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Review Article

A Review of Eczema: Current Therapies and Emerging Treatments

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ABSTRACT

Atopic dermatitis, commonly known as eczema, is a chronic relapsing skin disease characterized by pruritus, disruption of the epidermal barrier function, and immunoglobulin E-mediated sensitization to food and environmental allergens, which can lead to morbidity. Eczema is not always easy to control and every physician should know the essential principles of treatment. Symptoms, such as inflamed papules, plaques, and itching, and associated consequences, such as sleep disturbances, can have a significant impact on the quality of life of the patient and his family. Basic pharmacological treatment includes topical corticosteroids. Twice daily or more frequent application has not been shown to be more effective than once daily application. Maintenance treatment with topical corticosteroids may help prevent relapse in patients with moderate to severe atopic dermatitis. Topical calcineurin inhibitors (TCIs) are able to inhibit T-cell activation, thereby reducing inflammation.

INTRODUCTION

Eczema, also known as atopic dermatitis, is a persistent inflammatory skin disease characterized by itchy, red, erythematous, scaly skin lesions that often appear on the flexural surfaces of the body. It may present with asthma and allergic rhinitis as part of the allergic triad; approximately 30% of children with atopic dermatitis develop asthma later in life. [1] Clinically, the condition shows significant variation. AD is characterized by tender, intensely pruritic eczema-like lesions, which may be confined to a specific area or be widespread. Features such as juvenile plantar dermatitis, coin-shaped eczema, prurigo nodularis, and lesions. AD is not uncommon [2, 3]. The common factor linking these allergic disorders is atopy, the predisposition to immunoglobulin E (IgE)-mediated responses to environmental triggers. The term dermatitis comes from the Greek words “derma,” meaning skin, and “itis,” meaning inflammation. Inflammation and elevated immunoglobulin E (IgE) levels are seen in approximately half of all patients with this disease, and therefore atopic dermatitis is not a completely

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accurate term, as the complexity of AD is reflected in a pathogenesis involving a dysregulated immune system, an impaired skin barrier, and a variety of triggering factors. Examples of risk factors include mutations in the filaggrin gene, familial predisposition, and hard water in the home, with new risk factors being discovered continuously [4,5,6]. In most cases, AD occurs during early childhood, but late-onset forms account for 20% of adult AD cases. [7]

Etiology and Pathogenesis

Eczema, also known as atopic dermatitis, is a complex and multifactorial disease. The etiology of eczema involves a combination of genetic predisposition, environmental factors, an imbalanced immune response, skin barrier dysfunction, and micro biome imbalance. Genetic mutations that affect skin barrier function and immune response, as well as family history, play an important role. Environmental factors, such as exposure to allergens, irritants, and stress, can trigger or worsen eczema symptoms. The pathogenesis of eczema involves disruption of the skin barrier, activation of immune cells, release of cytokines and chemokine, inflammation, and skin damage. Chronic inflammation and skin remodeling lead to long-term skin damage and the development of associated diseases. The development of atopic dermatitis is thought to be due to a genetic defect in the filaggrin protein that causes atopic dermatitis to break down the epidermis. Scratching can lead to disruption and further inflammation of the epidermal barrier of the skin, the cycle called itch-scratch. [8] Additionally, the immunological imbalance theory states that atopic dermatitis results from dysregulation of T cells, particularly T helper cells types 1, 2, 17, and 22 as well as regulatory T cells.[9]

Genetic Factors

Many genes have been linked to atopic dermatitis, especially those involved in the structure of the epidermis and genes that encode key elements of the immune system. A remarkable recent discovery is the strong association documented between eczema and mutations in the filaggrin gene, on a specific chromosome [10]. The filaggrin genus is known as the largest Important genetic risk factor for eczema. Approximately 10% of people in Western populations carry mutations in the filaggrin gene, while approximately 50% of all patients have such mutations. Mutations in the filaggrin gene lead to functional deficiencies of the filaggrin protein that compromise the skin barrier. Clinically, this expression of these deficiencies results in dry or cracked skin and an increased risk of eczema. However, not all patients with atopic dermatitis carry these mutations and other genetic variants also contribute to the disease. [11]

Types of eczema

There are many types of eczema; some are unexplained forms and some types: [12, 13, 14]

Atopic Dermatitis

This is the most common form of eczema. It is closely related to asthma and hay fever. It is generally inherited and affects both adults and children. Symptoms include severe itching, burning, dryness and inflammation of the skin. The exact cause of AD is unknown, but it is thought to involve a combination of genetic, environmental, and immune factors. Triggers, such as allergens, irritants, stress, and hormonal changes, can aggravate the condition. Symptoms include itching, redness, swelling, and thickening of the skin. Diagnosis is based on physical examination, history, and skin tests. Treatment options include topical corticosteroids, moisturizers, immunomodulators, and systemic

therapies, as well as lifestyle changes such as avoiding triggers, maintaining a consistent skin care routine, and managing stress.

Allergic Contact Dermatitis

As the name suggests, this form of eczema is caused by an allergic reaction to substances that come into contact with the skin, such as nickel and perfume. Allergic contact dermatitis (ACD) is a type of skin inflammation that occurs when the skin comes into contact with an allergen, triggering an immune response. This response results in the release of chemical mediators, which cause blood vessels to dilate and lead to the characteristic symptoms of ACD, including redness, itching, swelling, blistering, and crusting. Common allergens that can cause ACD include metals such as nickel and chromium, perfumes, dyes, rubber, and certain plants such as poison ivy and poison sumac. Once the allergen is identified, treatment usually involves avoiding further exposure, using topical corticosteroids to reduce inflammation, and applying cold compresses to relieve itching and discomfort. In severe cases, oral antihistamines or corticosteroids may be needed to manage symptoms.

Irritant Contact Dermatitis

Again, as the name suggests, this form of eczema is caused by a reaction to substances that come into contact with the skin. Unlike allergic contact dermatitis, this form is caused by direct exposure to contact irritants caused by contact with detergents and chemicals. ICD can progress through acute, sub-acute, and chronic stages, and diagnosis is made by physical examination, medical history, and patch testing. Treatment involves eliminating the irritant, applying topical corticosteroids and moisturizers, and avoiding further irritation. Prevention is key, and people can protect themselves by using protective equipment, following safety protocols, and avoiding skin contact with irritants. In vitro models, such as reconstructed human epidermis and keratinocyte cell culture, can be used to study ICD, while in vivo models, including human patch tests and animal models, can also be used.

Infantile Seborrhoeic Dermatitis

This is a common form of eczema in babies under one year old, also known as cradle cap, which usually starts on the scalp or nappy area before spreading. It often affects It does not cause discomfort to the child as it is completely harmless and usually lasts a few months before disappearing completely. It is characterized by a thick, yellow, greasy crust on the scalp, face, and neck, accompanied by redness and inflammation. The exact cause is not known, but it is thought to be related to hormonal changes, sensitivity to soaps and shampoos, and fungal infections. Symptoms are usually mild and may include itching, scaling, and burning. Treatment usually includes gentle shampooing, application of olive oil, and topical antifungal creams. In severe cases, medicated shampoos or creams may be prescribed.

Varicose Eczema

This type of eczema usually affects the lower legs of middle-aged and elderly people and results from poor blood circulation. Varicose eczema, also known as stasis dermatitis or gravitational eczema is a type of skin inflammation that occurs in people with varicose veins or other circulatory problems. It usually affects the lower legs, especially the ankles and calves, where blood tends to pool due to gravity. Reduced blood flow and increased pressure cause fluid to leak out of the veins and into the surrounding tissues, leading to inflammation, itching, and thickening of the skin. Symptoms of varicose eczema include redness, swelling, warmth, and tenderness, as well as

crusting, scaling, and ulceration in severe cases. Treatment usually includes elevating the affected limb, wearing compression stockings, and applying topical corticosteroids to reduce inflammation. In some cases, surgery may be necessary to treat underlying circulatory problems.

Discoid Dermatitis

This form of eczema can also affect the lower legs of adults and is characterized by symptoms such as red, coin-shaped areas on the skin that may peel and be extremely itchy. Treatment for discoid dermatitis usually includes applying moisturizers to keep the skin hydrated, using topical corticosteroids to reduce inflammation, and avoiding triggers that can aggravate the condition. In some cases, phototherapy or oral antihistamines may also be recommended to manage symptoms.

Current therapies

Emollients

Xerosis (dry skin) is a common feature of atopic dermatitis, and many patients find that managing the xerosis controls their dermatitis. Emollients are topical agents used to moisturize and soften the skin, relieving dryness, itching, and irritation. They work by creating a physical barrier on the skin's surface to prevent water loss, attracting and retaining moisture from the air, and filling the spaces between skin cells to improve skin elasticity. Emollients come in a variety of forms, including creams, ointments, lotions, and gels, and are commonly used to manage dry skin conditions, such as eczema, psoriasis, and ichthyosis.[15] Regular use of emollients can help improve skin hydration, reduce itching and irritation, and prevent complications of dry skin. Therefore, patients are advised to apply emollients to the entire body, regardless of whether active symptoms are present. [16]

Topical corticosteroids

Topical corticosteroids are a treatment for flare-ups of atopic dermatitis. [17] Systemic corticosteroids are reserved for severe cases of atopic dermatitis that are refractory to treatment. While oral corticosteroids can reduce the lesions of atopic dermatitis, a flare-up of the disease can occur when these medications are discontinued. [18] When systemic corticosteroids are used for a severe flare of atopic dermatitis, the risk of rebound effect can be reduced by gradually reducing the dose of the drug, while they should always be used in combination with topical corticosteroids, because the combination is more effective than corticosteroids alone. [19]

Phototherapy

Phototherapy is effective in treating atopic dermatitis that does not respond to standard therapies. options that include ultraviolet A (UVA), ultraviolet B (UVB), or a combination of both. Psoralen plus UVA (PUVA) photochemotherapy may be considered in patients with extensive refractory disease. [20]

Calcineurin inhibitors

Current calcineurin inhibitors, such as pimecrolimus (Elidel) and tacrolimus (Protopic), are immunomodulatory agents and are used as second-line therapy. [21] Topical pimecrolimus and tacrolimus are both non steroidal anti-inflammatory drugs that are used specifically for areas such as the face and folds, including around the eyelids, to prevent skin atrophy and other complications. Caution is warranted when using potent topical corticosteroids in sensitive areas. [22, 23, 24]

Topical phosphodiesterase-4 inhibitors

The phosphodiesterase-4 inhibitor crisaborole, a topical inhibitor that also reduces inflammation,

Crisaborole Ointment 2%, has demonstrated efficacy and tolerability and was approved by the Australian Therapeutic Goods Administration (TGA) in 2019 as a treatment option for atopic dermatitis. Topical phosphodiesterase-4 (PDE4) inhibitors are a class of drugs that have shown promise in the treatment of various inflammatory skin conditions, including psoriasis, atopic dermatitis, and eczema. PDE4 is an enzyme that plays a key role in the regulation of inflammatory responses in the skin. By inhibiting PDE4, these topical agents reduce the production of pro-inflammatory cytokines and mediators, thereby decreasing inflammation and promoting skin healing. Topical PDE4 inhibitors, such as crisaborole and apremilast, have been shown to be effective and safe in clinical trials, with benefits including reduced inflammation, improved skin symptoms, and enhanced quality of life. [25]

Emerging treatment

Biologics

Atopic dermatitis is a chronic skin condition characterized by redness, lichenification, and itching. Historically, treatment options have focused primarily on local and systemic immunosuppressive therapies or UV light therapy; however, with the development of biologics, therapies that more specifically target atopic inflammation have become available. [26]

Dupilumab

Dupilumab is a fully human monoclonal antibody targeting the interleukin (IL)-4 receptor subunit (IL-4Ra) that blocks the signaling of the type 2 cytokines IL-4 and IL-13, approved for use in the treatment of adult patients with moderate to severe atopic dermatitis since 2017. [27] Recently, dupilumab has become the first systemic treatment for atopic dermatitis, offering superior efficacy and safety compared to traditional nonspecific immunosuppressive agents. [28,29] It is FDA-approved for the treatment of eczema and has a generally favorable safety profile, although some side effects are commonly reported, such as injection site erythema, conjunctivitis, and occasionally dermatitis of the head and neck (30-32). Dupilumab is more expensive than some older systemic drugs, such as eclosporin or methotrexate, but its safety profile and long-term efficacy in controlling severe disease outweigh the cost of dupilumab [33,34]

JAK inhibitors

Janus kinase (JAK) signaling and activation of transcription (STAT) pathways modulate several important immune pathways, including Th2 (IL-4, IL-5, IL-6, IL-10, IL-13, IL-31, CCL [chemokine). (C-C motif) ligand] 18), Th22 (IL-22, S100As), Th1 [IL-2, IFN-c and TNF (tumor necrosis factor)-b] and Th17 (IL-17A, IL-17F, IL-21, IL-22, IL-23R), [35] Oral JAK inhibitors such as baricitinib and upadacitinib were approved by the TGA in 2021, with upadacitinib added to the PBS from February 2022. These drugs are indicated for moderate to severe atopic dermatitis in patients aged 18 to 24 years. 12 years and older who require systemic treatment. However, JAK inhibitors carry potentially serious risks, including opportunistic infections, malignancies related to immunosuppression, myelosuppression, venous thromboembolism and cardiovascular events. The severity of these risks may vary between agents.[36]

Delgocitinib

Delgocitinib is a novel pan-JAK inhibitor specific for JAK1, JAK2, JAK3, and TYK2 kinases [37]. By disrupting multiple cytokine-mediated signaling pathways, it inhibits inflammation and may therefore be a suitable therapeutic agent for topical use in hand eczema. In a recent proof-of-

concept study, the efficacy of topical delgocitinib was evaluated compared to placebo in randomized patients. [38]

Eczema prevention [39, 40, 41, 42]

Preventing eczema requires a multifaceted approach that includes avoiding triggers, maintaining a healthy lifestyle, and seeking medical intervention when necessary. People with a family history of eczema, asthma, or allergies should be especially vigilant, as they are more likely to develop this condition. Avoiding allergens and irritants, such as pollen, dust mites, and harsh soaps, can help prevent symptoms. Keeping your skin moisturized, wearing breathable clothing, and avoiding scratching can also help. In addition, managing stress through exercise, getting enough sleep, and eating a balanced diet can help reduce the risk of eczema. Medical interventions, such as topical corticosteroids, immunomodulators, and phototherapy, can also be effective in preventing and managing symptoms. By taking a proactive approach to prevention and working with a healthcare professional to develop a treatment plan, people can reduce their risk of developing eczema and manage symptoms effectively.

CONCLUSION

Eczema, remains a challenging condition due to its chronic nature, impact on complex etiology. current therapies, including topical corticosteroids ,calcineurin inhibitors, emollients and new therapeutic options such as biologics, janus kinase inhibitors these treatments target the underlying immune dysfunction in eczema, providing more specific, long lasting and effective interventions. However, more research is needed to understand the long term effects of these therapies, and efforts must be made to improve access to these treatments. Further research is needed to fully understand the pathophysiology of eczema and to develop more effective and personalized treatments. This review highlights the current state of knowledge on eczema and its treatment, and identifies areas for future research.

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