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An Overview on Diagnosis and Management of Atopic Dermatitis in Primary Health Care Center

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ABSTRACT

Atopic dermatitis is considered one of the very common conditions that counter primary care physicians. It can affect any age; however, it tends to be more in the pediatric population. Diagnosis is mainly clinical, and the treatment depends on the stage of the disease. Different modalities are available in primary care. The PubMed database was utilized to select articles, and the following keys were used in the Mesh ((atopic dermatitis "[Mesh]) AND (primary care"[Mesh]) OR (atopic dermatitis in primary care Mesh])). Regarding the inclusion criteria, the articles were chosen to include one of the following topics: atopic dermatitis, recent diagnosis, and treatment in primary care. All other articles without any of the mentioned topics as their primary endpoint were the exclusion criteria.
The incidence, etiology, and management options were analyzed. Atopic dermatitis is a broad topic; many aspects are still unclear, and many need more work to improve patient outcomes.

Keywords: *Atopic dermatitis, Corticosteroid, Moisturization, Primary care*

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INTRODUCTION

Atopic Eczema (AE), also known as atopic dermatitis, is a complicated, recurrent skin condition involving an inflammatory, disturbingly itchy reaction. Its prevalence has been increasing lately (Galli *et al*., 2020). It can be acute or chronic. Mostly in children, but it can affect any age.

AE is common, and several factors can trigger or aggravate it. These factors can be environmental or due to diet, psychiatric challenges, or genetics. The causes are likely to be multifactorial yet remain unclear (Weidinger & Novak, 2016). Therefore, it is important to get AE diagnosed righteously in primary care. That is because the earlier, the better. Diagnosing AE is challenging, yet it should become familiar to primary care physicians. This article is dedicated to helping make that possible. Treatment mainly focuses on hydrating the affected area as AE results from defects in the function of the skin barrier, environmental and infectious agents, and immunological malfunction. Restoring the skin barrier will be the mainstay of treatment. This includes repairing and hydrating the skin, limiting pruritus, and reducing inflammation during flare-ups or when necessary. This will be possible by educating the patient or the caregiver, good skincare routines, being aware of any potential skin infections, and anti-inflammatory therapy using topical corticosteroids plus or minus topical calcineurin inhibitors (Fleming *et al*., 2020).

The purpose of this review is to discuss atopic dermatitis, the common etiologies, how to diagnose and treat in primary care, and when to seek emergency department help.

MATERIALS AND METHODS

The PubMed database was utilized to select articles, and the following keys were used in the Mesh ((atopic dermatitis

"[Mesh]) AND (primary care"[Mesh]) OR (atopic dermatitis in primary care Mesh])).

Regarding the inclusion criteria, the articles were chosen to include one of the following topics: atopic dermatitis, recent diagnosis, and treatment in primary care.

All other articles without any of the mentioned topics as their primary endpoint were the exclusion criteria.

Around 90 publications were chosen as the most clinically relevant out of 1,202 articles indexed in the previous two decades, and their full texts were evaluated. A total of 31 of the 90 were included after a thorough examination. Additional research and publications were found using reference lists from the recognized and linked studies. Expert consensus recommendations and commentary were added to help practicing physicians assess cirrhosis most simply and practically possible.

RESULTS AND DISCUSSION

Pathophysiology

The pathophysiology of AE is complicated and largely involves a lot of factors like epigenetic, genetic, and environmental factors, including dietary habits (obesity, breastfeeding, etc.) and vitamin D levels (Galli *et al*., 2020). Also, exposure to allergens, pollutants, or antibiotics can be risk factors and a cesarean section, immune system defects, skin barrier damage, and microbiome (Galli *et al*., 2020). The skin barrier inadequacy, the malfunction of all innate and adaptive immunity, and the changed skin pH play a primary role in the pathological mechanism. In contrast, the change of the skin microbiome catalyzes the acceleration of the faulty immune response. As mentioned, AE does not have any single genetic cause; nevertheless, mutations in Filaggrin (FLG) have a critical role (Drislane & Irvine, 2020). Inadequate Filaggrin weakens skin barrier integrity and enhances epidermal permeability, in addition to that predisposing individual with AE to irritant and allergic reactions (Drislane & Irvine, 2020). FLG mutations are positively correlated with the development of atopies, including asthma and rhinitis in children, which form the "atopic triad" (Drislane & Irvine, 2020). The classic atopic triad includes atopic eczema, asthma, and allergic rhinitis (or allergies in general) (Galli *et al*., 2020).

Clinical manifestations and differential diagnosis

AE is generally identified by these many erythematous, scaly, and ill-defined plaques. The neighboring unaffected skin can have itchiness and extreme dryness (xerosis). AE has two clinical presentations, an acute or a chronic presentation (Fleming *et al*., 2020). Also, it can be a combination of both, acute on top of chronic. Papulovesicular eruptions characterize acute presentations with erythema, oozing, edema, and excoriation, while chronic presentations are characterized by lichenification (skin thickening) and xerosis (Fleming *et al*., 2020).

When it comes to the age group that AE affects, it appears mostly early in our lives, yet it still can befriend us through our lifetimes (Fleming *et al*., 2020). It is most common in children and infants yet can still grow to adulthood. AE appears differently in each age group. Let us learn how it shows accordingly. Starting with infants, AE involves the neck, head, and extensor limb surface; the trunk can also be involved; even so, the diaper area is commonly spared (Weidinger & Novak, 2016). Second in line come the children. AE is presented with a shift towards chronic lesions on the flexural folds all over the hands, feet, ankles, wrists, and popliteal and antecubital fossae (Weidinger & Novak, 2016). There is a higher frequency of facial involvement in infants than in children. Finally, AE in adults. It usually affects the flexural areas just like in children and has a wider variety of secondary types, where the hands, nipples, genitalia, and eyelids are exclusively affected (Silverberg *et al*., 2019).

Regarding the common differential diagnosis of AE, the following can be counted: scabies infestation, plaque psoriasis, allergic contact dermatitis, irritant contact dermatitis, and seborrheic dermatitis.

Each of which had special morphologies, distributions, and signs and symptoms. *Seborrheic dermatitis* has the morphology of poorly defined areas of erythema with a greasy scale. The distribution of nose, nasolabial folds, eyebrows, glabella, and scalp; especially in children, the cradle-cap and diaper area are often affected. Its signs and symptoms in children resolve spontaneously within two weeks (Gupta & Bluhm, 2004). It has the morphology of acute to chronic eczematous lesions moving to irritant contact dermatitis. The distribution is restricted to the area of contact. Presents with burning sensation or itching (Barrett & Luu, 2017). At the same time, a*llergic contact dermatitis* has the same features except that its distribution is initially limited to the exposure area but is more probable to spread around. Then *plaque psoriasis* has the morphology of bright beefy-red, well-circumscribed plaques with silvery micaceous scales. The distribution is broad with extensor surface involvement, and the diaper area is often affected in children. The most important signs and symptoms are nail changes, including pitting and family history of psoriasis and genetics (Barrett & Luu, 2017).

Diagnosis

The diagnosis of AE remains solely clinical to its typical features on physical examination, as there are no specific laboratory or histologic markers to our date today. The most common criteria used for diagnosing AE consists of major and minor criteria. Major are four main elements: (1) dermatitis in infant or adult distributions, (2) pruritus, (3) dermatitis that is chronic or relapsing, and (4) atopic history. The minor criteria are all twenty-three features, allocated into facial features, triggers, complications, and a few others (Fleming *et al*., 2020). *Facial features are* infraorbital folds (Dennie-Morgan lines), hypopigmented patches, facial pallor, infraorbital darkening, cheilitis, erythema, anterior neck folds, and recurrent conjunctivitis. *The triggers include* skin irritants, environmental factors, psychological factors, and food. *The complications involved are* immediate skin reactivity, anterior subcapsular cataracts, predisposition to keratoconus, impaired cell-mediated immunity, and susceptibility to skin infections. Finally, the *other factors are* ichthyosis Vulgaris, white dermatographism, nipple dermatitis, early age of onset, perifollicular accentuation, xerosis, keratosis pilaris, dermatitis, hyper linear palms, hand or foot. Three minor and three major criteria are needed for a definitive diagnosis of AE.

The severity of AE is commonly categorized into three levels. First is mild-to-moderate AE, which involves limited affected areas of the body; second is the milder intensity of pruritus and sleep loss; third acute AE includes widespread areas of dry skin

and high-frequency pruritus and impacts the quality of life significantly.

Treatment

The mainstay management of AE in primary care is patient education, a good skincare routine, Topical Corticosteroids (TCS), Topical Calcineurin Inhibitors (TCIs), and topical phosphodiesterase (PDE)-4 inhibitors (Katoh *et al*., 2020). Nevertheless, if it was severe enough, treatment might level up. First of all, referral to dermatology, phototherapy, systemic immunosuppressants, or the use of biologics (such as Dupilumab). Let us go through each step of the management for further enlightenment. In mild to moderate AE, the following should be taken into consideration. Patients or caregivers should be educated about how often he/they should moisturize. Educate patients about the proper application and use of topical therapies, the significance of treatment adherence, and the chronic nature of the illness. Patient compliance is key to the success of the treatment (Lowe *et al*., 2018). Based on the AD severity, it is recommended for skincare to bathe in warm water for 10–15 min once or twice daily. That is to assist hydrate and cleanse the skin, better the penetration of topical treatments, and help in the debridement of infected skin. Patients should often moisturize right away after baths (Lowe *et al*., 2018). The moisturizers used shall contain different extents of humectant, emollient, and adjunct ingredients, including salicylic acid, lactate, urea, and ceramides. These adjunct ingredients are physiologic lipids of the stratum corneum and repair and hydrate it, decreasing infection, xerosis, flares, and pruritus (Draelos, 2018). Topical Corticosteroids (TCS) are the first-line treatment if uncontrolled by moisturizers or irritant avoidance; for mild lesions, use moderate potency for two to four weeks (Hoare, 2000). For acute flares, use high potency for up to two weeks. Either case, apply once or twice daily.

Most importantly, high potency TCS is acceptable for high absorption areas for five to seven days before tapering. Some side effects can be atrophy due to purpura, telangiectasia, striae, focal hypertrichosis (abnormal hair growth), the impairment of wound healing, allergic contact dermatitis, and rosacea-like eruptions (Hoare, 2000). Topical Calcineurin Inhibitors (TCIs) are the second-line treatment of mild to moderate atopic eczema in patients aged two years and above. They should be not be used on suspected eczema herpeticum. The most common side effect can be temporary application-site burning. Topical PDE-4 inhibitors are the first-line treatment of mild to moderate atopic eczema in patients aged two years and above. Also, the side effect is transient application-site burning (Boothe *et al*., 2017). Other treatments shall be considered if the AE is severe and primary care fails. Phototherapy is indicated if topical treatments fail. Patients should do it two to five sessions weekly. Its side effects are erythema, sunburn, advanced skin aging, itching, and nausea. Phototherapy can be inconvenient for some and expensive (Sidbury *et al*., 2014). Recent studies suggest using biologics if topical treatments fail and previous treatments are not enough. Dupilumab is a human monoclonal antibody approved for the treatment of moderate-severe AE and given subcutaneously for the interleukin-4 receptor-a subunit. Its dosage is 600 mg subcutaneous loading, followed by 300 mg subcutaneous dose every two weeks (Deleuran *et al*., 2020). It has a high cost, and no baseline investigations or routine laboratory monitoring is required. Possible side effects can be anaphylaxis, hypersensitivity reactions, conjunctivitis, and injection site reactions (Deleuran *et al*., 2020).

Complications

Complications that come from AE are basically due to the disturbed dried-out skin barrier. The skin is the first line of defense. Apparently, when it is disturbed, potential infections will rise! So this disturbed barrier performance and diminished skin antimicrobial peptides make patients suffering from AE predisposed to potential bacterial and viral skin infections. Meanwhile managing complications of AE, the two most common secondary infections are *impetigo* and *eczema herpeticum* (Weidinger & Novak, 2016). Staphylococcus aureus or Streptococcus pyogenes are the most often associated with secondary infection of dermatitis. It may present with yellow crusting, pustules, and bulla; hence, impetigo (Weidinger & Novak, 2016). On the other hand, the latent herpes simplex virus can cause the serious infection of eczema herpeticum. Its presentation can show as punched-out erosions, vesicles, hemorrhagic crusts, lymphadenopathy, pain, and with or without fever (Sun & Ong, 2017). Generally for impetigo, the treatment involves topical antibiotics and emollients. If it was severe can switch to oral antibiotics. While for eczema herpeticum this can be lethal (Sun & Ong, 2017). Suspected patients should undergo bacterial and viral microbiologic studies and get treated empirically with systemic antiviral medications with an urgent referral move from primary care to a dermatology/emergency room care (Sun & Ong, 2017).

CONCLUSION

Atopic eczema is a common skin disease that can be acute or mainly chronic. It begins early in life and may unfavorably impact the life quality of patients and their caregivers. The good news is its management starts with topical corticosteroids and optimal skincare practices. These remain the cornerstone of treatment for the illness. TCIs have been indicated to give an influential, second-line alternative to topical corticosteroids in patients prone to frequent flare-ups. In acute cases that cannot be controlled with topical therapies and proper skincare, systemic immunosuppressive agents may come in handy. Also, in patients showing a weak reaction to proper pharmacological therapy and optimal skincare practices, allergy testing to aeroallergens and foods may be considered that can also be based on patient history with such factors. In the end, biologics can be an option to treat severe cases. They have been certified to be used in moderate-to-severe AE and seem to have favorable effect and side-effect profiles based on randomized tests.

Biologics, like dupilumab, are being used and further looked into regarding AE. Studies on biologics can indicate hopeful future options for better managing this enervative skin disease.

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