Skin Anatomy and Function

Functions of skin:
- Prevent water, electrolyte and protein loss.
- Temperature control.
- Prevent infection.
- Barrier to irritants and allergens.

Skin disease affects about one quarter of the population at any one time.

The skin has 2 layers:
- Epidermis – outer layer, physical and biological barrier to external environment. Prevents loss of water and maintains homeostasis. Basal cell layer then mainly layers of keratinocytes (stratum spinosum, stratum granulosum, stratum corneum – flattened dead cells with lipids). The lipid barrier is important to protect skin – loss is important in eczema.
- Dermis – hair follicles, sebaceous glands, sweat glands.

Basic Terms

To describe an eruption:
- Distribution – extensor (e.g. psoriasis), flexor (e.g. atopic eczema), symmetrical (endogenous), asymmetrical (exogenous), acral (hands, head, feet), sun exposed, central, contact with skin or jewellery (contact dermatitis).
- Configuration – how individual lesions are grouped together – serpiginous (creeping eruption), herpetiform, seeding (Koebner phenomenon along scar), annular (tinea, granuloma annulare), ring shaped lesions (erythema multiforme), round lesions (psoriasis, discoid eczema, pityriasis rosea – oval red lesions with scaly edge, impetigo).
- Morphology – individual spots

Discrete lesions (SECTOR):
- Size, shape, site, surface, symmetry
- Edge
- Colour, consistency
- Tenderness, tethering, translucency, temperature
- Other features
- Regional lymphadenopathy

Skin eruptions (SCORN):
- Site, size, shape, surface
- Colour, consistency
- Oral
- Regions including scalp
- Nails

Pigmented lesions (ABCDEF):
- Asymmetry
- Border – risk of malignant melanoma if irregular or indistinct
- Colour – haphazard, different shades
- Diameter
- Elevation
- Fitting in with other moles
Eruption – rash
Lesion – single small area of skin change
Macule – flat area of colour change
Papule – small palpable mass
Plaque – flat topped larger (>1.5cm) lesion
Nodule – larger papule
Scale – visible white loosening of outermost skin
Crust – golden skin deposit due to dried plasma
Weal – smooth surfaced dermal swelling
Vesicle – small papule filled with clear fluid
Pustule – small papule filled with pus
Bulla – large blister
Erosion – partial loss of surface epithelium
Ulcer – complete loss of surface epithelium
Lichenification – thickening of skin due to chronic scratching or rubbing with increased markings
Comedone – blocked pore
Induration – thickening of dermis and subcutaneous tissues
Purpura – purplish lesion from free red blood cells, non-blanching

Skin history and examination

History
Rash – duration, site of onset, spread and distribution of lesions, itching or pain, aggravating factors.
Previous treatment.
Medical conditions and medication.
Family history.
Occupation.
Pets.

Skin and systemic disease

Various systemic diseases can present with skin signs.

Diabetes:
− Infections
− Ulcers – neuropathic (sites of pressure, non painful)
− Necrobiosis lipodica – shiny brown or yellow atrophic plaques on shin, may be ulcerated, telangiectasia.
− Granuloma annulare – erythematous circular lesions with well demarcated raised border, generally on hands and feet.
− Diabetic dermopathy – brown pigmentation and atrophic macules, may be precipitated by trauma.
− Acanthosis nigricans

Endocrine and metabolic disease:
− Thyroid – thyroid acropathy (like clubbing), pretibial myxoedema (raised red nodules).
− Cushing’s – buffalo hump, round moon face, striae, skin atrophy.
− Adrenal deficiency – hyperpigmentation (especially palms and buccal mucosa) due to stimulation of melanocytes.
Notes on Dermatology

Hyperlipidaemia – xanthelasmata (yellow plaques on periorbital skin), xanthomata (yellow papules – eruptive, tuberous(FH) or tendinous (FH)).
- Gout – sodium urate crystal deposition, tophi.

GI disease:
- Erythema nodosum – IBD, also sarcoid, infections (strep), drugs (OC, sulphonamides), painful red nodules on lower leg (lymphocytic vasculitis), resolve after 2-4 weeks.
- Pyoderma gangrenousm – inflamed ulcerated nodules, dark border, gun-metal bluish border, rapid expansion, due to IBD, also RA and blood disorders.
- Celiac disease – dermatitis herpetiformis, itchy blistering excoriated lesions on buttocks and elbows, IgA deposits.
- Hereditary haemorrhagic teleangectasia – epistaxis, GI bleeding and AV malformations in lung, liver and CNS, see vessels and face and oral mucosa.
- Liver – pruritus, spider naevi, palmar erythema, porphyria cutanea tarda (blisters in sun-exposed area related to excess alcohol).

Renal:
- Vasculitis (Henoch Schonlein) – nephrotic syndrome precipitated by strep sore throat. Get purpuric lesions with fever, malaise, arthralgia, haematuria, abdo pain.

Malignancy:
- Breast cancer – peau d’orange, Paget’s, nipple inversion
- Dermatomyositis – ovarian, lung, colorectal, pancreatic, lymphoma. Heliotrope periorbital rash or linear erythema on joints, muscle weakness, nailfold haemorrhages.
- Acanthois nigricans – hyperpigmented velvety skin change in flexure surfaces, related to stomach adenocarcinoma.
- Superficial thrombophlebitis – GI malignancy
- Acquired ichthyosis – generalised dry skin, lymphomas

Skin and connective tissue:
- Lupus – spectrum of skin disease, macular erythema, photosensitivity, scarring alopecia.
- RA – nodules, palmar erythema, pyoderma gangrenosum, vasculitic lesions (Bywaters).
- Sarcoidosis – erythema nodusum, lupus pernio (red or brown papules and plaques on face and back), nodules, papules, plaques.
- Systemic sclerosis – shiny skin, small beaked nose and mouth, perioral furrowing, Raynaud’s.

Medical conditions causing itching:
- Iron deficiency anaemia
- Lymphoma
- Thyroid disease
- Chronic liver disease
- Chronic renal failure
- Malignancy

Life-threatening skin disease:
- Toxic epidermal necrolysis – drug reaction (NSAIDS, antibiotics, antiepileptics), 35% mortality. Stop drugs.
- Erythrodermic psoriasis
Notes on Dermatology

Non-melanoma skin cancer

Non-melanoma skin cancer is the commonest cancer. Incidence is increasing. Aetiology is thought to be due to cumulative sun and UV exposure.

Basal cell carcinoma

80% of NMSC. Pathology:
- Malignancy of basal cells.
- Mainly occurs on sun-exposed sites.
- No precursor lesions.
- Infiltrates skin in 3D.
- Slow growing, locally invasive, relatively benign and indolent.
- Low mortality but can be high morbidity due to extensive local destruction needing mutilating surgery e.g. on face.
- High risk BCC – midface and ears, >2cm, aggressive subtype, neglected, immunosuppressed, previous radiotherapy.

History and examination:
- Typical patient is fair haired and skinned and has spent a lot of time outside.
- Many clinical presentations (superficial, nodular, cystic, nodulo-ulcerated, sclerosing, keratotic, pigmented) – well-defined, erythematous, pearly flesh coloured papules, surrounding telangiectasia, central ulcer. Can be exophytic nodule with pearly appearance and central ulcer. Can bleed, crust, bleed etc. like non-healing sore. Can be fibrotic like scar. If neglected can erode through skin, muscle and bone.
- Usually <1cm diameter.
Diagnosis:
- Clinical from history and examination.
- Biopsy.
- Histology once removed.
- Differential for superficial BCC – nummular eczema, psoriasis, Bowen’s disease, tinea, mycosis fungoides.
- Differential for nodular BCC – intradermal naevus, sebaceous hyperplasia.

Treatment:
- Curettage and cautery – 95% 5 year cure rate. Best for small, well-defined primary BCC.
- Cryotherapy
- Surgical excision – 5mm clearance.
- Mohs micrographic surgery – examine sections to ensure all excised. Time-consuming, costly but 99% 5 year cure rate.
- Radiotherapy – no histology.
- 5-fluorouracil – low risk BCC, good if multiple e.g. on trunk.
- PDT.

**Squamous cell carcinoma**

Pathology:
- Malignant neoplasm of keratinocytes.
- Rising incidence, due to UV exposure. Also related to smoking, ionising radiation and immunosupression, XP.
- Can arise from sites of chronic skin inflammation, high rates of metastases.
- Precursor lesions – actinic keratoses, Bowen’s disease.
- 5 year survival is about 80%. 25% with metastases. Bad prognosis associated with depth, ear or lip, larger size, invasion.
- Recurrence or metastases tend to occur within 5 years

History and examination:
- Typically sun-exposed sites.
- Firm flesh-toned papule or nodule. Smooth, scaly surface, ulcerated, crusted, hyperkeratotic.
- Sore, painful, bleeds.
- Rapidly enlarging.
- Can be infected in which case malodorous and fungating.

Treatment:
- Surgical excision – clearance margin (4mm for low risk, more if larger or high-risk site).
- Curettage and cautery – small, primary, low-risk tumours.
- Radiotherapy to avoid extensive or mutilating surgery.

**Keratoacanthoma**

Small papule with rapid growth to symmetrical dome-shaped flesh-toned nodule with central core filled with keratinous material. Surrounding telangiectasia. Then get indolent plateau phase and spontaneous involution.

Predisposing factors – sun exposure, tar exposure, petroleum oils.

Difficult to differentiate from SCC so generally surgical excision.
Actinic keratoses
Pre-malignant lesions which can develop to SCC.
Due to long-term sun over exposure. Very common.
Clinical – small brown/pink scaly papules. Can be rough, sore or irritable.
Cannot treat all AK, some may disappear spontaneously. Possible treatments include cryotherapy, 5-fluorouracil, curettage and cautery, surgical excision (only if SCC suspected), PDT.

5FU – erythema, vesiculation, erosion, ulceration, necrosis, healing. Healthy skin is unharmed.

Bowen's disease
Intra-epidermal carcinoma in situ – has not penetrated basement membrane. When this occurs becomes SCC.
Long history, about 3% progress to SCC.
Clinical – asymmetric, slow growing, well demarcated scaly erythematous patch.
Many differential diagnoses. Establish by punch biopsy.
Treatment as for AK.

Pigmented skin lesions
Describe by ABCDE.
Hutchinson sign is pigmented extension into nail fold, related to melanoma under the nail.
Signs suggesting melanoma:
- Change in size.
- Change in shape – especially if irregular.
- Change in colour – especially if irregular.
- Development of new pigmented lesion.
- Also inflammation, crusting, bleeding.

Benign melanocytic lesions
Ephelides (freckles):
- Lightly pigmented macules. Well-demarcated, on sun-exposed areas, not mucous membranes. Uncountable.
- Increased melanin without increased melanocytes.
- Occur in childhood, especially fair, and fade with age. Fluctuate with UV exposure.

Lentigines:
- Epidermal hyperplasia with increased melanin.
- Darker, larger macules. Sun-exposed areas, darken with UV exposure but do not fade again. Countable.
- Occur in adulthood and persist.

Lentigo simplex:
- Occurs in childhood.
- No relationship to sun exposure. Can occur on mucous membranes.
- Due to increased melanocytes.
Acquired melanocytic naevi (moles):
- Increased basal melanocytes with downgrowths at dermo-epidermal junction.
- Compound naevus – thicker slightly lighter brown papule. Melanocytes at junction and within dermis.

Congenital melanocytic naevi:
- Benign proliferations of melanocytes in epidermis or dermis.
- Categorised by size as small (1cm), medium or large (>20cm).
- Larger and more raised than acquired naevi.
- Risk of melanoma in large naevi (12% lifetime risk).

Benign naevi that mimic melanoma:
- Blue naevus
- Dysplastic naevus – at risk of developing melanoma.
- Naevus spilus – light brown macule with darker papules in it.
- Non-melanocytic lesions – seborrhoeic keratoses, dermatofibroses, haemorrhage, pigmented BCC.

**Melanoma**
Malignant neoplasm of melanocytes.
Curable at an early stage by excision.
No treatment once metastasised.

Lentigo maligna:
- Neoplastic melanocytes in basal layer.
- May become invasive.
- Usually on face of elderly patients.

In situ melanoma:
- Neoplastic melanocytes throughout epidermis but not invasive.

Superficial spreading melanoma:
- Most common subtypes – adults, usually sun-exposed areas.
- Macule with irregular border or colour.
- Slow horizontal growth phase then rapid vertical growth phase forming nodule.

Nodular melanoma:
- Older patients, any body site.
- Blue-black or red nodule which develops rapidly. May be ulcerated.
- Less surrounding macular pigmentation.

Amelanotic melanoma – absent pigment.

Treatment:
- Rapid surgical excision – 1cm margin for every 1mm depth.
- Staging.

Prognosis depends mainly on depth – if in situ 100% 5 year survival, if <1.5mm 90%, if >3mm 40%.
Leg ulceration

Most ulcers are of mixed cause. Gravity and immobility are important factors in most ulcers. Venous ulcers are the most common (about 80%). Arterial ulcers are less than 5%. It is often difficult to distinguish these clinically. Typically venous ulcers are superficial and around the medial malleolus, whereas arterial ulcers are punched out, deeper, necrotic and on the foot. This can be determined by ABPI:
- For arterial insufficiency to be a likely cause of ulceration will be <0.8.
- Compression to treat venous ulcers is safe if >0.9.

**Venous ulcers**
Veins in the leg are divided into deep (inside muscles) and superficial.
Venous insufficiency results from:
- Varicose veins – valves don’t work in surface veins so blood from deep veins flows back to the foot.
- DVT – damages valves. Can’t treat except by compression.
- Deep venous incompetence – calf pump failure.
Chronic venous hypertension leads to skin changes:
- White cell trapping, microcirculatory failure, lipodermatosclerosis, atrophie blanche, haemosiderin deposits, ulceration.
Treat by compression and sometimes superficial venous surgery.

**Arterial ulcers**
Ulceration due to poor blood supply. Typically related to arteriosclerosis, diabetes or vasculitis. Patients may still have drainage problems, especially if they get ischaemia when leg is raised. Treat conservatively, by angioplasty or bypass surgery.

**Neuropathic and traumatic ulcers**
Commonest cause of initial ulceration. Poor healing is then due to arterial or venous insufficiency. Can by diabetic neuropathy or leprosy causing repeated injury.

**Inflammatory and infective ulcers**
Cellulitis.
Necrotising fasciitis.
Pustular psoriasis.
Vasculitis e.g. polyarteritis nodosum.
Pyoderma gangrenosum.

**Neoplastic ulcers**
Important to remember for non-healing ulcers.
BCC, SCC.
Lymphoma.

**General treatment**
Skin grafting – unlikely to work unless treat underlying pathology.
Patch testing to check for allergy to medication complicating ulcer.
Larval therapy – debridement, anti-bacterial, affects cell communication.
Can get varicose eczema around ulcer – treat with moderately potent steroid.
Ulcers are always colonised with bacteria. Only need to treat if strep, pseudomonas or obvious clinical infection.
**Eczema**

Eczema is the same as dermatitis. Dermatitis means inflammation of the skin. Eczema therefore involves:

- Vasodilation – erythema and heat
- Exudates – oedema
- Release of inflammatory mediators – pain, itch

Eczema is a common condition with increasing prevalence.

**Classification of eczema**

Endogenous – atopic, seborhoeic, asteatotic, gravitational, discoid.
Exogenous – contact dermatitis (irritant, infective), allergic.

Can also classify as acute or chronic.

**Clinical features**

All patients – pruritus, xerosis.

Some patients, especially children – perifollicular accentuation, keratosis pilaris (extensor surfaces), angular cheilitis, pityriasis alba (hypopigmented macules), Dennie-Morgan lines (folds of skin from lower eyelid related to atopy).

Due to scratching – lichen simplex chronicus (lichenification and pigmentation in skin creases), prurigo nodularis (thickened firm nodules).

Acute eczema:
- Dermal vessels dilate, epidermal oedema, inflammatory exudates.
- Leads to erythema, vesicles and exudates, oedema, pruritus, papules and plaques, serous crust, scaling.
- Scratching rapidly changes appearance.

Sub-acute eczema:
- Less oedema.
- Epidermal cell malfunction causes acanthosis (thickening of epidermis) and hyperkeratosis.
- Fewer vesicles.
- Scaling.

Chronic eczema:
- Acanthosis, hyperkeratosis, lichenification, fissures.
- Hyper-/hypo-pigmentation.
- Persistent inflammation.
- Fissures

**History and examination**

History – aggravating and relieving factors, other atopic disease, family history, occupation, sleep disturbance, diet, previous treatment, effect on patient.

Examination – extent, severity, location, infection, growth in a child.
Atopic eczema
Atopy refers to a genetic predisposition to eczema, allergic rhinitis and asthma. This is due to hypersensitivity to certain allergens e.g. pollen.
Atopic eczema – chronic itchy inflammatory disorder of skin.
Epidemiology – mainly children, 15-20% of children, increasing prevalence.
History – relapses and remission, begins in early infancy, tends to improve with age.
Site - face and exposed surfaces in infants, flexures in children, generalised in adults.
Aetiology – increased levels of IgE, positive skin prick test to environmental allergens and house dust mite. Often colonised by staph aureus. Some children have food allergies e.g. to eggs which exacerbate eczema.

Diagnosis – itchy skin plus 3 of:
- Personal or family history of atopy
- Visible flexural dermatitis
- Dry skin
- History of flexural skin involvement
- Early onset.

First-line treatment:
- General – education, soap substitutes to protect skin barrier function, emmollients, avoid irritants and allergens.
- Emollients – creams, ointments, bath oils. Need to be applied in downward stroking motion.
- Topical steroids – reduce inflammation, strength depends on age, site and severity. Can cause thinning. Use sparingly with finger-tip unit.
  - Mild – hydrocortisone acetate
  - Moderate – hydrocortisone butyrate
  - Potent – betamethasone
  - Very potent – clobetasone
- Occlusive bandaging e.g. tar, zinc paste, wet wraps. Soothe the skin, stop itching and scratching.
- Antihistamines – may help.
- Antibiotics – if infected eczema (bright red, exuding, pustules) e.g. flucloxacillin, erythromycin.
- Topical immunomodulators e.g. tacrolimus. Calcineurin inhibitors. Used for facial eczema or to reduce steroid use.

Second-line treatment:
- Oral steroids
- PUVA
- Cyclosporine A – monitor BP and renal function, risk of malignancy.
- Azathioprine.
- Diet, herbs, psychological interventions.
- Antibiotics or acyclovir if infected.

Complications:
- Infection – staph, strep, molluscum, HSV.
- Psychological morbidity
- Poor growth in children
- Treatment side effects e.g. skin thinning, striae, adrenal suppression, malignancy, allergic contact side effects.
- Eczema herpeticum – HSV colonises skin, painful erosions, need oral or IV acyclovir, can be very severe.
Seborrhoeic dermatitis/ eczema
Affects areas rich in sebaceous glands e.g. scalp, face, upper trunk, especially young men. Severe in HIV patients. Due to abnormal response to commensal yeast.
Causes erythema, scaling and dandruff.
Treat with ketoconazole shampoo, imidazole and hydrocortisone.

In infants causes ‘cradle cap’. Treat with emollients, hydrocortisone, antifungal.

Asteatotic eczema
Occurs when skin fat content decreases (elderly, dry climate, over-washing, detergent). Skin dries out so get ‘crazy paving’ appearance due to loss of barrier function.
Sparres face and hands, rarely get vesicles or lichenification.
Treat with topical steroids and long term emollients.

Gravitational eczema
On lower legs due to chronic venous hypertension e.g. varicose veins, previous DVT, obesity. Get oedema, discolouration due to haemosiderin deposit, vesicles, induration, scaling, lipodermatosclerosis.
Treat with compression hosiery, emollients, topical steroids.
At risk of ulceration.

Discoid (nummular) eczema
Well demarcated disseminated chronic coin shaped scaly plaques, often infected.
Typically on limbs of middle aged men.
Needs potent steroid.

Contact dermatitis
Irritant – substances affect physiological integrity of the skin by direct chemical damage e.g. alkalis, detergents. Typically affects hands e.g. hair dressers, nurses etc. Atopic individuals are most susceptible.

Allergic – type IV hypersensitivity reaction to exogenous allergen. Distribution reflects contact with allergen. Use patch testing to identify allergen – nickel is common.

Treat by avoiding irritant or allergen e.g. gloves, emollients and topical steroids.

Psoriasis
Common chronic inflammatory disorder of the skin, typically affecting extensor surfaces.
Inflammation leads to increased turnover giving thickened skin with red scaly plaques.
In about 10% of cases is associated with arthritis.

Epidemiology – tend to develop either before 20 or in 50s. Genetic and environmental factors. Aggravated by trauma (Koebner phenomenon is new lesions appearing around local injury e.g. scratch, scar), infection (especially strep), endocrine factors, drugs (B blockers, steroid withdrawal, antimalarials), stress, alcohol, smoking.

Differential diagnosis – fungal infection, mycosis fungoides, seborrhoeic dermatitis.
Types of psoriasis

Plaque:
- 90% of cases.
- Typically salmon pink, well demarcated, symmetrical, erythematous scaly plaques with loosely adherent white scales.
- Removal of scales gives pinpoint bleeding (Auspitz sign).
- Can occur anywhere on body (including nails giving pitting) but typically extensor surfaces. Koebner phenomenon is new lesions occurring on trauma.

Guttate:
- Rain drop lesions, less scaling.
- Mainly on trunk.
- Occurs after strep throat infection especially in children.
- Self-resolving after 3 months but often heralds plaque psoriasis.

Pustular:
- Generalised – acute onset sterile pustules on background erythema. Painful with systemic malaise and fever. May be precipitated by steroid withdrawal.
- Localised (palmoplantar) – older people especially smokers, painful but no systemic features.

Erythrodermic:
- Affects >90% body area.
- Needs admission due to risk of hypothermia, high output cardiac failure, sepsis, dehydration, protein loss.
- Could be triggered by drug reaction on top of existing psoriasis.

Flexural:
- Scale removed by friction.
- Colonised by yeast and bacteria.
- Shiny, glistening well demarcated lesion.

Psoriatic arthropathy (5 different types):
- DIP joints
- Rheumatoid-like (sero-negative)
- Oligoarticular – knees, ankles, MCPs, assymetrical
- Spondyloarthropathy – spondylitis, sacroilitis
- Arthritis mutilans

Treatment
Remove triggers e.g. strep infection, drugs, alcohol, stress.

Topical:
- Tar – messy, folliculitis, irritation. Cleaner preparations now available.
- Dithranol – inhibit keratocyte proliferation. Messy, staining, irritation.
- Salicylic acid – keratolytic for thick plaques.
- Vit D analogues e.g. calcipotriol – stimulate differentiation, inhibit DNA synthesis and proliferation. Good for outpatients. Risk of hypercalcaemia.
- Steroids – good for facial psoriasis. Risk of rebound when stop, skin atrophy.
- Trimovate for flexural disease – steroid, antifungal, antibacterial.
Phototherapy:
- PUVA – UVA and psoralens (oral or topical, photosensitising).
- UVB – may have fewer side effects.
- Problems – erythema, pruritus, nausea, risk of skin malignancy.

Systemic:
- Methotrexate – folic acid antagonist, stops protein synthesis especially in rapidly dividing cells. Side effects of marrow suppression, liver fibrosis, teratogenic, drug interactions.
- Retinoids e.g. acitretin – suppress DNA synthesis and cell differentiation. Side effects include teratogenic, hyperlipidaemia, hepatotoxicity, dry skin.

Acne

Epidemiology
Very common – about 40% teenagers. Generally starts at puberty, 85% of sufferers aged between 12 and 24, 10% women 3% men continue until age 44. High risk with endocrine disorders and XXY.

Pathogenesis
Disorder of pilosebaceous unit:
- Comedogenesis (blocking of ducts) – comedones can be open (black heads) or closed (white heads). This may be due to abnormalities in sebum or keratinocytes, androgenic effect or cytokines.
- Seborrhoea – increased sebum production due to abnormalities in sebum or response to androgens.
- Infection with propionobacteria acnes – colonises pilosebaceous duct and thrives in sebum.
- Inflammation – due to P. acnes, cytokines or changes in epidermal barrier function.

Clinical signs
Generally face, chest and back. Lesions of acne:
- Inflammatory - pustules, papules, macules, nodules, cysts.
- Non-inflammatory - comedones.
- Scarring

Severity grading:
- Mild – just papules and some pustules, no inflammation or scarring.
- Moderate – some small nodules and cysts, some inflammation, no scarring.
- Severe – lots of pustules, mainly small nodules, inflammation and scarring.
- Very severe

Diagnosed clinically. If sudden onset, severe or associated with hirsuitism or menstrual disturbance, consider underlying endocrine disorder. Differential diagnoses – acne rosacea, peri-oral dermatitis, folliculitis, drug eruption.
**Treatment**

**Topical:**
- 1st line if mild or moderate disease.
- Benzoylperoxide – stings, bleaching, good combined with antibiotics, no resistance.
- Topical antibiotics – erythromycin, clindamicin, resistance is a problem as monotherapy.
- Retinoids

**Oral (if severe, widespread, scarring or oral fails):**
- Tetracyclines – skin and teeth staining, not if pregnant or breast feeding, post-inflammatory hyperpigmentation (especially minocycline).
- Erythromycin – resistance, GI side effects, safe in pregnancy.
- Hormone modulation – venous thromboembolism, only women.
- Isotretinoin (roaccutane).

**Treatment of scars:**
- Excision
- Laser resurfacing
- Intralesional steroid for keloid scars

**Isotretinoin**

Very effective.
4-6 month treatment.
Up to 30% relapse.

**Actions:**
- expulsion of mature comedones
- inhibition of comedone formation
- anti-inflammatory
- increases penetration of other agents
- decreases sebum production.

**Side effects:**
- needs specialist supervision
- teratogenic - need 2 methods of effective contraception for 3 months before and after
- irritation
- photosensitivity
- dry skin
- impaired liver function
- increased TG
- arthralgia
- decreased night vision
- initial flare

**Indications:**
- severe acne
- active acne with scarring
- resistant disease
- relapses after antibiotic treatment
- psychological consequences

Other acne variants – tropical, steroid, occupational (oils and tars), endocrine (Cushing’s, PCOS), infantile, drugs (anabolic steroids, phenytoin, isoniazid, androgens).
Bacterial and viral infections

**Commensal bacteria**
Present on the skin but not causing disease. Can cause disease and found in cultures. Micrococci, corynebacterium, propionibacteria, staph. Vary with age, site, humidity and antiseptic use.

Can become pathogenic if:
- Change in host surface, especially eczema or trauma.
- Interaction between commensals e.g. changed by antibiotics.
- Change in immune system – intercurrent illness, immunosupression.

Can get a primary infection or a secondary infection of underlying skin disease e.g. eczema, wound, ulcers.

Common pathogenic bacteria – staph, strep, pseudomonas (especially leg ulcers and green nails), corynebacteria, mycobacteria (lupus vulgaris, granulomatous lesions), spirochaetes.

**Staph aureus**
This is always pathogenic when cultured.
Coagulase positive.

Causes disease by direct invasion of epidermis and hair follicles and toxin production.


Hair follicle infections – folliculitis, furuncles (deeper), carbuncles (multiple adjacent follicles).

Ecthyma – infection below crust.

Toxins cause scalded skin syndrome, toxic shock and scarlatina.

Treatment:
- Topical – muprocin, fusidic acid
- Oral or IV - flucloxacillin

**Strep pyogenes**
Always pathogenic.
Many groups – Lancefield A is strep pyogenes.
Tends to be more acute onset and rapid spread than staph.

Cellulitis – pain, redness, warmth, typically lower leg and unilateral. Can blister or become haemorrhagic.

Erysipelas – sharply defined superficial infection with oedema involving face or leg, systemic symptoms e.g. fever.

Toxins cause scarlet fever and toxic shock-like syndrome.

Hypersensitivity to antigens can cause various conditions following an infections including – erythema multiforme, vasculitis, glomerular nephritis, erythema nodosum, guttate psoriasis.

Treatment:
- No topical
- Oral – penicillin V, erythromycin
- IV – benzylpenicillin
**Viral infections**

HSV:
- Type 1 – genital, type 2 – extra-genital especially lips.
- Primary infections – stomatitis, gingivitis; in most people very mild.
- Secondary infections – clusters of painful vesicles.
- Can complicate eczema (eczema herpeticum), which is life threatening – lots of monomorphic papules and vesicles.
- Generally no treatment needed but can use topical or systemic acyclovir.

VZV:
- Primary infection – chicken pox – severe in adults, immunosuppressed. Can cause pneumonia, hepatitis, encephalitis.
- Treatment – acyclovir for serious chicken pox, early shingles or shingles in elderly (increased risk of neuralgia).

Pox virus:
- Molluscum contagiosum
- Common in children.
- Self-limiting pearly umbilicated papules containing white material which can be expressed.

HPV:
- Self-limiting warts, common in children.
- Can be severe if immunocompromised.
- Can promote SCC.
- Treatment – salicylic acid paints, Duck tape, cryotherapy, podophylin, imiquimod, curettage and cautery.

**Fungi and infestations**

**Cutaneous fungal infections**
Fungi usually cause superficial infections affecting keratinised skin, hair and nails. Systemic fungal infections can occur in immunocompromised.

Dermatophytes (ring worm, athlete’s foot):
- Very common.
- Invade and multiply in keratinised tissue.
- 3 types – microsporum, trichophyton, epidermophyton.
- They can be transmitted from people, animals or soil.
- Named tinea + body site (cruris = groin, unguium = nails, capitis = scalp).
- Give well-defined scaly red annular lesions with a clear bit in the middle. Itchy, can be inflamed.
- On palms get hyperkeratotic lesions. Nails become thickened and yellowed. On scalp gets scaly, alopecia, pustules, hyperkeratosis (leads to scarring alopecia so needs treatment).

Investigate by skin and nail scrapings and hair samples – microscopy and culture.

Differential diagnoses – dermatitis (nummular, seborrheic), psoriasis (tinea is less symmetrical and widespread), folliculitis, candidiasis, granuloma annulare.
Notes on Dermatology  Author: Liz Tatman

Yeasts:
- Candida pityrosorum – group including C. albicans. Normal commensal but can cause symptomatic infection with infancy, old age, pregnancy, diabetes, AIDS, antibiotic use, steroid use etc. Candidiasis can be oral (curd like deposit), vulvovaginitis (itchy, white discharge), onychomycosis (discolouration and pitting) etc.
- Malassezia or Pityriasis versicolor - hypo or hyper pigmented discoid lesions on trunk, face and flexures. May complicate seborrhoeic dermatitis. Treat by selsun lotion or antifungals.

**Treatment of fungal infections**

**Topical:**
- Localised lesion.
- Azoles e.g. ketoconazole – stop fungal cell wall formation. Metabolised by liver.
- Polyenes e.g. nystatin

**Systemic:**
- Nails, scalp, hair affected or widespread.
- Terbinafine.
- Azoles e.g. itraconazole. Side effects systemically e.g. nausea, vomiting and impaired liver function with ketoconazole.
- Griseofulvin – only drug suitable for children. Binds to keratin and prevents infection so have to use for a long time to replace all keratin (6wks for skin, 6 months for nails). Fungistatic. Causes photosensitivity.
- Nails need longer treatment.

**Skin infestations**

**Scabies:**
- Mite
- Human to human transmission.
- Clinical signs – excoriated papules, burrows, crusting if severe e.g. immunosupression.
  Eruption is a reaction to mite saliva and faeces.
- Typical sites – finger webs, flexures, breasts, axilla, feet.
- Treatment – permethrin, malathion. Need 2 treatments a week apart to kill hatched nymphs.
  Treat all contacts together and wash linen in hot water.
- May get post scabetic eczema.

**Head lice:**
- Spread by close contacts.
- Main symptom is pruritus.
- Treat by removal e.g. fine comb or drugs e.g. permethrin.

**Other conditions**

**Vitiligo**
White patches, maybe with hyperpigmented borders.
Itch in sunlight.
Associated with autoimmune disease.
Alopecia
Hair loss.
Alopecia areata – autoimmune, lose hair, eyebrows, axillary hair etc.

Acne rosacea
Chronic relapsing and remitting condition of unknown aetiology. May be associated with H pylori. Particularly occurs in fair-skinned people. Chronic flushing, fixed erythema, rhinophyma, telangiectasia, papules, pustules. Can cause eye problems e.g. blepharitis. Manage by avoiding sun exposure, topical metronidazole, oral tetracyclines.

Lichen planus
Itchy disorder. Flat topped purple papules on wrists, ankles, nails, mouth, genitals. Use topical steroids.

Pityriasis rosea
Preceded by ovoid red scaly patch with scaly edge. Self-limiting.

Pemphigoid
Autoimmune blistering disorder in old people. IgG act against basement membrane. Tense blisters on urticarated base. Treat by oral prednisolone.

Pemphigus
Autoimmune blistering disorder in younger people. IgG antibodies damage desmosomes. Affects oral mucosa. Treat by oral prednisolone.

Photosensitivity
Polymorphic light eruption – idiopathic disorder, common in young women. Light exposure gives itchy red papules, vesicles and plaques which improves with continued exposure. Porphyria cutanea tarda – reduced function of liver enzyme breaking down uroporphyrinogen. Get photosensitivity, hypertrichosis, hyperpigmentation and skin fragility. SLE Drug induced – especially thiazides, tetracyclines, sulfonamides, phenothiazines, NSAIDS.

Important Note
These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources. Disclaimer: no responsibility can be taken by either the author or publisher for any loss, damage or injury occasioned to any person acting or refraining from action as a result of this information.

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