DERMATOLOGY

Clinical Correlation with Diagnostic Implications in Dermatology

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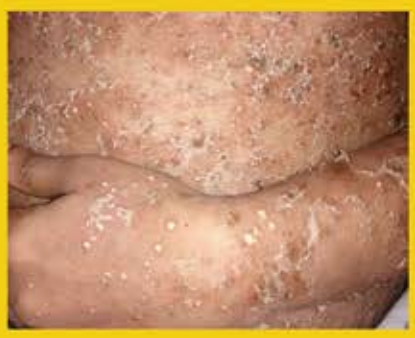
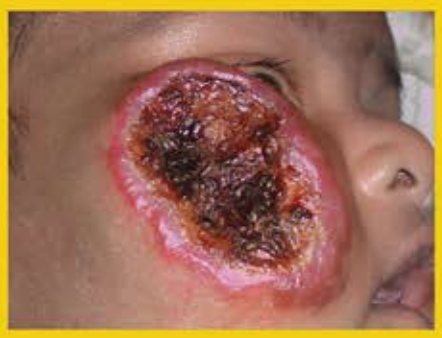
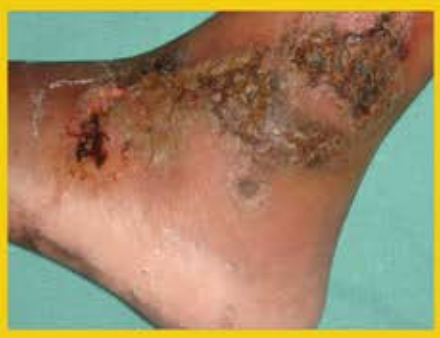
Biju Vasudevan

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JAYPEE

CLINICAL CORRELATION
with Diagnostic Implications in
DERMATOLOGY

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CHAPTER 68

Atopic Dermatitis

Tarang Goyal, Samipa S Mukherjee

Case study Atopic Dermatitis

Presenting Complaints

Doctor, our child is having a reddish rash all over her body with mild oozing from few lesions, for the last 15 days. She has severe itching and sneezes very frequently.

Setting

Dermatology outpatient department.

History of Present Illness

A 12-year-old girl child presented with complaints of itchy red rash all over the body since last 15 days. She has been having these episodes on and off since early infancy with a seasonal variation. The lesions show worsening in winter months. She also has a tendency for running nose and frequent sneezing. All her vaccinations are updated. Family history revealed asthma in maternal grandmother. Overall the itching is severe so as to disrupt the normal sleeping pattern of the child.

General Examination and Systemic Examination

A well-oriented child, with no lymphadenopathy, hepatosplenomegaly. Mild pallor was present.

Dermatological Examination

Edematous, erythematous, excoriated oozing ill-defined plaques predominantly present over face, periorbital area, neck, trunk, antecubital fossae, popliteal fossae and ankles (Figs 1 to 3). Scalp shows redness and fine scaling. Palms and soles show hyperlinearity. Nails, mucosae normal.

Q1. What is the clinical diagnosis?

a. Seborrheic dermatitis
b. Atopic dermatitis
c. Erythroderma
d. Nummular eczema

Ans: b (Atopic dermatitis)

Q2. What is atopic dermatitis (AD)?

Ans: Atopy is derived from the greek word "*atopos*" which means out of place or strange and refers to the predisposition to develop Allergic rhinitis, bronchial asthma, or AD with resultant surfaces where the body contacts the allergen, being "overreactive". Atopic dermatitis is a chronic, pruritic inflammatory skin disease that occurs most frequently in children.

Q3. What is the incidence of AD globally?

Ans: Atopic dermatitis affects up to 20% of children and up to 3% of adults; recent data show that its prevalence is still increasing, especially in low-income countries. It is very common in infancy and early childhood.

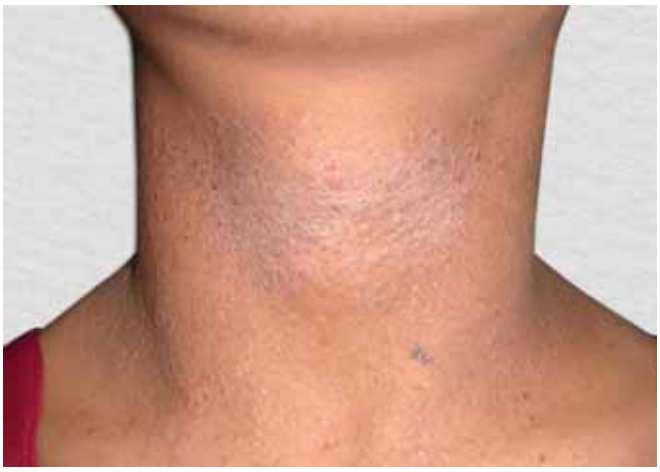

Fig. 1: Erythematous, excoriated ill-defined plaques predominantly present over neck

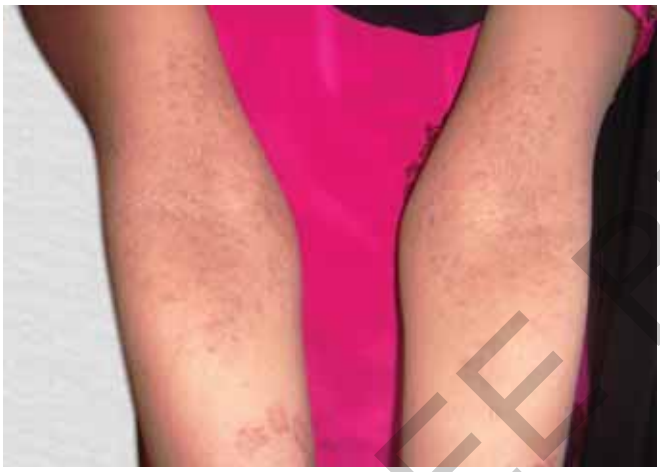

Fig. 2: Similar lesions on flexural aspect of elbows

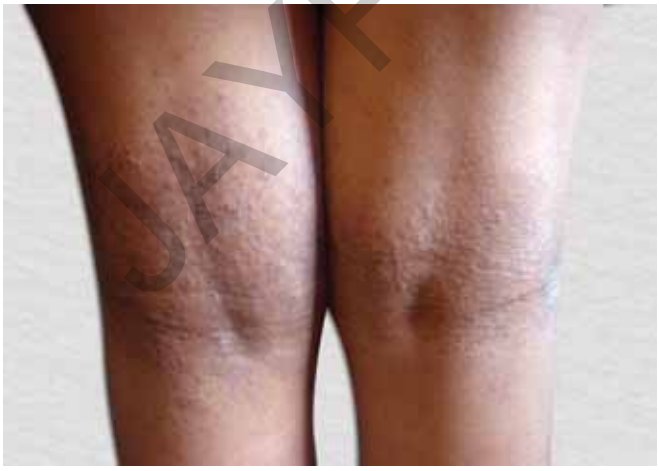

Fig. 3: Similar lesions on flexural aspect of knees

Q4. What is "atopic diathesis"?

Ans: Allergic rhinitis, bronchial asthma, or atopic dermatitis occurrence in patients.

Q5. What is atopic march?

Ans: The term "Allergic March" (also called "Atopic March") refers to the natural history of atopic manifestations, which is characterized by a typical sequence of immunoglobulin E (IgE) antibody responses and clinical symptoms which may appear early in life, persist over years or decades, and often remit spontaneously with age.

Q6. What are the diagnostic features of AD?

Ans: The Hanifin and Rajka criteria

- Major features:
  - Pruritus
  - Typical morphology and distribution
  - Chronically relapsing dermatitis
  - Personal or family history of atopy.
- Minor features (three or more):
  - Xerosis
  - Ichthyosis, palmar hyperlinearity, or keratosis pilaris
  - Immediate (type 1) skin-test reactivity
  - Raised serum IgE
  - Early age of onset
  - Tendency toward cutaneous infections (especially *Staphylococcus aureus* and herpes simplex) or impaired cell-mediated immunity
  - Tendency toward nonspecific hand or foot dermatitis
  - Nipple eczema
  - Cheilitis
  - Recurrent conjunctivitis
  - Dennie-Morgan infraorbital fold
  - Keratoconus
  - Anterior subcapsular cataracts
  - Orbital darkening
  - Facial pallor or facial erythema
  - Pityriasis alba
  - Anterior neck folds
  - Itch when sweating
  - Intolerance to wool and lipid solvents
  - Perifollicular accentuation
  - Food intolerance
  - Course influenced by environmental or emotional factors
  - White dermographism or delayed blanch.

Q7. What are etiopathological factors in development of AD?

Ans: The following are the etiopathological factors in development of AD:

- Decreased skin barrier function
- Immune responses (T helper-2 in acute AD and T helper-1 like in chronic AD)
- Genetic predisposition
- Altered skin barrier function
- Environmental factors and infectious agents.

Q8. What are the scoring systems used in AD?

Ans: The Diepgen score, EASI (eczema area scoring index), SCORAD (SCORing Atopic Dermatitis) and POEM (Patient-Oriented Eczema Measure) are used as scoring systems in AD.

Q9. What are the potential risk factors associated with rise in AD?

Ans: The following are the potential risk factors associated with rise in AD:

- Small family size
- Increased income and education
- Rural to urban migration
- Increased use of antibiotics.

Q10. What is "hygiene hypothesis" in AD?

Ans: Allergic diseases may be prevented by avoiding them acquiring "infections" from older siblings in early childhood. Reduced exposure to bacterial and parasitic infections in childhood leads to an abnormal development of the immune system, which later tends to overreact to innocuous antigens.

Q11. What are extrinsic and intrinsic types of AD?

Ans: Atopic dermatitis can be classified into two types based on the presence or absence of elevated IgE and specific IgE sensitization. (1) "Extrinsic type" of AD, also known as "allergic type", is associated with elevated IgE (45–80%). (2) In "intrinsic type", also known as "atopiform" dermatitis or "nonallergic" type, the IgE levels are normal (20–40%). Extrinsic type is more common in adult onset AD unlike intrinsic AD.

Q12. What is the role of decreased barrier function in AD?

Ans: The decreased barrier function leads to increased allergen absorption into skin and microbial colonization. The epicutaneous sensitization to allergen results in higher allergic immune responses in children.

Q13. In what ways the epidermal barrier function is compromised in AD?

Ans: The epidermal barrier function is compromised in AD in many ways:

- Downregulation of filaggrin and loricrin
- Decreased ceramide and antimicrobial peptide levels
- Increased levels of proteolytic enzymes
- Colonization by *S. aureus* and house dust mites
- Increased transepidermal water loss.

Q14. What are the strong and consistent risk factors in development of AD?

Ans: A family history of atopy and loss-of-function mutations in the filaggrin gene, which codes for filaggrin.

Q15. What cytokines are responsible for AD?

Ans:

- *In acute AD*: T helper-2 cytokines; interleukins (IL) such as IL-4, IL-13, and IL-31 are seen. IL-13 induces itching in AD patients. IL-31 correlates with severity of lesions
- *In chronic AD*: IL-5 and T helper-1 like cytokines (IL-12 and IL-18) are seen. IL-5 is responsible for eosinophil development and survival
- Cutaneous T cell attracting chemokine (CTACK); CC chemokine ligand 27 is also upregulated in AD.

Q16. What is the basis of pruritus in AD?

Ans: IL-31, IL-2, stress-induced neuropeptidases, proteases, substance P, acetylcholine, prostanoids, and calcitonin are responsible for itching in AD patients. Thereby the itching in AD is not controlled with antihistaminics; they have only a sedative role which is beneficial.

Q17. What are the patterns of AD presentation by age?

Ans:

- *Infantile (2 months–2 years)*: Symmetric involvement of cheeks, forehead, ears, scalp, extensor surfaces of extremities, and trunk. There is intense pruritis. Erythema, oozing, crusting, papules, and vesicles are commonly present.
- *Childhood (2 years to puberty)*: Wrists, ankles, neck, posterior thighs, buttocks, antecubital and popliteal fossae are commonly involved. Intense itching with poorly defined erythematous scaly plaques with secondary crusting, oozing, and erosions.
- Adults: Face, neck, arms, back, flexures commonly involved. Intense itching with thick lichenified plaques.

Q18. What infections are usually seen in AD?

Ans: The following infections have been shown to be commonly associated with AD:

- *Viral infections*: Herpes simplex (Kaposi varicelliform eruption or eczema herpeticum), small pox vaccination-induced eczema vaccinatum
- *Superficial fungal infections*: *Trichophyton rubrum, Malassezia sympodialis*
- *Bacterial infections*: *S. aureus* [particularly MRSA (methicillin-resistant *S. aureus*)].

Q19. What causes facial predominant AD?

Ans: Facial predominant atopy has a high colonization of the yeast *Pityrosporum sympodialis*.

Q20. What are the poor prognostic factors in AD?

Ans: The following are the poor prognostic factors in AD:

- Widespread AD
- Early onset AD
- Associated allergic rhinitis or bronchial asthma
- Family history of AD in parents or siblings
- Only child
- Very high serum IgE levels.

Q21. What physical findings are associated with AD?

Ans: The following physical findings are associated with AD:

- Xerosis
- Keratosis pilaris
- Pityriasis alba
- Hyperlinear palms and soles
- Hertoghe's sign
- Dennie-Morgan folds
- Dermatographism
- *Ocular complications*: Chronic blepharitis, atopic keratoconjunctivitis, vernal conjunctivitis, keratoconus, cataract (particularly anterior subcapsular cataract).

Q22. What is Hertoghe's sign?

Ans: Thinning or absence of hair on the lateral aspect of eyebrows.

Q23. What are the differential diagnoses of AD?

Ans:

- Allergic contact dermatitis
- Immunodeficiency
- Irritant contact dermatitis
- Lichen simplex chronicus
- Nummular dermatitis
- Scabies with eczematization
- Psoriasis
- Seborrheic dermatitis
- Extensive tinea corporis.

Q24. Are investigations necessary in AD?

Ans: Investigations are not needed in all cases of AD. Investigations may be done only in cases of suspected syndrome or as a pretreatment work up with systemic medications. IgE levels, skin prick test, radioallergosorbent testing (RAST), food-specific IgE levels, immunocap to know whether the child has outgrown the allergy may be done.

Q25. What are the common trigger factors of AD?

Ans: Certain foods especially cow's milk, milk products, soy, nuts, egg white can sometimes worsen AD. Aeroallergens such as animal dander, pollen, molds, and house dust mites should be avoided. Woolen clothes, carpets, furry toys, and pets are other common triggers. Psychological triggers such as stress of examination, anxiety, social pressure also play a role.

Q26. What are the general treatment plans for AD patients?

Ans: The following are the general treatment plans for AD patients:

- Avoid trigger factors
- Continue breastfeeding and delay exposure to solid foods (though not proven)
- Adequate moisturization of skin by an emollient which is customized according to patient
- Avoid long and very warm water baths, use bleach baths
- Use a mild soap or syndet
- Wear cotton clothes
- Keep humidity to 35-40%
- Avoid kissing the child
- Counseling and good psychological support remains the cornerstone.

Q27. What is "wet-wrap" therapy?

Ans: It is also known as "two-pajama", "two-caps", "two-gloves", "two-shirts", etc. treatment therapy according to the site involved. Immediately after bathing, put a mild-to-mid potency corticosteroid to skin. Soak one pajama in warm water and wear it wrung-out. Then wear a dry one on top of it and sleep.

After waking up in morning, remove both and apply moisturizer.

Q28. What are the topical treatments available for AD?

Ans: The following topical treatments are available for AD:

- Moisturizers and emollients
- Topical antiinflammatory therapies (topical glucocorticoids, topical calcineurin inhibitors)
- Topical antibiotics.

Q29. What is the role of probiotics in treatment of AD?

Ans: A Cochrane review done recently had concluded that probiotics are not an effective treatment for eczema in children and this could even lead to risk of few adverse effects.

Q30. What are the syndromes associated with AD?

Ans: Syndromes:

- Job's syndrome
- Hyper IgE syndrome
- Netherton's syndrome
- Wiskott-Aldrich syndrome.

Q31. What is the drug of choice for atopic erythroderma?

Ans: Cyclosporine is the drug of choice for atopic erythroderma due to its rapid onset of action, excellent control, and lesser side effects.

Q32. What are the other treatment options for AD?

Ans:

- Oral corticosteroids
- Cyclosporine
- Methotrexate
- Azathioprine
- Antihistaminics
- *Phototherapy*: Preferably narrowband ultraviolet B.

Q33. Which is the latest biological treatment that is undergoing phase 3 trial for AD?

Ans: Dupilumab: It is a monoclonal antibody against IL-4.

CHAPTER 69

Hand Eczema

Boby Krishna

Case study Hand Eczema

Presenting Complaints

Doctor, I have itchy raised lesions over both hands since the last 5 years with an acute exacerbation for 2 weeks.

Setting

Dermatology outpatient department.

History of Present Illness

A 29-year-old construction worker presented with history of recurrent episodes of numerous itchy raised lesions on the palms for the last 5 years. He consulted a local hospital and was treated with topical steroid creams and antihistamines, following which the lesions improved but to reappear later. He has noticed exacerbation of the skin lesions at work place. Presently since 2 weeks he has developed fresh lesions on hands with oozing, crusting and severe itching.

General Examination and Systemic Examination

No abnormality detected.

Dermatological Examination

Hyperkeratotic plaques on the palmar aspect of both hands along with oozy crusted lesions on the dorsum of both hands (Fig. 1). Excoriation marks were present. Edema of hands present.

Patch Test

Positivity to potassium dichromate.

Q1. What is the most likely diagnosis?

a. Psoriasis of the hands
b. Allergic contact dermatitis leading to chronic hand eczema
c. Dermatophytic infection of the hands
d. Irritant contact dermatitis leading to chronic hand eczema

Ans: b (Allergic contact dermatitis leading to chronic hand eczema)

Chronic relapsing course especially exacerbating at work place and patch test positivity to potassium dichromate (ingredient in cement) all favors allergic contact dermatitis to cement.

Q2. What is hand eczema?

Ans: Dermatitis largely confined to hands with or without minor involvement of other areas. It accounts for 20–35% of all dermatitis. The disease can be acute,

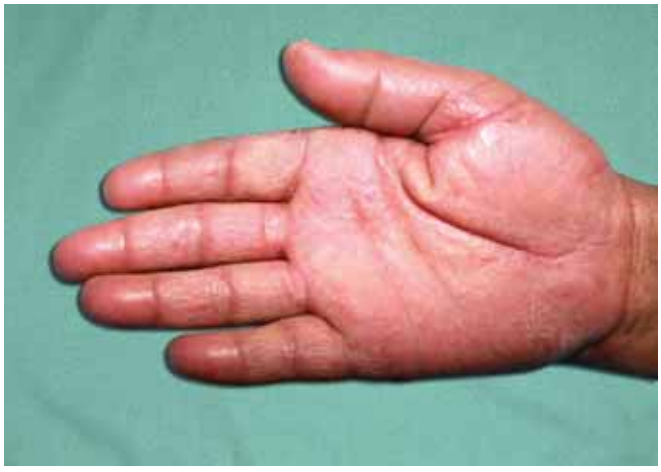

Fig. 1: Hyperkeratotic scaly lesions on palm with edema

lasting less than 3 months, or chronic or appear as recurrent flares in between periods with normal skin.

Q3. How do you classify hand eczema?

Ans: Classification of hand eczema:

Etiological

- Endogenous
  - *Immunological*: Atopy
  - *Psychosomatic*: Stress (e.g.: repeated picking, rubbing)
  - *Dyshidrosis*: Increased sweating
  - *Idiopathic*: Hyperkeratotic hand eczema
- Exogenous
  - Irritant contact dermatitis
    - *Chemical*: Detergents, soaps, etc.
    - *Physical*: Friction trauma, cold dry air, sweat
  - Allergic contact dermatitis
    - *Type I hypersensitivity reaction*: Contact urticaria to vesiculation
    - *Type IV hypersensitivity reaction*: Dermatitis to rubber, nickel, chromium, etc.
  - Ingested allergens
    - Food or drugs containing nickel chromium, etc.
  - Infection
    - Secondary to bacterial, fungal or viral infection.

Morphological

- Apron eczema
- Chronic acral dermatitis
- Discoid eczema (nummular eczema)
- Fingertip eczema
- Gut eczema
- Hyperkeratotic palmar eczema
- Pompholyx
- Recurrent focal palmar peeling
- Ring eczema
- Wear and tear dermatitis
- Others (patchy, vesiculosquamous).

Q4. What is the differential diagnosis of hand eczema?

Ans:

- *Psoriasis*: Well demarcated, erythematous, scaly plaques with nail changes
- *Palmoplantar pustulosis*: Persistent pustulosis usually on an erythematous base affecting central palm and instep of foot
- *Tinea manuum*: Unilateral patch, with scaly or pustular edge with positive mycology
- *Keratolysis exfoliativa*: Recurrent focal peeling on fingers, palms and soles
- *Bowen's disease*: Persistent red scaly patch resembling chronic dermatitis.

Q5. What are the risk factors for hand eczema?

Ans: Risk factors for hand eczema are listed in Table 1.

Table 1: Risk factors for hand eczema

Extrinsic factors
• *Wet work*: Ongoing exposure to liquids, frequent hand washing or occlusion with gloves • *Smoking*: Association between smoking and hand eczema • *Stress*: Exacerbates • *Site*: Back of hands more permeable than skin on palms therefore more vulnerable to irritants • Fat soluble chemicals enters skin even through undamaged horny layer • *Temperature*: High temperature—decreased barrier function, increased penetration of irritants and allergens • *Air flow*: Cold dry air—chapping of skin increased sensitivity of skin • *Low humidity*: Decreased water content and plasticity of stratum corneum—chapping and cracking • *Occlusion*: Promotes percutaneous absorption (gloves, wrist watch, straps, rings, shoes, boots, etc.). • *Occupation*: Healthcare workers, food service workers, beauticians, construction workers, homemakers, machinists and warehouse workers • *Medications*: Opiates—enhances allergic and irritant contact dermatitis • *Socioeconomic*: Exposure to cheap metals such as nickel and tattooing—dyes allergy.
Intrinsic factors
• *Age*: As age increases, barrier function deteriorates. • *Atopy*: Principal endogenous factor • *Dry skin*: more sensitive to irritants • *Ethnicity*: Whites more prone • *Sex*: Females more affected • *Genetic*: TNF α (tumor necrosis factor alpha) gene polymorphism • Coincidental diseases, e.g. cancer, HIV (decreased T lymphocyte function). • *Hormones*: Premenstrual flare up, e.g. nickel allergy.

Q6. How to differentiate irritant contact hand eczema and allergic contact hand eczema?

Ans: Both cannot be reliably distinguished based on clinical features alone (Table 2). Most often ICD (irritant contact dermatitis) precedes ACD (allergic contact dermatitis).

Q7. What are the features of vesicular hand dermatitis?

Ans: Popularly known as pompholyx or dyshidrotic eczema. There is no sweat gland dysfunction in dyshidrotic eczema, so it is a misnomer. Long-lasting, pruritic, vesicular and primarily involves the palmar surface of hand. Vesicles surmount on an erythematous base. Does not involve wrist and spares the dorsal surfaces of hands completely or limited to dorsal fingertips. Soles usually affected. Difficult to treat condition.

- Sudden onset of large bullae on palms can occur.
- They usually resolve in 2–3 weeks and recur at varying intervals.

Q8. Which among the following is not a synonym for housewives' dermatitis?

a. Wear and tear dermatitis
b. Apron eczema
c. Dermatitis palmaris sicca
d. Asteatotic eczema

Ans: b (Apron eczema)

Unlike housewives' dermatitis, apron eczema is an endogenous eczema which involves proximal palmar aspect of two or adjacent fingers and contiguous palmar skin over the metacarpophalangeal joints resembling an apron. Housewives' dermatitis occurs due to a combination of asteatosis, irritant exposure (soaps and detergents) and mild trauma. In this eczema, palmar skin feels dry and criss-crossed with superficial cracks along with chapping of the skin over the dorsa of knuckle joints.

Table 2: Difference between irritant contact hand dermatitis and allergic contact hand dermatitis

Irritant contact hand eczema	*Allergic contact hand eczema*
Finger web space involvement with extension to dorsal or ventral surfaces of hand	Fingertip, nail fold and dorsal hand involvement
Localization on palms and ball of thumb. Nonvesicular pattern	Frequently with vesiculation. Palms rarely primarily affected
Often begins around 3 months after exposure, usually wet work	Local reaction after 5–25 days
Upon re-exposure, quickly recurs	Re-exposure, reaction within 24–48 hours
Upon removal, the reaction improves—decrescendo pattern	Reaction often crescendo over 1–3 weeks before improving
Direct cell injury	Type IV hypersensitivity reaction. Patch testing helps to identify the antigen

Q9. How to diagnose hand eczema?

Ans:

- A thorough history including medical, occupational and social history is important.
- Physical examination
  - Hands
    - dorsal and palmar involvement
    - any vesicles or pustules
    - finger-tip and finger web involvement
    - wrist involvement
    - nail pitting
  - Any other area involved
- Patch testing
  - Considered in chronic hand dermatitis
  - Gold standard for diagnosing ACD
- Potassium hydroxide examination and fungal culture
- Biopsy to rule out other noneczematous skin diseases: Psoriasis, keratoderma, dermato-myositis, vesicular bullous pemphigoid, CTCL (cutaneous T-cell lymphoma)
- Radio allergosorbent testing (RAST) or prick testing for contact urticaria to foods, latex and other allergens.

Q10. What are the treatment modalities in hand eczema?

Ans: *Behavioral Changes*

- Elimination of known irritants and allergens
- Creating physical barriers with gloves
  - Vinyl gloves—for wet works and irritants
  - Rubber gloves/plastic—only with cotton lining especially if used for more than 10 minutes.
- Emollient barrier creams and ointments applied before work and reapplied after washing and whenever skin dries out
- Changing occupations
- Educating atopic patients to avoid irritants and wet work in home and at work is critical.
- Avoid excessive sweating and dry conditions which triggers hand eczema
- Minimize frequent hand washing with soap
- Stress avoidance

Topical Therapy

- *Topical corticosteroids*: Ultra potent creams/ointments for 2–4 weeks, then pulsed at weekends. Ointments are more effective than creams because of greater penetration and less

potentially sensitizing preservatives than creams. Preservatives such as formaldehyde releasers, parabens, MCI/MI in topical steroid creams are responsible for ACD.

- *Topical immunomodulators*: Topical pimecrolimus and tacrolimus inhibit inflammatory cytokine production by T-lymphocytes and mast cells. Twice daily application, with or without occlusion overnight. Transient skin burning and warmth are adverse effects. Pimecrolimus safer for long-term and widespread use due to lipophilicity and lower potential for systemic immunosuppression.
- *Intradermal injection of triamcinolone* (10 mg/mL): In recalcitrant localized patches.
- Nontraditional agents for topical use
  - Vitamin D_3 analogs
    - Calcitriol
    - Calcipotriol
    - Tacalcitol especially in hyperkeratotic palmar eczema
  - Tazarotene: Hyperkeratotic hand dermatitis
  - Tar and salicylic acid preparation: Hyperkeratotic hand dermatitis.

Wet dressings for early vesicular phase of pompholyx using 3% acetic acid or potassium permanganate compresses and the use of steroid-antiseptic or antibiotic preparation may help to lessen the risk of infection and improve the response.

Radiation and Light Therapy

- Grenz Rays
- PUVA (photochemotherapy), NBUVB (narrow-band ultraviolet B)

Systemic Therapy

In severe and recurrent cases of hand eczema.

- Systemic steroids: Start with 20–40 mg of prednisolone single daily dose in the morning for 3–7 days to a maximum of 10–14 days. This is repeated if necessary.
- Cyclosporin: 3 mg/kg per day for 6 weeks for resistant cases
- Mycophenolate mofetil: 2–3 g/day
- Methotrexate: 12.5–22.5 mg/week
- Azathioprine: 50–150 mg/day
- Oral antibiotics
- Retinoids: Acitretin proven effective. Alitretinoin, a new oral retinoid once daily treatment with 10, 20 or 40 mg/day. It can be used for severe, never go away completely or recurrent hand eczema.
- Biologicals: Etanercept in recalcitrant pompholyx and alefacept and ustekinumab in hyperkeratotic hand eczema
- Other therapies.

Iontophoresis

- *Botulinum toxin A injection*: Both are effective therapies for hyperhidrosis thereby, decreases sweat-induced irritation that occurs in occupational and atopic dermatitis.
- *Vitamin E, oral iron and oral zinc*: All showed benefits in hand eczema due to metal allergy.

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