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Clinical Correlation of *Taqashshur-E-Jild* with Psoriasis: A Literary Approach

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ABSTRACT

Psoriasis is one of the most common dermatologic diseases. It is a chronic non-infectious inflammatory disease of the skin, characterized by development of chronic, well defined, scaly, erythematous, plaques on the extensor aspects of the body, especially on the elbows, knees, trunk, back and scalp^{1,2,3,4}. Nail involvement is very frequent and often gives the clues to the diagnosis^{1,2,3,4,5,6}. Psoriasis can be localized or generalized. High variability and unpredictability are the hallmark of this chronic affliction. The estimated prevalence is 1.5% to 3% in general population. It has bimodal peak of incidence, at 16-22 years and 57-60 years. In Unani System of Medicines, Galen was the first person who described a skin disease in the name of psoriasis^{7,8}. But before and after the Galen many Unani physicians described the various names such as Sadafia, daus sadaf, baheq-e- siyah and Samakia⁹. In classics of Unani literature various references are found such as Sadafia, Da-us- Sadaf, Baheqe siyah, Samkia and qubae mutaqashshar which qualify for the disease of psoriasis^{1,10}. Celsus described impetigines and specified that the second species of impetigo was characterized by red skin covered with scales. This description suggested a type of papulosquamous disease, such as psoriasis. So, keeping the fact in mind want to established the clinical correlation with anaemia in the light of classical Unani literature as well as modern medicine.

Keywords: Taqashshur-E-Jild, baheq-e- siyah and Samakia, Sadafia, Da-us- Sadaf, Baheqe siyah, Samkia and qubae mutaqashshar.

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INTRODUCTION

Psoriasis is derived from Greek word so-ri-a-sis which means an itching. Psoriasis is a common, genetically determined, chronic, recurrent, inflammatory and proliferative disease of the skin characterized by round, circumscribed, erythematous, dry, dull red scaling plaques of various sizes, covered by greyish white or silvery white, imbricates and lamellar scales and typical extensor distribution. There is an accelerated rate of epidermal turnover, with hyper proliferation and defective maturation of epidermal Keratinocytes. The lesions have a predilection for the scalp, nails and extensor surfaces of the limbs especially the shins, elbows, knees, umbilical and the sacral region. The disease is enormously variable in duration and extent and morphological variants are common^{2,3,4,5}

Hippocrates (460-370 BC) was one of the first authors to write descriptions of skin disorders. He utilized the word *lopoi* to describe the dry, scaly, disfiguring eruptions of psoriasis, leprosy and other inflammatory skin disorders¹¹. Similar to Hippocrates' works, the Old Testament also lumped together many cutaneous disorders. The biblical term *tsaraat*, or *zaraath*, described a range of skin conditions including leprosy and psoriasis. Lepers were often ostracized because they were considered divinely punished and cruelty was imposed upon those who suffered from psoriasis and leprosy alike⁹.

Psoriasis is worldwide in distribution. It affects about 1.5 to 3% of population^{2,3,4,5,8,12,13}. It is most common in Caucasians of the western world¹⁴. The prevalence of psoriasis in southern Europe is lower than that in northern Europe, varying from 1.3% in Germany to 1.55% in Croatia, 1.58% in U.K, 1.3% in Turkey, 1.7% in Denmark and 2.3% in Sweden. In contrast to Europe & U.S a low prevalence of psoriasis is found in Asia and West Africa and in northern American blacks. The incidence is low in Japanese and Eskimos^{9,15,16}.

In India the reported incidence of psoriasis in different studies are 5.6%, 0.84%, 1.25%, 1.5%, 1.4%, and 2.8%. In India Jews and parsis have been found to be more susceptible than Hindus and Muslims^{16,17,18,19,20,21}.

So, Psoriasis is a major public help with this reason I have started correlation between *Taqashshur-E-Jild* and psoriasis with the help of classical Unani literature on the basis of causes clinical features pathogenesis and their management on the basis of modern parameter like clinical features, pathogenesis.

MATERIALS AND METHOD

Review material collected from the different ancient Unani books, PG Dissertation, online authentic research Journals & different websites and summarized with the help of computer.

LITERATURE REVIEW

The words Taqashshur-e-Jild are an Arabic term, which means "Peeling of scales from skin. It is a type of skin disease in which there is indurations of skin with silvery scaling like scale of fish. Unani scholars have mentioned the different aspects of disease in their respective works and also used the different terminologies for Da-us-Sadaf such as Qooba-e- Mutaqashsherah, Taqashshur-e-Jild, Qooba-e-Muzmin, Qashf-e-Jild, Sa'afa-e-qishri, Al-Sadafia, Chamba Talaq, Apras etc^{1,22}. Most of the Unani scholars have given the concept of Da-us-Sadaf or Taqashshur-e-jild as below:

Ibn-e-Rushd (1126-1198AD) described Da-us-Sadaf as a type of leprosy. He also mentioned that excessive formation of abnormal black bile (Ghair Tabai Sauda) in the body produces disease that cannot be removed easily²³.

Ibn-e-Hubal Baghdadi (1198AD) gave the description of Da-us- Sadaf. He mentioned that, Daus Sadaf is similar to Sa'afa-e-Yabisah, in which skin becomes dry, rough and wrinkled, from which red fluid oozes out and the lesion is covered with scales²⁴.

Ibn-e-Quaf described Taqashshur-e-Jild as a kind of Sa'afa-e-Qishri in his book, Kitab-ul-Umdah Fil-Jarahat as the disease of skin, in which white impetigo appears with formation of scales resembling the scales offish²⁵.

Akbar Arzani (1722AD) was a great Unani physician; he also described Taqashshur-e-Jild, in which the skin becomes dry, rough, thick, and scales cover the affected parts of the skin²⁶.

Azam Khan (1813-1902AD) described the disease as Qashf-e-Jild and Taqashshur-e-Jild in his book Ekseer-e-Azam²⁷.

Ghulam Jeelani discussed about Taqashshur-e-Jild with the name of Sadafia, Apras, and Chambal. He described etiology, clinical presentation, and management of Taqashshur-e-Jild in details²².

Zakaria Razi has described it in his book "Al-Hawi-Fil-Tib" as a roughness in skin with itching²⁸.

Majoosi described the characteristic of psoriasis as peeling of scales from the skin²⁹.

Rofas described a condition of Talaq, in which the lesion is surmounted by white scales resembling to Abrak³⁰.

Ibn-e-Zohr defined Daus Sadaf as the disease of skin, in which patient feels severe itching over the lesion³¹.

Pathology

Psoriasis has not been mentioned with this name in old Unani books, but it has been described as Taqashshur-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-Zohr 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 scales³¹.

Ali Ibn-e-Abbas Al-Majoosi has described that Tabiyat expels the khiit-e- Ghaleez towards skin from internal organs resulting in the dryness and itching of the skin, in this condition skin is unable to remove Khhit-e- Ghaleez leading to accumulation of Sauda in skin²⁹.

In classics of Unani literature various references are found such as Sadafia, Da-us- Sadaf, Baheqe siyah, Samkia and qubae mutaqashshar which qualify for the disease of psoriasis^{1,22}.

Ibn-e-Sina (Avicenna) (980-1037 AD) has referred it as qubae mutaqashshar which equally qualifies for the psoriasis. According to Unani literature, the causes of this disease is abnormal black bile or bile, which has burnt, bile in which there are qualitative as well as quantitative changes leading to psoriasis. The body excretes the abnormal humors in the form of viscid fluid, which moves towards the skin and forms crests that cause malnourishment of the skin. The toxins accumulate in the skin and decaying of the skin appears in the form of scales. Due to dryness scales comes out. Its cause is khilt Sauda. It is produced by the fluid which becomes dry like ash after burn and nature (Tabiyat) through it outside towards skin. If there is heat present it causes itching with dryness, otherwise there is no itching. Another reason of scaling of the skin is burnt Saudavi khilt, which is considered as a destructive humour. Which causes intense itching and scaling³².

Hkm. Kabeeruddin explained it, in Tarjuma-e-Kabir under the heading of Baheqe Aswad and more specifically Bars-e-Aswad, in which Khilt-e- Sauda is responsible for this skin disease. Khiit-e-Safra particularly in young people after burnt out changes into Khilt-e-Sauda, which gets deposited below the skin. The Sauda itself has properties of dryness, so it dries up skin. The dryness of skin gives rise to scaling and itching and burning sensation & sometimes skin gets cracked, bleeds and secondarily infected³³.

Abu-bakar Muhammad Bin Zakaria Razi, wrote in his book, "Al-Haavi fit- Tib" that, matter (material) of Qooba is less in quantity & sour and sticky. One of its types is very bad; there are red patches, itching and roughness in it. It is treated by leech therapy²⁸.

Ibn-e-Hubal Baghdadi wrote in his book, Al- Mukhtarat fit-tib" about Bars-e -Aswad, which is also called Qooba mutaqashshiir, its cause is thick material of Sauda, which is spread in skin and

scales, which itching and gives burning sensation. It is a bad disease, if becomes chronic, is difficult to treat. It is like leprosy of the skin. And also writes about Qooba & Taqashshur-e-Jild, this word is like dry Ganj of skin only difference is that matter of Qooba is on surface of skin and matter of Ganj is in deep. According to humour it is very bad. If Qooba is dry it is purely because of Sauda. If there is wetness and redness there is mixing of blood with Sauda. Wet qooda is easily treatable but sometimes it becomes chronic and bad.

In the detail of saafa he writes, one of the types of Ganj is dry and white, and scales shades from it. Its patients are called Ganja i.e. his head is like that salty (shore) soil where there is deposition of like algae, which is called saafa yaabis. Its material is dry like ash, which is made up of salty burnt phlegm or Sauda ²⁴.

Ibn-e-Sina writes in "Al- Qanoon fit Tibb" about Saafa (Ganj), initially it starts in the head of skin like beats on different places which change into scaly wound with redness. Sometimes there is secretion of fluid from it which is called wet saafa and sometimes presents in the form of dry Qooba especially in winter season and soon dissolves³⁴.

In Tarjuma-e- Qanoon the cause of Quba or Daad is described as a skin disease having scales filled with pungent fluid having high acidic nature, mixed with viscous or dense matter formed from black bile. Its further states that one of the few types of Quba exhibit scaling due to increased dryness and excess quantity of altered humour and the disease is more active during Kharife season³⁴.

Ibn Abbas Majoosi writes in his famous book "Kamil- us -Sana", that the old lesion of Daad or Quba peel off scales, which are round like those of a fish, and the cause of scaling and itching is attributed to admixing of khilt- e- Sauda with blood⁶⁸. In the same book it mentioned under the heading of leprosy that the dense or black bile when driven to the viscera, causes Sartaan (cancer) and if the black bile is thin it causes Bars (vitiligo) or Baheqe aswad (Pityriasis) or Quba e Daad etc. and if it gets dispersed throughout the body and is not infected it give rise to Juzam (leprosy) and if it gets infected it causes Humma-e-Saudavi.

In **Tibbe Akbar** it is mentioned that Quba or Daad sometimes presents as a chronic condition with scaling similar that of a fish²⁶.

In the **Firdaus-ul Hikmat** psoriasis has been described as Taqashshur-e- Jild in which skin becomes rough and scales are cast off which is due to combustion of black bile and dryness of skin. Its further states under the heading of chajan Vo Apras, it presents as red or black colored rough areas of skin of extremities, the course of which is fasade khoon that is altered blood or derivatives of combustion in the blood.

UNANI TREATMENT OF PSORIASIS

In Unani system of medicines the recommended lines of management to control psoriasis are Nuzuj wa tanqiy e akhlate gair tabiya (concoction and expulsion of abnormal humours) especially Sauda (melancholic humor) by aftimoon wilaiti (*Cuscuta reflexa* Roxb), Tukhme babchi (*Psoralea corylifolia* Linn), Bisfajj fistaqi (*Polypodium vulgare* Linn), Ghariqoon (*Polyporus officinalis* Fries), Turanjabeen (*Fraxinus ornus* Linn) along with tehleele auram (resolution of inflammation) by Makoh (*Solanum nigrum* Linn), Kasni (*Chicorium intybus* Linn), Brinjasif (*Artemisia vulgaris* Linn), Tasfeeya e dam (blood purification) by Shahetra (*Fumaric purviflora* Lam), Unnab (*Zizyphus vulgaris* Lam), Chiraita (*Swertia chirata* Buch-Ham), Sarphuka (*Tephrosea purpurea* Linn Pers), Ushba magrabi (*Smilax aspera* Linn), Karela (*Momordica chirantia* Linn), Indamale zakhm (cicatrization) by sendoor (plumbum), sange jarahat (silicate of magnesia), Mazu (*Quercus infectoria* Oliv), Hina (*Lawsonia inermis* Linn), Taskeene jild (Demulcification) by Behidana (*Cydonia vulgaris* Pers), Unnab, Tukhme kahu (*Lactuca scariola* Linn), Samage arabi (*Acacia arabica* willd), Tarteebe umoomi w muqami (general and local moisturization) by arqe gulab (rose water), Rogane badam (almond oil), Rogane zaitun (olive oil), Rogane narjeel (coconut oil) and use of Jali (cleanser) like neem (*Azadirachta indica* Linn), Haldi (*Curcuma longa* Linn), Kamela etc.

Modern Concept of Psoriasis

Psoriasis is derived from Greek word so-ri-a-sis which means an itching. Psoriasis is a common, genetically determined, chronic, recurrent, inflammatory and proliferative disease of the skin characterized by round, circumscribed, erythematous, dry, dull red scaling plaques of various sizes, covered by grayish white or silvery white, imbricates and lamellar scales and typical extensor distribution.

There is an accelerated rate of epidermal turnover, with hyper proliferation and defective maturation of epidermal Keratinocytes. The lesions have a predilection for the scalp, nails and extensor surfaces of the limbs especially the shins, elbows, knees, umbilical and the sacral region. The disease is enormously variable in duration and extent and morphological variants are common^{2,3,4,5}.

ETIOLOGY OF PSORIASIS

Provocative and exaggerative factors:

Many factors play a role in provoking a new episode of psoriasis or in exacerbating pre-existing disease. Triggering factors may be local or systemic.

Trauma:

The Koebner's phenomenon in 1872, Koebner described a patient who, after 5 years of developing psoriasis, noted that various traumatic insults to his skin resulted in lesions of psoriasis. Epidermal trauma alone will not induce the lesions; it should also involve the papillary dermis. Koebner's phenomenon usually occurs within 7 to 14 days, but the interval may be as short as 3 days or as long as 3 weeks. Psoriasis may occur at sites of bites, burns, drug reactions, dermatitis, lichen planus, malaria, skin test, vitiligo and herpes zoster. The disappearance of a psoriatic lesion following injury is known as: Reverse Koebner's phenomenon^{2,3,4,5}.

Infections:

Infections have long been reported as a trigger for the onset or exacerbation of psoriasis. Upper respiratory tract infections and tonsillitis especially when caused by streptococci, may cause flaring up of existing psoriasis or may precipitate an attack of acute Guttate psoriasis. An abnormal group A streptococcal infection may play an important role in exacerbation of psoriasis^{11,36}.

Endocrine Factors:

Psoriasis may remit during pregnancy, but there is exacerbation during the postpartum period. Generalized pustular psoriasis may exacerbate premenstrual and may also be provoked by high dose estrogen therapy^{37,38}.

Seasonal variation:

Most patients experience worsening of their skin lesions during winter. High humidity is usually beneficial. Although sunlight is generally beneficial, small minorities of psoriatic are provoked by strong sunlight and suffer summer exacerbations in exposed skin. Photosensitivity in psoriatic is present in advanced age and female sex. 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^{9,13}.

Psychogenic factors:

Psoriasis is a stress sensitive skin disease. Stress might induce alterations in the psoriatic lesions by increasing the neuropeptide content with a concomitant decrease in activity of neuropeptide degrading enzymes, especially mast cell^{39,40}.

Alcohol and Smoking:

The risk for psoriasis was higher in ex-smokers and in current smokers than in the patients who never smoked. The relation with smoking was stronger and more consistent among women than men. Alcohol is a risk factor for psoriasis in young and middle-aged men and psoriasis may sustain drinking. An effect of alcohol in lymphocyte transformation has been suggested⁴¹.

Acquired Immune Deficiency Syndrome:

The association between severe psoriasis, psoriatic arthropathy and human immunodeficiency virus (HIV) infection is well recognized. Psoriasis was found to flare severely or to appear *de novo* in explosive form as a feature of HIV infection develops⁴¹.

Drugs:

Many drugs can precipitate or exacerbate psoriasis. A number of beta-adrenergic blocking drugs such as propranolol, practolol and metoprolol exacerbate psoriasis. Lithium compounds may destabilize and exacerbate the psoriasis. Too rapid a withdrawal of corticosteroid therapy in patients with psoriasis may result in precipitation of generalized pustular psoriasis & may cause exfoliative dermatitis as a rebound phenomenon. Antimalarials may exacerbate psoriasis. Oral Phenylbutazone, Oxyphenbutazone, Indomethacin, Diclofenac and Ibuprofen were all reported to precipitate or exacerbate psoriasis^{42,43}.

Genetic basis of psoriasis:

Psoriasis is a disease of overactive immunity in genetically susceptible individuals. A decade of genome-wide linkage scans has established that PSORS1 is the strongest susceptibility locus demonstrable through family linkage studies; PSORS1 is responsible for up to 50% of the genetic component of psoriasis. More recently, human leucocyte antigen (HLA)-Cw6 has received the most attention as a candidate gene of the PSORS1 susceptibility locus on the MCH I region on chromosome 6p21.3. This gene may function in antigen presentation via MHC I which aids in the activation of the overactive T cells characteristic of psoriatic inflammation^{40,44}.

Genomic scans have shown additional susceptibility loci for psoriasis on chromosomes 1q21, 3q21, 4q32-35, 16q12, 17q25. Two regions on chromosome 17q were recently localized via mapping which demonstrated a 6 Mb separation thereby indicating independent linkage factors. Genes SLC9A3R1 and NAT9 are present in the first region while RAPTOR is demonstrated in the second region. SLC9A3R1 and NAT9 are players that regulate signal transduction, the immunologic synapse and T cell growth. RAPTOR is involved in T cell function and growth pathways. Using these genes as an example, we can predict that the alterations of regulatory genes, even those yet undetermined, can enhance T cell proliferation and inflammation manifested in psoriasis⁴⁴.

Pathophysiology:

Until recently psoriasis was considered a disorder of epidermal Keratinocytes; however, it is now recognized primarily as an immune-mediated disorder. In order to properly understand the immune

dysfunction present in psoriasis, it is imperative to understand the normal immune response of skin. Skin is a primary lymphoid organ with an effective immunological surveillance system equipped with antigen presenting cells, cytokine synthesizing Keratinocytes, epidermotropic T-cells, dermal capillary endothelial cells, draining nodes, mast cells, tissue macrophages, granulocytes, fibroblasts and non-Langerhan cells. Skin also has lymph nodes and circulating T lymphocytes. Together these cells communicate by means of cytokine secretion and respond accordingly via stimulation by bacteria, chemical, ultraviolet (UV) light and other irritating factors. The primary cytokine released in response to antigen presentation is tumor necrosis factor-alpha (TNF-a). Generally, this is a controlled process unless the insult to the skin is prolonged, in which case imbalanced cytokine production leads to a pathological state such as psoriasis^{45,46}.

Debate continues whether psoriasis is an autoimmune disorder or a T- helper 1 (Th1) immune dysfunction. T-cell activation, TNF-a, and dendritic cells are pathogenic factors stimulated in response to a triggering factor, such as a physical injury, inflammation, bacteria, virus, or withdrawal of corticosteroid medication. Initially, immature dendritic cells in the epidermis stimulate T-cells from lymph nodes in response to as yet unidentified antigen stimulation. The lymphocytic infiltrate in psoriasis is predominately CD4 and CD8 T-cells. Adhesion molecules that promote leukocyte adherence are highly expressed in psoriatic lesions⁴⁶.

T lymphocyte stimulation:

Both mature CD4+ and CD8+ T cells can respond, to the peptides presented by APCs. While the specific antigen that these T cells are reacting to has not yet been elucidated, several antigenic stimuli have been proposed. These include self-proteins, microbial pathogens and microbial super antigens. The premise that self-reactive T lymphocytes may contribute to the disease process is derived from the molecular mimicry theory in which an exuberant immune response to a pathogen produces cross-reactivity with self antigens⁴⁷.

Considering that infections have been associated with the onset of psoriasis, this theory merits consideration. However, it has also been observed that T cells can be activated without antigens or super antigens, but rather with direct contact with accessory cells. No single theory has clearly emerged, and thus researchers continue to search for the inciting stimulus that triggers the T lymphocyte and whether T cells are reacting to a self or non-self-derived antigen. T-cell activation which subsequently supports the production of growth factors and cytokines. These growth factors sustain neoangiogenesis, stimulate epidermal hyper proliferation, alter epidermal differentiation and decrease susceptibility to apoptosis that characterize the erythematous hypertrophic scaling lesions of psoriasis. Furthermore, the cytokines produced from the immunologic response, such as

tumor necrosis factor (TNF)-alpha and interferon (IFN)-gamma and IL-2, are correspond to cytokines that are up regulated in psoriatic plaques^{14,48,49}.

Dendritic cells:

Dendritic cells are professional APCs that process antigens in the tissues in which they reside after which they migrate to local lymph nodes where they present their native antigens to T cells. Multiple subsets of APCs including myeloid and plasmacytoid DCs are highly represented in the epidermis and dermis of psoriatic plaques as compared with normal skin¹⁰. While DCs play a pivotal role in eliciting an immune response against a foreign invader, they also contribute to the establishment of tolerance. Throughout their maturation, DCs are continuously sensing their environment which shapes their production of TH1 versus TH2 type cytokines and subsequently the nature of the T cell response. When challenged with a virus, bacteria or unchecked cell growth, DCs mature into APCs. However, in the absence of a strong stimulus, DCs fail to mature into APCs, but rather present self-peptides with MHC molecules thereby creating regulatory T cells involved in peripheral tolerance. If this balance between immunogenic APCs and housekeeping T cells is upset, inflammatory conditions such as psoriasis can result^{4,5,40}.

Action of Cytokines:

Psoriatic lesions are characterized by a relative increase of TH1 (IL-2, IFN-gamma TNF-alpha and TNF-beta) to TH2 (IL-4, IL-5, IL-6, IL-9, IL-10 and IL-13) type cytokines. As discussed previously, NKT cells stimulated by CD1d over expressing Keratinocytes increase production of Proinflammatory IFN gamma without effect on the anti-inflammatory IL-4. In addition to the cytokines produced by T cells, APCs produce IL-18, IL-23 and TNF-alpha found in the inflammatory infiltrate of psoriatic plaques. Both IL-18 and IL- 23 stimulate TH1 cells to produce IFN-gamma. Clearly, a TH1 type pattern governs the immune effectors cells and their respective cytokines present in psoriatic skin^{11,50,51}.

Action of TNF alpha:

Although a network of cytokines is responsible for the inflammation of psoriasis, TNF-alpha has been implicated as a master. Proinflammatory cytokine of the innate immune response due to its widespread targets and sources.

Histopathology:

The histology of psoriatic plaques is distinguished by excessive epidermal growth termed psoriasiform hyperplasia. This pattern includes a markedly thickened skin or acanthosis, elongated downward extensions of the epidermis into the dermis or rete pegs and aberrant Keratinocytes differentiation. Mitotic figures are visible at the basal layer of Keratinocytes demonstrating rapid

proliferation and maturation responsible for incomplete terminal differentiation. Thus, Keratinocytes retain their nuclei as visualized in the parakeratotic stratum corneum. The granular layer of the epidermis is also depleted⁵².

Additionally, the rapidly proliferating Keratinocytes fail to secrete lipids that normally adhere the corneocytes to each other, thereby producing the classic scale of a psoriatic plaque. The tortuous and dilated dermal blood vessels are responsible for the erythema exhibited by psoriatic plaques³³. In addition to epidermal hyperproliferation, an inflammatory infiltrate distinguishes psoriatic skin. Collections of neutrophils termed Munro's abscesses are found within the stratum corneum. Furthermore, an influx of T cells is found in both the epidermis and dermis along with increased numbers of dermal dendritic cells, macrophages and mast cells. These unique histological features of the psoriatic plaque represent the starting line for researchers determining the mechanisms that underlie the pathophysiology of psoriasis⁵³.

TYPES OF PSORIASIS:

Clinical types of Psoriasis⁵³

1. Chronic plaque psoriasis or psoriasis vulgaris.
2. Guttate (Eruptive) psoriasis.
3. Erythroderma (Exfoliative) psoriasis.
4. Follicular psoriasis.
5. Unstable psoriasis.
6. Generalized pustular psoriasis.
7. Annular pustular psoriasis.
8. Localized pustular psoriasis

Regional Variation:

1. Scalp psoriasis.
2. Penile Psoriasis.
3. Flexural Psoriasis.
4. Ungual Psoriasis.
5. Palm and sole (palmoplantar Psoriasis).

Clinical Features: Nail shows three types of lesions

- Pitting: found to be highest in fingernails, but toes are also involved.
- Separation of distal portion of the nail from the nail bed and wails.
- Thickening of the nails, accompanied by the collection of hyperkeratosis debris under the nail.

Signs of Psoriasis:**Wax candle sign (sign de la tache de baugie):**

When psoriatic lesion is scratched, candle grease like scales is produced. This is called Wax candle sign^{15,54}.

Grattage test:

Gentle scrapping the lesion with a glass slide produces silvery scales, this is called positive Grattage test⁵⁵.

Membrane of Bulkeley:

When the scales are completely scraped off, the stratum mucosum seen as moist red surface called as membrane of Bulkeley through these dilated capillaries can be seen as red spots^{8,55}.

Auspitz Sign:

On further scrapping the capillaries seen through membrane of Bulkeley are torn, leading to multiple bleeding points. It is due to paraeratsis, intracellular edema of the epidermal cells, supra capillary thinning of the stratum malphingi, elongation of dermal papillae and dilatation and tortuosity of papillary capillaries^{12,23,24,25,52}.

Koebner's Phenomenon:

Psoriatic lesions may develop along the scratch lines in the active phase, this is called Koebner's phenomenon. The trauma may be physical, mechanical, chemical or allergic. It usually occurs within 7-14 days after trauma. This phenomenon is not specific for psoriasis and can be seen in other conditions like lichen planus, vitiligo, herpes zoster etc.^{12,23,24,25,52}

Woronoff s Ring:

Occasionally a zone of hypo pigmentation may be seen around the psoriatic plaques. This is called as Woronoff's Ring^{8,56,57,58}.

Nail pit sign:

Circular areas of yellowish discoloration of nail bed and hyponychium may be seen through the nail plate, this is called as oil drop sign. Sometimes however, oval or round brownish red lesions resulting from accumulation of parakeratotic material in the nail bed can also be seen through the nail plate. This is called as "fleck phenomenon"^{8,19,39,56,58,59}.

Complications:

Exfoliative dermatitis, Infections, Nephritis, Renal Failure, Hepatic Failure Amyloidosis.

Co-Morbidities:

Psoriasis is associated with several co-morbidities, including decreased quality of life, depression, increased cardiovascular risk, type 2 diabetes mellitus, metabolic syndrome, cancer, Crohn's

disease, and psoriatic arthritis. It remains unclear whether cancers, in particular skin cancer and lymphoma, are related to psoriasis or its treatment. Phototherapy and immunosuppressive therapy can increase the risk of non-melanoma skin cancer^{10,36,60,61,62, 63}.

DIAGNOSIS OF PSORIASIS

Diagnosis of psoriasis is purely based on the clinical presentation, history and morphological evidence^{3,58,64}.

Clinical presentation:

- Presence of lesions at particular site e.g. elbows, knees, back, nails etc.
- Lesions covered with silvery scales.
- Candle Grease sign, Auspitz sign, and Koebner's phenomenon.
- Itching.
- Seasonal variation.

Henry H. has characterized the major, intermediate and minor stigmata for the diagnosis of Psoriasis⁶⁵.

Major signs⁶⁶

- Erythematous, usually sharply marginated Plaque that often have silvery scales in hairy areas.
- Severe dandruff, often with marginated plaque.
- Nail changes.
- Multiple pitting.
- Dystrophic nail and nail separation without evidence of fungus.
- Sero-negative arthritis.

Intermediate signs⁶⁶

- Hyperkeratosis, localized with or without scaling on elbows, knees, ankle and soles etc.
- Pruritic ani or other intertrigo with sharp margination of erythema.
- Corticoid-responsive penile macule, especially on the glans.
- Recalcitrant, scaly otitis externa.
- Persistent localized patches of nummular eczema.
- Sterile paronychia often multiple.

Minor sign⁶⁶

- Eczematous plaque of palms, soles or both.
- Acute onset of keratolysis like lesions of the palms or soles.

- Recurrent eczematous discoid eruptions of trunk and extremities.
- Koebner's phenomenon, new lesions appearing at the site of trauma.

Differential Diagnosis:

Lichen planus, Pityriasis rosea, Tinea corporis, Secondary syphilis, Lichen simplex, Reiter's syndrome, Atopic dermatitis, Pityriasis rubra pilaris, Candidiasis, Seborrheic dermatitis

Treatment goals:

It is helpful to set with patients a realistic goal of therapy. Generally, a goal of complete clearance of psoriasis is not realistic; few patients actually achieve prolonged clearance⁶⁷. To minimize adverse medication effects, it is safest to try to reduce the psoriasis to a manageable level, rather than to maximize doses in an attempt to obtain complete clearing. The histopathology of psoriatic lesions reveals inflammation, Keratinocytes hyper proliferation, and vascular dilation, offering multiple targets for intervention. Often, a combination of modalities can be utilized to enhance the therapeutic effect and minimize the adverse effects that could result from excessive use of one agent.

Treatment of localized psoriasis:

Tar, Topical corticosteroids, Topical calcipotriene, Topical Tazarotene, Anthraline, Corticosteroid tape, Intralesional Triamcinolone

Treatment of generalized Psoriasis:

UVB phototherapy, Psoraline +UVA (PUVA), PUVA + acetretine (Re-PUVA), Acetretine, Methotrexate, Cyclosporine, Mycophenolate mofetil, Hydroxyurea, Leflunomide

RESULTS AND DISCUSSION

Taqashshur-E-Jild (Psoriasis) is a common skin disease seen in clinical practice. It is mentioned that psoriasis is an autoimmune disease caused by abnormal immune system activated by T-cells in the skin. It can cause disfigurement, infection and many other complications. The estimated prevalence is 1.5% to 3% in general population. It has bimodal peak of incidence, at 16-22 years and 57-60 years.^{8,9,13}

Theory of psoriasis meets similarly on the basis of etiology, presentation, complications, pathophysiology and non-pharmacological management with Taqashshur-e-Jild. The Hippocrate,²⁵ Rofas,⁶⁸ Galen,²⁵ Razes²⁷ and Avicenna²² described the psoriasis (Taqashshur-e-Jild), which is due to the excess of the black bile (Khilt-e-Sauda). They describe the causes of psoriasis are heredity^{25,68,69,70}. luxurious life style (Rahat)⁷¹, diet (Ghiza)^{71,72,73}, excess of alcohol especially after meal^{12,22,68,74} and Yaboosat-e-Mizaj "Galen" recommended the main stay of treatment is to be evacuation (Istefragh by laxative), Nuzuj wa tanqiy-e- akhlate gair tabiya, Muhallil-e-Auram

(resolution of inflammation), Tasfeeya-e- dam (blood purification)²⁵. "Avicenna" emphasized on Istefragh, Indamale zakhm (cicatrization), Taskeene Jild (Demulcification) in the management of psoriasis²². Tartebe umoomi wa muqami (general and local moisturization), and use of Jali (cleanser). These concepts seem very true in the light of modern advances.^{7,12,75}.

CONCLUSION

Though the exact terminology and complete clinical picture of psoriasis had not mentioned in the ancient books of Unani, but the above references from these famous books are very close to modern description of the disease. Further the cause mentioned in these books, as admixing of blood with abnormal phlegm (balghame shor merari) pungent acidic fluid mixed with black bile, fasaad-e-khoon or altered blood and heredity, all of these correlates with causes mentioned in modern books of dermatology as a genetic or heredity cause, change in the biochemistry and the immunological cause of blood with Balgham-e-shor merari, such as abnormal blood is sent towards the skin via peripheral circulation³⁰.

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