Review of improving strategies for dermatitis in primary care

Mona Saeed AlZahrani, Nehal Saad AlThagafi, Ohood Saeed AlGhamdi

Abstract:

The aim of this study was to assess the knowledge, attitudes, and practices (KAP) of primary care residents in the diagnosis of the infectious aspects of AD and management methods. Search was performed in PubMed, EMBASE, and the Cochrane Library, for studies investigating the improving strategies for dermatitis in primary care published in English language until December, 2017.Atopic dermatitis is often the first presentation of an individual destined to a life time of allergic reaction and asthma. Considering that the skin is an extremely sensitising organ that contributes considerably to the systemic allergic response, extremely efficient treatments need to be developed to reduce skin inflammation in this disease. Advancements are most likely to require better definitions for the numerous clinical phenotypes of atopic dermatitis, consisting of identification of the genes resulting in the disease, a better understanding of the immunoregulatory abnormalities underlying it, and new paradigms for avoiding relapses of this skin problem. Such advancements will probably be tied to advancement of pharmacogenetics and targeting of effective therapies to subsets of patients with atopic dermatitis. Primary physicians should ask history of patient ,if patient predisposition for having dermatitis and trying to prevent, in case already having problems of skin the aim of primary physician is to diagnose properly and distinguish disease depending on clinical features and manage properly, as well as to educate patient for skin caring.

Introduction:

Atopic dermatitis (AD) is a chronic inflammatory skin disease with an occurrence of 15% to 20%. The illness predominantly affects children with 50% of situations arising in the first year of life and most others emerging in the very first 5 years. Because of this, AD is one of the most typical skin disease run into in pediatric primary care. There is enhanced susceptibility to skin infection with Staphylococcus aureus in AD, and such infections are connected with clinical degeneration. Infections are related to high rates of colonization with S. aureus (approximately 80%, including 16% methicillin-resistant S. aureus [MRSA] [1], [2].There are several published standards on the management of AD in children, consisting of the management of the infectious difficulties [3].While researches have assessed the knowledge, attitudes, and practices (KAP) of dermatologists in their management of AD, [4] including a recent research study comparing practices of pediatricians, allergists, and dermatologists, [5] there are no data on exactly how AD is handled among primary care providers in training, who care for a substantial part of affected kids.

The aim of this study was to assess the knowledge, attitudes, and practices (KAP) of primary care residents in the diagnosis of the infectious aspects of AD and management methods.

Methodology:

Search was performed in PubMed, EMBASE, and the Cochrane Library, for studies investigating the improving strategies for dermatitis in primary care published in English language until December, 2017. Moreover, references list of studies included were scanned for more relevant articles that could support our review.

Discussion:

- **CAUSES AND RISK FACTORS**

The greatest risk factor is a favorable family history for atopic illness, specifically for atopic dermatitis. Twin research studies suggested a heritability of greater than 80%, [6] although this percent might be an overestimation, given that gene-gene communication effects were not considered. Until now, 32 susceptibility loci have been identified with gene-mapping researches, but they discuss less than 20% of the estimated heritability [7].The strongest recognized genetic danger factor is null mutations in filaggrin (FLG), which encodes an essential epidermal architectural protein [8].FLG mutations cause the semidominant skin-scaling disorder ichthyoids vulgaris, which is defined by irregular skin dryness and palmar hyperlinearity- attributes typically seen in patients with atopic dermatitis (table). About 10% of people of European ancestry carry a single null mutation in FLG and have light ichthyosis vulgaris, and their danger for atopic dermatitis is raised by three times [8].Of note, most patients with atopic dermatitis do not have any FLG mutation, and up to 60% of service providers will not establish atopic illness [8]-- ie, FLG anomalies are neither necessary nor sufficient to trigger atopic dermatitis. Most of the various other well-known risk genes add to immune systems, specifically to innate immune signalling, T-cell activation, and T-cell specification. Only few of these genes have been implicated in other atopic qualities, however many of them have been connected to other inflammatory illness, suggesting that non-atopy-related molecular processes are functional [7].The inherited vulnerability is activated right into disease symptom by environmental and way of life aspects. Well established environmental threat variables are a supposed western diet plan with high amounts of sugar and polyunsaturated fatty acids, small family size, high education degree in the household, and living in urban settings and regions with reduced direct exposure to

ultraviolet radiation and reduced humidity [9].An organized review of 113 population-based research studies [10] identified no clear proof for a duty of certain infections or vaccinations, however that atopic dermatitis has a substantial positive organization with exposure to broad-spectrum anti-biotics while pregnant and early stage, especially constant programs; some much less consistent proof was reported for safety effects of very early daycare, endotoxin direct exposure, intake of unpasteurised farm milk, and exposure to dogs- ie, increased exposures to non-pathogenic microorganisms.

*Foods:*Food allergens induce skin breakouts in almost 40% of children with modest to severe atopic dermatitis [11].Food allergic reactions in patients with atopic dermatitis might generate dermatitis and add to extent of skin disease in some patients, whereas in others urticarial reactions, or non-cutaneous symptoms are evoked. Infants and little ones with food allergic reactions normally have favorable immediate skin tests or serum IgE directed to different foods, especially egg, milk, wheat, soy, and peanut [12]. Significantly, T cells specifc to food allergens have been duplicated from the skin lesions of patients with atopic dermatitis, giving direct evidence that foods can add to skin inflammation [13].In mice with atopic dermatitis, oral sensitisation with foods causes eczematous skin sores on repeat oral food difficulties.

- **CLINICAL DIAGNOSIS**

Atopic dermatitis provides a broad clinical spectrum varying from small kinds such as pityriasis alba (completely dry depigmented patches) or hand eczema to significant kinds with erythrodermic breakout. The most usual types consist of the scientific functions listed in table 1 [14].Of the significant attributes, pruritus and chronic or relapsing eczematous lesions with normal shape and circulation are crucial for diagnosis. Although pruritus could occur throughout the day, it is usually worse in the early evening and evening. Pruritis causes scratching,

lichenification, and prurigo papules. Patients with atopic dermatitis have a reduced threshold for pruritus. Allergens, decreased humidity, excessive sweating, and low focus of toxic irritants could worsen pruritus and scratching.

Acute and subacute skin lesions are frequently seen in kids and are defined by intensely pruritic erythematous papules related to excoriation and serous exudate. Chronic atopic dermatitis is characterized by lichenification, papules, and excoriations. In any way phases of this condition, patients typically have completely dry lackluster skin. The distribution and skin response pattern varies according to the patient's age and condition activity. During infancy, atopic dermatitis is typically more acute and primarily influences the face, scalp, and extensor surface areas of the extremities. In older kids and in those who have longstanding skin disease, the patient creates lichenification and localization of the rash to the flexural folds of the extremities. Chronic hand eczema can be the primary indication of many grownups with atopic dermatitis.

Table 1: Clinical features of atopic dermatitis [14].

Essential features
Pruritus Facial and extensor eczema in infants and children
Flexural eczema in adults
Chronic or relapsing dermatitis
Frequently associated features
Personal or family history of atopic disease
Xerosis
Cutaneous infections
Non-specific dermatitis of the hands or feet
Raised serum IgE concentrations
Positive immediate-type allergy skin tests
Early age of onset
Other features
Ichthyosis, palmar hyperlinearity, keratosis pilaris
Pityriasis alba
Nipple eczema
White dermatographism and delayed blanch response
Anterior subcapsular cataracts, keratoconus
Dennie-Morgan infraorbital folds, orbital darkening
Facial erythema or pallor

- **MANAGEMENT**

Effective management of atopic dermatitis needs a multipronged method including skin care, recognition and elimination of flare elements, and anti-inflammatory therapy [15].Randomised regulated trials are specifically vital in examining the effects of treating atopic dermatitis due to the significant placebo impact in this condition.

Skin care

In atopic dermatitis, the disrupted function of the skin barrier is most likely the result of reduced ceramide concentrations and results in dry skin (xerosis) and enhanced transepidermal water loss [16].Irritants such as soaps or detergents, contact with chemicals, smoke, alcohol and astringents found in toiletries, and rough clothing could intensify the xerosis. Soaps with minimum defatting task and a neutral pH are liked. Xerosis contributes to development of epithelial microfissures and fractures, which allows entry of skin pathogens, irritants, and allergens. Wet dressings can be used on severely affected or chronic lesions refractory to skin care. Dressings can be a reliable barrier against persistent scraping, allowing a lot more quick healing of excoriated sores. Every attempt must be made to enable patients to be as usually energetic as feasible. Some sporting activities such as swimming might be much better endured compared to various other sports involving intense perspiration, and physical get in touch with, but chlorine ought to be rinsed off right away after swimming and the skin lubricated.

Identification and elimination of triggering factors

Potential allergens can be determined by taking a mindful history and doing careful allergic reaction tests. Negative skin prick tests or serum examinations for allergen-specific IgE have a

high predictive worth for eliminating suspected allergens. Positive skin or in-vitro allergy examinations, especially to foods, often do not associate with clinical symptoms and need to be verified with controlled food challenges, removal diets, or atopy patch tests. Avoidance of foods implicated in controlled challenges results in clinical renovation [17].Generally, substantial removal diet plans, which sometimes can be nutritionally deficient, are useless. A lot of kids who dislike food outgrow their food hypersensitivity in the first few years of life, making it much less relevant as trigger element when older. Extended avoidance of house dust mites in sensitised patients with atopic dermatitis leads to improvement of their skin disease. Avoidance measures include use house dirt mite-proof encasings on cushions, mattresses, and boxsprings; washing bedding in hot water weekly; removal of room carpets; and decreasing indoor humidity levels [18], [19].Unlike allergic rhinitis and external asthma, immunotherapy with aeroallergens has not been confirmed to be reliable in therapy of atopic dermatitis. Well controlled research studies are still should determine the duty of immunotherapy in this disease, and this technique must be scheduled for individuals who have a clear cut verifiable background of aeroallergen caused atopic dermatitis-eg, seasonal exacerbations to pollen.

Topical anti-inflammatory agents

Glucocorticoids-Topical glucocorticoids are frequently utilized to manage acute exacerbation of atopic dermatitis. However, once control of atopic dermatitis is achieved with an everyday program of topical glucocorticoids, long-lasting control can be kept with twice regular applications of topical fluticasone to areas that have recovered but are prone to establishing eczema [20].Side-effects from topical glucocorticoids are straight connected to the effectiveness ranking of the substance and the size of use, so the clinician needs to instruct the patients and to balance the requirement for an extra potent steroid with the capacity for side-effects. Therefore,

ultra-high-potency glucocorticoids should be utilized only for very brief amount of times and in regions that are lichenified however not on the face or intertriginous areas.

Tacrolimus-Topically applied FK-506, or tacrolimus, a calcineurin prevention that acts by binding with high affinity to the 12 kDa macrophilin, has been effectively used in treatment of atopic dermatitis. Tacrolimus prevents activation of numerous vital cells involved in atopic dermatitis, including T cells, dendritic cells, mast cells, and keratinocytes [21].Multicentre blinded vehicle-controlled phase-3 trials with 0 - 03% and 0 - 1% tacrolimus ointment, have shown tacrolimus to be both effective and safe in adults and children. Local burning feeling has been the only usual adverse event and most patients have greatly minimized pruritus within 3 days of beginning therapy. In grownups, but not kids, a dose-response result was seen between 0 - 03% and 0 - 1% tacrolimus, particularly for patients with severe skin disease.

Pimecrolimus- Ascomycin substances such as pimecrolimus, which has the very same system of activity as tacrolimus, have been developed in topical and oral types. Like tacrolimus, they inhibit manufacturing of Th1 and Th2 cytokines, and hinder mediator release from mast cells and basophils [22] 1% pimecrolimus works and secure in grownups and children with atopic dermatitis [23], [24].Pimecrolimus cream lotion 1% has been approved for short-term and intermittent long-lasting use in mildmoderate atopic dermatitis for patients 2 years and older. When utilized as upkeep treatment, topical pimecrolimus decreases the variety of flares triggered by atopic dermatitis and minimizes requirements for corticosteroid therapy.

The approval of topical calcineurin inhibitors for therapy of atopic dermatitis is a terrific advancement in our management choices for this illness. Nonetheless, we ought to still create guidelines for use of topical corticosteroids versus calcineurin preventions. Topical calcineurin preventions can be useful over topical corticosteroids in some situations, consisting of treatment

of patients who do not respond well to topical steroids, patients with steroid phobia, and therapy of face and neck dermatitis where inefficient, low-potency topical corticosteroids are typically utilized due to fears of steroid-induced skin atrophy. The possible use of topical calcineurin preventions as maintenance treatment is additionally interesting for prevention of atopic dermatitis flares [25].However, although systemic absorption of these substances are low, the drugs need to be carefully kept track of to rule out the possibility that skin cancers and increased viral skin infections will show up when such agents are used long-lasting.

Emotional stressors-Patients with atopic dermatitis often reply to frustration, shame, or various other stressful events with enhanced pruritus and scraping, and stress can cause immunological changes in such patients [26], [27].Psychological analysis or counselling must be taken into consideration in patients who have problem with emotional triggers or mental problems contributing to problem in handling their disease. Relaxation, behavioural adjustment, or biofeedback can be helpful in patients with habitual scratching.

- **PREVENTION**

No primary prevention strategy has been developed today. Most interventions examined so far concentrated on allergen avoidance or immunomodulation, [28] but an overview of systematic reviews [29] did not report clear proof for efficiency of measures such as maternal nutritional antigen avoidance while pregnant and breastfeeding, long-lasting breastfeeding, hydrolyzed protein formulas, soy formulas, omega-3 or omega-6 fatty acid supplements, and interventions with prebiotics or probiotics. Similarly, arises from a German birth cohort study [30] showed no proof in support of a postponed introduction of solid foods. Prevention approaches intending to enhance skin barrier function have been established. One pilot research [31] and 2 randomized

regulated tests [32], [33] reported that a daily full-body emollient therapy from birth decreased the advancing incidence of atopic dermatitis in high-risk infants by 30- 50%.

Conclusion:

Atopic dermatitis is often the first presentation of an individual destined to a life time of allergic reaction and asthma. Considering that the skin is an extremely sensitising organ that contributes considerably to the systemic allergic response, extremely efficient treatments need to be developed to reduce skin inflammation in this disease. Advancements are most likely to require better definitions for the numerous clinical phenotypes of atopic dermatitis, consisting of identification of the genes resulting in the disease, a better understanding of the immunoregulatory abnormalities underlying it, and new paradigms for avoiding relapses of this skin problem. Such advancements will probably be tied to advancement of pharmacogenetics and targeting of effective therapies to subsets of patients with atopic dermatitis. Primary physicians should ask history of patient ,if patient predisposition for having dermatitis and trying to prevent, in case already having problems of skin the aim of primary physician is to diagnose properly and distinguish disease depending on clinical features and manage properly, as well as to educate patient for skin caring.

Reference:

1. Suh L, Coffin S, Leckerman KH, Gelfand JM, Honig PJ, Yan AC. Methicillin-resistant Staphylococcus aureus colonization in children with atopic dermatitis. Pediatr Dermatol. 2008;25:528-534.
2. Balma-Mena A, Lara-Corrales I, Zeller J, et al. Colonization with community-acquired methicillinresistant Staphylococcus aureus in children with atopic dermatitis: a cross-sectional study. Int J Dermatol. 2011;50:682-688.
3. Lewis-Jones S, Mugglestone MA. Management of atopic eczema in children aged up to 12 years: summary of NICE guidance. BMJ. 2007;335:1263-1264
4. Chan YC, Tay YK, Sugito TL, et al. A study on the knowledge, attitudes and practices of Southeast Asian dermatologists in the management of atopic dermatitis. Ann Acad Med, Singapore. 2006;35:794-803.
5. Saavedra JM, Boguniewicz M, Chamlin S, et al. Patterns of clinical management of atopic dermatitis in infants and toddlers: a survey of three physician specialties in the United States. J Pediatr. 2013;163:1747-1753.
6. Thomsen SF, Ulrik CS, Kyvik KO, et al. Importance of genetic factors in the etiology of atopic dermatitis: a twin study. Allergy Asthma Proc 2007; 28: 535–39.
7. Ellinghaus D, Baurecht H, Esparza-Gordillo J, et al. High-density genotyping study identifi es four new susceptibility loci for atopic dermatitis. Nat Genet 2013; 45: 808–12.
8. Irvine AD, McLean WH, Leung DY. Filaggrin mutations associated with skin and allergic diseases. N Engl J Med 2011; 365: 1315–27.
9. Flohr C, Mann J. New insights into the epidemiology of childhood atopic dermatitis. Allergy 2014; 69: 3–16.
10. Flohr C, Yeo L. Atopic dermatitis and the hygiene hypothesis revisited. Curr Probl Dermatol 2011; 41: 1–34.
11. Sampson HA. Food allergy. Part 1: immunopathogenesis and clinical disorders. J Allergy Clin Immunol 1999; 103: 717–28.
12. Lever R, MacDonald C, Waugh P, Aitchison T. Randomised controlled trial of advice on an egg exclusion diet in young children with atopic eczema and sensitivity to eggs. Pediatr Allergy Immunol 1998; 9: 13–19.

13. van Reijsen FC, Felius A, Wauters EA, Bruijnzeel-Koomen CA, Koppelman SJ. T-cell reactivity for a peanut-derived epitope in the skin of a young infant with atopic dermatitis. J Allergy Clin Immunol 1998; 101: 207–09.
14. Williams HC. Diagnostic criteria for atopic dermatitis: where do we go from here? Arch Dermatol 1999; 135: 583–86.
15. Hoare C, Li Wan Po A, Williams H. Systematic review of treatments for atopic eczema. Health Technol Assess 2000; 4: 1–191.
16. Imokawa G. Lipid abnormalities in atopic dermatitis. J Am Acad Dermatol 2001; 45 (suppl): S29–32.
17. Woodmansee DP, Christiansen SC. Improvement in atopic dermatitis in infants with the introduction of an elemental formula. J Allergy Clin Immunol 2001; 108: 309.
18. Tan BB, Weald D, Strickland I, Friedmann PS. Double-blind controlled trial of effect of housedust-mite allergen avoidance on atopic dermatitis. Lancet 1996; 347: 15–18.
19. Holm L, Bengtsson A, van Hage-Hamsten M, Ohman S, Scheynius A. Effectiveness of occlusive bedding in the treatment of atopic dermatitis—a placebo-controlled trial of 12 months' duration. Allergy 2001; 56: 152–58.
20. Van Der Meer JB, Glazenburg EJ, Mulder PG, Eggink HF, Coenraads PJ, for the Netherlands Adult Atopic DermatitisStudy Group. The management of moderate to severe atopic dermatitis in adults with topical fluticasone propionate. Br J Dermatol 1999; 140: 1114–21.
21. Wollenberg A, Sharma S, von Bubnoff D, Geiger E, Haberstok J, Bieber T. Topical tacrolimus (FK506) leads to profound phenotypic and functional alterations of epidermal antigen-presenting dendritic cells in atopic dermatitis. J Allergy Clin Immunol 2001; 107: 519–25.
22. Zuberbier T, Chong SU, Grunow K, et al. The ascomycin macrolactam pimecrolimus (Elidel, SDZ ASM 981) is a potent inhibitor of mediator release from human dermal mast cells and peripheral blood basophils. J Allergy Clin Immunol 2001; 108: 275–80.
23. Luger T, Van Leent EJ, Graeber M, et al. SDZ ASM 981: an emerging safe and effective treatment for atopic dermatitis. Br J Dermatol 2001; 144: 788–94.
24. Eichenfield LF, Lucky AW, Boguniewicz M, et al. Safety and efficacy of pimecrolimus (ASM 981) cream 1% in the treatment of mild and moderate atopic dermatitis in children and adolescents. J Am Acad Dermatol 2002; 46: 495–504.
25. Kapp, A, Papp K, Bingham A, et al. Long-term management of atopic dermatitis in infants with topical pimecrolimus, a non-steroid anti-inflammatory drug. J Allergy Clin Immunol 2002; 110: 277–84.
26. Schmid-Ott G, Jaeger B, Meyer S, Stephan E, Kapp A, Werfel T. Different expression of cytokine and membrane molecules by circulating lymphocytes on acute mental stress in patients with atopic dermatitis in comparison with healthy controls. J Allergy Clin Immunol 2001; 108: 455–62.

27. Schmid-Ott G, Jaeger B, Adamek C, et al. Levels of circulating CD8(+) T lymphocytes, natural killer cells, and eosinophils increase upon acute psychosocial stress in patients with atopic dermatitis. J Allergy Clin Immunol 2001; 107: 171–77.
28. Simpson EL, Keck LE, Chalmers JR, Williams HC. How should an incident case of atopic dermatitis be defi ned? A systematic review of primary prevention studies. J Allergy Clin Immunol 2012; 130: 137–44.
29. Foisy M, Boyle RJ, Chalmers JR, Simpson EL, Williams HC. The prevention of eczema in infants and children: an overview of Cochrane and non-Cochrane reviews. Evid Based Child Health 2011; 6: 1322–39.
30. Zutavern A, Brockow I, Schaaf B, et al, and the LISA Study Group. Timing of solid food introduction in relation to eczema, asthma, allergic rhinitis, and food and inhalant sensitization at the age of 6 years: results from the prospective birth cohort study LISA. Pediatrics 2008; 121: e44–52.
31. Simpson EL, Berry TM, Brown PA, Hanifi n JM. A pilot study of emollient therapy for the primary prevention of atopic dermatitis. J Am Acad Dermatol 2010; 63: 587–93.
32. Horimukai K, Morita K, Narita M, et al. Application of moisturizer to neonates prevents development of atopic dermatitis. J Allergy Clin Immunol 2014; 134: 824–30, e6.
33. Simpson EL, Chalmers JR, Hanifi n JM, et al. Emollient enhancement of the skin barrier from birth off ers eff ective atopic dermatitis prevention. J Allergy Clin Immunol 2014; 134: 818–23.