



Psoriasis – An Overview

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ABSTRACT

The main objectives of this review article are to discuss different aspects of psoriasis including its etiology, pathogenesis, co-morbidities, complications and management etc. and to discuss about psychosocial impact & quality of life in the patients of psoriasis. Psoriasis is one of the most common dermatologic diseases affecting upto 2.5 % of world population. The disease psoriasis, most commonly manifests on the skin of the elbows, knees, scalp, lumbosacral areas, intergluteal clefts and glans penis. In 30% of patients, the joints are also affected. The skin is the important organ of communication with the external world and have an eternal relationship with mind (psyche). Therefore, more than a cosmetic nuisance, psoriasis produces anxiety, depression and other psychological problems that affect the quality of life. Psychological factors have traditionally been associated with the onset, development and persistence of psoriasis. Stress is emphasized as one of the major important factors in the initiation or exacerbation of psoriasis. Patients of psoriasis always experience physical, mental and socio-economic embarrassment in the society. This embarrassment leads to mental stress which further causes aggravation of pre-existing disease. Psoriasis is now considered a complex, chronic, multifactorial, inflammatory disease with involvement of immunological, genetic and environmental factors.

Keywords: Psoriasis, Pathogenesis, Molecular inflammation, Psychosocial, *Rasayana*

INTRODUCTION

Psoriasis is a non-infectious chronic inflammatory skin disorder clinically characterizes by erythematous sharply demarcated papules and rounded plaques covered by silvery micaceous scales. Psoriasis is notoriously chronic and is well known for its course of remissions and relapses [1,2]. The word 'psoriasis', is derived from the Greek word 'psora' meaning "itch" or "scurf" or "rash", although most patients suffering from the condition do not complain of itching. It has been known since ancient times and was originally considered a type of leprosy [3].

Psoriasis is one of the oldest recorded skin diseases. The famous Hippocrates and his school (460–377 B.C.) produced objective and meticulous descriptions of many skin disorders. In their classification, dry scaly eruptions were grouped together under 'lopoi' (epidermis). This group probably included psoriasis and leprosy. The confusion between Psoriasis and leprosy remained for many centuries. From 1000 – 1400 A.D. the

prevalence of leprosy was very high. Many psoriatic patients, diagnosed as leprosy, received the same brutal treatment as leprosy patients and were isolated from the community. Psoriasis was again mentioned in the first century by Cornelius celsus, a Roman author. Celsus described it as the fourth variant of impetigo, a condition caused by *Staphylococcus pyogenes*. This condition appears as red patches with watery blisters on the skin. The English dermatologist, Robert Willan (1757 ~ 1812) recognized psoriasis as an independent disease. He identified two categories. "Leprosa Graecorum" was the term he used to describe the condition when the skin had scales. Psora Leprosa described the condition when it became eruptive [4].

Galen was the first who used the word 'Psoriasis'. Under this name he described a skin disorder characterized by a scaliness of the eyelids, corners of the eyes and the scrotum along with the itching and excoriations. Although, this type of clinical presentation was termed psoriasis, is in fact more similar with eczema. In 1841, Ferdinand von Herba

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definitely separated the clinical picture of psoriasis from that of leprosy. Robert Willan (1809) was the first person who offers an accurate description of psoriasis and its different manifestations. Psoriasis has been described in great detail by Pussy (1933) and Bechet (1936). Simon (1949) has studied Psoriasis in Tropics and reported that psoriasis was 30 times more common in Europeans. Wright and Reed (1964) had proved definite link between Psoriatic arthritis and Reiter's disease. In 1977-78 Seville has investigated the relationship between psoriasis and psychological stress [5].

The physical and mental functioning of patients with psoriasis is reported to be affected as much as that of patients with cancer, arthritis, hypertension, heart disease, diabetes and depression. Physical and mental functioning scores for psoriasis patients are among the lowest of all groups (10/11 for physical and 9/11 for mental functioning, 11 representing the lowest functioning) [6].

Psoriasis can be extremely debilitating, both physically and emotionally. Lack of proper knowledge and frequent misunderstanding in the general and medical communities about the disorder add to the stigma and emotional stress associated with it. It may also interfere with various aspects of the quality of life such as personal relationships, sports, sexuality, self-care activity and activities at work or school [7].

Psoriasis is a chronic skin disease with severe psychosocial effects. Its chronicity, frequent relapses, the absence of permanent cure and symptoms such as pruritus make it hard to live with. Furthermore, the cosmetic disfigurement has a negative impact on the quality of life due to psychological stress, disruption of social relationships and difficulties in daily life [8].

PSORIASIS

Although traditionally psoriasis has been considered a dermatologic disease, contemporary medical literature is accumulating to support the assertion that psoriasis is actually a multisystem disorder [9].

Etiology: Exact etiology of psoriasis has yet to be discovered, the immune system and genetics are known to play major roles in its pathogenesis and manifestation. According to most workers, it is a heredo-familial disease brought on by stress. Psoriasis is now considered a multi-factorial disorder that has several factors like genetic predisposition, environmental and immunologically mediated inflammation. Current researches suggest that the inflammatory mechanisms are immune

based and most likely initiated and maintained primarily by T cells in the dermis [10]. Several risk factors/triggers participated in the etiology of psoriasis are described which is as follows [11-19]-

- Trauma: Psoriasis at the site of injury is well known and the phenomenon is termed as Koebner phenomenon. A wide range of injurious local stimuli, including physical, chemical, electrical, surgical, infective and inflammatory insults have been recognized to elicit psoriatic lesion.
- Environmental factors: several studies validated that the interaction between genes and environment is important in manifestation of psoriasis. Many environmental factors have linked to psoriasis, and have been implicated in the manifestation of disease and exacerbation of pre-existing disease.
- Infection: Acute guttate psoriasis is strongly associated with preceding or concurrent streptococcal infection, particularly of the throat. There is evidence that streptococcal infection may be important in chronic plaque psoriasis, and treatment with rifampicin and penicillin may lead to clearance of skin lesions.
- Drugs: There are many drugs reported to be responsible for the onset or exacerbation of psoriasis. Chief amongst these are lithium salts, antimalarials, beta blockers, ACE inhibitors, NSAIDs, and the withdrawal of corticosteroids. Some authors and colleagues have suggested that NSAIDs and beta blockers have little adverse effect but the adverse effect of lithium salts and antimalarials may be severe.
- Metabolic factors: The early onset of psoriasis in the women, with a peak around puberty, changes during pregnancy and provocation of psoriasis by high dose estrogen therapy potentially indicate a role for hormonal factors in the disease. A questionnaire study has provided data from 65 females who had one or more pregnancies after diagnosis was made. Psoriasis was improved approximately in 40% of the pregnancies, and worsened in 14%. Hypocalcaemia has been reported to occur in severe forms of psoriasis, particularly generalized pustular psoriasis.
- Psychogenic factors: Considerable clinical evidence exists for the role of psychogenic factors in onset and exacerbation of disease. Seville reported consistent links between major stressful life events and disease manifestation. Gupta reported more exacerbations and worsening of disease related with stress reactivity. Some other studies also established the role of psychogenic factors in the initiation or exacerbation of psoriasis.

- Alcohol and smoking: It has long been suspected that both cigarettes and alcohol have a detrimental effect on psoriasis. When controlled for confounding variables, studies suggested that alcohol may exacerbate pre-existing disease but does not appear to induce psoriasis. This effect seems greater in men than women. Heavy drinkers tend to have more extensive and inflamed disease. Increased alcohol consumption is a recognized stress response. Excess drinking is undoubtedly also a consequence of disease and leads to treatment resistance and reduces therapeutic compliance.
- Weather: Winter tends to be the most challenging season for people living with psoriasis. Numerous studies indicate cold weather is a common trigger for many people and that hot and sunny climates appear to clear the skin. Cold winter weather is dry, and indoor heat robs the skin of needed moisture. This usually worsens psoriasis. Psoriasis can become even more severe when the stress of the holidays and winter illnesses combine to compromise immune system. While hot and sunny may help clear psoriasis, air-conditioning can dry out the skin and aggravate psoriasis.

Pathogenesis: The most evident pathogenetic change leading to psoriasis is alteration in the cell kinetics of keratinocytes i.e. abnormal differentiation and hyperproliferation of keratinocytes. Keratinocytes are cells in the epidermis that produce keratin, a “protein that helps to protect the skin and underlying tissues from heat, microbes, and chemicals.” Patients with psoriasis shed and replace these cells every two to six days, a quite bit faster than normal which is 21 to 28 days leading to buildup of dead and living cells.

Role of keratinocytes: There is epidermal hyperplasia along with abnormal maturation of keratinocytes that leads to the development of the thick scaly plaques that are so characteristic of psoriasis. Gottlieb AB suggested that the epidermal changes and the inflammatory infiltrate composed of T cells with interspersed neutrophils may be linked together by the cytokines produced by both keratinocytes and leukocytes. The proposal based partly on the fact that epidermal acanthosis and keratinocyte mitoses were often seen in delayed type hypersensitivity reactions and after the intradermal injection of gamma interferon. Gamma interferon and its induced proteins have been demonstrated in active psoriatic plaques. Increased levels of the keratinocyte autocrine cytokines, transforming growth factor (TGF)-alpha and

interleukin (IL)-6, have been detected in active plaques. The apparent overexpression of IL-6 in hyperplastic psoriatic tissue may explain features of psoriasis that link keratinocyte proliferation with immune activation and tissue inflammation. Both IL-6 and gamma interferon increased TGF-alpha expression in normal cultured keratinocytes. Cytokines produced during immune activation and other inflammatory processes may lead to epidermal hyperplasia. It indicates that keratinocytes have an important role to play in the pathogenesis of psoriasis [20].

Several markers of abnormal keratinocyte differentiation have been found, and all have implication in the pathogenesis of the disease. These include aberrations of keratinocyte transglutaminase type I (TGase K), skin-derived antileukoproteinase (SKALP), migration inhibitory factor-related protein-8 (MRP-8), Involucrin, Filaggrin and keratin expression. Several possible biomarkers responsible for the overproduction of the keratinocytes have been found in psoriatic skin like epidermal growth factor (EGF), bone morphogenetic protein-6 (BMP-6), transforming growth factor-alpha (TGF-a), ornithine decarboxylase, activating protein (AP1) and mitogen-activated protein kinase (MAPK) [21].

Immunopathogenesis: Psoriasis was long-considered either a disorder of keratinocyte growth or a chronic inflammation. However, advancement in immunologic techniques and in genetic analysis over the past four decades have resulted in a reappraisal of the pathophysiology of psoriasis. Some consider psoriasis as an organ-specific autoimmune disease that is triggered by an activated cellular immune system and is similar to other immune-mediated diseases such as Crohn's disease, rheumatoid arthritis, multiple sclerosis and juvenile-onset diabetes [22-23].

The pathogenesis of psoriasis is a complex interaction among genetic, immunological, and environmental components. It was previously assumed that Th1 cells played the dominant role in the manifestation but, in recent years, the current concept favors the role of Th17 cells. Innate immune cells produce key cytokines (TNF- α , IFN- α , IFN- γ , IL-1 β , and IL-6) that activate dendritic cells. Activated dendritic cells present antigens and secrete mediators such as IL-12 and IL-23, leading to the differentiation of Th1 and Th17. IL-23 serves as a key master cytokine regulator. T cells secrete mediators (e.g., IL-17 and IL-22) that activate keratinocytes and induce the production of antimicrobial peptides, pro-inflammatory cytokines and chemokines. These mediators feed back into the pro-inflammatory disease cycle and shape the

inflammatory infiltrate [24]. Scarpa suggested simplified model of traffic of activated T-lymphocytes moving from gut-associated lymphoid tissue to skin and/or joints. [25].

Nesterov suggested a complex model of pathogenesis of the chronic dermatoses based on trigger role of blastocystosis (BLC). The blastocystosis can entail serious dysbiotic deviations in the microflora of intestine and disturbance of its barrier function which occurs because of both blastocystosis and disbacteriosis of parietal microflora. As a result too many toxic products of vital activity of blastocystis and microflora (it is not specified, which exactly) get to blood flow and support chronic endointoxication. The chronic endointoxication in its turn breaks the work of immune system and the balance of the oxidizers-antioxidants system. There is hyperactivation of processes of free-radical oxidation and reduction in antioxidative activity. The author assumes that disturbance of work of these systems appears to be sufficient for the initialization and support of chronic dermatoses, including psoriasis [26].

Molecular pathways of inflammation: The ability to develop effective therapeutics by rational design is crucially dependent on elucidation of the molecular circuitry of inflammation in human autoimmune diseases. Cytokine interactions in psoriasis have previously been illustrated as a 'type-1 pathway', which assumes a linear relationship between proximal inducers (IL-23 or IL-12), production of IFN- γ and TNF by type-1 T cells, and downstream activation of numerous IFN-responsive genes through signal transducer and activation of transcription 1 (STAT1) [27]. Although this model is conceptually useful, it accounts for only a small fraction of the more than 1,300 genes that become upregulated in psoriatic lesions [28]. Clearly, STAT1, STAT3 and nuclear factor- κ B (NF- κ B) transcription factors are activated in psoriasis. Upstream activators may well be IFNs for STAT1, and TNF or IL-1 for NF- κ B, but more recently discovered cytokines such as IL-20 and IL-22 also have the ability to activate STAT and NF- κ B pathways, thus supporting the network concept [29,30].

Role of molecular genetics: About 30 percent of individuals with psoriasis have a family history of the disease in a first- or second degree relative. At least nine chromosomal susceptibility loci have been elucidated (PSORS1-9). HLA-Cw6 is a major determinant of phenotypic expression. An association with the PSORS has been found with functional polymorphisms in modifier genes that mediate inflammation (e.g., TNF- α) and vascular

growth (e.g., VEGF) [31,32]. Thus, the pathogenesis of psoriasis is considered a complex interaction among genetic, immunological and environmental components.

Types and Clinical features: Commonly psoriasis can be grouped into five categories which include plaque or psoriasis vulgaris, guttate, inverse or flexural, pustular and erythrodermic.

Psoriasis varies greatly in clinical presentation and ranges from mild disease with isolated patches to extensive disease with confluent plaques involving multiple areas of the body. Plaque psoriasis is the most common subtype, affecting 80% to 90% of those with psoriasis [33].

The disease may present in varying degrees of severity during its course. Individual lesions may range from pinpoint lesions to large plaques. The size of the lesions helps to determine the psoriasis type. Clinical features of different types of psoriasis are as follows [34] -

Plaque type or psoriasis vulgaris

- This is the most common variety of psoriasis, diagnosed in 80% to 90% of patients.
- The patients have stable, slowly enlarging plaques, which remains unchanged for long period of time.
- The lesions are of variable size, sharply circumscribed, red, dry, and usually covered with layers of silvery white, micaceous scales.
- The most common involved areas are elbow, knees, gluteal cleft, and the scalp. Involvement tends to be symmetric.
- Generally characterized by absence of itching, but in tropic countries patients complain of slight or moderate pruritis which, if accompanied by secondary psychogenic stress and lichenification, is more marked.
- Nail involvement in up to 55% of patients with findings such as onycholysis, subungual hyperkeratosis and "oil drops".

Guttate psoriasis (Eruptive psoriasis)

- Usually characterizes by small papules of short duration (weeks to months).
- It is an acute variant, often seen in younger patients and characterized by an abrupt eruption of small drop shaped, red, individual lesions on the skin that are not normally as thick or as crusty as lesions of plaque psoriasis.
- The word guttate is from the Latin word meaning "drop". Sometimes known as teardrop psoriasis or raindrop psoriasis.
- Plaques are usually small, not more than 1cm in diameter.
- It is usually associated with acute group A-beta haemolytic streptococcal infection of pharynx in the preceding 7 to 10 days.

- Plaques are fairly widespread and may develop anywhere in the body, except the soles and palms. Most commonly affects the trunk and limbs but not frequently on the face, ears and scalp.
- This form of psoriasis may resolve on its own, occasionally leaving a person free of further outbreaks, or it may clear for a time only to reappear later as patches of plaque psoriasis.

Inverse psoriasis

- More common in older adults than children and in obese or overweight individuals. The scaling is greatly reduced or absent.
- Inverse psoriasis is characterized by inflamed, bright red, smooth patches of skin.
- Most commonly affected areas include the axillae, groin, gluteal cleft, submammary folds, vulva and other body folds.
- In obese/overweight patients, there may be symptoms under the belly (where it folds over).
- The surface has a glaze hue and fissuring at the depth of the fold, which is a common presentation.
- If the skin rubs together in the folds, symptoms will be aggravated and sweating in the skin folds may also aggravate the symptoms.

Pustular psoriasis: Primarily seen in adults, pustular psoriasis is characterized by white blisters of noninfectious pus (consisting of white blood cells) surrounded by red skin. It generally develops quickly. Pustular psoriasis may be localized to certain areas of the body, such as the hands and feet, or covering most of the body. It begins with the reddening of the skin followed by formation of pustules and scaling. Pustular psoriasis may be triggered by internal medications, irritating topical agents, over exposure to UV light, pregnancy, systemic steroids, infections, stress and sudden withdrawal of systemic medications or potent topical steroids. Pustular psoriasis can be grouped into two categories, localized and generalized.

Acute generalized pustular psoriasis is characterized by following-

- The skin becomes dry and tender.
- The warning signs, not always present are followed by an abrupt onset of high fever with severe malaise.
- Pre existing lesions become fiery and developed pin point pustules.
- Any variety of pustular exanthemata may occur including isolated pustules, lakes of pus, circinate lesions, plaques of erythema, or a generalized erythroderma.

- The nails become thickened or separated by subungual lakes of pus.
- Relapse is common.

Complication of acute generalized pustular psoriasis include

- Hypoalbuminaemia
- Hypocalcaemia
- Oligaemia and foetal tubular necrosis.
- Deep vein thrombosis.
- Secondary infection by staphylococcus
- Inflammatory polyarthritis.
- Malabsorption in acute episode.
- Gross hair loss in chronic cases.
- Death may occur in acute stage in the absence of effective treatment.

Erythrodermic psoriasis

- Erythrodermic psoriasis is particularly an inflammatory form of psoriasis that often affects most of the body surface. It generally appears in the patients having unstable plaque psoriasis, where lesions are not clearly defined.
- It is characterized by periodic, widespread, fiery redness of the skin and the shedding of scales in sheets, rather than smaller flakes.
- There is widespread exfoliation (shedding of skin).
- The erythema (reddening) and exfoliation (shedding) of the skin are often accompanied by severe itching and pain.
- Erythrodermic psoriasis "throws off" the body chemistry, causing protein and fluid loss that can lead to severe illness like dehydration, malnutrition as well as heart failure.
- Edema (swelling from fluid retention), especially around the ankles, may also develop along with infection.
- The regulation of body temperature is often disrupted, producing shivering episodes.
- Infection, pneumonia and congestive heart failure brought on by erythrodermic psoriasis can be life threatening. People with severe cases of this condition are often hospitalized.
- Known triggers of erythrodermic psoriasis include the abrupt withdrawal of a systemic psoriasis treatment including corticosteroids, allergic reaction to a drug resulting in the Koebner response, severe sunburns, infection, medications such as lithium, anti-malarial drugs and strong coal tar products.

Complication of psoriasis

Complication of psoriasis include following

- Infections
- Eczematization
- Pustulization
- Itching

- Burning and Tightness
- Hypocalcaemia.
- Amyloidosis
- Arthritis
- Hepatic and Renal failure
- Tumour formation

Co-morbidities: Traditionally psoriasis has been considered a disease of the skin, but multiple reports attest to the important role of systemic inflammation with ramifications for other organ systems. Many studies suggest that patients of psoriasis tend to have concurrent illnesses (behavioral and systemic), termed as co-morbidities, which include psoriatic arthritis, cardiovascular disease, nonalcoholic fatty liver disease, inflammatory bowel disease, lymphoma, skin cancer, anxiety and depression [35]. These co-morbid conditions may occur concurrently or years after development of psoriasis [36,37]. Most common co-morbidity is psoriatic arthritis (PSA). Psoriatic arthritis is of five types [38] which is as follows-

1. Symmetric arthritis: Affects about 15% of PSA patients, involves multiple symmetric pairs of joints in the hands and feet and resembles rheumatoid arthritis.
2. Asymmetric arthritis: The most common type of PSA, found in about 80% of patients. Usually involves only 1-3 joints in an asymmetric pattern and may affect any joint (e.g. knee, hip, ankle, and wrist). Hands and feet may have enlarged "sausage" digits.
3. Distal interphalangeal predominant: This "classic type" occurs in only about 5% of PSA patients. Primarily involves distal joints of the fingers and toes. It is sometimes confused with osteoarthritis, but nail changes are common.
4. Spondylitis: Inflammation of the spinal column causing a stiff neck and pain in the lower back and sacroiliac area. Peripheral disease may be seen in the hands, arms, hips, legs and feet.
5. Arthritis mutilans: A severe, deforming type of PSA affecting <5% of patients with PSA. Usually affects a few joints in the hands and feet and is associated with pustular psoriasis.

The relationship between psoriasis and associated diseases has drawn particular interest in recent years. The awareness of co-morbidities associated with psoriasis has led to a paradigm shift in the understanding of the disease and its management.

Psychosocial impact: Psoriasis has a negative impact on physical, emotional, social, sexual, and financial well-being. Psychological stress occupies a special place among the factors that trigger psoriasis. In general, psychological stress has been frequently described as a variable that

triggers skin disease, and has been commonly associated with high levels of sympathetic activation and difficulties in regulating emotions [39-41].

In some studies Million compared the personality with the functioning of immune system and found that the personality can be studied as an interface between the outer and inner world, and between the social and biological levels. Personality would be a complex behavioural system that evolved due to the need to deal with a threatening environment undergoing constant change [42]. Million also suggested that the different ways of dealing with the environment may be more or less adoptive. These studies are supported by Quiroga *et al.* [43]. Psoriasis patients experience reduced quality of life usually due to social rejection as compared to healthy subjects. These patients also experience greater physical discomfort, mood swings, poor body image and self image, and restricted daily and social activities due to visibility of their lesions.

Therapeutics in psoriasis: Choice of treatment for psoriasis depends on many factors, including the extent of disease, its effect on quality of life, and the patient's perception of their illness. Conventional treatment of psoriasis is based on the degree of severity. There can be substantial variation between individuals in the effectiveness of specific psoriasis treatments. Medications with the least potential for adverse reactions are preferentially employed. If the treatment goal is not achieved then therapies with greater potential toxicity may be used. Medications with significant toxicity are reserved for severe unresponsive psoriasis. This is termed as 'psoriasis treatment ladder'. As a first step topical treatment is employed. If topical treatment fails to achieve the desired goal then the next step would be to expose the skin to ultraviolet (UV) radiation. This type of treatment is called phototherapy. The third step involves the use of systemic medications. Over time, psoriasis can become resistant to a specific therapy. Therefore, treatments may be periodically changed to prevent resistance and to reduce the chance of adverse reactions, this type of strategy is termed as treatment rotation.

Rotational and combination therapies increase efficacy and decrease toxicity of treatment. The future may bring stem-cell therapy and gene-based therapies, including "antisense" treatments that directly inhibit psoriasis specific genes. However, the adverse effects and toxicity of conventional psoriasis treatments necessitate safer and effective natural treatments that can be used as alternatives or in an integrative fashion.

Natural treatment: A study on five cases of psoriasis over a six month period validated the beneficial role of diet which is rich in fresh fruits and vegetables, small amounts of protein from fish and fowl, fiber supplements, olive oil and herbal teas when assessed by PASI (psoriasis area and severity index) score and PSS (psoriasis severity scale) score [44]. Some studies suggested the beneficial role of 'Gluten' free diet in different dermatological disorders including psoriasis [45-47].

Ayurvedic system of medicine propounds a separate concept of medicinal dietary supplements in the context of *Rasayana* (rejuvenation by nutraceutical action) in addition to food and diet. *Rasayanas* can be used as nutritional supplement as well as medicine depending upon its various types. The important *Rasayanas* which are part of our daily routine diet include *Rasona* (*Allium sativum* linn.), *Haridra* (*Curcuma longa* linn.) and *Pippali* (*Piper longum* Linn.). Use of some *Rasayana* like *Amalaki* (*Emblica officinalis* Gaertn) and *Haritaki* (*Terminalia chebula* Retz.) should be promoted as a part of our routine diet to impede dermatological disorders including psoriasis [48].

Severe psoriasis has been associated with nutritional deficiencies because of an accelerated loss of nutrients from the hyper-proliferation and desquamation of the epidermal layer of skin. A study carried out on 50 hospitalized patients with psoriasis, demonstrated that 18% had decreased total protein, 16% had decreased serum albumin, 38% had elevated mean corpuscular volume and 39% had decreased haematocrit [49-50]. Therefore, it results in increase demand of nutrients. Thus, in this state, patient should be recommend the diet like seeds, nuts, grains, vegetables and fruits, with emphasis on raw seeds and nuts and plenty of organically grown raw vegetables and fruits with special recommendation of dietary supplements rich in antioxidants specially by promoting the use of *Rasayanas*. These food types are easily digestible and can be assimilated faster and quicker in the human system. Some *Rasayanas* like *Pippali* etc. are good bio-availability enhancers and thus enhance the bio-availability of different nutrients from diet [51].

Some study suggested that psoriasis (an inflammatory disorder), should benefit from an anti-inflammatory diet, elimination and or rotation of allergenic foods, and therapeutic fasting [52].

It has been established that patients with disseminated psoriasis have significantly decreased serum levels of the biologically active form of vitamin D, 1-alpha, 25-dihydroxyvitamin D3 (1-

$\alpha,25(\text{OH})_2\text{D}_3$; calcitriol) compared to age and sex matched controls and also compared to patients with moderate psoriasis. Some studies suggested anti-proliferation, and immune-regulating properties of calcitriol (the active form of vitamin D) and its analogs that may inhibit the growth and maturation of keratinocytes on oral supplementation [53-54].

Lifestyle modification: Ayurvedic system of medicine places great emphasis on the impact of a positive life style on health preservation and the role of a negative life style in the promotion of disease. It is well acknowledged that positive and negative impact of lifestyle related factors like discipline of food intake, activity level, sleep, surrounding environment etc. play a major role in health and disease respectively. Lifestyle as well as life style modifications as described in Ayurveda include the application of *Achara Rasayana* (non-pharmacological rejuvenative measures), *Dincharya* (daily regimen), *Ritucharya* (seasonal regimen), *Sadvratta* (code of good conducts) and others [55].

There are some lifestyle related factors which influence the psychosomatic health of an individual, which are economic and social status, social support networks, education and literacy, surrounding social environment, family environment, sanitation, culture etc. [56]. Therefore, modification of these lifestyles related factors are very important for the prevention and the management of dermatological disorders including psoriasis especially by using natural principles of Ayurveda.

CONCLUSION

Psoriasis is a common, chronic, inflammatory, multifactorial disease with predominantly skin and joint manifestations affecting 2.5 % of world population. Initially it is considered as a disorder of keratinization but recent studies favor the role of immunological, genetic and environmental factors. Several studies suggest a strong relation between skin and mind (psyche). As a result, more than a cosmetic nuisance, psoriasis is associated with psychosocial effects that seriously affect quality of life and social relations. The impact of psoriasis on the physical, social, psychological, and financial aspects of life should not be trivialized and must be considered with the same importance as other chronic conditions. Advances in understanding the cellular immunology and biology of psoriasis, when coupled with the biotechnology revolution and rapid advances derived from human genetic studies of auto-immunity, have enhanced insights into the etio-pathogenesis and treatment of

psoriasis. Treatment of psoriasis remains challenging and no definite treatment is available till date. Ayurvedic system of medicine offers natural and cost effective way of management for a wide range of dermatological disorders including psoriasis which specially includes the use of wholesome diet enriched with *Rasayanas* and lifestyle modification in daily life. Use of *Rasayanas* (pharmacological as well as non-

pharmacological) and lifestyle modification as described in Ayurveda promote psychosomatic health and thus help in the management of psoriasis. If contemporary advance technologies for understanding the cellular immunology, biology and molecular genetics of disease coupled with natural management strategies available in Ayurveda, then it will open new vistas in the management of psoriasis.

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