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Review Article

Baraṣ (Vitiligo) in Historical and Unani Literature: A Comprehensive Review

Dr. Mohd Tarique[1]*, Dr. Naila Naaz[2], Dr. Mohammad Zubair[3], Dr. Shaikh Imtiyaz[3], Dr. Mohd Furqan Patel[3]

[1] Assistant Professor, Department of Medicine (Unani), Mohammadia Tibbia College & Assayer Hospital, Malegaon, Maharashtra, India

[2] M.D., Department of Medicine (Unani), National Research Institute of Unani Medicine for Skin Disorders, Hyderabad, India

[3] Associate Professor, Head, Department of Medicine (Unani), Mohammadia Tibbia College & Assayer Hospital, Malegaon, Maharashtra, India

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For Correspondence:

Mohd Tarique, Assistant Professor, Department of Medicine (Unani), Mohammadia Tibbia College & Assayer Hospital, Malegaon, Maharashtra, India.

Abstract

Baraṣ (Vitiligo) is a chronic depigmentary disorder marked by well-demarcated glossy white macules due to selective loss or dysfunction of melanocytes, causing significant psychosocial morbidity despite being nonfatal. The review traces historical descriptions of *Baraṣ* across Unani, Ayurvedic, GrecoRoman and other classical texts, and synthesizes classical Unani concepts including *Fasād al-Dam, Balghami Ghalīẓ*, weakness of *Quwwat-i Mughayyira/Mushabbiha/Dāfi'a*, and prognostic signs such as bleeding on pricking with contemporary pathophysiological models (autoimmune, neural, oxidative stress, genetic susceptibility and convergence theory). Clinical classification parallels modern nosology (nonsegmental, segmental, mixed), while Unani subtypes (*Baraṣ*-i *Uẓmā*, *Baraṣ-i Muntashir*) emphasize depth and chronicity of humoural involvement. Diagnosis relies on clinical examination complemented by Wood's lamp, targeted laboratory screening for associated autoimmune disorders (notably thyroid disease), and, when indicated, biopsy. Therapeutic strategies integrate Unani principles *Tanqiya-i Badan, Ta'dili Mizāj, Islāhi Haḍm*, topical *Jālī/Muḥammir/Musakhkhin* agents, and regimental therapies with evidencebased modern treatments such as topical corticosteroids, calcineurin inhibitors, NBUVB phototherapy, and surgical grafting for stable lesions. The review highlights areas of concordance (sun exposure/psoralen use historically and in PUVA), identifies limitations in prognostication, and advocates for rigorous clinical trials to evaluate Unani single and compound formulations alongside conventional modalities. Integration of traditional Unani approaches with modern dermatological care may offer a holistic, patientcentred pathway for managing *Baraṣ*, but requires standardized protocols and safetyefficacy data.

Keywords: Vitiligo, *Baraṣ*, Unani Medicine, PUVA

1. Introduction

Vitiligo is a common, acquired disorder of skin and mucous membranes characterized by the selective loss of functional melanocytes, resulting in clearly defined white patches or macules on the skin. [1] It can appear anywhere on the body, but is most frequently seen on the face, hands, genitals, and around body openings. Sometimes, friction in areas such as elbows, ankles, and neck can trigger vitiligo—this is known as the Koebner phenomenon.[2] Vitiligo is generally classified into two major types: Segmental Vitiligo, which features unilateral, band-shaped patches in specific segments, and Non-Segmental Vitiligo, which involves bilateral patches often concentrated on the face and limbs or scattered symmetrically across the body. Occasionally, both types may occur together, but the segmental form tends to respond poorly to treatment.[3,4]

Worldwide, vitiligo affects an estimated 0.5% to 2% of people.[5] These numbers vary depending on factors like geography, culture, and social awareness. Areas where vitiligo is more visible and stigmatized tend to report higher prevalence, especially in the Indian subcontinent, where rates can reach as high as 8.8% in some regions.[6] Conversely, a large population study on Bornholm Island in Denmark reported a prevalence of only 0.38%.[7] Vitiligo can develop at any age but typically emerges during periods of active growth, with 70-80% of cases occurring before the age of 30.[8,9] It can even be present from birth or appear later in life. [10] Some studies report a higher incidence among females, whereas others find more males affected.[9] Current evidence suggests that vitiligo affects males and females similarly,[11] though females often seek medical help more frequently due to social stigma. More recent modelling estimated physician- or dermatologist-diagnosed lifetime prevalence at 0.36% in the general population, 0.67% in adults, and 0.24% in children, underscoring the need to interpret older prevalence estimates cautiously.[61,65]

2. Historical Background and Terminology

The concept of ideal skin tone has existed in the societal structure since the beginning of time and has played an

important part in the perception of people.[12] The problem of skin discolorations has existed since centuries ago. It can be traced back as far as 2200 BC when, during the *Āshoorī Ṭibb* period in Iran, there were reports of *Baraṣ*, which stands for "white or yellow patches."[2] *Baraṣ* actually means "white skin" in Arabic language. There are instances in the Qur'an mentioning this kind of disorder where it was stated in *Surah Āl Imran* (Surah 3:49) and *Surah Al-Ma'idah* (Surah 5:110) that Jesus cured the ones affected with vitiligo through God's wish. This Arabic term *Al-abras*, used in the aforementioned Verses, has been translated to mean vitiligo by scholars like Tahar Ben Achour.[3]

There are several ancient literatures that describe skin disorders resembling vitiligo. The second pigmentary disorder listed in the Ebers papyrus written in Ancient Egypt (1500 BC) could refer to vitiligo. But according to the controversy of the translation of the contemporary texts, this assumption cannot be proved.[4] Skin disorders listed in the Indian literature including *Atharva Veda* (1400 BC), *Vinaya Pitika* (224-544 BC) in the Buddhism literature and *Kilasa*, *Sveta Khista*, and *Charka* as white or yellow spots could have occurred on the skin.[5] Similarly, there are other writings mentioning this disease like *Amarakosha* (600 AD), *Manusmriti* (200 BC), and *Charaka Samhita* (800 BC). [6] In addition, some writers have linked vitiligo with the term "*shira-bito*," meaning "white man" in 1200 BC, from the *Nakatomi no harai* of Shinto prayers.[7]

Nonetheless, in most cases, these symptoms could not be differentiated from leprosy and other skin conditions. Notably, even Hippocrates (460-377 BC) did not distinguish between vitiligo and leprosy. [8] In the Bible, there were mentions of these conditions using the terminology "*tzara'ath*." This condition is rendered in Greek translation of the Book of Leviticus (Chapter 13) as "white leprosy." Nevertheless, contemporary religious scholars and dermatologists have asserted that these descriptions most probably referred to vitiligo and other conditions affecting skin pigmentation.[2,8]

The term "Vitiligo" is considered to have originated either from the Latin word "vitium," meaning defect or blemish, or "vitilum," meaning small blemish. The term was first coined by the Roman physician, Celsus, in the 1st century AD, in his book "De Medicina." [9] Vitiligo's understanding was minimal during the medieval period in Europe. By the 16th century, the physician of Italian origin, Hieronymus Mercurialis, wrote about vitiligo in his book "De Morbis Cutaneis" and concluded that the disease results from the collection of phlegm under the skin layer, a conclusion akin to that drawn by the Persian physician, Zakariyya Rāzī. [10,11] In Korea during the 17th century, vitiligo and some other disorders of the skin, including tinea versicolor, nevus depigmentosus, and albinism, were all considered as one hypo-pigmentary disorder called vitiligo in the book Dongui Bogam.[12]

Novelty and contemporary scope:

This review updates the historical account of *Baraṣ* by placing classical Unani descriptions alongside contemporary disease classification, pathogenesis, diagnostic practice, and treatment recommendations. Importantly, recent clinical evidence includes a randomized non-inferiority trial of Unani formulations versus PUVAsol, while newer international guidance and therapeutic studies provide a current framework for interpreting these traditional approaches. The review therefore emphasizes where historical Unani concepts are concordant with modern observations and, equally, where traditional claims remain hypothesis-generating because robust comparative evidence is limited.[54,63,64,66]

A few accomplishments worth mentioning occurred in later years. One of them is when French doctor Claude Nicolas Le Cat in the 18th century described how a new lesion may appear on an intact skin because of a trauma, which became known as Koebner phenomenon. At the turn of the 19th century, *Moriz Kaposi* from Vienna found out that vitiligo results from the absence of pigmented granules.[13,14]

Revival in interest in modern treatment of vitiligo occurred in 1947 when Professor A. Medhat El-Mofty of Egypt employed methoxsalen (8-methoxypsoralen), which is extracted from various plants including *Psoralea corylifolia* (*Babchi*) and *Ammi majus* (*Atrilal*) along with sunlight in curing the disorder. Around the same period, Khellin, which does not belong to the group of psoralens, was found equally effective when employed along with sunlight. [15] In 1959, Dr. Aaron Lerner discovered that there is balance between the agent responsible for lightening and the pigmenting hormone secreted by the pituitary gland.[13]

By the late twentieth century, vitiligo was increasingly recognized as a progressive depigmenting disorder and characterized by white patches characterized by loss of melanocytes, with multifactorial causes. Its pathogenesis is now understood as multifactorial rather than attributable to a single mechanism. The only factor that is common between all the theories is the presence or absence of melanocytes. Some popular theories of causality include the neurogenic, self-destruction, and autoimmune theories.

3. Unani Concept of Vitiligo (*Baraṣ*)

Ancient Unani scholars recognized *Baraṣ* (Vitiligo) as a distinct skin disorder characterized by white patches due to depigmentation. However, the causes were described differently by various Unani scholars. Some of the important causes described in different classical Unani literature are as follows:

In his treatise *Firdaws al-Ḥikma fi'l Ṭibb*, Rabban Ṭabarī (810–895 CE) identified *Fasād al-Dam* (corruption of the blood) as the primary cause of *Baraṣ* (vitiligo/leucoderma). He also regarded weakness of the *Quwwat-i Hāḍima* (digestive faculty) as an important contributing factor. According to his theory, *Baraṣ* results from the combined effects of blood corruption, coldness of the blood, and an excess of *Balgham* (phlegm), whereas an excess of *Sawdā'* (black bile) is responsible for the development of *Bahaq-i Aswad*. Regarding prognosis, Rabban Ṭabarī stated that the

condition was considered curable if blood appeared after pricking the affected area; however, the absence of blood indicated an unfavourable prognosis and the prognosis was regarded as unfavourable.[13]

According to *Kitab al-Hāwī*, Zakariyya Rāzī stated that *Baraṣ* is due to the phlegmatic modification of *Laḥm* (flesh) and this leads to the modification of blood and prevents the nourishment of tissues, leading to the absence of colouring of skin and sometimes of hair and flesh underneath. He also noted the view held by *Shamūn* that an excess consumption of foods with much moisture or water may cause *Baraṣ*. [14] He also outlined several prognostic indicators for *Baraṣ* under different conditions:[14]

When rubbing the affected skin produces a hyperaemic reaction (redness from increased blood flow), the disease is generally considered curable.

If the patches are limited in extent and display a reddish or yellowish tint, recovery is usually faster. In contrast, when the condition is widespread and the patches appear milky white or cloudy, it is regarded as incurable.

If, upon pricking the affected area, a whitish fluid emerges instead of blood, the prognosis is unfavourable; the presence of blood, however, indicates a chance of cure.

Lesions located on the head and feet are especially slow to respond to treatment.

Ahmad bin Muhammad Ṭabarī, in his *Moʿālijāt-i Buqratiyyā* work, categorized the disease *Baraṣ* in two different types during the 10th century AD. In *Baraṣ-i Uẓmā* the disease was identified as a severe condition where the corrupted humors entered deep into the body and influenced the bones, thus making it tough or almost impossible to be treated. The other form was relatively a mild form involving just the area between skin and bone. For the identification of the disease, he said one should prick the diseased skin; the white colour fluid indicates *Baraṣ-i Uẓmā*.[15]

The stated objectives of this historical therapeutic approach include getting rid of toxins in the body, tempering the nature of the body, and adhering to a proper dietary regime for producing good blood. It recommends that one consumes food such as young kid meat, chicken, and chick instead of milk and dairy products.[16]

Kāmil al-Ṣanāʿa al-Ṭibbiyya by Al Majūsī (930 – 994 AD) has documented the condition of *Baraṣ* as the bleaching of the skin and hair, either local or all-over the body. The cause was excess phlegm in the blood, which weakened the *Quwwat-i Mughayyira* (colour regulating force). Another observation made by him was that the presence of white discharge from the diseased area implied an incurable condition, while bleeding indicated a curable one.

According to Al Majūsī, foods that cause the production of phlegm, like milk, fresh fish, and moist and cold foods, should be avoided, after which medicines would be used to get rid of the extra phlegm in the body.[17]

Baraṣ was characterized by Ibn Sīnā in *Al-Qānūn fi'l Ṭibb* as a gradual whitening of the skin and body that may even affect the bones. This condition was attributed to deficiency of the *Quwwat Mushabbiha* (colour-producing power) and the *Quwwat Dāfiʿa* (waste elimination power) because of an excess of harmful, thick substance called *Mādda-i Ghalīẓ*. Thus, the alteration of tissue nutrition results in pigmentation defects in the skin.[18]

Ibn-e-Hubal (1122–1213 AD) discussed *Baraṣ* in his book "*Kitāb al-Mukhtārāt*" as being an affection in which skin whitens due to a person having a cold and moist disposition along with the presence of thick phlegm (*Balgham-i Ghalīẓ*). He suggested that treatment be done early with the help of purgatives, hot and dry diet, and avoiding cold and moist foodstuffs.[19]

H. Jurjānī (12th century AD), in his classic work *Dhakhīrā Khawārẓam Shāhī*, described *Baraṣ* in the same way as Ibn Sīnā, attributing its cause to the weakness of the *Quwwat Mughayirra* (the faculty responsible for transformation) and *Quwwat Dāfiʿa* (the faculty of expulsion). This weakness, he said, arises from the excessive accumulation of thick phlegm (*Balgham-i Ghalīẓ*).[20] Daūd Antākī 1541–1599, also expressed a similar opinion regarding both the cause and treatment of the disease in his book, Tadhkirā ūlil-Albāb.[21]

Hakīm Akbar Arzānī (17th century), in *Ṭibb-i-Akbar*, and Hakīm Āʿẓam Khān, in *Iksīr-i-Āʿẓam*, described *Baraṣ* as a whiteness appearing on the outer surface of the skin and sometimes involving internal organs. In some cases, it spreads across the entire body and is then referred to as *Baraṣ-i Muntashir* (generalized vitiligo). When the condition becomes chronic (*Muzmin*) and keeps progressing, they acknowledged that treatment becomes extremely difficult. They also mentioned, much like Zakariyya Rāzī, that if the affected area looks shaggy, light white, turns red after rubbing, and bleeds when pricked, it offers a hopeful prognosis and can be treated.[22,23]

Abūl MH Qamārī defined *Baraṣ* as a whiteness of the skin and its hair caused by a sticky form of phlegm (*Lesdār Balgham*) mixed with the nourishing blood of the muscular parts. He emphasized that dietary irregularities are an important contributing factor. For treatment, he advised inducing vomiting for a few days at the beginning, followed by purgatives (*Mushil*) along with hot and dry foods (*Hār Yābis Aghdhiya*).[24]

Similarly, Sadīduddīn Gazrūnī, in *Al-Sadīdī*, described *Baraṣ* as white patches on the skin mainly resulting from weak digestion (*Ḍuʿf-i-Hazm*), and further quoted Ibn Sīnā's earlier account of the condition. [25]

In summary, *Baraṣ* is a disease that causes whitening of skin due to loss of pigmentation, which could spread to deeper layers like bones. The disease was caused due to the weakness of *Quwwat Mughayirra, Mushabbihā*, and *Dāfiʿa* due to the buildup of thick phlegm causing blood to get corrupted and blood to become cold (*Fasād al-Dam* and *Barūdat al-Dam*). Lesions with reddish or

yellowish discoloration or bleeding lesions were considered curable; however, lesions on the head and legs were difficult to cure.[13,15,17,18,20,23,25]

Recently, the Central Council for Research in Unani Medicine (CCRUM) has described *Baraṣ* in its standard treatment guidelines as a depigmentary disorder characterized by glossy white patches of the skin, sometimes extending to deeper tissues like muscles and bones. The condition is attributed to a *Su' Mizāj Bārid* (cold morbid temperament) of the affected area along with predominance of phlegm, which weakens the transformative faculty (*Quwwat Mughayyira*). In some cases, it even appears at sites of *Hijāmā* (cupping therapy). Hair in the affected area typically turns white, and pricking these patches may cause oozing of a whitish fluid.[26]

4. Etiopathogenesis

Vitiligo is a multifactorial skin disorder that arises from the interaction of several biological and environmental factors rather than a single causative mechanism. The convergence theory is widely recognized as the most comprehensive explanation for its pathogenesis, proposing that genetic susceptibility, autoimmune dysfunction, oxidative stress, neural factors, biochemical alterations, and environmental influences collectively contribute to the development of the disease. Oxidative stress and certain chemical exposures can damage melanocytes, increasing their vulnerability to destruction. This process is further intensified by immune dysregulation, particularly the activation of autoreactive $CD8^+$ T lymphocytes and a decrease in regulatory T cells, which promote melanocyte loss. Genetic variants associated with immune function and melanocyte survival also increase the likelihood of disease development. Furthermore, defects involving neural signaling, melanocyte stem cells, and biochemical pathways may influence both the initiation and progression of vitiligo. Overall, current evidence indicates that vitiligo results from the complex interaction of multiple pathogenic mechanisms acting together to produce depigmentation. [27,28] [29] Contemporary reviews further support a central role for oxidative stress, innate and adaptive immune activation, and genetic susceptibility, with autoreactive CD8+ T cells contributing to melanocyte destruction.[55–57,59]

5. Classification of Vitiligo

Baraṣ, known as vitiligo, according to the author, Ahmad bin Rabban Tabrī in his work *Mu'ālajāt-e-Buqrāṭiyya*, exists in two categories; the first is where the morbid humor (*Ruṭūbat-i fasidā*) has penetrated too deep inside the tissues and bones making it hard to treat, while in the second category it stays between the skin and bones. [15,18] Akbar Arzāni defined *Baraṣ*-i-*Muntashir* (universal vitiligo) as a disease due to debility of the Quwwat-Mughayirra (alterative faculty). He emphasized that its treatment became extremely difficult once it turned into a chronic problem.[23]

On the other hand, according to modern international consensus [3,30,63], vitiligo is broadly classified into three major categories: Non-segmental vitiligo (often referred to simply as "vitiligo"), Segmental vitiligo, and Mixed vitiligo.

Vitiligo classification recognizes several types: the common non-segmental form, the segmental type marked by distinct patch patterns and hair whitening, mixed forms combining both, and small localized patches that are still waiting for clearer classification as doctors learn more. This classification helps in understanding and managing the disease better.[30] [31]

Table 1. Classification of vitiligo.

Type of vitiligo	Subtypes	Remarks
Non-segmental (NSV)	Focal, mucosal, acrofacial, generalized, universal	Subtyping may be useful for clinical description and epidemiologic studies.
Segmental vitiligo (SV)	Focal, mucosal, unisegmental, bi- or multisegmental	Distribution patterns can be further described, although terminology is not fully standardized.
Mixed (NSV + SV)	According to the severity and distribution of SV	The segmental component is often clinically prominent.
Unclassified	Focal at onset; multifocal asymmetrical non-segmental; mucosal (one site)	May require longitudinal reassessment as the phenotype evolves.

Source: adapted from the classification framework cited in the manuscript and updated in accordance with contemporary consensus terminology.

6. Clinical Features of Vitiligo

The majority of Unani scholars defined *Baraṣ* as white spots that appear on the skin; according to Akbar Arzānī, these spots can affect the entire body's skin, starting with their little size. [23] The *Baraṣ* lesion could or not, and it is because to *Maddā-i Raddiya* and *Ghalīẓ Ruṭūbāt*, respectively, that they are smooth, lustrous, and delicate to the touch. *Ruṭūbāt-i-Fāsida* can occasionally cause a reddish-coloured lesion.[15]

The first sign of *Baraṣ* (Vitiligo) is typically a well-defined, milky white, depigmented macule that ranges in diameter from a few to several millimetres and that the patient may not detect. [32] Any part of the body may have lesions. but are usually seen on sites of stretch and pressure. The vitiligo lesions may occur in the areas subjected to trauma or friction, as an outcome of Koebner's phenomenon. [9] In generalized vitiligo, both sides of the body with symmetrical pattern, usually involved. [32] Because of the hyperpigmentation on the periphery, the macules typically have a round to oval form and slightly distinct margins.[32]

Within a vitiligo lesion, hypopigmented and normal-coloured patches can occasionally coexist with depigmented areas. The name "trichrome vitiligo" is frequently used to characterise this pattern; however, different degrees of hypopigmentation can result in trichrome, quadrichrome, or pentachrome vitiligo.[9,33] Therefore, "multichrome vitiligo" is the recommended phrase to describe these kinds of patterns.[32]

Typically, vitiligo does not impact hair and leaves it coloured, but in certain instances, it can cause grey or whitish hair. For instance, localised whitening of the scalp and eyebrows typically results in localised depigmentation of hair, but occasionally, complete whiteness of the scalp hair may occur. Since ancient times, depigmented body hair within vitiligo lesions has been regarded as a poor indicator of regimentation.[14,32] Darker-skinned patients are easier to spot vitiligo patches on than those with extremely pale skin.[32]

7. Prognosis of Vitiligo

Clinical assessments are limited to evaluating disease activity because there are currently no reliable laboratory markers for prognosis.[34,35]

As mentioned previously, Zakariyya Rāzī identified several clinical signs to help predict how vitiligo might progress, he further stated: [14]

When the lighter patches are limited in size and have a reddish or yellowish tint, recovery tends to be faster.

In contrast, widespread patches that look milky white or cloudy are generally considered difficult to treat.

Lesions on the scalp or feet tend to respond more slowly to therapy.

Tabrī also noted that bleeding after pricking a lesion signals a favorable prognosis, while the absence of bleeding is a negative sign. [13] Other factors linked with poor outcomes include, patches inside the mouth or other mucosal surfaces, the presence of white hair inside lesions,[36] a family history of vitiligo, concurrent autoimmune diseases or antithyroid antibodies,[37] nutritional deficiencies, digestive problems, and ongoing psychological stress, whether mental or emotional.[38]

8. Diagnosis of Vitiligo

At present, there are no dependable laboratory tests that can predict how vitiligo will progress or what the long-term outcome will be. For this reason, physicians primarily assess the disease activity through clinical observation. Wood's lamp examination is highly useful for identifying the areas affected in people with fair skin as well as the light-coloured palms and soles of people with darker complexions.[32] Current international recommendations likewise emphasize clinical assessment, disease activity, extent, subtype, and treatment goals when selecting management strategies.[58,63]

Knowing how many melanocytes are still alive in the skin helps guide treatment options and predict how the disease may progress. Wood's lamp exam can also reveal the first hints of repigmentation, often seen at the edges of a patch or around hair follicles.[32]

8.1 Recommended Diagnostic Procedures in Vitiligo

8.1.1 If Diagnosis Is Certain

Thyroid function is commonly evaluated through tests that measure thyroid stimulating hormone (TSH), anti-thyroid peroxidase (TPO) antibodies, and antithyroglobulin antibodies. In cases where disorders like Graves' disease are suspected, additional investigations such as anti TSH receptor (TSHR) antibodies may also be carried out. When a patient has a history or symptoms suggesting other autoimmune conditions, physicians often extend the workup to include broader autoantibody testing and may involve specialists such as endocrinologists or immunologists to ensure comprehensive management of overlapping autoimmune diseases.[39]

8.1.2 If Diagnosis Is Uncertain

A small round sample of skin is taken using a punch biopsy tool from both the affected and unaffected areas. If needed, further tests like fungal studies or molecular tests to check for lymphoma cells may also be carried out.[39]

9. Differential Diagnosis

If the diagnosis of vitiligo is uncertain, several conditions should be considered in the differential diagnosis. These include chemical- or drug-induced leukoderma, congenital and genetic disorders such as piebaldism and Waardenburg syndrome, post-inflammatory or post-traumatic hypopigmentation, infection-related depigmentation (e.g., leprosy and tinea versicolor), paraneoplastic conditions such as hypopigmented mycosis fungoides and melanoma-associated depigmentation, and idiopathic causes. Other disorders that may mimic vitiligo include nevus depigmentosus, pityriasis alba, idiopathic guttate hypomelanosis, halo nevus, progressive macular hypomelanosis, and amelanotic melanoma. Careful clinical evaluation, supported by relevant investigations, when necessary, is essential to distinguish these conditions from vitiligo and establish an accurate diagnosis.[1,30,40]

10. Treatment Options

The main aim of treating vitiligo is to stop the immune system from damaging the pigment-producing cells (melanocytes) and to encourage these cells to spread back from nearby skin and hair roots. Treatment

options usually fall into three groups—medications, surgical methods, and physical therapies—and in many cases, a combination of these approaches works best. [41]

10.1 Pharmacological Treatment

The management of vitiligo is guided by the extent, location, and activity of the disease. Topical corticosteroids are the first-line treatment for localized lesions, while tacrolimus and pimecrolimus are preferred for sensitive areas such as the face and skin folds because they do not cause skin atrophy. Calcipotriol may be combined with corticosteroids or phototherapy to enhance repigmentation. For rapidly progressive vitiligo, oral mini-pulse corticosteroid therapy can help halt disease progression. Narrowband UVB (311 nm) is the preferred phototherapy owing to its effectiveness and safety, whereas PUVA is used less often because of its greater risk of adverse effects. The 308-nm excimer laser is another effective option for localized disease. In stable vitiligo that is unresponsive to medical treatment, surgical procedures, including punch grafting, suction blister epidermal grafting, split-thickness skin grafting, and non-cultured epidermal cell suspension transplantation, can achieve durable repigmentation. More recent evidence has expanded the therapeutic landscape to include topical Janus kinase inhibition; phase 3 trials demonstrated efficacy of ruxolitinib cream for facial and total-body repigmentation, and international and Canadian consensus statements now incorporate topical steroids, calcineurin inhibitors, phototherapy, JAK inhibition, systemic approaches, and surgery according to disease characteristics. [58,60,64,67,68] [42]

10.2 Uṣūl-i- ʻIlāj (Unani Principles of Treatment)

In the Unani system of medicine, the treatment of *Baraṣ* (Vitiligo) is based on a few key principles. These include *Tanqiya-i Balgham* (cleansing the body of excess phlegm), *Ta'dil-i Mizāj* (restoring balance of temperament), and *Islāh-i Haḍm* (improving digestion). For local management, Unani scholars recommend applying medicines with cleansing (*Jālī*), rubefacient (*Muḥammir*), and warming (*Musakhkhin*) properties. These are usually given in the form of *Ṭilā'* (liniments), *Ḍimād* (pastes), or *Roghan* (medicated oils). Recent evidence is encouraging but remains limited: a randomized non-inferiority trial found that a Unani regimen was not inferior to PUVAsol in its per-protocol analysis, whereas later reviews have emphasized the need for larger, rigorously designed trials and standardized formulations.[54,66]

ʻIlāj bi'l Tadbīr (Regimental therapies) are also advised. One such practice is *Dalk Khashin*, where the affected skin is rubbed regularly with a rough cloth to stimulate it. *Nafsiyātī Tadbīr* is also advised, which involves supporting patients and their families by reassuring them that vitiligo is not contagious, while also helping the patient build self-confidence and cope positively with the condition.[26,43]

In terms of prevention (*Tahaffuẓ*), patients are cautioned against cautery (*Al-Kayy*), excessive sweating through hot baths, indulgence in frequent sexual activity, and the use of alcohol (except in very limited medicinal forms).[26]

ʻIlāj bi'l Ghidhā' (Dietary management) plays a central role as well. Warm-natured foods are recommended, while cold and moist foods are to be avoided.[15,24]

ʻIlāj bi'l Dawā' (Pharmacological treatment) usually begins with *Tanqiya'-i-Badan* (cleansing the body of harmful substances). This involves three steps: first, administering *Munḍij-i-Balgham* (concoctives of phlegm) until signs of ripening (*Nuḍj*) appear; second, giving three purgatives (*Mushil-i-Balgham*) in alternation with three cooling agents (*Tabrīḍ*).[26,44]

After *Tanqiyā-i Badan, Ta'dil-i Mizāj* (Correction of morbid temperament) should be performed by using drugs of hot temperament. *Adwiya-i Muḥammira* and *Adwiya-i Lādhiʻa* should be used locally to provide strength to *Quwwat Ghādhiya* by giving it *Tehrīk*. Sometimes *ʻAmal-i -Kayy* can be used to treat *Baraṣ*. [44] Buqrāṭ (Hippocrates) and Ibn-i Sarābiyūn advised that once the body has been cleansed through *Tanqiya* (evacuation/ purification), attention should then be given to strengthening and regulating the digestive system by using light and easily digestible foods.[45] Zakariyya Rāzī, one of the great Unani physicians, also emphasized that exposing the affected patches to sunlight can be highly beneficial.[14]

10.3 ʻIlāj (Treatment):

For centuries, numerous single and compound formulations have been used for vitiligo in the Unani system of medicine.

Single Drugs

Aṭrilāl (Ammi majus), Bābchī (Psoralea corylifolia, Chāksū (Cassia absus), Ḥabb al-Nīl (Ipomoea nil, Kherbaq Siyāh (Helleborus niger), Panwār (Cassia tora), Zanjabīl (Zingiber officinalis) Qust (Saussurea lappa), Shītraj (Plumbago zeylanicum), Anjīr dashtī (Ficus hispida).[46–48] Contemporary reviews of herbal medicine in vitiligo also support continued investigation of plant-derived agents while emphasizing the limitations of available clinical evidence.[62]

Compound Formulations

Oral Formulations: *Majūn Aṭrilāl, Iṭrīfal-i Kabīr*, (semi-solid preparations), *Ḥabb-i Ayāraj, Ḥabb-i Baraṣ, Ḥabb-i Sakbīnaj,*

Topical Formulations: *Ṭilā-i Hindī, Ṭilā-i Baraṣ (*liniments*), Safūf-i Hindī, Safūf-i Baraṣ, Safūf-i Kāla Bichua (*powdered formulations*), Roghan-i Baraṣ (*oil*), Marham-i Baraṣ (*ointment*), Ḍimād-i Baraṣ (*poultice*), Ḥabb-i Hindī* (pills) [17,49–53]

A 2025 case report described repigmentation in a child with poliosis associated with vitiligo following Unani treatment; however, case-report evidence cannot establish general efficacy or safety.[69]

11. Conclusion

Baraṣ (Vitiligo) is a pigmentary disorder. Although vitiligo is not life-threatening, it greatly influences the

psychological, social and emotional well-being of patients, particularly those with dark skin tones. Although there is no universally curative therapy, there are numerous options that can be considered to prevent its spread and encourage repigmentation of the affected skin areas. The Unani system of medicine provides for a holistic approach in treating *Baraṣ* by way of *Munḍij wa Mushil* therapy together with topical applications with properties such as *Jālī* (detergent), *Muḥammir* (rubefacient), *Muḥallil* (resolvent) and *Musakhkhin* (warming agents). There are a large number of single (*Mufrad*) and compounded (*Murakkab*) medicines of Unani system that have proven to be very useful in treating this chronic and stubborn disease. Additionally, the application of herbal treatments, proper diet, modification of lifestyles and psychological counselling play an important part in the successful treatment of this ailment. The integration of traditional medicines, particularly Unani, Ayurveda, Siddha, Homoeopathy and Yoga with conventional treatment can prove to be a safe and effective multidisciplinary treatment of *Baraṣ* (Vitiligo) in this era. The available evidence supports presenting Unani interventions as complementary or investigational options rather than as established substitutes for evidence-based dermatological care. Recent clinical and consensus literature also highlights the importance of shared decision-making, disease-activity assessment, and individualized treatment selection. [54,63,64,67]

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