| Inflammatory skin conditions | | | |
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| | pathophysiology | Clinical presentation | Treatment |
| Acne vulgaris | Increased androgen ? increased sebum production.<br>Comedones form due to accumulation of lipid and keratin in pilosebaceous unit.<br>Medium for bacterial overgrowth (p.acne) which convert sebum into fatty acids that elicit inflammatory response. | -Black heads (open comedones): oxidized keratin plug.<br><br>-White heads (closed comedones).<br><br>-Nodules: follicles rupture and inflame dermis.<br>- Nodulocystic lesions | -**Topical antiseptics** (benzoyl peroxide).<br>-**Oral antibiotics** (tetracycline e.g; doxycycline, minocycline).<br>-**Topical retinoids.**<br>-**Oral isotretinoin**: reserved for severe nodulocystic scarring acne. |
| Rosacea | not entirely understood.<br>Abnormal vasoactivity.<br>Usually in adults of nouthern European heritage<br>**Trigger factors**<br>**-Hot** weather, hot drinks, spicy food<br>Stress, **alcohol**, nicotine<br>*-Demodex* mites | -flushing, Papules and pustules often on central face.<br>-Telangiectasias<br>-Rhinophyma<br>- no comedones | -topical antibiotics (metronidazole).<br>-topical azelaic acid.<br>-oral antibiotics (tetracyclines)<br>-oral isotretinoin (for refractory disease) |
| Seborrheic dermatitis | may be associated with *Malassezia* species (M.furfur) colonization in areas with sebaceous glands. | -dandruff on the scalp (the mildest form).<br>- erythematous plaques, greasy yellow scales commonly on Forehead, hairline, retroauricular, nasolabial folds, eyebrows, trunk.<br>-In infants: on scalp “cardle cap”, diaper area, face, neck, axilla. | - topical antifungals (ketoconazole shampoo and cream).<br>- topical steroid<br><br>*There is no cure (problem recur when the treatment is stopped) |
| Atopic dermatitis | -Familial.<br>-Associated with asthma | -Acute eczema: intense erythema and itching, | - Topical: emollients, steroids, tacrolimus. |

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| (endogenous eczema) | and allergic rhinitis (atopic triad).<br>- Filaggrin gene mutation (epidermal protein that maintain skin barrier).<br>- Increased Th2 cell mediated response.<br>- Th2 vs. Th1/17 imbalance.<br>- Exposure to environmental triggers and IgE mediated hypersensitivity (type 1) | intraepidermal edema (spongiosis), Papulo-vesicles, oozing.<br>-Subacute: crusting and scaling, mild itching.<br>-Chronic: lichenification (thickening and hyperpigmentation), intense itching.<br>* Infants: face, scalp and extensor surfaces.<br>Children: flexor surfaces (antecubital fossa, popliteal fossa, wrist).<br>Usually resolves in childhood.<br>*Complications: eczema herpeticum (life-threatening), impetigo (most common).<br>*Variants: pityriasis alba, juvenile plantar dermatosis. | - <u>systemic:</u><br>Antihistamine, Immunosupressants (MTX, azathioprine, cyclosporine): if irresponsive to therapy.<br><br>Antibiotics or antivirals if superinfection.<br>Eczema herpeticum ? IV acyclovir. |
| Asteatotic eczema | Low humidity : desert, high altitude, travel.<br>Excessive bathing especially using soaps and detergents | dry, itchy ,scaly, cracked skin with network of shallow erythematous fissures in the epidermis **'crazy paving'.** Common on elderly usually on shins | Emollients + mild topical steroid |
| Pompholyx (vesicular eczema) | | * itchy rash presents on plams and soles centers and with tiny vesicles that may evolve into bullae.<br>Affect 20-40 year old.<br>* In severe cases : nail dystrophy + paronhychia<br>* 2ry bacterial infections | Potassium permanganate soaks (drying agent) + potent topical steroid.<br><br>Systemic antibiotics if needed. |

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| | | is common. | |
| Discoid eczema | | * Well defined, scattered coin – disc shaped plaques that are extremely itchy with vesicles or crusting. On the limbs, usually in middle age. | Emollients + potent topical steroid |
| Stasis eczema | * Common in patients with chronic venous HTN, DVT, varicose veins , obesity, pregnancy | Diffuse erythema, scaling, crusting and itching on lower third of both legs without pain. Hyperpigmentation, venous ulcers are common | Treatment of underlying condition,<br>Emollient + moderately potent topical steroid |
| Allergic contact dermatitis (exogenous eczema) | Type IV hypersensitivity reaction in response to specific allergen in a sensitized person.<br>Examples: nickel, poison ivy, neomycin..) | Pruritic erythematous papules and vesicles that later scales and crusts, at the site of exposure but spreading in the periphery.<br><br>Diagnostic test: positive patch test. | -avoidance of offending allergen.<br>- topical steroids |
| Irritant contact dermatitis (exogenous eczema) | a localized inflammatory skin reaction caused by a direct cytotoxic effect of a causal agent. occur in anyone above threshold level.<br>Examples: acids and alkalis, detergents, mineral oils, napkin dermatitis, wear and tear household eczema | Pruritic erythematous vesicles, crusting and scaling that is **strictly confined** to the site of exposure. | -avoidance of the causative agent.<br>-topical steroid. |
| Psoriasis | Multifactorial.<br>Genetic: (HLA-C) | - plaque psoriasis (most common): | -avoid smoking, excessive alcohol, reduce weight. |

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| | environmental: stress, infection, trauma, alcohol, smoking.<br>-**Dysregulation of Th1/ Th17.**<br>-Psoriatic Plaques are highly infiltrated with CD3+ T-cells & CD11c+ dendritic cells.<br>-Release of pro-inflammatory cytokines ? activate keratinocytes and other inflammatory cells? rapid turn over ? **hyper-keratosis & parakeratosis.** | Well demarcated Erythematous plaques with silver scaling.<br>mild itching.<br>Typically on elbows, knees, scalp, lower back.<br><br>Nail changes: onycholysis, subungual hyperkeratosis, pitting nails, beau's lines, splinter hemorrhages.<br><br>Psoriatic arthritis: DIP, hands, feet, ankles, spondylitis, sausage-shaped digits, telescoping (A. mutilans)<br><br>Koebner's phenomenon: new plaques in areas of skin trauma. | -Topical: emollients, coal tar, **vitamin D analogues**, **steroids**, salicylic acid..<br><br>-**Phototherapy:** UVB/ PUVA.<br><br>- systemic: **Immunosupressants** (MXT, cyclosporine, acitretin), **biological** (infliximab, adalimumab, etanercept). |

| Other types of Psoriasis | |
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| -Guttate psoriasis: | Widespread small plaques on trunk and limbs, usually in adolescents, preceded by strep throat. Usually resolves completely (acute form). |
| -Palmoplantar pustular psoriasis (PPPP): | Monomorphic yellowish pustules on palms and soles ? brown color, association with smoking. |
| -Generalized pustular psoriasis: | severe unstable form, may be precipitated by steroids. |
| -Flexural psoriasis (inversa): | erythema in areas of skin folds. |
| -Napkin psoriasis | |
| -Erythrodermic psoriasis: | -Life-threatening, erythema affects nearly all the skin. Scaling is absent. Triggers: withdrawal of |

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| | systemic steroids, infection, excessive alcohol, antimalarial drugs, lithium, low calcium.<br>Increased cutaneous blood flow & fluid loss, leading to: Heart failure, Hypothermia, Dehydration, Low protein & edema, Secondary infections, Death. |

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| Urticaria (hives) | Type I hypersensitivity.<br>Mast cell degranulation with release of histamine and other proinflammatory mediators increased capillary permeability and transient leakage of fluids into surrounding tissue and development of wheal (dermal edema).<br>In 50% of patients with ordinary urticaria no underlying cause is found.<br>Possible triggers of acute urticaria include infections, vaccinations, medications and food. | **Pruritic** raised wheels (smooth red papules or plaques) lasting minutes or hours (<24 hrs).<br><br>If >24 hrs, painful, resolve with bruising urticarial vasculitis.<br><br>-Cholinergic urticaria: pinhead-sized wheals with a red flare around them, following a warm shower/bath, or after exercise.<br>-Solar urticaria: sunlight causes an acute urticarial eruption.<br>-Pressure urticaria: at areas of pressure (soles, hands, buttoks).<br><br>Diagnosis: dermatographism,<br>Skin biopsy if suspecting urticarial vasculitits. | -identify possible triggers.<br>-Oral H1 antihistamine (e.g; cetirizine or loratadine)<br>-if severe: combination of H1 blockers + H2 blockers + leukotriene antagonists (montelukast).<br><br>- oral corticosteroids for very severe eruptions, for urticarial vasculitis.<br><br>Omalizumab (anti-IgE): for chronic refractory urticaria. |
| Angioedema | Type I hypersensitivity.<br>May occur with urticaria.<br>swelling of the subcutaneous tissues | Deep swelling of subcutaneous tissues and/or mucous membranes. Usually **painful.** | -identify possible triggers.<br>-Same as urticaria |

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| | due to increased vascular permeability.<br><br>Allergy to medications (ACE, NSIADS, bupropion, statins or PPIs) may be the cause. | Hereditary angioedema (C1 esterase deficiency)<br>: recurrent episodes of angioedema without urticaria, may be associated with GI symptoms and life-threatening laryngeal edema.<br>Diagnosis: C1 esterase and C3 level if suspecting hereditary angioedema. | |
| Alopecia areata | autoimmune condition affecting hair follicles causing hair loss. | Patchy alopecia areata (most common pattern): Well-demarcated single or several round/oval patches of normal-appearing skin.<br><br>Characteristic "exclamation point hairs" may be observed.<br><br>DDx: tinea capitis, telogen effluvium, androgenic alopecia | -Intralesional corticosteroids<br>-Topical steroids<br>-Systemic steroids |

| Bacterial skin infections | | | |
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| | Microorganism | Clinical | Treatment |
| Impetigo | Staph aureus or strep pyogenes | Superficial skin infection (epidermis)<br>develops rapidly into clusters of pustules and vesicles which break down into the classic honey colored crusts.<br>Bullous lesions occur with staph strains that produce exfoliative toxins A/B. | -Antiseptic<br>-Topical antibiotic (e.g. fusidic acid, mupirocin).<br>-systemic antibiotics (flucloxacillin or erythromycin): for bullous impetigo or severe widespread nonbullous impetigo. |

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| | | *Highly contagious. | |
| Folliculitis | Staph aureus<br><br>Hot tub folliculitis ? pseudomonas aeruginosa (contaminated water) | pustule and erythema around the follicular orifice which may be associated with mild irritation.<br>Deeper folliculitis is characterized by abscess formation, boils, furunculosis, carbuncle | |
| Erysipelas | Strep pyogenes | Infection of upper dermis. Develop rapidly as red, shiny, raised, spreading plaque with a well-demarcated edge. Most commonly affect the face and lower legs. | Oral amoxicillin or roxithromycin.<br>IV benzyl penicillin if severe.<br><br>Prophylactic penicillin V for recurrent attacks |
| Cellulitis | Staph aureus or Strep pyogenes | Infection of deeper dermis and subcutaneous tissue. develops slowly and has a poorly defined margin and marked regional lymphadenopathy. The lower leg is the most common site affected.<br>Patients may have fever and general malaise. | Oral amoxicillin or roxithromycin.<br>IV benzyl penicillin if severe. |
| Necrotizing fasciitis | Polymicrobial (anaerobes, staph, strep, others) ? in diabetics, immunocompromised.<br><br>Or monomicrobial (Strep pyogenes) ? in healthy people following | Infection of the deeper tissue causes dusky purplish erythema associated with extensive necrosis.<br>Pain out of proportion of physical exam, followed by anesthesia. Crepitus from methane and CO2 production | Urgent surgical debridement and broad spectrum antibiotics |

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| | skin injury. | | |
| Staphylococcal scalded skin syndrome (SSSS) | of S. aureus that produce exfoliative toxins A/B (target desmoglein 1 ? intraepidermal splitting) | Generalized cutaneous erythema is followed by widespread superficial blistering (Nikolsky sign positive) and exfoliation which may be most striking in the flexures. Occur in infants and children < 5 years | systemic antibiotics to treat Staphylococcus. If nonresponsive ? MRSA coverage. |
| Ecthyma | Strep pyogenes | small pustules that have adherent crust and underlying ulceration, most commonly occur on the lower legs of children and elderly people who live in humid climates. Lesions usually heal slowly with scarring. | |
| Erythrasma | Corynebacterium minutissimum | superficial scaling and mild inflammation, with a reddish-brown discoloration that affects the flexural skin sites, particularly the axilla and groin. Under Wood's ultraviolet light the affected skin fluoresces pink | oral erythromycin |