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A REVIEW OF ECZEMA RESEARCH AND MANAGEMENT

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ABSTRACT
Eczema, also known as atopic dermatitis, is a chronic inflammatory skin condition characterized by red, itchy, and scaly patches. It primarily affects the epidermis and can occur at any age, though it often begins in childhood. Eczema encompasses several types, including atopic dermatitis, contact dermatitis (both irritant and allergic), dyshidrotic eczema, nummular eczema, seborrheic dermatitis. Atopic dermatitis, a persistent inflammatory skin condition that impacts 10% of adults and up to 20% of youths around the world, affects over 200 million people. While adult-onset dermatitis accounts for one in four cases of atopic dermatitis, the condition is more commonin children and often worsens into maturity. The pathogenesis of eczema involves a complex interplay between genetic predisposition, immune system deregulations, and environmental factors. Genetic mutations affecting the skin barrier function, such as those in the filaggrin gene, contribute to increased susceptibility by allowing irritants and allergens to penetrate the skin. In severe cases, systemic treatments or immuno modulators may be required. While there is no cure, ongoing research into the underlying mechanisms and new therapeutic approaches aims to improve long-term management and patient outcomes.

KEYWORDS: Eczema, Types, Pathogenesis, Symptoms, Treatment.

1. INTRODUCTION

Eczema is a skin disorder that can result in oozing/weeping redness, swelling, scaling, and dryness. The most common kind of eczema is called atopic dermatitis, or atopic eczema. When the majority of medical professionals and patients refer to "eczema," they mean this. Atopic dermatitis, a persistent inflammatory skin condition that impacts 10% of adults and up to 20% of youths around the world, affects over 200 million people. While adult-onset dermatitis accounts for one in four cases of atopic dermatitis, the condition is more commonin children and often worsens into maturity. A compromised skin barrier, elevated immune cell activity in the skin, and the micro biome the community of microorganisms that live on the skin are every aspect of the development of atopic dermatitis.[1] About one in five children suffer from eczema, also known as atopic dermatitis or atopic eczema. The condition is becoming more common and has a significant negative impact on people's capacity and quality of life to pursue occupations. Eczema typically begins in childhood and often persists into maturity. Asthma, allergic rhinitis, and food allergies are among the atopic disorders that children with eczema are more likely to acquire. Since food allergies typically emerge after dermatitis, and because eczema with an early onset is closely linked to food allergies, treating eczema could stop food allergies from developing.[2]

2. CLASSIFICATION OF ECZEMA

- Contact Dermatitis
- Dyshidrotic Eczema
- Neurodermatitis
- Nummular Eczema
- Sebhorrheic Dermatitis
- Atopic Dermatitis

2.1. CONTACT DERMATITIS

Contact dermatitis is an inflammatory eczematous skin disorder. The syndrome is caused by contact allergens, which are tiny reactive compounds that change proteins and elicit immunological responses that are either innate and adaptive or contact irritants, which are substances or metal ions that injure without inducing a T-cell response.

Contact dermatitis can be divided into two categories: irritant-induced and allergy-induced. Irritant contact dermatitis is a generic skin reaction to direct chemical injury that releases mediators of inflammation

predominantly from epidermal cells, whereas allergic contact dermatitis is a delayed (type 4) hypersensitivity response to external contact antigens. The combination of T cells and cytokines induces an immunological response.[3]

2.2. DYSHIDROTIC ECZEMA

Mostly on the palmoplantar area are the lateral and ventral sides of the fingers, pruritic, tiny, tense vesicles are the hallmark of dyshidrotic eczema, often referred to as dyshidrotic dermatitis or pompholyx. Histological analysis reveals an eczematous reaction around the sweat ducts that is not connected with anomalies of the sweat ducts, despite the fact that its genesis appears to be related to sweating, as Dyshidrotic eczema typically happens to a person who has hyperhidrosis during the spring allergy season.[4]

2.3. NEURODERMATITIS

An itching patch of skin is the initial sign of neurodermatitis. It itches more when you scratch it. More scratching results in thick, leathery skin. Several itching spots may appear; these are usually on the neck, wrists, forearms, legs, or groin area.

However, the itching can be so severe that it impairs your quality of life, sleep, and ability to conceive.

2.4. NUMULAR ECZEMA

Well-defined round or oval plaques, or nummular dermatitis, are typically dispersed asymmetrically throughout the limbs (Fig. 1). Nummular dermatitis is uncommon before the age of five, in contrast to classic AD, and itching is not a prevalent symptom.48 Since many occurrences of nummular dermatitis are linked to micro trauma (such as bug bites or scratching), as well as other possible causes such S aureus infection, contact allergens or irritants, and xerosis, the etiology of the condition is unknown.[5]

2.5. SEBHORRHEIC DERMATITIS

With a 5% global prevalence, seborrheic dermatitis (SD) is a widespread, chronic, inflammatory illness that affects people of all ages, races, and ethnicities. SD lesions are thin, erythematous, scaly plaques and patches that usually affect the central face, nasolabial folds,ears, eyebrows, and scalp. They can also affect areas with facial hair. The quality of life is significantly impacted negatively by SD, especially in individuals who have moderate to severe illness. The goal of current SD treatments is to reduce inflammation or stop the growth of Malassezia spp. in order to treat SD signs and symptoms such pruritus, erythema, and scaling.[6]

2.6. ATOPIC DERMATITIS

Atopic dermatitis, sometimes known as atopic eczema, is a persistent inflammatory skin disorder marked by scaly, erythematous, and itchy skin lesions that are often restricted to the body's surfaces that bend. As part of an allergic triad, it can manifest as asthma and allergic rhinitis; asthma is thought to occur later in life in 30% of children with atopic dermatitis.[7]

Skin barrier failure, which causes dry, itchy skin and prompts scratching that causes mechanical damage, allergic sensitization to environmental antigens, and allergic skin inflammation, are the characteristics of AD.[8]

3. EPIDEMIOLOGY

Approximately 20% of people may experience atopic dermatitis at some point in their lives, while the disease's incidence varies widely across the globe. Between 1950 and 2000, the frequency rose significantly in various so-called industrialized countries, to the point where many describe to it as the “allergic epidemic.” However, current data suggests that in other nations, like the UK and New Zealand, where eczema prevalence was once extremely high, symptoms have leveled out or even declined. This suggests that the global epidemic of allergicdiseases is not growing steadily. However, atopic dermatitis continues to be a major health risk and is still becoming more common in many nations, especially in the developing world.[9]

4. ETIOLOGY OF ECZEMA

The etiology of atopic dermatitis is complex, involving both environmental and genetic variables that result in immune system and epidermal abnormalities. Genetic changes include loss of function mutations of the epidermal protein filaggrin (Filament Aggregating Protein), which is broken down into element of natural moisturization. Up to 30% of individuals with atopic dermatitis have mutations in the filament grin gene, which may also put them at risk for ichthyosis vulgaris, allergic rhinitis, and keratosis pilaris. In 10% to 30% of cases, food hypersensitivity may potentially be the cause of or a worsening of atopic dermatitis. In ninety percent of, cases, peanuts, milk, egg, wheat and soy are the trigger for these reactions or flares. It appears from recent research that smoking and adult-onset AD may be related.[10]

5. PATHOPHYSIOLOGY OF ECZEMA

The pathophysiology of AD is dynamic and complex and involves environmental variables, IgE-induced hypersensitivity, changes in cell-mediated immune responses, and barrier failure. Moreover, it is caused by a complicated interaction between the immune system, the host's environment, the altered genes, and drug abnormalities.

5.1. Skin barrier dysfunction

A multitude of physical, chemical, and biological factors that cause AD are kept out of the body by the physicochemical shield of the skin.[11] Atopic dermatitis is more common in people who have mutations in the filaggrin gene.

In the stratum granulosum and stratum corneum,

structural proteins encoded by the Filaggrin gene aid in maintaining the adhesion of keratinocytes. This keeps the stratum corneum moist and protects the skin barrier. Eczema is eventually caused by reduced Filaggrin production as a result of gene abnormalities, which also weakens the skin's protective barrier and promotes trans epidermal water loss.[9]

5.2. Immunological factors

It's still unclear which major pathway causes skin inflammation. This may be caused by T- cell interaction with allergens that have passed through the skin barrier, or it may be brought on by scratching the skin, which causes the keratinocytes to generate pro- inflammatory cytokines. In the pathophysiology of AD, both non-adaptive and adaptive immunity play significant roles.[11]

5.3. Environmental and microbial factors

AD is the outcome of genetic and environmental factors working together. Food, smoking, airborne allergens, bacteria, viruses, soaps, and detergents are just a few examples of environmental factors that can damage the epidermal barrier and initiate the inflammatory process in AD.

5. 3.1 Airborne allergens

Allergens in the air have the potential to cause inflammation in skin that is prone to atopic dermatitis. The most frequent cause of airborne allergens is house dust mites (HDM), which are derived from the species Dermatophagoides pteronyssinus and D.farina. The HDM's enzymatic activity can break strong epithelial connections, so destroying the skin barrier.

5.3.2 Microbial exposure

Atopic skin colonization by S. aureus is caused by a number of factors, including elevated skin pH, decreased ceramide levels, elevated fibronectin concentration, and low levels of antimicrobial peptides. Additionally, it is known that S. aureus secretes enterotoxins A, B, and toxin-1, which greatly exacerbates AD. Additionally, S. aureus secretes enzymes thatbreak down corneodesmosomes. Compared to non-AD patients, AD patients are more susceptible to skin diseases such as Verruca vulgaris and Molluscum contortus.[11]

6. MANAGEMENT OF ECZEMA

6.1. BATHING AND MOISTURIZERS

It makes sense to suggest bathing in warm, not hot, water for five to ten minutes once a day. When cleaning surfaces, soap should only be used absolutely necessary. In this case, it is recommended to use a mild cleanser such as Basis, Cetaphil, Dove, or Kiss My Face. As soon as possible after taking a bath, patients should apply a thick layer of moisturizer such as Baby Oil, Mineral Oil, Eucerin, Aquaphor, or Moisture to prevent their skin from drying out completely.[12]

6.2. OCCLUSION, SOAKS AND SKIN PROTECTION

Occlusion can provide the highest amount of moisture to severely damaged skin in addition toemollient treatment. Gloves or plastic wrap applied to small areas can offer hand protection. But keep in mind that the danger of adverse effects and systemic absorption is increased when occlusive techniques are used in addition to topical corticosteroids.

6.3. ANTIHISTAMINES AND ANTIDEPRESSANTS

Tricyclic antidepressants or antihistamines may be used to treat pruritus when moisturizers and other conservative measures are ineffective. Older sedatives like diphenhydramine (Benadryl) and hydroxyzine (Atarax) are more effective at controlling pruritus than are the more modern, nonsedating histamines.[13] One optionis to try a nonsedating antihistamine to see if it helps if tiredness is an issue. Sedative, antihistaminic, and antipruritic properties are also present in tricyclic antidepressants including amitriptyline (Elavil) and doxepin (Sinequan).[14]

6.4. ANTIBIOTICS

Secondary infections should be treated with antibiotics. First-generation cephalosporins, macrolide antibiotics, dicloxacillin (Pathocil), and clindamycin (Cleocin) are examples of appropriate medications. If a patient consistently contracts bacterial infections without taking medicine, they may be suffering from chronic immunosuppression. Using cloths soaked in a solution of aluminum acetate or saline, the afflicted regions are to be topically treated for secondary infected crusted lesions.

6.7. PHOTOTHERAPY

Phototherapy works well for atopic dermatitis that is recalcitrant. UVB, UVA, or a mix of UVA and UVB radiation can be used to deliver this treatment. Psoralen in conjunction with ultraviolet A (PUVA) photochemotherapy may be helpful for patients with severe refractory disease.[15]

6.8. IMMUNOSUPPRESSANTS AND ANTINEOPLASTICS

Patients with refractory atopic dermatitis have claimed success with cyclosporine (Sand immune). When therapy is interrupted, the problem sometimes resurfaces, though usually not to the same degree.[16] The oral medication tacrolimus is used to prevent organ transplant rejection; a topical form isalso marketed under the name Prograf. With minimal side effects, this topical medication seems to be beneficial in treating refractory atopic dermatitis.[27]

BIBILOGRAPHY

1. JAMA, 2023; 329(3): 268. doi:10.1001/jama.2022.21457
2. Chalmers, J. R., Haines, R. H., Bradshaw, L. E., Montgomery, A. A., Thomas, K. S., Brown, S. J., Williams, H. C. (2020). Daily emollient during

infancy for prevention of eczema: the BEEP randomised controlled trial. The Lancet. doi:10.1016/s0140-
3. Contact Dermatitis Graham Litchman; Pragya A. Nair; Amber R. Atwater; Beenish S.Bhutta
4. Nishizawa, A. (2016). Dyshidrotic Eczema and Its Relationship to Metal Allergy. Current Problems in Dermatology, 80–85. doi:10.1159/000446785 10.1159/000446785
5. Silverberg, N. B., & Durán-Mc Kinster, C. (2017). Special Considerations for Therapy of Pediatric Atopic Dermatitis. Dermatologic Clinics, 35(3): 351–363. doi:10.1016/j.det.2017.02.008 10.1016/j.det.2017.02.008
6. Roflumilast foam 0.3% for adolescent and adult patients with seborrheic dermatitis: A randomized, doubleblinded, vehicle-controlled, phase 3 trial Andrew Blauvelt, MD, MBA, a Zoe D. Draelos, MD, b Linda Stein Gold, MD, c Javier Alonso-Llamazares, MD, PhD, d Neal Bhatia, MD, e Janet DuBois, MD, f Seth B. Forman, MD, g Melinda Gooderham, MD, h Lawrence Green, MD, i Scott T. Guenthner, MD, j, kAdelaide AHebert, MD, l Edward Lain, MD, m Angela Y. Moore, MD, n, o Kim A. Papp, MD, PhD, p, q Matthew Zirwas, MD, r, s, tSaoriKato, BSN, u Scott Snyder, PharmD, u David Krupa, MS, u Patrick Burnett, MD, PhD, u David R. Berk, MD, u and David H. Chu, MD, PhDuhttps: //doi.org/10.1016/j.jaad.2023.12.065
7. Atopic Dermatitis: An Overview REBECCA BERKE, MD; ARSHDEEP SINGH, MD; and MARK GURALNICK, MD Kaiser Permanente's Woodland Hills Family Medicine Residency Program, Woodland Hills, California.
8. Cellular and Molecular Mechanisms in Atopic Dermatitis Michiko K. Oyoshi, RuiHe, Lalit Kumar, Juhan Yoon, and Raif S. GehaDOI:10.1016/S0065-2776(09)01203-6
9. Thomsen, S. F. (2014). Atopic Dermatitis: Natural History, Diagnosis, and Treatment. ISRN Allergy, 2014; 1–7. doi:10.1155/2014/354
10. A topic DermatitisLogan Kolb; Sarah J. Ferrer-Bruker.
11. Deepa S. Mandlik & Satish K. Mandlik (2021) Atopic dermatitis: new insightinto the etiology, pathogenesis, diagnosis and novel treatment strategies, Immunopharmacologyand Immunotoxicology, 43: 2, 105-125, DOI:10.1080/08923973.2021.1889583.
12. Lookingbill D, Marks JG.Eczematous rashes. In: Principles of dermatology. 2d ed. Philadelphia: Saunders, 1993; 127–30.
13. Wahlgren CF, Hagermark O, Bergstrom R. The antipruritic effect of a sedative and a non- sedative antihistamine in atopic dermatitis.Br J Dermatol, 1990; 122: 545-51.
14. Hanifin JM. Role of antihistamines in atopic dermatitis.J Allergy Clin Immunol, 1990; 86(4 pt 2): 666-9.
15. Fitzpatrick TB, ed. Dermatology in general medicine. 4th ed. New York: McGraw-Hill, 1993.
16. Berth-Jones J, Graham-Brown R, Marks R, Camp RD, English JS, Freeman K, et al. Long-term efficacy and safety of cyclosporin in severe atopic dermatitis. Br J Dermatol, 1997; 136: 76-81.
17. Boguniewicz M, Fiedler VC, Raimer S, Laurence ID, Leung DY, Hanifin JM. A randomized vehicle-controlled trial of tacrolimus ointment for treatment of atopic dermatitis in children.J Allergy Clin Immunol, 1998; 102: 637-44.