NOTES PAPULOSQUAMOUS DISORDERS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Heterogeneous skin disorders; scaly papules, plaques
 - Papule: circumscribed, solid elevation of skin < 1cm/0.39in
 - Plaque: broad papule/confluence of papules ≥ 1cm/0.39in
 - Scale: dry/greasy laminated masses of keratin

CAUSES

Inflammation

SIGNS & SYMPTOMS

See individual disorders

DIAGNOSIS

OTHER DIAGNOSTICS

Rash patterns

TREATMENT

May spontaneously resolve

MEDICATIONS

- Topical (e.g. corticosteroids), nonsteroidal anti-inflammatory drugs (NSAIDs), antihistamines
- Immunosuppressants, retinoids (e.g. acitretin)

OTHER INTERVENTIONS

Phototherapy, colloid baths

LICHEN PLANUS

osms.it/lichen-planus

PATHOLOGY & CAUSES

- Self-limiting chronic dermatosis
- Multifactorial pathogenesis
 - Environmental factors → genetic aberrations of immune system
 - CD8+ T cells respond to altered antigens in basal epidermidis/ dermoepidermal junction
 - Causal agent identified: lichenoid reaction (e.g. drugs)

- Chronic inflammation in mucosal lesions \rightarrow squamous cell carcinoma

SIGNS & SYMPTOMS

- Shiny, flat-topped, pink-purple, polygonal papules coalesce, form plaques with red scales
- Wickham striae (pathognomonic)
 - Interspersed grey-white lace-like pattern of lines

- Symmetrical peripheral distribution, esp. on flexural surfaces (e.g. wrists, elbows, ankles, shins)
- Severe pruritus
- Koebner phenomenon
 - Skin lesions induced by local trauma
- Nail involvement
 - 10% of individuals; subungual thickening/hyperpigmentation, thinning/ ridging/ grooving of nail plate, pterygium formation, onycholysis
- Mucosal involvement
- Oral mucosa
 - Asymptomatic/burning sensation,severe pain
 - Mostly bilateral, inner cheeks
 - Various types may coexist; oral variant of Wickham's striae, erosive/ulcerative, papular, plaque-like, atrophic, bullous
 - Possible secondary Candida infections
- Esophageal mucosa
- Dysphagia/odynophagia
- Genital mucosa (glans penis, vulva/vagina)
 - Lower urinary tract symptoms, dyspareunia, itching in individuals who are biologically female



MNEMONIC: 6 Ps

Clinical presentation of lichen planus Planar Purple Polygonal Pruritic Papules Plaques

DIAGNOSIS

LAB RESULTS

- Typical microscopic features
 - Acanthosis: epidermidis thickening
 - Interface dermatitis: continuous infiltrate of lymphocytes along dermoepidermal junction → sawtoothing (dermoepidermal interface with zig-zag contour)

 Basal keratinocytes degenerate, appear like stratum spinosum cells (squamatization)/undergo necrosis, become incorporated into inflamed papillary dermis (Civatte bodies)

OTHER DIAGNOSTICS

- Rash pattern distinctive at skin examination
- Skin biopsy

Rule out secondary malignancies

TREATMENT

 Cutaneous lesions spontaneously resolve in nine months, longer for mucosal lesions
 Leaves area of hyperpigmentation

MEDICATIONS

- Reduce symptoms, shorten duration
- Antihistamines (pruritus), corticosteroids
- Retinoids, immunosuppressants

OTHER INTERVENTIONS

- Occlusive dressings
- Phototherapy
 - Ultraviolet A radiation



Figure 6.1 Lesions on the shins of an individual with lichen planus.



Figure 6.2 The histological appearance of lichen planus. There is a lymphocytic infiltrate at the junction between the dermis and epidermis which is known as interface dermatitis.

PITYRIASIS ROSEA

osms.it/pityriasis-rosea

PATHOLOGY & CAUSES

- Self-limiting acute dermatosis
- Unknown etiology; may be viral in origin, related to human herpesvirus 7 (HHV7)

SIGNS & SYMPTOMS

- Upper respiratory tract infection may precede rash
- "Herald patch"
 - Solitary oval red plaque, usually located on trunk
 - First skin lesion
 - Spreads with central clearing, fading in 2–10 days
- 1–2 weeks after herald patch, multiple round/oval pink (white individuals of European descent)/dark brown (black individuals of sub-Saharan African descent) plaques with central scale appear

- Trunk, neck, upper arms, thighs; "Christmas tree" progression
 - Across chest, then rib-line
- Pruritus
- Systemic
 - Low-grade fever, headache, nausea, fatigue



Figure 6.3 A herald patch is often seen at the onset of pityriasis rosea. It is a slightly raised, erythematous patch with superficial scaling.

DIAGNOSIS

LAB RESULTS

- Skin biopsy (rare)
- Microscopic features
 - Dyskeratosis: abnormal premature keratinization
 - Extravasated erythrocytes within dermal papillae

OTHER DIAGNOSTICS

- Rash pattern
 - Distinctive at skin examination

TREATMENT

• May spontaneously disappear in 6–8 weeks

MEDICATIONS

Antihistamines for pruritis

OTHER INTERVENTIONS

- May spontaneously disappear in 6–8 weeks
- Colloid baths for pruritis



Figure 6.4 The clinical appearance of pityriasis rosea on the torso of an adult male.

PSORIASIS

osms.it/psoriasis

PATHOLOGY & CAUSES

- Chronic dermatosis of skin, nails, joints
- Multifactorial pathogenesis
 - \circ Environmental factors \rightarrow genetic abnormalities of immune system
 - CD4⁺ TH1, TH17, CD8⁺ T cells collect in epidermis, secrete cytokines (e.g. IFN-gamma, TNF-alpha, IL-17, IL-22), growth factors → abnormal microenvironment ("cytokine soup") accelerates keratinocyte proliferation → defective keratinization, epidermal thickening
- Unpredictable progression with

spontaneous remissions, sudden exacerbations (e.g. may worsen in winter lack of sun, humidity)

- Skin abrasion, infection, drugs (e.g. lithium, beta blockers, chloroquine), psychosocial stress → exacerbations
- 10–15% of individuals develop psoriatic arthritis
 - Inflammatory cells in joint tissue \rightarrow synoviocyte proliferation
 - Surrounding connective tissue also involved (e.g. enthesitis)

TYPES

- Plaque psoriasis, AKA vulgar psoriasis; 90%
- Guttate (eruptive), inverse (flexural), pustular, erythrodermic

SIGNS & SYMPTOMS

- Plaque
 - Pink, salmon-colored papules/plaques covered by loosely adherent silver-white scales
 - Any area of body, esp. extensor surfaces (e.g. knees, elbows), lumbosacral area, scalp, glans penis
 - Itching is mild/absent
- Nail involvement in 30% of individuals
 - Subungual thickening
 - Yellow-brown discolorations of nail plate (resembling oil slicks)
 - Crumbling/ridging/pitting of nail plate
 - Onycholysis: separation of nail plate from bed
- Guttate (eruptive)
 - Drop-like appearance, associated with group A streptococcus
- Inverse (flexural)
 - Skin folds
- Pustular
 - Blisters filled with non-infectious pus
- Erythrodermic
 - Total body inflammation, skin exfoliation, severe itching, swelling, pain; ability to regulate temperature, perform barrier functions impaired; possibly fatal
 - May develop from any type (e.g. plaque during corticosteroid rebound phenomenon)
- Auspitz sign
 - Pinpoint bleeding appears when scale removed
- Koebner phenomenon
 - Characteristic skin lesions induced by local trauma
- Psoriatic arthritis
 - Inflammatory arthritis: pain, red overlying area, swelling, hot to touch
 - Frequently occurs after onset of rash
 - Asymmetric peripheral oligoarthritis;

joints of hands, feet most affected, followed by sacroiliac bone, spine

- Fusiform swelling of digits (dactylitis); aka "sausage digits"
- Aggressive disease with joint damage, malformations not common

DIAGNOSIS

DIAGNOSTIC IMAGING

For psoriatic arthritis

X-ray

• Erosive changes, "fluffy" periostitis, presence of new bone formation

MRI

Inflammation in adjacent bone marrow and soft tissues

LAB RESULTS

- Skin biopsy (rare)
- Acanthosis
 - Epidermidis thickening
- Parakeratosis
 - Keratinization (retention of nuclei in stratum corneum)
- Neoangiogenesis with tortuous blood vessels below stratum corneum
- Accumulation of neutrophils in superficial epidermis (spongiform pustules), in stratum corneum (Munro microabscesses)
- Clinical diagnosis
 Psoriasis features, clinical pattern of joint involvement
- Confirmation
 - Elevated inflammatory markers, negative rheumatoid factor (RF), anticyclic citrullinated peptide antibody (anti-CCP)

OTHER DIAGNOSTICS

- Rash pattern
 - Distinctive at skin examination
- Differentiation from rheumatoid arthritis
 - Minority show polyarthritic pattern with no skin lesions; note asymmetry, distal interphalangeal joint involvement, mild joint destruction

TREATMENT

- No definitive cure
- Avoid triggers

MEDICATIONS

- Topical corticosteroids → antiinflammatory, antiproliferative
- Vitamin D derivatives (calcipotriene, calcipotriol) → limit keratinocyte proliferation
- Anthralin \rightarrow suppresses proliferation
- Combination therapy is most effective (e.g. betamethasone dipropionate + calcipotriene)
- Affected area > 10%, unsuccessful topical treatment, involves face, hands, genitals
- Immunosuppressant
 - Methotrexate, cyclosporine
- Systemic retinoids
 - ${}^{\rm o}$ Acitretin \rightarrow inhibits pro-inflammatory cytokines
- Biologic therapy
 - Anti-TNF (infliximab, etanercept, adalimumab), T-cells (alefacept), IL-12/23 (ustekinumab)
- NSAIDs, immunosuppressant/biologic therapy
 - For psoriatic arthritis

OTHER INTERVENTIONS

- Topical
 - \circ Coal tar \rightarrow inhibits cellular mitotic activity, proliferation
 - Moisturizers, emollients
- Phototherapy
 - Ultraviolet A radiation
 - Often combined with topical tar/ systemic acitretin/psoralen/methoxsalen
 - Immunosuppressive, antiproliferative



Figure 6.5 A large psoriatic plaque on the upper limb.



Figure 6.6 Psoriasis affecting the hand.