JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

A Comprehensive Review and Recent Highlights of *Nār-Farsī* (Eczema) in the Aspect of Unani Conception

Anjum Rasool [1], Zaffar Hussain [*2], Arsha Noor [1], Arshid Iqbal [3], Sabreena Bashir [4]

1. P. G. Scholars, Dept. of Moalajat, Regional Research Institute of Unani Medicine, Srinagar.
2. Professor, HOD, Dept. of Moalajat, Regional Research Institute of Unani Medicine, Srinagar.
3. MD. Coordinator, Dept. of Ilmul Advia, Regional Research Institute of Unani Medicine, Srinagar.
4. P.G. Scholar, Dept. of Dermatology, National Institute of Unani Medicine, Banglore.

Abstract: *Nār-Farsī* is a provocative or agitating reciprocation of skin which is identified by erythema, excoriation, dryness, exudation, blistering, pruritus etc which can either be acute or chronic. Prevalence of *Nār-Farsī* is estimated to 2 to 10% in developed countries. According to *Ibn-e-Abi Sadique NārFarsī is* named because *Naar* means '*Aag*' Patient feels more irritation and burning sensation in those lesions ,Farsi means it was more common among people of *Persia* (Mulk Faras)or referred to the physician who treat the *Nār-Farsī* first also was resident of *Persia* .It is caused by *safravi madda* (bilious matter) admixed with *damvi madda*(sanguineous matter). Nonetheless many theories are bound to demonstrate etiology but elementally two factors causing eczema are described, firstly an allergic or sensitive skin and secondly exposure to an irritant. Diagnosis is based on clear signs and symptoms, apart from this a clinical diagnostic criteria is used named Hannifin and Rajka's criteria and also some specific investigations are available.

Keywords: ***Nār-Farsī*, Eczema, *safravi madda***

INTRODUCTION

Eczema is known by different names such as ***Chajjan, Akota, Nar-farsi*** in Unani system of medicine.[1] But its Indian name is ***Chambal***.[2] The term Eczema comes from Greek word means to boil out (ec-out, zema-boil) because in Eczema skin is boiling out or oozing out. The term Dermatitis also comes from Greek word for inflammation of skin. Among some people Eczema and dermatitis are synonyms while among other people Dermatitis implies an acute condition and Eczema is a chronic one, so it is clear that all eczema are Dermatitis but not all Dermatitis are eczema.[3] In modern text book of dermatology *Nar-farsi* is described as an inflammatory response of the skin to multiple agents characterized by erythema, odema, vesiculation, oozing, crusting and lichenification.[4] The eczema is non-infectious and non contagious inflammatory dermatosis.[5] *Ali-bin-Abbas Majoosi, Muhammad Ibn Zakariya Razi and Ismaiel jurjani* in their respective compilation stated that in this skin disease, the liquid vesicles appear on the skin with intense burning sensation and itching.[6] *Majoosi* described *Nār-farsi* under the symptoms of *chichak* (small pox). He explained that if *chickak* is admixture of *sadded* (pus) and *khoon* (blood) and resembles the vesicles of boil the condition is known *Nār-farsi.*[7]

ETIOLOGY

Unani Scholars have described the cause of *Nar-farsi* are mixing of *khilt-e-safra* into blood, indigestion, general weakness, nerve weakness, arthritis, gout, intestinal worms , incomplete evacuation and extreme hot and cold.[8] According to *Unani* physician *nar-farsi* is a skin disease where rashes at the site of lesion occur and show peacock shaped linear flame of fire. Vesicle formation occurs after some time along with irritation and severe Itching occurs. In later stages these vesicles crack, rupture and change into dry state and finally crust formation and lichenification occurs.[9] *Razi* stated that there is burning sensation in *Nār-farsi* with pruritus after that blister is formed and fill with dilute substance.[10]

As per modern physician oedema is a characteristic change between the cells of epidermis known as spongiosis, leading to formation of vesicles. There is increased keratin layer and the whole epidermis become thickened.[11] Eczematous patients usually present with a history of allergy in the form of hay fever, asthma, allergic rhinitis.[5]

EPIDEMIOLOGY

Epidemiological studies play an essential character in representing the risk factors of eczema, as comprehensive prevalence and incidence data can reveal the burden of disease in the population of adults, adolescents, and children in different geographical regions.

Global epidemiology of eczema:

Eczema affects about one-fifth of all individuals during lifetime, but the prevalence of the disease varies greatly throughout the world. Currently there are 10-20% of children experiencing eczema. Other study demonstrates that the prevalence of eczema is estimated to be 10-30% in children. This disease was considered as a "children disease" for a long time. In early childhood, the incidence of disease is same in both sexes, and only around the age of 6 years the prevalence among girls become higher than in boys (3:2). Eczema most often commences in prior childhood. It is accepted that 60% of all cases commences in the first year of life and 90% earlier the age of 5. The disease tends to relapse before 5 years of age in 40-80% of patients and in 60-90% it recedes before 15 years of age.[12,13,14]

PATHOGENESIS [15]

Ali bin Rabban al Tabri described that in this disease ghilzat (increased viscosity of khilt) develops in the skin along with inflammatory condition and erythematous rashes which doesn't spread into deeper layers of skin rather spreads locally accompanied by severe pain. Therefore, after disappearance of the lesions, its effect remains because of dissolution (*tahleel*) of *raqeeq khilt* and *ghaleez khilt* remain at the site of lesion.

CLASSIFICATION [16]

In the classical literature according to shape of lesion and presence and absence of fluid in them the *unani* Scholars have classified *Nār-farsi* as

1. *Sada Nār-farsī* (simple eczema)
2. *Surkh mayal Nār-Farsī* (reddish eczema)
3. *Mutaqeeh Nār-Farsī* (pustular eczema)
4. *Abladar Nār-Farsī* (blustering eczema)
5. *Shiqaqi Nār-Farsī* (fissured eczema)
6. *Sulb Nār-Farsī* (hard eczema).

Further it is classified as *haad* (acute) and *muzmin* (chronic) [17]

In modern medicine eczema has been classified in the following manner depending upon the type of lesion:

- Acute phase: erythema, edema, vesiculation, oozing, crusting
- Sub acute: hyperpigmentation, scaling and crusting
- Chronic: lichenification

Nowadays eczemas are classified for practical use into 2 broad groups:

Exogenous eczema :

A. Irritant contact eczema
B. Allergic contact eczema
C. Photosensitive eczema
D. Infective eczema.

Endogenous eczema:

A. Atopic eczema
B. Seborrheic eczema
C. Nummular eczema
D. Asteatolic eczema
E. Stasis eczema

CLINICAL FEATURES [3]

ACUTE ECZEMA:

This is characterized by plaque which are ill defined, erythematous and oedematous surmounted by papules and vesicles which on rupturing show oozy look. Exudates dry to form scaly, crusts and spongiosis.

CHRONIC ECZEMA:

There may be less exudation, prominent scaling, lichenification and fissuring.

Typical menifestations of Eczema [18]

The clinical appearance of Eczema is often more detailed with a large variation in the morphology and dispersal of eczema combined with other various features. However many patients with Eczema have universal tendency to exist with dry skin (xerosis) due to the low water content and superfluity water loss through the epidermis. The skin is pastel because of enlarged tension in the capillaries and the power to sweat is reduced. There is an increased cholinergic response to scratch, presumed white demographism or skin-writing, steming in hives at the affected site. The palms of hands and feet may manifest hyperlinearity, and the individuals hair is dry and fragile. Frequently there is double skinfold beneath the lower eyelid (Dennie-Morgan fold) that becomes magnified in times of increased disease activity.

Eczema can be grouped into three clinical stages, although these may be difficult to reproduce in the individual person.

- **Eczema of infancy**- Infants experience eczema that is localized to the face, scalp, and extensor aspects of the arms and legs, but it can also be widespread.
- **Eczema of Childhood**- In toddlers and older children, the eczema lesions tend to shift location so that they are often confined to the flexures of the elbows and knees as well as the wrists and ankles, although it can occur at any site. In general the eczema becomes drier and lichenified with excoriations, papules and nodules.
- **Eczema of Adolescence and Adulthood** - In adult patients the lesions frequently localize to face and neck, head and neck dermatitis, and a considerable portion of patients, around 30%, develop atopic hand eczema, which may interfere with workplace activities.[86]

Risk factors for eczema:[19,20,21,22,23,24]

Intrinsic risk factors includes:

- Atopic history of parents
- Filaggrin mutations
- Decreased short chain fatty acids in the gut of children
- Underlying medical conditions such as keratoconus

Extrinsic factors include:[25]

- Low microbial exposure
- Antibiotic exposure
- Urban environment
- Stress
- Pollutants
- Food
- Smoke exposure

Aggravating factors for eczema[26]

Specific exposures are known for worsening eczema and these must be avoided

- Wollen clothes aggrevates itching and discomfort
- Hot water also worsens itching.
- Long baths
- Contact urticaria
- Suspected or documented food allergy.

Complications:[27]

Patients with AD are more vulnerable to viral and bacterial skin infections because of reduced skin antimicrobial peptides and poor barrier function. Impetigo and eczema herpeticum are the two secondary infections that are most frequently treated in AD patients.

The most common secondary infections that cause dermatitis are caused by Streptococcus pyogenes or Staphylococcus aureus. Bulla, pustules, and/or yellow crusting are possible symptoms, Methicillin-Resistant S. Aureus (MRSA) infections in AD are more common and create superantigens, which can exacerbate dermatitis.

Differential diagnosis of eczema:[28]

- Seborrheic dermatitis
- Atypical psoriasis
- Scabies
- Dermatophytosis
- Factitious dermatitis
- Ichthyosis
- Actinic prurigo
- Eczema like cutaneous drug eruption

DIAGNOSIS OF ECZEMA:[29,30,31]

As Eczema has different range of manifestations as distribution, severity and presentation, there is not any accepted gold standard diagnostic criteria or laboratory marker. The Hanifin-Rajka Criteria (HRC) are initially developed and most cited diagnostic criteria in studies. This criteria has been used to bring uniformity to the diagnosis. The American Academy of Dermatology (AAD) revised a consensus criteria which was actually developed by Hanifin and Rajika in 1980 for diagnosis of Eczema consisting of three sub-categories of essential, important and associated features.

Another diagnostic criteria which was extensively used in epidemiological research was evolved by UK Working Party in 1994, this criteria was similar to Hanifin and Rajika's criteria however it differs in some aspects like it includes family history, personal history of asthma, visible flexural dermatitis and onset under 2 years of age.

EASI (Eczema Area Severity Index) and SCORAD (SCORing Atopic Dermatitis) also broadly used clinical tool to examine the extent and severity of eczema. It measures objective signs as well as subjective symptoms.

The signs and symptoms of eczema are assessed on the basis of Eczema Area and Severity Index (**EASI**) score and patients oriented eczema measure (**POEM**) questionnaire. This is more feasible in obtaining a quick and simple understanding of severity of disease and responses to medication. A study recommend using **EASI** score with **POEM** questionnaire to assess "Objective and Subjective" diseases severity measument.

INVESTIGATIONS [32]

- **Patch-test**:

Patch test detects type 4 hypersensitivity (delayed type of reaction). This test gives specific clue about the antigen as there are specified antigens for every allergen due to atopy. In this process an allergen is enforced to the back of patient under occlusive dressing and left for 48 hours.

Then the patient is inspected for hypersensitivity reactions (edema, erythema or papulovesicles. This test is carried out by an expert physician. This test is usually done for evaluation of chronic dermatitis.

- **Prick-test**:

Prick test is useful for detection of type 1 hypersensitivity. Atopic patients bear a familial potency to develop abnormal hypersensitivity to common allergen examples include allergic asthma, allergic rhinitis, allergic conjuctivites, atopic eczema, urticaria. Type 1 hypersensitivity reaction is triggered by penetration of antigen through body surface for example skin, respiratory or gastrointestinal tract. This particular antigen combines with antibody on mast cell membrane. Bridging of IgE molecules on the mast cell surface provocates an instant release of vasoactive amines. This response can lead to asthma, eczema, urticaria when IgE levels reach on peak and cause release of histamine and other mediators.

- **IgE level in serum:**

It is useful to measure IgE level particularly when the usual presentation of acute eczema absent mainly when the distribution of eczema is atypical and there are no other phase of atopic illness. It gives pillar to clue about specified environmental allergens for example horse dust mite, pet dander, pollens etc. Levels are elevated according to the severity of disease.

MANAGEMENT:

Victorious treatment of eczema needs methodical, multilateral course that includes education about the condition of disease, skin hydration, pharmacologic therapy and the spotting and elimination of flare factors like allergens, irritants, infectious agents and emotional stressors.

Education intervention

A study from Australia found it important for eczematous patients to give adequate education as it decreases severity of eczema. Educate the patients about the appropriate use of topical therapies, the importance of treatment adherence and about the chronic nature of disease.

Skin-hydration

As there is malfunction of skin barrier in dermatitis which manifests as an increase in transepidermal water loss (TEWL) and increases penetration of allergens and infectious agents which can lead to inflammation and itching because of reduced natural skin lipids and filaggrin deficiency.

Moisturizers in the form of lotions, ointments or creams restore the ablity to retain, absorb and redistribute water.

Treatment:

The functional treatment of Dermatitis is build on amalgam of everyday use of emollients and anti-inflammatory treatment, while escaping contact with provocative allergens and irritants. Anti-inflammatory therapy should be suitably selected to include topical cortisteroids (TCSs) or topical calcineurin inhibitors (TCI) relying on the disease activity.

Management of eczema in Unani medicine :[33,34,35]

Usool-e-illaj (principles of treatment)

- *Izalasabab* (treat the cause)
- *Tanqiya-e-mawad* (for evacuation of bad elements)
- *Mussafiyat-e-dam* (blood purifiers)
- *Mana-e-uffonat-e-jild* (anti-infective)
- *Musakinat-e-jild* (sedative to skin)
- *Mulayanat wa mushilat* in case of constipation
- Bathing and cleaning of lesions

Ilaj-e-*Nar-e-Farsi* (treatment of eczema)

- ***Izalae-e-sabab*:** Treat and remove the cause which is responsible for Nār-Farsī
- ***Tanqiya-e-mawad*(for evacuation of bad elements):** For this purpose joshanda of Sana Makki5gm, Saqmooniya 5gm,HaleelaKabli 5gm, Aaloo Bukhara 5gm can be given to the patient before taking mussafiyat-e-dam
- ***Mussafiyat-e-dam***:

  Single drugs (*mufardat): Gul-e-mundi* (sphaerantusindicus Linn*)*

  Unnab (ziziphusjujuba Mill)

  Shahtra (FumareIndicaPugsley*)*

  Chiraita(swertiaChirayitaRoxb)
- **Compound drugs (murakabat):**

  Qurs Mussafi Khoon, Sharbat-e-Nelofar, Arq-e-Shahtra, Majoon-e-Ushba

***Mana-e-uffonat-e-jild* (sedative to skin) and *Musakinat-e-jild*(anti-infective)**

- Apply *Rasoot* mixed with *Rogan-e-Gul* locally.
- Apply *Sandal*, *Mudarsung* for sedation.
- Apply *Sandal*, *Mudarsung*, Kafoor after mixing in *Arq-e-Gulab* locally.

CONCLUSION

Eczema is an inflammatory, itchy skin condition that develops after a persistent, intermittent, relapsing course. Because of the itching, scratching, and lack of sleep caused by moderate to severe eczema, patients and their close family members experience a considerable reduction in quality of life that is comparable to that of childhood epilepsy or type-1 diabetes. About 40% of instances of eczema carry over into adulthood, which

can affect social and occupational functioning. Patients and their families experience a marked decline in quality of life as a result of the condition, which has major socioeconomic repercussions. So this disease has become a major health problem. Despite of widely available therapies for management of Nar-Farsi definite treatment is still a challenge. Unani Medicine in this regard can provide a safe and effective treatment.

REFERENCES

1. Jilani Ghulam, Makhzan-e-Hikmat Ist edition, Lahore: mercentile press 1923;2:31.
2. Kabiruddin, Tarjuman-e-Kabeer (Urdu translation of Sharah Asbabwaalamat). Hyderabad: Hikmat Book Depot, YNN, 3,256-257.
3. Hunter JAA, Savin JA, Dahl MV. Eczema and Dermatitis: Clinical Dermatology, Third Edition, Blackwell Publishing, 2003, 70-90.
4. Golwalla AF, Golwalla SA. Medicine of students 18th edition, Mumba Empress court 1999,700-702.
5. Behl P, Aggrawal A, Srivasta G, Practice of Dermatology 9th edition New Delhi : CBS Publisher and Distributor, 2004,126-135.
6. Jurjani1, Zakheera Khwarazam Shahi-urdu translation by Khan HH vol. 8. Delhi IdaraKitabul Shifa:2009:18
7. Majoosi AA. Tarjuma Kamilus Sana vol1; Lucknow:Munshi Naval Kishore 1889:430
8. Aleem Shagufta, Amraz-e-Jild, Second Edition, Adabistan publications, 2014, 86-93.
9. Chand Puri Kausar, Mojizul Qanoon, third edition, Qaumi council Barai Farogh Urdu zaban 1998,441.
10. Zakarya RAB Mb. Kitab al Mansoorie Urdu translationary CCRUM: CCRUM Ministry of health and family welfare Govt of India 1991,273.
11. Buxton Paul K. Eczema and Dermatitis: ABC of dermatology, Fourth edition BMJ Publishing ground London, 2003, 17-19.
12. Grize L, Gassner M, Wüthrich B, et al. Trends in prevalence of asthma, allergic rhinitis and atopic dermatitis in 5-7-year old Swiss children from 1992 to 2001. Allergy. 2006;61:556–62.
13. Weber AS, Haidinger G. The prevalence of atopic dermatitis in children is influenced by their parents' education: results of two cross-sectional studies conducted in Upper Austria. Pediatr Allergy Immunol. 2010; 21:1028–35.
14. Spergel JM. From atopic dermatitis to asthma: the atopic march. Ann Allergy Asthma Immunol. 2010;105:99–106
15. Tabri AM. Molaejat buqratiyah (Urdu translation by CCRUM). 1997; II. New Delhi: CCRUM.
16. Kabiruddin, Tarjuma-e- Kabeer (Urdu translation of Sharah Asbab wa alamat). Hyderabad: Hikmat Book Depot, YNM, 3, 256-257.
17. Khanna. N. Illustrated synopsis of dermatology and sexually transmitted disease-4th edition New Delhi. Elsevier. 85
18. Williams HC. Atopic dermatitis. New England Journal of Medicine. 2005;352(22):2314–2366
19. Alkotob SS, Cannedy C, Harter K, et al. Advances and novel developments in environmental influences on the development of atopic diseases. Allergy. 2020;75(12):3077-3086.
20. Hale G, Davies E, Grindlay DJC, Rogers NK, Harman KE. What's new in atopic eczema? An analysis of systematic reviews published in 2017. Part 2: epidemiology, aetiology and risk factors. Clin Exp Dermatol. 2019;44(8):868-873.
21. Boutin RCT, Sbihi H, Dsouza M, et al. Mining the infant gut microbiota for therapeutic targets against atopic disease. Allergy. 2020;75(8):2065-2068.]
22. Venter C, Meyer RW, Nwaru BI, et al. EAACI position paper: Influence of dietary fatty acids on asthma, food allergy, and atopic dermatitis. Allergy. 2019;74(8):1429-1444.
23. Chang YS, Weng SF, Wang JJ, et al. Association between keratoconus and the risk of adolescent- or adult-onset atopic dermatitis. Allergy. 2020;75(11):2946-2948.
24. Kantor R, Kim A, Thyssen JP, Silverberg JI. Association of atopic dermatitis with smoking: A systematic review and meta-analysis. J Am Acad Dermatol. 2016;75(6):1119-1125.e1111

25. Kantor R, Silverberg JI. Environmental risk factors and their role in the management of atopic dermatitis. Expert Rev ClinImmunol. 2017;13(1):15-2
26. F. J. Bath-Hextall, A. J. Birnie, J. C. Ravenscroft, and H. C. Williams, "Interventions to reduce Staphylococcus aureus in the management of atopic eczema: an updated Cochrane review," British Journal of Dermatology, vol. 163, no. 1, pp. 12–26, 2010
27. Fleming P, Yang YB, Lynde C, O'Neill B, Lee KO. Diagnosis and Management of Atopic Dermatitis for Primary Care Providers. J Am Board Fam Med. 2020 Jul-Aug;33(4):626-635. doi: 10.3122/jabfm.2020.04.190449. PMID: 32675275
28. Silvestre Salvador, J. F., Romero-Pérez, D., & Encabo-Durán, B. (2017). Atopic Dermatitis in Adults: A Diagnostic Challenge. Journal of investigational allergology & clinical immunology, 27(2)
29. Williams HC, Burney PGJ, Hay RJ, et al. The U.K. Working party's diagnostic criteria for atopic dermatitis—I. Derivation of a minimum set of discriminators for atopic dermatitis. *British Journal of Dermatology*. 1994;131(3):383–396.
30. Stalder JF, Taieb A, Atherton DJ, et al. Severity scoring of atopic dermatitis: the SCORAD index. Consensus report of the European Task Force on Atopic Dermatitis. *Dermatology*. 1993;186(1):23–31.
31. Hanifin JM, Thurston M, Omoto M, Cherill R, Tofte SJ, Graeber M. The eczema area and severity index (EASI): assessment of reliability in atopic dermatitis. *Experimental Dermatology*. 2001; 10(1):11–18.
32. Silvestre Salvador, J. F., Romero-Pérez, D., & Encabo-Durán, B. (2017). Atopic Dermatitis in Adults: A Diagnostic Challenge. Journal of investigational allergology& clinical immunology, 27(2), 78–88. https://doi. org/10.18176/jiaci.0138
33. Aleem Shagufta, Amraz-e-Jild, Second Edition, Adabistan publications. 2014;86-93.
34. Kirmani Nafees bin Auz, MoalijatSharahAsbab (Urdu translation by Allama Kabeeruddin), New Delhi: Aijaz Publication House. 2012; 3:256-257.
35. Khan DM, Rahman DA. Narfarsi in the light of unani conception: A Review. International Journal of Health Sciences and Research. 2019;11(9): (2249–9571).