



BARAŞ (VITILIGO) IN UNANI MEDICINE AND CONVENTIONAL MEDICINE: AN OVERVIEW

Mozakkir Husain^{1*}, Qamar Uddin² and Munawwar Husain Kazmi³

¹PG Scholar, Department of Moalajat (Medicine), National Research Institute of Unani Medicine for Skin Disorders (NRIUMSD), Hyderabad-500038, India.

²Professor & HOD, Moalajat (Medicine), National Research Institute of Unani Medicine for Skin Disorders (NRIUMSD), Hyderabad-500038, India.

³Professor & HOD, Iilmul Advia (Pharmacology), National Research Institute of Unani Medicine for Skin Disorders (NRIUMSD), Hyderabad-500038, India.

***Corresponding Author: Mozakkir Husain**

PG Scholar, Department of Moalajat (Medicine), National Research Institute of Unani Medicine for Skin Disorders (NRIUMSD), Hyderabad-500038, India.

Article Received on 16/05/2020

Article Revised on 04/06/2020

Article Accepted on 24/06/2020

ABSTRACT

Baraş (Vitiligo), sometimes referred to as leukoderma (from the Greek words leuco meaning white and derma meaning skin) is an acquired, chronic depigmenting disorder of the skin and/or mucosa, characterized by milky white, non-scaly macules and/or patches with distinct margins, and caused by destruction of melanocytes in lesional skin. According to Unani Medicine, Baraş (Vitiligo) is a white discoloration of the skin, which is caused by the weakness of Quwwat Mughayyira (transformative faculty), cold impaired temperament of organs, or it may be congenital. About 1-2% of the world's population, or 40-50 million people, have vitiligo. Vitiligo appears to be more common in people with certain autoimmune diseases. These patches are more common in sun exposed areas, including hands, feet, arms, face, and lips. Other common areas for white patches to appear are the armpits, groin, around mouth, eyes, nostrils, navel, and genitals. Vitiligo generally appears in one of three patterns. In one pattern (focal pattern), depigmentation is limited to one or only a few areas. Some people develop depigmented patches on only one side of their bodies (segmental pattern). But for most people who have vitiligo, depigmentation occurs on different parts of the body (generalized pattern). In addition to white patches on the skin, people with vitiligo may have premature greying of the scalp hair, eyelashes, eyebrows, and beard.

KEYWORDS: Baraş, Leukoderma, Unani, Segmental Vitiligo, Non-segmental vitiligo.

INTRODUCTION

Baraş (Vitiligo) is a common, acquired, idiopathic discoloration of the skin characterized by well-circumscribed, ivory/ chalky white colored macules, which are flush to the skin surface in contrast to leukoderma. The lesion may be surrounded by a ring of tan intermediate colour around which is the normal skin, 'the trichrome'. The hair over the patch may be either normal or white (leukotrichia).^[1] Celsus was the first to use the term vitiligo in his Latin medical classic *De Medicina* during the second century BCE.^[2] The name is believed to derive from the Latin *vitium*, meaning defect or blemish^[3] rather than *vitellus*, meaning calf.^[4] Typical vitiligo lesions can be defined as milky white, non-scaly macules with distinct margins. *Baraş* (Vitiligo) occurs worldwide, with a prevalence of 0.1% to 2.0% in the United States, the estimated incidence is 1%.^[5] In India, the incidence among dermatology outdoor patients is estimated to be between 3-4%. The incidence of *Baraş* (Vitiligo), as reported by various workers in different cities of India can be between 2.9% in Goa to 8.8% in

Delhi. However, most authors say that its incidence is around 4%, which is however, definitely more as compared to the world's population of 1%.^[6] This is a common condition, in which, completely white patches develop due to melanocyte destruction. It is probably an autoimmune disease.^[7] Vitiligo commonly begins in childhood or young adulthood, with peak onset of 10-30 years, but it may occur at any age. All races are affected, and both sexes are equally afflicted. A female preponderance has been reported, but the discrepancy has been attributed to a presumed increase in reporting of cosmetic concerns by female patients. Although familial clustering of cases is commonly seen, inheritance occurs in a non-Mendelian pattern. Approximately 20% of patients with vitiligo have at least one first degree relative with vitiligo, and the relative risk for first degree relatives of vitiligo patient is increase by 7- to 10-fold.^[5]

Concept of baraş (vitiligo) in unani medicine

Baraş (Vitiligo) has been discussed in detail in the classics of Unani Medicine. According to Unani

Medicine, Baraş (Vitiligo) is a white discoloration of the skin, which can appear anywhere in the body, but mostly occurs on hands and feet. Sometimes it occurs in few organs, sometimes it affects all organs. When it involves most of the body's skin, it is known as Baraş Muntashir (extensive vitiligo). Thus, the whole body becomes white.

According to Ibn Sīnā (Avicenna) (980-1037) in his medical encyclopaedia, *Al-Qānūn fi'l Ṭibb*, the perfectness of the tissue metabolism depends on four factors, including Quwwat Jādhiba (absorptive faculty), Quwwat Māsika (retentive faculty), Quwwat Mughayyira (transformative faculty) & Quwwat Mushabbiha (power of resemblance), and Quwwat Dāfi'a (expulsive faculty).^[8,9] Quwwat Jādhiba is the power which serves for the absorption of food. Quwwat Māsika is the power that retains the nutrients at tissue level, so that they may be well-integrated with the tissue. Quwwat Mughayyira and Quwwat Mushabbiha are the powers that bring changes and shape the nutrients into tissue power. Quwwat Dāfi'a is the power that excretes waste material from the tissue level and throws into the bloodstream for final decomposition and excretion from the body. Ibn Sīnā says that depigmentation occurs due to defects in the function of Quwwat Mushabbiha (power of resemblance) at the tissue level.

‘Ali ibn ‘Abbās Majūsī (930-994AD) in his famous book, *Kāmil al-Ṣanā'a al-Ṭibbiyya* says^[10], Baraş occurs due to domination of phlegmatic humour in the blood, and weakness in Quwwat Mughayyira (transformative faculty) in the organ. There is white discoloration of the skin, even the hair also turns white. On puncturing the skin with a needle, if, white fluid oozes in spite of blood, then there is no chance of cure; if blood or reddish fluid oozes, then there is no hopelessness for the cure. When Baraş (Vitiligo) becomes chronic, the treatment is difficult. The primary step in the treatment of this disorder is to restrict the intake of phlegm forming foods such as milk, fish and cold and wet foods. Besides, the patients should be given honey and such purgative drugs, which may expel Balgham (phlegm), like Ḥabb-i-Ayārij and M'ajūn made from Ghāryaqūn, Shaḥm-i-Ḥanzal, Ḥabb al-Nīl, Turbud, etc.

Zakariyya Rāzī (Rhazes) (850-925AD) in his famous book *Kitāb al-Hāwī fi'l Ṭibb* has given a comprehensive description of this disease.^[11] According to him, if white patches of Baraş (Vitiligo) do not turn red on rubbing or when, instead of blood, white fluid comes out on pricking them, the possibility of recovery is low and vice versa. If white patches are limited and non-extensive and the colour of the patches is yellowish or reddish, then early cure can be expected. Conversely, when Baraş (Vitiligo) is extensive and spreading and where the affected areas become bloodless and colour of the patches is cloudy, it is incurable. He also mentioned that the patches on the feet and head do not respond to treatment adequately.

Aetiology and pathogenesis

Vitiligo is a multifactorial, polygenic disorder, with a complex pathogenesis that is not yet well understood. Of various theories of disease pathogenesis, the most accepted is that genetic and nongenetic factors interact to influence melanocyte function and survival, eventually leading to autoimmune destruction of melanocytes. Other suggested explanations have included defects of melanocyte adhesion, neurogenic damage, biochemical damage, auto cytotoxicity and others.^[12] According to Unani Medicine, *Baraş* (Vitiligo) is caused by the weakness of *Quwwat Mughayyira* (transformative faculty), failure of *Quwwat Mushabbiha* (power of resemblance), cold impaired temperament of organs, or it may be congenital.

Epidemiology

Baraş (Vitiligo) is the most common depigmenting disorder. The largest epidemiological study was done in 1977 on the island of Bornholm in Denmark, where vitiligo was described to affect 0.38% of the population.^[13] The prevalence of vitiligo is often referred to as 0.5-1% of the world's population.^[14] Although the exact prevalence is difficult to estimate, the rates are as high as 8.8% in India, because these data referred to the prevalence of patients with vitiligo within one skin institute in Delhi.^[15] This high value could be due to the inclusion of cases with chemically-induced depigmentation. Overall, the highest incidence rates have been recorded in India (up to 8.8%), followed by Mexico (2.6-4%), and then Japan (1.68%). The disparity between prevalence and incidence data could be due to high reporting of data; places where social and cultural stigma are common, forcing patients to seek early consultation, or where lesions are more prominent in dark skinned populations.^[16] Adults and children of both sexes are equally affected, although women and girls often present for treatment more frequently, possibly because of the greater negative social effects for affected women and girls than for men and boys.^[17] Non-segmental vitiligo (NSV) develops at all ages, but usually occurs in young people between the ages of 10 years and 30 years.^[18] Childhood-onset vitiligo (before age of 12 years) is reported to be common and affects 32-37% of patients.^[19] compared with previously reported 25%.^[20] Non-segmental vitiligo (NSV) can occur at any age, whereas segmental vitiligo (SV) tends to occur at a young age, before the age of 30 years in 87% of cases and before the age of 10 years in 41.3%. Segmental vitiligo accounts for 5-16% of overall vitiligo cases.^[21] The proportion of patients with positive family history varies from one part of the world to another. In India, in particular, it ranges from 6.25-18%. In some studies, it is as high as 40%. The mode of transmission of vitiligo is quite complex. It is probably polygenic with a variable penetrance.^[22]

Classification of baraş (vitiligo)

On the basis of the polymorphic distribution, extension, and number of white patches, vitiligo is classified into

generalized (vulgaris, acrofacial, mixed), universalis, and localized (focal, segmental, and mucosal) types.^[23] Vitiligo is also classified as segmental and Non-segmental types, on the basis of distinctive clinical features and natural histories.

1. **Segmental vitiligo (SV):** It is characterized by macules having unilateral dermatomal distribution that does not cross the midline. It generally affects young children and typically remains localized, the depigmented lesions persisting unchanged for many years.^[12]
2. **Non-segmental vitiligo (NSV):** It includes all cases not classified as segmental, including localized, generalized, and acrofacial.
3. **Vitiligo vulgaris:** In this type, multiple scattered lesions are distributed in a more or less symmetrical pattern. It is the most common presentation of Generalized Vitiligo (GV)
4. **Acrofacial vitiligo:** It affects the distal end of fingers and facial orifices in a circumferential pattern. It is a subtype of GV.
5. **Mixed vitiligo:** It is a combination of acrofacial and vulgaris, or segmental and acrofacial types.
6. **Vitiligo universalis:** It is a complete or nearly complete depigmentation of the whole body. It is the most severe form of NSV.
7. **Focal vitiligo:** It is characterized by the presence of one or few macules in one area but not distributed in a segmental pattern. It is considered a precursor form of GV.
8. **Mucosal vitiligo:** It is a term reserved for depigmentation of mucous membrane alone.

Clinical features of *baraş* (vitiligo)

Vitiligo is characterized by the appearance of patchy discoloration evident in the form of typical chalky-white or milky macules. The macules are round and/ or oval in shape, often with scalloped margins.^[24] The size of the macules may vary from a few millimetres to several centimetres, with the lesions affecting the skin and/ or mucous membranes. By and large, the lesions are asymptomatic although itching/ burning may precede or accompany the onset of the lesions in a few patients.^[25] Vitiligo is a slow and progressive disease and may have remissions and exacerbations correlating with triggering events.^[26] Occasionally, the lesions of vitiligo may begin to form around a pigmented nevus (Sutton's Nevus/ Leukoderma Acquisitum Centrifugum) and then go on to affect distant regions.^[27]

Associated diseases

Amongst autoimmune diseases, the strongest association is with thyroid disease, including hypothyroidism and hyperthyroidism. Systemic disorders like, diabetes mellitus, pernicious anaemia, Addison's disease, lymphoma, leukaemia, human immunodeficiency virus (HIV) infection, and Sjogren's syndrome are a few of the diseases associated with vitiligo.^[28, 29] Autoimmunity and

immune responses are of paramount significance in vitiligo.^[30]

Cutaneous associations

Cutaneous associations of vitiligo are important as they commonly provide circumstantial indication to its possible aetiopathogenesis. Halo nevus, lichen planus, alopecia areata, leukotrichia, and premature greying of hair are frequently reported associations.^[31] Of these, leukotrichia (poliosis) is found in up to 45%, premature greying of hair (canities) in 37%, followed by halo nevus in 35% and alopecia areata in up to 10% of cases.^[22] Rarely, other skin disorders like nevus depigmentosus, dermatitis herpetiformis, giant congenital melanocytic nevus with neurotization, chronic urticaria, polymorphic light eruption and malignant melanoma have also been recorded in association with vitiligo.^[32] Moreover, psoriasis vulgaris confined to vitiligo patches and occurring contemporaneously in the same patient has recently been described. Stress is associated with vitiligo in many patients.^[33]

Differential diagnosis

Differential diagnosis of *Baraş* (Vitiligo) includes several dermatoses like, nevus depigmentosus, pityriasis alba, pityriasis versicolor, post-inflammatory hypopigmentation, tuberous sclerosis, idiopathic guttate hypomelanosis, Waardenburg syndrome, systemic sclerosis, borderline tuberculoid leprosy^[34], chemical leukoderma, and melanoma-associated leukoderma.^[35]

Diagnosis

The diagnosis of vitiligo is based essentially on clinical examination, because the lesions have a typical appearance. However, if the lesions are not distributed in the pattern of classical vitiligo, confusion with other hypomelanotic disorders can arise. Inspection with the aid of a Wood's light can then be helpful. The presence of a family history of vitiligo, the Koebner phenomenon, leukotrichia or associated autoimmune disorders such as thyroid disease can help to support a clinical diagnosis of vitiligo.^[36]

Management of *baraş* (vitiligo) in conventional medicine

Current treatments for vitiligo are largely unsatisfactory, as the aetiopathogenesis of vitiligo remains poorly understood. First-line treatments consist of topical treatments (corticosteroids and calcineurin inhibitors). Once daily application of potent topical corticosteroid preparations (e.g., 0.1% betamethasone valerate and 0.05% clobetasol propionate) is recommended, but should preferably be applied in a discontinuous scheme (e.g., 15 days per month for 6 months) to avoid local side-effects (skin atrophy, telangiectasia, hypertrichosis, acneiform eruptions and striae). The use of topical calcineurin inhibitors (tacrolimus or pimecrolimus) mainly for the face and neck is an alternative to topical steroids. Twice daily applications are recommended, initially for 6 months.^[37]

Second-line treatments consist of phototherapy (NB-UVB and psoralen and UVA [PUVA]) and systemic steroid treatment. Treatment with phototherapy is effective in some cases. NB-UVB (311 nm) phototherapy is at least equally effective as PUVA, with fewer side-effects because of intake of psoralens.^[38] Targeted phototherapies can be used for localised vitiligo, e.g., Excimer Lamps or Lasers (peak at 308 nm). No consensus exists as to the optimum duration of phototherapy. Irradiation will most often be stopped, if no repigmentation occurs within the first 3 months of treatment, even though repigmentation sometimes starts later on. Oral Mini-Pulse (OMP) of moderate doses of betamethasone or dexamethasone (2.5mg/day) for 3-6 months can be considered in fast spreading vitiligo to stop progression.^[39]

Third-line treatments consist of surgical grafting techniques and depigmenting treatments. Surgical methods are planned as a therapeutic option in patients with segmental vitiligo and those with non-segmental vitiligo with stable disease for at least 1 year after recognized non-response of medical interventions and absence of Koebner's phenomenon. Only a few patients are therefore suitable for these interventions. The surgical techniques that are mentioned in the European guidelines consist of tissue grafts (full-thickness punch, split-thickness, and suction-blister grafts) and cellular grafts (cultured melanocytes and non-cultured epidermal cellular grafts). The three tissue grafting methods seem to have much the same success rates of repigmentation. Additionally, cellular grafting techniques were, in general, equally effective, although the percentages of repigmentation were slightly inferior to the tissue grafts.^[40] However, important advantages of cellular grafting are the possibility of treating large areas and better cosmetic results than with tissue grafts.^[41] Furthermore, adverse events appear to be less frequently related with cellular grafts than with punch grafting, followed by split-thickness grafting.^[42] Depigmenting treatment of residual areas of pigmentation should only be considered in widespread (>50% body surface area), obstinate, and disfiguring vitiligo, or highly visible recalcitrant facial or hand vitiligo. Skin-bleaching methods reported are monobenzone ethyl ester or 4-methoxyphenol.^[43]

Management of *baraş* (vitiligo) in unani medicine

According to Unani system of medicine, there are four primary methods of treatment. These are *Ilāj bi'l Ghidhā* (Dietotherapy), *Iāj bi'l Tadbīr* (Regimen Therapy), *Ilāj bi'l Dawā* (Pharmacotherapy), and *Ilāj bi'l Yad* (Surgery). *Baraş* (Vitiligo) is a chronic disease and, hereafter, all the Unani physicians are of the view that its treatment should be started with *Tanqiya* (Removal of harmful material from the body) with *Munđij* and *Mushil* (MM Therapy). The goal of *Munđij* and *Mushil* Therapy is to correct the metabolic errors such as humoral imbalance in the body. This therapy is very exclusive which is employed to patients with persistent, chronic,

systemic diseases. There are different types of *Munđij* and *Mushil* therapies which are prescribed after clinical examination of patients and determining the dominating Humour (Khilt) as causative factor. Mostly *Munđij-e-Balgham Advia* are given in the management of *Baraş* (Vitiligo).^[44] The management of *Baraş* (Vitiligo) includes to maintain and balance the deranged Balgham (Phlegm), *Ta'dil-i Mizāj* and use of appropriate ointment locally.^[45] For the treatment of *Baraş* (Vitiligo), various single and compound drugs are used like, *Atrilāl* (*Ammi majus*)^[46], *Babchi* (*Psoralea corylifolia*)^[47], *Waj* (*Acorus calamus*), *Gandhak Amlasar* (Sulphur), *Geru* (Silicate of alumina & oxide of iron)^[48], *Habb-i Baraş*, *Habb-i Hindi*^[49], *Safūf Baraş*^[50, 51], etc.

CONCLUSION

Vitiligo affects millions of people, regardless of their ethnic background. Advances in medical and surgical treatments have been made, but there is no cure for vitiligo. It is psychologically disturbing especially in the dark skin. The treatment of vitiligo depends upon the duration of vitiligo and whether it is localized or generalized. If more than two-thirds of the body is affected by vitiligo, then it is better to bleach the whole body. The patients most likely to respond are those of recent onset, and who have lesions on the face. Long standing vitiligo with white hair is most unlikely to be cured. Lips and finger tips have a poor response to treatment because of the absence of hair follicles.

Adverse effects of conventional treatment are more common, which have led to search of new alternatives. A better option for the management of vitiligo may be Unani Therapy. The advantage of Unani therapy compared with conventional therapy is that it is more effective in vitiligo and does not produce major adverse effects as evidenced through various clinical studies. Moreover, it also improves the patients' quality of life.

Until further advances are made, there should be a holistic, multi-disciplinary approach to the management of vitiligo, which includes information about the disease, cosmetic camouflage, as well as referral to psychotherapy and appropriate alternative therapies, which are promising for patients afflicted with this traditionally stigmatizing and challenging condition, like Unani therapy.

REFERENCES

1. Sehgal VN (2010). Textbook of clinical dermatology, 5th edition; Jaypee Brothers Medical Publishers, New Delhi; pp. 107-110.
2. Nair BK. Vitiligo, a retrospect. *Int J Dermatol*, 1978; 17: 755-57.
3. Koranne RV, Sachdeva KG. Vitiligo. *Int J Dermatol*, 1988; 27: 676-81.
4. Panda AK. The medico historical perspective of vitiligo (Switra). *Bull Indian Inst Hist Med Hyderabad*, 2005; 35: 41-46.

5. Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz S (2003). Fitzpatrick's Dermatology in General Medicine, 6th edition, McGraw-Hill Professional, New York, pp. 953-960.
6. Datta K and Mandal SB (1969). A clinical study of 650 vitiligo cases and their classification. *Ind. J. Dermatol.* 14: 103-111.
7. Burton JL (1989). *Essentials of Dermatology*, 2nd edition, Churchill Livingstone, London, pp. 29-31.
8. Unani. National Health Portal of India. Therapeutic approaches and treatment modalities. [Cited 2020 May 6]. Available from: https://www.nhp.gov.in/unani_mty.
9. Ibn Sīnā (1906). *Al-Qānūn fi'l Ṭibb* (Urdu Version), Vol.4, Munshi Nawal Kishor Press, Lucknow, pp. 389-391.
10. 'Ali ibn 'Abbās Majūsī (1889). *Kāmil al-Ṣanā'a al-Ṭibbiyya* (Urdu version), vol. 1, Munshi Nawal Kishore Press, Lucknow; p. 196.
11. Rāzī ABMIZ. *Kitāb al-Ḥāwī fi'l Ṭibb* (Arabic version) Vol. 23. Dairatul Marif, Osmania University, Hyderabad, 1970; pp. 72-75.
12. Lowell Goldsmith, Stephen Katz, Barbara Gilchrest, Amy Paller, David Leffell, Klaus Wolff (2012). *Fitzpatrick's Dermatology in General Medicine*, 8th Edition, Vol. 1, McGraw-Hill Professional, p. 792.
13. Howitz J, Brodthagen H, Schwartz M, Thomsen K. Prevalence of vitiligo. Epidemiological survey on the Isle of Bornholm, Denmark. *Arch Dermatol*, 1977; 113: 47-52.
14. Ezzedine K, Lim HW, Suzuki T, et al. Revised Classification/ Nomenclature of vitiligo and related issues: the Vitiligo Global Issues Consensus Conference. *Pigment Cell Melanoma Res*, 2012; 25: E1-13.
15. Behl PN, Bhatia RK. 400 cases of vitiligo. A clinic therapeutic analysis. *Indian J Dermatol*, 1972; 17: 51-56.
16. Sehgal VN, Srivastava G. Vitiligo: compendium of clinico-epidemiological features. *Indian J Dermatol Venereol Leprol*, 2007; 73: 149-56.
17. Singh M, Singh G, Kanwar AJ, Belhaj MS. Clinical pattern of vitiligo in Libya. *Int J Dermatol*, 1985; 24: 233-35.
18. Ezzedine K, Diallo A, Leaute-Labreze C, et al. Pre- vs. post-pubertal onset of vitiligo: multivariate analysis indicates atopic diathesis association in pre-pubertal onset vitiligo. *Br J Dermatol*, 2012; 167: 490-95.
19. Nicolaidou E, Antoniou C, Miniati A, et al. Childhood- and later-onset vitiligo have diverse epidemiologic and clinical characteristics. *J Am Acad Dermatol*, 2012; 66: 954-58.
20. Halder RM, Grimes PE, Cowan CA, Enterline JA, Chakrabarti SG, Kenney JA Jr. Childhood vitiligo. *J Am Acad Dermatol*, 1987; 16: 948-54.
21. Hann SK, Lee HJ. Segmental vitiligo: clinical findings in 208 patients. *J Am Acad Dermatol*, 1996; 35: 671-74.
22. Behl PN, Aggarwal A, Srivastava G. Vitiligo In: Behl PN, Srivastava G, editors. *Practice of Dermatology*. 9th ed. CBS Publishers: New Delhi; 2003. pp. 238-241.
23. Hann S-K, Nordlund JJ: Clinical features of generalized vitiligo. In: *Vitiligo*, edited by S-K Hann, JJ Nordlund. London, Blackwell Science, 2000, p. 35.
24. Shwartz RA, Janniger CK. Vitiligo. *Cutis*, 1997; 60: 239-44.
25. Arata J, Abe-Matsuura Y. Generalized vitiligo preceded by a generalized figurate erythematous-squamous eruption. *J Dermatol*, 1994; 21: 438-41.
26. Goudie RB, Spence JC, Mackie R. Vitiligo patterns stimulating autoimmune and rheumatic diseases. *Lancet*, 1979; 2: 393-5.
27. Fisher AA. Differential diagnosis of idiopathic vitiligo from contact leukoderma. Part II: Leukoderma due to cosmetics and bleaching creams. *Cutis*, 1994; 53: 232-4.
28. Sehgal VN, Rege VL, Mascarenhas F, Kharangate VN. Clinical pattern of vitiligo amongst Indians. *J Dermatol*. Tokyo, 1976; 3: 49-53.
29. Kim YC, Park HJ, Cinn YW. Phakomatosis pigmento vascularis type IIa with generalized vitiligo. *Br J Dermatol*, 2002; 147: 1028-1029.
30. Sehgal VN, Srivastava G. Vitiligo: Auto-immunity and immune responses. *Int J Dermatol*, 2006; 45: 583-590.
31. Lee D, Lazova R, Bologna JL. A figurate papulosquamous variant of inflammatory vitiligo. *Dermatology*, 2000; 200: 270-4.
32. Shin JH, Kim MJ, Cho S, Whang KK, Hahm JH. A case of giant congenital nevocytic nevus with necrotization and on set of vitiligo. *J Eur Acad Dermatol Venereol*, 2002; 16: 384-6.
33. Midelfart K, Moseng D, Kavli G, Stenvold SE, Volden G. A case of chronic urticaria and vitiligo, associated with thyroiditis, treated with PUVA. *Dermatologica*, 1983; 167: 39-41.
34. Halder RM, Grimes PE, Cowan CA, Enterline JA, Chakrabarti SG, Kenney JA Jr. Childhood vitiligo. *J Am Acad Dermatol*, 1987; 16: 948-54.
35. Verma S, Kumar B. Contact leukoderma of the scalp or an unusual variant of vitiligo. *J Dermatol*, 2001; 28: 554-6.
36. Van Geel N, Speeckaert M, Chevolet I, et al. Hypomelanoses in children. *J Cutan Aesthet Surg*, 2013; 6: 65-72.
37. Taieb A, Alomar A, Bohm M, et al. Guidelines for the management of vitiligo: the European Dermatology Forum consensus. *Br J Dermatol*, 2013; 168: 5-19.
38. Westerhof W, Nieuweboer-Krobotova L. Treatment of vitiligo with UV-B radiation vs topical psoralen plus UV-A. *Arch Dermatol*, 1997; 133: 1525-28.
39. Singh A, Kanwar AJ, Parsad D, Mahajan R. Randomized controlled study to evaluate the effectiveness of dexamethasone oral mini-pulse therapy versus oral minocycline in patients with

- active vitiligo vulgaris. *Indian J Dermatol Venereol Leprol*, 2014; 80: 29-35.
40. Njoo MD, Westerhof W, Bos JD, Bossuyt PM. A systematic review of autologous transplantation methods in vitiligo. *Arch Dermatol*, 1998; 134: 1543-49.
 41. Van Geel N, Goh BK, Wallaey S, De Keyser S, Lambert J. A review of non-cultured epidermal cellular grafting in vitiligo. *J Cutan Aesthet Surg*, 2011; 4: 17-22.
 42. Falabella R. Surgical treatment of vitiligo: why, when and how. *J Eur Acad Dermatol Venereol*, 2003; 17: 518-20.
 43. Kim YJ, Chung BS, Choi KC. Depigmentation therapy with Q-switched ruby laser after tanning in vitiligo universalis. *Dermatol Surg*, 2001; 27: 969-70.
 44. Hakīm Muhammad A'zam Khān (1885). *Iksir-i-A'zam* (Persian version). Munshi Nawal Kishore Press, Lucknow, 4: 475-487.
 45. Qamri MH (2008). *Ghinā Munā*, CCRUM, New Delhi; p. 556.
 46. Anonymous (1987). *Standardisation of Single Drugs of Unani Medicine Part-1*, CCRUM, Ministry of Health & Family Welfare, Govt. of India; pp. 1-7.
 47. Anonymous (2007). *The Unani Pharmacopoeia of India, Part-1, Vol-1*, Dept. of AYUSH, Ministry of Health & Family Welfare, Govt. of India, pp. 13-14.
 48. Ghani N. *Khazain-ul-Advia, Idāra Kitāb al-Shifā'*, Darya Ganj, New Delhi, pp. 401, 723, 1146.
 49. Anonymous (2006). *National Formulary of Unani Medicine*, Dept. of AYUSH, Ministry of Health & Family Welfare, Govt. of India, Part IV, 1st Edition, pp. 10-11.
 50. Kabīruddīn M (2006). *Al-Qarābādīn*, CCRUM, New Delhi, 2nd Edition, pp. 222: 1141-42.
 51. Jīlaniī G. *Makhzan al-Murakkabāt wa Muallam Dawāsāzī*, Islāmīa Setum Press, Makkī Darwāza, Lahore; pp. 158-159.