IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

Review on Atopic Dermatitis

Rajlaxmi Deolekar, Vaibhav J. Gajbe, Bilal Sufi, Vivek K. Gohane
New Montfort Institute of Pharmacy Asthi, Wardha, Maharashtra, India
vaibhavgajbe2468@gmail.com

Abstract: *Atopic dermatitis (AD) is becoming a major public health problem due to its increasing prevalence and growing evidence that it may cause other allergic reactions. Atopic dermatitis is a common inflammatory skin disease, with typical recurrent eczema lesions. This can be frustrating for children's patients, parents and healthcare providers. Pediatrics treats most children with atopic dermatitis because many patients do not have access to pediatric subspecialty physicians such as pediatric dermatologists and pediatric allergic doctors.*

Keywords: Atopic Dermatitis

I. INTRODUCTION

Atopic dermatitis is a common chronic inflammation of the skin that affects children mainly.[1] Atopy is defined as a inherited tendency to produce immune globulin E antibodies (igEs) in response to a small amount of common environmental proteins, such as pollen, household dust and food allergens. Dermatitis stems from the Greek Derma (derma) (inflammation). Dermatitis and eczema are generally used as synonyms, but eczema is sometimes reserved for acute symptoms of the disease (Greek: eczema boils).[2] Because AD is linked to significant morbidity, a decline in quality of life (QoL), and conditions similar to other chronic diseases such diabetes, fibrosis, and epilepsy, it is a global health concern.[3,4]. The human body's physical and functional barriers are provided by the epidermis, and the most significant pathological findings in AD skin are deficiencies in the skin's skin barrier.[5,6] The illness, which is apparent and chronic, can have a significant effect on patients' and their families' lives. Symptoms include severe rash, discomfort from skin injuries, and insomnia.[7,8] Additionally, AD may be the beginning of a "atopic march," which results in various forms of atopic dermatitis (AD), which typically begin in childhood and become extremely persistent and chronic, including rhino conjunctivitis and asthma.[9] Atopic dermatitis (AD) is a juvenile onset condition. Clinical features might vary widely, making accurate diagnosis challenging at times.[10] Ten to thirty percent of people suffer from AD, which is extremely frequent.[11,12] AD is becoming more and more common, at least in emerging nations. Environmental variables have a profound influence on the genetic basis of AD. Current developments Genetics research on AD patients has identified a mutation of Filaggrin (FLG) in those individuals.[13] The general patterns in which patients acquire adult rhinitis and early AE childhood asthma are referred to as the "atopy march."[14] The primary signs of AD include lichenification, dry skin, papules, inflammation of eczema, and severe rhinitis, which is thought to be a hallmark of AD. It is thought that pruritus, which starts a vicious cycle of itching, scratching, and worsening eczematous lesions, is a key factor in maintaining and exacerbating AD.[15,16]

EPIDEMIOLOGY

Approximately 25% of people may experience atopic dermatitis at some point in their lives, while the disease's incidence varies widely across the globe.[17] About half of all cases of atopic dermatitis occur in the first year of life, and the majority—probably ninety-five percent—occur before the patient is five years old.[18] Of those whose eczema began in childhood, about 75% experience a spontaneous remission before to puberty, with the remaining 25% developing eczema until adulthood. Atopic dermatitis that recurs in maturity or develops in adulthood often manifests primarily as hand eczema in many cases. According to World Health Organization data on global disease initiatives, AD affects at least 230 million people around the world and is considered to be the main cause of skin diseases that do not lead to fatal disease[19] and between 50 and 75 percent of children with early atopic dermatitis are sensitive to one or more allergens, such as food allergens, home dust fungi or pets.[20.21] Adult AD epidemiology is largely ignored. Many studies

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

have been conducted on the prevalence of AD in children and adolescents, but only a few studies have been conducted on AD in adults.[22,23]

PATHOGENESIS

AD pathogenesis is complex and faceted. Skin barrier failure, environmental factors, genetic predisposition and immune failure play an important role in their development and are closely related. In the past, the emphasis was placed on the disregulation of the T-helper cell, the production of immune globulin E (IgE) and the hyperactivity of mast cells leading to inflammation of vascular diseases and characteristic skin disorders.[24] SC is the main epidermal component of the so-called epidermal barrier and is directly involved in some barrier functions. Some known abnormalities in SC barrier disorders can contribute to the destruction of epidermal barriers, enabling mechanisms to be effective in AD pathophysiology[25] and recent genetic studies have made real progress in AD disease research, with many patients with AD finding FLG mutations.[26]

PATHOPHYSIOLOGY

The immune hypothesis. Immune imbalance theory argues that atopic dermatitis is caused by imbalances between T cells, especially T assistant cells 1, 2, 17 and 22, and regulatory T cells.[27]

Hypotheses skin barrier. The theory of skin barrier defects is more recent and stems from the observation that individuals with filaggrin mutations are more likely to develop atopic dermatitis.[28]Flaggrin genes encode structural proteins in the corneal and granulosa layers and contribute to the connection of keratinocytes. This maintains an intact skin barrier and a hydrated corneum layer. Genetic defects cause fewer filaggrins, which causes skin barrier failure and transepidermal water loss, causing eczema. There is evidence that inadequate skin barriers lead to dry skin, increased skin penetration of allergens, allergies, asthma and hay fever.[29]

CLINICAL FEATURE

Clinical diagnosis is usually easy, but with the exception of infants, babies, and older people, the clinical features are more abnormal. Skin biopsy can help to exclude other common diseases that mimic AD, co-exist with AD, or complicate AD, such as T cell lymphoma in the skin or rare diseases such as primary immunodeficiency and childhood nutritional deficiencies. In order to support the diagnosis, several criteria have been proposed over time, but original Hanifin and Rajka criteria have been proposed.[31] Adverse brain injury occurs in any part of the body, although it usually shows age- related distribution patterns. Children often have extensive, more severe skin damage and show severe erythema, swelling, excretion and serous exudates characterized by oily and cracked surfaces, facial/chin and trunk regions and reduced diaper areas. In childhood AD is more localized and chronic, with pale erythema, xerosis and thickened skin due to repeated scratches that usually affect flexor surfaces. Adolescents and adults may have diffuse AD models, but also localized disorders that generally affect hands, eyelids and legs. Adults can only show chronic AD or head and neck subtypes, including the upper trunk, shoulders and head.[31,32,33,34,35] Other common features, but not prerequisites for diagnosis, include early onset (usually during the first year of life), the history of atopic disease in individuals and/or families, specific IgE reactions, and the presence of generalized skin dryness.[36,37]One of the reasons for increased risk of skin infection in AD is the decrease in secretion of important antimicrobial peptides, such as antimicrobial peptides such as defensin or Catholic, which are important for natural immunity.[38,39]

DIAGNOSIS

In general, clinical diagnosis is easy, but clinical features are more unusual in babies, infants, and elderly patients. Skin biopsy can help to eliminate other common diseases that imitate, coexist or complicate AD, cancers such as T cell skin lymphoma or, unlike in childhood, other rare diseases such as primary immunity and nutritional deficiencies. Some criteria have been proposed over time to support diagnosis, but the original Hanifin and Rajka criteria[40] are still the most widely used worldwide,[41] and patients with non-typical conditions require a skin biopsy to eliminate other skin diseases similar to AD. These include other inflammatory diseases (seborrheic disease, psoriasis, allergic or irritant contact

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

disease, lichenoide disease), primary ichthyosis, scabies, infections (fungi, human immunodeficiency virus (HIV), malignant disease (mainly skin T-cell lymphoma) and metabolic diseases.[42]

TREATMENT

Atopic dermatitis is untreated and many patients are suffering from chronic disease. Consequently, the treatment of atopic dermatitis has the following objectives:[43]

reducing the number and severity of a flare; and

reducing the duration and severity of a flare.

The first objective mainly refers to prevention and the second objective to treatment. Prevention is best achieved by reducing skin dryness, especially by daily use of hydrators and skin emollients, and by avoiding specific and non-specific irritants such as allergens and non-cotton clothing. When dryness decreases, the desire to scratch decreases and the risk of skin infection decreases. Avoiding long hot baths further prevents skin aging, but when bathing, apply moisturizer directly to the skin and increase skin moisture. Reduce flares when there is real eczema or when a mild intermittent eczema is exacerbated. Treatment of eczema often requires corticosteroids to be taken. In addition to current treatments, severe acute or chronic eczema often requires systemic immunosuppressive drugs or UV light therapy.

Emulsifiers: Maintain an effective skin barrier. Emollient use is an essential part of the treatment of atopic dermatitis. It has been shown that they should be used several times a day, and their systematic use has reduced the need for corticosteroids.[44,45]

Skin-based therapy should be the first method of treatment. The method consists of four main components, each focused on specific symptoms of AD: 1) skin repair and maintenance, 2) topical anti-inflammatory drugs aimed at suppressing inflammation responses. 3) Control of itch; 4) Control of infectious triggers, detection and treatment of flares associated with itch. Education of patients and their families is another key factor, which must not be neglected. AD is a frustrating disease because it has a repeated nature, even when it comes to excellent treatment plans. When primary care providers set realistic expectations of the outcome, parental compliance improves and frustration reduces. Most children experience more symptoms or at least more symptoms, so discussing the disease's[46] outlook helps, and the patient receiving a complete diagnosis of AD and his care can improve. The severity of the disease is better than the comprehensive diagnosis and care of the patient,[47] with the aim of treating AD, the onset of the disease, known as "flash" is the lowest, and the duration and severity of the outbreak is reduced.[48,49]Adult patients generally treat AD, and in 2012, Ring and colleagues proposed an updated guideline for treating AD (not just adult AD).[50]

TOPICAL TREATMENT

It is recommended to use topical steroids in acute flares and maintain topical calcium kinergic inhibitors.[51]The topical tachymus is more effective than the pimecrolimus in moderate to severe AD patients, but both have similar safety profiles and can be applied to sensitive and thin skin (face, eyeslids)[52]

PHOTOTHERAPY

Since most patients with atopic dermatitis improved in summer, artificial ultraviolet radiation is often used in AD therapy. At present, UV sources include devices capable of emitting selective radiation spectrums:

UVA + UVB (about 280–400 nm).

The broad-band ultraviolet B (BB-UVB = 280–320 nm) The narrowband UVB (nbUVB = a peak of 311–313 nm) A narrowband UVB. UVA1 (340–400 nm)

Usually, phototherapy is not indicated in acute AD stages (except UVA1) and is more appropriate for chronic, painful and lichenic conditions, and should not be used for patients who are exposed to the sun to exacerbate skin diseases. In fact, the availability of photographic therapy equipment limits the selection of a specific UV treatment: for example, UVA1 devices are expensive to buy and maintain.[53]

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

SYSTEMIC TREATMENT

Sedation of antihistamines can reduce itch during recurrence, but they affect driving skills and should not be combined with alcohol or certain antidepressants[54] Antihistamines have been used for decades to relieve pain in AE patients. However, only a few randomised controlled trials have been conducted, and in most of them have shown little or no effect on the reduction of pruritus. The first generation of sedatives, such as hydroxyzine, Clemastine Fumigate, and Dimetinden Maleate, may help in acute situations where the symptoms of eczema are exacerbated (Evidence Level D). As regards the new non-sedative antihistamines, in AD , only studies with loratadine, cetirizine or fexofenadine showed no or only weak relief from pruritus.[55]

OTHER MEDICATION

In a recent placebo-controlled study of adults with asthma, severity was improved after four months of specific immunotherapy for asthma, particularly in patients with severe asthma.[56] Interferon beta, interferon gamma, and infliximab have been tried for severe asthma, but there are few well-controlled studies to justify their use in adults.[57]

MEDICATION

Moisturizers are externally applied compounds that contain several components in order to maintain the integrity and appearance of the skin.[58] Previous studies have shown that moisturizers affect the function of normal skin's barrier to reduce TEWL and sensitivity to irritants.[59,60] Moisturizers are generally classified on the basis of their mechanism of action as occlusive, humidifying and emollient. The two main mechanisms used to rehydrate SC are occlusive and humectant.[61]

Occlusives are generally oily substances that can degrade the TEWL by forming water-sensitive layers on the surface of the skin and the SC's surface interstation. Occlusives are very effective on moist skin due to water intoxication.[62]

The prototype of occlusives is the most effective occlusive moisturizing, petrolatum. Lanolin and mineral oil are also commonly used, but are less effective to cover. Silicone, including dimethicone and cyclomethycane, are new synthetic fillers that are popular as ingredients for oil-free moisturizers due to their low-allergenic, non-comedogenic, non-greasy and odorless properties. In addition to the above-mentioned traditional oils, some hydrating chemicals can contain physiological skin barrier lipids, cytric acid, cholesterol, and free fatty acids. These three key lipids, unlike external closures, must penetrate deeper into SC and ultimately become part of SC's lipid barrier, restoring the normal balance of the epidermal barrier.[63]

Low molecular weight compounds that attract water are known as humectants.[64]They improve the SC's ability to absorb water from the deeper dermis and epidermis. Rarely do humectants absorb water from the surrounding air until the relative humidity is higher than 70%.[65]The most efficient humectant is glycerol.[66]

In skin care, moisturizing plays a number of important tasks, such as helping to restore the skin's damaged barrier, minimizing transepidermal water loss, keeping the skin hydrated, easing dryness, and lowering the need for topical corticosteroids (TCSs). The main job of the stratum corneum is to stop water from evaporating through the skin.[67]

ANTI-INFLAMMATORY

Systemic anti-inflammatory drugs may be necessary when first-line and second-line therapies do not provide benefit to the patient.[68] Skin inflammation immune responses manifest themselves in AD eczematosis. Because moisturization is unlikely to be effective in treating skin eruptions, the treatment in these episodes focuses on reducing the inflammatory reaction. In the past 40 to 50 years, topical steroids have been the first and most widely used drugs to treat active AD.30 If used properly, they are safe and effective.[69]

On the other hand, there are some dangers of systemic absorption with consequent adrenal suppression, telangiectasia, striae, and cutaneous atrophy when taken improperly. When applied near the lips (periorificial dermatitis) or eyes (intraocular hypertension, cataracts), there are additional strong local effects. Parents and healthcare professionals actually experience "steroid phobia" as a result of these possible hazards. This fear does not correspond with the severity of AD, but it does cause the skin condition to be undertreated.[70,71]

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

ANTI-MICROBIAL

Patients with bacterial infections should receive antibacterial or oral treatments to prevent the development of antimicrobial resistance, but their use should usually be limited to short periods. First- generation cephalosporins may be used to treat S aureus infections that produce superinfection in AD patients, according to some data. Other medical professionals will prescribe antibiotics like doxycycline or cotrimoxazole that have anti-inflammatory properties in addition to their ability to fight staphylococcus. As an adjuvant therapy, bleach (sodium hypochlorite) baths may also be suggested for people with AD who experience recurrent or severe secondary bacterial infections. It has been proposed that bleach's antiseptic properties can lessen S aureus's tendency to colonize skin.[72]

Eczema herpeticum is characterized by numerous painful, single-sided and broken pieces (bleeding with bleeding pieces). Patients with facial injuries should be referred to ophthalmology to assess the possibility of retinal involvement. Cutaneous lesions must be examined to identify polymerase chain reactions in herpes simplex virus and varicellazoster virus. If the result is not obtained within hours of examination, or if the morphology of the lesions is consistent with the Herpes simplex virus, an acute treatment should be initiated. . Treatment includes antiviral acyclovir or its derivatives, famciclovir and valacyclovir. Patients with severe or persistent infections such as fever, insomnia and lymphadenopathy should take oral products. Intravenous acyclovirs are generally reserved for patients with food and drinking restrictions, low immune capacity, vision impairment or systemic involvement. Patients with dermatophytic (fungal) M-furfur infections (the best micro-examination of scaled skin scraps) should be treated with antifungal drugs (such as topical benzodiazepines). Some studies suggest that when pregnant or developing, mothers can supplement probiotics in the first three years of pregnancy, and that early AD can be delayed or delayed by 20%.[73,74]

ANTI-HISTAMINE

Scratching causes the release of histamines and other mediators, thereby exacerbating pruritus. This may be frustrating because patients may have difficulty sleeping. Antihistamines that are sedative and non-sedative are often prescribed, with non-sedative antihistamines that are less useful in AD management to control pruritus. It has been shown that oral antihistamines (e.g., Hydroxyzine, Diphenhydramine, Doxepin) stimulate the quality of sleep of patients.[75]

PREVENTION

Because childhood diseases are more common, prevention is concentrated in pregnancy. Prevention should be initiated as soon as possible, including skin barriers, immunity/allergens and environmental aspects. Early prevention methods, such as nutrition supplements, breastfeeding, hydrogenated milk, prebiotics and prebiotics, have shown inconsistent results and have not yet shown significant impact on reducing the risk of AD.[76,77,78]

Although there have been long-term studies of general interventions aimed at reducing the incidence of allergic diseases, particularly AD, some studies clearly focus on atopic movements. The main intervention currently being investigated and the treatment of the atopic march hypothesis is the construction and maintenance of the function of the newborn skin barrier.[79]

II. CONCLUSION

Atopic dermatitis is a skin disorder in which skin infections, inflammation and redness occur, and in order to cure this patient, you must maintain good hygiene.

After reviewing medical interventions and lifestyle adjustments, improved care and support for atopic dermatitis patients may be improved, ultimately enhancing patients' quality of life.

Atopic dermatitis improves the quality of life of atopic dermatitis patients through appropriate management and treatment

REFERENCES

[1]. Danby S, Cork MJ. A New understanding of atopic dermatitis: the role of epidermal barrier dysfunction and subclinical inflammation. J Clin Dermatol. 2010;1:33-46.

[2]. Torres T. Atopic dermatitis: the new therapeutic revolution in dermatology. Acta Med Port. 2017;30:669–70

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

[3]. Weidinger S, Beck LA, Bieber T, Kabashima K, Irvine AD. Atopic dermatitis. Nat Rev Dis Primers. 2018;4:1

[4]. . Nakatsuji T, Chen TH, Narala S, Chun KA, Two AM, Yun T, et al. Antimicrobials from human skin commensal bacteria protect against Staphylococcus aureus and are deficient in atopic dermatitis. Sci Transl Med 2017;9:eaah4680.

[5]. van Smeden J, Bouwstra JA. Stratum corneum lipids: their role for the skin barrier function in healthy subjects and atopic dermatitis patients. Curr Probl Dermatol 2016;49:8-26.

[6]. de Bruin Weller MS, Rockmann H, Knulst AC, Bruijnzeel-Koomen CA. Evaluation of the adult patient with atopic dermatitis. Clin Exp Allergy. 2013 Mar;43(3):279-91. [PMID: 23414536].

[7]. Finlay AY. Quality of life in atopic dermatitis. J Am Acad Dermatol. 2001 Jul;45(1 Suppl):S64-6. [PMID: 11423879].

[8]. Illi S, von Mutius E, Lau S, Nickel R, Grüber C, Niggemann B, Wahn U; Multicenter Allergy Study Group. The natural course of atopic dermatitis from birth to age 7 years and the association with asthma. J Allergy Clin Immunol.2004 May;113(5):925-31. [PMID: 15131576].

[9]. Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. Acta Derm Venereol Suppl (Stockh) 1980;59:44-7.

[10]. Bieber T. Atopic dermatitis. N Engl J Med 2008;358:1483-94.

[11]. Choi WJ, Ko JY, Kim JW, Lee KH, Park CW, Kim KH, Kim MN, Lee AY, Cho SH, Park YL, Choi JH, Seo SJ, Lee YW, Roh JY, Park YM, Kim DJ, Ro YS. Prevalence and risk factors for atopic dermatitis: a cross-sectional study of 6,453 Korean preschool children. Acta Derm Venereol 2012;92:467-71

[12]. Palmer CN, Irvine AD, Terron-Kwiatkowski A, Zhao Y, Liao H, Lee SP, Goudie DR, Sandilands A, Campbell LE, Smith FJ, O'Regan GM, Watson RM, Cecil JE, Bale SJ, Compton JG, DiGiovanna JJ, Fleckman P, Lewis-Jones S, Arseculeratne G, Sergeant A, Munro CS, El Houate B, McElreavey K, Halkjaer LB, Bisgaard H, Mukhopadhyay S, McLean WH. 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.

[13]. Gustafsson D, Sjoberg O, Foucard T. Development of allergies and asthma in infants and young children with atopic dermatitis – a prospective follow-up to 7 years of age. Allergy 2000: 55: 240–245.

[14]. Boguniewicz M, Leung DYM: New concepts in atopic dermatitis. Compr Ther 1996;22:144– 151.

[15]. Morren M-A, Przybilla B, Bamelis M, Heykants B, Reynaers A, Degreef H: Atopic dermatitis: Triggering factors. J Am Acad Dermatol 1994;31:467–473.

[16]. M. I. Asher, S. Montefort, B. Bjorkst ¨ en 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," The Lancet, vol. 368, no. 9537, pp. 733–743, 2006.

[17]. H. C. Williams, "Atopic dermatitis," New England Journal of Medicine, vol. 352, no. 22, pp. 2314–2366, 2005.

[18]. J. M. Spergel, "From atopic dermatitis to asthma: the atopic march," Annals of Allergy, Asthma and Immunology, vol. 105, no. 2, pp. 99–106, 2010.

[19]. Weidinger S, Beck LA, Bieber T, Kabashima K, Irvine AD. Atopic dermatitis. Nat Rev Dis Primers. 2018;4:1

[20]. Hay RJ, Johns NE, Williams HC, Bolliger IW, Dellavalle RP, Margolis DJ, et al. The global burden of skin disease in 2010: an analysis of the prevalence and impact of skin conditions. J Invest Dermatol. 2014;134:1527–34.

[21]. Saeki H, Oiso N, Honma M, Iizuka H, Kawada A, Tamaki K. Prevalence of atopic dermatitis in Japanese adults and community validation of the U.K. diagnostic criteria. J Dermatol Sci. 2009 Aug;55(2):140-1. [PMID: 19427173].

[22]. Deckers IA, McLean S, Linssen S, Mommers M, van Schayck CP, Sheikh A. Investigating international time trends in the incidence and prevalence of atopic eczema 1990-2010: a systematic review of epidemiological studies. PLoS One. 2012;7(7):e39803. [PMID: 22808063].

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

[23]. Elias PM, Steinhoff M. "Outside-to-inside" (and now back to "outside") pathogenic mechanisms in atopic dermatitis. J Invest Dermatol. 2008;128(5):1067–1070

[24]. Levin J, Friedlander SF, Del Rosso JQ. Atopic dermatitis and the stratum corneum: part 2: other structural and functional characteristics of the stratum corneum barrier in atopic skin. J Clin Aesthet Dermatol. 2013 Nov;6(11):49-54. [PMID: 24307926].

[25]. Palmer CN, Irvine AD, Terron-Kwiatkowski A, Zhao Y, Liao H, Lee SP, Goudie DR, Sandilands A, Campbell LE, Smith FJ, O'Regan GM, Watson RM, Cecil JE, Bale SJ, Compton JG, DiGiovanna JJ, Fleckman P, Lewis-Jones S, Arseculeratne G, Sergeant A, Munro CS, El Houate B, McElreavey K, Halkjaer LB, Bisgaard H, Mukhopadhyay S, McLean WH. 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.

[26]. K. Eyerich and N. Novak, "Immunology of atopic eczema: overcoming the Th1/Th2 paradigm," Allergy, vol. 68, pp. 974– 982, 2013.

[27]. C. N. A. Palmer, A. D. Irvine, A. Terron-Kwiatkowski et al., "Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis," Nature Genetics, vol. 38, no. 4, pp. 441–446, 2006.

[28]. A. de Benedetto, A. Kubo, and L. A. Beck, "Skin barrier disruption: a requirement for allergen sensitization," Journal of Investigative Dermatology, vol. 132, no. 3, pp. 949–963, 2012.

[29]. Hanifin J, Rajka G. Diagnostic features of atopic dermatitis. Acta Derm Venereol. 1980;92:S44–7.

[30]. Weidinger S, Beck LA, Bieber T, Kabashima K, Irvine AD. Atopic dermatitis. Nat Rev Dis Primers. 2018;4:1.

[31]. Waldman AR, Ahluwalia J, Udkoff J, Borok JF, Eichenfield LF. Atopic dermatitis. Pediatr Rev. 2018;39:180–93.

[32]. Nutten S. Atopic dermatitis: global epidemiology and risk factors. Ann Nutr Metab. 2015;66:S8–16.

[33]. Kapur S, Watson W, Carr S. Atopic dermatitis. Allergy Asthma Clin Immunol. 2018;14:S52.

[34]. Silverberg JI. Adult-onset atopic dermatitis. J Allergy Clin Immunol Pract. 2019;7:28–33

[35]. Weidinger S, Beck LA, Bieber T, Kabashima K, Irvine AD. Atopic dermatitis. Nat Rev Dis Primers. 2018;4:1

[36]. Tsakok T, Woolf R, Smith CH, Weidinger S, Flohr C. Atopic dermatitis: the skin barrier and beyond. Br J Dermatol. 2019;180:464–74.

[37]. Ong PY, Ohtake T, Brandt C, Strickland I, Boguniewicz M, Ganz T, Gallo RL, Leung DY. Endogenous antimicrobial peptides and skin infections in atopic dermatitis. N Engl J Med 2002;347:1151-60.

[38]. Hata TR, Gallo RL. Antimicrobial peptides, skin infections, and atopic dermatitis. Semin Cutan Med Surg 2008;27:144-50.

[39]. Hanifin J, Rajka G. Diagnostic features of atopic dermatitis. Acta Derm Venereol. 1980;92:S44–7.

[40]. Wollenberg A, Barbarot S, Bieber T, Christen-Zaech S, Deleuran M, Fink-Wagner A, et al. Consensus-based European guidelines for treatment of atopic eczema (atopic dermatitis) in adults and children: part I. J Eur Acad Dermatol Venereol. 2018y;32:657–82.

[41]. Siegfried EC, Hebert AA. Diagnosis of atopic dermatitis: mimics, overlaps, and complications. J Clin Forensic Med 2015;4(5):884-917

[42]. J. Ring, A. Alomar, T. Bieber et al., "Guidelines for treatment of atopic eczema (atopic dermatitis)—part I," Journal of the European Academy of Dermatology and Venereology, vol. 26, pp. 1045–1060, 2012

[43]. E. L. Simpson, "Atopic dermatitis: a review of topical treatment options," Current Medical Research and Opinion, vol. 26, no. 3, pp. 633–640, 2010

[44]. G. Ricci, A. Dondi, and A. Patrizi, "Useful tools for the management of atopic dermatitis," American Journal of Clinical Dermatology, vol. 10, no. 5, pp. 287–300, 2009.

[45]. Illi S, von Mutius E, Lau S, et al; Multicenter Allergy Study Group. The natural course of atopic dermatitis from birth to age 7 years and the association with asthma. J Allergy Clin Immunol. 2004;113(5):925–931

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

[46]. Staab D, Diepgen TL, Fartasch M, et al. Age related, structured educational programmes for the management of atopic dermatitis in children and adolescents: multicentre, randomised controlled trial. BMJ. 2006; 332(7547):933–938

[47]. . Asher MI, Montefort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, Williams H; ISAAC Phase Three Study Group. Worldwide time trends in the prevalence of symptoms asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. Lancet. 2006 Aug 26;368(9537):733-43. [PMID: 16935684].

[48]. Schultz Larsen F, Diepgen T, Svensson A. The occurrence of atopic dermatitis in north Europe: an international questionnaire study. J Am Acad Dermatol. 1996 May;34(5 Pt 1):760-

[49]. 4. [PMID: 8632070].

[50]. Ring J, Alomar A, Bieber T, Deleuran M, Fink-Wagner A, Gelmetti C, Gieler U, Lipozencic J, Luger T, Oranje AP, Schäfer T, Schwennesen T, Seidenari S, Simon D Ständer S, Stingl G, Szalai S, Szepietowski JC, Taïeb A, Werfel T, Wollenberg A, Darsow U; European Dermatology Forum; European Academy of Dermatology and Venereology; European Task Force on Atopic Dermatitis; European Federation of Allergy; European Society of Pediatric Dermatology; Global Allergy and Asthma European Network. Guidelines for treatment of atopic eczema (atopic dermatitis) Part II. J Eur Acad Dermatol Venereol. 2012 Sep;26(9):1176-93. [PMID:22813359].

[51]. Katsarou A, Armenaka M. Atopic dermatitis in older patients: particular points. J Eur Acad Dermatol Venereol. 2011 Jan;25(1):12-8. [PMID: 20569298].

[52]. Fleischer AB Jr, Abramovits W, Breneman D, Jaracz E; US/Canada tacrolimus ointment study group. Tacrolimus ointment is more effective than pimecrolimus cream in adult patients with moderate to very severe atopic dermatitis. J Dermatolog Treat. 2007;18(3):151-7. [PMID: 17538803].

[53]. Ring J, Alomar A, Bieber T, Deleuran M, Fink-Wagner A, Gelmetti C, Gieler U, Lipozencic J, Luger T, Oranje AP, Schäfer T, Schwennesen T, Seidenari S, Simon D Ständer S, Stingl G, Szalai S, Szepietowski JC, Taïeb A, Werfel T, Wollenberg A, Darsow U; European Dermatology Forum; European Academy of Dermatology and Venereology; European Task Force on Atopic Dermatitis; European Federation of Allergy; European Society of Pediatric Dermatology; Global Allergy and Asthma European Network. Guidelines for treatment of atopic eczema (atopic dermatitis) Part II. J Eur Acad Dermatol Venereol. 2012 Sep;26(9):1176-93. [PMID:22813359].

[54]. Dickel H, Bruckner TM, Schmidt A, Diepgen TL. Impact of atopic skin diathesis on occupational skin disease incidence in a working population. J Invest Dermatol. 2003 Jul;121(1):37-40. [PMID: 12839561].

[55]. Ring J, Alomar A, Bieber T, Deleuran M, Fink-Wagner A, Gelmetti C, Gieler U, Lipozencic J, Luger T, Oranje AP, Schäfer T, Schwennesen T, Seidenari S, Simon D Ständer S, Stingl G, Szalai S, Szepietowski JC, Taïeb A, Werfel T, Wollenberg A, Darsow U; European Dermatology Forum; European Academy of Dermatology and Venereology; European Task Force on Atopic Dermatitis; European Federation of Allergy; European Society of Pediatric Dermatology; Global Allergy and Asthma European Network. Guidelines for treatment of atopic eczema (atopic dermatitis) Part II. J Eur Acad Dermatol Venereol. 2012 Sep;26(9):1176-93. [PMID:22813359].

[56]. Werfel T, Breuer K, Ruéff F, Przybilla B, Worm M, Grewe M, Ruzicka T, Brehler R, Wolf H, Schnitker J, Kapp A. Usefulness of specific immunotherapy in patients with atopic dermatitis and allergic sensitization to house dust mites: a multi-centre, randomized, doseresponse study. Allergy. 2006 Feb;61(2):202-5. [PMID: 16409197].

[57]. Schmitt J, Schäkel K, Schmitt N, Meurer M. Systemic treatment of severe atopic eczema: a systematic review. Acta Derm Venereol. 2007;87(2):100-11. [PMID: 17340015].

[58]. Draelos ZD. Therapeutic moisturizers. Dermatol Clin. 2000;18:597- 607

[59]. Buraczewska I, Berne B, Linberg M, Torma H, Londen M. Change in skin barrier function following long-term treatment with moisturizers, a randomized controlled trial. BJD. 2007;156:492-8.

[60]. Buraczewska I, Berne B, Linberg M, Torma H, Londen M. Change in skin barrier function following long-term treatment with moisturizers, a randomized controlled trial. BJD. 2007;156:492-8

IJARSCT
ISSN (Online) 2581-9429
International Journal of Advanced Research in Science, Communication and Technology (IJARSCT)
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
Impact Factor: 7.301
Volume 3, Issue 1, December 2023

[61]. Draelos ZD. Therapeutic moisturizers. Dermatol Clin. 2000;18:597- 607
[62]. Anderson C, Dinulos JG. Are the new moisturizers more effective? Curr Opin Pediatr. 2009;21:486-90.
[63]. Saijic D, Asiniwasis R, Skotnicki-Grant S. A look at epidermal barrier function in atopic dermatitis: Physiologic lipid replacement and the role of ceramide. Skin Therapy Lett. 2012;7:6-9.
[64]. Rawlings AV, Canestrari DA, Dobkowski B. Moisturizer technology versus clinical performance. Dermatol Ther. 2004;17:49-56
[65]. Draelos ZD. Therapeutic moisturizers. Dermatol Clin. 2000;18:597- 607
[66]. Anderson C, Dinulos JG. Are the new moisturizers more effective? Curr Opin Pediatr. 2009;21:486-90.
[67]. Lucky AW, Leach AD, Laskarzewski P, et al. Use of an emollient as a steroid-sparing agent in the treatment of mild to moderate atopic dermatitis in children. Pediatr Dermatol 1997; 14:321-4
[68]. . Sidbury R, Davis DM, Cohen DE, et al. Guidelines of care for the management of atopic dermatitis: section 3. Management and treatment with phototherapy and systemic agents. J Am Acad Dermatol 2014;71:327-49.
[69]. Callen J, Chamlin S, Eichenfield LF, et al. A systematic review of the safety of topical therapies for atopic dermatitis. Br J Dermatol. 2007;156(2):203–221
[70]. Aubert-Wastiaux H, Moret L, Le Rhun A, et al. Topical corticosteroid phobia in atopic dermatitis: a study of its nature, origins and frequency. Br J Dermatol. 2011;165(4):808–814
[71]. Kojima R, Fujiwara T, Matsuda A, et al. Factors associated with steroid phobia in caregivers of children with atopic dermatitis. Pediatr Dermatol. 2013;30(1):29–35
[72]. Huang JT, Abrams M, Tlougan B, et al. Treatment of Staphylococcus aureus colonization in atopic dermatitis decreases disease severity. Pediatrics 2009;123:e808-14
[73]. Doege K, Grajecki D, Zyriax BC, Detinkina E, Zu Eulenburg C, Buhling KJ. Impact of maternal supplementation with probiotics during pregnancy on atopic eczema in childhoodVa meta-analysis. Br J Nutr 2012;107:1-6
[74]. Pelucchi C, Chatenoud L, Turati F, et al. Probiotics supplementation during pregnancy or infancy for the prevention of atopic dermatitis: a meta-analysis. Epidemiology 2012; 23:402- 14
[75]. Klein P, Clark R. An evidence-based review of the efficacy of antihistamines in relieving pruritus in atopic dermatitis. Arch Dermatol 1999;135(12):1522-5.
[76]. Tsakok T, Woolf R, Smith CH, Weidinger S, Flohr C. Atopic dermatitis: the skin barrier and beyond. Br J Dermatol. 2019;180:464–74.
[77]. Bath-Hextall FJ, Jenkinson C, Humphreys R, Williams HC. Dietary supplements for established atopic eczema. Cochrane Database Syst Rev. 2012;15:CD005205
[78]. Pelucchi C, Chatenoud L, Turati F, Galeone C, Moja L, Bach JF, et al. Probiotics supplementation during pregnancy or infancy for the prevention of atopic dermatitis: a meta- analysis. Epidemiology. 2012;23:402–14
[79]. Heimall J, Spergel JM. Filaggrin mutations and atopy: consequences for future therapeutics. Expert Rev Clin Immunol 2012;8:189–197