NOTES
DERMATITIS & ECZEMA

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Inflammatory skin disorders
- Immune-mediated skin damage

SIGNS & SYMPTOMS

- Rashes
  - Pruritus (itching), burning, pain

DIAGNOSIS

LAB RESULTS
- Skin biopsy, blood tests

OTHER DIAGNOSTICS
- Rash
  - Appearance, distribution

TREATMENT

MEDICATIONS
- Corticosteroids
- Immunosuppressants

ATOPIC DERMATITIS (ECZEMA)

osms.it/atopic-dermatitis

PATHOLOGY & CAUSES

- Allergic, inflammatory skin condition
- Common for children; may affect adults
- Associated with elevated serum IgE levels
  - *Atopy:* predisposition to IgE antibody release after trigger exposure

TYPES

Type 1 hypersensitivity
- Epidermal barrier dysfunction
  - Skin barrier defects (e.g. filaggrin mutation) → antigen entry → inflammatory cytokines

Type 4 hypersensitivity
- Primary immune dysfunction
  - T cell subset imbalance → Th2 predominance → increased inflammatory cytokine production (IL-4, 5, 13) → increased release of IgE from plasma B-cells, recruitment of mast cells, eosinophils

RISK FACTORS
- Family history of atopy (eczema, asthma, allergic rhinitis)
- Environmental allergen sensitivities
- Loss of function mutation in filaggrin gene (skin barrier function)

- Higher incidence in urban populations, high-income countries
- Low levels of early life exposure to endotoxin (immunogenic component of gram-negative bacteria)

COMPLICATIONS

- Skin infections
  - *Staphylococcus aureus* common commensal organism → impetigo
- Eczema herpeticum
  - Rapid spread of herpes simplex virus on affected skin
- Social stigma, anxiety

SIGNS & SYMPTOMS

- Acute
  - Pruritic erythematous papules, vesicles with exudate, crusting
- Chronic
  - Dry, excoriated erythematous papules with scaling; lichenification (hyperplasia)
- Dry skin
- Pruritus → chronic scratching → skin thickening, increased infection risk
- Cutaneous hyperreactivity to environmental antigens/stimuli (e.g. stress)
- 0–2 years old
  - Erythematous, pruritic, scaly, crusted lesions +/- vesicles, serous exudate
  - Extensor surfaces, cheeks, scalp
- 2–16 years old
  - Lichenified plaques (thickened epidermis)
  - Flexural distribution (e.g. antecubital, popliteal fossae); volar aspect of wrists, ankles, neck
- Adults
  - Localized lichenified plaques
  - Flexural surface involvement
  - Uncommonly involves face/neck/hands

DIAGNOSIS

LAB RESULTS

- Elevated level of Serum IgE

OTHER DIAGNOSTICS

- Morphology, distribution of lesions

United Kingdom working group atopic dermatitis criteria

- Mandatory
  - Evidence of pruritic skin with rubbing/scratching
- ≥ three following criteria
  - Skin crease involvement (antecubital fossa, popliteal fossae, neck, around eyes, ankles)
  - History/first degree relative with asthma/hay fever
  - Dry skin in past year
  - < two years old before symptoms arose (not applicable to children < four years old)
  - Visible dermatitis of flexural surfaces (< four years old → examine cheeks, forehead, outer aspects of extremities)

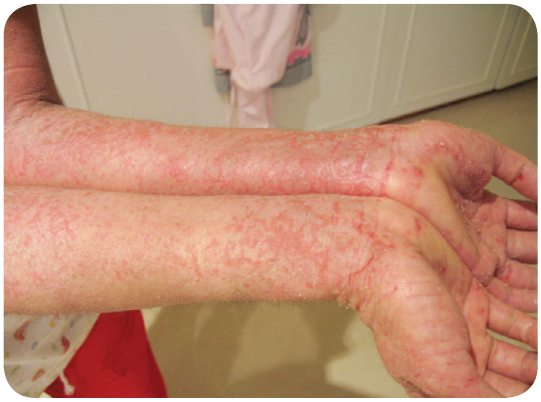

Figure 2.1 Atopic dermatitis affecting the flexural surfaces of the forearms.

TREATMENT

MEDICATIONS

- Control pruritus
  - Antihistamines
  - Topical calcineurin inhibitors (tacrolimus ointment, pimecrolimus cream)
  - Antibiotics to treat associated skin infections
- Immune suppression
  - Topical → systemic corticosteroids

- Topical calcineurin inhibitors
- Oral cyclosporine
- Dupilumab (IL-4 receptor antagonist)

OTHER INTERVENTIONS

- Reduce exposure to environmental allergens
- Avoid triggers
  - Heat, low humidity
- Manage stress/anxiety
- Maintain skin hydration
  - Thick, unscented creams with low water content/ointments without water
  - Apply after bathing/hand washing
  - Avoid lotions with high water/low oil content (evaporation dries out skin, triggers outbreak)
- Control pruritus
  - Prevent scratching; keep fingernails short (esp. young children)

CONTACT DERMATITIS

osms.it/contact-dermatitis

PATHOLOGY & CAUSES

- Inflammation of skin after contact exposure to allergens/irritants
- Localized
- Exposure to foreign substance triggers immune response
- *Most common form:* irritant contact dermatitis

CAUSES

- Exposure to irritant (irritation may be mechanical/chemical/physical)
  - *Acute:* strong irritant
  - *Chronic:* recurring exposure to weak irritant
- Detergents, surfactants, extreme pH, organic solvents, water
  - *Altered epidermal barrier function:* Fat emulsion → defatting of dermal lipids → cellular damage to epithelium → DNA damage, transepidermal water loss → cytotoxic cell damage → cytokine release from keratinocytes → activation of innate immunity
- Plants with spines/irritant hairs
- Low humidity
  - Skin loses moisture more easily

Allergic contact dermatitis

- *Anacardiaceae* family plants
  - Poison ivy, poison oak, poison sumac
- Nickel, fragrances, dyes
  - *Induction phase:* immune system primed for allergic response to antigen
  - *Elicitation phase:* contact allergens are typically haptens → small, can cross stratum corneum of skin to associate with epidermal proteins → form complete reactive antigen → dendritic cells recognise antigen → internalise antigen, transport to lymph nodes → present to T lymphocytes → trigger immune response → cell mediated immune response (Type IV delayed hypersensitivity) → memory cells remain within skin. Future exposure → triggers memory cells → immune response (cytokines, chemokines, TNF, lymphocytes, granulocytes migrate)

RISK FACTORS

- Age
  - *Infants:* highest risk
  - *> 65 years old:* lowest risk
- Body site exposure
  - Difference in thickness of stratum corneum, barrier function
  - Face, dorsum of hands, finger webs are prone to irritation
- Atopy

- Chronically impaired barrier function
- Occupational exposure
  - Continuous moisture exposure, repeated cycles of wet-to-dry from frequent handwashing
- Allergic contact dermatitis
  - Occupation (health professionals, chemical industry, beauticians, hairdressers, machinists, construction)
  - Increases with age
  - History of atopic dermatitis

SIGNS & SYMPTOMS

- Erythematous rash (can develop ≤ 72hrs after exposure)
- Vesicles/bullae/wheals occur at exposure site
- Glaze/parched/scaled presentation
- Scaling, hyperkeratosis, fissuring
- Itching (favors allergic etiology); burning (favors irritant)

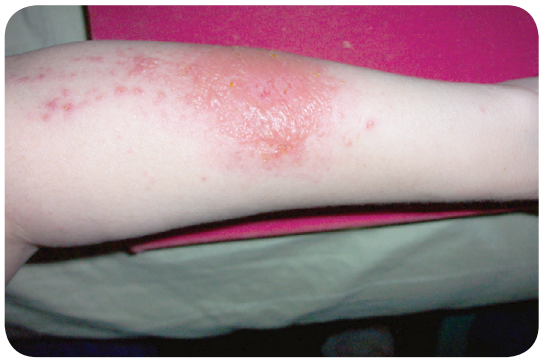

Figure 2.2 Contact dermatitis secondary to poison ivy exposure.

DIAGNOSIS

OTHER DIAGNOSTICS

- History of possible exposure to irritant/allergen
- Patch allergen testing

TREATMENT

MEDICATIONS

- Pruritus
  - Calamine lotion
- Mild topical corticosteroid (hydrocortisone)
- Oral antihistamine
- Allergic contact dermatitis
  - High potency topical corticosteroids
  - Oral corticosteroids
  - Topical calcineurin inhibitors (tacrolimus/pimecrolimus)
  - Systemic immunosuppression (azathioprine, mycophenolate mofetil, cyclosporine)

OTHER INTERVENTIONS

- Remove/avoid trigger
- Treat blistering
  - Cold compress
- Avoid scratching
- Retain moisture, protect skin
  - Barrier cream (e.g. zinc oxide)
- Irritant contact dermatitis
  - Mild acidic solutions (e.g. acetic acid) may neutralize alkali irritants/vice versa
  - Emollients (e.g. Aquaphor)
  - Gloves
- Allergic contact dermatitis
  - Phototherapy (narrow band UVB radiation)

SEBORRHOEIC DERMATITIS

osms.it/seborrhoeic-dermatitis

PATHOLOGY & CAUSES

- Sebaceous gland-centered skin inflammation
- Response to fungal antigens/irritants
- Chronic/relapsing
- Typically mild form of dermatitis

CAUSES

- Occurs in sites with greater density of sebaceous glands
- Not a disease of sebaceous glands, nor increased sebum production
- Suspected connection to lipid-dependent fungal genus *Malassezia*
  - Immune response to fungus
  - Local irritants produced by fungus
- Children
  - Nutritional deficiencies of biotin, pyridoxine (vitamin B_6), riboflavin (vitamin B_2)

RISK FACTORS

- Age (biphasic incidence: 2–12 months of age to adolescence; adulthood: peaks 30s–40s)
- Hyperandrogenism
- Biological males > biological females
- HIV
- Parkinson's
- Stress
- Cold, dry weather
- Sleep deprivation
- Poor general health

SIGNS & SYMPTOMS

- Scaling erythematous plaques
- Scales yellow, oily in appearance

Distribution

- Areas containing significant number of sebaceous glands
  - External ear, center of face, upper trunk, areas where skin rubs together
- Scalp
  - *Infants:* aka cradle cap; self-resolving
  - *Adults:* aka dandruff (pityriasis sicca); mildest form
  - Fine, white scaliness without erythema +/- pruritus
  - *Severe cases:* inflammation; patchy orange plaques with yellow, oily scales (pityriasis steatoides); may progress to oozing/crusting fissures affecting outer canal, concha of ear (vulnerable to superinfection)
- Face
  - Forehead, eyebrows, glabella, nasolabial folds; may affect cheeks/malar area in butterfly distribution
  - Frequently affects areas of facial hair distribution

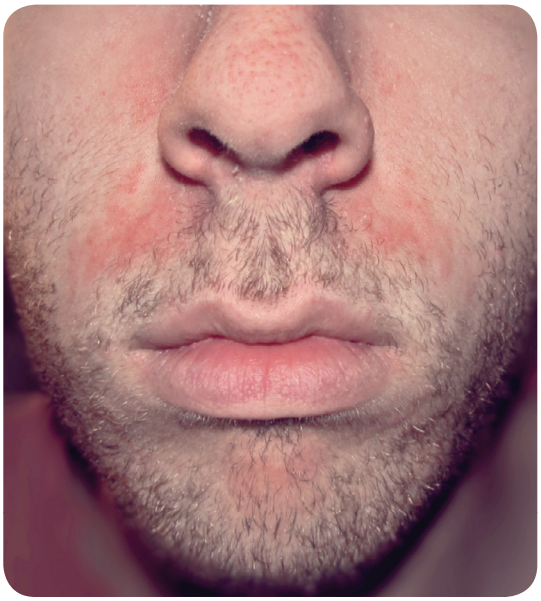

Figure 2.3 Seborrhoeic dermatitis affecting both nasal folds.

- Periocular
  - Blepharitis, free margin redness
  - Yellow crusting between lashes
  - Can occur in isolation/part of larger distribution
- Trunk (five distinct patterns of distribution)
  - *Moist, skin-contact regions:* axillae, inframammary folds, umbilicus, genitocrural
  - *Petaloid pattern:* fine, scaling plaques over sternum/interscapular
  - *Annular/arcuate:* round, scaly plaques, may have hypopigmented central clearing
  - *Pityriasiform pattern:* mimics pityriasis rosea, 5–15mm oval, scaly lesions along lines of skin tension
  - *Psoriasiform pattern:* large, rounded erythematous plaques with thick scales

DIAGNOSIS

OTHER DIAGNOSTICS

- History, appearance, distribution

TREATMENT

MEDICATIONS

- Topical antifungals
- Antifungal shampoo
- Topical corticosteroids
- Topical calcineurin inhibitors
- Oral antifungals
- Antiandrogens
  - Reserved for individuals for whom feminization/male infertility is unproblematic
  - *Sexually-active, uterus-bearing people:* combine with contraception to avoid risk to fetus

OTHER INTERVENTIONS

- Cradle cap
  - Apply emollient (petroleum jelly, vegetable oil, baby oil) to scalp overnight to loosen scales → remove scales with soft toothbrush
  - Frequent shampooing with mild, non-medicated baby shampoo → remove scales with soft toothbrush
  - Extensive/persistent cases → medical therapy
- Topical
  - Coal tar shampoo/ointment