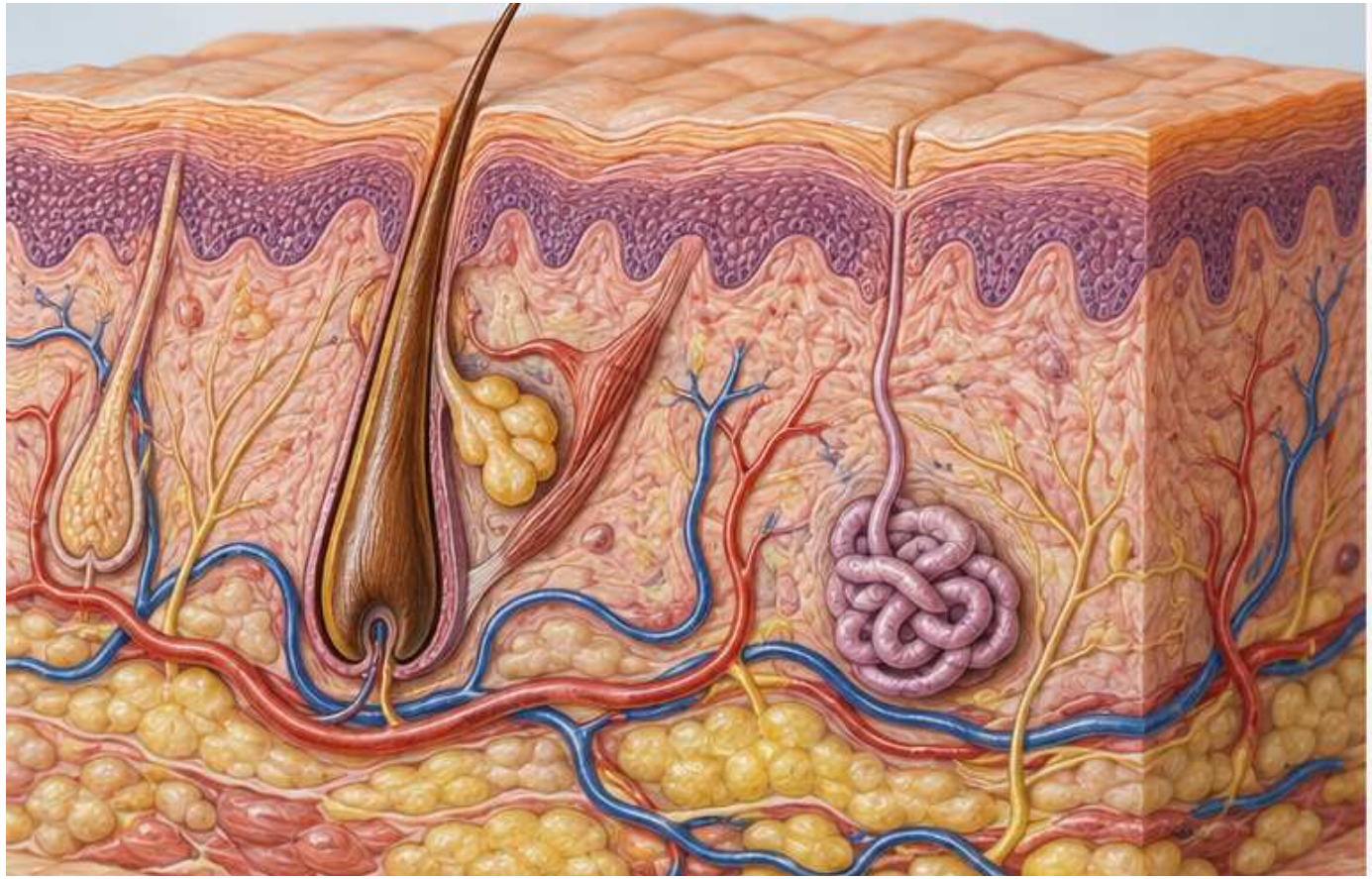
Skin architecture and barrier



EPIDERMIS, DERMIS AND APPENDAGES • SIMULATED

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.

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

RECOGNISE

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.

TEST / INTERPRET

Basement-membrane zone links epidermis to dermis via hemidesmosomes. Intraepidermal acantholysis produces fragile superficial blisters; subepidermal separation produces tense blisters.

TREAT

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.

MONITOR / COMPLICATION

Skin of colour may show less visible erythema and more post-inflammatory dyspigmentation. Assess warmth, scale, induration, tenderness and distribution, not colour alone.

BAD; dermatopathology core

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Lesion morphology and pattern recognition



MACULE, PAPULE, PLAQUE AND VESICLE • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

Map distribution, symmetry, flexures/extensors, photoexposure, dermatomes, palms/soles, mucosa and nails. Check if lesions blanch.

TREAT

Use history of onset, itch/pain, drug timing, fever and exposure to rank the differential. Photograph with consent and include a size scale.

MONITOR / COMPLICATION

A single diagnosis can produce several morphologies. A new painful purpuric or blistering eruption with fever, mucosal disease or systemic compromise is an emergency.

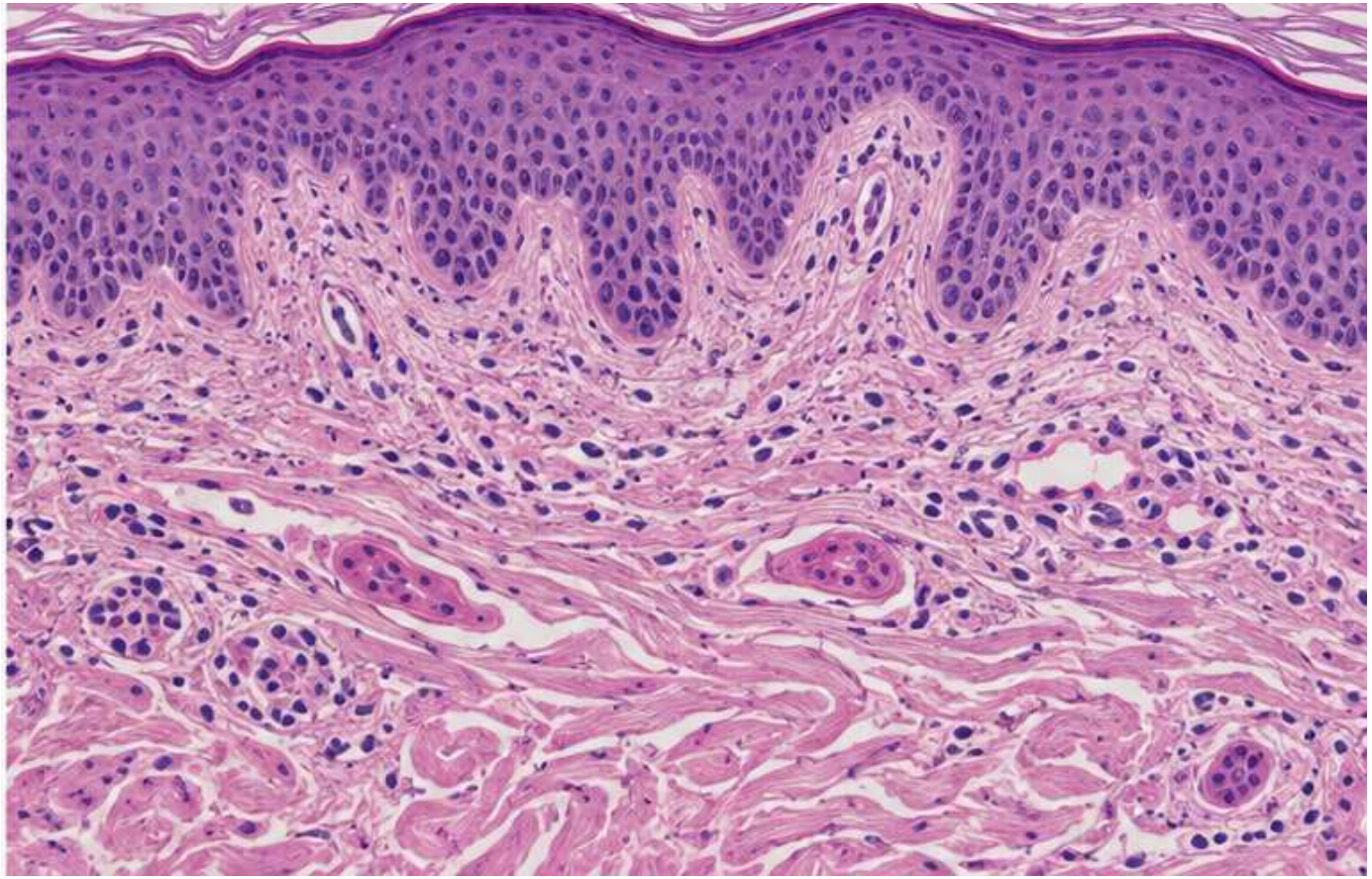
AMT MEMORY AID: lesion → distribution → time course → systemic clues → targeted test.

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

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

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Biopsy, histology and immunofluorescence



SKIN BIOPSY TEACHING MICROGRAPH • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

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.

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

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

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

BAD skin biopsy/DIF; NICE NG14

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Atopic dermatitis and skin barrier failure



FLEXURAL ECZEMA ON DARK SKIN • SIMULATED

RECOGNISE

Pruritic relapsing eczema often affects flexures, with xerosis, excoriation, lichenification and infection susceptibility. Atopic history supports but is not essential.

TEST / INTERPRET

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.

TREAT

Use liberal emollient, soap substitute, trigger reduction and site-appropriate topical corticosteroid. Proactive intermittent treatment of recurrent sites may reduce flares.

MONITOR / COMPLICATION

Review steroid technique, potency and quantity before escalation. Systemic immunomodulators require screening, vaccination planning and monitoring.

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.

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

NICE eczema CKS; BAD

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Allergic and irritant contact dermatitis



GEOMETRIC WATCH-STRAP DERMATITIS • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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

MONITOR / COMPLICATION

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.

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

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

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

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Psoriasis: immunology, phenotypes and treatment



PLAQUE PSORIASIS ON MEDIUM-BROWN SKIN • SIMULATED

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.

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

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

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.

NICE CG153 (2025 review)

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Acne, rosacea and follicular disorders



ACNE WITH POST-INFLAMMATORY CHANGE • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

Grade acne severity and scarring, pregnancy possibility, psychosocial harm and medication triggers. PCOS can contribute when acne coexists with hyperandrogenism.

TREAT

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.

MONITOR / COMPLICATION

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.

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

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

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

NICE NG198; BAD

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Urticaria, angioedema and mast-cell pathways



TRANSIENT WHEALS ON MEDIUM-BROWN SKIN • SIMULATED

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

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

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

RECOGNISE

Urticarial wheals usually resolve within 24 hours without residual marking. Angioedema affects deeper skin or mucosa.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

Review NSAIDs and physical triggers. Airway angioedema needs immediate escalation, irrespective of cause.

BAD urticaria; NICE TA339

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Adverse cutaneous drug reactions and SJS/TEN



DUSKY LESIONS WITH EPIDERMAL DETACHMENT • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

Assess mucosa, skin pain, BSA detachment, hydration, renal/liver function, blood count and infection. Record all recent drugs including intermittent antibiotics, anticonvulsants and allopurinol.

TREAT

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.

MONITOR / COMPLICATION

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.

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.

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

BAD SJS/TEN guideline 2016; MHRA

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Autoimmune blistering disease



TENSE PEMPHIGOID-TYPE BLISTERS • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

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.

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

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

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

BAD bullous disease guidance

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Cutaneous lupus and dermatomyositis



MALAR-PATTERN LUPUS TEACHING IMAGE • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

Photoprotection, smoking cessation and topical agents help skin disease. Hydroxychloroquine is a common systemic option for cutaneous lupus with retinal monitoring.

MONITOR / COMPLICATION

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

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

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

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

BAD cutaneous lupus; EULAR SLE

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Purpura and cutaneous vasculitis



PALPABLE PURPURA ON DARKER SKIN • SIMULATED

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

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

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

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

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

BAD vasculitis; EULAR ANCA

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Bacterial skin infection and necrotising disease



CELLULITIS TEACHING IMAGE • SIMULATED

RECOGNISE

Cellulitis is usually unilateral, warm, tender and spreading, often after a skin break. Impetigo causes superficial crusts; erysipelas is more sharply demarcated.

TEST / INTERPRET

Assess sepsis, portal of entry, oedema, tinea pedis, diabetes and vascular supply. An abscess requires drainage.

TREAT

Use local/NICE antibiotic guidance by severity and allergy, with elevation and oedema management. Incise/drain an abscess when appropriate.

MONITOR / COMPLICATION

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

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

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

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

NICE NG141; UKHSA

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Viral and fungal skin disease



DERMATOMAL VESICLES TEACHING IMAGE • SIMULATED

RECOGNISE

Herpes zoster causes unilateral dermatomal grouped vesicles and neuropathic pain; ophthalmic involvement threatens vision. HSV often recurs at mucocutaneous sites.

TEST / INTERPRET

Swab atypical vesicles for PCR when diagnosis alters treatment. KOH or fungal culture helps uncertain tinea, especially before systemic therapy.

TREAT

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.

MONITOR / COMPLICATION

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

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

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

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

BAD; NICE CKS

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Scabies, infestations and pruritus



INTERDIGITAL SCABIES TEACHING IMAGE • SIMULATED

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

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

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

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

Reinfection usually reflects untreated contacts or incomplete application. Persistent new burrows differ from residual pruritus.

UKHSA scabies; BAD

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Pigment disorders and skin of colour



VITILIGO ON DEEPLY PIGMENTED SKIN • SIMULATED

RECOGNISE

Vitiligo produces sharply depigmented patches from melanocyte loss; Wood-lamp examination may clarify subtle areas. Post-inflammatory hyperpigmentation follows eczema, acne or trauma.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

Skin cancer and inflammatory signs may present differently across skin tones. Examine palms, soles, nails and mucosa when relevant.

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

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

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

BAD vitiligo; NICE

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Hair, scalp and nail disease



ALOPECIA AREATA ON TEXTURED HAIR • SIMULATED

AMT MEMORY AID: first ask whether follicular openings remain.

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

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

RECOGNISE

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.

TEST / INTERPRET

Examine scalp for scale, pustules, perifollicular erythema and retained ostia. Consider iron deficiency, thyroid disease, drugs and nutritional status where appropriate.

TREAT

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.

MONITOR / COMPLICATION

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.

BAD alopecia guidance

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Keratinocyte cancer: basal and squamous



BASAL CELL CARCINOMA TEACHING IMAGE • SIMULATED

RECOGNISE

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.

TEST / INTERPRET

Assess size, site, recurrence, immunosuppression and invasive features; confirm histology. Ear, lip, central face, perineural symptoms and poorly differentiated pathology alter risk.

TREAT

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.

MONITOR / COMPLICATION

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.

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

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

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

BAD skin cancer; NICE

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Melanoma and suspicious pigmented lesions



ACRAL PIGMENTED LESION TEACHING IMAGE • SIMULATED

RECOGNISE

Melanoma may show asymmetry, border irregularity, colour variation, evolution or a new lesion unlike others. Nodular disease may be symmetrical but rapidly growing.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

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.

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

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

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

NICE NG14 (2022 update)

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION

Dermatological emergencies and systemic clues



ERYTHRODERMA TEACHING IMAGE • SIMULATED

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

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

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

RECOGNISE

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.

TEST / INTERPRET

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.

TREAT

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.

MONITOR / COMPLICATION

Watch for sepsis, electrolyte loss, thrombosis and ocular complications in severe blistering reactions. Document drug allergy precisely.

BAD SJS/TEN; NICE CG153

© Dr Blessing Dziwa MRCP(UK) MBA | APPROVED EDITION