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A Review on Atopic Dermatitis: Etiology, Clinical Features, Pathogenesis, Diagnosis, and Various Treatments

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ABSTRACT

Atopic dermatitis is a recurrent, chronic inflammatory skin disease which affects children. Dermatitis is derived from the Greek words "derma" (skin) and "itis" (inflammation). It affects 10-20% of adolescents and 1-3 percent of adults over their lifetime are caused by a complicated interaction between relevant genes, the environment of the host, pharmacological aberrations, and immunological variables. Two primary theories have been postulated. The first theory is that the adaptive immune system is out of balance, and the second is that the skin barrier is impaired. There is no specific test for Atopic dermatitis. Topical or systemic treatments are indicated depending on the severity of the Atopic dermatitis. It includes emmolients, corticosteroids, calcineurin inhibitors, phosphodiesterase 4 inhibitors, biological and wet wrap therapy. Once remission has been achieved, proactive maintenance therapy should be used to limit the number of flare-ups.

KEYWORDS: *Atopic Dermatitis, Filaggrin, Flare-ups, Human monoclonal antibody*

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INTRODUCTION

Wise and Sulzberger coined the term Atopic Dermatitis to describe a "puzzling kind of local or broad lichenification of skin. (1) Atopic dermatitis is a recurrent, chronic inflammatory skin disease which affects children. Atopy is a genetic predisposition for producing immunoglobulin E antibodies in reaction to minute levels of common environmental proteins including pollen, house dust mites, and food allergies. Dermatitis is derived from the Greek words "derma" (skin) and "itis" (inflammation). (2)As part of an allergic triad, it can occur with asthma and allergic rhinitis; an estimated 30% of children with atopic dermatitis acquire asthma later in life. (3) A variety of mechanisms contribute to the pathogenesis of Atopic dermatitis, including skin barrier deficiencies, disruption of innate immune responses, adaptive immune response defects with the development of robust type 2 immunity, and changes in the skin microbiome. (4) Atopic dermatitis begins in childhood and progresses from acute lesions affecting the face and dorsal portions of the limbs in infancy to lesions affecting the face, neck, and flexures in older children. Currently, ten to twenty percent of youngsters have Atopic dermatitis. (5) All people with Atopic dermatitis experience pruritus, as well as pain, sleep disturbances, and mental health issues. The severe symptoms and skin lesions can have a serious influence on one's health and quality of life (QOL). (6) In this paper, the fundamentals of Atopic dermatitis epidemiology, genetics, pathophysiology, and management have been examined, as well as evidence of progress in recent decades. (1)

EPIDEMIOLOGY:

Atopic dermatitis affects 10-20% of adolescents and 1-3 percent of adults over their lifetime. (1) In comparison to developing countries, Atopic dermatitis has been more prevalent in industrialized countries. Its frequency is also higher in metropolitan areas than in rural or agricultural areas in developed countries. (7) The International Study of Asthma and Allergies in Children (ISAAC) has revealed the most

important patterns in Atopic dermatitis around the world; Atopic dermatitis is not increasing or has plateaued in countries with the highest prevalence (e.g., the United Kingdom). (8) According to research, greater income, small family size, education, movement from rural to urban areas, and increased antibiotic use are the key supporting factors for Atopic dermatitis. (9) Chan et al. provided a correlation between a mother's stress and the likelihood of Atopic dermatitis in the fetus or newborn child in a recent review. (10)

ETIOLOGY:

Atopic dermatitis development is influenced by a variety of variables. Family history (particularly maternal history) is a substantial predictor of risk, but there appear to be other external factors as well, including insufficient early childhood contact to microbes and high airborne pollution exposure. (11)

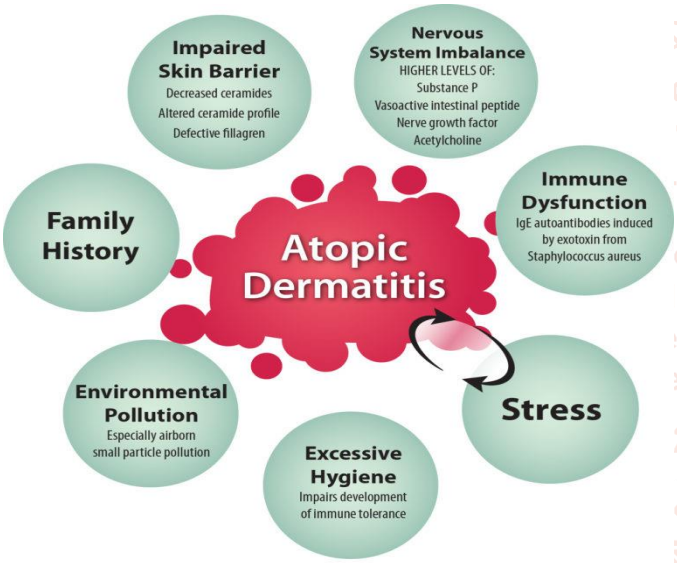

FIGURE 1: Causes of Atopic Dermatitis(11)

PATHOPHYSIOLOGY:

Atopic Dermatitis is caused by a complicated interaction between relevant genes, the environment of the host, pharmacological aberrations, and immunological variables. (12) For the focal lesions in atopic dermatitis, two primary theories have been postulated. The first theory is that the adaptive immune system is out of balance, and the second is that the skin barrier is impaired. These research theories are not mutually exclusive, but they may be complementary. (13)

IMMUNOLOGICAL HYPOTHESIS: Atopic dermatitis is thought to be caused by an imbalance of T cells, notably T helper cell types 1, 2, 17, and 22, as well as regulatory T cells, according to the immunological imbalance theory. Th2 activation of naive CD4+ T cells occurs predominantly in the allergy (atopic dermatitis) state, particularly in acute eczema. This induces an increase in the production of interleukins, especially IL4, IL-5, and IL-13, which leads to an increase in Immunoglobulin E levels and inhibits Th1 differentiation. (13)

SKIN BARRIER HYPOTHESIS: The skin serves as a physicochemical barrier against the physical, chemical, and biological elements that cause Atopic dermatitis. Proksch et al. discussed the crucial components of skin barrier disruption in Atopic dermatitis. (14) A number of molecular risk loci important for skin barrier function have been identified through genome-wide linkage studies. (15)

Three major causes of barrier malfunction in Atopic dermatitis patients have been identified.

- Filaggrin gene expression is reduced - Filaggrin is a vital skin protein involved in the bridge of keratin filament into tight bundle and the hydration of the stratum corneum. Filaggrin gene (encoding filaggrin protein) mutations were found in approximately 50% of Atopic dermatitis patients, and over twenty mutations in the gene have been identified so far. (16-19)
- Ceramide deficit in the skin - Phospholipids are important for water retention in the stratum corneum, the outer layer. Ceramide levels in the stratum corneum had an inverse connection with transepidermal water loss (TEWL) in Atopic dermatitis patients' stratum corneum (20,21)
- Overexpression of epidermal proteases - Kallikrein (KLK) 5, KLK 7, and KLK 14 are the proteases that cause corneodesmosome membrane damage. The effect is pH-dependent and increases when the pH of the stratum corneum is enhanced (22, 23). The lymphoepithelial Kazal-type 5 serine protease inhibitor regulates the activity of these proteases (LEKTI). Serine Protease Inhibitor Kazal type 5 (SPINK5) genes produce the LEKTI. The genetic mutation in SPINK5 has been associated to the onset and exacerbation of Atopic dermatits symptoms (24).

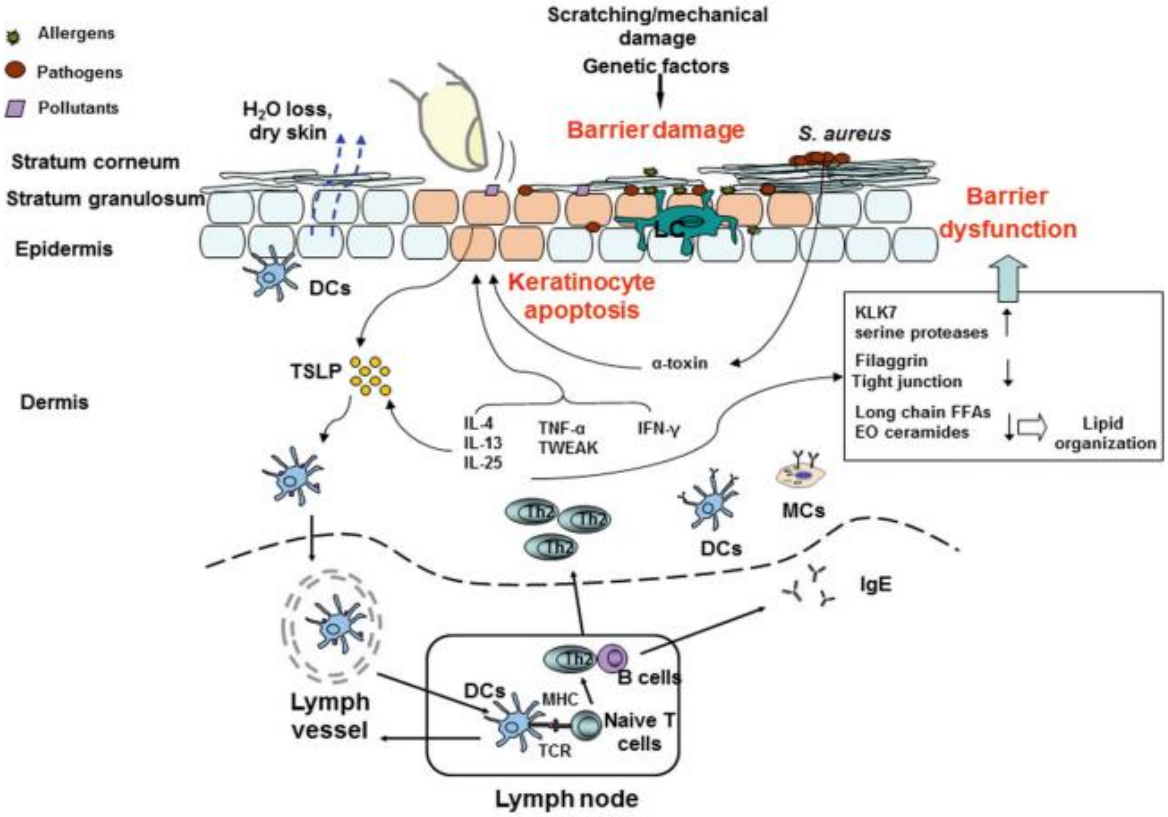

FIGURE 2: Effects of skin barrier on the pathogenesis of atopic dermatitis (25)

Skin barrier degradation is caused by genetic and immunologic factors, as well as biomechanical causes such as scraping, which allows allergens, bacterial and viral antigens, and other environmental elements to contact skin resident antigen-presenting cells. Antigen-presenting cells that have been activated move to lymph nodes and induce naive T cells to become Th2 cells. Increased Th2 cytokines, in combination with TNF-a and IFN-c, degrade epidermal barrier functions by promoting keratinocyte death, decrease tight junction function, and increase Th2 responses by increasing epithelial cell TSLP expression. Furthermore, infections that colonize the skin, such as Staphylococcus aureus, degrade barrier function by releasing virulence factors that cause keratinocyte mortality and increase Th2-type inflammation. Genetic and immunologic variables work together to cause epidermal barrier disruption and play a key role in the etiology of Atopic dermatitis. (25)

GENETIC FACTORS: A genetic deficiency in the filaggrin protein, which disrupts the epidermis, is thought to be the cause of atopic dermatitis. As a result of this disturbance, antigens from the surrounding factors come into touch with immune cells in the dermis, causing acute stinging, rubbing, and irritation. The itchy scratch cycle describes how scratching can cause further damage and swelling of the epidermal skin barrier. (26)

CLINICAL FEATURES:

Clinical features of AD can be categorized into three as follows

- Acute-(erythema, vesicles, bullae, weeping, crusting)
- sub acute - (scaly plaques, papules, erosions, and crusts)
- chronic- (lichenification, scaling, and hyperpigmentation or hypopigmentation).(4)

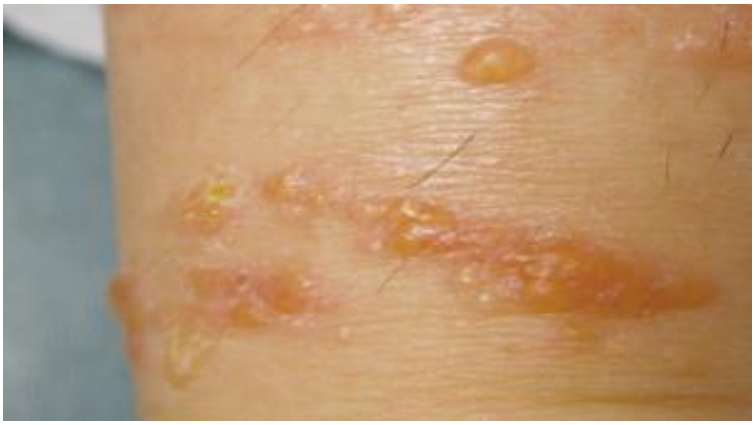

Figure 3: Acute atopic dermatitis in its weeping, blistering form. (3)

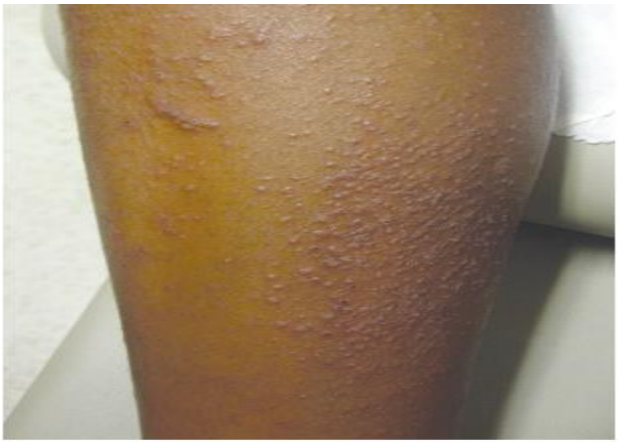

Figure 4: Sub acute atopic dermatitis in its dry, scaly, papular form. (3)

Clinical Sign	Description
Pruritus	The unpleasant sensation of the skin that provokes the urge to scratch; it is the primary hallmark of atopic dermatitis. Scratching the skin can aggravate existing dermatitis, causing excoriations that are either linear or punctate. Skin becomes leathered, rough, hard, and thickened upon scratching.
Xerosis	Dry skin in areas without clinically apparent inflammation. More common during periods of low humidity (eg, winter) and primarily affects the legs. Dysfunctional epidermal barrier function leads to dehydration of the stratum corneum layer that should have a 10% moisture content.
Ichthyosis vulgaris	Fish-like dry scales that can often look extremely thick and dry. Affected patients may alternately have excessively thin, whitish to brown scaling that classically affects the lower legs and shins while sparing the flexures. It is inherited in an autosomal semidominant manner.
Keratosis pilaris	Patients will have thick scale and redness around the hair follicles that may be surrounded by a patchy erythema. This condition most frequently affects the lateral cheeks, extensor (outer) aspect of the upper arms, and anterior thighs. The onset is typically during childhood and can persist into adulthood.
Follicular prominence	Follicles have a goose-bump appearance. Most commonly seen on the trunks of children and in darker-skinned individuals of any age.
Palmar and plantar hyperlinearity	Patients more often have exaggerated palmar hand creases than plantar creases.
Dennie-Morgan lines	Also known as atopic pleats, this refers to dark, symmetric, double horizontal folds below the lower eyelids as a consequence of intermittent edema of the eyelids.
Periorbital darkening ("allergic shiners")	Refers to gray to violet-brown discoloration and swelling around the eyes because of intermittent edema and rubbing of the region
Anterior neck folds	Horizontal folds or lines across the middle of the anterior neck.
Hertoghe sign	Loss of the lateral third of the eyebrows because of constant scratching.
White dermatographism	A blanching response as a result of stroking of the skin with the back of a fingernail that leads to white streaks. This reaction reflects excessive capillary vasoconstriction and local edema. This sign is reproduced in the scapular area where histamine is not depleted with trauma as a prestored mediator.
Pityriasis alba	Consists of multiple ill-defined light (hypopigmented) patches with fine scaling that are often located on the face and neck and occasionally appear on the shoulders and arms. These lesions are most obvious in darkly pigmented individuals and/or following sun exposure. This condition mostly affects children and young adults.

TABLE 1: Clinical signs of Atopic Dermatitis (8)

DIAGNOSIS:

There is no specific test for Atopic dermatitis. The diagnosis is made using specified criteria that consider the patient's medical history as well as clinical manifestations. (27) Several diagnostic standards and guidelines for Atopic dermatitis have been developed. (28) In a hospital context, the Hanifin and Rajka criteria were utilised as the usual diagnostic tool, with a sensitivity of 93-96 percent. (29) SCORAD (SCORing Atopic Dermatitis) is a commonly used clinical tool for determining the severity and extent of eczema. It assesses both objective and subjective symptoms such as irritation and insomnia. (30)

Must have three or more basic features described below 1) Pruritus 2) Typical morphology and distribution Flexural lichenification in adults Facial and extensor eruption in infants and children 3) Chronic or chronically relapsing dermatitis 4) Personal or family history of atopy (asthma, allergic rhinitis, atopic dermatitis)
Must have three or more following minor features 1) Xerosis 2) Ichthyosis/palmar hyper linearity, keratosis pilaris 3) Immediate (type I) skin test reaction 4) Elevated serum IgE 5) Early age of onset 6) Tendency toward cutaneous infection (especially *staph. Aureus* and *herpes simplex*), impaired cell-mediated immunity 7) Tendency toward non-specific hand or foot dermatitis 8) Nipple eczema 9) Cheilitis 10) Recurrent conjunctivitis 11) Dennie-Morgan infraorbital fold 12) Keratoconus 13) Anterior subscapular cataracts 14) Orbital darkening 15) Facial pallor, facial erythema 16) Pityriasis alba 17) Anterior neck folds 18) Itch when sweating 19) Intolerance to wool and lipid solvents 20) Perifollicular accentuation 21) Food intolerance 22) Course influenced by environmental and emotional factors 23) White dermographism, delayed blanch

TABLE 2: Diagnostic Standard of Hanifin and Rajka (5)

DIFFERENTIAL DIAGNOSIS:

- Candidiasis
- Contact dermatitis
- Dermatitis herpetiformis
- Impetigo
- Lichen simplex chronicus
- Nummular eczema
- Psoriasis
- Scabies
- Seborrheic dermatitis
- Urticaria
- Xerosis (31)

TREATMENT:

Topical or systemic treatments are indicated depending on the severity of the Atopic dermatitis. Stepladder treatment, appropriate for the clinical severity, is indicated. Once remission has been achieved, proactive maintenance therapy should be used to limit the number of flare-ups. (33)

FIRST LINE THERAPY:

MOISTURIZERS AND EMOLLIENTS

Patients with Atopic dermatitis have dry skin and a faulty skin barrier. The use of a moisturizer on a regular basis is essential in the treatment of Atopic dermatitis. A moisturizer restores the lipid barrier's ability to recruit, retain, and disperse water by repairing the skin barrier, maintaining skin integrity and appearance, reducing transepidermal water loss, and repairing the skin barrier's ability to recruit, hold, and redistribute water. (33) Moisturizer should be applied twice daily at the very least, and more frequently during extreme flare-ups. Adults with Atopic dermatitis should use about 250 g of moisturizer every week. It's best to apply moisturizer three minutes after taking a bath, while the skin is still wet. (34).

Acute lesions are treated with emollient treatment. It is recommended that emollients be used twice daily to the skin. Because of irritation and kidney impairment, urea-based emollients are not recommended for children. Propylene glycol irritates children's skin and should not be used. Emollients containing tannins and ichthammol are advised. Emollient formulations (lanolin/wool alcohol or methylisothiazolinone) that are free of protein allergies and haptens should be utilized. Flavonoids, saponins, and riboflavins from protein-free oat plant extracts, or lysates from Aquaphilus dolomiae or Vitreoscilla filiformis have been advised as non-medicated 'emollients' for Atopic dermatitis. (1)

TOPICAL CORTICOSTEROIDS

Topical corticosteroids, are significant anti-inflammatory medicines for controlling Atopic dermatitis, particularly during the acute stage. TCSs are the first-line treatment for people with Atopic dermatitis who have failed to maintain good skin care, including the use of moisturizers. (32)

Class	Potency	Generic name and strength
Class I	Very potent	Clobetasol propionate 0.05%
Class II	Potent	Beclometasone dipropionate 0.025%
		Betamethasone valerate 0.1%
		Betamethasone dipropionate 0.05%
		Diflucortolone valerate 0.1%
		Fluocinolone acetonide 0.025%
		Hydrocortisone butyrate 0.1%
		Mometasone furoate 0.1%
		Triamcinolone acetonide 0.1%
Class III	Moderate	Alclometasone dipropionate 0.05%
		Betamethasone valerate 0.025%
		Clobetasone butyrate 0.05%
		Fluocinolone acetonide 0.00625%
		Fluocortolone 0.25%
Class IV	Mild	Hydrocortisone 0.1%-2.5%
		Fluocinolone acetonide 0.0025%

BNF: British National Formulary

TABLE 3: classification of topical corticosteroids (32)

Anti-inflammatory, antiproliferative, and immunosuppressive properties of these drugs efficiently reduce atopic flare-ups. (27) Long-term administration of a low-potency glucocorticoid preparation on the face, genitalia, and intertriginous area, applied intermittently. (35)

ADVERSE EFFECTS:

- Striae,
- Petechiae,
- Telangiectasia,
- Skin thinning,
- Atrophy
- Worsening acne. (36)

Application frequency: For the treatment of atopic dermatitis, topical corticosteroids should be used twice day. (32)

TOPICAL CALCINEURIN INHIBITORS

From 2002, anti-inflammatory medication with topical calcineurin inhibitors (pimecrolimus and tacrolimus) has been recommended for Atopic dermatitis. (32) Both medicines block calcineurin phosphatase, which prevents T cells and mast cells from being triggered, as well as cytokine production and release. (1) Topical calcineurin inhibitors are a reasonable alternative to corticosteroids, especially for delicate skin regions such as the face and neck. (37) Topical calcineurin inhibitors do not have the same adverse effects as corticosteroids, such as skin thinning; therefore they can be used on a regular basis for extended lengths of time. In addition to oral calcineurin inhibitors, topical calcineurin inhibitors can be employed as part of a proactive therapy plan. (2) In investigations evaluating the efficacy of these drugs in the treatment of moderate to severe atopic dermatitis, tacrolimus was found to be at least marginally more successful than pimecrolimus. (38)

ADVERSE EFFECTS:

- Skin burning,
- Risk of viral infections such as herpes simplex virus is slightly elevated.
- There was a boxed warning based on a theoretical risk of malignancy (39,40)

WET WRAP THERAPY

Wet wrap therapy is a common treatment for acute flares and resistant illness, and it can significantly lower Atopic dermatitis severity immediately. In children with severe Atopic dermatitis, a recent randomized controlled trial found that a 4-week proactive regimen of Wet wrap therapy with diluted topical corticosteroids was better than Wet wrap therapy with moisturizer. Applying topical medications to the lesion is the initial step in Wet wrap therapy. A moist inner layer of tubular bandages is applied to the skin, followed by a dry outer layer. As an alternative, you may use gauze or a cotton suit. The Wet wrap therapy can be maintained for a period of time ranging from a few hours to a whole day. (41)

SECOND LINE THERAPY:

CYCLOSPORINE

Cyclosporine is an oral calcineurin inhibitor that inhibits the production of a variety of cytokines, including IL-2, by suppressing the action of the T-cell transcription factor, nuclear factor of activated T cells. (32) Treatment with cyclosporine has been linked to a reduction in skin disease and a better quality of life. Treatment withdrawal may result in a fast recurrence of skin disease, while some individuals may have long-term remission. (5)

DOSE: The medication should be taken twice a day at a dose of 3–5 mg/kg/day. (42)

ADVERSE EFFECTS:

- Reversible nephrotoxicity
- Infection
- Hypertension
- Electrolyte disturbances
- Dyslipidemia
- Tremor
- Hypertrichosis
- Headache
- Gingival hyperplasia
- Nonmelanoma skin cancer(43)

THIRD LINE THERAPY:

PHOTOTHERAPY

If topical therapies fail to manage the condition, phototherapy (typically 4–8 weeks) may be used. Narrow-band ultraviolet B radiation and medium-dose ultraviolet A1 are the most effective options. (44) Phototherapy can be used alone as well in conjunction with topical steroids and emollients. (32)

- UVA and UVB (about 280–400 nm).
- Broadband ultraviolet B (BB-UVB) wavelengths (about 280–320 nm).
- UVB with a narrow band (nbUVB = peak: 311–313 nm).
- UVA1 (wavelength 340–400 nm).

Longer wavelengths have not been well investigated in the treatment of atopic dermatitis and should not be used. When phototherapy is administered, it is generally part of a larger treatment plan. Phototherapy should not be used on children under the age of 12. (39)

ADVERSE EFFECTS:

- Actinic damage,
- Local erythema
- Tenderness,
- Pruritus,
- Burning, and stinging
- Nonmelanoma skin cancer,
- Melanoma (mostly with PUVA),
- Lentigines,
- Photosensitive eruptions,
- Folliculitis,
- Photo-onycholysis,
- HSV reactivation,
- Facial hypertrichosis.(45,46,47)

Skin type	Initial UVB dose	Dose increment after each treatment	Maximum dose
I	130 mJ/cm²	15 mJ/cm²	2000 mJ/cm²
II	220 mJ/cm²	25 mJ/cm²	2000 mJ/cm²
III	260 mJ/cm²	40 mJ/cm²	3000 mJ/cm²
IV	330 mJ/cm²	45 mJ/cm²	3000 mJ/cm²
V	350 mJ/cm²	60 mJ/cm²	5000 mJ/cm²
VI	400 mJ/cm²	65 mJ/cm²	5000 mJ/cm²

UV: Ultraviolet

TABLE 4: Guidelines for narrowband UVB according to skin type

AZATHIOPRINE

Adult patients who are resistant to, contraindicated to, or suffering harmful effects from Cyclosporine might explore Azathioprine. When administered as monotherapy in people with Atopic dermatitis, double-blind placebo-controlled trials show that Azathioprine improves both Quality of life and disease signs and symptoms. Before starting therapy, a test for the enzyme thiopurine methyltransferase is advised. (48, 49)

DOSE: 1–3 mg/kg/day is recommended depending on TPMT activity

ADVERSE EFFECTS:

- Nausea and vomiting
- Headache
- Hypersensitivity reactions
- Elevated liver enzymes
- Leukopenia. (50,51)

METHOTREXATE

Methotrexate is an antimetabolite that disrupts the metabolism of folic acid, which controls the immune system and inflammation. In a 2016 research, half of the patients with severe Atopic dermatitis benefitted from Methotrexate therapy, and after a year of usage, cessation owing to subjective side-effects is unusual, suggesting that treatment is long-lasting and effective. (52)

DOSE: 10–22 mg/week (32)

FOURTH LINE OF THERAPY: BIOLOGICS AND EMERGING THERAPIES

CRISABOROLE

Crisaborole is a nonsteroidal anti-inflammatory drug that can target PDE4 in a specific way. It efficiently up-regulates intracellular cyclic adenosine monophosphate, which is also a modulator of nuclear factor, by inhibiting Phosphodiesterase-4 activated B cell light-chain enhancer and activated T-cell nuclear factor signaling pathways 80 As a consequence, numerous proinflammatory cytokines are suppressed, and inflammation is controlled. (8)

DUPILUMAB

Dupilumab is a completely human monoclonal antibody that inhibits the signaling of both IL-4 and IL-13, the two primary drivers of type 2 immune response. (32)

DOSE: After an initial loading dose of 600 mg, the recommended dose of dupilumab is 300 mg

Every two weeks (two 300 mg injections at different sites). (53)

ADVERSE EFFECTS:

- Injection site reactions,
- Conjunctivitis,
- Blepharitis,
- Keratitis,
- Eye pruritus,
- Dry eye,
- Herpes simplex virus infection (53)

OTHER BIOLOGICALS

There is minimal experience with Ustekinumab, Rituximab, Tocilizumab, and Alefacept in Atopic dermatitis, based on favorable case reports. Except in rare circumstances, current data does not support the use of these biological in Atopic dermatitis. (32)

ORAL PHOSPHODIESTERASE-4 INHIBITOR

In September 2014, the Food and Drug Administration authorized Apremilast, an oral phosphodiesterase-4 inhibitor, for the treatment of moderate-to-severe plaque psoriasis. (32) An open-label pilot trial of its safety and effectiveness in Atopic dermatitis adult patients revealed a significant reduction in itching and an increase in life quality. Its favorable usage in Atopic dermatitis and other inflammatory skin disorders has been reported since then, but more rigorous randomized controlled research is needed. (54)

HIGH-DOSE INTRAVENOUS IMMUNOGLOBULIN

According to a study of the literature, 61 percent of individuals with Atopic dermatitis who were given high-dose intravenous immunoglobulin improved. Adults tended to be less likely (48%) than children

(90%) to response, and the duration of action was significantly longer in children. Adjunctive treatment was shown to be more successful than monotherapy in adults (59 percent vs. 0%), although monotherapy was found to be beneficial in 90 percent of children. High-dose intravenous immunoglobulin might be a safe and effective treatment option for resistant Atopic dermatitis, especially in youngsters, but it has to be tested further in double-blind, placebo-controlled studies. (55)

INTERFERON GAMMA

Interferon-has been shown to be effective in the treatment of Atopic dermatitis in a few trials. (32)

ADJUNCTIVE TREATMENT:

Alitretinoin – 9-cis-retinoic acid, or 9-cis-retinoic acid, is a newly produced retinoid derivative. Alitretinoin is used to treat atopic hand eczema that is chronically hyperkeratotic. (32)

Probiotics/prebiotics - When administered in the prenatal and postnatal periods, probiotics (Lactobacillus alone or Lactobacillus plus Bifidobacterium) appeared to serve a protective function in the prevention of Atopic dermatitis. However, there is no proof that probiotics are beneficial to newborns. (56)

Essential fatty acids - Evening primrose oil or an omega-3 fatty acid (docosahexaenoic acid) supplementation in the diet is safe and may aid with Atopic dermatitis, however there are inadequate randomized controlled trial data analyzing clinical efficacy for this practice to be advised. (32)

Vitamin D - Vitamin D may have a role in alleviating the symptoms of Atopic dermatitis. The findings of this study imply that vitamin D supplementation may help reduce the severity of Atopic dermatitis and is a safe and manageable treatment option. (57)

TREATMENT DURING PREGNANCY AND LACTATION:

Despite the fact that atopic dermatitis has no adverse consequences on pregnancy, treating pregnant or breastfeeding women, as well as those who are attempting to conceive, is difficult. (58) This was addressed in the European Task Force for Atopic Dermatitis position document on the management of the illness during preconception, pregnancy, and breastfeeding. Moisturizers, class II and III topical corticosteroids, topical calcineurin inhibitors, and phototherapy with uva and narrow-band uvb are all recommended in the study. Systemic therapy, on the other hand, should be used only when absolutely necessary. Short-term systemic steroids and cyclosporine are regarded reasonably safe when used with thorough supervision, however azathioprine should be avoided, and methotrexate and mycophenolate mofetil should be avoided in pregnant and lactating women. (59)

NON PHARMACOLOGICAL TREATMENT:

- Informing the patient on proper skin care and hygiene when bathing and dressing.
- Patient counselling about the negative impact of indoor and outdoor air pollution on long-term Atopic dermatitis management.
- Cleansing of the bedroom floor and mattresses according to standard procedures.
- Pollen filters and air conditioners for a comfortable interior atmosphere.
- If a patient is sensitive to a pet and comes into touch with it, avoidance is recommended.
- The American Academy of Dermatology recommended egg restriction in AD patients
- Who were found to be severely allergic to eggs.
- The use of probiotics such as lactobacillus or a prebiotic combination has been found to ameliorate the skin lesions associated with Atopic dermatitis.(1)

CONCLUSION:

In affluent nations, the rising prevalence of Atopic Dermatitis has become a severe public health concern since it greatly reduces the patient's quality of life. Furthermore, if not adequately controlled, it can lead to lifelong allergies and asthma. Improved medications and management therapy will be developed as a result of a better knowledge of its genetics and immunoregulatory cascade, reducing flare-ups and improving quality of life. Human monoclonal antibody-based medicines have made significant progress in the therapy of Atopic dermatitis, but further clinical studies are necessary for an effective treatment regimen to be established. Atopic dermatitis prevention and management can be improved with advanced solutions.

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