeopathy.

Neither the safety nor the effectiveness of homeopathic medicine is tested by the Food and Drug Administration (FDA).

Homeopathy comes with several risks. First, some products may be labeled with incorrect amounts of active ingredients. Higher amounts of the active ingredient can cause side effects, allergic reactions, or drug interactions. Some substances used in homeopathic medicines are toxic at any dose.

Never use homeopathic medicine in place of a medication that your doctor prescribes. Tell your doctor about any medications you take, including homeopathy, and talk to your doctor about any changes in your symptoms or overall health.

Conclusion & Summary

Psoriasis is a complex multifactorial disease for which various novel therapies have arisen in the past years. In spite

of the refinement of the targeted therapies, psoriasis remains a treatable but so far not curable disease. The targeted therapies show high clinical efficacy for the inhibition of IL-23 and IL-17. Some degree of a persistent antipsoriatic effect by these therapies could be demonstrated after drug discontinuation, and argue for disease modification concept. This important finding will be followed up in ongoing and future studies. However, in other cases, an initial clinical response is only short lived, requiring treatment with a different biologic. Clearly, more research is required to answer the question of why the drug survival of some biologics is limited. The therapeutic arsenal for psoriasis is likely to increase in the near future, with studies on orally applied new small molecules such as inhibitors targeting RORt. In spite of the safety and efficacy of targeted therapies, due to economic factors, dosage regimes, and adverse effect profiles, broader-acting drugs remain the mainstay of psoriasis systemic therapy in many clinical scenarios around the world. The role of genetics remains to be elucidated not only in the context of predisposition to disease, but also in the profiling of distinct psoriatic types based on cytokine signatures, and in identifying therapy response markers. Clearly, psoriasis is currently the best understood and the best treatable Th17-biased chronic inflammatory disease. After achieving excellent clinical responses for the majority of patients with available therapeutic approaches, the stratification of psoriasis patients to the optimal drug and ensuring the sustainability of our treatments are the major tasks to be resolved.

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