

	Pathogenesis	Clinical Picture	Diagnosis	Management
Atopic Dermatitis – Chronic relapsing eczema associated with intense pruritus.	<ul style="list-style-type: none"> – Genetic predisposition [atopy]. – Allergy – Immune mediated [IgE & T-cell] – Impaired skin barrier. – Dry skin [decrease production of moisturizing lipids; sebum] 	Acute: erythema, papules and vesicles & oozing Subacute: scales and excoriation. Chronic: lichenification & hyperkeratosis Stages & clinical variant: Infantile Dermatitis: Acute itchy inflammation involving face and extensors [elbows&knees]. Improves with age. Childhood Dermatitis: Subacute or chronic dermatitis more over extensors [elbow & knee folds, neck, wrist & ankle, groin]. Disappears in 70% of patients by the age of 6. Adults: Chronic dermatitis & more on flexures.	Clinical diagnosis. Criteria for atopic dermatitis: Major: Pruritus Typical morphology & distribution. Chronicity Personal or family history of atopy [Minor Criteria page 3] Labs: 1. IgE normal IgE does not exclude the disease. 2. CBC to exclude the infections Biopsy: –Depend on the stage –Spongiosis (oedema) –Exocytosis of lymphocytes Complications: 1. 2ndry infection: Bacterial by Staph aureus [impetigo & folliculitis] Viral by HSV [Eczema herpeticum] Fungal by Trichophyton Rubrum.	Education: Avoid alkali soaps Avoid woolen clothes & wear cotton Skin care: Emollient: Moisturizing the skin 1st line of treatment: •Topical therapy: Topical steroids Topical immuno-modulators [Tacrolimus, Pimecrolimus] [alternative treatment for steroids] •Oral antihistamine [sedating effect] •Oral antibiotics [2ndry infections] 2nd line [sever or resistant] Phototherapy Systemic therapy: steroids, Cyclosporin, Methotrexate, Azathioprine
Seborrhic Dermatitis – Common mild chronic eczema typically confined to skin regions with high sebum production & body folds	<ul style="list-style-type: none"> – Increased Sebum production by the sebaceous glands – Genetically or familial tendency – Over growth of commensal yeast Malassezia furfur (pityrosporum ovale) 	Presentation: Erythematous red-yellow, poorly circumscribed patches & thin plaques with bran-like to flaky (greasy) scales. Cradle Cap: Coherent scaly & crusty mass coring most of the scalp, seen in infantile SD. Timing: Periods when sebaceous glands are active [androgen dependent]: 1st few months of life & post puberty. Location: Areas rich in sebaceous glands e.g: scalp, face, ears, presternal region & flexural areas (axillae, inguinal & infra-mammary folds, umbilicus).	Seborrhic Dermatitis has a mild course with moderate discomfort [some are asymptomatic].	<ul style="list-style-type: none"> •Topical anti-fungals & anti-fungal shampoos •Topical low potent steroid [anti-inflammatory effect] •Topical immuno-modulators [if steroids are contraindicated] •Salicic Acid preparation 2–5% [Keratolytics] used in cradle scalp.

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Contact Dermatitis:	Allergic Contact Dermatitis: Dermatitis resulting from type 4 reaction following exposure to topical substances in sensitized individuals.	Presentation: Acute form present with crusted erythematous papules, vesicles & bullae that is well demarcated Location: localized to the site of contact with the allergen e.g. Nickel, rubber, fragrances & preservatives It can be more diffuse in distribution.	Clinical diagnosis PATCH testing remain the gold standard for accurate diagnosis.	<ul style="list-style-type: none"> •Avoidance. •Topical steroid •Systemic steroid [in sever cases] •Oral antihistamine [sedating effect]
	Irritant Contact Dermatitis: Localized non immunologically mediated inflammatory reaction. Results from direct cytotoxic effect d.t single or repeated application of a chemical substance to the skin.	<ul style="list-style-type: none"> – Same as allergic dermatitis but never extend beyond the area of contact – Tends to be painful rather than pruritic – Occurs from the 1st exposure 		
Discoïd Dermatitis [nummular]:	Microbial in origin i.e. 2ry to bacterial colonization or dissemination of bacterial toxins	<ul style="list-style-type: none"> – Sharply circumscribed eczema, nummular means (coin-shaped) – Usually very pruritic 	Clinical diagnosis	<ul style="list-style-type: none"> •Topical steroid •Topical antibiotic •Oral antibiotic
Dyshidrotic Dermatitis [pompholyx] – Housewife's dermatitis	<ul style="list-style-type: none"> –Not considered as a separate disease –Can be associated with atopy in 50% of patients. –Exogenous factors (e.g. contact dermatitis to nickel, chemicals) also play a role. 	<ul style="list-style-type: none"> – Acute dermatitis which is often vesicular with tiny deep seated vesicles – Associated with pruritus Location: Along the sides of the fingers & toes		<ul style="list-style-type: none"> •Avoidance of triggering factor. •Topical steroid.
Stasis Dermatitis		<ul style="list-style-type: none"> – Seen in patients with venous hypertension; chronic lower limb edema & varicose vein. – Can be complicated by superimposed allergic contact dermatitis 		
Neurodermatitis	Stress and anxiety that results in repeated rubbing & scratching of the skin. Common in atopic patients.	Chronic itching and scratching can cause the skin to thicken and have a leather texture with exaggeration of skin marking. Example of Neurodermatitis: Lichen Simplex Chronicus: Thick hyperkeratotic plaque with accentuation of skin marking. Location: Any site that the patient can reach, including the following: <ul style="list-style-type: none"> –Scalp& nape of neck –Extensor forearms and elbows –Vulva and scrotum – thighs, knees, lower legs & ankles 		Control itching (break itch scratch cycle). Topical or intra-lesional steroid. Oral antihistamine Oral Anxiolytic

Criteria to diagnose Atopic Dermatitis:**Major Criteria:**

Pruritus
 Typical morphology & distribution.
 Chronicity
 Personal or family history of atopy

Minor Criteria:

- Xerosis
- Ichthyosis/hyperlinear palms/keratosis pilaris.
- IgE reactivity
- Elevated IgE level
- Early onset
- Skin infection
- Chelitis
- Nipple eczema
- Recurrent conjunctivitis
- Keratoconus
- Dennie morgan fold
- Anterior cataract
- Orbital darkening
- Facial erythema
- Pityriasis alba
- Food hypersensitivity
- White dermatographism

Side Effects of prolonged use of steroids:**Cutaneous side effects:**

Impaired wound healing
 Skin infection
 Striae & atrophy
 Telangiectasia

Systemic side effects:

Adrenal insufficiency & Cushing's syndrome
 Hyperglycemia
 Hypertension
 Osteoporosis
 Psychosis

N.B: These side effects can be caused by prolonged use of topical steroids in children because of their large surface area.

	Atopic Dermatitis	Seborrheic Dermatitis
Presentation	Starts at 3 months of life	Starts shortly after birth [2 weeks] Improves and comes back in puberty
Symptoms	Markedly pruritic	Asymptomatic
Morphology	Erythema, papules and vesicles	Greasy scales & erythematous base
Location	Face & extensors in infants More on extensors in childhood More flexures	Scalp, nasolabial folds & body folds.
	Allergic Contact Dermatitis	Irritant Contact Dermatitis
Pathophysiology	Immune mediated; Type 4 delayed hypersensitivity	Non-immunological From direct cytotoxic effect of a chemical
Irritants	Person must be sensitized [2nd exposure] Irritates only those allergic to it	1st exposure. Irritates everyone.
Symptoms	Pruritic but not painful	Painful
Location	Site of contact but could be diffuse.	Never extends beyond the site of contact