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A review study on the efficacy of homoeopathic medicines in the management of atopic dermatitis

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Abstract

Atopic Dermatitis is a spectrum of chronic, pruritic, inflammatory skin conditions that emerge as eczematous eruptions or lesions, primarily affecting the face, and upper and lower extremities but sparing the groin and axillary regions. Atopic Dermatitis possesses secondary health risks, including secondary bacterial or viral infections and potentially severe food allergies, and it is associated with elevated levels of immunoglobulin and is often related to the conditions of non-cutaneous organs such as Allergic rhinitis, Inflammatory Bowel Disease and Bronchial asthma. The disease affects individuals of different ages and ethnicities and may be more prominent in Asians and Black, more than Caucasians. This article systematically overviews the patho-physiology, risk factors and causes, classification of Atopic Dermatitis, difference between pediatric and adult Atopic Dermatitis, clinical features and diagnostic criteria, and a strong emphasis on the Individualized homoeopathic medicines and its Repertorial approach.

Keywords: Atopic dermatitis, allergic rhinitis, inflammatory bowel disease and bronchial asthma, individualized homoeopathic medicines, repertorial approach

Introduction

Atopic Dermatitis (AD), frequently referred to as eczema, is one of the most prevalent chronic skin diseases, which is typically presented clinically as pruritis, erythematous plaques, scaling, crusting, fissuring, thickening, and lichenification, with lesion distribution varying in children and adults afflicted with AD. It can also be seen in close association with other co-morbidities like Inflammatory Bowel Disease (IBD), asthma, kidney diseases, etc. It may even have a negative impact upon the quality of lives of individuals with moderate to severe diseases through decreased sleep, productivity, behavioral issues, low self-esteem, anxiety, depression, and stress on caregivers ^[1].

The incidence of Atopic dermatitis has increased to about two to three folds globally, out of which 15 to 20 percent are children and 10 percent are adults ^[2]. The studies over the years show that the disease can be categorized into subtypes depending on various parameters such as age, skin phenotypes, skin barrier status, genetic alterations, and levels of IgE. AD is a spectrum of morbidities, but not a uniform entity, as a high degree of symptomatic heterogeneity is reflected in wide varieties of skin types, and therefore the histopathology varies in each case.



Fig 1: Atopic dermatitis on hands



Fig 2: Pediatric dermatitis

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Pathophysiology

Although the patho-physiology of AD is not entirely understood, it is considered to be caused by skin barrier malfunction and dysregulation of the immune system. The recent studies show that the abnormalities in the epidermal lipid layer and changes in the microbial distribution also play a key role in the occurrence of AD.

1. Skin-barrier malfunction

The Filaggrin (FLG) gene located on chromosome 1q2 encodes the protein FLG, a major component of stratum corneum, a layer of skin. Any mutations to this gene, especially homogenous mutations, have a major impact on the onset of AD with increased risk [3].

2. Dys-regulation of immune system

Some of the triggering parameters, like exposure to environmental factors such as allergens, microorganisms, smoke, chemical irritants, gases, etc., may induce the production of Type 2 cytokines, e.g., IL-4 and IL-3, which may thereby increase the production of chemokines and lower the antimicrobial proteins in the skin (AMP), causing the disruption in the function of the skin barrier, and therefore, colonization of bacteria occurs, such as *Staphylococcus aureus*, *Propionibacteriae*, and *E. coli*, eventually leading to itching of the skin in AD [4].

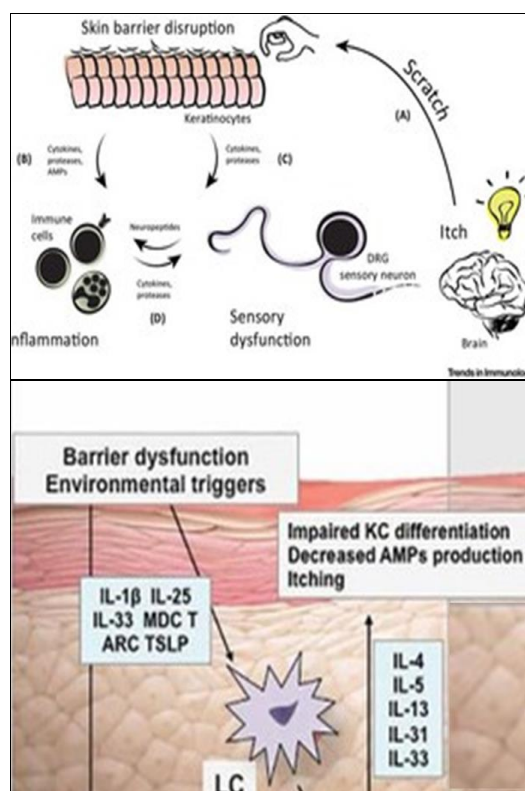


Fig 3: Shows the pathophysiology of atopic dermatitis (Skin barrier malfunction)

3. Abnormalities in the epidermal lipid layer

Lipids such as ceramides, long-chain FFAs, and cholesterol constitute the integrity of the epidermal barrier. When the levels of cytokines elevate, this lipid composition is also altered. It may reduce ceramides and long-chain FFAs, which may in turn destroy the epidermal barrier and may lead to the colonization of microbes and eventually cause lesional or non-lesional AD.

4. Changes in microbial distribution

Recent studies and experiments have shown a predominance of *Staphylococcus aureus* in severe cases of AD and *Staphylococcus epidermidis* in less severe cases. When the balance between the *Staphylococcus aureus* and commensal bacteria is disrupted, it leads to exacerbations of AD in the form of skin inflammations and allergic reactions [5].

Risk factors

The environmental risk elements implicated in AD include the following

In utero

Maternal exposures during pregnancy predisposing towards the increased risk of AD in childhood are the following:

- Maternal stress
- Cigarette Smoking & Alcohol Consumption
- Antibiotic exposure
- Omega-3 long-chain polyunsaturated fatty acids & diet [6].

Early life exposure to dirt and pathogens

The 'hygienic hypothesis' emphasizes the statement that the exposure to different kinds of pathogens, dirt, and unhygienic conditions has a way to steer away from developing AD in the early stage. The children living in developing nations and in rural areas are less likely to contract the disease [7].

Climate

The geographic variables such as changes in temperature, pressure, moisture, rainfall, UV radiation, etc. are also playing an important role in AD. The increase in temperature and moisture may provoke profuse perspiration, which may aggravate AD in children. The Ultraviolet radiations UVB and UVA are meant to be used in phototherapy treatment for AD. Therefore, it is logical that exposure to UV radiation can protect against AD.

Air pollutants and food allergens

The indoor and outdoor pollutants such as NO₂, SO₂, SO₃, CO, etc., are likely to cause adverse effects on skin, thereby causing genetic alterations, which may eventually lead to an increased risk of acquiring AD. They produce oxidative stress in the skin, which may lead to skin barrier malfunction or immune dys-regulation, thereby causing AD. Certain food items like eggs, nuts, shellfish, meat, and certain fruits can also aggravate AD and produce adverse effects [8].

Clinical manifestations

It presents clinically in three phases

- 1) Acute AD pruritic, vesicular eruptions with oozing and later crusting.
- 2) Subacute AD dry, scaly, erythematous papules and plaques.
- 3) Chronic AD repeated itching and scratching cause lichenification and thickening of skin [9].

It is commonly localized to the flexor aspects of the body, wrist, hands, neck, face, forehead, back, etc. As there are a broad variety of skin conditions, AD has to be differentiated from other diseases. Seborrheic dermatitis is a common skin condition difficult to distinguish from AD. The SD lesions are basically thick and greasy, white or yellow, and resolve in 2 years. Psoriasis is another disease with a distribution of

lesions, including the face, groin, and nails, which helps to distinguish it from AD. Scabies is an allergic reaction caused by *Sarcoptes scabiei*, manifested as small, red, papulo-vesicular lesions. It may spread among the family group during the same time. Contact dermatitis is the most common dermatitis, characterized by erythema and edema. Inflammations due to scabies infestation should also be distinguished from AD.

AD associated conditions and complications

There are a few skin conditions associated with AD. ^[10]

- 1) Pityriasis alba is a skin condition presented with red, scaly patches that leaves hypo-pigmentation when resolved and commonly occurs on the face, upper extremities, and trunk.
- 2) Keratosis pilaris occurs as small eruptions on the skin, which are harmless and caused by the accumulation of dead cells.
- 3) Ichthyosis vulgaris results in a fish-scale appearance most often in legs.
- 4) Dermographism, also called skin writing, occurs as a result of scratching the skin, resulting in a raised wheal within 5 minutes. The complications include bacterial, viral, and fungal infections and show atopic march in the form of an atopic triad, causing allergic rhinitis, asthma, and food allergies.



Fig 4: Paediatric dermatitis behind knee

Difference between pediatric AD & adult AD

Pediatric AD

In infants, AD is presented as xerosis, which can involve the whole body except the groin area. It may be presented as erythematous papules in flexural areas. It can gradually progress to inflammation, with oozing, scaling, and thickening of the site. It shows a centrifugal pattern, especially on the cheeks, neck, scalp, etc., and eventually spreads to lower areas as well. Itching is a characteristic feature, where the infant scratches the area, and it may result in excoriation and secondary infections. Lichenification may be seen in adolescents ^[11].

Adult AD

The clinical manifestations of AD vary with age. The adult AD manifested as marked xerosis and lichenification of skin. The lesions may be more diffuse along with more prominent underlying erythema.

The most affected parts in adults include the face being the common area. It is presented as dry and scaly lesions. Itching is uncontrollable and may produce oozing of discharges and eventually cause scaling and thickening of skin in the form of lichenification. Along with the above, a characteristic feature is also noted in adults, which is a brown macular ring made of amyloid deposited around the neck.



Fig 5: Adult dermatitis behind neck

Diagnostic criteria

The criteria which helps in the diagnosis can be categorized into the following

- 1) Characteristic features like pruritus, erythema, acute or chronic presentation, age-specific variations, relapsing episodes.
- 2) Personal and family history.
- 3) Complications and other conditions associated with AD like keratosis pilaris, pityriasis alba, ichthyosis, dermatographism, etc. However, the above categories of AD depiction are not always reliable, as the disease often shows variations in clinical presentation. IgE levels, prick tests, and RAST tests along with ELISA help in further investigation to find the allergen ^[12].

Lifestyle management ^[13]

- 1) Moisture of the skin has to be protected. Bathe using warm water and a gentle cleansing bar, and avoid scrubbing. Apply moisturizer.

- 2) Avoid allergens, exposure to causative factors, including changes in diet.
- 3) Keep away major life stressors.
- 4) Wear loose fitting clothes made of cotton and linen. Avoid wool as it might irritate skin Wash your clothes.

Repertorial study

Rubrics taken into account are the following.

- Mind, Anxiety
- Skin, Inflammation
- Face, Itching
- Face, Itching, eruptions.
- Skin, eruptions, eczema
- Skin, discoloration, red, scratching
- Skin, eruptions, crusty
- Skin, Itching, violent
- Skin, Itching, Scratching, agg.
- Skin, eruptions, bend of joints.

- Extremities, Itching, joints.
- Generals, Allergic Constitution.
- Skin, eruptions, erythema.
- Skin, eruptions, scaly
- Skin, dry
- Head, Itching of scalp.

Repertorial analysis (Using synthesis repertory) ^[14]

| | | | | | |
|---------|----|----|----------|------|------|
| mez. | 12 | 25 | Remedies | ΣSym | ΣDeg |
| graph. | 12 | 24 | merc. | 14 | 24 |
| sep. | 12 | 22 | psor. | 14 | 22 |
| rhus-t. | 11 | 29 | sulph. | 13 | 29 |
| calc. | 11 | 24 | ars. | 12 | 26 |
| phos. | 11 | 22 | | | |
| nat-m. | 11 | 21 | | | |

Fig 6: Repertorization sheet from synthesis app

| | | | | | |
|---------|---|----|---------|----|----|
| bov. | 9 | 15 | lyc. | 11 | 20 |
| ant-c. | 9 | 14 | apis | 11 | 18 |
| kali-c. | 9 | 14 | puls. | 10 | 19 |
| staph. | 9 | 14 | petr. | 10 | 17 |
| agar. | 9 | 13 | dulc. | 10 | 16 |
| carb-v. | 9 | 13 | kreos. | 10 | 13 |
| am-c. | 9 | 11 | nux-v. | 10 | 11 |
| lach. | 9 | 11 | sil. | 9 | 19 |
| ph-ac. | 9 | 11 | caust. | 9 | 18 |
| zinc. | 9 | 10 | hep. | 9 | 17 |
| calc-s. | 8 | 18 | anac. | 9 | 16 |
| olnd. | 8 | 16 | nit-ac. | 9 | 16 |
| kali-s. | 8 | 15 | bell. | 9 | 15 |

Fig 7: Repertorization sheet from synthesis app

Homoeopathic remedies

Merc sol: Eczema of chest, arms, and legs. Profound anemia in individuals with weak memory and slowness in answering. Weary of life. Yellow crusts with red areola; pustules with bloody, purulent discharge; foul body smell; dry, scaly skin; intolerable itching; aggravated at night, perspiring, wet damp weather, better by warmth, morning. Eruption is copper colored and may ulcerate ^[15].

Psorinum: Eczema, dry, scaly, moist or suppurating; in low-spirited, despondent, patients with no hope; profuse perspiration, sensitive to cold; wants to keep the head warm;

lack of reaction, dread cold, dry, dirty looking skin; eczema of scalp, flexures, behind the ears; in pale, peevish, delicate children; fetid, thin, excoriating discharges; itching worse in the evening and the open air; better by warmth and rest ^[16].

Sulphur: Psoric patients with dry skin and acne, never perspire; dirty filthy people with aversion to bathing, prone to skin affections; Even rags seems beautiful; irritable, thin, weak and aversion to business; they have scanty bloody discharges, burning, and intense itching that is worse at night and in the warmth of bed, and scratching relieves burning and starting; they have voluptuous itch, aggravated by bathing, suppressed eruptions, and an empty stomach between 10 and 11am.

Ars alb: Debility, exhaustion, restlessness, with nightly aggravation. Unquenchable thirst. Fear fright and worry. Chronic cases; skin dry, rough like parchment; skin white and thin; vesicles with watery exudation that excoriates the parts passed over; eruptions with intense itching and burning, better by warmth or heat in general.

Mezerium: Eczema in latent syphilitic patients, thick crusts oozing a bloody, purulent secretion; chilliness with pruritis, worse in bed. Ulcers itch and burn, surrounded by vesicles and shining, fiery red areola; itching worse at night and by warmth, cold skin covered with white scabs, and bleeding easily when touched; worse touch, warm food, motion; better, open air.

Graphites: Eczema accompanied by erysipelas; patients never perspire; skin is dry, dirty, thickened, rough, chapped, horny, and fissured, and it bleeds, and a watery or serous, sticky discharge oozes out. Children impudent, teasing, laughing at reprimands. Tendency to obesity. Want of disposition to work. Aggravation in damp or cold climate; mostly occur on the hands, face, behind ears, joints, and flexures ^[17].

Apis: Erythematous eruptions with swelling, stinging pain, and marked edema; extreme sensitiveness to touch and general soreness is marked; Apathy, indifference and unconsciousness, fidgety, jealous, awkward, drops things readily; never given after Rhus-tox; more on face, lips, nose, hands, and feet; circumscribed spots with itching and burning; skin looks pale, waxy, or dirty; red or purple rash or papules, worse in warm room and open air.

Causticum: Yellowish skin; eczema about the alae of the nose. Pustular eczema with burning and biting, preventing sleep. Restlessness at night; children are slow to talk; child does not want to go to bed. Ailments from long-lasting grief, sudden emotions. Thinking of complaints aggravates. Soreness in folds of skin, back of ears, between thighs Skin prone to intertrigo during dentition.

Sepia: Dark complexion, dark-haired; indifference to dear ones, averse to occupation, to family. Irritable; easily offended. Dreads to be alone. eczema accompanied by pelvic or uterine complaints or alternates with them. Lesions with brownish discoloration; eruptions dry, scaly, and in circular or ring forms. More in flexures of joints.

Rhus-tox: Dark, erythematous eruptions with intense burning and itching; vesicular with thin, watery or yellow straw-colored, offensive exudates; tendency to scale formation; listless, sad, thoughts of suicide, extreme restlessness, with continued change of position; dark, hard, and thick crusts; more on scalp, face, and upper and lower extremities; worse by warmth and damp climate and better by motion.

Calc carb: Scrophulous, blue-eyed, fair, pale, weak women

or fat, flabby children with distended abdomens; apprehensive, worse towards evening; aversion to work or exertion, increased perspiration, cold, clammy hands and feet; dry or moist eruptions; thick yellow crusts on scalp, face, behind ears, forearms; syphilitic diathesis; and yellow, putrid, pus-like oozing from eruptions.

Dulcamara: Scrophulous, phlegmatic constitutions. Mental confusion. Patients living or working in damp, cold basements. Hot days and cold nights towards the close of summer are favorable for the action of Dulcamara. Vesicular eruptions on an erythematous base; watery discharge; itching worsened by warmth, washing, winter, and cold and wet weather. Adenitis, pruritis worse in winter red spots, urticaria worse from cold exposure or sour stomach.

Natrum mur: Despondent, low-spirited, tubercular people, craving salt and sour things; consolation aggravates; emaciation, noted more on neck; greasy, oily, especially on hairy parts, fine vesicular eruptions all over the body, and on bends of limbs, margins of scalp, behind ear; intense itching; vesicles dry up, become a thin crust; worse by cold air; worse in edges of hair, flexures, genitals.

Lycopodium: Melancholy; afraid to be alone. Extremely sensitive. Loss of self-confidence. Apprehensive, weak memory, weeps easily even when thanked. Skin becomes thick and indurated. Eruptions vesicular, then dry, humid, suppurating, and full of deep rhagades, with a tendency to ulcerate. Eczema of the face, genitals, or any part of the body, with thick moist crusts, bleeding easily, and itching worse at night while in bed.

Petroleum: Strumous diathesis, especially the dark type, who suffer from catarrhal conditions. Very marked skin symptoms, acting on sweat and oily glands. Marked aggravation from mental emotions. Eruption dry, scaly, leathery or moist, disappearing in the summer and reappearing in the winter or during cold weather. Eczema fissure on hands, face, or behind ears; a moist, purulent, offensive discharge; itching, aggravated in the morning and open air; itching combined with chilliness. Intertrigo; psoriasis of hands; thick, greenish crusts burning and itching.

Bovista: Adapted to stammering children, old maids with palpitations, tottery patients. Awkward, sensitive, everything falls from hands; marked languor and lassitude. Moist, vesicular eruptions dry or moist, forming thick crusts; itching not relieved by scratching; rough, dark red; back of hands on the bends of knees; appear during full moon; more frequent in women with dysmenorrhea. Urticaria on excitement, on walking in the morning with rheumatic lameness. Itching on getting warm.

Silicea: Scrophulous, rachitic children, with large head, open fontanelles and sutures, distended abdomen, slow in walking, with defective assimilation; marked suppuration & bad effects of vaccination; yielding, faint hearted, anxious. Sensitive to all impressions, brain-fag, fixed ideas. Patient is chilly and the symptoms are worse in winter and cold climates; Rose-colored blotches on skin, offensive pus, Icy offensive cold and sweaty feet; Chilliness and very sensitive to cold delicate, pale, waxy, dry, cracks in fingers; rose colored blotches; eruptions itch in daytime and evening; coppery spots ^[18].

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