



The Faces of
Atopic Eczema
among Malaysian Children



Author Leong Kin Fon

Acknowledgement

- I would like to express particular thanks to all the patients & mentors who inspired me in making a simple and practical book on Atopic Eczema.

Dr Leong Kin Fōn

FROM CONFUSION TO CLARITY, ONE CONSULT AWAY

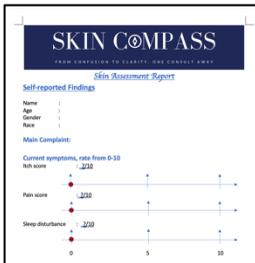
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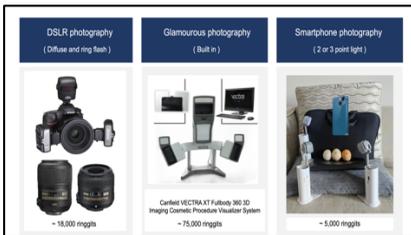
Are you lost and confused by several different clinical diagnoses?"

“The bottleneck in treating many skin problems is often misdiagnosis and misleading myths.

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FORE WORD

The Faces of Atopic Eczema among Malaysian Children

Atopic dermatitis or atopic eczema is a very common skin condition affecting one in five school children¹. It is a disabling disorder with pruritus and sleep disturbance being significant challenges, affecting the quality of life.

Dr. Leong Kin Fon, a senior experienced paediatric dermatologist shares his experience in the management of eczema in this book, which is divided into three sections. The first section details the terminology used in describing a skin rash. The second section answers 10 common questions, from the definition of eczema, the role of food allergy, the selection of moisturizers to useful management tips. The third section describes 15 clinical case scenarios commonly encountered from infected eczema to contact dermatitis, and the role of education and empowerment. Each case begins with a history, physical signs, diagnosis/differential diagnoses and management, similar to what happens in the clinics.

I congratulate Dr. Leong Kin Fon for putting together this practical and concise handbook which will benefit general practitioners, paediatricians, dermatologists and all doctors who care for patients with eczema.

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23 April 2016

Reference:

Tay YK, Kong KH, Khoo L, (et al), The prevalence and descriptive epidemiology of atopic dermatitis in Singapore school children. Br J Dermatol. 2002 Jan;146(1):101-6

PREFACE

Patients are our teachers. This is certainly the case in the stimulus for this simple atlas that illustrates the myriad faces of Atopic Dermatitis among children in Malaysia. Over the years, many patients with mismanaged Atopic Eczema have inspired me to share the challenges that I encounter in my daily practice.

This atlas is not a textbook of paediatric dermatology with detailed information and references on Atopic Eczema, but designed to provide practical information and sharing common pitfalls for all health care practitioners.

The format includes

- 1. Clinical history – brief but relevant information**
- 2. Clinical course (acute / subacute / chronic)**
- 3. Skin findings
(morphology & its details, distribution, pattern, local arrangement)**
- 4. Clinical diagnosis & differential diagnosis**
- 5. Management & clinical pearls**

Each case is presented in a similar format to emphasize the importance of a “step by step” approach in dermatology and not making a spot diagnosis on the first sight.
(Love at first sight is risky too!)

On the other hand, visual memory is an important tool in learning dermatology and good photographic documentation of skin lesions at different stages of evolution is vital in diagnostic dermatology.

Over the last 2 years, I have devoted a great deal of effort and time compiling the clinical photographs and sharing my own experiences in the session of “clinical pearls” to materialize this project. No labour of this type would be possible without the support of my parents.

Editor & Author

Dr. Leong Kin Fon

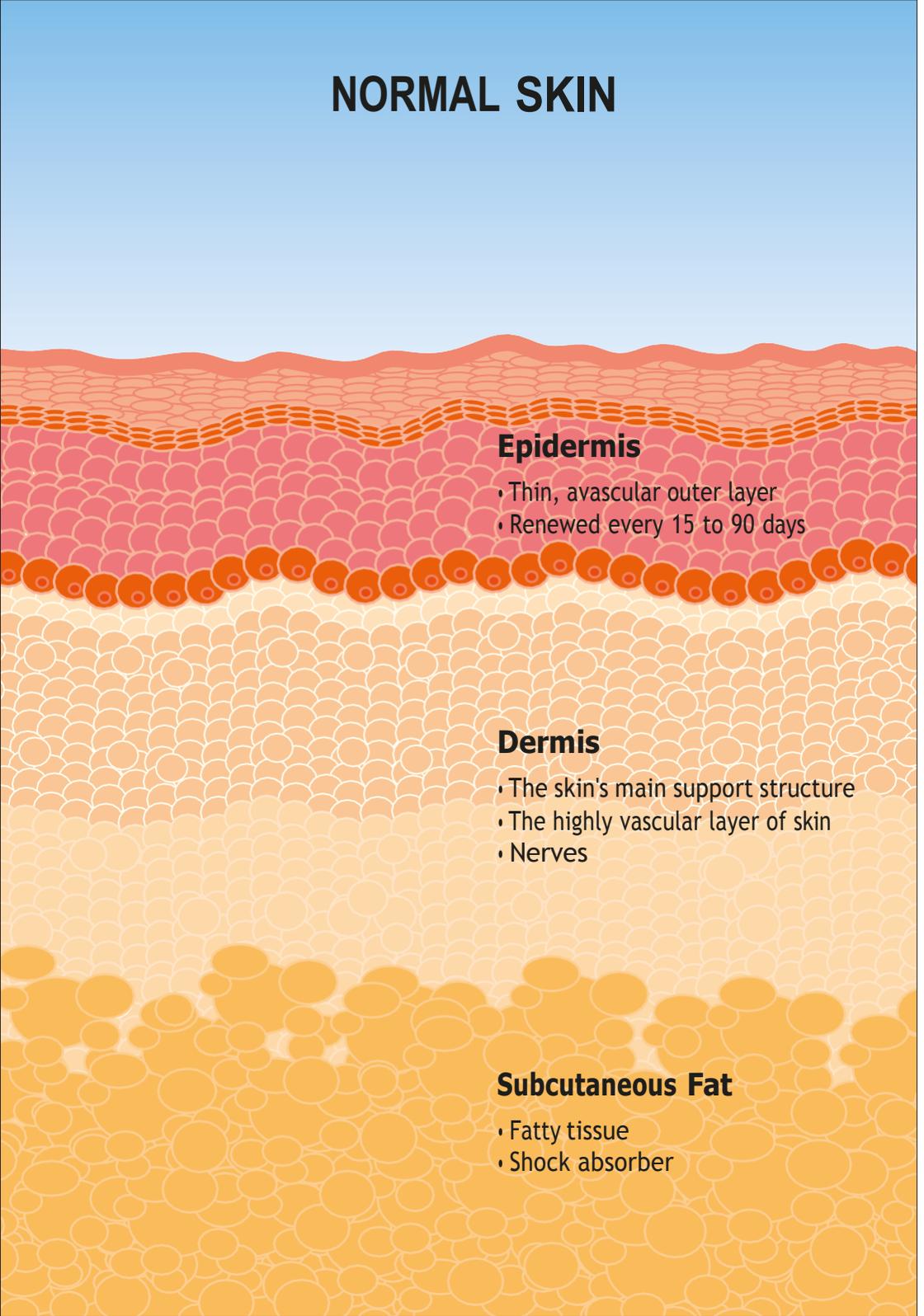
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(Asia Paediatric Dermatology Interest Group)

NORMAL SKIN



Epidermis

- Thin, avascular outer layer
- Renewed every 15 to 90 days

Dermis

- The skin's main support structure
- The highly vascular layer of skin
- Nerves

Subcutaneous Fat

- Fatty tissue
- Shock absorber

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CHAPTER 1
Introduction



INTRODUCTION

In dermatology, many physicians often prefer to look at skin lesions and making a spot diagnosis before obtaining history about the progression of the skin lesions and systemic review.

Although spot diagnosis based on classical images is very helpful in approaching skin problems, diagnosis of a dermatological problem is most reliably achieved by a “step by step” approach to patient evaluation that begins with an examination of

- 1. Morphology and its details** (refer to Figure 7 - 21)
- 2. Local arrangement** (refer to Figure 22 - 23)
- 3. Distribution & Pattern** (refer to Figure 24 a - h)
- 4. Clinical course**
- 5. Correlation to extracutaneous findings**

Progression of the morphology is very important in the diagnostic process of skin problems as every disease has different faces according to the stages of the disease evolution. For example in *Figure 1 - 6*, all are eczematous eruptions of different chronicity.



Figure 1-6 : Eczematous eruptions of different chronicity

MORPHOLOGY (I)

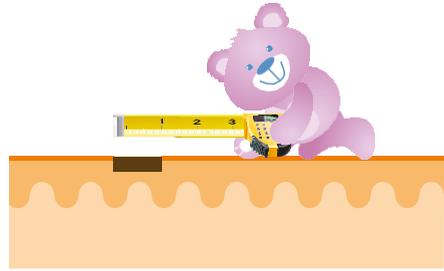


Figure 7
Macule is a non palpable skin lesion that is <1cm in diameter.

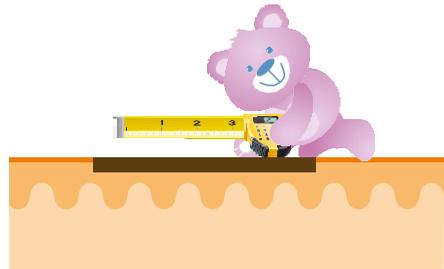


Figure 8
Patch is a non palpable skin lesion that is >1cm in diameter.



Figure 9
Papule are small palpable lesion that is <0.5 cm diameter

MORPHOLOGY (II)



Figure 10
Nodule is a palpable lesion that is
>0.5 - 2cm diameter

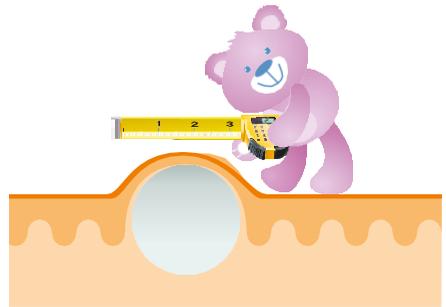


Figure 11
Tumour is a palpable lesion that is
>2cm diameter



Figure 12
Plaque is a palpable flat lesion greater than
0.5 cm diameter. It may be scaly or non scaly.

MORPHOLOGY (III)

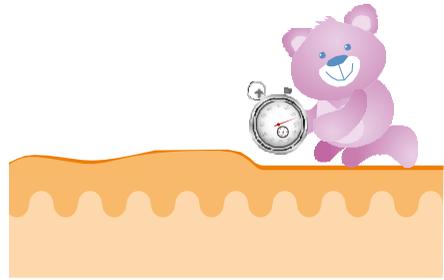


Figure 13
Wheal is an edematous papule or plaque caused by swelling in the dermis.



Figure 14
Vesicle is a small fluid-filled blisters less than 0.5cm in diameter.

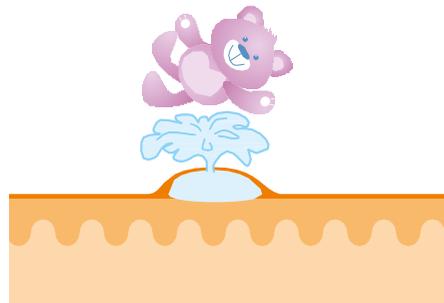


Figure 15
Bullae is a bigger fluid-filled blisters more than 0.5cm in diameter.

MORPHOLOGY (IV)



Figure 16
Pustule is a pus filled vesicle that can be follicular or non follicular.



Figure 17
Erosion is a partial loss of epidermis which heals without skin atrophy.



Figure 18
Ulcer is full thickness loss of epidermis & part of dermis / subcutaneous fat. It may heal with skin atrophy.

MORPHOLOGY (V)



Figure 19
Crust occurs when plasma exudes through an eroded epidermis. It is yellow or brown in colour.



Figure 20
Scaling is an increase in the dead cells on the surface of the skin.

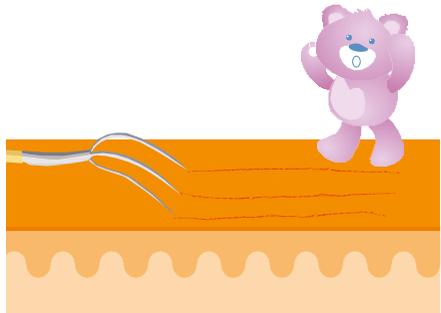


Figure 21
Excoriation is a linear or a picked scratch mark.

LOCAL ARRANGEMENT

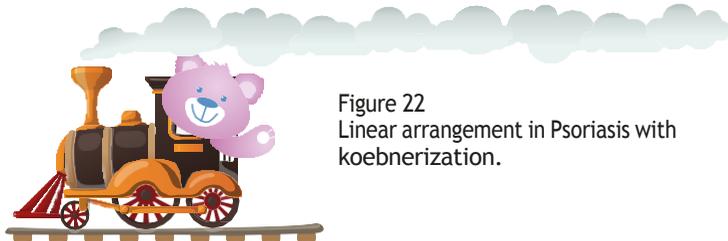


Figure 22
Linear arrangement in Psoriasis with
koebnerization.

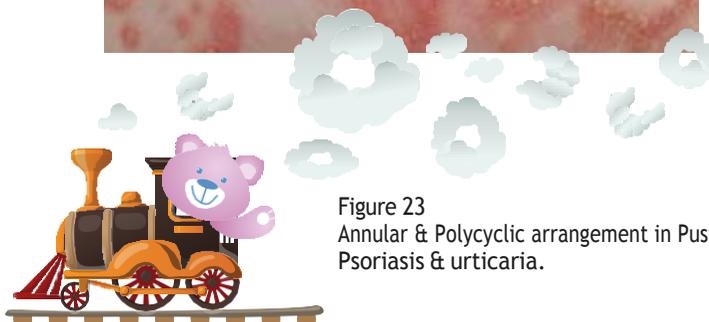
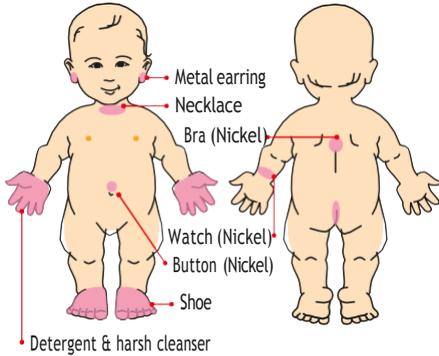
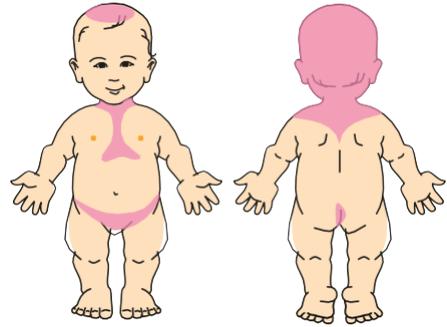


Figure 23
Annular & Polycyclic arrangement in Pustular
Psoriasis & urticaria.

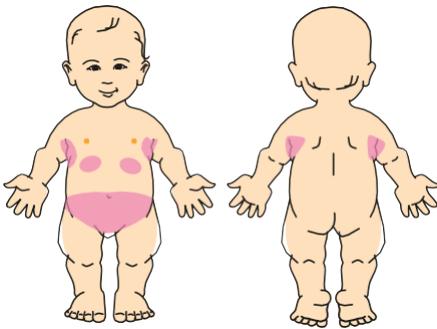
DISTRIBUTION & PATTERN (I)



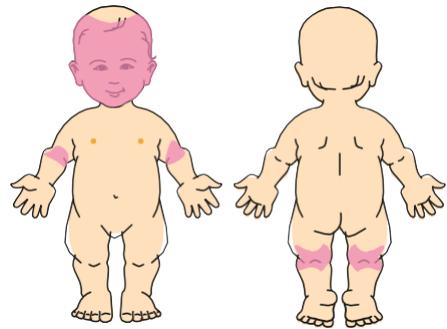
Contact Eczema Dermatitis
Cosmetic, jewellery and cleanser



Seborrhoeic Eczema



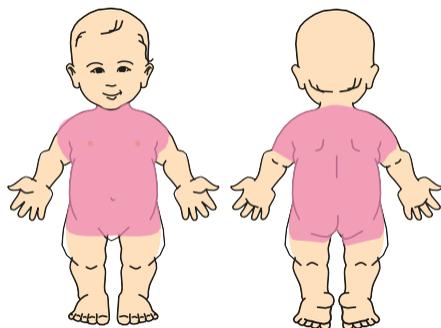
Scabies



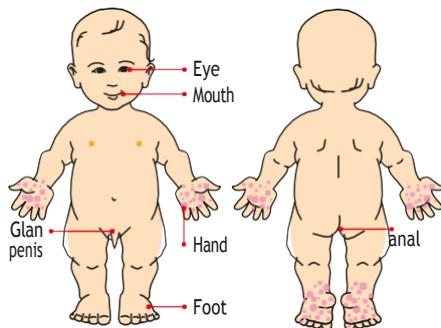
Atopic Eczema

Figure 24 : Distribution & Pattern

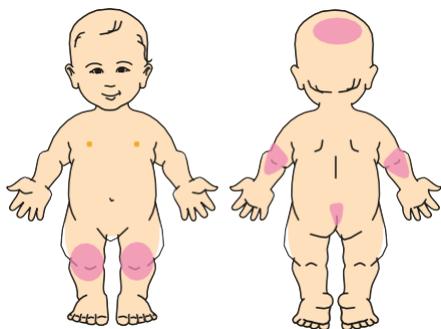
DISTRIBUTION & PATTERN (II)



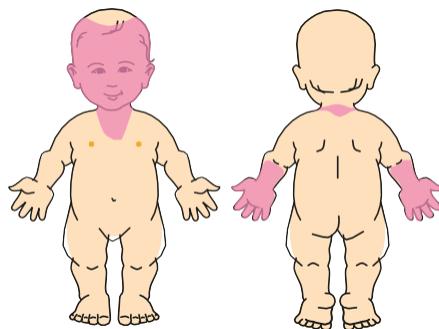
Pityriasis Rosea



Erythema Multiforme



Psoriasis



Photosensitivity Dermatitis

Figure 24 : Distribution & Pattern



CHAPTER 2

Ten common questions about Atopic Eczema (AE)



Q1: What is eczema / dermatitis ?

Eczema means inflammation of the skin that affects the epidermis and upper dermis. It has many clinical faces & can be classified based on the following:

A. DEGREE OF INFLAMMATION (*figure 1.1a-e & figure 1.2a-c*)

Normal skin → Dry skin → Subclinical eczema → Mild eczema → Severe eczema

B. CHRONICITY OF THE INFLAMMATION

Lesions pass through acute ↔ subacute ↔ chronic

Practically, there are overlaps among these stages and are bidirectional.

Eczema is the result of relative contributions of different primary & secondary components as listed in *figure 1.3*.

Acute eczema

- erythematous weepy or crusted erosions +/- intact vesicles +/- excoriations (*figure 1.4a*)

Subacute eczema

- dry, erythematous and scaly (*figure 1.4b*)

Chronic eczema

- dyspigmentation (hypo or hyper), thickened plaque and prominent skin markings (*figure 1.4c*)

C. UNDERLYING MECHANISM

- Exogenous, Endogenous or Mixed

Endogenous

1. Atopic eczema
2. Seborrheic eczema
3. Discoid eczema
4. Dyshidrotic eczema

Exogenous

1. Irritant contact
2. Allergic contact
3. Photodermatitis

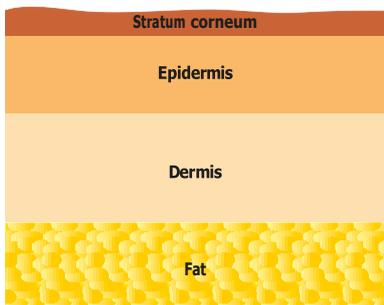


Figure 1.1a : Normal skin

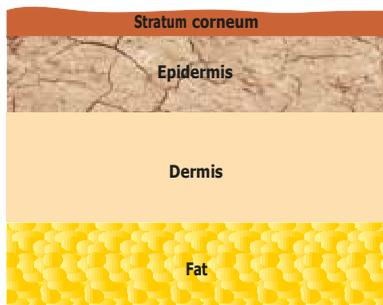


Figure 1.1b : Dry scaly skin

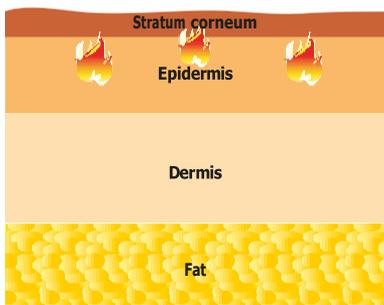
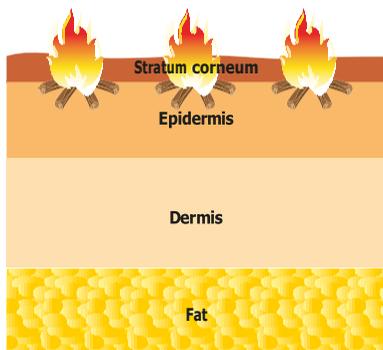
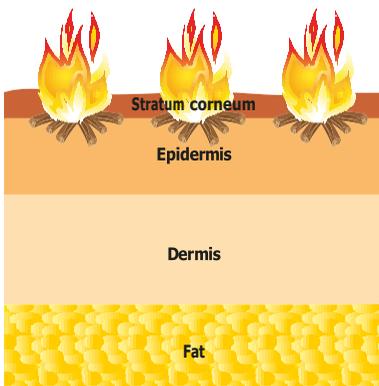


Figure 1.1c : Subclinical Eczema



**Figure 1.1d : Clinical Eczema - mild
(Mild Dry, Redness & Scaly)**



**Figure 1.1e : Clinical Eczema - severe
(Severe Weepy & Redness)**



Figure 1.2a : Severe eczema



Figure 1.2b : Mild eczema



Figure 1.2c : Subclinical eczema

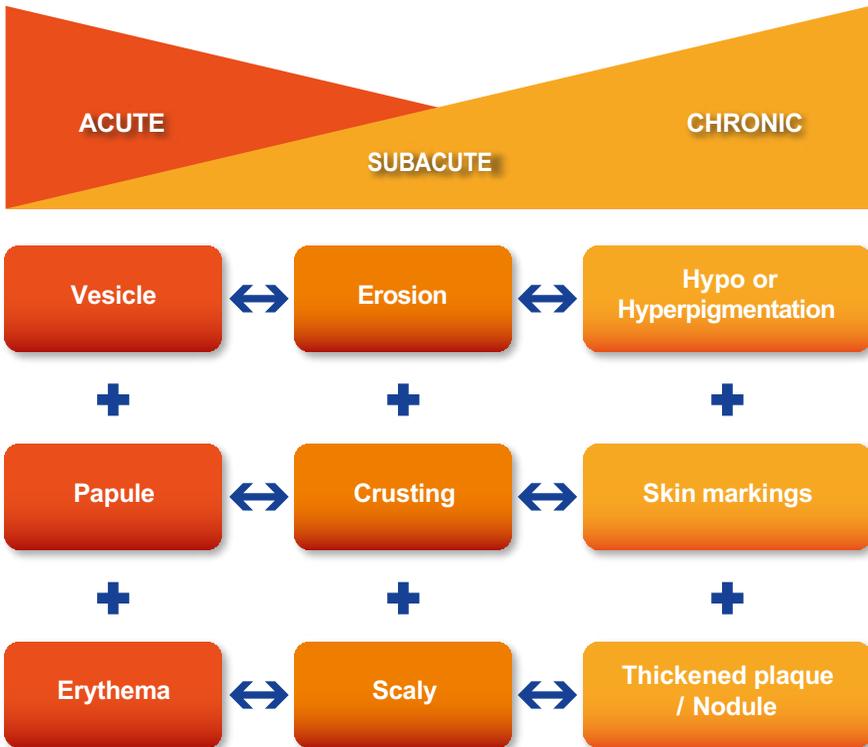


Figure 1.3 : Basic components of eczema

STAGES OF ECZEMA

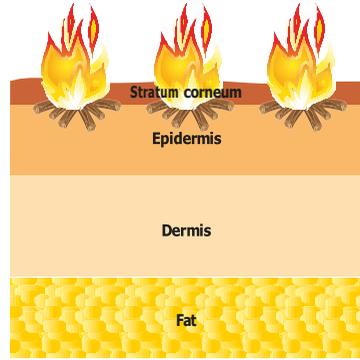


Figure 1.4a : Acute Eczema

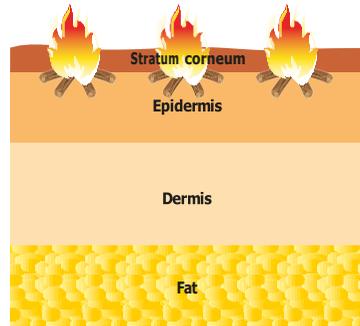


Figure 1.4b : Subacute Eczema

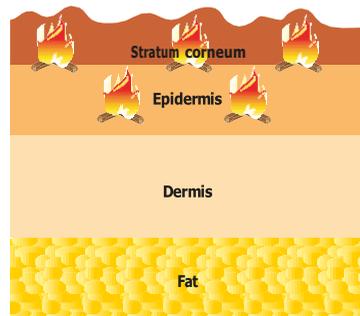


Figure 1.4c : Chronic Eczema

Q2: What is Atopic Eczema ?

Atopic Eczema is a form of endogenous eczema with multiple exogenous triggers that change dynamically with age & local environment. (*figure 2.1*)

In terms of clinical description and diagnosis, Atopic Eczema is a form of eczema with special features. (*figure 2.2a & 2.2b*)

The steps to clinical diagnosis is further elaborated in *Table 2*.

The diagnosis of Atopic Eczema is made clinically and is based on relevant history, morphology and distribution of skin lesions, and associated clinical signs. Occasionally histological features are helpful.

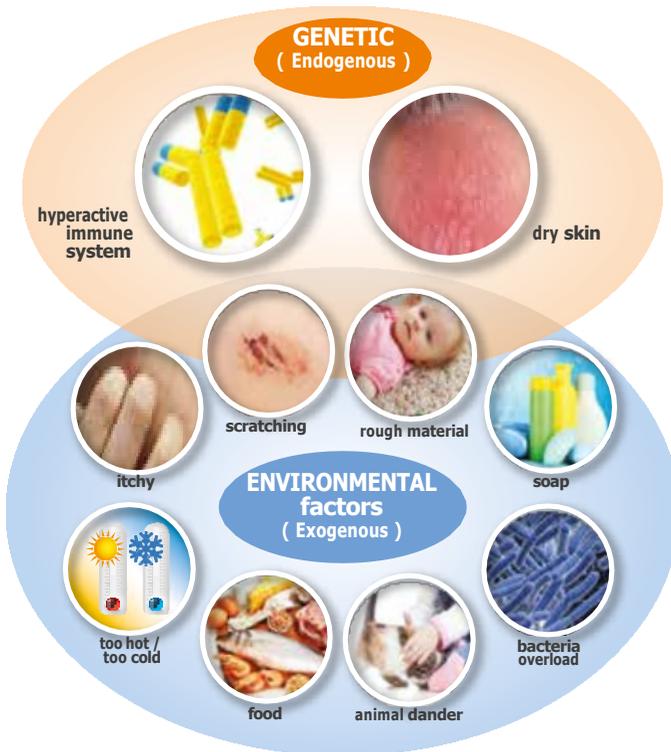


Figure 2.1 : Dynamic interplay between Genetic and Enviromental Factors in Atopic Eczema

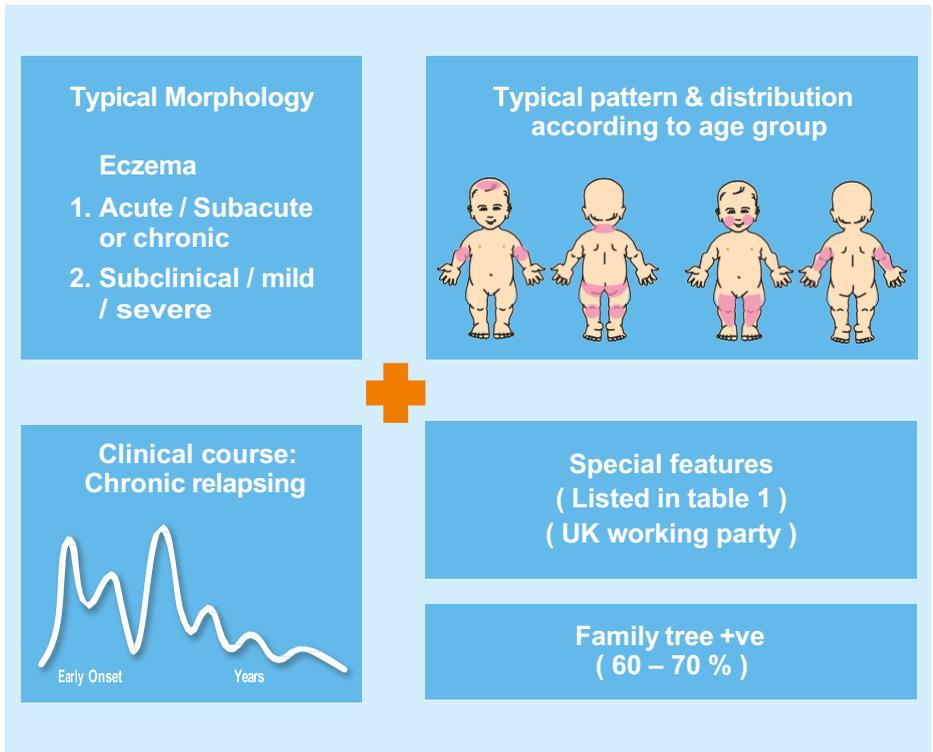


Figure 2.2a : Clinical criteria of Atopic Eczema

a. Must have:

- Pruritus

b. Plus 3 or more of the following:

- History of involvement of skin creases (front of elbows, back of knees, front of ankles, neck, around the eyes)
- History of generally dry skin in the past year
- Personal history of asthma or hay fever
- Onset under the age of 2 years
- Visible flexural dermatitis

Figure 2.2b : UK working party diagnostic criteria for Atopic Eczema

Step 1 : To identify the skin lesion as eczema

1. **Pruritus**
2. **Eczema with typical morphology according to**
Chronicity : Acute / subacute / chronic
Degree : subclinical / mild / moderate / severe

Eczema is a clinical morphological diagnosis based on its clinical stages of progression. Occasionally, skin biopsy is needed to offer histological support to differentiate it from psoriasis, lichen planus and mycosis fungoides and ichthyosis.

Step 2 : To differentiate it from other form of eczema by identification of these major features of Atopic Eczema

1. Eczema with typical pattern & distribution according to their age group (*figure 2.3*)
2. Xerosis or dry skin
3. Age of onset : usually in first 5 years of age
4. Clinical course : chronic or relapsing history with tendency to improve over months to years
5. Personal and / or Family history of atopy based on clinical assessment (asthma, allergic rhinoconjunctivitis / atopic eczema) or high serum IgE level

Step 3 : To look for other minor supportive features

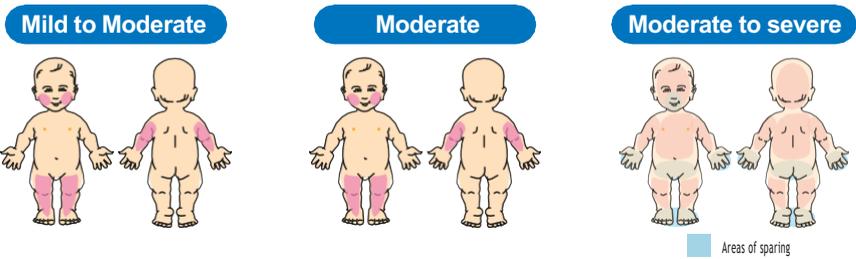
Minor features of Atopic Eczema are

A. Helpful in the diagnosis

Associated features are helpful to suggest the diagnosis of Atopic Eczema but are too non specific

1. Atypical vascular responses
2. Keratosis pilaris
3. Pityriasis alba
4. Ichthyosis Vulgaris
5. Ocular and periorbital changes - anterior subcapsular cataracts, keratoconus, recurrent conjunctivitis
6. Perioral and periauricular eczema
7. Perifollicular accentuation
8. Lab Test : Positive skin prick test and high IgE

Table 2 : Practical steps in diagnosing Atopic Eczema



Bilateral symmetrical pattern

1. cheeks,
2. extensor surfaces of extremities



Generalized but minimal over

1. Perioral & nasal, groin,
2. Palmoplantar

Figure 2.3a Major patterns according to age (0 – 2 year-old)

| | | |
|---|--|--|
| <p>Forehead & flexural (Simple)</p> | <p>Forehead & flexural (Plus 6 additional patterns)</p> | <p>Forehead & flexural (Generalized)</p> |
| | <ol style="list-style-type: none"> 1. Abdominal & other folds (Michelin signs) 2. Toilet seat pattern 3. Rhinoconjunctivitis 4. Scalp and neck pattern 5. Both Hands 6. Shoe related dorsal feet | |
| <p>Forehead & flexural (Simple)</p> | <p>Forehead & flexural " Michelin sign "</p> | <p>Forehead & flexural " Allergic rhinoconjunctivitis "</p> |
| | | |
| <p>Forehead & flexural (Toilet seat)</p> | <p>Forehead & flexural (Hands)</p> | <p>Forehead & flexural (both dorsal feet)</p> |
| | | |

Figure 2.3b Patterns according to age (2 year-old & above to Adolescent)

Q3: What is the mathematics of eczema ?

Eczema is not a single disease entity.

It may usefully be classified into 2 broad groups based on its main triggering factor(s).

1. **Exogenous**
2. **Endogenous**

The concept of endogenous & exogenous eczema is useful practically, but there is no absolute division. In many cases, the inflammation may be due to dynamic interaction between endogenous and exogenous factors. (*figure 3.1a, b & c*)

In Atopic Eczema, the primary event of skin inflammation is endogenous but secondarily triggered by multiple exogenous irritants, allergens & emotional stress. (*figure 3.2*)

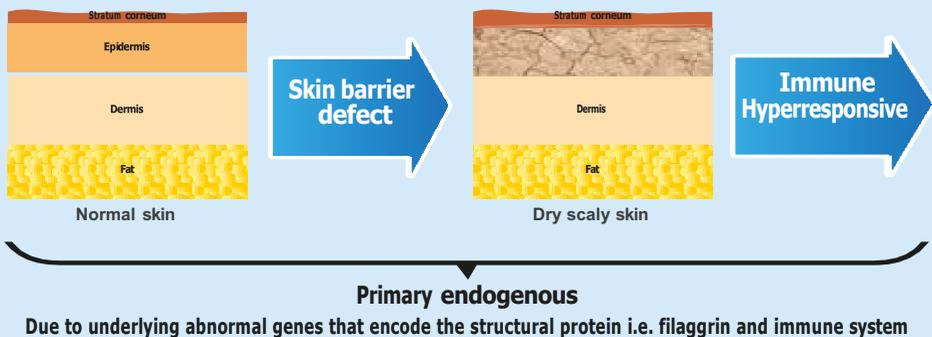
There is no known single cause for Atopic Eczema.

Atopic Eczema is thought to arise from a dynamic interaction between environmental and genetic factors. (*figure 3.3*)

As genetic factors are relatively stable, the dynamic environmental factors are dynamic with age group (*figure 3.4*) and responsible for the increasing prevalence and severity among urbanized communities.

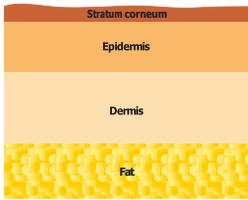
Atopy refers to the tendency towards asthma, eczema and hay fever, and is largely inherited (genetic). It is characterised by an overactive immune response to environmental factors.

Yet despite their genetic background, some children from an atopic family never develop Atopic Eczema and children with no family history can suffer from it.



Due to underlying abnormal genes that encode the structural protein i.e. filaggrin and immune system

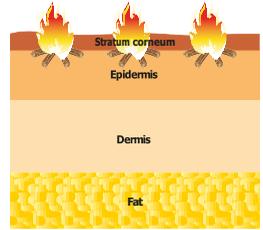
Figure 3.2 : Atopic Eczema (Primary endogenous + Secondary exogenous)



Normal skin

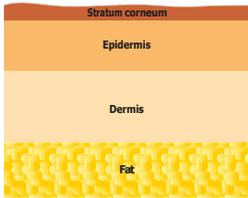


5% bleach detergent
+
1 hour



Clinical Eczema

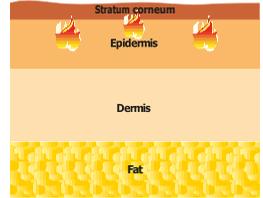
Figure 3.1a : Exogenous : Irritant contact eczema (clinical)



Normal skin



0.5% bleach detergent
+
15 minutes



Subclinical Eczema

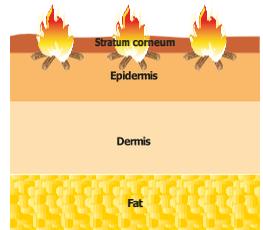
Figure 3.1b : Exogenous : Irritant contact eczema (subclinical)



Dry scaly skin

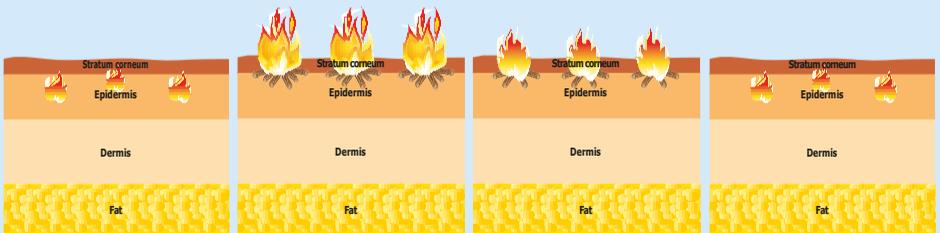


0.5% bleach detergent
+
15 minutes



Clinical Eczema

Figure 3.1c : Endogenous + Exogenous eczema (mixed)



Clinical Eczema

Secondary exogenous

- a. Harsh cleansers
- b. Hot, humid & sweating
- c. Itch and physical scratching
- d. Contact, food and aeroallergens
- e. Stress
- f. Prolonged air conditioning

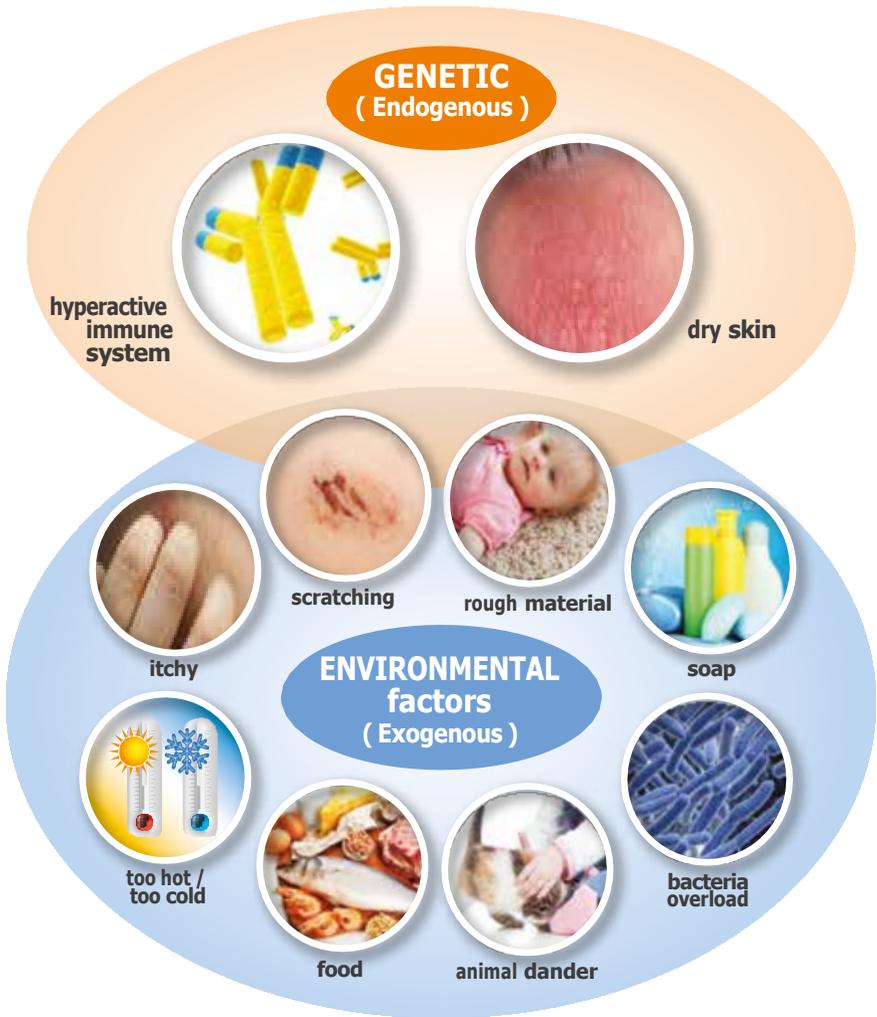
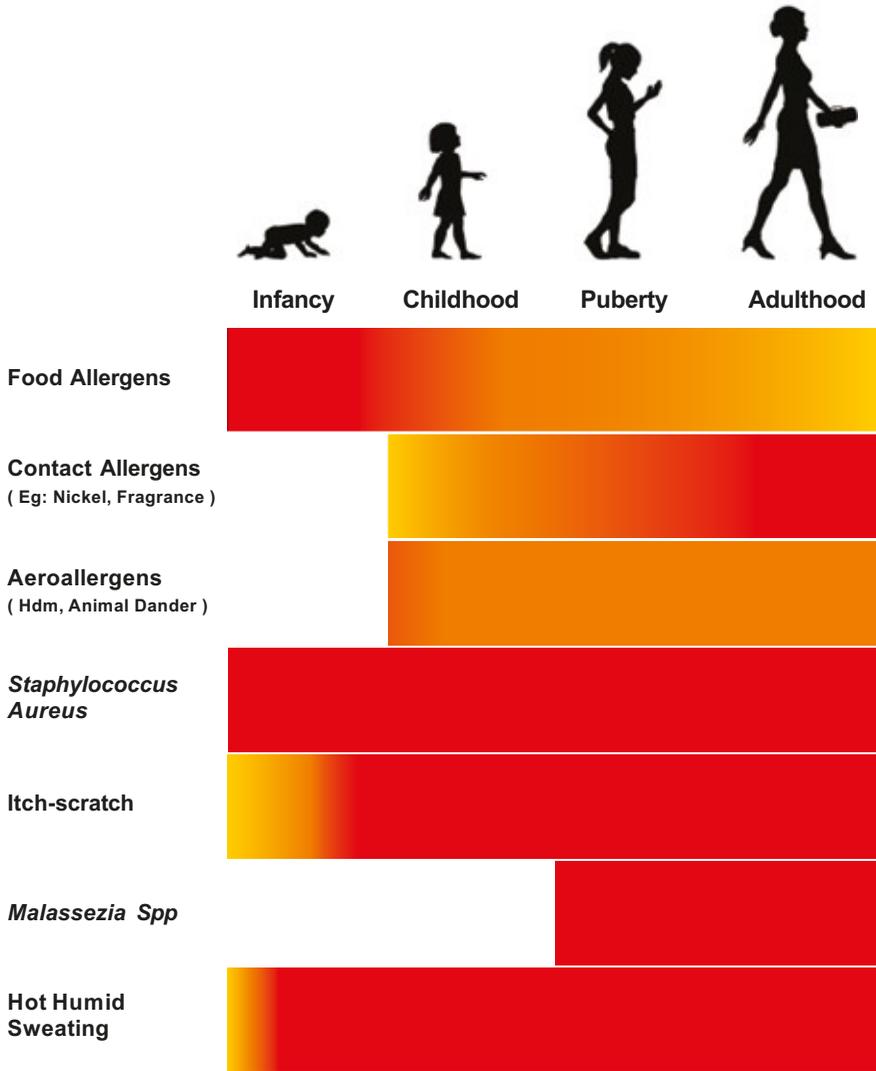


Figure 3.3 : Dynamic interplay between Genetic and Enviromental Factors in Atopic Eczema

TRIGGERING FACTORS OF AE



Need to adjust them according to

1. Age Group
3. Social Background
2. Local Climates
4. Potentially Modifiable

Figure 3.4 : Triggering factors of AE

Q4: What is the role of food allergy among patient with Atopic Eczema ?

Facts about the relationship between food allergy and Atopic Eczema.

1. **Atopic Eczema is a dynamic multifactorial inflammatory skin condition and food allergen is one of its triggering factors (not causative role)** (*figure 4.1 / 4.2*)
2. **Food allergy is more common among children with Atopic Eczema.** (*table 3*) **The role of food allergen in children with Atopic Eczema varies with:**
 - a. Age group (*figure 4.3*)
especially < 5 year-old as food allergy often improves over years in general. i.e. cow milk, chicken eggs, soya etc.
 - b. Severity of Atopic Eczema (*figure 4.4*)
about 30% of children with moderate to severe degree of atopic eczema.
 - c. Local lifestyle and exposure
The top food allergens are cow's milk, chicken eggs, soya milk, crustacean and fishes.
3. **Food manipulation and restriction is not the first line measure in children with Atopic Eczema.**

A balanced diet is important, but routine food restriction without guidance is not encouraged as food allergy is just one of the players in the pathogenesis of Atopic Eczema.

Consider food allergy and diet manipulation among children with Atopic Eczema in:

- A. Moderate to severe Atopic Eczema children < 5 year-old as research suggests that up to 30% of children with moderate & severe Atopic Eczema may have food allergy as one of its trigger.
- B. Immediate onset (IgE) reaction within 2 hours consistently to a particular group of food that shares the common allergenic component. Eg. Cow's milk, goat's milk and soya milk. Allergen specific serum IgE level, skin prick test and open food challenge are helpful in this form of food allergy.
- C. Late onset GI symptoms e.g. : Diarrhea, regurgitation, abdominal pain (24 - 48 hours after exposure to a particular group of food)
Single or double blinded food challenge after 4 weeks of elemental aminoacid / extensively hydrolysed formula is the gold standard for this form of food allergy.
- D. Failure to thrive due to food restriction, high demand and protein losing enteropathy.

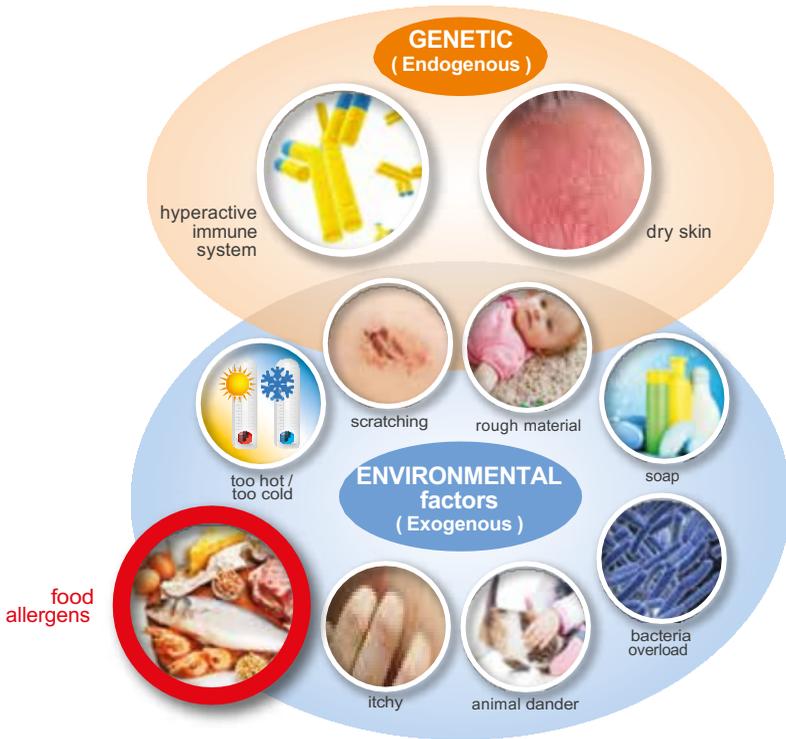


Figure 4.1 : Triggering factors of atopic eczema and food allergens are just one of the player.

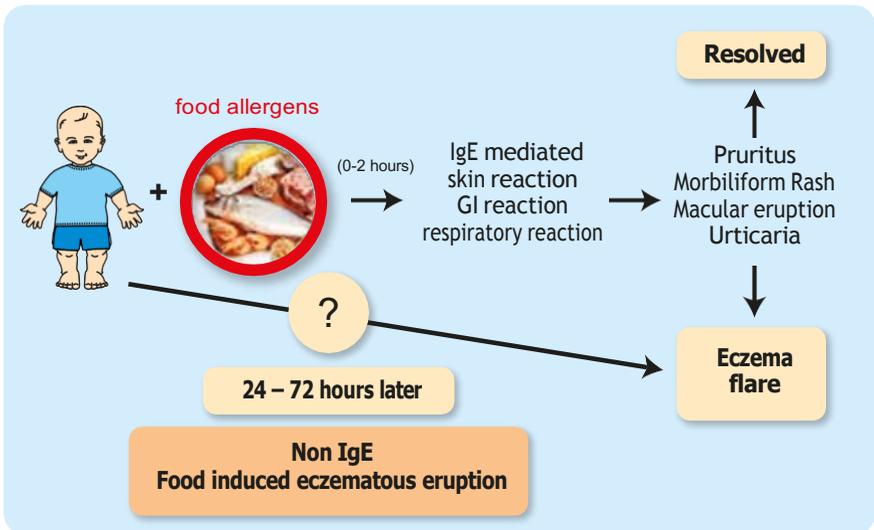


Figure 4.2 : What is often observed in food allergy

| Specific Food Allergen | Age of onset | Risk of anaphylaxis | Prognosis |
|------------------------|---------------------------|---------------------|---|
| Cow's milk | < 2 years old | Possible | 80% likely to outgrow by 4 years old |
| Hen's eggs | < 2 years old | Low | 80% likely to outgrow by 4 years old |
| Soybean | < 2 years old | Low | Most children outgrow soy allergy by 3 years old |
| Wheat | All ages | Possible | Unclear |
| Peanut | < 2 years old | High | Only 20% likely to outgrow by 5 years old; 80% with persistent peanut allergy |
| Crustace | Older children and adults | High | Tends to persist |
| Tree Nuts | Older children and adults | High | Tends to persist |

Figure 4.3 : Adapted from Singapore Ministry of Health, AMS-MOH Clinical Practice Guidelines

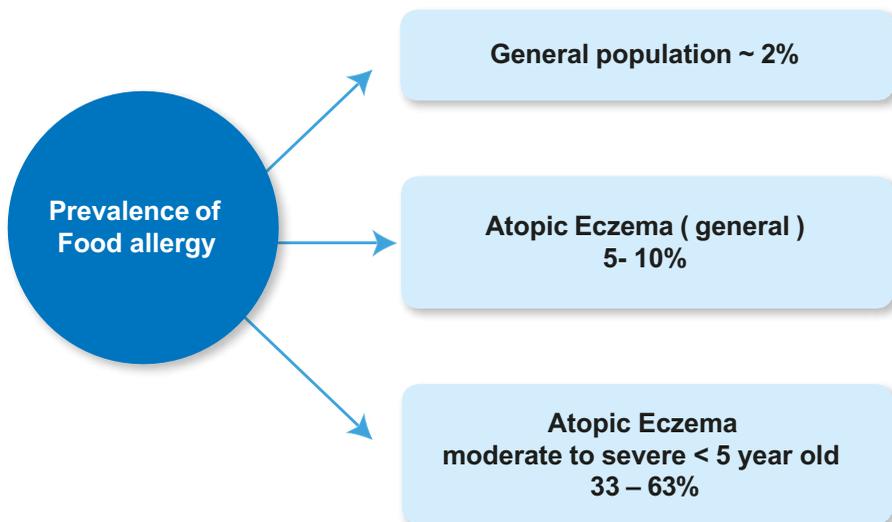


Figure 4.4 : Prevalence of food allergy in different setting

| Authors | Food allergy % |
|--------------------------|-----------------------|
| Sampson & McCaskill 1985 | 56 |
| Burks 1988 | 33 |
| Sampson 1992 | 63 |
| Eigenmann 1998 | 37 |
| Burk et al. 1998 | 39 |
| Niggemann 1999 | 51 |
| Eigenmann & Calza 2000 | 34 |
| Breuer et al. 2004 | 46 |

(Adapted from Harper's Textbook of Pediatric dermatology 3rd Edition)

Table 3 Prevalence of food allergy in children with eczema proven by DBPCFC

Q5: What is the role of allergy tests among patients with Atopic Eczema ?

Allergy tests are helpful & indicated only in a subset (15 - 20 %) of patients with Atopic Eczema as Atopic Eczema is not caused by allergens alone. As described earlier, Atopic Eczema is due to dynamic interaction among genetic (skin barrier dysfunction & immune dysregulation) and environmental triggers (allergens, irritants & microbial dysbiosis). (*figure 5.1*)

Allergy tests listed in *Table 4* are useful to identify the allergens (not irritants) that act as one of the triggering factors for Atopic Eczema via either IgE or non IgE mediated mechanism.

Consider allergy tests when the allergic components are suspected based on:

- 1. History of immediate onset reaction to a particular group of food**
- 2. Pattern analysis of his eczema** (*Figure 5.2*)
i.e. Central face, periorbital and nasal philtrum - consider aeroallergen testing
- 3. Moderate to severe Atopic Eczema**
- 4. Lacking of clinical response despite adequate microbial & irritants modification.**

There are many myths and controversies regarding Atopic Eczema, allergy & allergy tests, even within the medical profession. Despite its name, Atopic Eczema itself is not an IgE mediated allergy, nor is it necessarily associated with allergic sensitization. About 20% of adult patients with Atopic Eczema have normal total serum IgE level. However, overall, data indicates that allergy plays a triggering role in selected patients with Atopic Eczema.

People who have Atopic Eczema associated with elevated IgE or positive skin prick test are likely to have sensitized (exposed) to certain foods and aeroallergens in the environment.

The specificity of these tests are about 50%. But even if a true allergy is confirmed after food challenge, elimination of the food allergen may not directly affect the severity of the eczema as it may be a relatively minor trigger or an Independent condition that co exists with Atopic Eczema.

| | IgE & immediate onset (< 2 hours) | Non IgE & delayed onset (< 2 hours - 48 hours) |
|---------------------------|--|---|
| History assessment | ++ | (-/+) |
| Serum allergen IgE | +++ | (-) |
| Skin prick test | +++ | (-) |
| Food challenge | ++++ | ++++ |
| Patch test | - | ++ |

Table 4 : Accuracy & functions of these tests in assessing the allergic component of Atopic Eczema

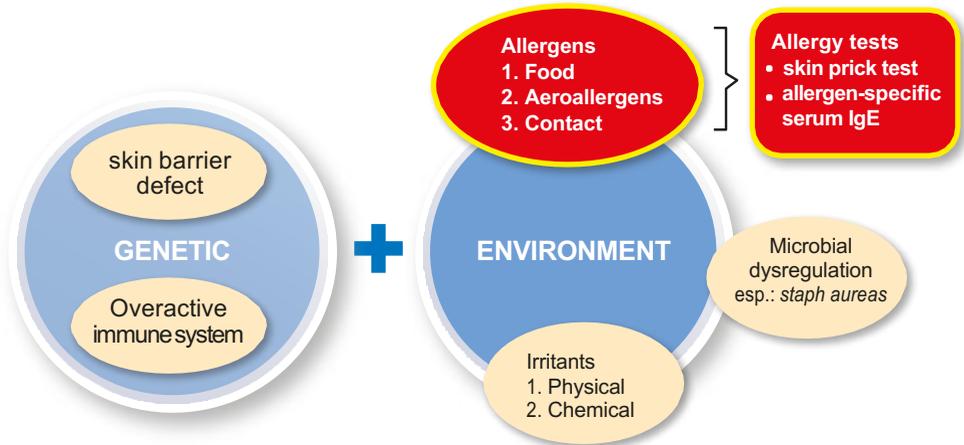


Figure 5.1 : The exogenous components of Atopic Eczema

Microbial - *Staphylococcus aureus*, *Malasezia sps* etc.

Irritants - Physical (scratching , clothing) or chemical
e.g. : sweating, harsh cleansers

Allergens - Food, aeroallergens & contact allergens



**Figure 5.2 : Eczema is noted over both his periorbital & nasal philtrum.
It was triggered by allergic rhinoconjunctivitis.**

Q6: Will my child outgrow Atopic Eczema ?

As we know, Atopic Eczema is a chronic relapsing disease with tendency to improve over years. (figure 6.1)

Poor prognostic features include a family history of the condition, early disseminated infantile disease, female gender and coexisting allergic rhinitis and asthma.

Based on these observed data (figure 6.2), we need to counsel our patients/caretakers based on the their initial expectation.

- If caretakers/patients have set an unrealistic short term plan i.e. shopping for “magic“ creams and expecting a cure from them, our primary objective in our counseling is to convince them that Atopic Eczema is a chronic relapsing disease and it requires lots of patience and lifestyle modification to control the disease activity.
- On the other hand, if the parents/patients feel hopeless after knowing that Atopic Eczema is a chronic relapsing disease, we need to reassure the patients/caretakers that there is light at the end of the tunnel i.e. 2/3 in clinical remission by puberty.

For Atopic Eczema patients in clinical remission, due to genetic predisposition, some experience a relapse of symptoms especially hand eczema after some symptom-free years. Hence, it may still affect their choice of career or employment in future.

A child with moderate to severe Atopic Eczema may have as much as 50% risk of developing asthma and 75% risk of developing hay fever.

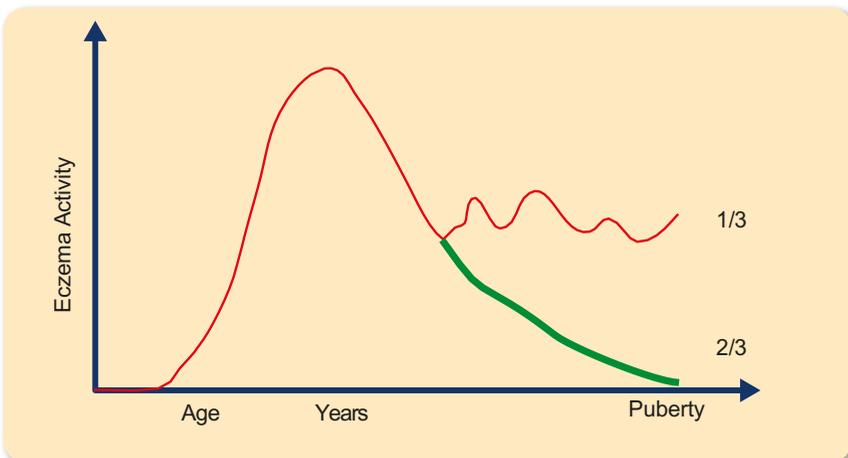


Figure 6.1 : Atopic Eczema is a chronic relapsing disease with a tendency to improve over years.

| Country | Study design | Follow up period | Resolution (%) |
|---------|-------------------------------|---|-----------------------|
| Canada | High risk birth cohort | Birth to 7 yr-old | 58% |
| Taiwan | Population based birth cohort | Birth to 10 yr-old | 69.8% |
| Germany | Birth cohort | Birth to 7 yr-old | 43.2% at age of 3 |
| Italy | Long term follow up | Over 20 years | 60.5% at the age of 6 |
| Korea | AE of first year onset | 1 st to 5 th year | 70.6% at the age of 5 |

Carlsten C, Dimich-Ward H, Ferguson A, Watson W, Rousseau R, Dybuncio A, et al. Atopic dermatitis in a high-risk cohort: natural history, associated allergic outcomes, and risk factors. *Ann Allergy Asthma Immunol* 2013; 110:24-8.
Hua TC, Hwang CY, Chen YJ, Chu SY, Chen CC, Lee DD, et al. The natural course of early-onset atopic dermatitis in Taiwan: a population-based cohort study. *Br J Dermatol* 2014; 170:130-5.
Chung Y, Kwon JH, Kim J, Han Y, Lee SI, Ahn K. Retrospective analysis of the natural history of atopic dermatitis occurring in the first year of life in Korean children. *J Korean Med Sci* 2012;27:723-8.

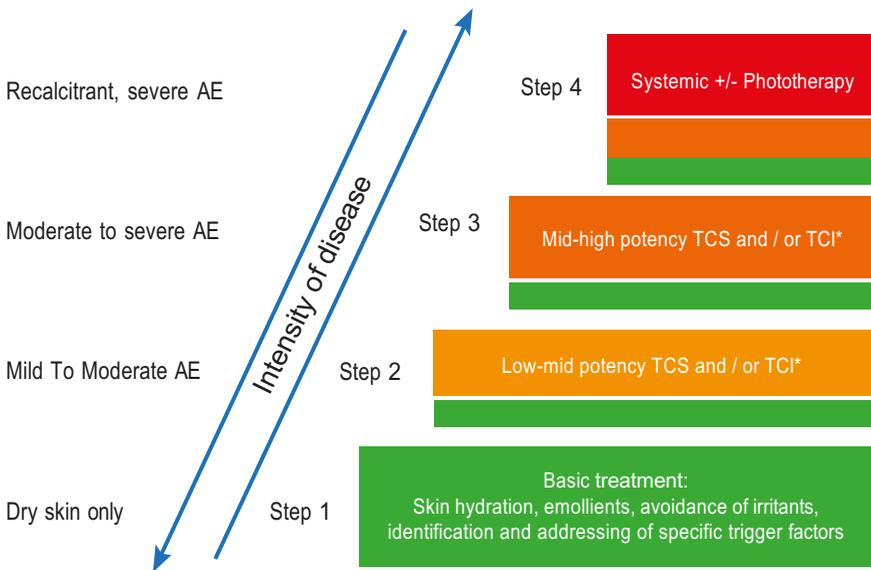
Figure 6.2 : Age & rate of resolution of Atopic Eczema



Q7: How do you manage Atopic Eczema ?

Once the clinical criteria of Atopic Eczema are fulfilled and parents are well informed about the diagnosis, we need to further stratify them according to their severity (chronicity), physical & psychosocial complications. The Stepwise approach is often recommended. (*figure 7.1*)

As Atopic Eczema is a chronic, relapsing, dynamic, multifactorial condition with a lot of misconceptions, the most important aspect in the management of Atopic Eczema is education and empowerment. The other pillars in the management of Atopic Eczema are listed in (*figure 7.2*)



(1) TCS = Topical corticosteroids,

(2) TCI = Topical calcineurine inhibitors

* over the age of 2 years

(3) Systemic : Prednisolone / Azathioprine / Cyclosporin / Methotrexate

Figure 7.1 : Long term stepwise approach based on disease severity

1. Education and empowerment of patients and caregiver(s)
2. Modification of environmental trigger factors
3. Control & modification of the itch-scratch cycle
4. Repair and restoration of skin hydration & barrier functions
5. Control skin inflammation
6. Clear clinical infections & restore skin microbiota



Figure 7.2 : Main pillars in the management of Atopic Eczema

A. TO RESTORE SKIN HYDRATION

Regular emollients - Hypoallergenic and low irritation (*Question 8*)

Avoid harsh cleansers

Properly humidify the room air if prolonged period in air conditioned room

B. ANTI ITCH PACKAGE

Unlike urticaria, histamine is not the main mediator that mediates the itch related to Atopic Eczema.

Anti histamines act primarily by blocking the H1 receptors and ameliorating histamine-induced pruritus. However, histamine is only one of many itch mediators in Atopic Eczema, minimizing benefit from anti histamine treatment. (*Figure 7.3*)

C. ANTI MICROBIAL

- Clinical bacteria infection

Localized: Topical mupirocin, fusidic acid and retapamulin

Extensive: Systemic cloxacillin, 1st and 2nd generation cephalosporin and macrolides.

Antiseptic bath - to reduce the density of *Staph aureus*

i.e. 0.005% Sodium Hypochlorite 10 minutes 2-3 times daily

- Eczema herpeticum - Oral / IV Acyclovir is indicated

- Secondary fungal infection requires either topical or systemic anti fungal i.e. topical Azoles or oral griseofulvin, terbinafine

D. ANTI INFLAMMATORY AGENTS (*Question 9*)

Often require combination rotational therapy of steroid, calcineurine inhibitor and emollient with anti inflammatory property

For resistant cases, consider systemic immune modulators and phototherapy

E. AVOID AND MODIFY THE TRIGGERING FACTORS (*refer question 10*)

F. EDUCATION & EMPOWERMENT

A structured, patient centered education program that is dynamic and tailored to local setting is essential.

It has 3 steps:

Step 1 : Actively debunk the myths & fallacies about Atopic Eczema

- its cause(s), trigger(s), magic creams etc

Step 2 : Infuse the facts about Atopic Eczema to all the major caretakers and patients

Step 3 : Empower the patient/caretaker with eczema action and monitoring plan that is tailored to the patient's social background.

ANTI ITCH PACKAGE

1. Avoid provoking factors (scrubbing, bathing >10 minutes, hot water bathing, harsh cleansers, low humidity air conditioned room, copious sweating)
2. Adequate hydration with regular moisturizers. Some of the moisturizers have ingredients with direct anti itch effects, for example, Glycyrrhethenic acid, Licochalcone, Palmitoethanolamide etc.
3. Gentle cleansers
4. In air conditioned rooms, use a humidifier to keep humidity between 35-40% and use thicker moisturizer.
5. Topical anti inflammatory creams i.e. TCS & TCI have both anti inflammatory and anti itch properties. But is limited to two applications each day
6. Wet wrap treatment
7. During acute flares, sedative anti histamine (5-7 days) may be helpful but not for chronic itch management. Non sedative anti histamines have variable results but may be helpful in the subset of atopic eczema patients with cholinergic or other forms of urticaria
8. Behavioural modification (Itch-scratch to Itch-tap) & keep the nails short
9. For resistant cases, consider systemic immune modulators treatment or phototherapy
10. Other options: Gabapentin, amitriptyline, aprepitant etc.

Fig 7.3 : Anti Itch Package

Q8: Can I keep pets, swim, bath 3 times perday, buy an expensive vacuum cleaner with hepa filter or.... if I have Atopic Eczema ?

The answer simply put, is both yes and no as there is no single fixed recipe for all patients with Atopic Eczema in modification of triggering factors.

We need to tailor it according to their age groups, clinical pattern of their eczema, the local climate and their social background. (*figure 8.1*)

As many of these triggering factors are minor and only partially modifiable, selective and “moderate” modification is preferred instead of “total pan-avoidance or elimination” i.e. total house dustmite elimination, extensively hydrolysed milk formula for all infants with Atopic Eczema etc.

TWO EXAMPLES ARE LISTED BELOW

(A) Frequency and duration of Bathing & Atopic Eczema

According to US guidelines, there is no clear frequency or duration of bathing that is optimal for patients with Atopic Eczema. However, it is generally recommended that up to **once-daily bathing** be performed to remove serous crust, as long as moisturizers follow as above; the duration should be limited to short periods of **time (e.g. 5-10 minutes)** with use of warm water.

But in Malaysia with tropical hot and humid climate, it is generally recommended for up to twice daily bathing (5-10 minutes each). In addition to this, short showers (1-2 minutes) to rinse off sweat is allowed after any outdoor activity.

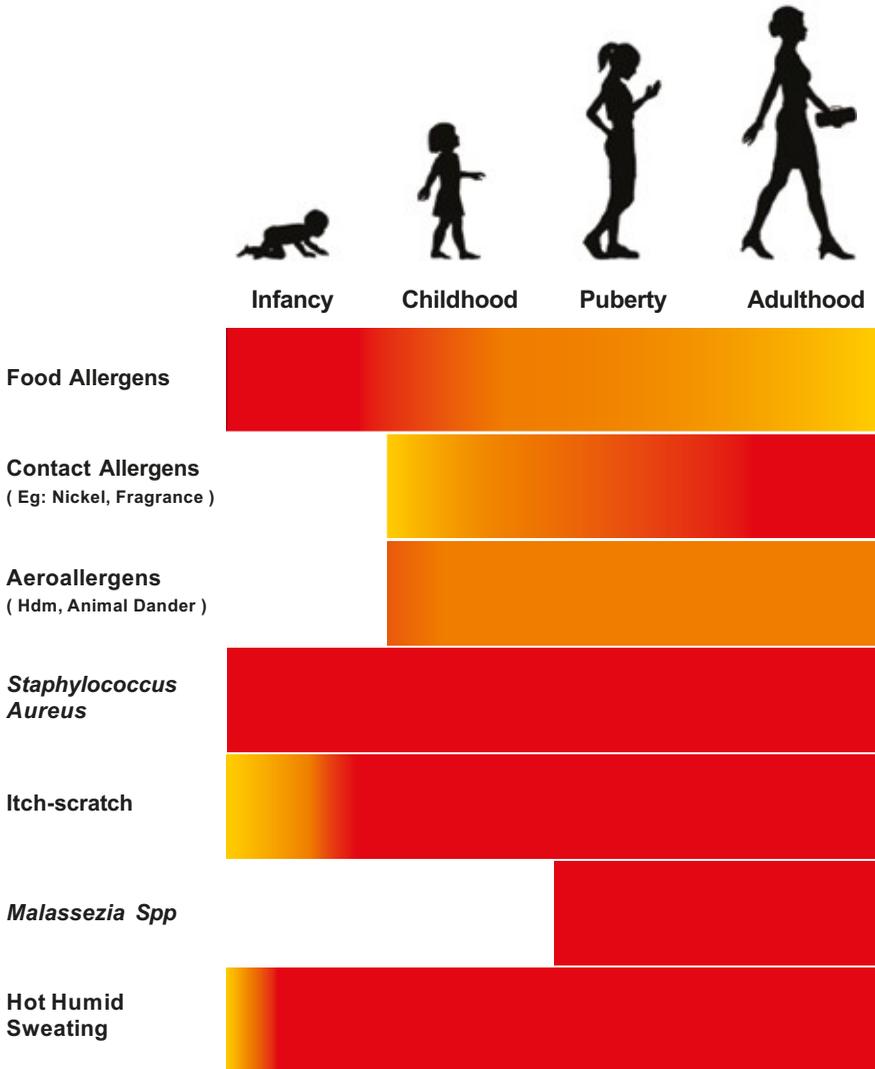
(B) Swimming & Atopic Eczema

Bleach bath at 0.005% sodium hypochlorite concentration for 5-10 minutes twice weekly is recommended & proven effective in the management of Atopic Eczema. This is similar to swimming in an optimally chlorinated (0.005%-0.01% sodium hypochlorite) indoor swimming pool for 15 minutes twice weekly plus immediate rinse off and moisturizer application.

Although swimming is beneficial among children with Atopic Eczema, the benefits of the antiseptic activity of a bleach bath may be neutralized by skin dryness & irritation

1. induced by prolonged swimming if the duration & frequency is increased to daily for 1 hour (instead of 10 minutes twice weekly)
2. without immediate rinse-off and adequate moisturizer.
3. Swimming under the hot sun may further aggravate eczema due to copious sweating.

TRIGGERING FACTORS OF AE



Need to adjust them according to

1. Age Group
3. Social Background
2. Local Climates
4. Potentially Modifiable

Fig 8.1 : Factors that contribute to the activity of Atopic Eczema according to age group

Q9: How do you manage the inflammatory component of Atopic Eczema ?

Basic information & principles about management of the inflammatory component of Atopic Eczema.

1. Understand the different stages of skin inflammation in Atopic Eczema:

Clinical inflammation → subclinical inflammation → True remission

(refer question 1)

Subclinical or microscopic inflammation is often under-recognized & inadequately managed.

Practically, treatment of inflammatory stage of Atopic Eczema can be divided into 2 stages:

- A. Acute flare - from clinical inflammation to subclinical inflammation
- B. Maintenance - from subclinical inflammation to true remission & prevent relapse

2. Among children with Atopic Eczema, many have a few “hotspots” or areas where eczema tends to recur (figure 9.1)

3. There are many effective medications with both anti-inflammatory & anti-itch effects. (figure 9.2)
Corticosteroid is the mainstay of treatment during acute flare but combination proactive therapy is preferred during maintenance phase to improve its safety profile.
For resistant cases, systemic agents and phototherapy are needed.

4. Selection is based on the body sites (face < palm), chronicity & thickness (acute < chronic) & extent of the eczematous lesions. (figure 9.3 & 9.4)

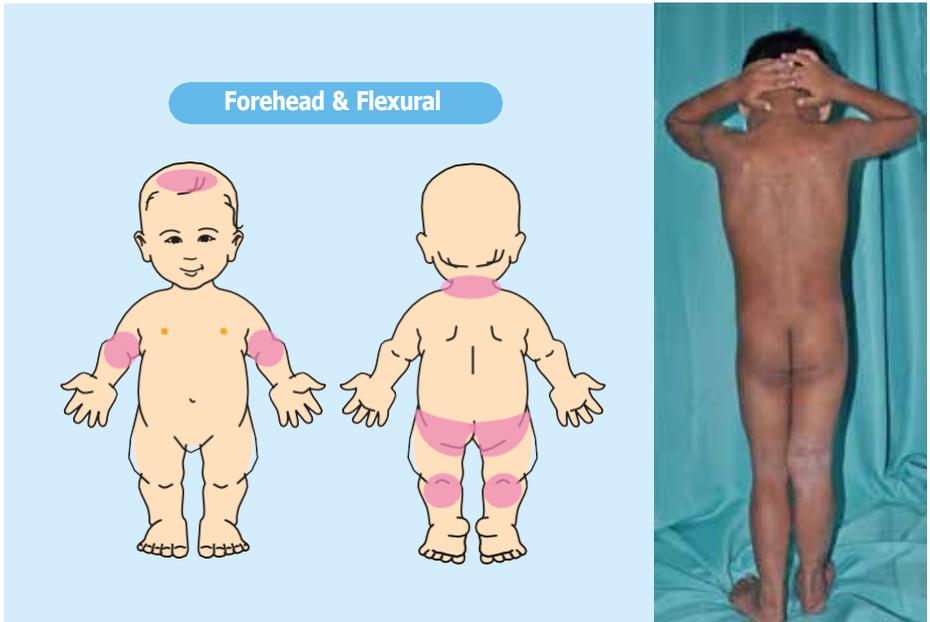


Figure 9.1 : Hotspots recognition

ANTI-INFLAMMATORY AGENTS FOR ATOPIC ECZEMA

1. Topical corticosteroid (TCS)

TCS has been the mainstay of treatment of inflammation. It is safe & effective if we understand its

- A. Potency based on its anti inflammatory effects (Grade 1-4)
- B. Degree of absorption according to our body sites (*Figure 9.5*)
- C. Strategy – Proactive or Reactive

2. Topical calcineurine inhibitor (Tacrolimus & Pimecrolimus)

3. Regular emollient especially those with anti itch & anti inflammatory features

4. Phototherapy especially NB UVB

5. Systemic immune modulators

Prednisolone, Azathioprine, Cyclosporine & Methotrexate & Mycophenolate mofetil

6. Other options : Biologics (anti IL-4, Omalizumab, IV immunoglobulin)

Fig 9.2 : Anti-inflammatory medication



Figure 9.3a :
Anti inflammatory agents for eyelids eczema
Class 1 TCS < 2 weeks + TCI + Emollient



Figure 9.3b :
Anti inflammatory agents for eyelids eczema
Class 1 / 2 TCS + TCI + Emollient



Figure 9.3c :
Anti inflammatory agents of body eczema
Class 2 / 3 TCS + TCI + Emollient



Figure 9.3d :
Anti inflammatory agents of body eczema
Class 3 / 4 TCS + TCI + Emollient

| | | |
|---|---|--|
| <p>Class 4 Clobetasol Propionate</p> <p>Class 3 Mometasone Furoate Fluticasone Propionate Betamethasone Valerate</p> <p>Class 2 Clobetasone butyrate Desonide</p> <p>Class 1 Hydrocortisone acetate/ base</p> | Age group: Adolescent > 12 yr old | <p>Palm / Soles</p> <p>Limbs</p> <p>Flexures</p> <p>Face</p> <p>Scrotum / Periorbital</p> |
| | Chronicity of eczema: Chronic Lichenification plaque | |
| | Formulation: Ointment > Cream > Lotion | |
| | Chronicity of eczema: Acute eczematous patches | |
| | Age group: Young baby | |

Figure 9.4 : Potency & Formulation ↔ Skin thickness, stage

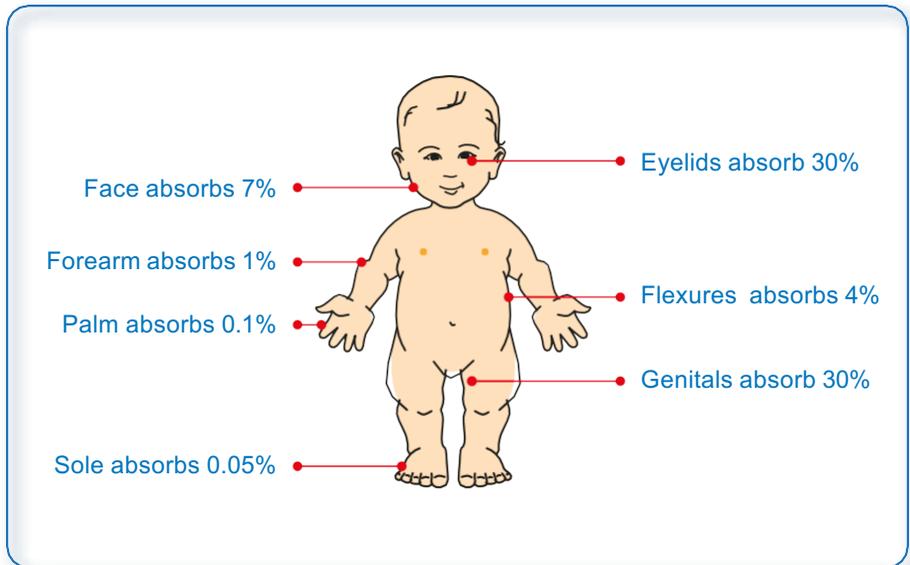


Figure 9.5 : Skin absorption of topical steroids

Reactive treatment approach

It is well established, widely accepted and for all licensed TCS and TCI. In this approach, combination of emollients with symptomatic anti-inflammatory therapy consisting of topical glucocorticosteroids (TCS) or topical calcineurin inhibitors (TCI) on an “as needed basis“.

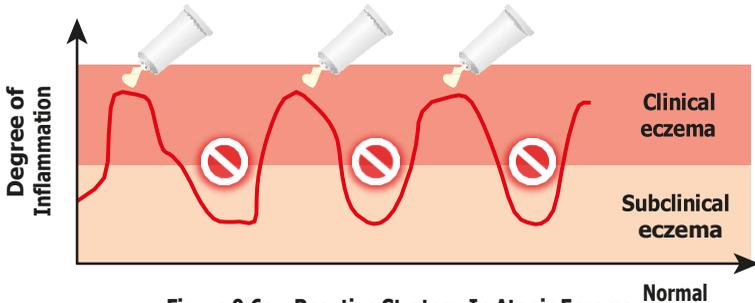


Figure 9.6a : Reactive Strategy In Atopic Eczema

Practically, the eczema first needs to be brought under control before a 2-3 times weekly regimen can be instituted. Proactive studies with TCS up to 16 weeks and TCI up to 52 weeks have shown some favorable results.

Proactive treatment approach

It is helpful in patients with hotspots or when eczema tends to recur in the same locations.

Clinical eczema is featured by redness, itchiness, dry or weeping +/- crusting. After 1-2 weeks of topical corticosteroid or calcineurine inhibitor, the redness and itchiness will reduce gradually and subclinical eczematous stage starts on the day that...

The redness disappears visually.

This non erythematous normal-looking resolving Eczema skin is not normal at all, but is characterized by a clinically meaningful barrier function defect and microscopic inflammation.

After a period of stabilization, topical inflammatory therapy (TCS +/- TCI +/- Emollient) is instituted in the hotspots, rather than waiting for a flare of eczema in a traditional reactive strategy.

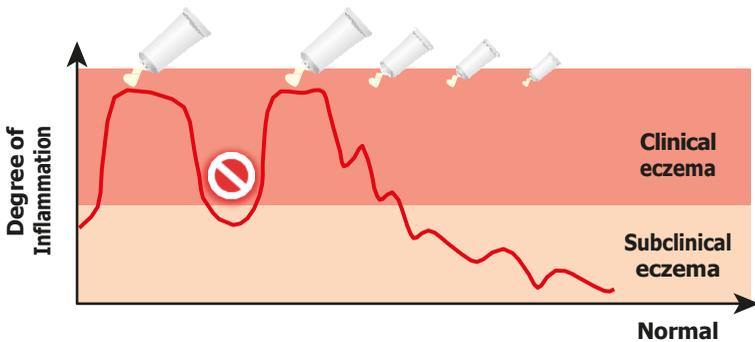


Figure 9.6b : Reactive vs Proactive Strategy In Atopic Eczema

Q10: How do you select your moisturizer?

Dry skin almost always accompanies Atopic Eczema and moisturizers are crucial to the successful management of Atopic Eczema. Hydrating the dry skin can lessen stinging and itching, provides a steroid-sparing effect and reduces the number of flares.

The differences of the ingredients used in normal moisturizer and formula designed for Atopic Eczema are briefly illustrated in (*figure 10.1*).

During the education and empowerment session, we need to help them to select their own “ideal“ moisturizer(s) & cleanser. (*figure 10.2*) The different emollient textures and forms should depend on the body sites, degree of dryness, stages of eczema and the humidity of the climate and personal preference.

Moisturizers are available in few forms: oil, lotion, cream and ointment. (*figure 10.3*)

Ointment based moisturizer is the most occlusive, least preservatives and suitable for dry lichenified eczema & for overnight protection in air conditioned room. On the other hand, in a hot, humid environment, they may trap sweat with associated irritation of the skin. Over application can lead to folliculitis and miliaria.

Creams are less greasy, easier to spread on the red inflamed eczematous lesions & over the hairy sites compared to occlusive ointment. Furthermore, creams are often more cosmetically acceptable.

Lotions are less occlusive than creams & ointments. These are best applied over hair bearing areas or during outdoor activities.



Figure 10.1 : Type of moisturizers

A large variety are available, reflecting that there is no 'right' moisturiser for all weathers & all patients. Below is a guide:

A. EFFICACY:

Hydration effect of a moisturizer is contributed by the occlusive and humectant components (*figure 10.4*) in it that can be measured by TEWL (Transepidermal water loss).

Anti inflammatory effect is contributed directly by the added active ingredients and indirectly by improvement of skin barrier function.

B. SAFETY PROFILE:

Hypoallergenic, non sensitizing, fragrance free

Can be assessed based on its ingredients, pre marketing patch test & post marketing surveillance. Trial on one side of his forearm for 2 - 4 weeks is encouraged before total body application for any new product.

C. COSMETICALLY

elegant & long lasting are important to improve the compliance.



D. COST

5 g / single application(Infant) to 20g / single application (Adult)
Monthly requirement for an infant is about 300g for twice daily application and 1.2 kg for an adult.

E. CLIMATE

(Indoor and outdoor)

Emollients should be applied two to three times daily or as frequently as the skin gets dry depending on the climate or the use of air conditioning. A less greasy formulation is preferred in hot & humid climate.

F. PERSONAL PREFERENCE

Figure 10.2 : Selection criteria of an ideal moisturizer for Atopic Eczema

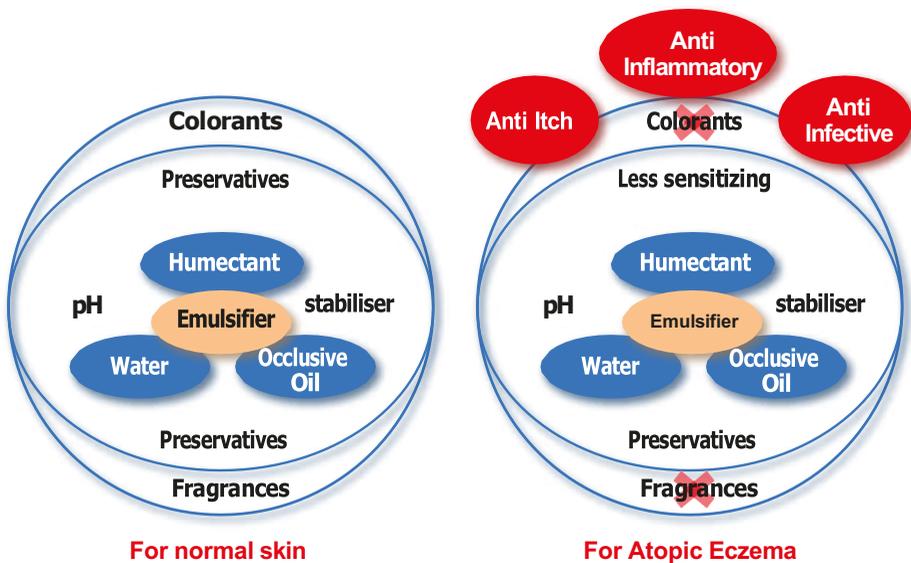


Figure 10.3 : Basic components of the moisturizers

| Class | Mode of action | Examples |
|--------------------------------------|--|--|
| Humectant | Water holding capacity of stratum corneum | Saccharide isomerate Glycerin Urea Sodium PCA Arginine |
| Occlusives | Form a hydrophobic layer to reduce water evaporation | Mineral oils Petrolatum Ceramide Squalene, Dimethicone Coconut oil, Kernel Oil Shea butter Lanolin |
| Anti Itch & anti inflammatory agents | Anti inflammatory Anti itch | Glycyrrhethic acid, Palmitoyl ethanolamine Coconut oil |

Figure 10.4 : Basic ingredients of a moisturizer

MOISTURIZER : TAKE HOME MESSAGES FOR PATIENTS / CARETAKERS

1 Patients should be advised to cleanse with a gentle non-irritant cleanser, moisturize all over two to three times daily or as frequently as the skin gets dry.

2 Emollients should be used regularly even when no actual inflammatory skin lesions are seen, and also during active disease flares together with topical anti-inflammatory agents (TCI & TCS)

3 Apply before swimming and after bathing while the skin is still moist (within 5 minutes) in order to trap the moisture on the skin.

4 The sequence of application is to first apply the topical steroid or immunomodulators, and then followed by the moisturizer. This is recommended but not evidence based.

5 Avoid inserting fingers into moisturizer pots to avoid contamination.

6 Cooling the creams may help provide a soothing sensation over the dry and inflamed skin.



CHAPTER 3

Fifteen Clinical Cases of Atopic Eczema (AE)



CASE 1

Infantile Atopic Eczema



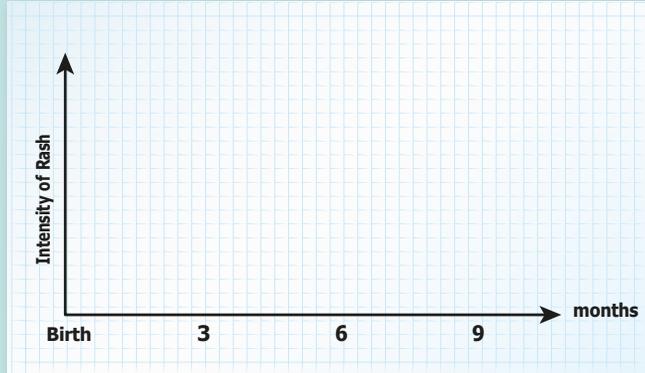
Fig1.1 / Fig1.2 : Dry, mild redness and scaly papules & patches are noted over his forehead, both cheeks and his back.
Fig1.3 / Fig1.4 : Symmetrical eczematous rash noted over the lateral aspect of his both shins.

History

7 month-old baby boy presented with itchy rash for 12 weeks.
He has positive family history of Atopic Eczema.

Clinical Course

— Chronic relapsing rash for 3 months



Distribution & Pattern

Fig 1.5 : Acute & Subacute Eczema
- Ill defined red scaly patches noted over his forehead and cheeks with excoriated papules and vesicles.

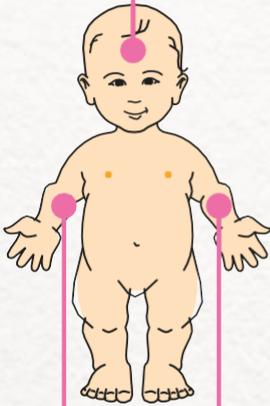


Fig 1.6 : Acute Eczema
- Red edematous scaly plaque with excoriations in linear arrangement.



Clinical Diagnosis

ATOPIC ECZEMA

1. Acute & Subacute Stage

2. Infantile Pattern

- Bilateral symmetrical over his face, extensor surfaces of upper and lower extremities
- Atopic Eczema is a clinical diagnosis that is based on a careful and thorough history examination of the child and follow up for its chronic relapsing course.
- There is little need for allergy tests and investigations in term of diagnosis.

Main differential diagnosis of Infantile onset Atopic Eczema are Seborrheic dermatitis & Scabies infestation.

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment

Facts about Atopic Eczema need to be addressed clearly and misconceptions need to be debunked actively in infantile onset Atopic Eczema. Common misconceptions about infantile onset Atopic Eczema are:

A. The Real Connection Between Atopic Eczema and Food Allergy

(Cause or Caused or Trigger or Co-exist)

Since Infantile Atopic Eczema commonly starts during the first year of life that coincides with introduction of weaning diet, the clinician is often faced with parents' overriding concern about food allergy in the infant with Atopic Eczema.

Misconception is that food allergy is the single factor that “causes” Atopic Eczema, lending support to the role of food exclusion in the management of Atopic Eczema. (from cow milk to goat milk, soy based milk etc.) In some cases, the misconception could become pathological as unwarranted food avoidance, particularly in infants, can produce nutritional deficiencies and adversely affect growth.

Many think that Atopic Eczema is caused by the food allergy, whereas it can be the other way around as eczematous skin may act as the primary site of food allergen exposure and contact sensitization.

A 5-year multicenter study in infants age 3-18 months found that in mild cases of Atopic Eczema, roughly 15% of infants had definite food allergies (Haniffin et al 2011).

But bear in mind that among these 15% of the cases, food allergy either acts as a trigger or just coexists with Atopic Eczema with minimal interaction.

B. Clinical course of Atopic Eczema

Atopic Eczema is a chronic relapsing disease with tendency to improve over years.

In practice, the doctor is often faced with caretakers that practice “doctor shopping” & “magic cream hunting” and their main aim is to cure their Atopic Eczema babies over days to weeks without further relapse.

C. Role of allergy testing (Skin prick test & serum allergen specific IgE)

Atopic Eczema is diagnosed based on a clinical criteria.

In the identification of triggering factors of Atopic Eczema, these tests are useful only in a subset of patients with moderate to severe Atopic Eczema or those with history of immediate onset reaction to certain groups of food or aeroallergens.

D. Steroid concern, steroid fear and steroid phobia is common among children with

Atopic Eczema especially among the infantile onset subtype.

2. Modification of triggering factors based on age group, clinical pattern & social background (refer question 8 ; figure 8.1)

3. Skin dryness & restoration of skin barrier (refer question 10)

Emollients should be prescribed in adequate amounts and these should be used liberally and regularly.

Apply it after bathing while the skin is still moist (within 5 minutes).

Emollients should be used during active disease flares in conjunction with topical anti-inflammatory agents, and also as maintenance therapy.

A regular use of emollients has a short and long term steroid sparing effect & anti-itch effects.

Recently, two pilot studies have shown that early moisturization may delay or prevent the onset of Atopic Eczema.

4. Anti-itch strategies (refer question 7 ; figure 7.3)

Sedating antihistamines may be used for a short term (7 - 14 days), under supervision where itch of eczema causes sleep disturbance. But it has a minimal role in the management of chronic itch related to Atopic Eczema.

Non sedative anti histamines may be helpful for a subset of Atopic Eczema patients with cholinergic urticaria and allergic rhinoconjunctivitis.

5. Anti-inflammatory – topical & systemic

Class 1 and Class 2 Topical corticosteroid are preferred for infantile Atopic Eczema patients. Class 3 TCS is reserved for lichenified lesions and only for short durations.

Topical calcineurine inhibitor is licensed for children aged more than 2 years and adults.

6. Antimicrobial and restoration or skin microbiota



CASE 2

Infantile Atopic Eczema with Bacteria Infection



Fig2.1 / Fig2.2 : Eczematous rash noted over his forehead, both cheeks and his shin.

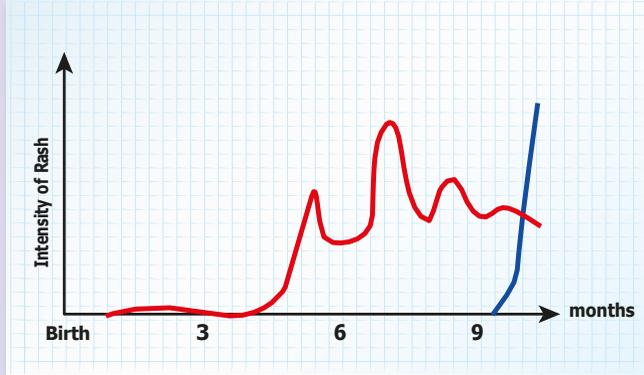
Fig2.3 / Fig2.4 : Eczematous rash noted over both lower limbs (left > right).

History

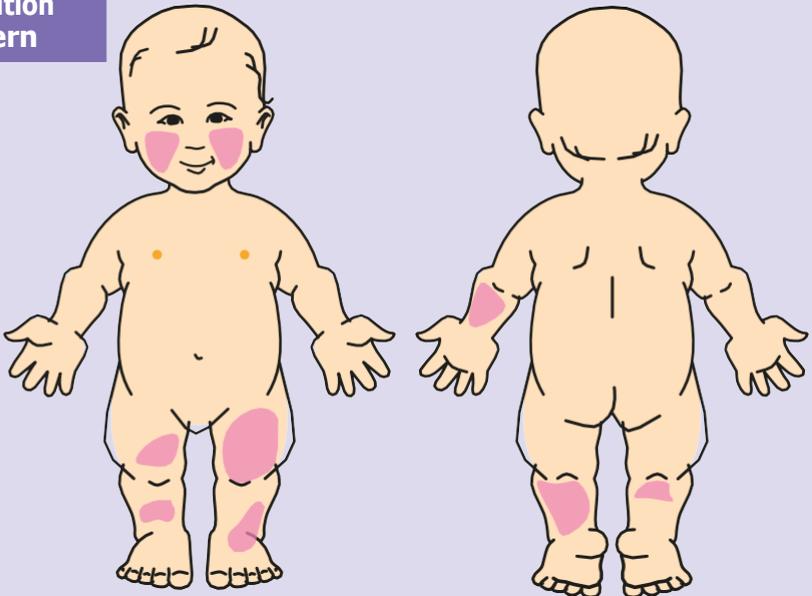
11 month-old baby boy presented with itchy rash for 3 months. About a week ago, his rash has worsened with yellowish discharge and low grade fever. The rash was unresponsive to his usual routine.

Clinical Course

- Chronic relapsing rash for 3 months
- New weepy rash for 1 week



Distribution & Pattern



Morphology & Progression

Fig 2.5 : Red scaly papules & crusted erosions are noted over his cheeks, nares & chin.



Fig 2.6 : Red papules, vesicles & crusted erosions surrounded by ill defined scaly patches are noted over his left lower limb.



Fig 2.7 : Red papules, vesicles & crusted erosions surrounded by ill defined scaly patches are noted over his left lower limb.



Clinical Diagnosis

ATOPIC ECZEMA

1. Infantile
2. Acute & Subacute Stage
3. With Secondary Bacteria Infection

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment about the relationship between Atopic Eczema & *S. aureus*

Besides itch and skin dryness, *S. aureus* colonization is another hallmark of Atopic Eczema. It is estimated that patients with Atopic Eczema carry *S. aureus* in 90% of clinically affected areas and 75% of clinically uninvolved areas. A small study in Singapore reported that *S. aureus* was isolated in 53% of patients with mild dermatitis and 100% of those with moderate to severe dermatitis. (Goh *et al* 1997)

Even extensive eradication steps with antibiotic and antiseptic are only transiently successful, with a recurrent rate of almost 100% in Atopic Eczema patients (Breuer *et al* 2002). *S. aureus* can contribute to persistent skin inflammation in Atopic Eczema by secreting toxins with superantigenic properties, exogenous proteases and reduces sensitivity of steroid receptor.

2. Modification of triggering factors based on their age group, clinical pattern & social background

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser (refer question 10)

4. Anti-itch strategies (refer question 7)

5. Anti-inflammatory – topical & systemic agents (refer question 9)

6. Restoration of skin microbiota and anti-infective therapy

Although antibiotic treatment usually reduces or totally eliminates *S. aureus* colonization, clinical improvement is not sustained, and recolonization usually recurs within 4 to 8 weeks. (Gilani *et al*, 2005)

Hence, antibiotic treatment, whether systemic (Cloxacillin, 1st & 2nd generation cephalosporine etc) or topical (Fusidic acid, Mupirocin, etc) should be reserved for cases in which explicit signs of infection are present.

Secondary infection should be suspected in patients with weeping, pustules, crusts, atopic eczema failing to respond to therapy, rapidly worsening of Atopic Eczema, fever.

Topical combination creams with steroids and antibiotics are widely used for Atopic Eczema flare-ups, but their superiority over anti-inflammatory treatment alone is not well established.

A diluted bleach bath treatment (0.005% Sodium hypochlorite; 2 times/week; 10 minutes) is recommended for Atopic Eczema patients with recurrent bacterial infection & moderate to severe Atopic Eczema.

CASE 3

Atopic Eczema with Molluscum contagiosum

Fig3.1



Fig3.2



Fig3.3



Fig3.4

Fig3.1 / Fig3.2 : Scattered areas of erythema, scaly patches over his trunk and upper limbs especially over the neck flexure.

Fig3.3 / Fig3.4 : Scattered areas of erythema, scaly patches over his lower limbs especially the lateral aspects of both his shins.

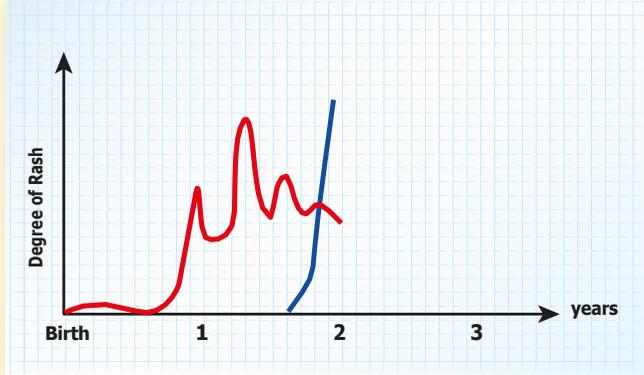
History

3 year-old boy has chronic relapsing itchy rash over his face, body and extremities since 6 month-old.

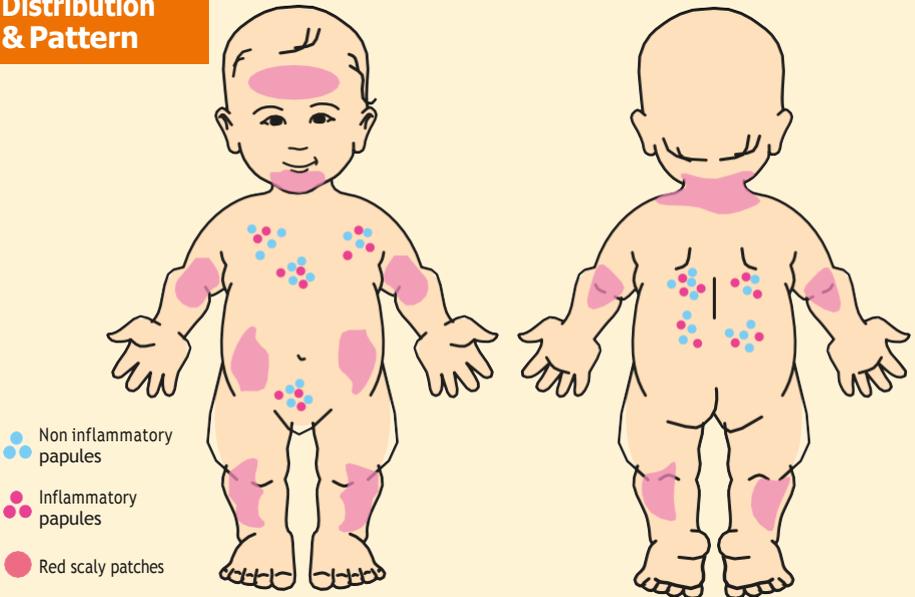
About 6 months ago, multiple new skin lesions with different appearance have erupted over his body followed by his extremities.

Clinical Course

-  Chronic relapsing rash for 18 months
-  New rash for 6 months



Distribution & Pattern



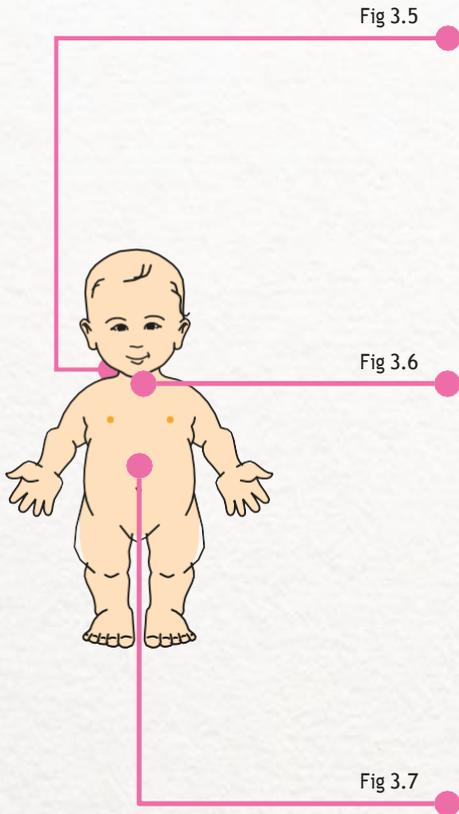


Fig 3.5 / Fig 3.6 / Fig 3.7 :
A few well defined inflamed papules surrounded by ill defined, Red, scaly patches over his neck & abdomen. Some of the papules are excoriated.

Clinical Diagnosis

ATOPIC ECZEMA

- Flexural & lateral shins pattern
- Complicated by extensive poxvirus infection

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment about the relationship between Atopic Eczema & Molluscum contagiosum

Molluscum contagiosum (MC) is a self-limiting pox virus infection that is very common in children.

It is widely accepted that molluscum contagiosum tends to be more intense & higher in number among Atopic Eczema patients.

Clinical challenges in the management are:

- a. Molluscum contagiosum infection can trigger an eczematous reaction around molluscum papules known as a hypersensitivity reaction and causes eczema symptoms to flare up.
- b. As molluscum contagiosum is usually spread by direct contact, itch-scratch cycle of Atopic Eczema increases the chance of spread within the individual (auto inoculation). Hence, treatment of molluscum contagiosum has to go hand in hand with anti itch and anti inflammatory medications i.e. topical steroids and calcineurine inhibitors.
- c. Most of the treatment modalities for molluscum contagiosum may induce physical and chemical irritation on the Molluscum contagiosum lesions. i.e. 5%-10% KOH, Benzyl peroxide, salicylic acid, cryotherapy, pulse dyed laser & curettage etc.

As a result, his eczema may get exacerbated by the treatment used.

Considerable debate exists about the management of Molluscum contagiosum and, according to a recent Cochrane review, no single intervention has been shown to be convincingly effective in clearing this disease.

2. Modification of triggering factors based on their age group, clinical pattern & social background

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser

4. Anti-itch strategies

As Molluscum contagiosum virus is highly contagious and can be transmitted by direct inoculation, modification of the itch-scratch cycle among Atopic Eczema patients is essential.

5. Anti-inflammatory – topical & systemic agents

Topical steroids or immunomodulators may potentiate the spread of the primary Molluscum contagiosum infection.

However, in symptomatic patients, treatment should not be withheld & short-course treatments of topical corticosteroids may be used.

6. Restoration of skin microbiota and anti-infective therapy

Generally, secondary bacterial infections caused by scratching is one the most common complications and requires antibiotics.

CASE 4

Adolescent Atopic Eczema with lichenification



Fig4.1 / Fig4.2 : Generalized erythema, scaly patches over his trunk and upper limbs.

Fig4.3 : Eczema was noted over his upper limbs. It is more prominent over the his wrists and elbows symmetrically.

Fig4.4 / Fig4.5 : Lichenified eczema was noted over his lower limbs. It is more prominent over the his knees and ankles symmetrically.

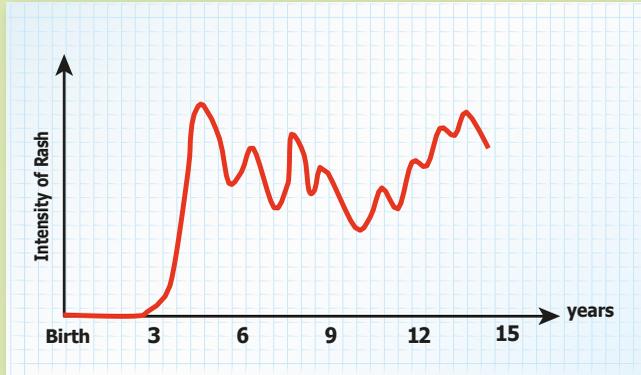
History

15 year-old boy with chronic relapsing and pruritic rash over his trunk and extremities since 2 year-old.

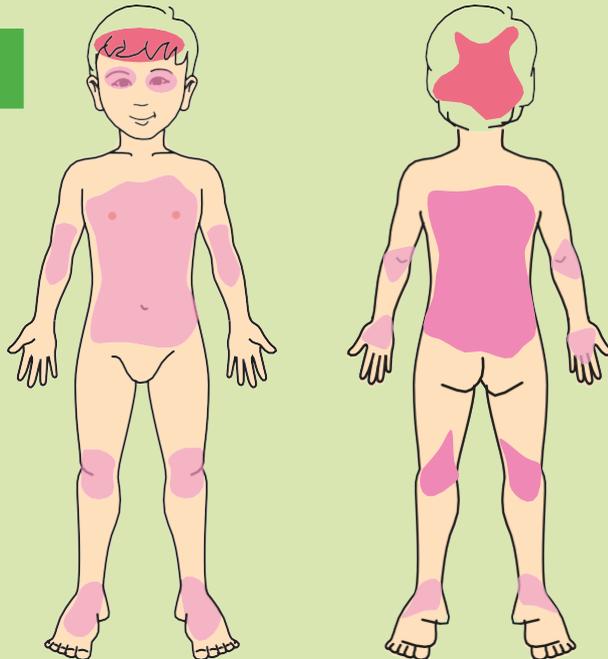
The rashes have extended to his scalp, forehead, around his eyes & hands since 2 years ago & getting less responsive to his routine home medications.

Clinical Course

— Chronic relapsing rash for 12 years



Distribution & Pattern



Morphology & Progression



Fig 4.8 : Mild redness, scaly palms and soles with hyperlinearity noted.



Fig 4.9 : Red, thick, scaly plaques with excoriations were noted over his knee & popliteal fossa.



Fig 4.10 : Dry, mild redness and scaly skin is noted over his shins.

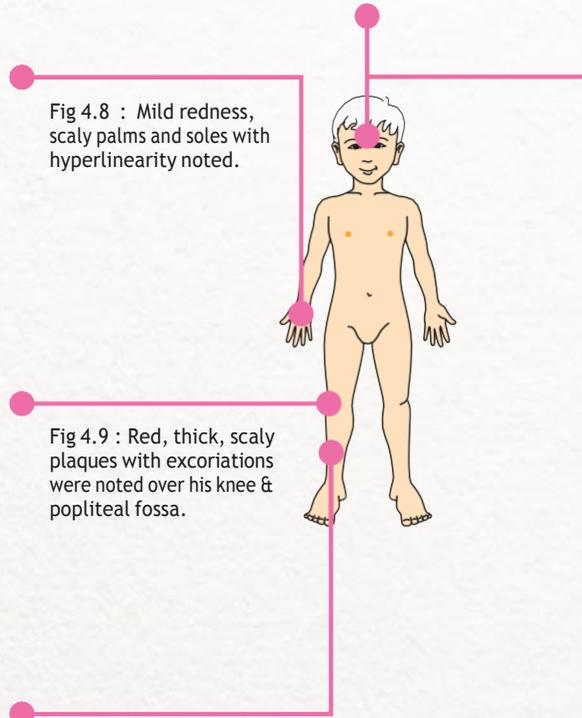




Fig 4.6 / Fig 4.7 : Ill defined red scaly patches & plaques were noted over his forehead, cheeks & eyelids.



Fig 4.11 : Ill defined red scaly patches & plaques were noted over his elbows and wrists.

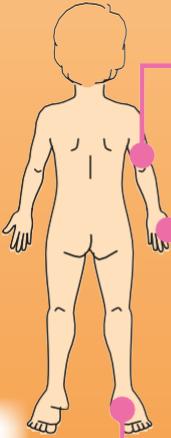


Fig 4.12 : Ill defined red scaly patches & plaques were noted over his elbows and wrists.



Fig 4.13 : Mild redness, scaly palms and soles with hyperlinearity noted.



Clinical Diagnosis

ATOPIC ECZEMA

- Chronic relapsing & persisted into adolescence
- Chronic Lichenification
- Generalized especially over the scalp, face, flexures and hands
- Ichthyosis Vulgaris & Palmoplantar hyperlinearity
- Low self esteem and poor sleep quality

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment

The goal of eczema treatment in teens is to allow him or her to maintain their usual pursuits with minimal disruption: good sleep, concentration in school, normal social activities and future career.

Most will get upset when they (~ 30%) do not outgrow their childhood eczema and carry it into their adolescent years. Adolescents may feel embarrassed when the appearance of their inflamed skin sets them apart from friends, leading to social isolation and lack of self confidence.

In addition, the lack of sleep and psychological pressure many teens experience as they face academic challenges can worsen Atopic Eczema.

We need to empower them (not the parents) about daily skin care and eczema action plan based on their lifestyle, from what time they get up in the morning and go to sleep at night to academic, after school and social activities.

2. Modification of triggering factors based on their age group, clinical pattern & social background

For adolescents with Atopic Eczema, two additional clinical patterns are identified and are potentially modifiable.

- a. Hand dermatitis is more common among adolescents with Atopic Eczema. This is due to frequent contact with harsh cleansers and other contact allergens (Nickel, perfumes etc) in their daily activities. Patch test may be helpful for identification of contact allergens in this subgroup.
- b. Head and Neck Atopic Dermatitis (HNAD) is a subgroup of postpubertal Atopic Eczema. *Malassezia*, the predominant skin microbiota fungus, is considered to be an important player in HNAD. (Baker, 2006; Faergemann, 2002). It is supported by
 - clinical improvements in Atopic Eczema symptoms, particularly Atopic Eczema of the head and neck with systemic anti fungal agents i.e. Itraconazole.
 - Patients with HNAD are more likely than healthy controls to have specific IgE antibodies against *Malassezia spp.*

And the serum anti-*Malassezia* IgE level is correlated well with the disease severity.

Management & Clinical Pearls

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser that is selected by the patient (not the parents) during counseling.

4. Anti-itch strategies

Wet wrap and behavioral modifications are highlighted in the anti itch regimen for this boy.

5. Anti-inflammatory – topical & systemic agents

- Class 3 & short duration of Class 4 Topical corticosteroid is needed for his lichenified eczema
- Proactive anti inflammatory creams for the hotspots
i.e. topical Corticosteroid +/- topical Calcineurine inhibitors 2-3 times/week for 16-20 weeks is effective to prevent early relapse

6. Restoration of skin pH & skin microbiota

Skin barrier repair creams

Antimicrobial during clinical infection

Antiseptic bath - bleach bath regimen is 2-3 times/week for 10 minutes at 0.005% concentration



CASE 5

Atopic Eczema with toilet seat eczema & auto eczematization



Fig 5.1 / Fig 5.2 : Acute eczematous rash noted over both his antecubital and abdominal folds. Sudden onset of new papulovesicles noted over his back.

Fig 5.3 / Fig 5.4 : Acute eczematous rash noted over his posterior thighs, lateral aspects of buttocks and lower back.

Fig 5.5 : Acute eczematous rash is more prominent over the abdominal folds.

Fig 5.6 : Michelin sign +ve as demonstrated.

History

12 year-old boy with chronic relapsing flexural atopic eczema for 4 years.

He complained of appearance of new rashes for past 3 months.

About his new rash: His eczema usually involves the flexures of the limbs but lately he developed new rashes over the back of both thighs symmetrically.

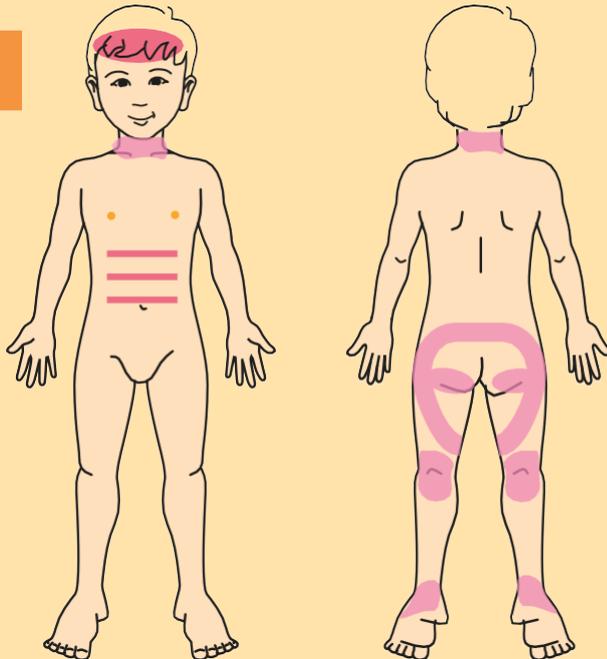
These rashes were very itching and aggravated after using the seating toilet
Maternal history of allergy rhinitis is documented.

Clinical Course

- Chronic rash for 4 years
- Acute on chronic rash for 4 months



Distribution & Pattern



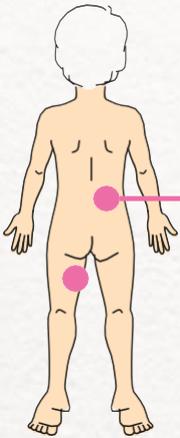


Fig 5.9 : Multiple crusted papules and vesicles are noted over his back.



Fig 5.10 : Well defined, red scaly patches are noted over his posterior thighs with multiple excoriations.



Clinical Diagnosis

Atopic Eczema

- Flexural pattern
- Toilet seat eczema with auto eczematization

Differential diagnosis :
Atopic Eczema with secondary Tinea infection
Atopic Eczema with allergic contact dermatitis to topical creams

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment (refer question 7)
2. Modification of triggering factors based on their age group, clinical pattern & social background

Recently, there has been an increased use of **harsh toilet seat detergents**, containing ingredients such as didecyl dimethyl ammonium chloride and alkyl dimethyl benzyl ammonium chloride, which have previously been documented to cause severe skin irritation. Avoiding direct skin contact with the toilet seat (Fig 5.11) is important to prevent further relapse beside the general recommendations that are described in question 8.

3. Skin dryness & restoration of skin barrier (refer question 10)
4. Anti-itch strategies (refer question 7)
5. Anti-inflammatory – topical & systemic (refer question 9)

Since auto eczematization is a systemic reaction, systemic steroids for 1-2 weeks are helpful beside treating the toilet seat eczema (herald eczema patch) with topical steroid till clinical resolution.

6. Antimicrobial and restoration or skin microbiota



Fig 5.11 : Avoid direct skin contact with the toilet seat.

CASE 6

Atopic Eczema with secondary tinea infection

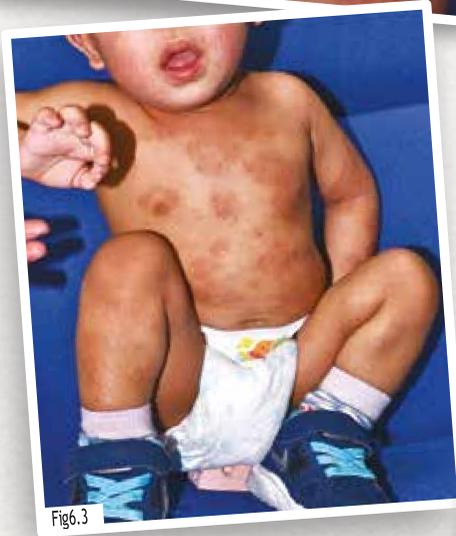


Fig6.1 : Acute & subacute eczema. Ill defined red scaly patches noted over his forehead and cheeks.

Fig6.2 : Ill defined red scaly patches noted over the lateral aspects of his both lower limbs.

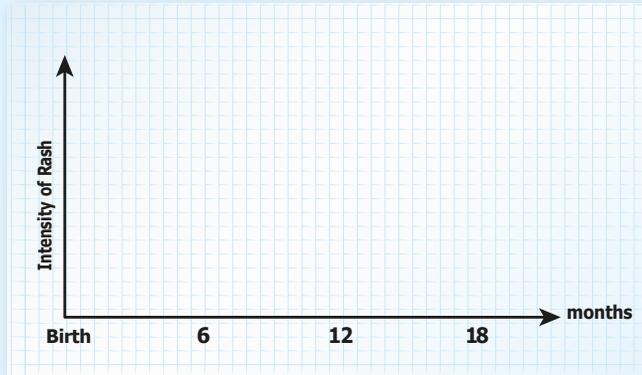
Fig6.3 / Fig6.4 : Well defined red scaly patches noted over trunk (back > front) & limbs.

History

14 month-old baby boy was presented with itchy facial rash for 6 months before it extended down to his body and limbs since 6 weeks ago. His chronic rash was under control initially with combination therapy of topical steroid and emollient but failed to do so for the last 6 weeks.

Clinical Course

- Chronic relapsing rash for 6 months
- Acute new rash for 6 weeks



Distribution & Pattern

Morphology & Progression

Fig 6.5

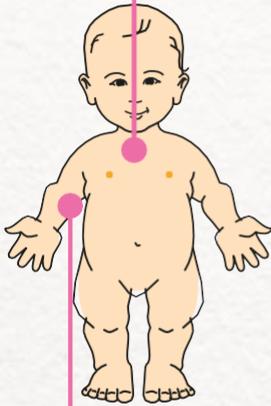


Fig 6.6



Fig 6.5 / Fig 6.6 :

Facial rash :

Ill defined red scaly patches noted

Truncal rash :

Well defined red scaly rash with expanding margin.

Clinical Diagnosis

INFANTILE ATOPIC ECZEMA

1. In acute to subacute
2. Tinea incognito

Subacute & chronic stage of Atopic Eczema often manifests as ill defined, pruritic, red scaly patches or plaque. In contrast to Atopic Eczema, Tinea corporis has an active, well defined advancing margin. Tinea corporis usually begins as a pruritic, well defined, circular or oval, erythematous, scaling patch or plaque that spreads centrifugally with central clearing. When Atopic Eczema is complicated by dermatophytes infection and steroid cream, it results as tinea incognito.

Tinea incognito is a dermatophytic infection which has lost its typical clinical appearance because of improper use of steroids or calcineurin inhibitors.

The steroid cream dampens down inflammation (redness, scaly surface & itch) so the condition feels less irritable. But when the cream is stopped for a few days the itch gets worse & the more steroid is applied, the more extensive the fungal infection becomes.

Tinea infection among Atopic Eczema patients is suspected when the margin of dermatitis becomes well defined and expanding slowly over weeks despite routine treatment with steroid and emollient.

The diagnosis of tinea is made by taking skin scrapings for microscopy and culture.

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment about Atopic Eczema (*refer case 1*)
2. Modification of triggering factors based on their age group, clinical pattern & social background
3. Skin dryness & restoration of skin barrier (*refer question 10*)
4. Anti-itch strategies (*refer question 7*)
5. Anti-inflammatory – topical & systemic (*refer question 9*)
6. Antimicrobial and restoration or skin microbiota

Tinea is usually treated with topical anti fungal agents, but if the treatment is unsuccessful or extensive as in this patient, systemic anti fungal agents, including terbinafine, itraconazole or Griseofulvin are preferred.

CASE 7

Atopic Eczema with prurigo nodularis & allergic rhinoconjunctivitis



Fig7.1 / Fig7.2 : 1. Multiple excoriations & nodules are noted over his limbs more than his body.
2. Ill defined red scaly patches over his neck.

Fig7.3 / Fig7.4 : Excoriations & nodules are noted symmetrically over his lower limbs.



Fig7.5 / Fig7.6 / Fig7.7 / Fig7.8 : The patterns & local arrangement is consistent with scratching pattern as the excoriated papules & nodules are more over the lateral > medial aspects & arranged in linear pattern.

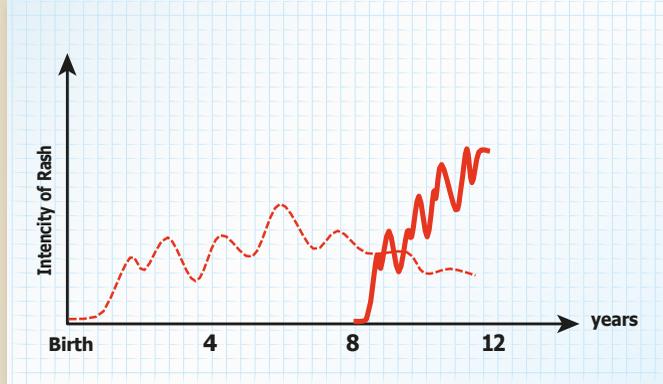
History

11 year old boy presented with chronic itchy relapsing rash since infancy.

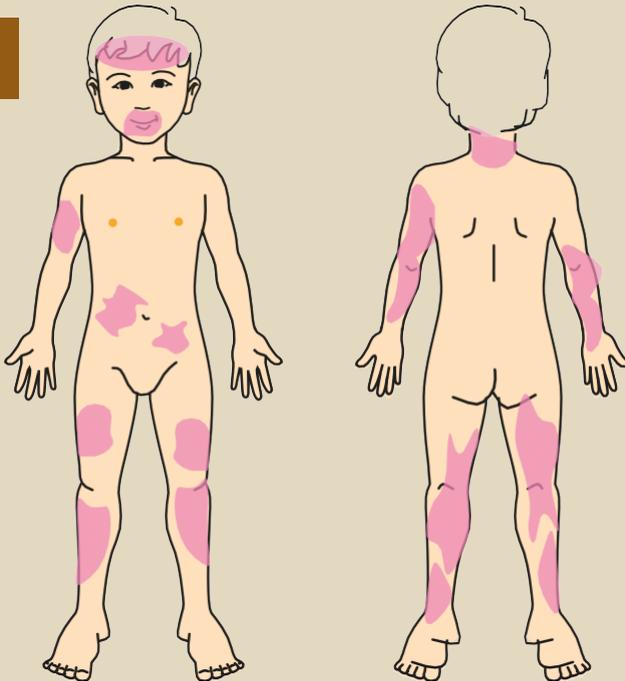
His rash has worsened & is resistant to routine treatment regimen since 2 years ago. Besides that, a few new rash patterns have appeared.

Clinical Course

- Chronic relapsing rash for 11 years
- Onset of new rash around his eyes / nasal philtrum for 2 years



Distribution & Pattern



Morphology & Progression



Fig 7.10 / Fig 7.11 / Fig 7.12 :
Forehead :
Red scaly and lichenified plaques are noted.
Periorbital & nasal philtrum :
Ill defined red scaly patches noted over this areas.

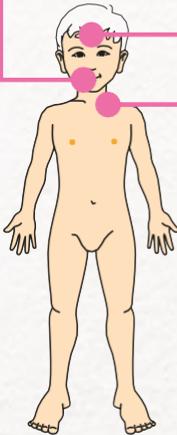


Fig 7.13 :
Ill defined red scaly patches with hyperpigmentation along the skin creases are noted over his neck (dirty neck appearance)



Morphology & Progression

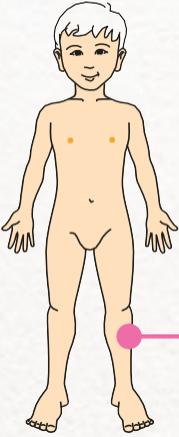


Fig 7.14 / Fig 7.15 :
Well defined excoriated papules
/ nodules arranged in linear
pattern .



Clinical Diagnosis

ATOPIC ECZEMA

Morphology : Lichenification & Prurigo nodularis Patterns are

1. Forehead, flexural
2. Scratching pattern
Extremities > body,
Extremities: lateral > medial aspects & linear arrangement
3. Allergic rhinoconjunctivitis with bilateral periorbital and nasal philtrum

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment (refer question 7)

2. Modification of triggering factors based on their age group, clinical pattern & social background

A few specific & modifiable clinical patterns are identified in this case

- Allergic rhinoconjunctivitis with periorbital and nasal philtrum areas
- Scratching pattern: lateral aspects of upper and lower limbs in linear pattern
- Forehead, neck and other flexures are related mainly due to occlusive folds and sweating

3. Skin dryness & restoration of skin barrier

Please refer *question 10* for selection of ideal moisturiser for this child

4. Anti-itch strategies (refer table 7.3)

Empower this boy with the anti-itch measures listed in *table 7* which is mandatory to arrest the formation of new pruritic nodules

5. Anti-inflammatory – topical & systemic (refer question 9)

Superpotent topical corticosteroid for 1-2 weeks followed by potent topical corticosteroid/ topical calcineurin inhibitor till clinical remission. Occlusive wet wrap therapy or intralesional triamcinolone are indicated for recalcitrant lesions

6. Antimicrobial and restoration or skin microbiota

CASE 8

Atopic Eczema - follicular variant

Fig8.1



Fig8.2



Fig8.3



Fig8.4

Fig8.1 / Fig8.2 : Hyperpigmented scaly papules& plaques are noted over his neck, lower back & posterior aspect of elbows symmetrically.

Fig8.3 / Fig8.4 : Hyperpigmented scaly papules & plaques are noted over posterolateral aspect of his both lower limbs. Sparing his knee flexures, palms & soles

History

6 year-old Indian boy with chronic relapsing itchy rash over his limbs more than his body and face since 12 month-old

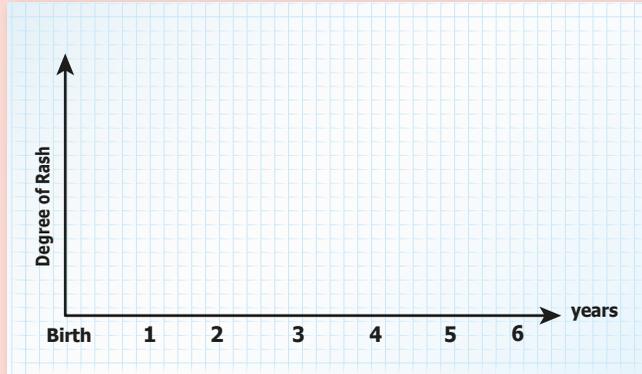
His itch has disturbed his sleeping quality.

Positive family history of atopic diseases is present.

Clinical Course

— Chronic relapsing rash for 18 month

Degree of Scratching



Distribution & Pattern

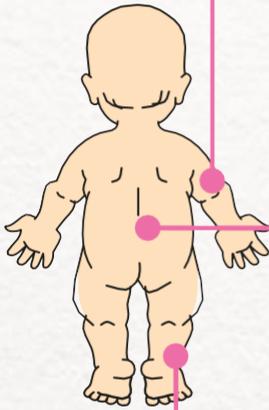


Fig 8.5



Fig 8.6

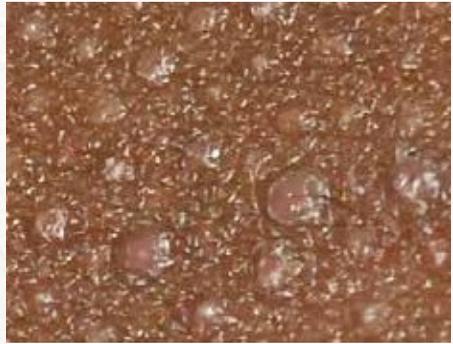


Fig 8.7



Fig 8.5 / Fig 8.6 / Fig 8.7 : Hyperpigmented scaly follicular papules are noted over his elbows and some of these papules have coalesced to become plaque over the shins.

Clinical Diagnosis

ATOPIC ECZEMA

Follicular pattern among the darker skin type

Vicious itch scratch cycle

Differential diagnosis are keratosis pilaris, lichen nitidus, Pityriasis rubra pilaris, id reactions & Gianotti Crosti syndrome

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment (refer question 7)

2. Modification of triggering factors based on their age group, clinical pattern & social background

Among the Atopic Eczema children with darker skin type, follicular pattern eczema that is exacerbated by harsh cleansers, dry climate & scratching is not uncommon. To avoid flare-ups, refrain from scratching affected areas, as this tends to exacerbate the condition. Also try to wear clothing made of natural fibers, as this may irritate skin less. Keep skin moisturized.

3. Skin dryness & restoration of skin barrier

Please refer to *question 10* for selection of an ideal moisturiser. In this case, there is accumulation of dead skin cells at the follicular openings. Hence, a combination of mild keratolytic agents (5-10% urea cream) & intensive emollient may be helpful for non erythematous scaly follicular papules.

4. Anti-itch strategies (refer table 7.3)

Empower this boy with the anti-itch measures listed in *table 7* to prevent the disease progression from scaly papules → inflamed papules → inflamed plaques with 

5. Anti-inflammatory – topical & systemic (refer question 9)

Potent topical corticosteroid for 1-2 weeks followed by proactive strategy (3 times/week) with potent topical corticosteroid/topical calcineurin inhibitor until full clinical remission. Wet wrap therapy is indicated for localized recalcitrant sites to break the itch-scratch cycle.

6. Antimicrobial and restoration or skin microbiota

CASE 9

Generalized exfoliative eczema

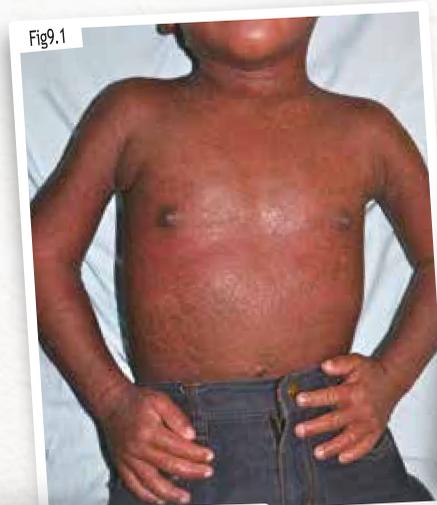


Fig9.1 / Fig9.2 / Fig9.3 / Fig9.4 : Generalized erythematous, scaly patches and lichenified plaques that coalesce together over his face, scalp, trunks and limbs.

Fig9.5 / Fig9.6 : Generalized exfoliative scaly rash with minimal involvement of his thick palmoplantar surfaces.

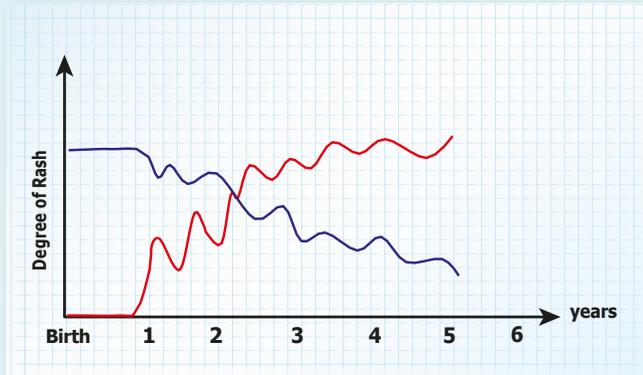
History

5.5 year-old boy with chronic persistent pruritic rash since 6 month-old.

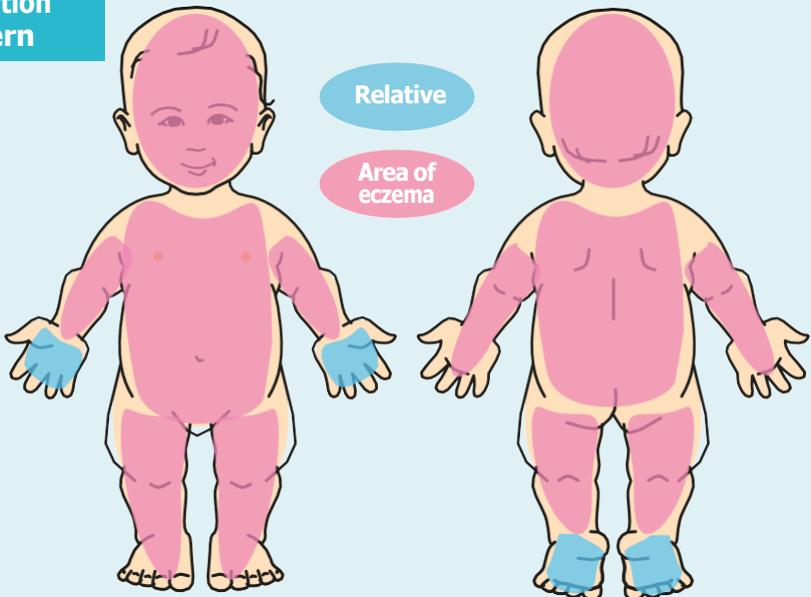
His sleep is greatly disturbed by the itchy rash.
He is always absent from school and has low self esteem.

Clinical Course

- Chronic relapsing rash for 4 years
- Sleeping quality



Distribution & Pattern



Morphology & Progression

Fig 9.7 :
Ill defined, red, scaly
patches noted over his face



Fig 9.8 :
Ill defined, red, scaly patches &
lichenified plaques that coalesce
together to form
generalized exfoliative eczema.
Secondary
erosions and
fissures are noted.



Fig 9.9 :
Ill defined, red, scaly patches &
lichenified plaques that coalesce
together to form generalized
exfoliative eczema.
Secondary erosions and
fissures are noted.



Clinical Diagnosis

Generalized exfoliative Atopic Eczema

- Acute on chronic lichenification
- Severe social & emotional disturbance
- Protein losing dermopathy & mild anemia
- Recurrent bacterial infection

Differential diagnosis for infantile and childhood erythroderma

- a. Atopic Eczema
- b. Psoriasis
- c. Autosomal recessive congenital ichthyosis
- d. Severe adverse drug reaction
- e. Primary immune deficiency or metabolic diseases

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

The concept of generalized exfoliative eczema and decompensated skin function (skin failure) is new to non-dermatologist clinicians and the need for intensive care in dermatology is overlooked. Skin failure is a dermatological urgency and is no less serious than visceral dysfunctions like cardiorespiratory, renal or liver failure.

Skin failure has been defined as the loss of thermoregulatory function, and failure to prevent percutaneous loss of fluid, electrolytes and protein, with resulting imbalance, and renders itself much more vulnerable to infection & spread of *Staphylococcus aureus*.

The principal nutrients lost in skin failure are protein and iron.

The main causes of hypoproteinemia are continuous loss through shed scales, increased BMR and dermatogenic enteropathy leading to protein loss. In exfoliative dermatitis, diffuse scaling leads to protein loss of approximately 20-30 g/m² BSA/day. In the presence of exudative skin lesions, the combined protein loss through oozing from the skin surface is even higher. His hypoalbuminemia would be exacerbated further by unwarranted food restrictions due to misconceptions about the role of food allergens in Atopic Eczema.

CASE 10

Atopic Eczema & Shoe contact dermatitis



Fig10.1 / Fig10.2 : Erythema, scaly patches are seen over the extensors and flexors of his elbows

Fig10.3 / Fig10.4 : Erythema, scaly patches & plaques are noted over the extensors and flexors of his knees & lateral aspects of his shins & dorsal feet

Fig10.5 : Lichenified plaques are found over the dorsal aspects of his first three toes symmetrically

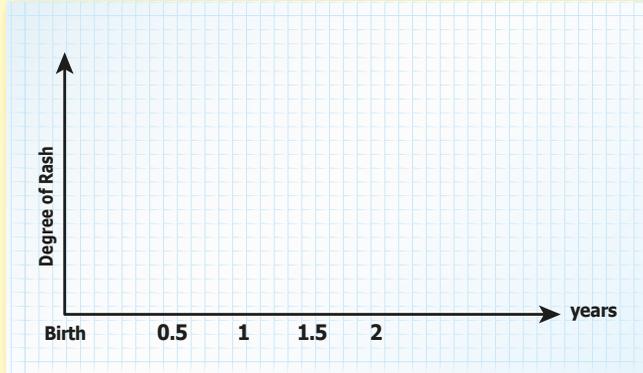
History

4 year-old girl has chronic relapsing itchy rash that started over her face and extensor surfaces of her extremities since 6 month-old and is showing some degree of improvement until 6 months ago with new rashes over her feet. Positive family history of atopic diseases among her first degree relatives

Clinical Course

— Chronic relapsing rash for 3 years

New rash for 6 months over her feet



Distribution & Pattern

Morphology & Progression



Fig10.7 :
Coin-shaped, well defined scaly plaques with excoriation are noted over the knees



Fig10.8 :
Coin-shaped, well defined scaly plaques with excoriation are noted over the knees

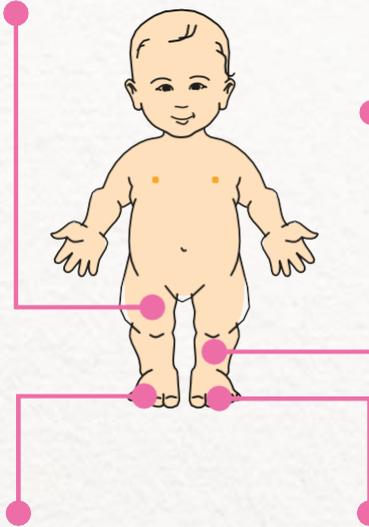


Fig10.9 :
Hyperpigmented lichenified scaly plaques with fissures are noted over the areas of contact with her shoes

Fig10.10 :
Hyperpigmented lichenified scaly plaques with fissures are noted over the areas of contact with her shoes



Clinical Diagnosis

ATOPIC ECZEMA

Flexural & Extensor pattern

Discoid shaped eczema

Shoe related contact allergens and irritants

Shoe dermatitis is usually begins on the top surface of the big toe and spreads to the upper surfaces of the foot bilaterally. Dermatitis may also be found on the sole of the foot, the side of the feet & heels.

Juvenile plantar dermatosis

Presents as a red, scaly lesion that affects the weight-bearing regions of the sole of the foot. The lesions are a symmetrical eczematous rash that can be found on the bottom of the foot. One unique finding in JPD is that the toe webs & arch of the feet are spared. To consider Tinea Pedis if the pattern is asymmetry with webspaces and nails involvement.

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment

2. Modification of triggering factors based on their age group, clinical pattern & social background

Shoe dermatitis is a form of contact dermatitis resulting from exposure to shoes and/or sandals. Different chemicals, in conjunction with a hot and humid environment within the shoe, give rise to allergic and/or irritant dermatitis. Allergic shoe dermatitis is commonly caused by constituents of rubber, rubber accelerators, glues, and rarely by linings and dyes. Chromium that is gradually liberated from leather collagen by the action of hydroxyl acids in sweat especially when shoes are used without stockings may act as an allergen too.

Some steps you can take to reduce shoe contact dermatitis reactions include:

- Controlling foot perspiration using antiperspirants
- Wear stockings to reduce friction between the skin surface and the shoes
- Avoiding all footwear that contains allergens detected by Patch test

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser

4. Anti-itch strategies listed in *figure 7.3*

5. Anti-inflammatory

Potent topical corticosteroid is needed for her chronic lichenified eczema, followed by moderate potent corticosteroid (2- 3 times/week) until clinical remission

6. Restoration of skin microbiota and anti infective therapy with topical antibiotic

CASE 11

Atopic Eczema with secondary bacteria & Herpes simplex infection



Fig11.1 / Fig11.2 : Generalized dry, red scaly patches are found over his trunk and upper limbs.

Multiple vesicles & erosions are noted mainly over the extremities, upper chest and abdomen

Fig11.3 / Fig11.4 : Dry, red and ill defined scaly patches are found over both his lower limbs together with multiple vesicles & erosions

History

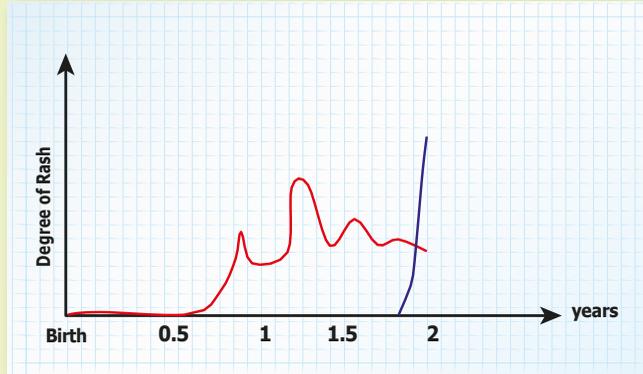
2 year-old boy with chronic relapsing itchy rash over his face, body and extremities since 6 month-old.

There is a sudden eruption of new blisters for 5 days together with fever and malaise. His current flare up was not responsive to his routine anti inflammatory and antibiotic treatment.

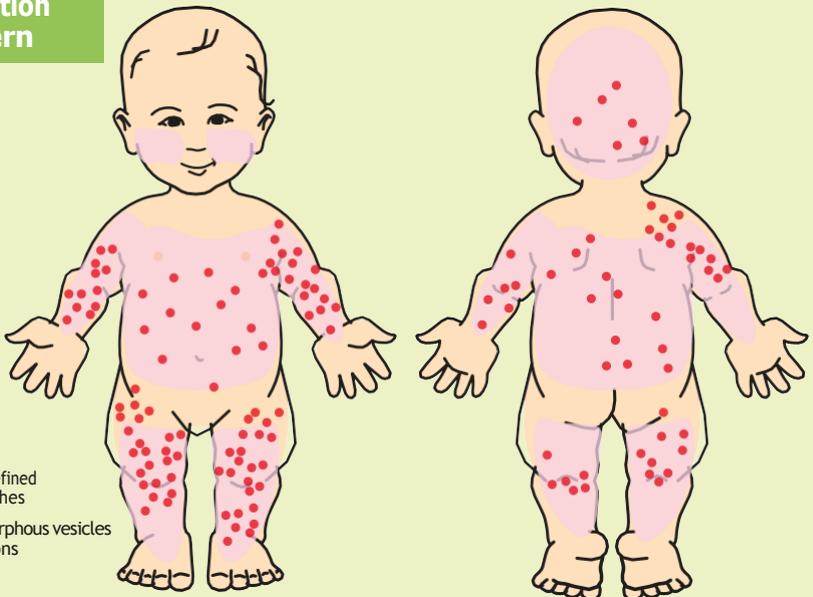
Positive family history of atopic diseases among his first degree relatives is noted.

Clinical Course

- Chronic relapsing rash for 18 month
- New rash for 5 days with fever



Distribution & Pattern



- Dry, ill defined red patches
- Monomorphous vesicles & erosions

Fig11.5 :
Multiple well defined erosions
with an erythematous scaly
background are noted over
his abdomen. Some of these
erosions have coalesced.

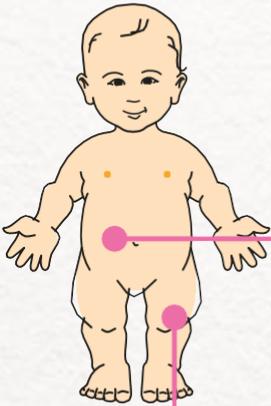


Fig11.6 :
Multiple well defined erosions
with an erythematous scaly
background are noted over
his lower extremities.



Clinical Diagnosis

ATOPIC ECZEMA

Generalized

Complicated by secondary infection

- Extensive herpes simplex virus
- Staph. aureus

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. **Education & Empowerment**
2. **Modification of triggering factors based on their age group, clinical pattern & social background**
3. **Restoration of skin barrier hydration with moisturizer & gentle cleanser**
4. **Anti-itch strategies as listed in *figure 7.3***
5. **Anti-inflammatory agents**

There is a concern that application of topical corticosteroids to the skin that is affected by eczema herpeticum may facilitate dissemination of HSV and worsen the disease. Wollenberg et al has reported that majority of EH occurs in patients with untreated Atopic Dermatitis, arguing against a role for topical corticosteroids in the development of EH. (Wollenberg et al, 2003)

If clinical diagnosis of EH is certain and patient is under close observation, combination of topical corticosteroid and systemic acyclovir is safe.

6. **Restoration of skin microbiota and anti microbial therapy**

Eczema herpeticum is considered as a dermatological emergency (medical urgency). Eczema herpeticum usually arises during a first episode of Primary herpes simplex infection.

Blisters can occur in normal skin or in sites actively or previously affected by Atopic Eczema.

Eczema Herpeticum is suspected based on:

1. Atopic Eczema failing to respond to their routine therapy i.e. anti inflammatory agents and 48-72 hours of appropriate antibiotic.
2. Sudden onset of clustered blisters that rupture into punched-out erosions. These erosions are usually 1-3 mm that are monomorphic in appearance i.e. they all appear uniform & similar to each other (Polymorphic in chickenpox infection). They may coalesce to form larger areas of erosion with crusting
3. Systemic symptoms like fever, lethargy and poor oral intake are common.

Tzanck smear and Direct fluorescent antibody stain are helpful.

In terms of treatment, systemic Acyclovir or if available, valaciclovir for 10 to 14 days is required.

Intravenous aciclovir is indicated if the patient is too sick to take tablets, or if the infection is deteriorating despite treatment.

To consider combination of systemic anti viral & antibiotic among cases of eczema herpeticum as it is difficult to exclude secondary bacteria infection clinically.

Patients with recurrent eczema herpeticum should be offered prophylactic antiviral therapy.

CASE 12

Atopic Eczema with Ichthyosis Vulgaris



Fig12.1 / Fig12.2 : Extensive dry & scaly skin are found mainly over her trunk and extremities. Face is minimally affected.

Fig12.3 / Fig12.4 : Dry and scaly skin is found over her lower limbs especially the lateral aspect of both her shins.

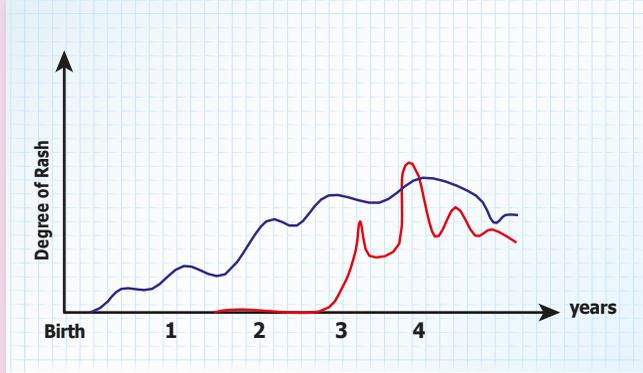
History

4 year-old girl with generalized dry & flaky skin especially over her lower limbs is noted since early infantile period.

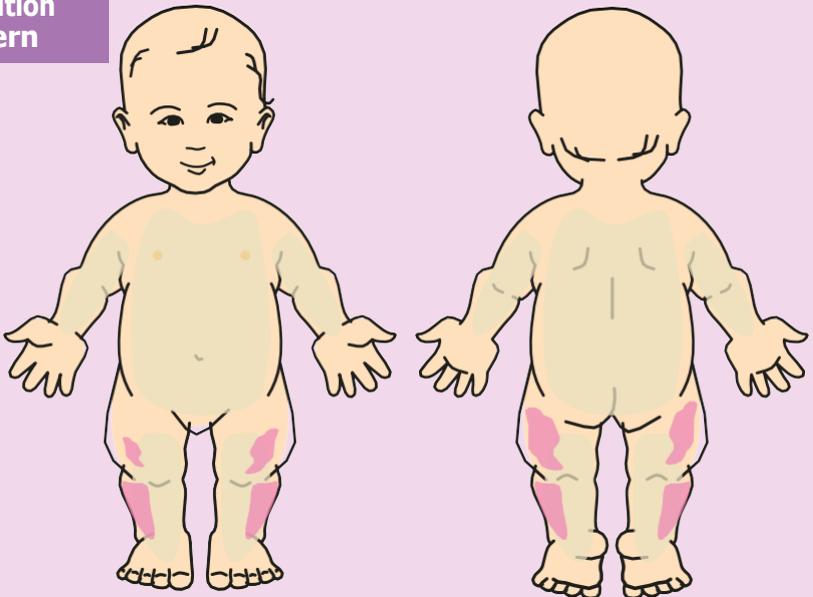
A year ago, she developed red itchy rashes over the lateral aspects of both her shins and occasionally get infected.

Clinical Course

- Chronic relapsing rash for 12 months
- dry skin since 3 month-old



Distribution & Pattern



- Dry skin
- Eczema

Fig12.5 :
Dry, scaly skin with peri
follicular accentuation is
found mainly over her
abdomen & lower
extremities.

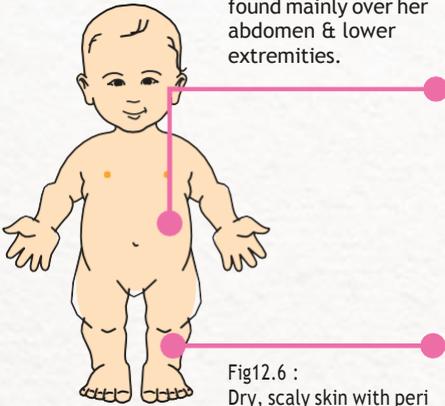


Fig12.6 :
Dry, scaly skin with peri
follicular accentuation is
found mainly over her
abdomen & lower
extremities.

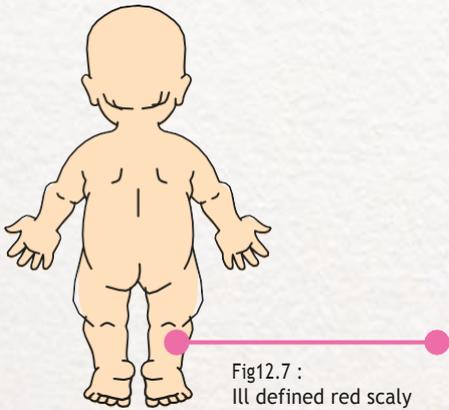


Fig12.7 :
Ill defined red scaly
patches with superficial
erosions are noted over
the lateral aspect of both
her shins.



Clinical Diagnosis

ATOPIC ECZEMA

Underlying skin dryness and ichthyosis vulgaris

Peri follicular accentuation

Filaggrin gene mutation is the cause of Ichthyosis Vulgaris.

It is the strongest known endogenous (genetic) risk factor for the development of Atopic Eczema

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

- 1. Education & Empowerment**
- 2. Modification of triggering factors based on their age group, clinical pattern & social background**

Skin dryness is one of the hallmarks of Atopic Eczema. Skin dryness in Atopic Eczema is due to dynamic interaction between endogenous & exogenous factors.

Endogenous factors include Filaggrin & ceramide deficiency in Stratum corneum.

In this case, she has Ichthyosis Vulgaris that is due to filaggrin gene mutation.

Exogenous factors are

- Air conditioning, central heating or sitting close to a fire or fan heater.
- Excessive prolonged bathing, especially in hot water.
- Contact with harsh soaps, detergents and solvents.

- 3. Restoration of skin barrier hydration with moisturizer & gentle cleanser**

Adequate moisturization with a hypoallergenic emollient that has both anti-itch and steroid sparing effects.

- 4. Anti-itch strategies as listed in *figure 7.3***
- 5. Anti-inflammatory agents**
- 6. Restoration of skin microbiota and anti microbial therapy**

CASE 13

Atopic Eczema “forehead, neck, flexural pattern” related to occlusion & sweat

Fig13.1



Fig13.2



Fig13.5



Fig13.6



Fig13.3



Fig13.4



Fig13.1 / Fig13.2 : Extensive dry, ill defined, red and scaly skin are found mainly over the neck, upper chest, abdominal folds, around the elbows and wrist joints, and his back.

Fig 13.3 / Fig13.4 : Michel sign positive as the eczematous rashes concentrate around the abdominal folds when the child leans forward.

Fig 13.5 / Fig13.6 : Erythematous scaly patches are noted over his popliteal fossa and dorsal feet.

History

9 year-old boy with chronic relapsing itchy rash over his neck, abdominal folds and his extremities since 3 year-old.

His rash always get itchy and worsens after any outdoor activities with profuse sweating. Hence, his social life is significantly restricted. His mother stopped him from taking seafood, eggs and many other self diagnosed food allergy.

Clinical Course

— Chronic relapsing rash for 6 years



Distribution & Pattern

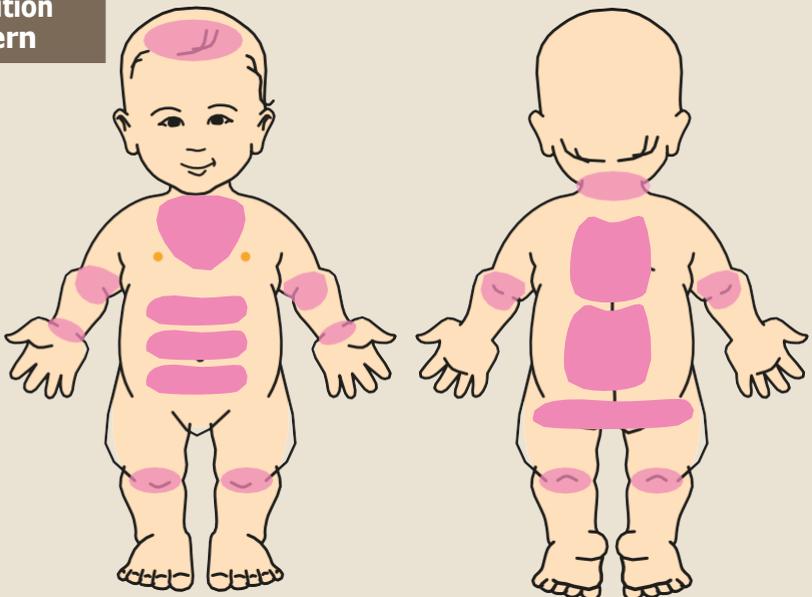


Fig13.7

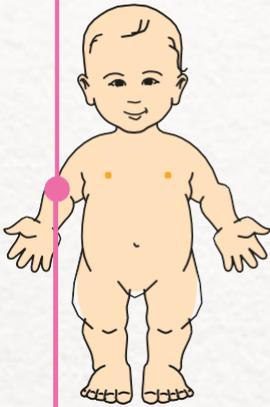


Fig13.8

Fig13.7 / Fig13.8 :
dry, erythematous, ill defined and
scaly patches are noted over his
antecubital fossa. A few crusted
erosions are seen.



Clinical Diagnosis

ATOPIC ECZEMA

Occlusion & Sweat induced pattern

(Forehead, neck, flexures)

Acute and subacute

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment
2. Modification of triggering factors based on their age group, clinical pattern & social background

Atopic Eczema patients often report that sweating leads to a prickly heat sensation & itch that results in eczema flare-ups.

Since sweating goes hand-in-hand with exercise especially in tropical countries, many Atopic Eczema patients dislike hot temperatures and high humidity as it brings on eczema.

Eczema that is exaggerated by sweat is more severe over the forehead, neck, around the joints and body folds.

This clinical pattern (Forehead, neck, body folds & lower back pattern) can be explained by:

- A. Sweat production rate varies with body sites. (Forehead > Neck > other body sites)
- B. Occlusion sites i.e. abdominal folds, neck, limb flexures etc have increased the duration of contact between the sweat that is trapped on the skin surfaces.
- C. Skin thickness is thinner over face & limbs flexures. Hence, it is more susceptible to external irritants like sweat & harsh cleanser.

You can't control the weather, but you can try to minimize the symptoms by reducing the duration of contact between the sweat and their skin.

- Use air conditioning (24-26 degree Celsius) in your home & bedroom when it is hot and humid. When sleeping, air conditioning is useful to avoid night sweats and eczema flares. On the other hand, avoid dry skin induced by cold and low humidity air produced by air conditioning.
- Ask for a seat in the classroom that is away from sunny windows.
- During an eczema flare, avoid strenuous exercise outside during hot, humid weather. Take short showers to rinse off sweat after exercising or playing outside.
- Wear breathable natural-fiber clothing that is loose fitting. Tight fitting clothing can irritate eczema by rubbing on the affected area. Cotton is a good option.

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser
4. Anti-itch strategies as listed in *figure 7.3*
5. Anti-inflammatory agents

Proactive strategy with topical corticosteroid or calcineurin inhibitor is preferred & effective to prevent frequent relapses over the hotspots i.e. forehead & flexures

6. Restoration of skin microbiota and anti microbial therapy

CASE 14

Atopic Eczema with Malignant misconceptions

Fig14.1



Fig14.2



Fig14.3



Fig14.4



Fig14.1 / Fig14.2 : Extensive dry, erythematous patches and plaques are noted over her trunk.

Fig14.3 / Fig14.4 : Extensive dry, ill defined red scaly patches are noted over her extremities.

History

A 18 month-old girl has developed generalized itchy rash with disturbed sleep pattern.

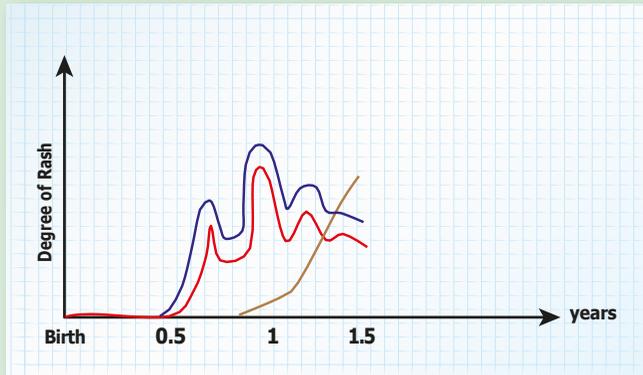
At 6 month-old, a red itchy rash started over her face, spreading towards body and limbs rapidly over the next 12 months.

She is on unwarranted food restriction based on her parent's own observation and direct interpretation of the levels of allergen specific serum IgE level.

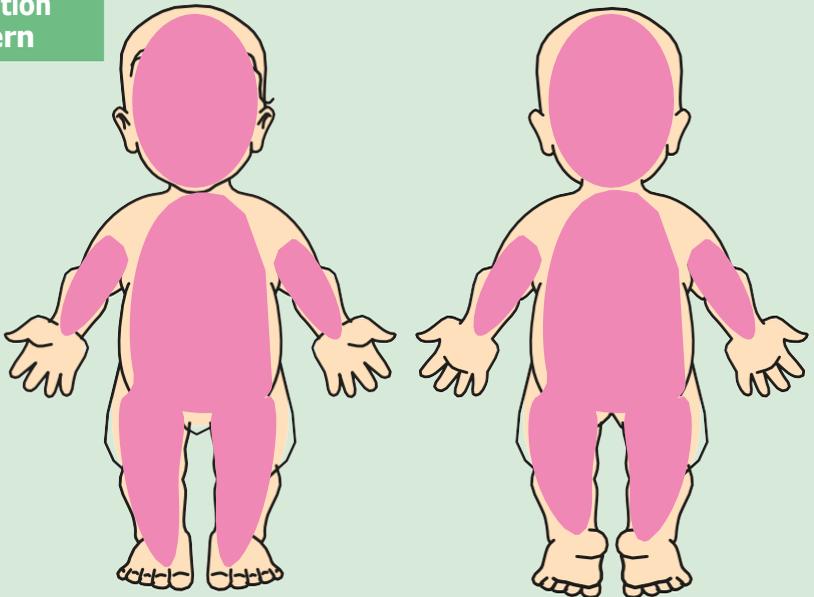
Positive family history of atopic dermatitis among her first degree sibling.

Clinical Course

- Chronic relapsing rash for 18 month
- Degree of Itch
- Malnutrition



Distribution & Pattern



Morphology & Progression

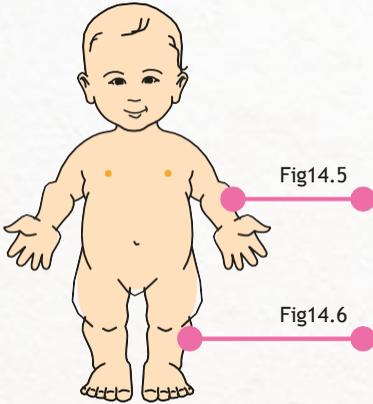


Fig14.5 / Fig14.6 :

Extensive dry, ill defined red scaly patches & plaques are noted over her extremities. Excoriations due to itch scratch cycle are noted too.

MICROBIOLOGY (IMMUNOLOGY) ALLERGY SPECIFIC TEST - ENZYME IMMUNOASSAY

SPECIMEN: Serum

| | |
|------------------|---------------|
| D. pteronyssinus | > 100.00 kU/L |
| D. farinae | > 100.00 kU/L |
| D. microceras | > 100.00 kU/L |
| Egg white | 2.39 kU/L |
| Milk | 8.58 kU/L |
| Fish (cod) | 0.84 kU/L |
| Wheat | 1.11 kU/L |
| Crab | 0.38 kU/L |
| Shrimp | 0.88 kU/L |
| Pacific Squid | 0.41 kU/L |
| Chicken Meat | 0.58 kU/L |
| Peanut | 0.52 kU/L |
| Soya Bean | 1.77 kU/L |

Associated systemic findings

| Test | Result | Unit | Ref. Range | Alarm |
|----------------------|-----------|------------|----------------------|------------|
| UREA | 2.00 | mmol/L | 0.00 - 8.00 | |
| SODIUM | 143 | mmol/L | 132.00 - 145.00 | |
| POTASSIUM | 5.34 | mmol/L | 3.10 - 5.10 | High |
| CHLORIDE | 108 | mmol/L | 96.00 - 111.00 | |
| CREATININE | 27 | mmol/L | 0.00 - 88.00 | |
| TOTAL PROTEIN | 43 | g/L | 66.00 - 87.00 | Low |
| ALBUMIN | 20 | g/L | 30.00 - 54.00 | Low |
| ALKALINE PHOSPHATASE | 108 | U/L | 0.00 - 346.00 | |
| ALANINE TRANSAMINASE | 39 | U/L | 0.00 - 54.00 | |
| TOTAL BILIRUBIN | 3 | umol/L | 0.00 - 17.00 | |

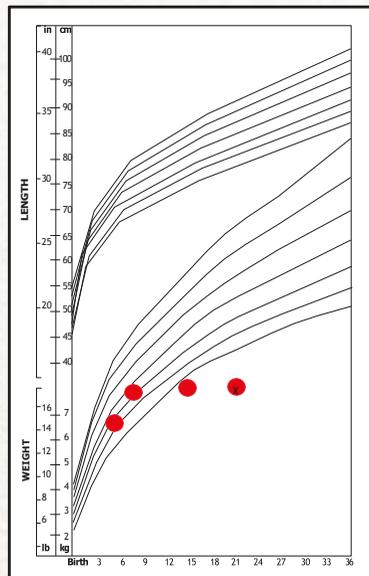


Fig14.7 : Allergy specific test : Multiple food allergens and aeroallergens sensitization are noted. (Normal value is < 0.35 kU/L)

Clinical Diagnosis

ATOPIC ECZEMA

1. Severe generalized
2. Multiple food allergen "sensitization" that is identified based on skin prick test / Allergen specific serum IgE is misinterpreted as multiple food allergies
3. Unwarranted food restriction is complicated by malnutrition & hypoalbuminemia

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment
2. Modification of triggering factors based on their age group, clinical pattern & social background

Allergy tests for aeroallergens and food allergens are indicated for this girl based on:

- a. Moderate to severe Atopic Eczema
- b. Lacking of clinical response despite adequate education, modification of microbial burden & irritants exposure
- c. History of 2 episodes of generalized itchy maculopapular rash within 6 hours after cow milk ingestion

Unfortunately, the post test counseling was not properly performed and she ended up with unwarranted food restriction i.e. avoidance of cow milk and cow milk products, chicken eggs, peanut, seafood, wheat etc.

People who have Atopic Eczema associated with elevated IgE or positive skin prick test are likely to have sensitized (exposed) to certain food and aeroallergens in the environment. But the specificity of these tests are about 50%.

These results need to be correlated to her age, diet history, exposure history and finally confirmed by food challenge test before dietary restriction is initiated.

During our care, she was then put on extensively hydrolysed cow milk for 6 weeks and re-challenged under a controlled setting.

In her food challenge test with cow milk, her eczema had flared up about 4 hours after re-introduction of cow milk.

No clinical reaction to soy based milk & egg white were noted although the se-IgE level (Soya bean & egg white) was slightly elevated.

3. Restoration of skin barrier hydration with moisturizer & gentle cleanser
4. Anti-itch strategies as listed in *figure 7.3*
5. Anti-inflammatory agents

For severe generalized Atopic Eczema, systemic immune modulators play an important role to put the inflammation into remission. This girl was given a 8 weeks course of systemic prednisolone followed by oral Azathioprine for 18 months before we managed to control her eczema.

During the maintenance phase, the inflammatory component is further controlled by a combination of rotational proactive therapy of topical corticosteroid, calcineurin inhibitor and emollient with anti-inflammatory property.

6. Restoration of skin microbiota and anti microbial therapy

CASE 15

Atopic Eczema & the pathological sequence of Itch-scratch inflammation infection



Fig 15.1 / Fig15.2 : Multiple excoriations & erosions are noted over the his upper limbs, especially the posterior aspect of his elbows.

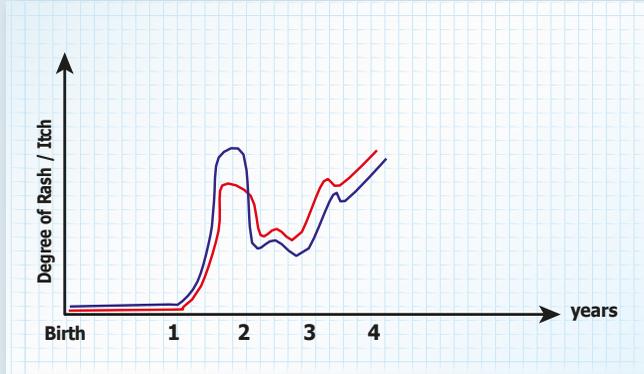
Fig 15.3 / Fig15.4 : Multiple excoriations & crusted erosions are noted over the his lower limbs, especially over the knees and lateral aspect of his shins.

History

4 year-old boy with chronic relapsing itchy rash mainly over his extremities since 1 year-old. His sleep quality is greatly reduced. The disease has altered the emotional and social functioning of the child and his family.

Clinical Course

- Chronic relapsing rash for 3 years
- Degree of Itch



Distribution & Pattern

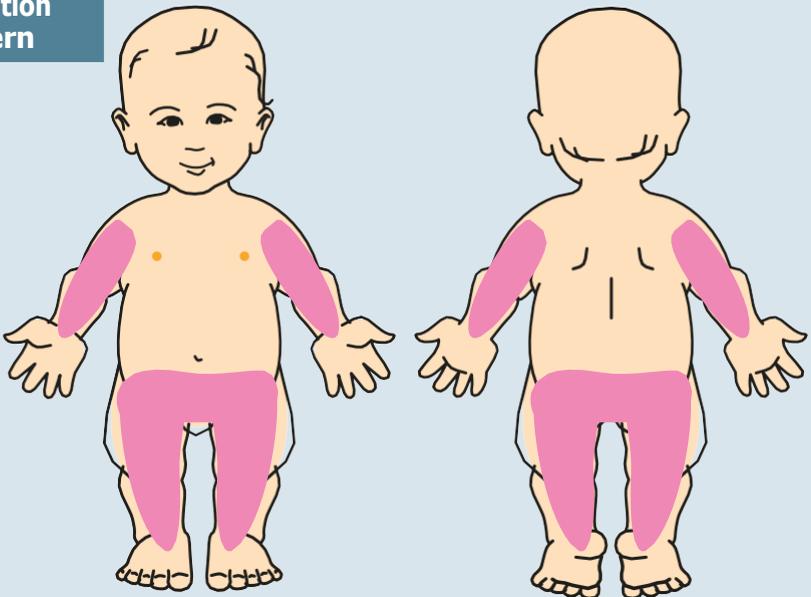
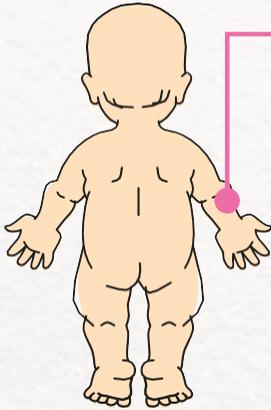


Fig15.5/ Fig15.6 : Multiple excoriations & crusted erosions in linear arrangement are seen at the back of his elbows. These lesions are surrounded by red scaly patches.



Clinical Diagnosis

ATOPIC ECZEMA



Differential diagnosis of acute eruption of vesicles, pustules & erosions in a AE patient are:

- a. Folliculitis
- b. Id reaction
- c. Papulovesicular stage of acute eczema with scratching
- d. Eczema herpeticum
- e. Secondary bacteria infection

Excoriations and erosions that are due to scratching can be differentiated from the others based on:

1. Clinical course: Slower onset than eczema herpeticum & its severity is consistent with degree of scratching noted by history / direct observation
2. Pattern and local arrangement: linear and non follicular (folliculitis)
3. Distribution: more severe over the posterior aspect of both elbows & lateral aspects of both shins

Management & Clinical Pearls

SIX PILLARS APPROACH OF ATOPIC ECZEMA

1. Education & Empowerment

Itch is the hallmark of Atopic Eczema. It is defined as “an unpleasant sensation, eliciting the urge to scratch”.

In severe cases, patients scratch the involved skin areas until bleeding excoriations result. It may then be followed by secondary bacteria infection.

Nocturnal prolonged scratching with sleep loss is a common problem in Atopic Eczema patients.

Intensity of pruritus was significantly related to the stress experienced by the Atopic Eczema patients and has impaired the emotional and social functioning of the child and his family.

2. Modification of triggering factors based on their age group, clinical pattern & social background

3. Skin dryness & restoration of skin barrier

Please refer *question 10* for selection of an ideal moisturiser for this child

4. Anti-itch strategies (refer *table 7.3*)

Empowering the patient with the anti-itch measures listed in *table 7.1* is mandatory in order to modify the itch - scratch - inflammation - infection sequence

5. Anti-inflammatory - topical anti-inflammatory medications are helpful (refer *question 9*)

6. Antimicrobial and restoration or skin microbiota

Topical antibiotic i.e. Fusidic acid, Mupirocin for localized crusted erosions due to secondary bacteria infection, Topical antiseptic (KMnO₄ soak and diluted bleach bath) is useful to prevent recurrent bacterial infection.

