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Concise review on atopic dermatitis and Unani medicine

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Abstract
Atopic dermatitis (AD) is a chronic and relapsing disease affecting an increasing number of patients. It is a common inflammatory condition of the skin that is usually seen in childhood, but can onset or persists into adulthood. Atopic dermatitis (AD) has a prevalence of 1%-3% in adults and 10%-20%, in children. Environmental factors or genetic-environmental interactions seem to play a key role in disease progression. AD is often found in individuals with a personal or family history of atopy, including allergic rhinitis, asthma and AD. Signs and symptoms of AD: pruritus, dry skin, edema, excoriations, lichenification and oozing. AD's presentation may vary depending on the patient's age, disease severity and chronicity. Recently, more attention is given to a proactive therapeutic by regular intermittent application of low potency steroids or topical calcineurin inhibitors to prevent new flares. Principle of treatment in Unani medicine is based on multidimensional approach i.e. *Ilaj Bil Ghiza, Ilaj Bil Tadbeer and Ilaj Bil Dawa*.

Keywords: atopic dermatitis, inflammatory condition, skin disease, unani medicine

Introduction
Atopic dermatitis, commonly known as eczema, is a common chronic, relapsing skin disease characterized by pruritus, disrupted epidermal barrier function, and immunoglobulin E-mediated sensitization to food and environmental allergens [1]. Atopic dermatitis (AD) is the most common chronic inflammatory skin disease [2]. Lifetime prevalence of approximately 20% [3]. The prevalence of AE in adults is about 1%-3%, and 10%-20%, in children [4].

Atopic dermatitis affects about one-fifth of all individuals during their lifetime, but the prevalence of the disease varies greatly throughout the world [5].

About 60% of cases will develop within the first year of life [6]. Atopic dermatitis is more common in rural rather than urban areas [7]. This incidence which emphasizes the link to lifestyle and environment factors in the mechanisms of AD [8]. About 50% of patients with severe atopic dermatitis will develop asthma, and 75% will develop allergic rhinitis [9].

Around 50% of all those with atopic dermatitis develop symptoms within their first year of life, and probably as many as 95% experience an onset below five years of age [3].

The causes of AE remain unclear, but are likely to be multi-factorial in nature; involving genetic, socioeconomic, and environmental factors [10]. Environmental factors or genetic-environmental interactions seem to play a key role in disease progression [11].

The disease significantly reduces the quality of life of patients and their families, which leads to serious socioeconomic consequences [12].

One of the most vital elements of taking medical history is identifying potential aggravating factors [9]. Common food allergens triggering AD are milk and milk products, peanuts, eggs, soy, wheat, seafood, and shellfish [13].

Pathophysiology of Atopic dermatitis
The pathophysiology of atopic dermatitis is complex and multifactorial, involving elements of barrier dysfunction, alterations in cell mediated immune responses, IgE mediated hypersensitivity, and environmental factors [14].

Nemeth V *et al*, reported that there is a genetic component to atopic dermatitis. One common mutation has been observed in the gene Filaggrin, a vital gene for skin cell maturity. This gene is responsible for creating the tough, flat corneocytes that form the outermost protective layer of skin [15].

AD shares general features of barrier dysfunction and skin inflammation with other inflammatory diseases of the skin such as psoriasis or allergic contact dermatitis, but a plethora of disease-specific genetic, immunologic and environmental factors have been identified in AD as well [16]. Some study reported that, different dendritic cells subtypes, such as Langerhans cells, inflammatory dendritic epidermal cells and plasmacytoid dendritic cells, play a key role in AE and impact on the mechanisms underlying AE, such as the recruitment of inflammatory cells, T-cell priming, and cytokine and chemokine release. In addition, allergens in combination with bacterial and viral stimuli influence the course and severity of AE [17].

Histopathology
The histopathology seen in atopic dermatitis is non-specific. In the acute phase lesions, characterized by intensely pruritic, erythematous papules, histopathology reveals mild epidermal hyperplasia, infiltrations of lymphocytes and macrophages along the venous plexus in the dermis and intercellular edema of the epidermis (spongiosis) [15]. The histopathology of systemic allergic contact dermatitis is usually characterized by spongiosis. A number of other much less common to rare histological findings have also

been reported, including erythema multiforme-like eruption, vasculitis, urticaria, and miscellaneous groups [18].

Histology of both forms of dermatitis is highly similar to that of allergic contact dermatitis and has no fundamental impact on the diagnosis of AD. Clinically "normal" appearing skin of AD patients contains a sparse perivascular T-cell infiltrate suggesting minimal inflammation [19].

Clinical Features

The clinical phenotype of AD varies with age and may differ during the course of disease [4]. The eczematous lesions may present with acute (oozing, crusted, eroded vesicles or papules on erythematous plaques), subacute (thick and excoriated plaques), and chronic (lichenified, slightly pigmented, excoriated plaques) forms. Furthermore, xerosis and a lowered threshold for itching are usual hallmarks of AD [20].

Pruritus attacks can occur throughout the day and worsen during the night, causing insomnia, exhaustion, and overall substantially impairs quality of life. Three different stages can be distinguished clinically: infancy, childhood and adolescent/adulthood [19].

Diagnosis

Additional diagnostic tests, such as the patch test, prick test, skin biopsy, or blood test, are usually necessary to rule out other diseases or other types of eczema appearing concomitantly with AD [21].

Management

Standard medical treatment with a pharmacologic approach may be necessary if basic treatment fails to control symptoms satisfactorily [22]. Recently, more attention is given to a proactive therapeutic by regular intermittent application of low potency steroids or topical calcineurin inhibitors to prevent new flares [11].

While treating these disease Unani physicians laid emphasis on Tabiyat by which body works physiologically and resists against infections. It is also called Madicatrix naturae. If it is strong, the body functions smoothly; if it is weak, the risk of illness increases. According to the principles and philosophy of Unani Medicine, disease is natural process. Its symptoms are the reactions of the body to the disease and the chief function of the physician is to aid the natural forces of the body [23]. According to principle of treatment remove the cause which is responsible for atopic dermatitis Tanqiya-e-Muwad; for this rationale Turbud 5 gm Sana makki 5gm, Haleela Kabli 5gm, Saqmooniya 5gm, can be given in the form of Joshandah with 20 gm Gulqand to the patient before meal [24, 25].

After tanqiy-e-Mavad Musaffit-e-Dam Advia used like Chiraita Chobchini, Gule Mundi each 5 gm in the form of Joshandah with Itriphal Shahtara 7 gm twice a day.

For soothing and cooling effect on the surface of skin applied Roghan Gul, Roghan Kaddu, Roghan Narjeel with Kafoor [26-30].

Differential Diagnosis

The differential diagnosis for atopic dermatitis includes may eczematous dermatitides including contact dermatitis, fungal infections, seborrheic dermatitis, and drug eruptions. During childhood, the differential may consist of scabies, psoriasis, ectodermal dysplasia, Hyper IgE syndrome, Netherton's syndrome, Wiskott-Aldrich syndrome and other immuno deficiencies, and enzyme deficiencies [8, 9, 10].

Conclusion

Atopic Dermatitis a worst disease; it is an inflammatory response to the skin. Unani physicians have explained the AD in the name of Fasad-Khoon and were well aware about dermatitis and have described the etiological factor, types, pathology, clinical features in detail. Principle of treatment in Unani medicine is based on multidimensional approach i.e. Ilaj Bil Ghiza, Ilaj Bil Tadbeer and Ilaj Bil Dawa.

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