



UNANI AND WESTERN PERSPECTIVE OF DA'- US- SADAF (PSORIASIS): A REVIEW

Suhail Akhtar¹, Taufiq Ahmad*², Naseem Ahmad³ and Mohd Zubair²

¹Medical Officer, Central Government Health Scheme, New Delhi.

²Associate Professor, Mohammadia Tibbiya College Malegaon, Nashik, Maharashtra.

³Assistant Professor, Mohammadia Tibbiya College Malegaon, Nashik, Maharashtra.

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*Corresponding Author

Taufiq Ahmad

Associate Professor,
Mohammadia Tibbiya
College Malegaon, Nashik,
Maharashtra.

ABSTRACT

Da'- us- Sadaf (Psoriasis) is a chronic, non communicable, painful, disfiguring and disabling disease for which there is no cure and with great negative impact on patient's quality of life. Social exclusion, discrimination and stigma are psychologically devastating for individuals suffering from psoriasis and their families. It can occur at any age, and it is most common in the age group of 50 to 69 years. The reported prevalence of psoriasis in countries ranges between 0.09% and 11.4%, making psoriasis a serious global problem. The aetiology of psoriasis remains unclear, although there is evidence for genetic predisposition. The role of the immune system in psoriasis causation is

also a major topic of research. Although there is a suggestion that psoriasis could be an autoimmune disease, no auto antigen that could be responsible has been defined yet. Its treatment is still based on controlling the symptoms. Topical and systemic therapies as well as phototherapy are available. The need for treatment is usually lifelong and is aimed at remission. So far, there is no therapy that would give hope for a complete cure of this problem. In Unani system of medicine, Psoriasis is described under the various heading such as *Da'- us- Sadaf Taqashshur-e- Jild*, *Qooba-e-Mutaqashsherah*, *Chambal*, *Apras*, *Talaq*, *Sa'af-e-Qishri* and *Al-Sadfiah*. It is caused by deposition of *Khilte ghaleez* (Abnormal Sauda) in skin, and treated by various modes of treatments. Ibn Zohr and Majoosi clearly explained the pathogenesis of *Da'- us- Sadaf* on the basis of Humoral theory. According to them, excessive amount of *Khilte ghaleez* (Abnormal Sauda) gets accumulated in the skin and interrupt its nutrition and function, resulting the skin becomes dead and fallout in the form of scales. Unani physicians are successfully treating this ailment since ancient times by adopting

various mode of treatment like evacuation of *Khilte Ghaleez* (Abnormal sauda), this may be done through *Irsal-e-Alaq* (Leeching), *Hijama* (Cupping) *Fasd* (Venesection), *Tarīque* (Sweating) *Munzij* and *Mushil* therapy. The Paper aims to explore the each and every aspect of this disease described in Unani and Conventional medicine.

KEYWORDS: Unani Medicine, *Da'-us-Sadaf*, Psoriasis, *Taqashur-e- Jild*, Conventional Medicine.

INTRODUCTION

Over the years the effective treatment of psoriasis has eluded the researchers as such as its etiopathogenesis. Recently the advent of modern investigative procedures has thrown more light on the pathophysiology of this chronic relapsing papulosquamous disease. Earlier it was thought to be a disorder of hyperproliferation of keratinocytes but psoriasis is purely an autoimmune disorder in which there is T-cell mediated inflammatory disorder. On the basis of this a lot of therapeutic agents are evolved with the help of modern science and technology. These agents may be psoralen ultra violet therapy (PUVA), corticosteroid, coal tar preparations and immunosuppressive agents.

Now a days for extensive, more chronic and persistent psoriasis the haemodialysis is also a trend in few therapeutic centers. But all these therapeutic measures have an extensive as well as exhaustive procedures and also hazardous at greater extent. Therefore evolving a new and effective therapy is always a challenge before clinicians as well as for pharmacologists. In unani system of medicine where the pathogenesis is defined on the basis of humoral theory there is involvement of several humours to develop the psoriatic lesions but ultimately there is predominance of melancholic humour may be derived either by bilious humour, phlegmatic humour, sanguinous humour and by melancholic humour itself. Ultimately there is typical presentation mimicking with the features already described for such ailments in classical unani literatures. That is extremely dryness with hyperkeratotic scaling lesions. Usually there is hyperproliferative conditions in melancholic diseases and all uncontrolled mitotic changes are mend for abnormal melancholic temperament (*Soo-e-Mizaj Saudavi*), such as various malignancies, auto immune disorders, hyperplastic lesions etc. In psoriasis there are rapid keratinocytes turnover due to mitotic changes in basal layer of dermis, which is somewhat similar to hyperplastic lesions. The psoriasis is one of the disease that can be categorised under the melancholic predominant pathological conditions. On the same speculated guidelines the treatment can also be carried out. Unani drug acts in this regard by

expelling out the rotten abnormal humour, thereby correcting the normal physiology of cells, tissues, organs and systems etc. Therefore the drugs are also effective in relieving the symptoms, presentations, as well as relapse at larger extent. The drugs are used in either form, (oral and systemic) since time immemorial on human beings in various form and formulations.

Definition

Da' - us- Sadaf is an Arabic term, which is composed of two separate words, Da' - us- Sadaf Da' having meaning of disease and *Sadaf* means Pearl. Because the scales, peeling out from the lesions, look like the pearl, so it is called Da' - us- *Sadaf*.

Psoriasis is derived from Greek word "psora" meaning, scales. Because the scales are the pathognomic feature of psoriasis so it is called Psoriasis.^[14,64,85]

In fact Da' - us- Sadaf has not been described in any ancient Unani book with this name, while other skin diseases having similar properties have been mentioned. Therefore eminent Unani physicians have tried to correlate the psoriasis with their signs and symptoms and described it in their books with different names as mentioned above.

ZakariaRazi has described it in his book *Al-Hawi-Fil-Tib* as a roughness in skin with itching.^[2] Ibn-e-Hubal mentioned that Da' - us- Sadaf is similar to *Sa'afa-e-Yabisah*, in which skin becomes dry, rough and wrinkled, from which red fluid oozing out and the lesion is covered with scales.^[39] Majoosi characterized it as peeling of scales from the skin.^[46] Unani TabeebRoofas described a condition of *Talaq*, in which the lesion is surmounted by white scales resembling to Abrak.^[84]

Ibn-e-Zohr, defined Da' - us- Sadaf as the disease of skin, in which patient feels severe itching over the lesion.^[42] Ibn-Al-Quf described Da' - us- Sadaf in his book, *Kitab-Al-Umdah Fi-Al-Jarahat*, as the disease of skin, in which white impetigo appears with formation of scales resembling the scales of fish.^[43] Akbar Arzani explained the psoriasis as a disease of skin, in which the skin becomes dry, rough, thick, and scales cover the affected parts of the skin.^[8]

However, in modern system of medicine, psoriasis is defined as a papulosquamous and inflammatory disorder characterized by sharply demarcated plaques of various shape and size, with characteristic silvery lustrous scaling.^[16,25,53,62,6,90]

Some pathologists characterize psoriasis as an inflammatory dermatoses that exhibit epidermal hyperplasia with elongation of rete ridges and club shaped dermal papillae.^[59,91] Psoriasis, in other words, is a chronic inflammatory cell-mediated disease affecting skin and joints, histologically characterized by epidermal hyperplasia, abnormal differentiation of keratinocytes, angiogenesis and presence of neutrophils.^[20,35,56,86]

Synonyms

Qooba-e-Mutaqashsherah^[41,44,48]

Taqashshur-e-Jild^[2,8,39,42,46,48]

Qooba-e-Muzmin^[44,48]

Qashf-e-Jild^[8,48]

Sa'af-e-Qishri^[43]

Da' - us- Sadaf^[30,45,87]

Al-Sadfiah^[45,78]

Chambal^[30,45,63,87]

Talaq^[84]

Apras^[45,78]

Prevalence

Psoriasis is a chronic inflammatory disease characterized by a great variation in prevalence within and between the countries because of geographical, environmental and genetic factors.^[28,61] It is a worldwide disease affecting about 1 - 2 or 1.5 and 3% of population.^[7,12,17,14,26,28,29,62,71,77,88] Psoriasis seems to be most prevalent in Caucasian population, less frequent in yellow brown individuals and in black population.^[26] The prevalence of psoriasis in Southern Europe is lower than that in Northern Europe. In Northern America the prevalence is ranging between 0.5 – 2% in Caucasians, where as it is 0.97% in South America.^[24] In contrast to Europe and US, a low prevalence of psoriasis has been found in Asia, West Africa and in Northern American blacks. The incidence of disease is low in Japanese and Eskimos. Psoriasis is nearly absent in North American Indians, and in examination of 26000 South American Indians, not a single case was seen.^[28] In a large study conducted in Australia, the estimated prevalence was 2.3 %, however the percentage was higher in south than in North.^[21] Although it can appear just after birth or in old age as having its onset at the age of 103 years, but it most commonly involves adults in the second and fourth decade of life.^[28,54,88] The mean age of onset was about 28 years in one study in the United States.^[26,88] Other Indian studies have reported the highest incidence to be in second decade or in the reproductive age group.^[54,8] A study of the onset of psoriasis in 2400 patients showed a peak incidence at 22.5 years of age, a second peak of onset around age 55 years was found in 11.5% of the patients.^[28,37] A North Indian study found that the mean age of onset was higher for males than females that are 29 years in males and 27 years in females.^[28]

There is high concordance between monozygotic twins than dizygotic twins for the disease that indicates inherited factor.^[3,28] According to studies by Lomholt in Faeroe island, the probability of a child having the disease if one parent has psoriasis is about 25%, where as it is 60 – 70% if both parents have psoriasis.^[3,14] The HLA CW6 antigen was significantly more frequent in patients who manifested the disease before the age of 40, while in patients with a later onset the HLA CW2 and HLA-B27 are found more frequently.^[14,34] Psoriasis attacks are more common in winter season than summer, but its prevalence is more in tropical areas and temperate climate. Several studies showed no sex predisposition to the disease, though the women develop the disease earlier than the men.^[3,60,62,71]

Etiology

The exact etiology is still unknown, however it is considered as multifactorial inflammatory disease with wide variety of triggering factors which involved in causation of psoriasis, just like Humours, Genetic, Environmental, Anxiety, Mechanical damage (Koebner phenomenon) Ultra violet rays, Bacterial and Viral infections, Smoke and Drugs.^[7,12,29,51,57,68,71,77]

Humours

Sauda-e-GhairTabai (abnormal black bile)^[4,5,6,40,42,48] *MuhtaraqKhilt-e-Sauda*^[1,48] *Khilt-e-Hareefwa Laza*^[84] *Balgham-e-shor*^[40] *Zojaji Balgham*⁴³ *Khushkboraqi Madda*^[39] and *Mirrah Sauda*.

Emotional stress

Stress may be one of the most important precipitating factors capable of triggering psoriasis (Seville RH), though its mechanism is difficult to understand but it has been known that cytokines such as IL-1, IL-2, TNF- α and IFN- γ all have the potential to stimulate the hypothalamic Pituitary –adrenal axis resulting in the release of corticotrophin releasing factor, Adrenocorticotrophic hormone and corticosteroids.^[16,69,72] This system provides a negative feedback mechanism that can counteract an otherwise exaggerated immune response but stress can perturb this normal response and responsible for exacerbation of psoriasis. According to Christopher and Mrowietz, psoriasis is made worse by stress in approximately in 30- 40 percent cases.^[18, 26,28,32,52]

Trauma

Psoriatic lesion may develop at the site of injury to skin, the induction of lesion by such injury is known as Koebner phenomenon or Isomorphic phenomenon.^[30,88,90] The injury may

be of various types Physical, Chemical, Mechanical and Allergic.^[88] In 1872, Koebner^[50] described a patient who developed psoriatic lesion after a horse bite on the arm. It is plausible that the traumatized keratinocytes may play a role in the development of this phenomenon by secreting cytokines basic fibroblast growth factor (b-FGF), which is essential to induce both the proliferation of keratinocytes and the endothelial cells.^[50,79] Psoriatic patches usually occur 1-2 weeks after cutaneous injury, but may be as short as 3 days or as long as 2 years depending on the degree of sensitivity of patient's skin.^[28,88]

Infections

It is well documented that upper respiratory tract infection and tonsillitis when caused by streptococci may cause a flare up of existing psoriasis or may precipitate an attack of acute guttate psoriasis.^[3,51,88] According to Rosenberg and Noan, the presence of microbial products in the skin could be a pre-requisite for the development of psoriatic lesion in genetically susceptible individual.^[88] Weisensealet *al.* demonstrated that group A streptococcal infection was found only in type 1 psoriasis, in HLA Cw6 allele bearing patients. The incidence of positive streptococcal parameter in this group was higher than individual with no HLA Cw6 allele.^[93] Therefore, it indicates that the presence of HLA Cw6 significantly influences the susceptibility of a distinct immune response pattern to streptococcal antigen and the possibility of the disease.

Hereditary

Genetic factors play a key role in predisposition of psoriasis. As the recent studies have proved that there is deep relation between inheritance and psoriasis. Therefore, it was found that when one parent had psoriasis, the disease developed in 8.1 percent of the offspring, this value increased to 41 percent when both the parents had psoriasis and in monozygotic twins the occurrence rate is higher (65%) than dizygotic twins (30%).^[3,28,29,66] The main factor responsible for that inheritance is human leukocyte antigen (HLA) that is located on chromosome 6. The HLA types, more frequently reported to be associated with psoriasis, are HLA-B13, HLA-Bw57, HLA Cw6 and HLA DR7 and HLA associated with early onset of disease is HLA Cw6 which was positive in 85 % patients with early onset disease. HLA-B27 is related to generalized pustular psoriasis.^[14,28,88,90] Unani physician Ibn-e-Rushd has described the inheritance as a factor in the development of disease, which is produced by genetic basis temperamental imbalance of humour. Ibn-e-Rushd explained that abnormal

black bile included in the blood and provide nutrition to the body organs, which results in different types of disease such as fever and inflammatory disorder.^[40]

Diets

Among Unani physicians Ibn-e- Rushd has described that some humoural diseases are caused by intake of cold and dry food. Excessive use of spices and salty food may also be responsible for the disease.^[40,47]

Drugs

Many drugs can precipitate and exacerbate the psoriasis by reducing the cyclic AMP level and via chemical interaction with genetically susceptible individual. Alteration of polymorphonuclear leucocyte chemotaxis, increased or decreased synthesis of arachidonic acid metabolites, modifications of cyclic nucleotide system and lymphokine production are probably involved in this phenomenon. For example β blockers.^[3,7,15,17,28,33,61,65,77,88,89,92] Propranolol, Practolol, Metoprolol), Lithium.^[3,28,33,61,83,65,92] Anti depressive agent^[17,88] (Trazodone), Hypolipidaemicagent^[17] (Gimfibrozit), Immunesuppressive agent^[88] (Cyclosporin) ACE inhibitor^[28,65] (Enalapril, Captopril), Anti-Malarial^[28,33,65,88] (Chloroquine), NSAID^[3,65,66,70] (Indomethacin, Salicylates, Maclofenamate, Phenylbutazone, Oxyphenylbutazone, and Ibuprofen. Others^[3,33,88] (Clonidine, Potassiumiodide, Amioderone, Digoxin, Penicillin, Terfenamide). Sudden withdrawal of corticosteroid therapy in psoriasis may result in precipitation of generalized pustular psoriasis (GPP) as a rebound phenomenon, occasionally more potent topical steroids also cause such precipitation.^[33,88]

Alcohol

Alcoholism may aggravate the disease, because alcohol is thought to act as suppressing the cell mediated immunity, and alcoholic patients show indeed more severe, extensive and inflamed disease.^[17,38,45,62,63]

Seasonal Variation

Most of the patients experience worsening of the disease during winter season. While high humidity is usually beneficial for the lesions.^[88]

Sun Light

Some person gets relief from sun light, while those who are sensitive to sun light or ultra violet rays, experience worsening of the condition.^[13,17,77]

Endocrinal factors

Psoriasis may subside during pregnancy while generalized pustular psoriasis may be precipitated due to increased level of progesterone in later half of pregnancy.^[17,77]

Smoking

Smoking is thought to be a triggering factor in the onset of the disease, particularly in palmopustular psoriasis. The proposed mechanism of action for smoking include morphological and functional alteration of polymorphonuclear cells, and their skin migration and oxidative tissue damage.^[3,38,51]

Pathogenesis

Psoriasis has not been mentioned with this name in old Unani books, but it has been described as *Taqasshur-e-jild*, which indicates the disease, in which scales peel out from the skin. Therefore no any defined and clear pathology has been established till now. However, Unani physicians revealed the pathogenesis of the disease having similar properties. According to Ibn-e-Zohar excessive amount of morbid melancholic humour (*Khilt-e-sauda*) is accumulated in the skin, which leads to malfunctioning of skin and it becomes unable to take proper nutrition and to remove morbid melancholic humour (*Khilt-e-sauda*). As a result of that, skin tissues become dead and fallout in the form of scales.^[42] Ali Ibn-e-Abbas Almajoosi has described that *Tabiyat* expels the *khilt-e-Ghaleez* towards skin from internal organs resulting in the dryness and itching of the skin. In this condition skin is unable to remove *Khilt-e-Ghaleez* leading to accumulation of *sauda* in skin. But according to Modern concept Pathogenesis of psoriasis is still not fully understood, however, it represents excessive but controlled proliferation and differentiation, inflammation and immune dysregulation. Because a characteristic feature of involved skin is hyperproliferation. Currently there is evidence of more than eight fold shortening of the epidermal cell cycle (36h versus 311h for normal) in involved skin of patients with psoriasis.^[56, 90] Further there is a two-fold increase in the proliferative cell population, and 100% germinative cells of the epidermis appear to enter the growth fraction compared with 60 – 70% for normal subjects. These alteration results in hyperplastic epidermis generating 35000 cells/mm² per day from a proliferative compartment containing approximately 52000 cells /mm² of skin surface. While the normal skin produces only 1218 cells /mm² per day from a proliferative compartment of 26000 cells/ mm². There was a great acceleration of the transit time of cells from the basal layer to the upper most row of the squamous cell layer, from approximately 53 days in

normal epidermis to only seven days in the epidermis of active psoriatic lesion.^[14,23,28,56,64,75,90]

Immunopathology

It has been shown that T lymphocytes predominate in the inflammatory infiltrate. They are mainly CD4⁺ lymphocytes, and they are in psoriatic lesion known to be in an activated state and express HLA-DR and interleukin-2 receptors. The activation of T lymphocytes may be due to bacterial super antigen such as streptococcal enterotoxin B.^[23] Activated CD4⁺ T lymphocytes produce a variety of cytokines including interleukin-2 (IL-2), tumour necrosis factor- α (TNF- α) and gamma interferon (γ -IFN).^[23,64,75] γ -IFN is thought to play important role in the initiation of psoriatic lesion as demonstrated by the induction of pinpoint lesion of psoriasis at the site of γ -IFN injection in previously uninvolved skin.^[23,27] γ -IFN induces the expression of intercellular adhesion molecule-1 (ICAM-1) in keratinocytes and endothelial cells. This molecule mediates the adhesion and trafficking of lymphocytes into the epidermis by binding to its ligand LFA-1 (Lymphocyte function associate antigen-1) expressed on lymphocyte membrane. γ -IFN inducible protein is detected in epidermis during cellular immune responses and may have chemotactic as well as mitogenic properties. Keratinocytes from lesional psoriatic skin show altered response to γ -IFN, and not responsive to the growth inhibitor effects of γ -IFN. These findings suggest that decreased responsiveness of keratinocytes to γ -IFN may contribute to the hyperproliferation and altered differentiation of epidermal cells in psoriasis.^[23,88] Treatment that specifically targeted on T lymphocytes such as Anti-CD3, Anti CD4 and CD25 have confirmed the relevance of T lymphocytes in the pathogenesis of psoriasis.^[31,49,61,74,90]

Clinical features

In all the cases of psoriasis, certain features should be examined carefully to look for characteristic changes that permit the proper diagnosis, because the psoriatic lesion is characterized by its particular morphology and the site of predilection.

Morphology of psoriatic lesion

Each psoriatic lesion starts as a papule and extends peripherally to form nummular and discoid plaques. Many such discoid lesions coalesce to form large plaque.^[88] There may be many numbers of lesions or only a single one, and, when multiple, may be symmetrically distributed. So the size of single lesion varies from a pin-point to plaque,^[29,87,88] which is oval

and irregular in shape with well-defined and clear-cut borders. This is important for diagnosis in flexural and glans psoriasis, when other features are absent.^[17,28] The colour is full rich red, and often referred to as salmon pink, not normally seen in eczema, seborrhoeic dermatitis or lichen simplex. This quality of colour is of special diagnostic value in lesions on the palms, soles, and scalp. On the legs a bluish tint is often present.^[17] The scales are abundant, loose, dry and silvery white or micaceous. It may be waxy yellow in rupoid form and less in flexural and glans psoriasis.^[17,30,62,71,83,87,88]

Site of predilection

Although no region is exempted from involvement, the areas most commonly affected by psoriasis are the pressure points e.g. elbows, knees, scalp (from where it may spill on to the forehead and nape of neck), extensor surface, lumbosacral area and back. This common localization has been attributed to Koebner or Isomorphic phenomenon.^[53,58,62,71,88]

Signs

- 1) **Candle grease sign:** When a psoriatic lesion is scratched with the help of glass slide, candle grease like scales are produced, which is also known as ‘Signe de la tache de bougie’.^[12,14,50,88]
- 2) **Grattage test:** Gentle scraping the lesion with a glass slide produces the silvery scales, and then grattage test is positive.^[68,82]
- 3) **Membrane of bulkeley:** When the scales are completely scraped off, the stratum mucosum (basement membrane) is exposed and a moist red surface is seen, which is known as Membrane of Bulkeley.^[77,82,88,90]
- 4) **Auspitz sign:** On deep scraping, the capillaries at the tip of elongated papillae are torn leading to multiple bleeding points. This is characteristic of psoriasis and known as Auspitz sign.^[17,28,29,30,33,58,77,82,88,90]
- 5) **Holo or Woronoff sign:** A zone of hypopigmentation seen around the plaque which is evident after treatment with ultra violet radiation or topical steroid due to deficiency of prostaglandin E.^[17,58,77,82,88,90]
- 6) **Koebner’s or Isomorphic phenomenon:** Development of isomorphic lesion at the site of local trauma of uninvolved skin, the lesion develops 7 to 14 days after the injury. The injury to skin should involve both epidermis and dermis to induce koebner’s phenomenon.^[28,29,33,53,58,62,76,77,82,88,90]

Nail changes in psoriasis

1. Pitting of nail plate: Pitting of the nail plate is the most common finding, which results from focal psoriasis in that part of nail matrix, leaving a small pit like deformity.^[17,28,29,53,62,71,77,88]

2. Oil drop sign: There are subungual patches of psoriasis of a few millimeters in size, which because of their yellow color look like oil spots in the nail.^[14,17,28,71,77]

Classification

The analytical studies have shown that there is strict correlation between early manifestation and HLA-CW6 antigen, so the non-pustular psoriasis initially can be defined into two as follows.^[14]

1. Type I Psoriasis (Early onset psoriasis)

2. Type II Psoriasis (Late onset psoriasis)

Type I Psoriasis – It is related to early age of life, which occurs before the age of 40 years and associated with the HLA-CW6 antigen, there is an increased familial occurrence in it.^[13,14,76]

Type II Psoriasis – It occurs after the age of 40 years and there is no relation with HLA-CW6 antigen.^[13,14,76]

Psoriasis further can be classified clinically as below^[7,12,14,15,17,28,29,33,53, 62,71,77,88]

1) Guttate psoriasis (2) Chronic plaque psoriasis (3) Psoriatic erythroderma (Exfoliative psoriasis) (4) Pustular psoriasis (5) Mucous membrane psoriasis (6) Arthropathic psoriasis (7) Regional psoriasis.

1) Guttate psoriasis: This form is characteristic of psoriasis of an early age of onset and as such is found frequently in young adults, the lesion is pinhead to pea sized (0.5-1.5cms in diameter). These rain drops like erythematous papules erupt abruptly and distributed bilaterally symmetrically all over the body, especially on the trunk and upper extremities, sparing the palms and soles. Streptococcal throat infection may frequently precede the onset or flare up of the disease by 1-2 weeks and other predisposing factors are aggressive local therapy or withdrawal of systemic glucocorticoids. Throat swab should be taken to rule out the streptococcal infection and elevated Antistreptolysin O (ASO) titer is usually found in this condition.^[7,11,12,14,15,17,18,28,29,33,53,58,62,71,76,77,81,88,90]

2) Chronic plaque psoriasis / Nummular psoriasis/Psoriasis vulgaris: The commonest type of the disease is the plaque or nummular type that occurs in more than 80% of the patients.^[87] It is characterized by well defined erythematous-squamous plaque, the lesion is round and oval in shape, variable in size as coin size to large palm size, from few to numerous in number and typically affects elbows, knees, scalp, lumbo-sacral area, retro auricular, intergluteal cleft and umbilical area.^[1,15,55,58,68,81,82,90] If palm-sized lesions predominate, it is called as psoriasis geographica, where as if coin-sized lesions predominate, it is known as nummular psoriasis. The lesion can be localized, minimal psoriasis or generalized, involving whole of the body, known as generalized or universal psoriasis.^[14,17,28,33,62,88]

3) Pustular psoriasis: This is the severe clinical variant of psoriasis; characterized by flat, sterile, non-follicular pustules of variable diameter, ranging from 1-5 mm. The pustular psoriasis is divided into two.^[15,57,76,81,88]

1. Generalized pustular psoriasis (Von Zumbush psoriasis)

2. Localized pustular psoriasis (Barber's psoriasis)

Generalized pustular psoriasis (Von Zumbusch)

This is the most severe form of generalized pustular psoriasis (GPP). It is most common in children between 1-5 years of age. The skin lesions start abruptly as multiple erythematous, tender plaques, which soon become pinhead size, tiny sterile pustules. Pustules are erupted over the trunk and extremities. The attacks are accompanied by high-grade fever, leucocytosis, arthralgia, malaise and burning sensation prior to the appearance of erythema and pustules.^[7,14,17,28,59,62,77,81,88] The nail changes are also common, subungual collection of pus is present there. The tongue and buccal mucosa may also be involved. Pustular psoriasis is precipitated by systemic steroids (Braverman et al 1972, Champion, 1959) Iodide, Salicylates, Progesterone (Shelley, 1972) Penicilline (Privat et al, 1969) and Nystatin (Petrozzi, and Witkostein, 1971).^[23,43]

Localized pustular psoriasis (Barber's psoriasis)

Since the lesions usually affect the palms and soles, so it is also called as palmo-plantar pustulosis. It is more common in females. The lesions are erythematous, well defined with many tiny pustules on thenar and hypothenar eminence of the palms, soles and side of heels. The area is scaly, red with tendency to fissure; the pustules may also be hemorrhagic.

Associated arthropathy, especially of the distal interphalangeal type, may occur in some.^[7,14,17,28,57,59,62,76,77,81,88]

Hallo peau continuous acrodermatitis

It is characterized initially by perisubungual pustules. This is usually induced by trauma and localized at thumbs. Sometime it may involve whole fingers and nails. Systemic symptoms are absent in localized pustular psoriasis.^[14,17,28,76,88]

Erythrodermic psoriasis

It is characterized by universal erythema and scaling, involving face, hands, feet, nails, trunk and extremities. It may occur in patients with previous chronic disease or appear as a reaction to non-tolerated topical therapy e.g. Chloroquine or β -adrenergic receptor blockers or as a result of too vigorous light therapy e.g. Ultraviolet B or UVB. All the symptoms of psoriasis are present in this condition but erythema is the most prominent feature. Besides this, some systemic symptoms are also found like hyper or hypothermia, dehydration, hypoproteinaemia, electrolyte imbalance, anaemia, hypocalcaemia, renal and cardiac failure may also occur.^[1,14,17,28,33,62,76,77,81,88,90]

Psoriasis inversus (Flexural psoriasis)

About 2-4 % of patients suffer from inverse form of psoriasis. It affects major skin folds such as axillae, groins, sub mammary folds, vulva, gluteal cleft, periumbilical region, retroauricular area, glans of uncircumcised penis.^[14,17,22,33,81,88,90] It is common in older adults than children. The lesions are well defined, less scaly, smooth, having glazed hue with a few deep painful fissures. The plaques are often confined to the area of skin-to-skin contact. Flexural psoriasis may occur as a primary disorder or as a koebner phenomenon on top of infective or seborrhoeic dermatoses.^[7,11,14,17,33,53,62,71,76,88]

Psoriasis unguis

Nail changes are seen in about 20 – 50% of all the patients of psoriasis, more frequently in psoriasis arthropathica. It is uncommon in children (7 –13%).^[5,16,36,81,88,90] Fingernails are involved more frequently than the toe nails, 50 and 35% respectively, and show typical psoriatic finding than the toe nails.^[10,71] The common changes seen in nails are pitting of nail plate, onycholysis, oil spot, ridging of nail plate, subungual hyperkeratosis and splinter haemorrhage.^[14,17,28,33,53,62,76,88]

Psoriasis arthropathica /Arthritis psoriatica

Psoriatic arthritis is an autoimmune inflammatory disorder, associated with psoriasis with a negative test for rheumatoid factor. It occurs in 5 – 10% of psoriasis patient and its peak occurrence is between 20 – 40 years of age, and rarely occurs in children. Both the sexes are equally affected. It may precede or accompany the skin manifestations. Genetically it is found that HLA B 27, DR3, A26, and B38 haplo type are significantly associated with psoriatic arthritis. [14,28,33,53,62,71,77,81,88,90]

There are five clinical types of psoriatic arthritis

1. Involvement of distal interphalangeal joints of fingers and toes with nail changes (16%).^[29,77]
2. Symmetrical polyarthritis like rheumatoid arthritis.^[29,77]
3. Arthritis mutilans with osteolysis or destruction of bones of hands and feet (5%).^[77]
4. Oligoarthritis with a single or a few interphalangeal or metacarpophalangeal joints (70%).^[77]
5. Ankylosing spondylosis with association of peripheral arthritis.^[77]

Rupoid psoriasis

It is a limpet like cone shaped lesion.^[17]

Elephantine psoriasis

This term is used to describe unusual but very persistent, thickly scaling, large plaques that sometimes occur on the back, limbs, hips or elsewhere.^[17]

Ostraceous psoriasis

It is a ring like hyperkeratotic lesion with a concave surface, resembling an oyster shell.^[17]

Complications

- 1) **Infection:** Secondary infection may occur in psoriatic lesions during topical steroid therapy, particularly staphylococcal infection occurs in 50% of patients if surgical procedure is carried out through psoriatic plaque.^[12,17,76]
- 2) **Itching:** It is more common in unstable psoriasis. The degree of itching reflects the emotional state of the patient and, if severe, may be a symptom of anxiety or depression.^[14,17,76]

3) Arthritis: It rarely occurs with psoriasis and usually involves distal interphalangeal joints.^[14,17,76]

4) Alcoholism: It was found that the male patients, suffering from severe form of psoriasis, had the history of heavy drinking. It may be a symptom of stress caused by severe skin disease.^[12,17,76]

5) Nephritis and Renal failure: Streptococcal infection of throat may rarely spread through blood to the kidney and causes glomerulonephritis. Renal failure due to acute tubular necrosis may rarely result from the oligoemia after loss of albumin into and from the skin in acute pustular psoriasis.^[17,76]

6) Hepatic failure: Severe abnormalities of liver function may occur in erythrodermic or pustular psoriasis, and likely to be related to oligoemia, drugs and alcohol intake.^[17,10,77]

7) Apical pulmonary fibrosis: It is non-articular complication of ankylosing spondylitis, and has been reported in association with a case of psoriatic spondylitis.^[17]

8) Amyloidosis: Amyloidosis is a rare sequel of secondary arthropathic, generalized pustular and severe non-pustular psoriasis.^[17]

Investigations: The following investigations are carried out for the diagnosis of psoriasis.

1) E.S.R. – Usually it is normal in psoriasis but in generalized pustular psoriasis it may be elevated.^[17]

2) T. L. C – Its value is raised in psoriasis.^[17]

3) Serum uric acid – It is elevated in up to 89% patients.^[17,77]

4) Serum calcium – In pustular and erythrodermic psoriasis the calcium is decreased in serum.^[17]

5) Immunoglobulin – It is generally normal but IgA deficiency and monoclonal gammopathy are documented in association with psoriasis.^[77]

6) Anti nuclear antibody – It is found in rheumatic arthritis but negative in psoriatic arthritis.^[77]

7) Throat swab – It is useful in guttate psoriasis.^[13]

8) Nail dipping and Skin scraping – It is carried out to exclude the fungal infection because it is negative in psoriasis. It is also called KOH smear.^[29]

9) Skin biopsy – It is performed to confirm the diagnosis by histopathological examination of psoriasis.^[7,13,17,68]

Diagnostic Points

Diagnosis of the Psoriasis is based on the following points. Essential to this diagnosis is the presence of the stigmata of psoriasis and the patient must have morphologic evidence of psoriasis.

1. Family history of psoriasis.
2. Presence of lesions at particular sites e.g. Elbow, Knee, Scalp, Back and Nails.
3. Lesions covered with silvery scales
4. Candle grease sign, Auspitz sign and Koebner Phenomenon
5. Itching
6. Seasonal variations

Differential Diagnosis

1. Lichen planus: The thickened and violaceous colour of lesion, glistening surface, less scales sticky to the lesions and presence of oral changes are the characteristic features of the disease. The lesions are usually present on the outer surface of wrist and front of calf. Nails pitting are absent.^[7,9,12,13,15,17,25,36,57,76]

2. Pityriasis rosea: It is characterized by appearance of multiple, oval, well defined, erythematous scaly eruptions, disposed along the body cleavages, resembling an inverted christmas tree. The initial lesion a herald patch is the diagnostic hallmark of the disease. The lesions are found on the trunk and run along the ribs.^[7,9,12,13,15,25,36,71,90]

3. Tinea corporis: It starts as erythematous itchy papules, that progress to form a annular or arcuate lesion with relative clearing in the center and studded at periphery with papules and papulovesicles. It particularly affects head, scalp, face, trunk, groin, hands and nails.^[9,13,15,17,76,77,90]

4. Secondary syphilis: The secondary syphilis is usually preceded by history of painless genital sore, cutaneous rashes associated with mucosal lesions, lymphadenopathy and coppery scaly papules on the palms and soles. Serological test for treponemapallidum is confirmatory.^[9,12,76]

5. Lichen simplex: It can resemble psoriasis closely, particularly on the scalp and near the elbow, but intensified skin markings, ill-defined edges and marked itching are characteristic of lichen simplex.^[12,36,90]

6. Reiter's syndrome: This disorder occurs as a sequel to non-specific urethritis in men and, less commonly to bowel infection, and probably results from infection with Mycoplasma organisms. There is usually an accompanying arthritis and spondylitis and occasionally

conjunctivitis. Psoriasiform skin lesions develop on the soles and toes. Inflamed, red, scaling patches may also develop on the glans penis.^[9]

7. Atopic dermatitis: This is a very common, extremely itchy disorder of unknown cause that characteristically involve the face and flexures of infants, children, adolescents, and young adults. The patient is constantly itchy and restless. The itchiness is made worse by change in temperature, by rough clothing and by other minor environmental alterations.^[71]

8. Pityriasis rubra pilaris: The colour of lesion is less distinct, deeply red follicular lesions are present and horny thickening has yellowish tinge.^[7,9,12,13,15,71,76]

9. Candidiasis: It is characterized by less demarcated with frayed edges, less erythematous lesion with satellite pustules at margins.^[12,64,80,90]

10. Seborrheic dermatitis: This is a common eczematous disorder that characteristically occurs in hairy areas, on the flexures and on the central part of the trunk, reddened itchy patches either scaly or exudative and crusted. It usually involves scalp as ‘dandruff’ when severe the eyebrows. Other facial areas may be involved such as the nasolabial folds, the paranasal sites, the external ears and the retroauricular folds.^[7,12,13,17,71,76,77,90]

Management

Complete cure and effective therapy is always a challenge before clinicians. In unani system of medicine where the pathogenesis is defined on the basis of humoural theory there is involvement of several humours to develop the psoriatic lesions but there is predominance of melancholic humour that may be derived either by bilious humour, phlegmatic humour, sanguinous humour and by melancholic humour itself. So there is typical presentation mimicking with the features already described for such melancholic ailments in classical unani literatures. That is extremely dryness with hyperkeratotic scaling lesions. Usually there are hyper proliferative conditions in melancholic diseases and all uncontrolled mitotic changes are mending for abnormal melancholic temperament (*Soo-e-Mizaj Saudavi*), such as various malignancies, auto immune disorders, hyperplastic lesions etc. In psoriasis there are rapid keratinocytes turnover due to mitotic changes in basal layer of dermis, which is somewhat similar to hyperplastic lesions. The psoriasis is one of the disease can be categorised under the melancholic predominant pathological conditions. On the same speculated guidelines the treatment can also be carried out. Our drug acts in this regard by expelling out the rotten abnormal humour, thereby correcting the normal physiology of cells, tissues, organs and systems etc. Therefore the drugs are also effective in relieving the symptoms, presentations, as well as relapse at larger extent. The drugs are used in either

form, (local and systemic) since time immemorial on human beings in various form and formulations.

To achieve the above mentioned objectives the disease can be treated under following three headings (1) *Ilaj bit tadbeer* (Regimen Therapy) (2) *Ilaj bil ghiza*(Diet Therapy) (3) *Ilaj bid dawa*(Drug Therapy).

Ilaj bit tadbeer: Following *tadabeer* should be carried out.

- Hygiene should be maintained.
- Keep the patient away from the stress and anxiety.
- Stop alcohol and Smoking.
- To evacuate the morbid humours following measures should be performed

Fasd (Venesection)

Fasd is one of the oldest classical modes of treatment in unani system of medicine. In *fasd* usually the particular vein is incised and blood is removed, along with the blood the morbid humour, responsible for the disease, is expelled out. So It is used for cleansing and evacuation of morbid humour from the body and indirectly relieving the inflammatory congestion. Hence it is highly effective method in various diseases. The unani physician Muhammad Tabri has mentioned in its book “*Moalajat-e-Buqratiyah*” that in *Taqashshur-e-jild* Basalic vein of both hands should be venesected to remove the morbid humour.^[84]

Taleeque (Leeching)

Taleeque is also a way of treatment using medicinal leech to relieve inflammation, to correct imbalance of the four humours. Leech treatment was very popular during the middle ages. According to the unani system of medicine an imbalance in the proportion of four humours produces diseases. So Ibn-e-Sina has proved leeching beneficial in skin diseases and chronic non healing wound and ulcers. Leech sucks approximately 5 to 10 ml of blood so it also removes the morbid humour responsible for the diseases like venesection.^[40]

Tareeque (Sweating)

Tareeque is a method of *istifragh* and cleansing of body. In which the waste and morbid material are removed through skin. Ibn-e-Abi Usaibah has quoted saying of Hippocratein his book “*Uyoon-ul-Anba fi Tabaqaat al Atibba*” that part between epidermis and dermis should be treated through sweating and diaphoresis. Tareeque can be carried out by three ways.(1)

Hammam(Bathing) (2) Inkibab(Vapour bath) (3) Aabzan (Sitz bath).These three methods produce hot and humid climate that dissolve the morbid melancholic humour beneath the skin and due to hot condition the pores of skin dilate and the dissolved morbid material comes out and improve the condition.^[94]

Ilaj bil Ghiza (Diet Therapy)

- Use easily digestible diet.^[39]
- Avoid high iodine diet.
- Avoid salty, pungent, cold and dry diet eg. Salty dry meat.^[39]
- Avoid diet, producing melancholic humour, like cow meat, salty fish, and cheese.^[39]

Ilaj bil Dawa(Drug Therapy)

In fact Da'- us- Sadaf has not been described in any classical Unani books with this name, while other skin diseases having properties like psoriasis (Da'- us- Sadaf) have been mentioned. Therefore eminent Unani physicians have tried to correlate the psoriasis with their signs and symptoms and described it in their books with different names as mentioned above.

In Unani system of medicine the management of psoriasis is very effective. The eminent Unani physicians like Muhammad, Tabri, Ibn-e-Sina, Ibn e Zohr, Ibn e Hubal Baghdadi, Ghulam Jeelani, Akbar Arzani and Azam Khan has described the basic principles of treatment under the following headings.

Principles of treatment (Usool-e-Ilaj)

Excretion of Fasid Akhlat (morbid humours)

The disease is caused by morbid humours, mostly black bile (*Sauda*) that must be excreted from the body for maintaining the balance of humours of the body. Excretion (*Tanqiyah*) can be done, after giving *Munzij khilt-e-Sauda*, by use of purgatives like *joshandah-e-Afsanteen*.^[8,19,41,45,48]

Use of blood purifying drugs

As per the Unani system of medicine, skin diseases can be due to accumulation of unwanted and waste metabolic products in blood. So the drugs, which help in purification like *Musaffiyat-e-Dam* should be used.^[8,19,41,45,48]

Local application of emollients

Many of the Unani physicians have emphasized to apply any emollient over the lesions frequently in the form of ointment or oil.^[40,41,42,45,46,47]

Anti-inflammatory drugs

Anti-inflammatory drugs should be applied locally to promote early healing. Moreover they advised to avoid foods like sour, sweet in the diet of patient.^[40,41,42,45,46,47]

Digestive drugs should be used: Jawarish Jalinoos, Majoon Dabeed-ul-Ward, Jawarish Aamla, Jawarish Kamooni, Jawarish bisbasah.

Munzija-e-Sauda Adviyah used in Da' - us- Sadaf

Shahatrah, Unnab, Aftimoon vilayati, Badranjboyah, Bisfaaij Fistaqi, Badyaan, Maweez Munaqqa, and Gauzaban.^[8,19,41,45,48]

Mushil-e-Sauda adviyah used in Da' - us- Sadaf

Turbud, Ghareeqoon, Sana Makki, Shahme Hanzal. Decoction of Munzija e Sauda drugs is used for 15 to 40 days, after that mushil e sauda drugs is added to munzija drugs and used for 1-2 days.^[8,19,41,45,48]

Musaffiyat e Dam adviyah used in Da' - us- Sadaf

Shahatrah, Sarphookah, Charaitah, Gul e Mundi, Nagand-e-Babri, Unnab, Barg-e-Hina, Sandal safaid wa surkh.^[8,19,41,45,48]

Unani physician Muhammad Tabri has mentioned in his book Moalajat-e-Buqratiyah that Taqashshur-e- Jild should be treated as follows. Venesection should be performed in both the hand with the gap of 7 days in between, after that following decoction is used. Afsanteen 25 gm, Shahatrah 40 gm, parsiyaonsha 35 gm, Tamar e hindi 35 gm, Halilah Zard 40 gm, Turanjabeen 52 gm, Injeer 3, Unnab 40, Luk neem kob 7 gm, Revand 7 gm, Maweez e Munaqqa 82 gm, barg e makoh 5 gm. All these drugs is boiled in 1500 ml of water until 400 ml of water is remaining, after that sugar and Roghan-e-badaam 17 gm each is added to the decoction, and then use it until the patient feels weakness. It will remove the morbid humour from the body, and then use energy rich diet.^[84]

Majoosi has described the treatment of Taqashshur e Jild in his book Kamil us Sana'ah that following decoction is used after complete nuzj of morbid humour. Banafshah 17gm, Halilah

Kabuli 17 gm, Halilah Zard 17 gm, Halilah Siyah 17 gm, Sana Makki 24 gm, Bisfaaij Fistaqi 10gm, Turbud Mujawwaf 10 gm, Gul e Surkh 10 gm, Gul e neelofar 10 gm, Tukhm e Kasni 10 gm, Aslusoos Muqashshar 10 gm, Ustukhuddoos 10 gm, Maweez e Munaqqa 35 gm, Unnab 20, Sapistan 20. Boil all the above drug in 1200ml of water until it remains 400ml and then stirr Maghz Floos e Khayar e Shambar and Turanjabeen 52 gm each in the above decoction, and take it in the morning. After complete evacuation of morbid humour use Maa ul Jubn as an internal liquored.^[95]

Hakim Akbar Arzani mentioned the treatment of Psoriasis in his book Tibb e Akbar. Remove the morbid humour from the body with the help of decoction of Aftimoon and Maa ul jubn. Halilah Siyaah 24gm, Halilah Kabuli 24gm, Hlilah Zard 24gm, balilah 10gm, Sheer e Aamlah 10gm, Shahatrah 30gm, Afsanteen Roomi 30gm, Gul e Ghafis 17gm, Qantooriyoon 17gm, aftimoon vilayati 24gm, maweez Munaqqa 52gm, Ghariqoon 3gm, Turbud 3gm. Boil all drugs in 2litre of water until 500 ml of water is remaining, after adding 24gm of sugar, take the decoction.^[8]

Emollient drugs used in psoriasis for local application

Roghan Badam(Almond oil), Roghan Zaitoon(Olive oil), Roghan Nargeel(Coconut oil), Roghan Banafshah(). Marham Daa us Sadaf, (Ingridients: Murdaarsang(Monoxide of lead), Raskapoor, Kafoor(Camphor), Mom(Wax)).^[40,41,42,45,46,47]

Compound drugs used in Psoriasis

Itrifal e Shaharah, Majoon e Usbah, Khameerah e Sandal, Sharbat e Sandal, Sharbat e Unnab, Sharbat e murakkab Musaffiy e Khoon, Arq e Shahatrah. Marham Gulabi, Marham Daa us Sadaf, Marham Hina, Roghan Babchi, Roghan Chalmoghra, Roghan Kamelah for local application.^[8,19,41,45,48]

Digestive drugs can be used in psoriasis

Jawarish Jalinoos, Majoon Dabeed-ul-Ward, Jawarish Aamla, Jawarish Kamooni, Jawarish bisbasah.^[8,19,41,45,48]

The following drugs are used in modern system of medicine**Antimitotic or cytotoxic agents**

Methotrexate, a competitive antagonist of tetrahydrofolate reductase, blocking the formation of thymidine and thus DNA. It inhibits cellular proliferation and is highly effective in many patients with psoriasis. It is thought that this antiproliferative activity may be important in reducing epidermal and lymphocyte proliferation. It is administered on a weekly schedule in doses of 5-25mg orally or intramuscularly.^[14,17,28,29,33,53,71,77,88,90]

Cyclosporin, It inhibits CMI due to inhibition of lymphocyte mitosis and release of lymphokines. It has antiproliferative effect on the keratinocytes. It is dramatically effective in psoriasis when given in doses of 3-5 mg/kg/day.^[14,17,28,29,33,53,71,77,88,90]

Retinoids like Acitretin, It is a retinoid with profound effects on keratinization. It regulates growth and terminal differentiation of keratinocytes. It is administered orally on a daily basis and is particularly effective for pustular psoriasis. It often improves but seldom clears the more common type plaque psoriasis.^[14,17,28,29,33,53,71,77,88,90]

Topical therapy**Antipsoriatic agent**

Topical corticosteroids:^[20,23,40,41,49,104,113,131,149,151]are both antimitotic and anti-inflammatory. Various topical corticosteroids are successfully used in the treatment of psoriasis. Clobetasone and momentasone is the most potent of the currently available topical corticosteroids. But the recurrence is more common when the local steroid treatment is withdrawn.^[14,17,28,33,62,88]

Anthralin also called Dithranol. It has a cytostatic and a strong anti-inflammatory effect, it also reduces DNA synthesis. It is used in two concentrations. Low concentration(0.05%) is applied for 18-22 hours daily, higher concentration(0.25-2%) used as short contact therapy(30 minutes applications and then washed off). Drithocreame, ranging in concentration from 0.1% to 1.0%.^[14,17,28,33,62,71,77,88,90]

Tars. It acts as an anti-bacterial, anti-fungal, anti-pruritic, anti-acanthotic, atrophogenic and photosensitizing agent. A compound preparation a tar liquid-5% liquor carbonis detergens (LCD)-contained in Aquaphor.^[14,17,28,29,33,71,77,88]

d) Vit-D Analogue such as Calcipotriol (Dovonex ointment, cream, and Lotion). It reduces the epidermal cell proliferation and inhibits T cell proliferation in response to IL-1, and used as a 0.005% ointment for the treatment of stable plaque psoriasis.^[17,29,33,62,67,71,77,88,90]

Tazarotene,(Tazorac gel 0.05% and 0.1%) is a retinoid (Vitamin a derivative). It is a keratoplastic and keratolytic agent, reduces scaling and plaque thickness, but not erythema. Topical tazarotene is applied at bed time, often in conjunction with a topical steroid applied in the morning.^[17,29,33,62,67,71,77,88,90]

Keratolytic or descaling agent: Salicylic acid used in the treatment of psoriasis, generally as a 2% to 10% ointment applied twice daily over a thick lesion. It causes shedding of scales of psoriatic lesions, by dissolving the intercellular matrix between corneocytes and softening the stratum corneum.^[14,17,28,62,67,77,88,90]

Biological Agents

Such as alefacept, efalizumab, etanercept, adalimumab are recently introduced agents, which are used in certain clinical setup but they are long term benefit outlays. The hazards, is still matter question. Therefore these alternative biological agents need further exploration.^[31,73]

Dialysis

There are recommendations by few dermatologist as well as nephrologists that dialysis improves the psoriasis in patients with normal renal functions. Although its mechanism is not fully understood and it is supposed that there is removal of psoriatic factors through dialysis.^[88]

Photochemotherapy

PUVA Therapy

A treatment with oral or topical psoralen and subsequent long-wave ultraviolet-A radiation is known as PUVA therapy. Parrish and coworkers introduced it in 1974.^[9,12,14,17,28,33,62,67,71,77,88,90]

BATH PUVA

The photo sensitizer (8-MOP or 5-MOP) is delivered to the skin by addition to bath water.^[7,9,12,14,28,33,53,62,67,71,77,88,90]

Goeckerman's regime

Ultra violet-B with topical coal Tar.^[14,17,62,77]

Ingram regime

Ultra violet-B with topical Anthralin.^[14,17,62,77]

CONCLUSION

Da-us-sadaf is a complex, multifunctional inflammatory skin diseases caused by accumulation of abnormal sauda in the skin and characterized by T-cell activation, local vascular changes, abnormal keratinocyte proliferation and neutrophil activation. In this article we have tried to highlight the historical background, pathophysiology and current Unani and western approach of management, which will be hopefully, help the students and researchers working in this area.

Bibliography

1. Aleem, Tabeeba Shagufta: "Amraz-e-Jild" Saba Publishers Aligarh, 2002; 65-67.
2. Al-Razi, Abu Bakar Mohammad Bin Zakariyya: "Kitab-Al-Hawi Fil-Tibb", 1stEd; Dairatu-Maarif, Hyderabad, 1970; 23: 72.
3. Andrea L Neimann, Steven B Porter and Joel M Gelfand: "The epidemiology of psoriasis, Expert Rev. Dermatol, 2006; 1(1): 63-75.
4. Angarooni, Mulla Sadid-Al-Din: "Sadeedi Sharah Al Mujiz", Pub. and year not mentioned, 823.
5. Antaki, Daud-Al-Zaheer: "Tazkirat-ulul-Albab Wa Al- Jame Lil Ajab-il-Ajaib", Pub. and year not mentioned, 1: 168.
6. Aqsarai, Jamaluddin: "Al-Aqsarai", 1st ed; Matab Al-Nami, Lucknow, 1908, 4:169
7. Arnold, Harry L et al: "Andrew's Disease Of the Skin Clinical Dermatology", 8th Ed; W B Saunders Company, 1990; 1-13: 198-204.
8. Arzani, Hakim Akbar: "Tibb-e-Akbar", Urdu Faisal Publication Deoband. Year not mentioned, 739.
9. Ausiello, Goldman: "CECIL Text Book of Medicine", 23rd Ed; Saunders Elsevier. Philadelphia, 2007; 2: 2940-41.
10. Baran R, Dawber RPR: The Nail in Dermatological Diseases. In: diseases of the nails and their management 2nd ed. Oxford: Blackwell scientific Publication; 1984: 135-137.

11. Barker AJ and Wilkinson DS: Psoriasis: In: Rook AJ, Wilkinson DS and Ebling FJ eds. "Text Book of dermatology". Blackwell Scientific Publications, Oxford: 1999; 1315-1409.
12. Behl, P.N: "Practice of Dermatology", CBS Publishers and Distributors, NEW Delhi, 2000; 14-18: 253-256.
13. Boon A. Nicholas et al: "Davidson's Principles and Practice of Medicine", 20th ed; Churchill, Livingstone, 2006; 1287-92.
14. Braun-Falco, O. et. Al: "Dermatology" Springer- Verlag Berlin Heidelberg New York, 1991; 417-34.
15. Braunwald, et al: "Harrison's Principles of Internal Medicine", 17th McGraw Hill Medical Publishing Division, New York, London, New Delhi, 2008; 1: 315-16.
16. Buske-Kirschbaum A, and Hellhammer H: Endocrine and immune responses to stress in chronic inflammatory skin disorder. *Ann NY Acad. Sci.*, 2003; 992: 231-240.
17. Champion, R.H. et al: "Text Book of Dermatology", 6th ed; Black well science Ltd. etc., 1998; 2: 1598-1643.
18. Christophers E, Mrowietz U: Psoriasis. In: Fitzpatrick TB, Eisen AZ, Wolff K, eds. *Dermatology in general Medicine*. McGraw Hill, New York: 2003; 489-511.
19. Chughtai, Ghulam Muhiuddin and Chughtai, Fasihuddin: "Desi Tibbi Pharmacopeia", 1st ed; Shiekh Mohammad Bashir and Sons, Lahore, Pakistan 2: 166-67.
20. Dirckx JH: Dermatologic term in the medicina of Celsus. *Am J Dermatopathol*, 1938; 5(4): 363-369.
21. Duffy DL, Spelman LS, Martin NG et al: Psoriasis in Australian twins. *J Am Acad Dermatol P. Book.*, 1993; 29: 428.
22. Duweb. GA, Eldebani S, Alhaddar J: Calcipotriolcream in the treatment of flexural psoriasis. *Int. J tissye react*, 2003; 25(4): 127-130.
23. Elder David, Elenitsas Rosalie, et al: "Histopathology of the skin" 8th Ed. Lippincott, Philadelphia. New York.
24. Elder JT, Rajan P, Nair P et al: The genesis of psoriasis. *Arch Dermatol*, 1994; 130: 216-224.
25. Epstein, Owen, et al: "Clinical Examination", Mosby USA: 4.14-4.16.
26. Farber EM, Nall L: Guttate psoriasis. *Cutis* 1993; 51(3): 157-159,163-164 and The Natural History of Psoriasis in 5600 patients. *Dermatologica*, 1974; 148: 118-122.

27. Fierlbeck G, Rassner G, Muller C: Psoriasis induced at the injection site of recombinant interferon gamma. Result of immunohistologic investigations. *Arch Dermatol*, 1990; 126(3): 351-355.
28. Fitzpatrick, Freedberg, Irwin M et al: "Dermatology in general Medicine" 6th Ed. Medical publishing division McGraw Hill. 1: 407-420.
29. Fitzpatrick, James E. et. Al: "Dermatology Secrets" Hanley & Belfus, INC Medical Publisher, Philadelphia, 1996; 1-5: 15-21, 39-42.
30. Ghaznivi, Khalid: "Amraz-E-Jild Wa Ilaj-e-Nabvi", Azeem Publisher, New Delhi, 1993; 257-60.
31. Ghosh Sanjay: "Recent advances in Dermatology" Jaypee brothers, 2007; 2: 277-85.
32. Gupta MA, Gupta AK, Kirby S et al: A psychocutaneous profile of psoriasis patients who are stress reactors: a study of 127 patients. *Gen hosp Psychiatry*, 1989; 11: 166-173.
33. Habif P. Thomas: "Clinical Dermatology", 4th ed. Mosby Toronto, 2004: 209-39.
34. Hallman-Mikolaiczak A: Ebers Papyrus. The book of medical knowledge of the 16th century B.C. Egyptians. *Arch Hist. Filoz. Med.*, 2004; 67(1): 5-14.
35. Hamid, Isa Pasha: "Kitab-Al-Meraj Fi-Al-Tibb", 1st ed; Bil Matab-Al-Wataniyyah Bil-Misr, 1303H.; 278-79.
36. Henseler T, Christopher E: Psoriasis of early and late onset: Characterization of two types of psoriasis vulgaris. *J Am. Acad. Dermatol*, 13: 450, 1985.
37. Higgins E: Alcohol, Smoking and Psoriasis. *Clin. Exp. Dermatol*, 2000; 25: 215-223.
38. Hulse EV: The nature of biblical "leprosy" and the use of alternative medical terms in modern translation of the Bible. *Med Hist*, 1976; 20(2): 203.
39. Ibn-e-Rushd, Abul Walid Mohammad: "Kitab-ul-Kulliyat", Urdu Tarjamah CCRUM New Delhi, 1980; 89-90.
40. Ibn-e-Sina, Abu Ali Ibn-Al-Hussain: "Al-Qanoon", Matba-Al-Nami, Lucknow, 1906; 4: 303-05.
41. Ibn-e-Zehr, Abu Marvan Abdul Malik: "Kitab-al-Taiseer Fil Madavat-e-Wa-Al-Tadbeer", 1st ed; Urdu Tarjamah CCRUM New Delhi, 1986; 204-05.
42. Ibn-ul-Quf, Abu-Al-Farj: "Kitab-Al-Umdah Fil Jarahat", Urdu Tarjamah CCRUM New Delhi, 1: 100, 174-75.
43. Israili, Mohammad Ayyub: "Tarjamh Urdu Shareh-e- Asbab", Munshi Naval Kishore, Lucknow, 2: 414-15.
44. Jeelani, Hakim Ghulam: "Makhzan-e-Hikmat", Matba S.H Offset Press, New Delhi, 1996; 2: 698.

45. Kabeeruddin, Hakim Mohammad: "Tashreeh-e-Kabeer", Qarol Bagh, Delhi, 1924; 2: 563-70.
46. Kareem, N.M: Maknzanul-Advia, Urdu, Vol 1, 1765 Matba'a Munshi Naval Kishore Press, Kanpur. Jild-2 39, 40 part-1 266 Jild 1 part 2: 526,661,722,723.
47. Khan, Mohammad Azam: "Akseer-e-Azam", Matba Nizami, Kanpur, 1289 Hijri, 4: 436,511.
48. Khan, Najm-ul-Ghani: "Khazain-ul-Advia", edition and year of pub not mentioned, Idara-e-Ketab-al-Shifa, New Delhi, 187-89, 316-17, 421-22, 832-34, 894-95, 1133-34, 1284-85, 1352-55.
49. Koebner H. Zur: Aetiology psoriasis. *Vjschr Dermatol* 1876; 3: 559.
50. Kormeili, T. et. al: "Psoriasis Immunopathogenesis and Evolving Immunomodulations and systemic therapies;U.S. Experiences" *British Journal Of Dermatology*, No.1 July. 2004; 151: 12.
51. Kritkar, K.R., et al: "Indian Medicinal Plants", 2nd ed; International book Distributors, Dehradun, 1975; 2: 138-40 607-09.
52. Kumar, Praveen and Clark, Michael: *Clinical Medicine*, 6th Ed, Elsevier Saunders Pub. USA, 2005; 1322-1324.
53. Lal S. Clinical pattern of psoriasis in Punjab. *India J Dermatol Venereol*, 1966; 35: 5-12.
54. Lebwohl M: Psoriasis. *Lancet*, 2003; 361: 1197-1204.
55. Lerman S: Ocular side effects of accutane therapy. *Lens Eye Toxic Res*, 1992; 9(3-4): 429-38.
56. Marks, R: "Roxburgh's Common Skin Disease", 17th ed; Arnold international student's edition, 2003; 128-42.
57. Mathew Meier. Pranab B. Sheth Yawalkar N: (ed) *Management of psoriasis Curr. Probl. Dermatol. Basal Karger*, 2009; 38: 1-20.
58. McKee HP: "Essential skin pathology". London: Mosby international Ltd., 1999; 71-72.
59. Melski JW, Stem RS: The separation of susceptibility to psoriasis from age of onset. *J invest Dermatol*, 77: 474, 1981.
60. Michael Traub, ND, and Keri Marshal MS, ND: "Psoriasis-Pathophysiology" *Alternative Medicine Review Number 4* 2007; 12: 319-330.
61. Ministry of Health: "Pharmacopoeia of India", 2nd ed; Manager of Publications, Delhi, 1970; 83: 376.
62. Multani, Hakim Harichand: "Taj-ul-Hikmat", Malik Book Depot Lahore, Pakistan, Year not mentioned, 471.

63. Murphy GF: "Dermatopathology" 1st ed. Philadelphia W. B Saunders Company; 195: 73-77.
64. Nakayama J: Four cases of sebopsoriasis or seborrheic dermatitis of face and scalp successfully treated with 1a-24(R)-dihydroxycholecalciferol (tacalcitol) cream. *Eur J Dermatol*, 2000; 10(7): 528-532.
65. Ogilvie, Colin, et al: Chamberlain's Symptoms and Sign in Clinical medicine, 12th ed. Butterworth Heinemann, 1997; 59-60.
66. Pasricha J.S: Treatment of Skin disease, 4th ed. 2000, Oxford and IBH Publishing Co. Pvt. Ltd., 83-89.
67. Pasricha, J.S., et. al: "Illustrated Text Book Of Dermatology", 3rd Ed; Jaypee Brothers, New Delhi, 2006; 141-50.
68. Pereira, P. Rocha, et. al: "The inflammatory Response in mild and in Severe Psoriasis" *British Journal Of Dermatology*, No.5, May. 2004; 150: 927.
69. Pereira, P. Rocha, et. al: "Erythrocyte Damage in Mild and Severe Psoriasis" *British Journal Of Dermatology*, No. 2, Feb. 2004; 150: 242.
70. Rastogi, Ram P. et al: *Compendium of Indian Medicinal Plants*, (Reprint) CDRI, Lucknow and National Institute of Science Communication, New Delhi, 2001; 3: 522, 569, 591.
71. Ratan, Vidya: *Handbook of Human Physiology*, Jaypee Brothers Medical Publishers (P) Ltd., New Delhi. Year of Pub. not mentioned 173-174.
72. Richards, H.L. et.al: "Detection of psychological Distress in patients with Psoriasis: Low Consensus between Dermatologist and Patients" *British Journal Of Dermatology*, No. 5 May, 2004; 150: 927.
73. Ritu Saini, William D. Tutrone and Jeffrey M. Weinberg: "Advances in Therapy for Psoriasis" *Current Pharmaceutical Design*, 2005; 11(2): 273-280.
74. Rizova H, Nicolas JS, Morel P et al: "The effect of anti CD4 monoclonal antibody treatment on immunopathological changes in psoriatic skin". *J Dermatol Sci*, 1994; 7(1): 1-13.
75. Robbins, Stanley L: *Pathological Basis of Diseases*, 4th ed. 1984, W.B. Saunders Co. Philadelphia, USA, 1309.
76. Roenigk, Henry H., et. al: "Psoriasis", 2nd Ed; Marcel Dekker INC. New York, Hong Kong, 1990; 3-7: 45, 51, 11, 131-135, 157, 171-173, 177, 181, 201-03, 209-19, 255, 228-31, 233-35.

77. Sainani, Gurumukh S., et.al: "API Text Book Of Medicine", 6th Ed; Association of Physicians of India, Mumbai, 1999; 1198-99.
78. Salim, Izzat Salim Beg: "Wasail-ul-Ibtihaj Fil-Tib-il-Batini Wal-Ilaj", Al Misr, Year not mentioned, 599-603.
79. Sharpe RJ, Arndt KA, Bauer SI et al: Cyclosporin inhibits basic fibroblast growth factor-driven proliferation of human endothelial cells and keratinocytes. Arch Dermatol 1989; 125: 1359-1362
80. Smoller B R, Horn TD: "Dermatopathology" in systemic disease. New York: Oxford University Press; 2001; 25-29.
81. Souhami RL. Moxham J: "Text book of medicine" 3rd ed. London: Churchill Livingstone; 1997; 33-35.
82. Stern RS: Psoriasis. Lancet 1997; 350: 349-353.
83. Swash, Michael: Hutchison's Clinical Method, 22nd ed. (ELBS), W.B. Saunders Company Ltd., London, 2007; 251-55.
84. Tabari, Ahmad Bin Mohammad: Al-Moalejat-ul-Buqratiya, Urdu translation, Vol. 1st part I-5, 183-85. 2, Part 6-8, CCRUM, New Delhi, 1997; 151-55,185.
85. Thomas, Clayton L: "Taber's Cyclopedic Medical Dictionary", 18th ed. F.A. Davis Company Philadelphia, 1997; 519.
86. Toonasi, Mohammad: "Akseer-e-Hikmat", Urdu Translation by Abdul Haque, A.R.W. Company, Lahore, Pakistan, Year not mentioned, 194.
87. Toor S.H. et al: "Kamil-ut-Tashkhees" Pub. Bu Idarah Tarjuman-Al-tib Lahore. Year not mentioned. 94-95.
88. Valia, R. G: IADVL, "Text Book and Atlas Of Dermatology", 2nd Ed; Bhalani Publishing House Mumbai, India., 2: 812-34.
89. Van de Kerkhof PC, De Hoop D, De Korte J et al: Scalp psoriasis, clinical presentations and therapeutic management. Dermatology, 1998; 197(4): 326-334.
90. Virendra N Sehgal: "Text book of clinical Dermatology" 4th Ed. New Delhi: Jaypee Brothers Medical Publishers; 2004; 126-39.
91. Weedon, David: "Skin pathology" Churchill Livingstone, 1999.
92. Weiss G, Shemer A, Trau H: The koebner phenomenon: review of the literature. J Eur Acad Derm Venereol, 2002; 16: 241: 248
93. Weisenseel, P. Laumbacher B, Besgen P. et al staptococal infection distinguishes different type of psoriasis. J. Med. Genet, 2002; 39: 767-768.

94. Usaibah, ibn e abi, uyoon ul ambaa fi Tabaqaat al atibbaa, urdu translation ccrum, New Delhi, 1990; 1: 77.
95. Almajoosi, Ali ibn e Abbas Kamil us Sana, ah Al-Tibbiyah, CCRUM, New Delhi, 2005; 1: 151-154, 185.