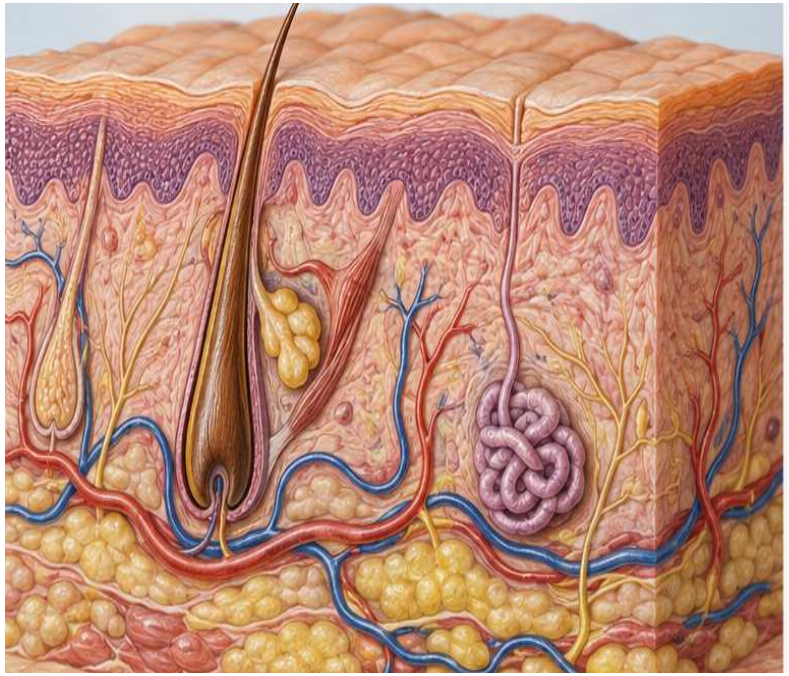
Clinical morphology, disease mechanisms and management

See the lesion. Name the process. Assess the patient.

This illustrated chapter links skin anatomy and lesion morphology to pathology, investigation, treatment and systemic disease. It is written for MRCP(UK) Parts 1 and 2, with original simulated clinical images showing a range of skin tones.



PART 1 CORE

Layer and barrier → morphology → immune pathway → histology → pharmacology.

PART 2 APPLICATION

Presentation → danger signs → differential → biopsy/test → management → follow-up.

All images are simulated educational examples, not patient studies or diagnostic standards.

For clinical decisions, consult current local prescribing guidance and relevant specialist advice.

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Skin architecture and barrier

ANATOMY / MECHANISM / PRESENTATION

The epidermis contains keratinocytes, melanocytes, Langerhans cells and Merkel cells. Basal renewal, spinous cohesion, granular lipid processing and stratum-corneum barrier function explain the appearances of blistering, scaling and barrier disease. The dermis contains vessels, nerves, collagen, hair follicles and sweat glands.

DIFFERENTIAL / INVESTIGATION

Basement-membrane zone links epidermis to dermis via hemidesmosomes. Intraepidermal acantholysis produces fragile superficial blisters; subepidermal separation produces tense blisters. Histology level and direct immunofluorescence distinguish the major autoimmune bullous disorders.



Epidermis, dermis and appendages • SIMULATED

TREATMENT / PHARMACOLOGY

Barrier repair depends on regular emollients and avoiding irritants. Topical drug vehicle matters: ointment favours dry skin, cream is more acceptable in weeping sites, and scalp preparations aid hair-bearing skin. Choose steroid potency by site, severity and duration.

COMPLICATIONS / NEXT STEP

Skin of colour may show less visible erythema and more post-inflammatory dyspigmentation. Assess warmth, scale, induration, tenderness and distribution, not colour alone. Document occupational exposure, systemic symptoms, medication and mucosa.

CLINICAL PEARL

Transepidermal water loss increases when the stratum corneum is disrupted. Emollient choice and adherence often determine whether anti-inflammatory treatment can work.

AMT MEMORY AID: layer predicts lesion: epidermis = scale/fragile blister; deeper split = tense blister.

MRCP TRAP: absence of bright redness does not exclude severe inflammation in dark skin.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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Lesion morphology and pattern recognition

ANATOMY / MECHANISM / PRESENTATION

Name the primary lesion before naming a disease: macule is flat colour change; papule is raised and small; plaque is broad and palpable; vesicle contains clear fluid; pustule contains purulent material; wheal is transient dermal oedema. Secondary scale, crust, excoriation, erosion and ulcer follow evolution or injury.

DIFFERENTIAL / INVESTIGATION

Map distribution, symmetry, flexures/extensors, photoexposure, dermatomes, palms/soles, mucosa and nails. Check if lesions blanch. Palpable non-blanching purpura suggests small-vessel inflammation; retiform purpura suggests occlusion or deeper vascular pathology.



Macule, papule, plaque and vesicle • SIMULATED

TREATMENT / PHARMACOLOGY

Use history of onset, itch/pain, drug timing, fever and exposure to rank the differential. Photograph with consent and include a size scale. Dermoscopy helps triage pigmented or vascular structures; uncertain lesions need specialist assessment, not image diagnosis alone.

COMPLICATIONS / NEXT STEP

A single diagnosis can produce several morphologies. A new painful purpuric or blistering eruption with fever, mucosal disease or systemic compromise is an emergency. Consider distribution before selecting a biopsy site.

CLINICAL PEARL

A lesion that does not blanch needs a vascular or haemorrhagic differential. Document whether it is palpable, painful or associated with fever.

AMT MEMORY AID: lesion → distribution → time course → systemic clues → targeted test.

MRCP TRAP: “rash” without primary morphology prevents a useful differential.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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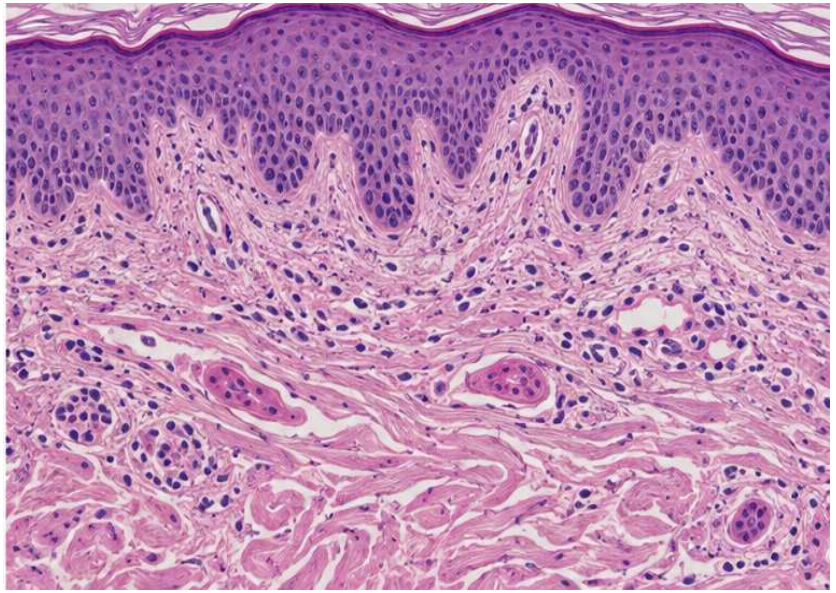
Biopsy, histology and immunofluorescence

ANATOMY / MECHANISM / PRESENTATION

Spongiosis points toward eczematous reaction; psoriasiform acanthosis may accompany psoriasis; interface injury suggests lichenoid, lupus or drug processes; leukocytoclastic vasculitis shows vessel-wall injury and neutrophil debris. Histology is interpreted with timing and clinical phenotype.

DIFFERENTIAL / INVESTIGATION

For suspected autoimmune blistering, sample lesional skin for routine histology and perilesional intact skin for direct immunofluorescence in the correct transport medium. For vasculitis, a fresh purpuric lesion improves diagnostic yield; assess urine, renal function and systemic features.



Skin biopsy teaching micrograph • SIMULATED

TREATMENT / PHARMACOLOGY

Choose punch, shave or excision by question and anatomic site. Suspected melanoma generally needs complete diagnostic excision with a narrow clinical margin where feasible, retaining full depth for Breslow measurement. Discuss exceptions with the skin cancer team.

COMPLICATIONS / NEXT STEP

False-negative biopsy occurs with wrong site, old lesion or corticosteroid-altered tissue. DIF patterns are not interchangeable: intercellular IgG in pemphigus; linear basement-membrane staining in pemphigoid; granular IgA in dermatitis herpetiformis.

CLINICAL PEARL

Tell pathology the suspected diagnoses, lesion age and treatment exposure; morphology alone rarely resolves a poorly contextualised sample.

AMT MEMORY AID: one specimen for architecture, another for immune deposits.

MRCP TRAP: formalin destroys the diagnostic utility of a DIF specimen.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD skin biopsy/DIF; NICE NG14

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Atopic dermatitis and skin barrier failure

ANATOMY / MECHANISM / PRESENTATION

Pruritic relapsing eczema often affects flexures, with xerosis, excoriation, lichenification and infection susceptibility. Atopic history supports but is not essential. On dark skin, inflammation can appear violaceous or grey-brown rather than bright red.

DIFFERENTIAL / INVESTIGATION

Assess extent, sleep loss, quality of life, infection, occupation and allergic contact contribution. Weeping or crusting may reflect secondary infection, but not every flare needs antibiotics. Eczema herpeticum is a painful monomorphic vesicular eruption with systemic illness.



Flexural eczema on dark skin • SIMULATED

TREATMENT / PHARMACOLOGY

Use liberal emollient, soap substitute, trigger reduction and site-appropriate topical corticosteroid. Proactive intermittent treatment of recurrent sites may reduce flares. Calcineurin inhibitors can spare sensitive sites; phototherapy and systemic biologic/JAK therapy are specialist options for refractory moderate-severe disease.

COMPLICATIONS / NEXT STEP

Review steroid technique, potency and quantity before escalation. Systemic immunomodulators require screening, vaccination planning and monitoring. Eczema herpeticum needs urgent systemic antiviral treatment and eye assessment if periocular.

CLINICAL PEARL

A painful, uniform crop of vesicles in eczema raises eczema herpeticum. Periocular involvement adds urgency for ophthalmic assessment.

AMT MEMORY AID: itch–scratch–barrier loop; treat inflammation and restore barrier together.

MRCP TRAP: widespread eczema with punched-out painful erosions is not an ordinary flare.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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Allergic and irritant contact dermatitis

ANATOMY / MECHANISM / PRESENTATION

Irritant dermatitis reflects direct barrier injury and is common with wet work; allergic contact dermatitis is delayed type IV hypersensitivity after sensitisation. Both may itch and scale, but sharply localised or spreading geometric contact patterns suggest exposure.

DIFFERENTIAL / INVESTIGATION

Ask about occupation, cosmetics, fragrance, preservatives, nickel, gloves and topical medicines. Patch testing identifies relevant delayed allergy; it is not a test for immediate urticaria. Review actual products and relevance of positive reactions.



Geometric watch-strap dermatitis • SIMULATED

TREATMENT / PHARMACOLOGY

Avoid the culprit and protect the barrier, with emollients and appropriate topical anti-inflammatory treatment. Hand eczema often needs occupational adjustment and glove strategy. Treat fissures and secondary infection when present.

COMPLICATIONS / NEXT STEP

Repeated exposure can cause chronic lichenification and work disability. A negative standard patch series does not rule out an untested allergen; irritant dermatitis can coexist with allergy. Distinguish from tinea before prolonged steroid use.

CLINICAL PEARL

Hand dermatitis may have both irritant and allergic drivers; patch results require exposure correlation, not merely a positive laboratory report.

AMT MEMORY AID: contact shape + exposure chronology + patch relevance.

MRCP TRAP: positive patch test means sensitisation, not automatically current causation.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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Psoriasis: immunology, phenotypes and treatment

ANATOMY / MECHANISM / PRESENTATION

IL-23/Th17-driven inflammation accelerates keratinocyte turnover. Plaque disease has sharply demarcated scale on extensor sites and scalp; guttate disease may follow streptococcal infection; pustular and erythrodermic forms can be unstable emergencies.

DIFFERENTIAL / INVESTIGATION

Assess body surface area, PASI or equivalent severity, DLQI, nail disease, inflammatory joint symptoms and cardiometabolic risk. A painful swollen digit or inflammatory back pain prompts psoriatic arthritis assessment. Biopsy is reserved for uncertainty.



Plaque psoriasis on medium-brown skin • SIMULATED

TREATMENT / PHARMACOLOGY

Topical corticosteroid/vitamin D combinations, phototherapy and conventional systemic agents (methotrexate, ciclosporin, acitretin) are selected by phenotype and comorbidity. Biologics targeting TNF, IL-17 or IL-23, or targeted oral agents, follow current NICE eligibility and screening.

COMPLICATIONS / NEXT STEP

Methotrexate risks marrow and liver toxicity; ciclosporin causes nephrotoxicity and hypertension; acitretin is highly teratogenic. Biologics require infection/TB/HBV screening and class-specific monitoring. Avoid abrupt systemic steroid use as routine psoriasis therapy.

CLINICAL PEARL

A biologic choice depends on joint disease, bowel disease, infection history and local access criteria, as well as PASI and DLQI.

AMT MEMORY AID: skin severity + life impact + joints + metabolic risk decide escalation.

MRCP TRAP: a small area on hands, genitals or face can have high functional burden.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

NICE CG153 (2025 review)

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Acne, rosacea and follicular disorders

ANATOMY / MECHANISM / PRESENTATION

Acne involves follicular plugging, sebum and inflammation; distinguish comedones from rosacea, which lacks comedones and may have flushing, telangiectasia and ocular disease. Hidradenitis suppurativa produces recurrent painful nodules, tunnels and scarring in intertriginous sites.

DIFFERENTIAL / INVESTIGATION

Grade acne severity and scarring, pregnancy possibility, psychosocial harm and medication triggers. PCOS can contribute when acne coexists with hyperandrogenism. Investigate atypical rapid virilisation. Consider ocular rosacea if gritty or red eyes.



Acne with post-inflammatory change • SIMULATED

TREATMENT / PHARMACOLOGY

Use combination topical retinoid/benzoyl peroxide or other guideline-based first-line therapy, avoiding antibiotic monotherapy. Oral antibiotic courses need finite review and topical non-antibiotic maintenance. Severe scarring acne may need isotretinoin with current MHRA safeguards and pregnancy prevention.

COMPLICATIONS / NEXT STEP

Isotretinoin requires lipid/liver and mental-health monitoring and strict teratogenic precautions. For hidradenitis, combine smoking/weight support, pain management, local care, medical therapy and surgery for established tunnels; selected biologics require specialist eligibility.

CLINICAL PEARL

Isotretinoin safety includes pregnancy prevention and psychiatric review; the drug should be prescribed through the current specialist safeguards.

AMT MEMORY AID: comedone = acne clue; recurrent tunnels = hidradenitis clue.

MRCP TRAP: long-term topical or oral antibiotic monotherapy drives resistance.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

NICE NG198; BAD

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Urticaria, angioedema and mast-cell pathways

ANATOMY / MECHANISM / PRESENTATION

Urticarial wheals usually resolve within 24 hours without residual marking. Angioedema affects deeper skin or mucosa. Bradykinin-mediated angioedema, including ACE inhibitor or hereditary C1 inhibitor deficiency, typically lacks wheals and responds poorly to antihistamines.

DIFFERENTIAL / INVESTIGATION

First exclude anaphylaxis: airway, breathing, circulation and trigger chronology. Lesions lasting over 24 hours with bruising or pain suggest urticarial vasculitis; check complement and biopsy where appropriate. Chronic spontaneous urticaria usually needs limited, targeted tests.



Transient wheals on medium-brown skin • SIMULATED

TREATMENT / PHARMACOLOGY

Second-generation non-sedating H1 antihistamines are first-line, with specialist up-dosing where indicated. Omalizumab is a specialist option for refractory chronic spontaneous urticaria under current access criteria. Anaphylaxis needs intramuscular adrenaline and emergency care; bradykinin disease has distinct treatment.

COMPLICATIONS / NEXT STEP

Review NSAIDs and physical triggers. Airway angioedema needs immediate escalation, irrespective of cause. Avoid repeated systemic corticosteroids as a chronic control strategy.

CLINICAL PEARL

Airway swelling needs emergency assessment. Wheals with hypotension or respiratory compromise indicate anaphylaxis, not simple chronic urticaria.

AMT MEMORY AID: wheal <24 h; persistent bruise/pain → consider vasculitis.

MRCP TRAP: isolated ACE-inhibitor angioedema is not histamine-mediated urticaria.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD urticaria; NICE TA339

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Adverse cutaneous drug reactions and SJS/TEN

ANATOMY / MECHANISM / PRESENTATION

Morbilliform eruptions are common; identify timing, culprit drugs and systemic illness. DRESS usually follows a delayed latency with fever, facial oedema, eosinophilia and visceral injury. SJS/TEN features painful dusky lesions, mucositis and epidermal detachment.

DIFFERENTIAL / INVESTIGATION

Assess mucosa, skin pain, BSA detachment, hydration, renal/liver function, blood count and infection. Record all recent drugs including intermittent antibiotics, anticonvulsants and allopurinol. SCORTEN aids prognosis in SJS/TEN but does not replace urgent expert assessment.



Dusky lesions with epidermal detachment • SIMULATED

TREATMENT / PHARMACOLOGY

Stop suspected culprit immediately. SJS/TEN requires admission to a specialist dermatology/burns or intensive-care service for fluids, wound care, pain control, ophthalmology and genital/mucosal care. Immunomodulatory treatment choices remain specialist and evidence-dependent.

COMPLICATIONS / NEXT STEP

DRESS may relapse during steroid taper and can cause delayed thyroid or other autoimmune complications. Document the culprit and avoidance precisely; distinguish severe reaction from uncomplicated exanthem for future drug safety.

CLINICAL PEARL

The culprit may be a drug started weeks earlier. Record start/stop dates and mucosal findings before the medication history becomes unclear.

AMT MEMORY AID: pain + mucosa + dusky skin = urgent SJS/TEN pathway.

MRCP TRAP: do not rechallenge a culprit drug after a severe cutaneous adverse reaction.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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Autoimmune blistering disease

ANATOMY / MECHANISM / PRESENTATION

Bullous pemphigoid causes pruritic tense blisters, often in older people; autoantibodies target BP180/BP230 at the basement-membrane zone. Pemphigus vulgaris causes fragile erosions and common oral involvement via desmoglein antibodies and intraepidermal acantholysis.

DIFFERENTIAL / INVESTIGATION

Biopsy active lesion for H&E and perilesional skin for DIF; serology helps classify and monitor in context. Linear basement-membrane IgG/C3 suggests pemphigoid; intercellular IgG suggests pemphigus. Dermatitis herpetiformis produces intensely itchy grouped lesions and granular IgA, linked to coeliac disease.



Tense pemphigoid-type blisters • SIMULATED

TREATMENT / PHARMACOLOGY

High-potency topical corticosteroids can control pemphigoid; systemic therapy depends on extent, frailty and infection risk. Pemphigus usually needs systemic corticosteroids plus steroid-sparing immunotherapy, often rituximab in specialist care. Assess nutrition, mucosal pain and infection.

COMPLICATIONS / NEXT STEP

Blistering therapies carry risks of infection, hyperglycaemia, osteoporosis and drug toxicity. Tense versus flaccid is a useful clue but biopsy and DIF establish the diagnosis.

CLINICAL PEARL

Pemphigus often causes painful oral erosions before skin blisters. Mucosal disease and fluid loss change admission decisions.

AMT MEMORY AID: pemphigoid = below epidermis, tense; pemphigus = within epidermis, fragile.

MRCP TRAP: sample intact perilesional skin for DIF, not the eroded centre.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

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Cutaneous lupus and dermatomyositis

ANATOMY / MECHANISM / PRESENTATION

Cutaneous lupus can be acute photosensitive malar, subacute annular/psoriasiform or chronic discoid with scarring. Dermatomyositis causes heliotrope change, Gottron papules and photo-distributed poikiloderma; muscle weakness may be absent in amyopathic disease.

DIFFERENTIAL / INVESTIGATION

Assess systemic lupus features including urine protein/blood, creatinine, complement, dsDNA and organ involvement. For dermatomyositis evaluate proximal strength, CK, myositis antibodies, lung disease and age-appropriate malignancy screening.



Malar-pattern lupus teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Photoprotection, smoking cessation and topical agents help skin disease. Hydroxychloroquine is a common systemic option for cutaneous lupus with retinal monitoring. Dermatomyositis often needs immunosuppression and multidisciplinary assessment of muscle, lung and swallowing.

COMPLICATIONS / NEXT STEP

An evolving rash may be a marker of renal or pulmonary disease. Avoid assuming a normal CK excludes amyopathic dermatomyositis; distinguish drug/photosensitivity mimics.

CLINICAL PEARL

Dermatomyositis can be amyopathic. Photosensitive rash with dysphagia or breathlessness still warrants muscle and lung evaluation.

AMT MEMORY AID: skin clue → examine muscle, lung, kidney and malignancy context.

MRCP TRAP: a malar eruption alone does not diagnose systemic lupus.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD cutaneous lupus; EULAR SLE

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Purpura and cutaneous vasculitis

ANATOMY / MECHANISM / PRESENTATION

Palpable purpura on dependent legs suggests small-vessel vasculitis; causes include drugs, infection, IgA vasculitis and systemic autoimmune disease. Retiform purpura, necrosis or livedoid change may signal occlusive disease, emboli or larger-vessel pathology.

DIFFERENTIAL / INVESTIGATION

Check urine dipstick/protein, renal function, blood pressure, full blood count, inflammatory markers, medication and infection history. Biopsy a fresh lesion for H&E and DIF; screen ANCA, complement, hepatitis and cryoglobulins selectively when clinical features justify them.



Palpable purpura on darker skin • SIMULATED

TREATMENT / PHARMACOLOGY

Treat the cause and assess organ involvement urgently. Skin-limited disease may resolve with support and trigger removal; nephritis, pulmonary haemorrhage, neuropathy or bowel ischaemia need specialist systemic therapy. Do not start immunosuppression before evaluating infection mimics.

COMPLICATIONS / NEXT STEP

IgA deposition supports IgA vasculitis but is time- and site-dependent. Normal platelets distinguish purpura from thrombocytopenic bleeding only in context; both can coexist.

CLINICAL PEARL

Purpura with proteinuria or haematuria is potentially systemic vasculitis even if the skin eruption appears limited.

AMT MEMORY AID: palpable purpura → urine, BP and systemic review.

MRCP TRAP: a skin biopsy does not substitute for renal assessment.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD vasculitis; EULAR ANCA

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Bacterial skin infection and necrotising disease

ANATOMY / MECHANISM / PRESENTATION

Cellulitis is usually unilateral, warm, tender and spreading, often after a skin break. Impetigo causes superficial crusts; erysipelas is more sharply demarcated. Bilateral red legs commonly suggest stasis or eczema rather than bilateral infection.

DIFFERENTIAL / INVESTIGATION

Assess sepsis, portal of entry, oedema, tinea pedis, diabetes and vascular supply. An abscess requires drainage. Pain out of proportion, rapid progression, bullae, anaesthesia or shock suggests necrotising infection despite deceptively mild early skin findings.



Cellulitis teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Use local/NICE antibiotic guidance by severity and allergy, with elevation and oedema management. Incise/drain an abscess when appropriate. Suspected necrotising fasciitis needs immediate surgery, resuscitation and broad antimicrobials; imaging must not delay exploration.

COMPLICATIONS / NEXT STEP

Recurrent cellulitis prevention includes treating interdigital fungal disease and chronic oedema. Consider DVT, lipodermatosclerosis, gout and contact dermatitis in a red painful limb.

CLINICAL PEARL

Bilateral red swollen legs more often reflect venous inflammation than bilateral cellulitis; compare warmth, tenderness, fever and symmetry.

AMT MEMORY AID: red leg = infection, inflammation, vascular or oedema mimic.

MRCP TRAP: normal early imaging does not make necrotising infection safe to observe.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

NICE NG141; UKHSA

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Viral and fungal skin disease

ANATOMY / MECHANISM / PRESENTATION

Herpes zoster causes unilateral dermatomal grouped vesicles and neuropathic pain; ophthalmic involvement threatens vision. HSV often recurs at mucocutaneous sites. Dermatophyte infection causes advancing scaly border; candida favours moist folds with satellite pustules.

DIFFERENTIAL / INVESTIGATION

Swab atypical vesicles for PCR when diagnosis alters treatment. KOH or fungal culture helps uncertain tinea, especially before systemic therapy. Widespread or atypical infection in an immunosuppressed patient prompts broader evaluation.



Dermatomal vesicles teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Antivirals are time-sensitive, particularly with ophthalmic, disseminated or immunocompromised zoster. Use topical antifungal for limited tinea and systemic treatment for selected hair/nail or extensive disease after confirmation and safety review. Avoid steroid monotherapy in tinea incognito.

COMPLICATIONS / NEXT STEP

Ophthalmic zoster needs same-day specialist assessment. Disseminated zoster or eczema herpeticum can cause severe systemic disease; assess isolation and IV therapy indications.

CLINICAL PEARL

Ophthalmic zoster, disseminated vesicles and immunosuppression lower the threshold for urgent referral and systemic antiviral therapy.

AMT MEMORY AID: grouped vesicles + dermatomal pain = zoster; ring scale = tinea.

MRCP TRAP: a steroid can mask tinea and enlarge the infection.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD; NICE CKS

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Scabies, infestations and pruritus

ANATOMY / MECHANISM / PRESENTATION

Scabies causes nocturnal pruritus with burrows or papules in finger webs, wrists, waist and genital skin; infants and older patients may have different distributions. Crusted scabies is highly contagious, often with immunosuppression or impaired sensation.

DIFFERENTIAL / INVESTIGATION

Clinical diagnosis uses contact history and examination; dermoscopy or microscopy can confirm mites when uncertain. Consider atopic disease, urticaria, cholestasis, renal failure, iron deficiency and haematological disease for generalised itch without a primary rash.



Interdigital scabies teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Treat the patient and close contacts simultaneously, using current local regimen with complete skin coverage and repeat timing; launder bedding/clothing appropriately. Crusted scabies needs specialist combined therapy and infection control. Post-scabetic itch can persist after eradication.

COMPLICATIONS / NEXT STEP

Reinfection usually reflects untreated contacts or incomplete application. Persistent new burrows differ from residual pruritus. Evaluate secondary bacterial infection in excoriated skin.

CLINICAL PEARL

An entire household treatment cycle can fail if one close contact is missed or treatment is washed off too soon.

AMT MEMORY AID: night itch + webs + household contacts.

MRCP TRAP: persistent itch alone does not prove treatment failure.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

UKHSA scabies; BAD

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Pigment disorders and skin of colour

ANATOMY / MECHANISM / PRESENTATION

Vitiligo produces sharply depigmented patches from melanocyte loss; Wood-lamp examination may clarify subtle areas. Post-inflammatory hyperpigmentation follows eczema, acne or trauma. Melasma is patterned facial hyperpigmentation linked to hormonal and light exposure.

DIFFERENTIAL / INVESTIGATION

Assess onset, distribution, mucosal or hair involvement, active spread, psychosocial impact and relevant autoimmune symptoms. Distinguish vitiligo from pityriasis versicolor, post-inflammatory hypopigmentation and rare depigmenting dermatoses.



Vitiligo on deeply pigmented skin • SIMULATED

TREATMENT / PHARMACOLOGY

Photoprotection is important for depigmented skin and dyspigmentation. Topical corticosteroid or calcineurin inhibitor and narrowband UVB are options for selected vitiligo; emerging targeted treatments depend on current eligibility. Address underlying inflammation before pigment therapies.

COMPLICATIONS / NEXT STEP

Skin cancer and inflammatory signs may present differently across skin tones. Examine palms, soles, nails and mucosa when relevant. Avoid judging inflammatory severity solely by redness.

CLINICAL PEARL

Post-inflammatory pigment change can outlast active eczema or acne; reassure accurately while controlling the primary inflammation.

AMT MEMORY AID: pigment lost (vitiligo) versus pigment redistributed after inflammation.

MRCP TRAP: acral or nail melanoma can occur in any skin tone.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD vitiligo; NICE

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Hair, scalp and nail disease

ANATOMY / MECHANISM / PRESENTATION

Alopecia areata causes non-scarring patches and may include exclamation-mark hairs and nail pitting. Androgenetic loss follows patterned miniaturisation; telogen effluvium is diffuse shedding after systemic stress. Scarring alopecia destroys follicular openings and needs early diagnosis.

DIFFERENTIAL / INVESTIGATION

Examine scalp for scale, pustules, perifollicular erythema and retained ostia. Consider iron deficiency, thyroid disease, drugs and nutritional status where appropriate. Tinea capitis needs fungal sampling and systemic treatment; traction and central centrifugal alopecia require culturally informed hair-practice history.



Alopecia areata on textured hair • SIMULATED

TREATMENT / PHARMACOLOGY

Intralesional or topical anti-inflammatory therapy may help limited alopecia areata; extensive disease can need specialist systemic treatment under current eligibility. Scarring alopecia warrants prompt biopsy of active edge and disease-specific treatment to prevent irreversible loss.

COMPLICATIONS / NEXT STEP

Nail pitting suggests psoriasis or alopecia areata; onycholysis has fungal, traumatic and psoriatic causes. Dark longitudinal nail bands with irregularity or periungual extension need melanoma assessment.

CLINICAL PEARL

Loss of follicular openings indicates scarring. Delay in diagnosis risks permanent alopecia, so biopsy the active margin.

AMT MEMORY AID: first ask whether follicular openings remain.

MRCP TRAP: do not label a scarring alopecia as simple telogen effluvium.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD alopecia guidance

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Keratinocyte cancer: basal and squamous

ANATOMY / MECHANISM / PRESENTATION

BCC is often pearly with rolled border and telangiectasia, though pigmented variants occur. Cutaneous SCC can be keratotic, indurated or ulcerated and carries greater metastatic potential, especially at high-risk sites or with immunosuppression.

DIFFERENTIAL / INVESTIGATION

Assess size, site, recurrence, immunosuppression and invasive features; confirm histology. Ear, lip, central face, perineural symptoms and poorly differentiated pathology alter risk. Actinic keratosis is a premalignant marker of UV damage and may be multiple.



Basal cell carcinoma teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Surgical excision is central for most invasive tumours; Mohs or specialist approaches suit selected high-risk BCC/SCC. Non-surgical options may suit carefully selected superficial BCC or field change. Advanced disease needs skin cancer MDT and current systemic treatment eligibility.

COMPLICATIONS / NEXT STEP

Hedgehog inhibitors for advanced BCC can cause muscle cramps, dysgeusia and alopecia; immunotherapy for advanced SCC can cause immune-mediated colitis, hepatitis, pneumonitis and endocrinopathy. Confirm current indication and monitoring.

CLINICAL PEARL

Perineural symptoms in SCC or high-risk BCC warrant urgent specialist review. Anatomical site changes management even when the lesion is small.

AMT MEMORY AID: BCC locally destructive; SCC has greater nodal risk.

MRCP TRAP: an ulcer that repeatedly “heals” and recurs may be malignancy.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD skin cancer; NICE

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Melanoma and suspicious pigmented lesions

ANATOMY / MECHANISM / PRESENTATION

Melanoma may show asymmetry, border irregularity, colour variation, evolution or a new lesion unlike others. Nodular disease may be symmetrical but rapidly growing. Acral melanoma affects palms, soles or nails and can be missed on dark skin.

DIFFERENTIAL / INVESTIGATION

Urgent specialist assessment and dermoscopy precede diagnostic excision, ideally full-thickness so Breslow depth and ulceration can be measured. Histology drives staging, wide local excision margins and sentinel node discussion. Imaging is stage-dependent.



Acral pigmented lesion teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Surgery is primary for localised disease. Higher-stage or unresectable disease is managed by MDT with surveillance, adjuvant/neoadjuvant systemic options and molecular testing as indicated. Immune checkpoint therapy and BRAF/MEK-targeted therapy have distinct eligibility and toxicities.

COMPLICATIONS / NEXT STEP

Checkpoint toxicity can involve colitis, hepatitis, pneumonitis and hypophysitis; BRAF/MEK therapy can cause pyrexia, rash, cardiac or ocular effects. A changing acral or longitudinal nail lesion needs biopsy, not reassurance from skin tone.

CLINICAL PEARL

Acral melanoma may be overlooked when clinicians focus only on sun-exposed skin. Examine soles, palms and nails deliberately.

AMT MEMORY AID: evolving lesion + depth + stage determines the pathway.

MRCP TRAP: ABCDE is imperfect for nodular and acral melanoma.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

NICE NG14 (2022 update)

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Dermatological emergencies and systemic clues

ANATOMY / MECHANISM / PRESENTATION

Erythroderma affects most skin surface and can result from eczema, psoriasis, drug reaction or lymphoma. Loss of barrier causes fluid and heat dysregulation, infection and high-output cardiac stress. Generalised pustular psoriasis and SJS/TEN also require urgent multidisciplinary care.

DIFFERENTIAL / INVESTIGATION

Assess vitals, temperature, fluid balance, mucosa, skin pain, detachment, drug exposures and lymphadenopathy. Examine for systemic clues: dermatomyositis, vasculitis, lupus, secondary syphilis or cutaneous lymphoma. Biopsy may require repeat sampling as inflammation can obscure cause.



Erythroderma teaching image • SIMULATED

TREATMENT / PHARMACOLOGY

Admit unstable patients, stop suspected drug, support fluids/nutrition/temperature and treat the cause with dermatology and relevant specialty input. Avoid reflex systemic corticosteroids before infection and drug reaction are characterised.

COMPLICATIONS / NEXT STEP

Watch for sepsis, electrolyte loss, thrombosis and ocular complications in severe blistering reactions. Document drug allergy precisely. Early escalation and repeated skin examination matter more than a single label.

CLINICAL PEARL

Erythroderma requires repeated fluid, temperature and haemodynamic assessment. The underlying diagnosis may remain uncertain on day one.

AMT MEMORY AID: extensive skin failure behaves like organ failure.

MRCP TRAP: diffuse redness is a severity sign, not a diagnosis.

AMT 60-SECOND RECALL: identify lesion, site, time course, systemic risk, targeted test and next action.

BAD SJS/TEN; NICE CG153

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Evidence, versions and clinical review

NICE CG153	Psoriasis assessment and management; reviewed 4 March 2025 https://www.nice.org.uk/guidance/cg153
NICE NG198	Acne vulgaris management; isotretinoin safety clarification 2023 https://www.nice.org.uk/guidance/ng198
NICE NG14	Melanoma assessment and management; 2022 update https://www.nice.org.uk/guidance/ng14
NICE NG141	Cellulitis and erysipelas antimicrobial prescribing https://www.nice.org.uk/guidance/ng141
BAD	Clinical guideline collection and disease guidance https://www.bad.org.uk/guidelines-and-standards/clinical-guidelines
BAD	SJS/TEN UK treatment guideline 2016 https://cdn.bad.org.uk/uploads/2021/12/29200149/SJS-TEN-summary-of-treatment-2-pages.pdf
MHRA	Isotretinoin safety measures; check current Drug Safety Update https://www.gov.uk/drug-safety-update

AUTHOR REVIEW AND APPROVAL

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Approval scope: production edition of this chapter. Clinical content and simulated educational illustrations retained from v1.

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