



A COMPREHENSIVE REVIEW OF ATOPIC ECZEMA

Byale Rutuja. B¹, S.N. Nagoba², Sumit Awale³, Asmita Zodage⁴, A.V. Moholkar^{1*}

Department of Pharmaceutics, Channabasweshwar Pharmacy College (degree) Latur-413512

ABSTRACT:

Atopic eczema (AE) is a persistent, inflamed skin condition that typically appears in infancy. The causes of AE are still unknown after many studies, but they are probably complex in nature. The development of diseases appears to be significantly influenced by environmental variables or genetic-environmental interactions play a crucial part in the spread of the illness.

Cuticle hydration should be used as a first line treatment for AEs since it helps to improve barrier function and decrease itching. This may help to minimise the requirement for topical steroid administration.

Atopic dermatitis' acute and subacute lesions are frequently marked by painful, erythematous papules and vesicles with excoriations and a serous discharge. Plaques and papules with excoriations that are lichenified are typical of chronic atopic dermatitis. Patients with atopic dermatitis also have an increased chance of developing skin diseases, such as superinfections caused by bacteria and viruses.

Keywords: Eczema, Atopic Dermatitis, Atopic Eczema, Treatment, Nanoemulsion, Epidemiology

INTRODUCTION:

Atopic eczema, also known as atopic dermatitis, is a persistent, relapsing, itchy, and inflammatory eczematous eruption that typically begins in infancy.

Although the exact origins of AE are unknown, it is likely that a combination of genetic, socioeconomic, and environmental factors is responsible.

For instance, it has been discovered that AE development is related to FLG mutations. Recently, the prevalence of AE has been rising, although the cause of this is still unknown.

According to several researches, environmental variables may have a role in the rise in AE prevalence. Smaller families, higher incomes, better education, emigration from rural to urban areas, and an increase in antibiotic use could all be linked to the decrease in AE.

Recent studies have shown that tobacco smoke in the environment, indoor air pollution, and exposure to allergens outside are all thought to be environmental variables. A significant global public health issue, AE affects 1%–20% of persons globally. AE affects between 1% and 3% of adults and between 10% and 20% of youngsters.

The most typical type of childhood eczema is AE. The prevalence of AE has more than tripled since the 1960s. Uncertainty still exists on the causes of the increased prevalence.

Types of Eczema:

- 1) Atopic Dermatitis
- 2) Contact Dermatitis
- 3) Hand Eczema
- 4) Seborrheic Dermatitis
- 5) Stasis Dermatitis
- 6) Neurodermatitis
- 7) Dyshidrotic Eczema
- 8) Nummular Eczema

EPIDEMIOLOGY:

Over the past three decades, there has been a sharp rise in the prevalence of AD, which now affects 15% to 30% of children and 2% to 10% of adults. About 45% of all cases of AD (early onset AD) start in the first six months of life, 60% in the first year, and 85% before the age of five. Before puberty, more than two thirds of these kids will experience a spontaneous remission. The illness can, however, also develop in adults (late-onset AD as opposed to early-onset AD). Standardised questionnaire data from 486,623 children aged 13-14 years in the international study of asthma & allergies in childhood (ISAAC) suggest that atopic eczema is not just a problem confined to western Europe, with high prevalence found in many developing cities undergoing rapid demographic change.

There is reasonable evidence to suggest that the prevalence of atopic eczema has increased two-to-three-fold over the past 30 years, the reasons of which are unclear. No reliable incidence estimates are available for atopic eczema.

PATHOPHYSIOLOGY OF ECZEMA:

The pathophysiology of atopic dermatitis is complex & multifactorial, involving elements of barrier dysfunction, alterations in cell mediated immune response. Ige mediated hypersensitivity and environmental factors. A number of mechanisms & the cells are thought to be important in atopic eczema & these are reviewed in details elsewhere. Microscopically, the characteristics appearance of eczema is that of excess fluid between the cells in the epidermis (spongiosis). When severe, this fluid eventually disrupts the adjacent cells in the epidermis to form small collections of fluid, which are visible to the naked eye as vesicles. In the chronic phase, atopic eczema is characterised by gross thickening of the epidermis (acanthosis) & an infiltrate of lymphocytes in the dermis.

TREATMENT OF ECZEMA:

AE is a chronic skin inflammation and its symptoms wax and wane with various manifestations. Personalized treatment should be given to the patient based on their age, the degree and extent of their AE, and the location of their lesions. In addition to the primary pharmaceutical treatment for AE, other approaches such cutaneous hydration, identifying and removing aggravating factors, relieving pruritus, and patient education should be taken into account. Flares need to be avoided through a variety of systemic strategies.

In a 1995 report co-authored by a British Association of Dermatology and Royal College of Physicians Working Party, the therapy of atopic eczema in the UK was summarised.

There are three stages: As part of the initial course of treatment, it was necessary to adequately explain the nature of the disease and offer suggestions for avoiding irritants. Emphasis was placed on the importance of using emollients in sufficient amounts and on treating secondary infections quickly.

Topical steroids were emphasised as the backbone of treatment; however, caution was advised regarding the length of treatment, location, and age of the person being treated. Antihistamines were only advised because of their sedative effects. The importance of cognitive behavioural approaches for particular families was also addressed.

Allergen avoidance was referred to as a second-line treatment, along with ultraviolet light therapy administered under a doctor's supervision and dietary interventions like reducing house dust mites.

Short bursts of systemic corticosteroids, cyclosporin A, evening primrose oil, and Chinese herbal remedies were among the third-line treatments (always administered under the supervision of a physician).

Avoiding irritants and probable allergies is part of conventional therapy, as does maintaining skin moisture with fatty emollients. Most frequently, topical immunomodulators and corticosteroids are utilised. In some circumstances, other medications such phototherapy, antimicrobials, antihistamines, and systemic immune suppressive are also a possibility.

Based on the general agreement of a large number of practitioners and patient advocates, these suggestions were made. While many recommendations lacked RCT support, several did. Therefore, it is unknown how many of these suggestions are actually helpful to patients. A double layer of protective bandages (also known as "wet wraps") with or without topical steroids, "newer" once-daily topical corticosteroids like mometasone and fluticasone, and possibly some increased use of potent systemic medications like cyclosporin A are recent developments.

Another AD treatment option is phototherapy. Numerous papers demonstrate how effective phototherapy is for treating mild to severe AD. Currently, psoralen with UVA (PUVA), 311-nm narrowband ultraviolet light B (NBUVB), and ultraviolet light A1 (UVA) are the most often used. The most effective of them has been found to be PUVA, which also offers the longest duration of remission. NBUVB and medium- and high-dose UVA1 are essentially equally effective.^{82–84} Although it has been tested, combining UVA and UVB has been demonstrated to be less effective than UVA at a modest dose¹. The rise in skin cancer growth has led to a decrease in PUVA use despite the drug's great efficacy. Additionally, studies have shown that about 50% of those who receive UVA1 treatment respond to it, and for those who do, the response is transient. More recently, medicated device creams and foams that restore the epidermal barrier's normal function have hit the market. These pharmaceutical devices offer antioxidant, antiprotease, anti-inflammatory, and help restore the natural balance of lipids, one of the reasons for the epidermal abnormalities associated with AD. The medication plays a crucial role in the long-term therapy of AD, extending the interval between relapses, but it differs from moisturisers in that it acts as a single entity rather than as a combination of active chemicals dispersed across inactive ones. Device creams and foams have the potential to moisturize, Furthermore, prescription devices might be more strictly regulated than over-the-counter medications. Furthermore, it can cause new parents considerable stress because nocturnal itching can keep kids up late and keep their parents up as well. Alternative approaches to improve patient and family awareness and education should be included in treatment programmes in addition to conventional ones.

Alongwith conventional therapies, alternative approaches to raise patient and family awareness and education should be included in treatment programmes.

Nanoemulsion/Submicron emulsions (SMEs)/ Mini-emulsions are thermodynamically stable transparent or translucent dispersions of oil and water stabilized by an interfacial film of surfactant and co surfactant molecules having a globule size of less than 100nm. Recently nanoemulsion are frequently used for delivery of vaccine, DNA encoded drug, antibiotics, cosmetics and topical preparations and are administered via various routes like oral, pulmonary, intranasal, and ocular, and transdermal etc. Nanoemulsion are categorized as multiphase colloidal dispersion, and are characterized by its stability and clarity. The dispersed phase typically comprises small particles or droplets, and they have very low oil/water interfacial tension.

CONCLUSION:

AE is more common than it used to be, with prevalence rates today ranging from 1% to 20% of the general population. Environmental and genetic factors may both contribute to AE development. The majority of AE patients experience remissions and sporadic flares as part of a chronic, relapsing disease course. Consequently, managing chronic AE symptoms is still difficult. Adjuvant therapy, regular medical treatment with pharmaceutical agents like topical steroids and topical immunomodulators, and third-line therapeutic alternatives such applying emollients are all available for treating AE. Additionally, it's crucial to avoid triggers and/or aggravators at all times, such as woollen clothing, emotional stress, and uncomfortable weather conditions. Additionally, systemic immunosuppressants such cyclosporine, azathioprine, and mycophenolate mofetil as well as phototherapy should be taken into consideration.

REFERENCES:

1. Bieber T. Atopic dermatitis. *N Engl J Med* 2008; 358: 1483–1494.
2. Williams HC. Clinical practice: atopic dermatitis. *N Engl J Med* 2005; 352: 2314–2324.
3. Leung DY, Bieber T. Atopic dermatitis. *Lancet* 2003; 361: 151–160.
4. Bologna JL, Jorizzo JL, Rapini RP, eds. *Dermatology*. Vol. 1. St. Louis, MO: Mosby; 2003: 2584.
5. Spergel JM, Paller AS. Atopic dermatitis and the atopic march. *J Allergy Clin Immunol* 2003; 112(6 suppl): S118–S127.
6. Miyake Y, Kiyohara C, Koyanagi M, et al. Case-control study of eczema associated with IL13 genetic polymorphisms in Japanese children. *Int Arch Allergy Immunol* 2011; 154: 328–335.
7. Williams HC, Strachan DP. The natural history of childhood eczema: observations from the British 1958 birth cohort study. *Br J Dermatol* 1998; 139:834-9.
2. Friedmann PS. The pathogenesis of atopic eczema. *Hosp Med* 2002; 63:653-6.
3. Pyun BY. Natural history and risk factors of atopic dermatitis in children. *Allergy Asthma Immunol Res* 2015; 7:101-5.
4. Palmer CN, Irvine AD, Terron-Kwiatkowski A, Zhao Y, Liao H, Lee SP, et al. Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis. *Nat Genet* 2006; 38:441-6.

5. Asher MI, Montfort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. *Lancet* 2006; 368:733-43.
6. von Mutius E. Gene-environment interactions in asthma. *J Allergy Clin Immunol* 2009; 123:3-11.
7. Lee JH, Lee HS, Park MR, Lee SW, Kim EH, Cho JB, et al. Relationship between indoor air pollutant levels and residential environment in children with atopic dermatitis. *Allergy Asthma Immunol Res* 2014; 6:517-24.
8. Gruber R, Janecke AR, Grabher D, et al. Lower prevalence of common filaggrin mutations in a community sample of atopic eczema: is disease severity important? *Wien KlinWochenschr* 2010; 122: 551–557.
9. Sampson HA. Food allergy. Part 1: immunopathogenesis and clinical disorders. *J Allergy Clin Immunol* 1999; 103(5 part1): 717–728.
10. Tan BB, Weald D, Strickland I, et al. Double-blind controlled trial of effect of housedust-mite allergen avoidance on atopic dermatitis. *Lancet* 1996; 347: 15–18.

