2019-2020
Dermatology Handbook

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Course Description

2 weeks of Dermatology teaching including lectures, seminars, online modules, and practical experience of the dermatology ward and outpatient areas.

Week 1: Lectures 1-4

Week 2: Lectures 5, 8, 9. Seminars.

Assessment: Short quiz on the last day (Thursday). One case report (see syllabus for details). Seminars are graded. Final exam will be 50% of the mark.
The Structure & Function of the Skin

- The largest organ in the body
- Barrier to chemicals, infectious organisms
- One of the special senses: touch, pressure, vibration, heat, cold, pain
- Thermoregulation – heat & water control
- Vitamin D synthesis

Diseases are caused by:

- **External Factors**
  - Sunshine eg sunburn
  - Heat eg miliaria
  - Cold eg perniosis
  - Chemicals (allergens & irritants) eg eczema
  - Infections
  - Trauma eg ulcers, blisters
- **Internal Factors**
  - Psychological
  - Genetic
  - Internal disease
  - Drugs
  - Infections

Epidermis

- Stratified squamous epithelium
- Thickness varies 0.1-1mm
- Basement membrane
- Stratum basale
  - Single layer columnar cells
- Stratum spinosum
  - Keratinocytes
- Stratum granulosum
- Stratum corneum

Functions

- Barrier – prevents loss of interstitial fluid, barrier to harmful substances
- Vitamin D synthesis
  - 7-dehydrocholesterol in keratinocytes
  - Converted by sunlight to cholecalciferol
  - 25-hydroxylation in kidneys to active metabolite
- Melanogenesis
  - Melanocytes migrate from neural crest by 8 weeks’ gestation
  - Basal layer, hair bulb, retina, pia arachnoid
  - Melanin protects surrounding keratinocytes from harmful UVR
- Immune response
  - Langerhans Cells – present antigens to T lymphocytes in skin or local lymph nodes
- Sensation
  - Merkel cells – fine touch

Dermo-epidermal junction

- Basement membrane
- Stains with PAS on histology
- Functions:
  - mechanical support
  - adhesion of basal layer
  - filter of nutrients from dermis to epidermis

Dermis

Cells:
- Fibroblasts – synthesise collagen
- Phagocytes, lymphocytes, dermal dendritic cells, mast cells – immune function

Fibres:
- Collagen (70-80%)
  - 3 polypeptide chains in a triple helix
  - Every 3rd amino acid is glycine
- Reticulin
  - Fine collagen fibres
  - Around blood vessels and appendages (&fetal skin)
- Elastic fibres (2%)
  - Amorphous elastin core with surrounding elastic tissue microfibrillar component

Others:
- Ground substance: mainly Hyaluronic acid & Dermatan sulphate
- Muscles
  - Smooth muscle ie arrector pili muscles
  - Striated – facial expression muscles eg platysma
- Blood vessels
  - Thermoregulation (deep plexus & superficial plexus)
- Cutaneous lymphatics
- Nerves
  - Free sensory nerves – related to itch
  - Pacinian corpuscle - pressure
  - Meissner corpuscle – vibration, touch
Diagnosis of Skin Disease

History
- Duration
- Site of onset
- Evolution / spread
- Associated symptoms: itch, burning, pain
- Appearance: wet, dry, blisters, pustules
- Medications tried: otc / prescribed, topical / systemic
- Other symptoms: fever, weight loss, night sweats
- Past skin history
- Past medical history
  - Asthma, hayfever
- Medications
- Allergies
- Family history of skin disease or atopy
- Social history – smoking, occupation, travel
- Sun exposure – skin type, hobbies, work, travel, sunbeds, sunburn

Examination:
- Distribution
  - Sites affected
  - Localised or generalised
  - Check mouth, hair, nails
- Morphology
  - Macule
  - Patch
  - Papule
  - Nodule
  - Plaque
  - Vesicle
  - Bulla
  - Pustule
  - Abscess
  - Folliculitis
  - Furuncle
  - Carbuncle
  - Wheal
  - Excoriation
  - Fissure
  - Erosion
  - Ulcer
  - Petechia
  - Purpura
  - Ecchymosis
  - Haematoma
  - Scale
  - Crust
  - Keratosis
  - Sinus
  - Scar
• Atrophy
• Lichenification
• Stria
• Pigmentation

Nails
• Nail disorders can be isolated or as part of a skin condition
• Examination of the nails is as important as examining the skin
• Clubbing
• ONycholysis
• Longitudinal Ridging
• Onychogryphosis
• Longitudinal melanonychia
• Onychogryphosis
• Habit tic / median canaliform dystrophy
Inflammatory Skin Conditions

Psoriasis
• Inflammatory disease of the skin, nails and joints
• Associated with increased risk of cardiovascular disease
• Multifactorial disease with complex genetics
• Two peaks of inheritance:
  • Type 1 – 2nd/3rd decade, FH+
  • Type 2 – late adulthood

Genetics
• Polygenic inheritance
  • 1 affected parent – 14% risk of disease
  • 2 affected parents – 41% risk of disease
  • 0 affected parents, 1 sibling – 10% risk of disease
  • 70% concordance in monozygotic twins (20% in dizygotic twins)
• PSOR1 on 6p21 (HLA-Cw6 allele) – associated with Type 1
• 11 other genes (PSORS2-12)

Pathogenesis
• Hyperproliferation of keratinocytes
  • Associated angiogenesis in the superficial dermis in response to over-production of VEGF
• Inflammatory cell infiltrate – neutrophils, TNF, T lymphocytes

Precipitating factors
• Trauma – Koebner phenomenon
• Infection – commonly Group A beta-haemolytic strep eg tonsillitis (also HIV associated with sudden onset severe psoriasis)
• Hormonal – improves in pregnancy, relapses post-partum
• Sunlight – 10% worsen
• Drugs – antimalarials, beta-blockers, IFN-a, lithium, steroid withdrawal
• Stress

Histology
• Parakeratosis (nuclei in stratum corneum)
• Irregular thickening of the epidermis, with thinning over dermal papillae
• Epidermal infiltration by leukocytes (Munro microabscesses)
• Dilated, tortuous capillary loops in dermal papillae
• T-lymphocyte infiltrate in upper dermis

Plaque Psoriasis
• Well-demarcated plaques
• Red – variable intensity
• Silvery scale
  • Auspitz sign
• Single or multiple
• Variable size and shape
• Symmetrical distribution
  • Elbows, knees, lower back, scalp

Guttate Psoriasis
• Triggered by streptococcal tonsillitis
• Many small round red macules, with overlying scale
• Clears within a few months
• May develop plaque psoriasis later

Special Sites
- These may be isolated, or in association with other types / sites
- Scalp
- Nails
- Flexures / genital
- Palms and soles
  - If pustules associated ‘palmoplantar pustulosis’

**Investigations**
- None generally needed
- Biopsy in difficult / atypical cases
- Throat swab in guttate psoriasis
- Skin scrapings / nail clippings if tinea suspected
- Rheumatology input if joint symptoms (X-ray, blood tests)
- Objective measure of severity (there is still some user variation)
  - PASI score (out of 72, >10 considered severe)
  - DLQI score (out of 30, >10 considered severely affecting QOL)

**Management**

**Topical Therapies**
- Vitamin D analogues eg calcipotriol, calcitriol
  - Reduce epidermal proliferation
- Topical corticosteroids
  - Reduce scaling and redness – risk of skin thinning and early relapse / unstable psoriasis
- Local retinoids eg tazarotene
  - Reduce epidermal proliferation, reduce inflammatory infiltrate in dermis
- Dithranol
  - Inhibits DNA synthesis
  - Stains normal skin & bathtubs! Irritant to face & flexures
- Coal tar
  - Used for thousands of years BUT messy, smelly
  - Inhibits DNA synthesis, photosensitises the skin
- Salicylic acid
  - Debrides scale, enhances penetration of other topical therapies
- Calcineurin inhibitors eg pimecrolimus, tacrolimus
  - Useful in face, flexures

**Phototherapy**
- UVB
  - Narrowband used mainly nowadays
- Psoralen + UVA

**Systemic Therapy**
- Retinoids ie. Acitretin
  - Vitamin A analogue, teratogenic
- Methotrexate
  - Teratogenic
- Ciclosporin
  - Short term use mainly – risks of hypertension and renal damage
- Fumaric acid esters
- Mycophenelate mofetil, azathioprine..

**Biological Therapy**
- New biologics are being developed all the time
- Every new gene and immune pathway determined can be a target for a biologic therapy
• Patients must have severe psoriasis and have failed / intolerant of standard systemic therapy / phototherapy
• Risks of opportunistic infections, MS, congestive HF, lupus-like syndromes, lymphomas
• Most established are:
  • Etanercept (anti-TNF-a)
  • Adalumimab (anti-TNF-a)
  • Infliximab
  • Ustekinumab (anti-IL12/23)
  • Secukinumab (anti-IL17)

Lichen Planus
• Affects skin, genitals, mouth & nails +/- scalp
• Violaceous, itchy, flat-topped papules
• Overlying white lacy streaks – Wickham's striae
• Wickham's striae – may be intraoral
• Koebner phenomenon occurs
• Many forms - hypertrophic, erosive/ulcerative, annular, etc
• Nails – longitudinal ridging, pterygium

Management
• Potent topical steroids
• Systemic steroids
• Phototherapy UVB, PUVA
• Systemic therapy – ciclosporin

Eczema

Histology
• Spongiosis (oedema in epidermis)
• Intraepidermal vesicles
• Acanthosis (thickening of stratum spinosum)
• Hyperkeratosis (thickening of stratum corneum)
• Parakeratosis (retained nuclei in stratum corneum)
• Vasodilatation
• Lymphocyte infiltration

Clinical Features
• Red, scaly, itchy patches
• May have weeping, crusting or vesicles in the acute stage
• May have lichenification and fissuring in the chronic stage

Management
• Topical emollients
  • Cream Vs Ointment
  • Wet wrap dressings
  • Soap substitutes
• Topical corticosteroids
  • Mild, Moderate, Potent, Superpotent
  • Ointments have less preservatives
  • Side effects include skin thinning and easy bruising
• Topical calcineurin inhibitors
• Phototherapy (whole body or localised, UVB or PUVA)
• Systemic therapy eg Azathioprine, Ciclosporin
• Systemic therapy *hands* Alitretinoin
• +/- antihistamines, antibiotics
• +/- support
• +/- avoid irritants

Atopic Eczema
• Inflammatory disease
• Barrier dysfunction
• Raised IgE levels
  • More likely to have persistent eczema, asthma
• Associated with other atopic conditions — allergic rhinitis, asthma, food allergies
• Rising prevalence worldwide (20% western Europe/USA/Aus, 2% developing) — “hygiene hypothesis”
• Genetic factors
  • Monozygotic twins 80%, dizygotic twins 22%
  • 75% risk if both parents affected (higher risk if mother than father)
  • Filaggrin loss of function mutations (barrier)
    • More severe eczema, asthma, peanut allergy

Presentation
• 75% before age 6m, 80-90% by 5 years
• 60-70% clear by teens (may relapse later, skin dryness persists)
• Itch is the main symptom, and greatly affects quality of life of children and parents

Infection
• Staph Aureus colonisation common
• Can cause infection leading to eczema flare (eg impetigo), or worsening despite treatment
• Herpes simplex causes eczema herpeticum

Pityriasis Alba
• Light patches on pigmented skin +/- fine scale
• Strong association with atopic dermatitis
• Topical steroids or calcineurin inhibitors can be helpful
  • Hypopigmentation will take months to improve

Contact vs Irritant Eczema
• Irritant eczema is common — mainly hand dermatitis
  • Water, dust, detergents, chemicals
• Allergic contact dermatitis — Type IV delayed type hypersensitivity
  • Once sensitised, permanent allergy
• Pompholyx — recurrent vesicles/blisters on palms/fingers/soles
  • Can be caused by stress, irritants
• Differentiating between them is an important part of management
  • History eg hobbies, occupation, wearing of jewellery or particular uniforms

Patch testing
• Standardised concentrations of common allergens
• Applied to the back
• Readings at 48 and 96 hours (Day 2 & 4)
• Photopatch testing — similar but UVA applied
  • Day 0 patches applied
  • Day 1 UVA applied
  • Day 3 readings

Common Allergens
• Nickel — cheap jewellery, belt buckles, etc
• Chrome — cement, leather
• Fragrance
• Hair dye — paraphenyldiamine
• Emollients — cetostearyl alcohol, lanolin
Preservatives – parabens, formaldehydes
Topical steroids – tixocortol, budesonide
Rubber mixes – carba, mercapto, thiuram, black rubber
Plants – sesquiterpene lactone, primin
Glue/adhesives – colophony

Management

“The Usual” plus:
Irritant Avoidance
Allergen Avoidance (if relevant)
Protective clothing eg gloves

Juvenile Plantar Dermatosis

Sweat gland blockage causing red, itchy, dry, fissured soles of feet
Clears in teens
Treat flares using topical steroids and emollients
Mainstay of management is reducing time spent in occlusive footwear

Seborrhoeic Dermatitis

Overgrowth of yeast Malassezia Furfur
Scalp, ears, forehead, nasolabial folds, eyebrows
Red, scaly
Mild known as “dandruff”
Can affect babies “cradle cap” – tends to clear spontaneously
Presternal and interscapular
Red, scaly and/or follicular papules/pustules
Axillae, groins, umbilicus
* HIV Indicator Disease (severe, not responding to treatment)

Management
Topical ketoconazole cream
Ketoconazole shampoo
Selenium sulfide shampoo
“trimovate” cream for flexures
Oral itraconazole

Ichthyosis Vulgaris

Autosomal dominant disorder
Mutation in Filaggrin gene -> loss/reduction of profilaggrin
Skin dryness develops over first years, may improve in adulthood
Small scales on limbs, prominent palmar creases
Management involves emollients after shower/bath
Dermatology Emergencies

Erythema Multiforme

- Annular, non-scaly red plaques
  - Lesions expand and clear centrally, new one forms in the middle
  - “target” lesion
  - May blister
  - Clear over a few days
- Mainly palms, soles, forearms, legs
- New lesions appear for 1-2 weeks after infection/drug has been stopped
- Can affect mucosa: oral, genital, ocular, pharyngeal (EM major)

Causes

- Infection
  - HSV – can cause recurrent EM
  - Mycoplasma
  - Hepatitis
  - Bacterial eg URTI
  - Fungal, Parasitic, etc
- Drugs
- Pregnancy
- Malignancy
- Idiopathic

Management

- Investigate for cause eg swabs, PCR, CXR & treat if found
- EM
  - Emollients
  - Topical steroids
  - Antihistamines
- EM Major
  - Multi-disciplinary involvement
  - Oral corticosteroids
- Recurrent EM
  - 6 months prophylactic antivirals trial

Drug Reactions

- Drug reactions are variable – almost any morphology
- The commonest is the exanthematous drug reaction
- The most serious is Stevens-Johnson syndrome – which when severe leads to Toxic Epidermal Necrolysis (TEN)
- These are the 2 drug reactions you MUST be able to recognise

Drug Exanthem

- Morbilliform “measles-like” eruption starts on the trunk and spreads to extremities
- Associated itch, fever
- Caused by antibiotics, sulfonamides, barbiturates, phenylbutazone, para-aminosalicylate
- Starts 2-3 days after drug administration

Management

- Stop the drug
- Topical emollients
- Topical steroids
- Calamine lotion, antihistamines
Stevens-Johnson Syndrome / Toxic Epidermal Necrolysis

- Drugs implicated
  - Sulfonamides, anti-epileptics, nevirapine, allopurinol, sulfasalazine, NSAID
  - Onset 4-28 days after
- Body surface area affected
  - 1-10% SJS
  - 10-30% SJS/TEN overlap
  - >30% TEN

Presentation

- Prodrome: fever, malaise, cough
- Onset of painful macular exanthem -> red, intense pain, detachment of skin -> erosions
- Split is subepidermal
- Nikolsky +
- Starts on trunk and face, spreading to limbs
- Mucous membranes affected (may include GI tract & bronchial tree)

Assessment

- Body surface area affected
- SCORTEN predicts mortality
- Death due to infection, loss of fluids & electrolytes
- If survive – will occur again if retakes the drug
- May develop blindness due to corneal scarring

Management

- Identify the cause
- Symptomatic management
- Burns unit or ICU care
- Fluid & electrolyte balance
- Wound care
- Pain management
- Nutritional support
- IVIG
- Plasmapharesis

Urticaria

- This is not an emergency but patients worry
- Pink, itchy wheals on the body
- Each wheal lasts <24hours
- Acute urticaria – lasts less than 6 weeks
- Chronic urticaria – 2-3 episodes per week for more than 6 weeks
  - May settle spontaneously

Angio-oedema

- Angioedema involves subcutaneous tissues, deeper swellings eg periorbital, perioral, tongue
- This can last up to 7 days
- It can occur with chronic urticaria or isolated
- If isolated it may be due to
  - Hereditary Angioedema
    - AD
    - deficiency of C1 esterase leads to generation of vasoactive mediators
    - Plasma C4 levels will be low
  - ACE inhibitor (due to inhibition of bradykinin breakdown)

Pathogenesis

- Mast cell degranulation releases histamine & other vasoactive substances
• Increased capillary permeability
• Leakage of fluid into tissue
• Wheal forms

Classification
• Acute vs Chronic
• Physical urticarias
  • Cold urticaria
  • Solar urticaria
  • Heat urticaria
  • Cholinergic urticaria (exercise/stress)
  • Dermographism (immediate pressure)
  • Delayed pressure (3-6 hours after pressure, lasts up to 48 hours)

Key Facts
• Type 1 hypersensitivity reactions (IgE mediated) can also lead to urticaria
• Opiates, aspirin, NSAIDs can cause urticaria
• Contact urticaria eg from food or food additives, latex
• Urticarial reactions can occur to insect bites
• A common cause of acute urticaria is infection

Management
• Antihistamines (H1 blocking)
• H2 blockers
• Montelukast
• Systemic corticosteroids
• Systemic therapy
• Biologic – omalizumab

Pityriasis Rosea
• Not an emergency but sudden onset causes patients to worry
  • Is it serious?
  • Is it contagious?
  • Is there any treatment?
• Due to reactivation of HHV7 / HHV8
• Affects children & young adults usually

Presentation
• ‘Herald patch’
  • Few cm red scaly plaque
• Few days later, for 1-2 weeks
  • Onset of small, oval, salmon pink macules
  • Collarette of fine scale
  • On the back can see “fir tree” pattern
• Can be itchy
• Resolves spontaneously over 2-10 weeks

Management
• Correct diagnosis
• Emollients
• Calamine lotion or antihistamines for itching
• Topical steroids can be tried
• UVB phototherapy can be helpful for persistent cases

Erythroderma
• One of the few true dermatological emergencies
• Definition: >90% body surface area affected (red, scaly)
• May have associated lymphadenopathy / hepatosplenomegaly (may be reactive)
• Impaired temperature regulation: fever, shivering
• Sequelae: oedema, high output cardiac failure, tachycardia, anaemia, dehydration

Causes
• Causes:
  • 40% idiopathic
  • Psoriasis
  • Dermatitis
  • Mycosis fungoides / CTCL
  • PRP
  • Drug eruptions
  • Others
• Many idiopathic turn out to be due to MF/CTCL – repeat biopsies usually required

Management
• Supportive care
  • 50/50 WSP/LP
  • Fluid balance
  • Temperature control
  • Swabs of broken skin
• Specific management depends on the condition
Bullous Diseases

Bullous Pemphigoid
- Autoimmune disease
- IgG targets basement membrane antigens BP230 & BP180
- Older patients
- Self-limiting condition within 1-2 years
- Risk of mortality – related to age, steroid use

Presentation
- ‘Prebullous’ – itchy red plaques, may have vesicles
- Tense clear or red blisters
  - Can be localised or generalised
  - Rarely can involve oral mucosa
- Nikolsky negative
- Can get secondary infection

Management
- Biopsy for diagnosis
  - H&E: subepidermal blister, eosinophils
  - DIF: linear IgG & C3 along basement membrane zone
- Very potent topical steroids (localised forms)
- Systemic steroids
  - High dose then reduced to lowest maintenance dose
- Oral doxycyline (anti-inflammatory antibiotic) has been proven to be of benefit
- Immunosuppression: AZT, MTX, dapsone

Pemphigus Vulgaris
- Autoimmune disease
- IgG antibodies target desmoglein 1 in the epidermis
- Middle aged patients
- Common in Mediterranean, Indian origin, Ashkenazi Jews
- Mortality 15%
- One third remission within 3 years

Presentation
- Flaccid blisters which rupture on the trunk, flexures, scalp
  - Crusted or weeping erosions seen
- Nikolsky sign positive
- Mucosal involvement- mainly oral
- Can get secondary infection

Management
- Biopsy to confirm diagnosis
  - H&E: intra-epidermal vesicles, acantholysis
  - DIF: intercellular deposits of IgG and C3
- High dose systemic steroids
- Immunosuppression: AZT, MMF, cyclophosphamide
- New: Rituximab, dapsone, plasmapheresis

Dermatitis Herpetiformis
- Prevalent in North Europeans
  - Association with HLA DQ2 & DQ8
- Gluten-sensitive enteropathy is present
  - May not have bowel symptoms
• Serum autoantibodies present to tissue transglutaminase
  • These cross-react with epidermal transglutaminase
• Longterm condition
• Risk of small bowel lymphoma
• Associated with other autoimmune diseases

Presentation
• Intensely itchy grouped vesicles and urticated papules
  • Excoriations and erosions seen due to scratching
• Elbows, knees, buttocks, shoulders

Management
• Biopsy
  • H&E: subepidermal vesicle, neutrophils
  • DIF: granular deposits of IgA and C3 in dermal papillae tips and superficial dermis
• Gluten free diet
  • Skin disease takes months to improve
• Dapsone

Erythema Nodosum
• Inflammation of subcutaneous fat
• It is a sign of underlying disease
  • Infection eg strep, TB, EBV, HBV, Mycoplasma, Chlamydia
  • Drugs eg sulfonamides, penicillins, OCP
  • Systemic disease eg IBD, Sarcoid
  • Pregnancy
  • Malignancy

Presentation
• Tender red nodules usually on anterior legs (+/- arms, others)
• May have associated fever and arthralgia
• Resolves over 2 weeks, may recur for 6-8 weeks

Investigations
• Search for the underlying cause
  • CXR
  • ASOT
  • Throat swab
  • Pregnancy test

Management
• Treat the cause!
• Bed rest, leg elevation
• NSAIDs

Subacute Cutaneous LE
• Well-demarcated annular plaques on face, chest, hands, and sun-exposed sites
  • Heal without scarring
• Photosensitive
• 50% also have systemic disease
• Antibodies to Ro commonly
• May be drug-induced
• Treatment includes photoprotection, topical steroids, antimalarials (HXQ)

Discoid LE
• Few plaques on sun exposed sites
• Erythematous, scaly, plaque with follicular plugging
  • Heals with scarring, telangectasia
• Can affect the scalp leading to scarring hair loss
• 5-15% develop systemic disease
• Treatment: potent/very potent steroids, tacrolimus, photoprotection, antimalarials

Dermatomyositis
• Subset of polymyositis
  • Skin changes include heliotrope eyelid discolouration, neck and presternal poikiloderma (shawl sign), lilac atrophic scaly plaques over knuckles (Gottron’s papules)
  • Proximal muscle weakness
  • Subcutaneous calcification in juvenile form
• Autoimmune
• Age >40 may be a sign of malignancy in 25%
• Treated with high dose systemic steroids, immunosuppression, photoprotection

Scleroderma
• Systemic sclerosis always affects the skin
  • Reduced elasticity
  • Thickening/hardening
• Localised (CREST)
  • Extremities and face
  • Calcinosis (calcium over pressure points)
  • Raynauds (may precede skin changes)
  • Sclerodactyly (immobile hard fingers)
  • Telangectasia (periungual and face)
• Diffuse
  • Raynauds, Sclerodactyly
  • Rapid onset of disease, widespread skin involvement
  • Beak-like nose, perioral wrinkles, alopecia, dryness
  • Multi-system involvement

Vasculitis
• Cutaneous small vessel vasculitis
  • Painful palpable purpura, may have necrotic centre
  • Usually lower legs and arms
  • Resolves Spontaneously after removing the cause
  • May be associated with systemic vasculitis eg renal, GI (check urine & BP)
  • Investigations include infection screen, CXR, antibody screen, complement
• Henoch-Schonlein purpura
  • Children following URTI
  • Palpable purpura of lower limbs, arthritis, abdominal pain, nephritis

Necrobiosis lipoidica
• Affects <3% of diabetics
• Non-diabetic patients should be tested
• Shiny yellowish atrophic plaques on shins
• May ulcerate (slow to heal)

Sarcoid
- Erythema nodosum
- Sarcoid granulomas in skin
  - Brown-red papules, nodules, plaques
  - Scar sarcoid
  - Dusky plaques on nose/fingers

**Hair Loss**
- Diffuse hair loss can occur due to systemic disease
- Endocrine (pituitary, thyroid, parathyroid, androgens)
- Drugs (antimitotic eg chemotherapy, retinoids, anticoagulants, OCP)
- Androgenetic
- Iron deficiency
- Severe chronic illness
- Malnutrition
- Other causes: telogen effluvium, androgenetic, diffuse alopecia areata

**Hirsutism**
Growth of terminal hair in female patient in a male distribution
- Racial/familial
- PCOS
- Cushings
- Ovarian/adrenal tumour
- Anabolic steroid use

**Hypertrichosis**
Excessive growth of terminal hair
- Malnutrition
- Drugs (minoxidil, phenytoin, ciclosporin)
- Cutaneous porphyrias
- Others eg rare syndromes

**Paraneoplastic**
- Acanthosis nigricans (GI) – smooth, velvety hyperpigmentation in axillae, groins
- Erythema gyratum repens (bronchial, oesophageal) – ‘woodgrain’ appearance
- Necrolytic migratory erythema (glucagonoma) – scaly advancing edge
- Dermatomyositis
- Pruritus
Sebaceous Disorders

Sebaceous Glands

- Associated with hair follicles (face, behind ears, upper chest and back) or in the epidermis (eyelid, mucous membranes, nipple, genitalia)
- Multilobed gland with lipid-containing cells
- Stimulated by androgens
- Sebum: triglycerides, free fatty acids, wax esters, squalene, cholesterol
- Sebum functions: waterproof, mildly bactericidal, mildly fungistatic

Acne

- Inflammatory disorder of the pilosebaceous unit
- Affects mainly teenagers
- Increasing persistent and late onset acne
- Multifactorial
  - Over-production of sebum
  - Androgens – excessive response to normal levels
  - Occlusion of skin pores – genetic, cosmetics
  - Increased bacterial colonisation – P.Acnes
  - Genetics
  - Diet – dairy?
- Has marked effects on QOL
- Clears by age 23-25 in 90%

Presentation

- Face, shoulders, upper chest, back
- Seborrhoea – oily skin
- Open comedones – blackheads
- Closed comedones – whiteheads
- Inflammatory papules, pustules, nodules, cysts
- Atrophic or hypertrophic scarring, post-inflammatory erythema and/or pigmentation

Treatment

Topical

- Gentle cleansing
- Salicylic acid (comedones)
- Benzoyl peroxide (inflammatory)
- Vitamin A analogues (comedones)
  - Tretinoin, isotretinoin, adapalene
- Azelaic acid (inflammatory, bactericidal)
- Antibiotics *resistance*
  - Clindamycin, erythromycin

Systemic

- Erythromycin
  - Erythromycin resistance in P.Acnes
- Tetracyclines
  - Oxytetracycline, tetracycline, lymecycline, doxycycline, minocycline
  - Avoid in pregnancy and <9 years
  - Minocycline side effects pigmentation & lupus-like syndrome
- Trimethoprim
- Co-cyprindiol (dianette)
  - Risk of VTE – use only for 3 months after acne clears
Isotretinoin
- Oral retinoid
- Inhibits sebum excretion, inflammation and P.Acnes
- Used in severe nodulocystic acne, scarring acne, non-responsive acne
- May flare on initiation – can use lower dose in first month(s)
- Teratogenic
- Risk of depression and suicidality
- Dry skin, dry nose/lips/eyes, facial erythema, muscle aches, hyperlipidaemia, hair loss
- Rare serious: loss of night vision, pancreatitis, hepatotoxicity, pseudotumour cerebri, hearing loss

Rosacea
- Females; peak age of onset 30-40
- More common in fair skinned patients
- Abnormally high levels of cathelicidin leading to inflammation and vasodilatation
- Symmetrical: cheeks, nose, mid-forehead, chin
- Flushing in response to warmth, sunlight, spicy food, alcohol, caffeine, embarassment
- Fixed erythema and telangetasias
- Discrete papules, papulo-pustules, plaques, nodules
- Overgrowth of sebaceous glands and connective tissue (nose – rhinophyma)
- Ocular – blepharitis, conjunctivitis, keratitis

Treatment
- Topical – metronidazole, azelaic acid
- Oral antibiotics – tetracyclines, erythromycin
- Systemic isotretinoin
- Sunscreens – if sunlight aggravates
- Lasers or IPL for telangetasia / erythema
- Brimonidine for flushing
- Surgical excision / laser for rhinophyma
- **Avoid topical steroids – withdrawal will lead to severe rebound flare**
Skin cancer & skin surgery

Benign

Seborrhoeic Keratosis
- Single or multiple
- Older patients
- Warty stuck-on appearance

Skin tag
- Soft skin-coloured or pigmented pedunculated papules
- Neck, flexures

Milia
- Small subepidermal keratin cysts on the face
- May be multiple
- May occur after trauma or blister

Melanocytic Naevi
- Localised benign collection of melanocytes
- Congenital + early childhood onset, increase in numbers during adolescence & after sunburn
- Risk of melanoma in congenital naevi >20cm
- Junctional – compound – intradermal
- Atypical naevi can occur sporadically or familial (CDKN2A mutation has risk of MM)
- Halo navi – naevus involutes, then skin repigments

Precancerous

Actinic Keratosis
- Discrete pink macule with overlying scale
- Middle-aged and elderly
- Cumulative sun exposure
- Risk of progressing to SCC
- Treatment with cryotherapy, curettage, topical 5-FU, topical imiquimod, topic ingenol mebutate, PDT

Bowens disease
- Squamous cell carcinoma in situ
- Single, slowly evolving pink scaly plaque
- Treatment with cryotherapy, curettage, excision, PDT, topical 5-FU/imiquimod

Cancer

Basal Cell Carcinoma
- Most common form of skin cancer
- Middle-aged & elderly patients
- Risks: prolonged sun exposure, radiation, arsenic
- Multiple in Gorlin’s syndrome
- Commonest is the nodular type – presents as a skin-coloured papule with telangiectasia, rolled edge, +/-central ulceration +/- pigmentation
- Treatment is with excision (Mohs for high risk lesions)
- Alternative is radiotherapy
• Superficial subtype – can treat with cryotherapy, PDT, imiquimod

Squamous Cell Carcinoma
• Malignant tumour of keratinocytes
• Risk of metastasis
• Risks: UV, tar, arsenic, organ transplant (immunosuppression)
• Present as rapidly growing scaly nodules +/- ulceration
• Treatment is with surgical excision
• Patients require follow-up

Melanoma
• Rising incidence in the UK/USA
• Occurs on palms/soles/mucosa in Asian/Black skin
• Risks: family history (CDKN2A), fair skin types, atypical naevi, sunlight/sunburn.
• Sun protection is key in prevention (sun screen, clothing, sun avoidance)
• Management is surgical excision (+/- SNB)

Mole Assessment
• A – Asymmetry
• B – Border (Irregular)
• C – Colour (>3)
• D – Diameter (>6mm)
• E – Elevation (new)

Skin Surgery

Excision
• Lesions are excised under local anesthetic
• Aseptic technique
• Scar follows the relaxed skin tension lines (skin crease)
• Margin of surrounding skin removed (4mm BCC/SCC, 2mm pigmented lesion)
• Elliptical shape -> straight scar

Shave excision
• Exophytic benign lesions

Curettage
• Benign or precancerous lesions

Mohs microsurgery
• Narrow excision with marking of the edges
• Immediate processing into histological slides
• If tumour extends to the margin then more tissue is removed and examined again
• And so on ..
**Photodermatology, Psychodermatology, Lasers & Cosmetic Dermatology**

**Photodermatology**
- Sunlight has several benefits: vitamin D synthesis, creation of nitric oxide which lowers BP, treatment of diseases eg eczema, psoriasis
- Excess increases the risk of developing melanoma, and causes photoaging.
- Sunlight can worsen some skin disorders eg Rosacea, lupus, porphyria
- Sunlight can cause some skin disorders eg PLE, CAD, photocontact dermatitis (photoallergy), photosensitive drug eruption (eg doxycycline, naproxen, amiodarone)

**Polymorphic Light Eruption**
- Most frequent photodermatosis
- Genetic predisposition
- Recurs annually in Spring after sun exposure
  - Some develop ‘hardening’ after initial few exposures and therefore less episodes
  - Clears during Winter
- Onset 2hrs – 5 days after sun exposure
- Itchy red papules, vesicles, eczematous plaques
- Sun exposed areas only
- Face may be spared
- Prevention
  - Sunscreens
  - Protective clothing
  - Phototherapy ‘desensitisation’
- Treatment
  - Potent topical steroids
  - Oral steroids in severe episodes

**Psychodermatology**
1. Stress and emotion can aggravate many/most skin conditions
2. The presence of a skin condition can have an effect on patient’s mood and quality of life
3. Psychodermatoses

**Psychodermatoses**

**Dysmorphophobia**
- Patient imagines/exaggerates their lesion/skin disease

**Neurotic excoriations**
- Patients admit picking skin lesions
- Release of tension with scratching/picking felt
- May have mood disorder or OCD

**Acne excorii**
- Pre-existing acne vulgaris (usually mild) is picked/squeezed leading to scarring

**Delusional Infestation**
- Convinced that they are infested
- Sensations of crawling, picking/cutting out, fixed belief of fibres coming out of the skin, “matchbox sign”
- May have underlying drug or alcohol addiction, vitamin deficiency, cerebrovascular disease
- Management includes atypical antipsychotics
Dermatitis Artefacta
- Skin lesions are caused by the patient
- Denied by the patient
- Lesions heal under occlusive dressings
- No primary lesions seen
- Unusual shape or unexplained distribution
- Usually young women, some medical knowledge e.g. nurses, may have an underlying mood/personality disorder

Hair Pulling / Trichotillomania
- Usually children
- Pulling/twisting of hair leads to irregular patches of hair loss
- Residual broken/bent hairs seen
- Resolves in children spontaneously
- May be associated with learning difficulties and difficulties in adolescence

Ageing
- Ageing leads to
  - Thin skin (bruising)
  - Clumping of elastic fibres (elastosis)
  - Pigmentation (lentigines, freckles)
  - Loss of dermal elastic recall (wrinkles)
  - Loss of collagen (1% per year)
  - Reduced fibroblasts (less collagen synthesised and slower wound healing)
  - Telangiectasia
  - Redistribution of subcutaneous fat
- Caused by: time, genetics, repeated muscular contraction
- Aggravated by:
  - Smoking
  - Chronic UV damage

Ways to “reverse ageing”
- Tretinoin (retinoid)
- Chemical peels
- Lasers
- IPL
- Botox
- Fillers
- Plastic surgery
- Dermabrasion

Botox
- Clostridium Botulinum produces Botulinum Toxin
- Irreversibly binds to the presynaptic junction blocking the release of acetylcholine -> paralysis of the muscle
- Indications: strabismus, dystonia, wrinkles
- Effects start up to 2 weeks after injection, last 3-4 months
- Contraindication: myasthenia gravis, pregnancy, breastfeeding, aminoglycoside antibiotics

Fillers
- Natural or synthetic polymers e.g. collagen, hyaluronic acid
• Indications: filling areas which have lost volume eg nasolabial folds
• Side effects: bruising, erythema, oedema, foreign body reaction, granulomas, lumps, infections, local tissue necrosis due to vascular compromise

**Melasma**
• Acquired symmetrical hyperpigmentation
  – Darkens after sun exposure
• Sun-exposed skin -> face usually
• Women > men
• Darker skin types affected more frequently
• Causes:
  – Sunlight
  – Pregnancy
  – Oestrogens / OCP
• Treatment
  – Sun avoidance & sunscreen
  – Hydroquinone (alone or combined with retinoid/steroid)
  – Azelaic acid / Kojic acid
  – Chemical peels
  – Laser

**Principles of Phototherapy**
• Mainly narrowband UVB and Psoralen-UVA (PUVA)
• Pre-treatment phototesting / skin typing to determine starting dose
• UVB
  – Less carcinogenic than PUVA
  – 3 times weekly treatment
• PUVA –
  – 8-methoxypsoralen (oral/topical) used
  – Twice weekly treatment
  – Carcinogenic risk
  – More effective than UVB & penetrates into the deep dermis

**Principles of Photodynamic Therapy**
• Indications: AK, Bowens, superficial BCC
• Photosensitiser applied topically (ALA, MAL)
• Irradiated 3 hours later with red of blue light
• ‘Daylight’ PDT regimens available
• Can treat several lesions at once
• Side effects: pain, erythema, crusting, rarely ulceration

**Laser**
• Indications: vascular birthmarks/lesions, tattoo removal, epidermal naevi, pigmented lesions, seborrhoeic keratoses, warts
• Laser Amplification by the Stimulated Emission of Radiation
• High intensity coherent light sources of a single wave length
• Light photons are absorbed by the target chromophore (ie tattoo pigment, melanin, etc) leading to tissue destruction