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Herbal Drugs in the Treatment of Psoriasis

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Abstract

Psoriasis is an inflammatory skin condition characterized by scaling with inflammation, resulting in regions of thick, red skin covered in silvery scales. It can be itchy or painful. Systemic treatment, topical therapy, and phototherapy are used to treat psoriasis, but they have negative and potentially fatal side effects. Patients with psoriasis are more likely to acquire other conditions such as psoriatic arthritis, anxiety and depression, cancer, metabolic syndrome, cardiovascular disease, and Crohn's disease. Psoriasis is estimated to be around 2-3% of the general population. In Europe, the prevalence ranges from 0.1% to 3.2%, depending on the country. In Asia, the prevalence is generally lower, with estimates ranging from 0.1% to 1.5%. The study aims to highlight these plants, herbal formulations, and associated therapies to develop safer and more effective treatments for psoriasis. This review summarizes the current knowledge on herbal products used topically for psoriasis treatment, including their mechanisms of action such as inhibition of keratinocyte hyperproliferation, immune-inflammatory reaction. It also discusses the penetration of herbal products through the psoriatic skin barrier, novel drug delivery systems, and possible adverse effects of herbal therapy. Here, we provide a comprehensive review of the background of EP, assess the available clinical data on the efficacy of targeted therapies, and aim to provide a foundation for clinical decision making for this rare form of psoriasis.

Keywords: Psoriasis, Pathogenesis, Herbal extract.

Introduction

Psoriasis is a chronic inflammatory cutaneous disease characterized by the formation of scaly, indurated, erythematous plaques. Psoriasis has three principal histologic features: epidermal hyperplasia; dilated, prominent blood vessels in the dermis; and an inflammatory infiltrate of leucocytes, predominantly into the dermis[1]. The pathogenesis of psoriasis is characterized by increased production of inflammatory cytokines that cause hyperkeratosis. Briefly, interleukin (IL)-23 and IL-12 are produced by myeloid dendritic cells, and these cytokines activate naïve T cells to differentiate into Th1, Th17, and Th22 cells, which then produce cytokines responsible for the development of psoriatic plaques such as IL-17, IL-22, tumor necrosis

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factor-a, and interferon-g. Cutaneous lesions most commonly develop on the elbows, knees, scalp, umbilicus, and lumbar regions, and are typically characterized by erythematous plaques covered with silvery-white scales, termed chronic plaque psoriasis[2]. Less frequently, psoriasis can occur on € the nails (23–27%), face (49%), palms and soles (12–16%), or intertriginous regions (21–30%), and management of psoriasis in these areas can be challenging [(5)].

Epidemiology

psoriasis is estimated to be around 2-3% of the general population. In Europe, the prevalence ranges from 0.1% to 3.2%, depending on the country. In Asia, the prevalence is generally lower, with estimates ranging from 0.1% to 1.5%. Psoriasis can occur at any age, but it is most commonly diagnosed in individuals between the ages of 15 and 35[3].

Types of psoriasis

T here are different types of psoriasis and it's possible to have more than one type:

1.Chronic Plaque Psoriasis

Among different types of psoriasis, the most common one is plaque psoriasis or psoriasis vulgaris. Almost 85 percent of people with psoriasis have plaque psoriasis which is characterized by thick red patches of skin, often with a silver or white flaking layer[4].

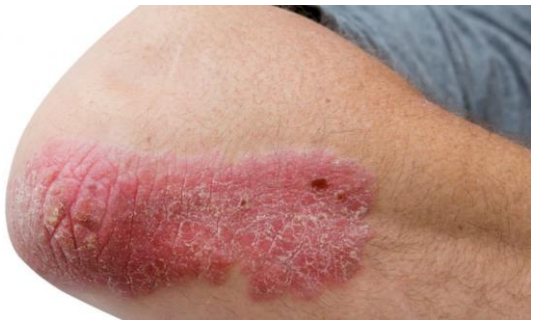

Figure 1: Plaque psoriasis

2.*Guttate Psoriasis*

Guttate psoriasis is often triggered by a bacterial infection, such as strep throat. It appears as small, red, drop-like lesions on the skin. Guttate psoriasis is more common in children and young adults. In this case, patients severely present small drop like lesions which respond well to topical treatments and phototherapies[4].

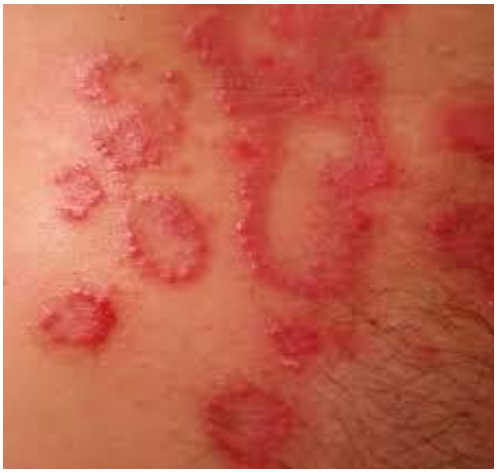

Figure 2: Guttate psoriasis

3. Flexural Psoriasis

The quality of life of a psoriatic patient may be impaired considerably by facial and flexural psoriasis. This type of psoriasis is an extrapolative marker indicating a poor prediction of psoriasis. Facial and flexural psoriasis cannot be considered as dissimilar disease entities but rather as site differences[4].

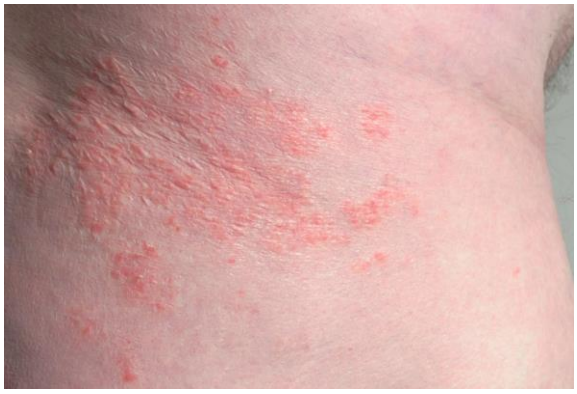

Figure 3: Flexural psoriasis

4. Erythrodermic Psoriasis

Erythroderma is a scaly erythematous dermatitis that involves 90% or more of the cutaneous surface. The most mutual dermatoses underlying erythroderma are psoriasis and eczema. Erythroderma may be also caused by cutaneous T cell lymphomas[4].

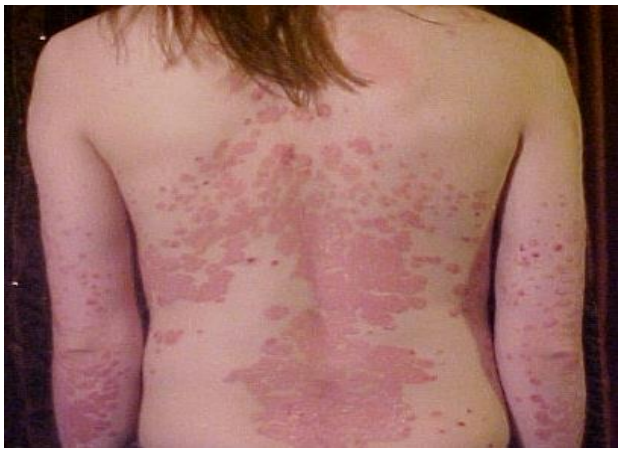

Figure 4: Erythrodermic psoriasis

5. Pustular Psoriasis

The patients who are suffering from pustular psoriasis or related pustular diseases may genetic abnormalities which impair the function of crucial players of the innate skin immune system. Detection of these irregularities has changed the paradigm of these diseases recently[5].

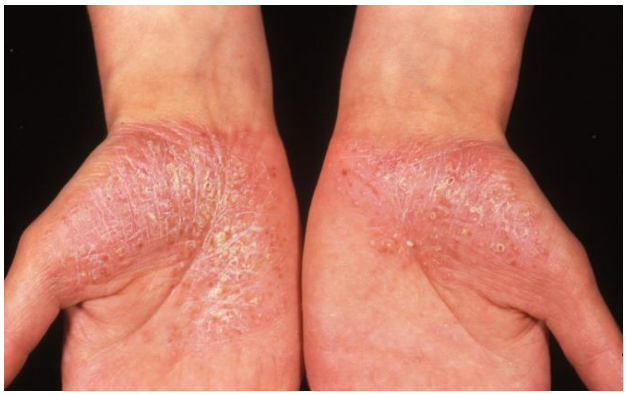

Figure 5: Pustular psoriasis

7. Palmoplantar Psoriasis

Plaque psoriasis that involves the palms and soles is characterized as palmoplantar psoriasis. This type of psoriasis is a challenge for dermatologists that is difficult to be treated with topical and systemic therapies[5].

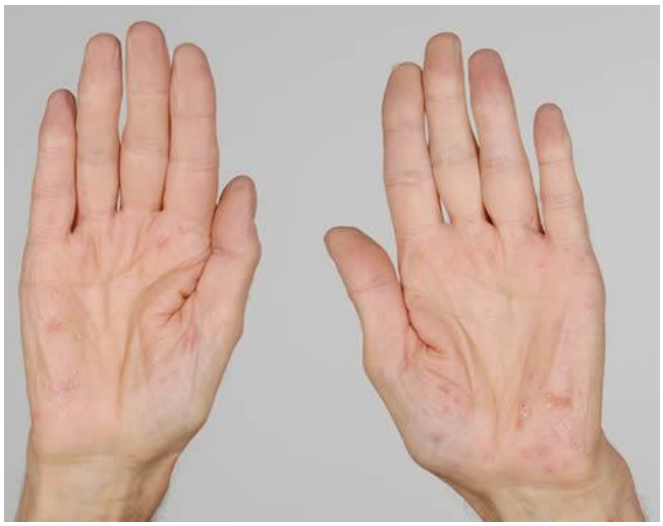

Figure 7: Palmoplantar psoriasis

8. Scalp Psoriasis

Scalp psoriasis can affect patients' lives harmfully and is often resistant to the treatment that is not been a major focus of a scientific study. The activity of secukinumab of patient-reported outcomes of scalp psoriasis is evaluated by this analysis[6].

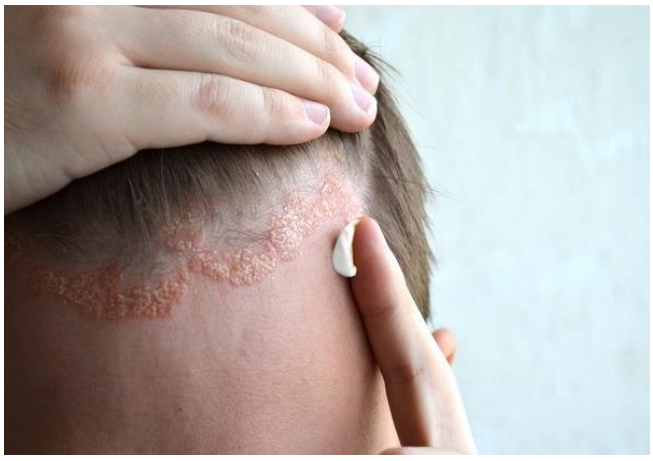

Figure 8: Scalp psoriasis

9. Nail Psoriasis

About 80% patients with psoriasis are likely to develop nail psoriasis as a result of the conditions of their nails as nails are considered epidermal appendages. Psoriasis can cause nail disorders of two patterns[6].

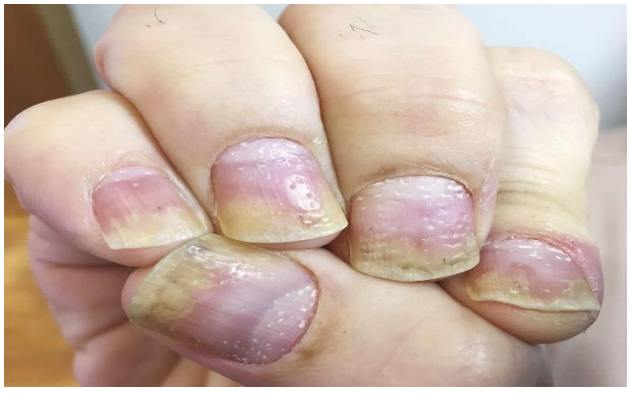

Figure 9: Nail psoriasis

10. Psoriatic Arthritis

An inflammatory rheumatic disorder of unknown etiology occurring in patients with psoriasis is named as psoriatic arthritis. An authenticated set of classification criteria for psoriatic arthritis having specificity of 98.7% and sensitivity of 91.4% and has recently established by The Classification Criteria for Psoriatic Arthritis group[6].

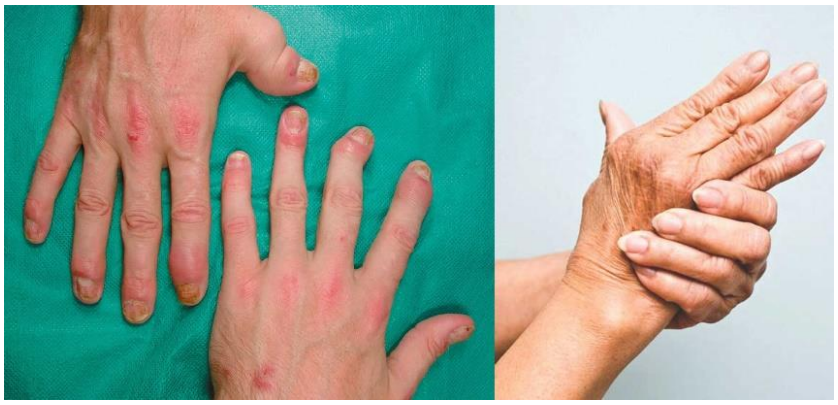

Figure 10: Psoriatic arthritis

Method

PubMed, Scopus and Google Scholar were searched for articles published from 2000 up to the present. Search terms included “herbal products and psoriasis”, “herbal treatments for psoriasis”, “topical herbal medication for psoriasis”, and “herbal drug delivery systems in psoriasis treatment”. References from reviews about herbal products and psoriasis were examined for additional articles and case reports. A manual search was also conducted, based on citations in scientific literature.

Inclusion And Exclusion Criteria

Selection criteria included articles which are examining herbal products used for the topical treatment of psoriasis by means of animal studies and clinical trials, and are comparing herbal products treatment vs. control treatments (placebo or active therapy). Other forms of psoriasis treatment than topical administration of herbal products (e.g., oral, systemic) were excluded from the study. Also, publications in languages other than English were excluded[7].

Pathology of Psoriasis

The pathophysiology of psoriasis involves both skin cells and immune cells. Psoriasis is typically characterized as inflamed skin with surface scales, thickening of the epidermis (acanthosis; granular layer is reduced or absent) caused by parakeratosis, which is a consequence of nuclei retention in SC keratinocytes caused by abnormal differentiation and hyperproliferation of epidermal keratinocytes[8]. Some scientific reports consider nitric oxide (NO), released from keratinocytes at high concentrations, as a key inhibitor of cellular proliferation and inducer of cell differentiation in vitro. Although a high-output NO synthesis is suggested by the expression of inducible NO synthase (iNOS), mRNA, and proteins in psoriasis lesions, the pronounced hyperproliferation of psoriatic keratinocytes may indicate that iNOS activity is too low to effectively deliver antiproliferative NO concentrations[9]. As a consequence, the impairment of corneocyte differentiation, including an impaired formation and secretion of lamellar body contents and the processing of lamellar body contents into lamellar bilayers, causes a reduction in the psoriatic skin barrier function. Moreover, skin barrier problems in psoriasis are not only the excessive growth and aberrant differentiation of corneocytes but also almost absent normal moisturizing factors (NMFs) like water, an imbalance of skin lipids (rise in the levels of cholesterol and fall in the levels of ceramides), and dry and sensitive skin[10]. The role of the immune system and its interactive network of leukocytes and cytokines in disease pathogenesis was also described. Psoriatic lesions are highly infiltrated with immune cells, most notably CD3+ T cells and CD11c+ dendritic cells. Proinflammatory cytokines produced by these cells, including tumour necrosis factor-α (TNF-α), interferon-γ

(IFN-γ), interleukin17 (IL-17), IL-20, IL-22, IL-23, IL-12, and IL-1b, have been linked to the pathogenesis of psoriasis through causing keratinocytes hyperproliferation. Moreover, IFN-γ and IL-15 seem to increase the apoptotic resistance of the keratinocytes. Also, growth factors and genetic factors like transforming growth factor (TGF)-β, toll-like receptor (TLR)-2, signal transducer and activator of transcription (STAT-3), coiled-coil alpha-helical rod protein 1 (CCHCR1), steroidogenic acute regulatory protein (StAR), and vitamin D receptor (VDR) are suggested to be the most critical factors governing the exacerbation of psoriasis. The essential transcription factor in psoriasis, nuclear factor kappa B (NF-κB), has been shown to be a key regulatory element occurring in a variety of immune and inflammatory pathways, in cellular proliferation and differentiation, and in apoptosis. An imbalance between the proapoptotic and antiapoptotic activities of NF-κB proteins has been demonstrated to cause differentiation and hyperproliferation in psoriatic lesions rather than in normal cells[11].

Topically Used Herbal Products for the Treatment of Psoriasis

Many herbal topical formulations have been marketed worldwide to prevent psoriasis. There are many advantages of using natural drugs, including patient compliance, less side-effects, easy availability, low-costs, and more than one mode of biochemical action for psoriasis treatment. Therefore, researchers are searching for new herbal products, which have the potential to be an alternative for synthetic drugs in psoriasis therapy.

Araroba Tree (Vataireopsis araroba (Aguiar) Ducke)

Dithranol, a potent topical treatment for psoriasis, is derived from the anthracene compound. This compound was originally extracted from chrysarobin, which is obtained from the bark of the araroba tree, found in the Amazon rainforests. Dithranol's therapeutic effect is attributed to its ability to inhibit the release of pro-inflammatory cytokines and the proliferation of keratinocytes. A multicenter study involving 106 patients with chronic psoriasis plaques revealed that short contact treatments with dithranol (ranging from 15 to 45 minutes) were significantly more effective than the standard treatment using calcipotriol ointment (applied twice daily, containing 50 µg/g of calcipotriol). This treatment was administered once daily for a duration of 12 weeks (Level of Evidence - A)[12].

Lace Flower (Ammi majus (L.) and Ammi visnaga (L.):

Ammi majus (L.) and Ammi visnaga (L.) Lam yield the furanocoumarins 8-methoxypsoralen (8-MOP) and 5-methoxypsoralen (5-MOP) for therapeutic purposes. These psoralens are considered phototoxic compounds, and their phototoxicity is activated when exposed to long-wave ultraviolet A (UVA) radiation. This activation can result in significant phototoxic skin reactions. In the context of PUVA therapy (psoralen plus UVA), these compounds have several therapeutic properties, including the inhibition of keratinocyte proliferation and immunosuppressive effects. These attributes are harnessed for the treatment of severe inflammatory skin conditions, such as psoriasis. Multiple clinical studies have provided evidence of the efficacy of PUVA therapy, including systemic PUVA (Level of Evidence - A), bath PUVA (Level of Evidence - A), and cream PUVA therapy (using 0.1% 8-MOP) (Level of Evidence - A) in the management of psoriasis[13].

Turmeric (Curcuma longa (L.):

Turmeric has been a significant component in Traditional Chinese Medicine (TCM) and Ayurvedic Medicine. In laboratory settings, both turmeric and its primary active compound, curcumin, have demonstrated anti-inflammatory, antimicrobial, and antioxidant properties. Recent years have seen laboratory and clinical investigations into the potential therapeutic benefits of curcumin for psoriasis. Curcumin's potential benefits for psoriasis may include inhibiting phosphorylase kinase, reducing the expression of pro-inflammatory cytokines like IL-17 and TNF-α, and enhancing the epidermal barrier by increasing the expression of involucrin and filaggrin in vitro. However, it's important to note that there is a lack of randomized, placebo-controlled studies involving turmeric and curcumin for psoriasis treatment[14].

Herbal Drug Use to Treat Psoriasis

Centella asiatica

Plant description. C. asiatica, commonly used in Southeast Asian countries, is a traditional Chinese medicine with broad medicinal value. C. asiatica from Apiaceae plant family is a small, perennial, herbaceous creeper. The genus Centella consists of 50 species distributed in tropical and subtropical regions of the world. The species is original to tropical countries such as India, Sri Lanka, China, Indonesia, Malaysia, South Africa, and Madagascar. It is native to the warmer regions of both hemispheres. This plant can be found along the sides of rivers, streams, ponds, and irrigated fields and grows wild in moist, gloomy locations up to 7000 feet. In India and Sri Lanka, it grows among stone walls or other rocky regions at the height of around 2000 feet. The plant has several synonyms such as C. coriacea Nannfd., Hydrocotyle asiatica L., H. lunata Lam., and Trisanthus cochinchinensis Lour[15].

Safety and Toxicity of Centella asiatica

Assessing the toxicity of Centella asiatica is a crucial aspect of pharmacological research and the quality control of plant-based health products. The most commonly reported toxicity test for Centella asiatica is acute toxicity, with a particular focus on its leaves. One study explored the use of whole plants as the test samples. Ethanol was predominantly used as the solvent, while acetone was employed in one experiment. The test subjects included mice, rats, and zebrafish, with most research findings indicating a high level of safety. Notable findings in toxicity studies include LD50 values of 1250 mg/kg and over 2000 mg/kg. In experiments using acetone extract, the LD50 value was notably higher at 4000 mg/kg. Whole-plant trials involving C. asiatica juice demonstrated that even at a dose of 7000 mg/kg, there were no signs of toxicity.

In clinical studies, oral dosages of C. asiatica extract at 250 mg and 500 mg were well tolerated in both single and repeated doses. Modern pharmacological assessments indicated minimal interaction potential between C. Asiatica physiologically active substances and cytochrome (CYP) isoenzymes. Additionally, the extract's heavy metal concentration remained within acceptable limits.

However, a few clinical instances reported adverse effects. In particular, three women who developed jaundice after 30, 20, and 60 days of C. asiatica use were diagnosed with granulomatous hepatitis. Their symptoms improved upon discontinuation of the medication. While preclinical studies have highlighted the pharmacological effects and safety of C. asiatica, these isolated clinical reports suggest the need for further research to determine the safest clinical dosages. It's worth noting that some plants, such as Germander, Skullcap, and Glycyrrhizin, contain di- or triterpenic active compounds that can lead to liver damage through apoptosis promotion and cell membrane changes. These pathways may provide insights into the mechanisms behind C. asiatica-related injuries[14].

Boswellia serrata Resin

Boswellia serrata resin, also known as olibanum or frankincense, is derived from the Burseraceae family. This resin is primarily sourced from regions characterized by dry and arid climates, including Yemen, Oman, India, and northeast Africa. Boswellia serrata resin has a long history of use in traditional medicine and is believed to possess various medicinal properties, including antiseptic, anti-inflammatory, antimicrobial, anxiolytic, and anti-cancer effects. These medicinal effects are attributed to a range of aromatic compounds present in the resin, with boswellic acid being the primary active principle. Additionally, the resin contains volatile oils composed of sesquiterpenes and monoterpenes, as well as diterpenes like cembrenol (serratol), incensole, and incensole acetate[16].

Lipophilic pentacyclic triterpene acids of the oleanane (α-boswellic acids), ursane-(β-boswellic acids), and lupane -type (lupeolic acids) are also found in Boswellia serrata resin, along with an ether-insoluble fraction containing polysaccharides such as arabinose, galactose, and xylose .Importantly, Boswellia serrata resin is considered safe for use, and it is permitted as a feed additive by the US Food and Drug Administration

(USFDA).The medicinal properties of Boswellia serrata have been extensively studied and documented by various researchers .However, despite its established medicinal potential, there is limited information regarding its suitability for use in aquaculture and its potential effects on the growth, immune response, and overall health of fish, such as Oreochromis niloticus[18].

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