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A comprehensive review on comparative study of Ayurveda and allopath in management of psoriasis

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Abstract

A common skin disorder called psoriasis causes thickened patches of the skin. Typically, it is discovered to be covered with silvery scales. Although psoriasis is regarded as a skin condition, it is actually the outcome of an immune system dysfunction. Types of psoriasis-Plaque psoriasis (psoriasis vulgaris), Pustular psoriasis, Nail psoriasis, Guttate psoriasis, Flexural psoriasis, Erythrodermic psoriasis. Activated T cells go into the skin from the circulation and lymph nodes and release cytokines (INF, IL-2) that cause pathologic alterations. Other cytokines like TNF- and IL-8 are produced locally by neutrophils and keratinocytes. Keratinocyte proliferation is a result of T-cell generation and activation. Histocompatibility studies show relationships between histocompatibility antigens and HLA-C6, TNF, and IL-3.1)

- T-helper ½,
- Th17 & Th17 Cytokines,
- Dendritic cells, IL-23 and TNF-α.

They have many causes that help in develop the psoriasis are

- Genetics,
- Lifestyle,
- HIV,
- Microbes,
- Medications.

They have many treatment therapies are-

Topical therapy-

- Corticosteroids,
- Calcipotriene,
- Tazarotene.

Keywords: Psoriasis; Keratinocyte; Cytokines; Pustular psoriasis; Allopathy; Guttate psoriasis

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1. Introduction

A common skin disorder called psoriasis causes thickened patches of the skin. Typically, it is discovered to be covered with silvery scales [1]. Although psoriasis is regarded as a skin condition, it is actually the outcome of an immune system dysfunction [1,2]. Before they can mature, skin cells rapidly emerge from underneath the skin's surface and build up on the surface. In psoriasis, this process—also known as turnover—might take just a few days instead of the typical month. These spots develop into crimson, swollen patches with white scaling over them. People who have cancer, AIDS, or autoimmune diseases typically experience it the hardest [3]. Scratching and minor skin injuries aggravate the psoriasis in affected regions, or irritations. Having psoriasis can itch or burn. In regions where the skin bends, it may split or break [3]. As an autoimmune condition, psoriasis is thought to be influenced by both genetic and environmental factors. Psoriasis is a non-contagious, dry, inflammatory, and unsightly skin condition that can affect the entire body [3]. The scalp, tips of fingers and toes, palms and soles, umbilicus, gluteus, under the breasts and genitals, elbows, knees, and shins are the area's most usually afflicted [3,4]. The knees, elbows, trunk, and scalp are the most typical areas of the body to develop a rash with itchy, scaly patches due to psoriasis. Psoriasis is a typical, persistent (chronic), incurable illness. It may hurt, disrupt your sleep, and make it difficult for you to focus. The illness typically flares up for a few weeks or months, then subsides for a period. Infections, cuts, burns, and specific drugs are common triggers in persons with a genetic propensity to psoriasis. You can manage your symptoms with the help of treatments. To improve your quality of life while dealing with psoriasis, you can also attempt new lifestyle practices and coping mechanisms. A chronic, non-contagious [5] autoimmune condition known as psoriasis is characterized by elevated, abnormal skin patches. These spots are scaly, dry, irritating, and red, pink, or purple [6]. The severity of psoriasis ranges from small, localized spots to total body coverage. The Koebner phenomenon describes how an injury to the skin might result in psoriatic skin changes there. [7] The five main kinds of psoriasis are erythrodermic, pustular, guttate, inverse, and plaque. [8] The majority of cases of psoriasis are plaque psoriasis, sometimes referred to as psoriasis vulgaris. [9] It often appears as white scales on top of crimson areas. The backs of the forearms, shins, navel region, and scalp are the body parts that are most frequently impacted. Lesions in guttate psoriasis have a drop form. Pustular psoriasis manifests as tiny, non-infectious blisters that are filled with pus. Opposite Red areas of psoriasis appear in skin creases. Erythrodermic psoriasis can arise from any of the other kinds and manifests as a very broad rash. Most psoriasis sufferers eventually develop problems with their fingernails and toenails. Examples of this could be nail pits or variations in nail color. Psoriasis does not have a proven cure, although a number of therapies can help manage the symptoms. Examples of these therapy include biologic therapies that target particular immunologic pathways, immunosuppressive medications like methotrexate, vitamin D3 cream, UV radiation, and steroid creams. Using only creams, around 75% of skin involvement gets better. Between 2 and 4% of people have the illness. [7] Equal numbers of men and women are affected. Although the disease can start at any age, it usually does so in adulthood. Psoriasis is linked to a higher incidence of depression, lymphomas, Crohn's disease, cardiovascular disease, and psoriatic arthritis. Up to 30% of people with psoriasis also have psoriatic arthritis.

2. Ayurveda versus allopathy

- Allopathy means "other than the disease" and is derived from the Greek words állos (other or different) and pathos (disease or suffering).
- The word "Ayurveda" comes from the Sanskrit roots "Ayus," which means "life," and "Veda," which indicates "knowledge" or "science." Ayurveda, thus, is defined as "The wisdom of Life [10]," as a result.

2.1. Types of psoriasis

2.1.1. Plaque psoriasis (psoriasis vulgaris):

It is psoriasis' most prevalent variant. The majority of those who have psoriasis are affected. Plaque psoriasis commonly manifests as elevated, reddish patches of skin that are coated with silvery-white scaly skin. Plaques are these regions. Plaque psoriasis, the most prevalent type of psoriasis, results in scale-covered, dry, elevated skin patches (plaques). They could be few or numerous. They typically show up on the scalp, lower back, elbows, and knees. Depending on the skin tone, the patches have different colours. On dark or Black skin, the afflicted skin may heal with transient color changes (post-inflammatory hyper pigmentation). The most prevalent form of psoriasis, psoriasis vulgaris, which is also known as chronic stationary psoriasis or plaque-like psoriasis, affects 85–90% of psoriasis sufferers. [11] Plaque psoriasis often manifests as elevated, inflammatory patches of skin that are covered in scaly, silvery-white skin. The elbows, knees, scalp, and back are the most typical locations for these plaque-like growths.[12]

2.1.2. Pustular psoriasis

It features raised bumps that are surrounded by pus that is not contagious (pustules). Red and sensitive skin can be found under and around pustules. Pustular psoriasis can be localized, most frequently affecting the hands and feet, or generalized, causing random, wide-spread patches to appear on any area of the body.[13] Pustular psoriasis manifests as raised bumps that are not contagious (pustules). [14] The skin is red and sensitive beneath and around the pustules. [15] Pustular psoriasis may only affect a certain area of the body or it may affect the entire body. Psoriasis pustules palm plantar is and acrodermatitis continua of Hallopeau are two examples of localized pustular psoriasis; they both affect the hands and feet. [16] Skin patches that are discolored and scaly are a symptom of psoriasis. Although it can happen anywhere on the body, it frequently happens near the knees and elbows. Psoriasis can strike at any age, although most cases occur in adults between the ages of 15 and 35. Children under the age of 10 are rarely affected by the illness. Along with other types of psoriasis, such as plaque psoriasis, pustular psoriasis can occur. It can manifest as in isolated regions, such as the hands and feet, or over the entire body. Rarely does it appear on the face. Usually, it starts out with a patch of skin that is painful and discolored. The characteristic big blisters of non-infectious pus appear within a few hours. In time, these blisters become crusty and brown. Skin can look glossy or scaly after they peel off.

2.1.3. Nail psoriasis

Make a variety of alterations to the finger and toe nails' appearance. These alterations include nail pitting, nail discoloration behind the nail plate, thickening of the skin beneath the nail, nail loosening (onycholysis), and nail crumble. [17] Psoriasis can alter the nails and cause a number of variations in how fingernails and toenails look. When psoriasis affects the skin, 40–45% of patients develop nail psoriasis; in patients with psoriatic arthritis, the lifetime prevalence is 80–90%. [29] These changes include crumbling of the nail, whitening of the nail, small areas of bleeding from capillaries under the nail, yellow-reddish discoloration of the nails known as the oil drop or salmon spots, pitting of the nails (pinhead-sized depressions in the nail are seen in 70% with nail psoriasis), and thinning of the skin under the nail (subungual hyperkeratosis).[18] An autoimmune condition known as nail psoriasis results in nail discoloration, pitting, and structural abnormalities. Although you can polish your nails and buff your nails to make them look better, it might still make you feel self-conscious. The symptoms of nail psoriasis can improve with therapy, and it is not communicable. An autoimmune condition known as nail psoriasis makes your skin cells grow rapidly. It's a form of psoriasis that impacts your finger and toe nails. Psoriasis of the nails frequently coexists with psoriatic rashes on other regions of the body. Psoriasis of the nails is an autoimmune disease. Your fingernails and toenails change color, become pitted, and undergo other alterations as a result. Although there is no cure, therapies can lessen the associated symptoms. Psoriasis of the nails is prevalent. More than 50% of persons with psoriasis and about 86% of those with psoriatic arthritis are affected by it. Not a fungus, nail psoriasis. Psoriasis of the nails is an autoimmune disorder. Your immune system overreacts, which causes the premature growth of new skin cells.

2.1.4. Guttate psoriasis

Numerous small, oval (teardrop-shaped) dots are its defining feature. They cover a substantial portion of the body, including the scalp, limbs, and trunk. Strep throat infection and guttate psoriasis are related. A skin condition called as guttate psoriasis frequently manifests suddenly and usually follows an infection like strep throat. Although it can occur in adults, it more frequently affects young individuals. Small, red scaly areas of skin irritation are the condition's signature symptom. Children account for the majority of cases with guttate psoriasis. However, it can also occur in young adults, particularly those under 30. About 2% of all psoriasis cases have guttate psoriasis. Chronic psoriasis and guttate psoriasis are related but not the same disease. The majority of sufferers of guttate psoriasis will fully heal. However, it's thought that roughly one-third of those who get guttate psoriasis go on to get chronic psoriasis, which results in larger patches "Plaques" are scaly regions. Psoriasis is an inflammatory condition, which means that for some reason, your immune system overreacts and results in the symptoms of psoriasis. Numerous tiny, scaly, droplet-like lesions that are red or pink are present in guttate psoriasis (papules). Large portions of the body, particularly the trunk but also the limbs and scalp, develop these many psoriasis lesions. A streptococcal infection, usually streptococcal pharyngitis, frequently causes guttate psoriasis.[19]

2.1.5. Flexural psoriasis (inverse psoriasis)

It shows as skin areas that are smooth and irradiated. It is mostly found in skin folds between the thighs and under a chubby stomach (pannus), and beneath the breasts (infra mammary fold). [20] A condition called inverse psoriasis makes your skin cells grow very quickly. A specific type of psoriasis called skin fold psoriasis manifests itself in these places, as shown in Fig 1 such as:

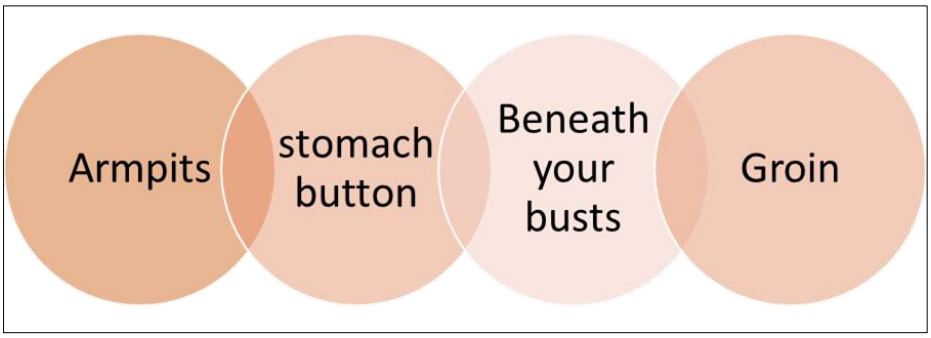

Figure 1 Skin fold psoriasis in places

It resembles a smooth, shiny, discolored (brown, red, or purple) rash with a wet feeling. The term "autoimmune illness" is frequently used to describe inverse psoriasis. However, the disease-causing antibody has not yet been found. Therefore, it falls within the category of an immune-mediated disease. This indicates that while the precise explanation is unknown, doctors think it may be related to how your immune system reacts. Sometimes it is referred to as inter-trig nous psoriasis (inter-trij-uh-nus) by medical professionals. Your skin can be afflicted with psoriasis and inverse psoriasis. Thick, discolored areas of skin with white or silvery scales are the hallmark of psoriasis. Plaques are the big, scaly spots. Because it affects moist parts of your body, inverse psoriasis doesn't have the thick, scaly plaques that other varieties of psoriasis do. A psoriasis rash appears shinier than an inverse psoriasis rash does. Inflammatory skin diseases that cause rashes in your skin folds include inverse psoriasis and intertrigo. However, the causes and therapies of intertrigo and inverse psoriasis are distinct. Inter-trigo is a result of skin rubbing against itself. Your skin's surface surfaces clump together in the folds of your skin as a result of trapped moisture. Moisture makes the friction worse, which damages the skin and causes inflammation. Keeping the affected area cool, dry, and clean can help with intertrigo. Immune-mediated diseases include inverse psoriasis. This implies that instead of defending your body from pathogens like bacteria or viruses, your immune system is attacking certain areas of your skin. Inverse psoriasis symptoms can either be eliminated or reduced with the use of treatments. However, since inverse psoriasis is a persistent (chronic) disorder, flare-ups may happen at any time during your lifetime.

2.1.6. Erythrodermic psoriasis

The majority of the body's surface is extensively inflamed and exfoliated, and there may be intense itchiness, edema, and discomfort. Especially after the abrupt discontinuation of systemic therapy, it frequently results from the worsening of unstable plaque psoriasis. Because more severe inflammation and exfoliation interfere with the body's capacity to regulate temperature and the skin's ability to conduct barrier functions, this type of psoriasis may be lethal.[21] A rare skin disorder called erythrodermic psoriasis results in a crimson rash covering much of your body. The rash resembles a burn and can be just as deadly because it can lead to dehydration, fever, and chills. Erythrodermic psoriasis needs to be treated right away by a doctor. An estimated 3% of Americans, usually adults, suffer with psoriasis. Plaque psoriasis affects as many as 9 out of 10 patients with psoriasis. The erythrodermic variant of psoriasis is rare, affecting just around 3% of those with the condition. Plaque psoriasis is present in about 1 in 3 individuals who develop erythrodermic psoriasis. Autoimmune diseases include psoriasis. It occurs when your immune system overreacts incorrectly and damages your own body. This reaction generates inflammation, which speeds up the formation of new skin cells. The greatest risk for developing erythrodermic psoriasis is in those with poorly managed plaque psoriasis. Erythrodermic psoriasis can develop if psoriasis medications, such as corticosteroids or immunosuppressants, are abruptly stopped. Symptoms can also result from excessive use of drugs like retinoids, which are related to vitamin A, or topical steroids.Erythrodermic psoriasis can appear in certain people after: as shown in Fig 2.

In a few of days, erythrodermic psoriasis can develop suddenly (as an acute rash). Plaques typically develop from an existing psoriasis rash. A few months may pass before erythrodermic psoriasis fully develops. On more than 90% of your body, you experience redness and inflammation that resembles a severe burn or sunburn. The skin rash is extremely irritating and could feel scorching. Additionally, your skin could start to peel off in big sheets. Toenails and fingernails fall out in some persons. Symptoms of erythrodermic psoriasis might come and go. Treatments can manage the condition and even put it into remission, which results in a minimal or even absent rash and other symptoms. However, flare-ups of the diffuse rash and symptoms can happen.

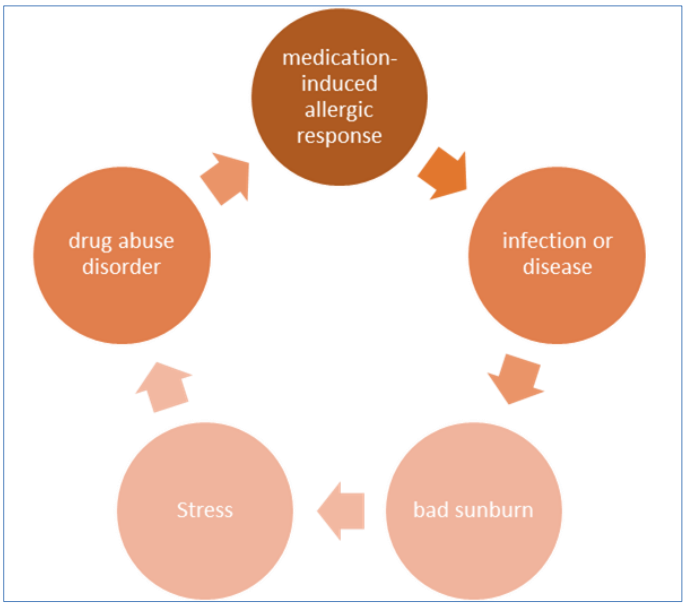

Figure 2 Erythrodermic psoriasis can affect following people

3. Pathophysiology

Needs for cutaneous immune T-cell-mediated activation 2 signals that are mediated through surface cell-cell contact proteins and cells that deliver antigens, such as macrophages or dendritic cells: (2) Co stimulation, which is mediated by different surface contacts, and (1) interaction between T-cell receptor and antigen. Activated T cells go into the skin from the circulation and lymph nodes and release cytokines (INF, IL-2) that cause pathologic alterations. Other cytokines like TNF- and IL-8 are produced locally by neutrophils and keratinocytes. Keratinocyte proliferation is a result of T-cell generation and activation. Histocompatibility studies show relationships between histocompatibility antigens and HLA-C6, TNF, and IL-3.[22]

3.1. T-helper 1/2-

It was discovered in 1979 that cyclosporine A (CsA) reduced psoriatic skin eruptions, indicating that the immune system and keratinocytes are both involved in the pathogenesis of psoriasis. Large numbers of activated CD4+ and CD8+ lymphocytes were seen in the skin and peripheral blood of psoriatic patients, leading to the early assumption that they played an equal role in the inflammation associated with psoriasis. [23-24] As a result, it was discovered that CD4+ T-helper (Th) cells were more crucial than CD8+ lymphocytes in the development of skin lesions similar to psoriasis in mice that received transplants of activated Th cells from psoriatic patients.[25-26] Additionally, psoriatic lesions had higher levels of Th1 cytokines like c-interferon (IFN-c), tumor necrosis factor-a (TNF-a), and interleukin (IL)-12, but no such Th2 cytokine expression levels (IL-4, IL-5, and IL-10) increased.[27-28] These results defined psoriasis as a Th1-type illness. [29] However, neither IFN-c nor TNF-a can cause keratinocyte growth. [30-31] It was hypothesized that additional important players would be involved in the development of psoriasis because the pathophysiology of the condition could not be fully understood based just on Th1 functions. [32]

3.2. Th17 & Th17 Cytokines

Th17 cells have gained notice recently as an important component of psoriasis. It has been suggested that Th17 is a novel Th subtype that produces IL-17A and cannot be classified as either Th1 or Th2 using the conventional paradigm. 16 It was discovered that activated memory CD4+ T cells produce IL-17A. 17 In addition to Th17 cells, CD8+ cells, cd-T-cell receptor cells, and natural killer T cells also secrete tiny amounts of IL17A. Six cytokines make up the IL-17 family (IL-17A to IL-17F). IL-17 cytokines, particularly IL-17A and IL-17F, play a part in defense against external pathogen infection. Studies on mice lacking the IL-17 receptor revealed that IL-17 has significant roles in defending the body against infection by Gram-negative bacteria and fungi. 33-34 It has been established that IL-17 and psoriasis are closely related. High quantities of IL-17 mRNA were found in the lesion skin of those with psoriasis but not in the non-lesion skin. IL-17 increased the keratinocytes' production of the pro-inflammatory cytokines IL-6 and IL-8, which aggravate psoriasis. Additionally, imiquimod, a Toll-like receptor (TLR)7/8 ligand and strong immune stimulator, when applied topically to mice, caused psoriasis-like dermatitis and the production of IL-17A and IL-17F. Further evidence that pro-

inflammatory cytokines, such as IL-17A, are involved in the onset of psoriasis comes from the fact that both CsA and anti-TNF-a agents reduced the levels of IL-17A, IFN-c, IL-23, and chemokine (C-C motif) ligand 20 in psoriatic lesions in conjunction with the improvement of psoriatic eruptions. These data imply that the IL-17 family is significant during psoriasis. Interleukin-22, a cytokine belonging to the IL-10 family, is intimately related to the inflammation associated with psoriatic skin (Fig. 1). Th17 cells are the primary producers of IL-22. T22, a cell type that only makes IL-22, has also been discovered. The IL-22 receptor is primarily found on the expressed on keratinocytes, and by promoting keratinocyte proliferation, IL-22 causes epidermal hyperplasia. Patients with psoriasis have higher levels of IL-22 in their peripheral blood compared to healthy individuals. In contrast, IL-22 expression is downregulated in connection with ant psoriatic therapy-induced remission and upregulated in psoriatic skin lesions. [33] These results imply that IL-22 plays significant roles in the pathophysiology of psoriasis and increases the proliferation of keratinocytes.

3.3. Dendritic cells, IL-23 and TNF-α

In a study looking for members of the IL-6 cytokine family, interleukin-23 was found. Dendritic cells (DC), activated monocytes, macrophages, T cells, and B cells all produce IL-23, a heterodimer made up of IL-23p19 and IL-12p40 (IL-12/23p40) chains. [34,35] IL-23 interacts to its heterodimeric receptor, which is expressed on memory T cells, natural killer T cells, monocytes, and DC and is made up of IL-12Rb1 and IL23R subunits. By encouraging Th17 expansion, IL-23 controls the growth and upkeep of the Th17 population. Using animal models of collagen-induced arthritis, autoimmune encephalomyelitis, and inflammation related to Th17, the involvement of IL-23 in Th17 was revealed. These conditions were markedly improved by the absence of IL-23 receptors, which are made up of IL-23p19 and IL-12p40 (IL-12/ 23). [36] Both in humans and mice, the function of IL-23 in cutaneous inflammation has been studied. Human psoriatic skin lesions have been demonstrated to overexpress IL-23 and IL-12p40 (IL-12/23p40), with mature DC, monocytes, and DC generated from monocytes producing the majority of the IL-23p19 in the papillary dermis. It has been demonstrated that IL-23 causes an increase in the production of TNF-a, IL-12p40 (IL-12/23p40), IL-23p19, and IL-20R2 in mice, which in turn mediates acanthosis and hyperkeratosis (IL-20 receptor subunit). [37-38] In psoriatic skin lesions, DC is the predominant source of IL-23.

4. Causes

4.1. Genetics

Researchers have discovered genetic loci linked to the disorder, and about one-third of persons with psoriasis indicate a family history of the ailment. According to studies on identical twins, there is a 70% likelihood that one of the twins will end up with psoriasis if the other sibling does. Nearly 20% of nonidentical twins run this risk. These results imply that psoriasis development involves both a genetic predisposition and an environmental response. Although numerous genes are linked to psoriasis and the condition has a significant genetic component, it is unclear how these genes interact. The majority of the discovered genes are connected to the immune system, particularly the T cells and the major histocompatibility complex (MHC). Genetic investigations are useful because they can reveal biological mechanisms and pathways for more research and possible drug targets. [39]

4.1.1. Lifestyle

Chronic infections, stress, and alterations in season and climate are among the factors said to make the condition worse. Hot water, scratching psoriasis skin lesions, dry skin, excessive alcohol intake, smoking, and obesity are additional variables that may exacerbate the illness.

4.1.2. HIV

Human immunodeficiency virus (HIV)-positive individuals have a psoriasis prevalence that is equivalent to HIV-negative individuals', but psoriasis in HIV-positive individuals is typically more severe. Psoriatic arthritis is substantially more common in HIV-positive people with psoriasis than in people without the virus. While the immune response in psoriasis vulgarism is characterized by a pattern of cellular signals typical of Th1 subset of CD4+ helper T cells and Th17 helper T cells, the immune response in HIV-infected individuals is typically characterized by cellular signals from Th2 subset of CD4+ helper T cells [40]. It is believed that the decreased CD4+-T cell numbers lead to an overactivation of CD8+-T cells, which are responsible for the worsening of psoriasis in HIV-positive individuals. Psoriasis is frequently severe and could not respond to conventional medication in people with HIV/AIDS. A severe flare-up of psoriasis and/or psoriatic arthritis can occur in people who have had long-term, well-controlled psoriasis in response to a new HIV infection.

4.1.3. Microbes-

According to some accounts, psoriasis develops after strep throat and may become worse if Staphylococcus aureus, Malassezia spp., or Candida albicans are present on the skin or in the gut. Children and teenagers are frequently affected by guttate psoriasis, which can be brought on by a recent group A streptococcal infection (tonsillitis or pharyngitis).

4.1.4. Medications-

Beta blockers, lithium, antimalarial drugs, nonsteroidal anti-inflammatory drugs, terbinafine, calcium channel blockers, captopril, glyburide, granulocyte colony-stimulating factor, interleukins, interferons, lipid-lowering drugs, and paradoxically TNF inhibitors like infliximab or adalimumab can all cause drug-induced psoriasis. Due to the rebound effect, stopping corticosteroids (a topical steroid cream) might make psoriasis worse.

4.2. Treatment

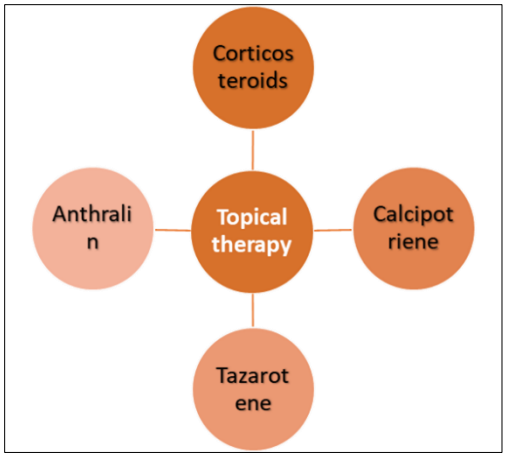

Figure 3 Treatment used in Allopath

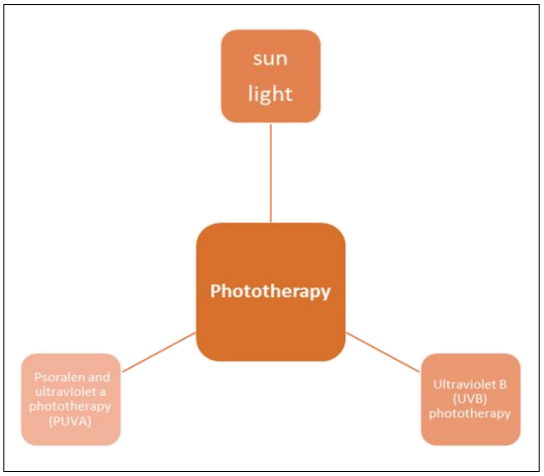

Figure 4 Therapies included in Psoriasis Patient

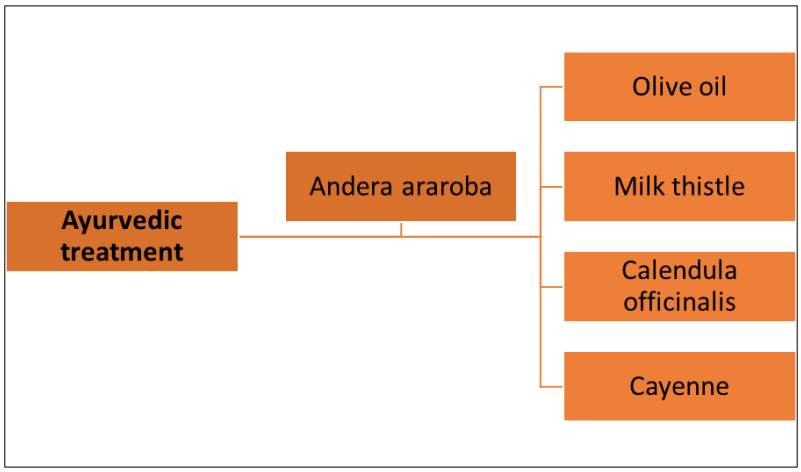

Figure 5 Overview of treatment used in Ayurveda

5. Conclusion

A multifaceted, extremely complex inflammatory skin condition, psoriasis. The development of focused biological treatments, which are not without potential risks, has been sparked by a new understanding of this complex disease. Options for improving safety and efficacy in the therapy of psoriasis are presented by a review of alternative natural remedies. In India, Ayurveda has long known about medicinal plants, herbs, spices, and herbal treatments. Their use will play a significant role in the treatment of this illness. Psoriasis pathogenesis involves a variety of cell types, such as inflammatory cells, keratinocytes, and antimicrobial peptides. The creation of new treatment drugs for psoriasis will be facilitated by additional basic research into the pathophysiology. Therefore, to prove the reliability and potency of Ayurvedic medications, high-quality clinical trials are necessary. The value to the patient comes from using the two systems in a balanced, evidence-based manner; this must be encouraged, especially in developing nations like India with a low doctor-to-patient ratio. This review will bring some insights into the disease's pathogenesis while also giving researchers additional options to create effective and reasonably priced psoriasis medications, especially in underdeveloped nations.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest.

References

[1] Shaikh G, Ali S, Talmale S, Surwase US (2012) Alternative Medicine For Psoriasis –Natural Herbal Ayurvedic Treatment – A Review. Int J Ayu Herb Med 2: 455-463.

[2] Deshkar N (2008) A Comphrehensive Review of RubiaCardifolialinn. Pharmacog 2: 124-134.

[3] Kumar A (2016) Fight Psoriasis Naturally Through Ayurveda. 6: 2231-6876.

[4] Khyade M (2009) Pharmacognostical and Physiochemical Standardization of Leaves of WrightiaTinctoria R.Br. Int J Pharma Res Dev 8: 1-10. Link: http://bit.ly/2CACNhJ.

[5] Boehncke WH, Schön MP (September 2015). "Psoriasis". Lancet. 386 (9997): 983–94. Doi:10.1016/S0140-6736(14)61909-7.

[6] National Institute of Arthritis and Musculoskeletal and Skin Diseases. 12 April 2017. Archived from the original on 22 April 2017. Retrieved 22 April 2017.

[7] LeMone P, Burke K, Dwyer T, Levett-Jones T, Moxham L, Reid-Searl K (2015). Medical-Surgical Nursing. Pearson Higher Education AU. p. 454.

[8] Ely JW, Seabury Stone M (March 2010). "The generalized rash: part II. Diagnostic approach". American Family Physician. 81 (6): 735–9.

[9] Jain S (2012). Dermatology: illustrated study guide and comprehensive board review. Springer. pp. 83–87.

[10] Parisi R, Symmons DP, Griffiths CE, Ashcroft DM (February 2013). Identification and Management of Psoriasis and Associated ComorbidiTy (IMPACT) project team. "Global epidemiology of psoriasis: a systematic review of incidence and prevalence". The Journal of Investigative Dermatology. 133 (2): 377–85. doi:10.1038/jid.2012.339

[11] Palfreeman AC, McNamee KE, McCann FE (March 2013). "New developments in the management of psoriasis and psoriatic arthritis: a focus on apremilast". Drug Design, Development and Therapy. 7: 201–10. doi:10.2147/DDDT.S32713.

[12] Colledge, N.R.; Walker, B.R.; Ralston, S.H., eds. (2010). Davidson's principles and practice of medicine (21st ed.). Edinburgh: Churchill Livingstone/Elsevier. pp. 1260–1.

[13] Karodi R, Jadhav M, Rub RA, Bafna A (2009) Evaluation of the Wound Healing Activity of a Crude Extract of RubiaCordifolia (Indian madder) in Mice. Int J Applied Res Natural Products 2: 12-18.

[14] Robinson A, Van Voorhees AS, Hsu S, Korman NJ, Lebwohl MG, Bebo BF, Kalb RE (August 2012). "Treatment of pustular psoriasis: from the Medical Board of the National Psoriasis Foundation". Journal of the American Academy of Dermatology. 67 (2): 279–88. doi:10.1016/j.jaad.2011.01.032.

[15] Raychaudhuri SK, Maverakis E, Raychaudhuri SP (January 2014). "Diagnosis and classification of psoriasis". Autoimmunity Reviews. 13 (4–5): 490–5. doi:10.1016/j.autrev.2014.01.008.

[16] Rendon A, Schäkel K (March 2019). "Psoriasis Pathogenesis and Treatment". International Journal of Molecular Sciences. 20 (6): 1475. doi:10.3390/ijms20061475.

[17] A Case Report of Severe Psoriasis in a patient with AIDS: The Role of the HIV Virus and the Therapeutic Challenges involved. 13 National Skin Center.

[18] Tan ES, Chong WS, Tey HL (December 2012). "Nail psoriasis: a review". American Journal of Clinical Dermatology. 13 (6): 375–88. doi:10.2165/11597000-000000000-00000.

[19] Weigle N, McBane S (May 2013). "Psoriasis". American Family Physician. 87 (9): 626–33.

[20] Kameshwari MNS, Paramasivam G (2012) Urgineaindica and its role in psoriasis: A Review. Int J Pharm Life Sci 3: 2236-2242.

[21] Lebwohl M (1999) The role of salicylic acid in the treatment of psoriasis. Int J Dermatol 38: 16-24.

[22] Dipero JT, Wells BG, Schwinghammer TL, DiPiro CV (2015) Pharmacotherapy hand book; 9th edtn, MC Graw Hill Publications.

[23] Bos JD, Hagenaars C, Das PK, Krieg SR, Voorn WJ, Kapsenberg ML. Predominance of “memory” T cells (CD4+, CDw29+) over “naive” T cells (CD4+, CD45R+) in both normal and diseased human skin. Arch Dermatol Res 1989; 281: 24–30.

[24] Ferenczi K, Burack L, Pope M, Krueger JG, Austin LM. CD69, HLA-DR and the IL-2R identify persistently activated T cells in psoriasis vulgaris lesional skin: blood and skin comparisons by flow cytometry. J Autoimmun 2000; 14: 63–78.

[25] Wrone-Smith T, Nickoloff BJ. Dermal injection of immunocytes induces psoriasis. J Clin Invest 1996; 98: 1878–1887.

[26] Boyman O, Hefti HP, Conrad C, Nickoloff BJ, Suter M, Nestle FO. Spontaneous development of psoriasis in a new animal model shows an essential role for resident T cells and tumor necrosis factor-alpha. J Exp Med 2004; 199: 731–736.

[27] Nestle FO, Turka LA, Nickoloff BJ. Characterization of dermal dendritic cells in psoriasis: autostimulation of T lymphocytes and induction of Th1 type cytokines. J Clin Invest 1994; 94: 202–209.

[28] Schlaak JF, Buslau M, Jochum W et al. T cells involved in psoriasis vulgaris belong to the Th1 subset. J Invest Dermatol 1994; 102: 145–149.

[29] 29. Austin LM, Ozawa M, Kikuchi T, Walters IB, Krueger JG. The majority of epidermal T cells in psoriasis vulgaris lesions can produce type 1 cytokines, interferon-c, interleukin-2, and tumor necrosis factor-a, defining TC1 (cytotoxic T lymphocyte) and TH1 effector populations: a type 1 differentiation bias is al. J Invest Dermatol 1999; 113: 752–759.

[30] Hancock G, Kaplan G, Cohn Z. Keratinocyte growth regulation by the products of the immune cells. J Exp Med 1988; 168: 1395– 1402.

[31] Detmar M, Orfanos CE. Tumor necrosis factor-alpha inhibits cell proliferation and induces class II antigens and cell adhesion molecules in cultured normal human keratinocytes in vitro. Arch Dermatol Res 1990; 282: 238–245.

[32] Krueger G, Callis K. Potential of tumor necrosis factor inhibitors in psoriasis and psoriatic arthritis. Arch Dermatol 2004; 140: 218–225.

[33] Ye P, Garvey P, Zhang P, Nelson S, Bagby G, Summer W. Interleukin-17 and lung host defense against Klebsiella pneumoniae infection. Am J Respir Cell Mol Biol 2001; 25: 335–340.

[34] Huang W, Na L, Fidel PL, Schwarzenberger P. Requirement of interleukin-17A for systemic anti-Candida albicans host defense in mice. J Infect Dis 2004; 190: 624–631.

[35] Oppmann B, Lesley R, Blom B et al. Novel p19 protein engages IL-12p40 to form a cytokine, IL-23, with biological activities similar as well as distinct from IL-12. Immunity 2000; 13: 715–725.

[36] 36 Parham C, Chirica M, Timans J et al. A receptor for the heterodimeric cytokine IL-23 is composed of IL-12Rbeta1 and a novel cytokine receptor subunit, IL-23R. J Immunol 2002; 168: 5699– 5708.

[37] Pirhonen J, Matikainen S, Julkunen I, Alerts E. Regulation of virusinduced IL-12 and IL-23 expression in human macrophages. J Immunol 2002; 169: 5673–5678.

[38] Lee E, Trepicchio WL, Oestreicher JL et al. Increased expression of interleukin 23 p19 and p40 in lesional skin of patients with psoriasis vulgaris. J Exp Med 2004; 199: 125–130.

[39] Kopp T, Lenz P, Bello-Fernandez C, Kastelein RA, Kupper TS, Stingl G. IL-23 production by cosecretion of endogenous p19 and transgenic p40 in keratin 14/p40 transgenic mice: evidence for enhanced cutaneous immunity. J Immunol 2003; 170: 5438–5444