Dermatology and Envenomation

Skin signs of systemic disease, eczema, snake, scorpion and spider envenomation, cutaneous infections and infestations, psoriasis, the blistering and drug emergencies, skin tumours, and sexually transmitted infections.

Cases 70–79 · 10,054 words

This block covers the dermatology teaching of both internal medicine courses. The **first course** supplies cases 70 and 72; the **second course** contributes ten dermatology lectures, covered here by cases 73 and 79. Dermatology is the block where the second course adds most, and it is taught largely by pattern recognition — so these cases lean more on comparison tables than the rest of the book.

Case	Lecture it serves
System opener: describing the skin	Introduction and terminology in dermatology
Case 70 — Cutaneous manifestations of systemic disease	Cutaneous manifestations of systemic disease
Case 71 — Eczema	Eczema and papulosquamous disease
Case 72 — Snake, scorpion and spider envenomation	Snake and spider envenomation
Case 73 — Bacterial and viral infections of the skin	Viral and bacterial infections of the skin
Case 74 — Fungal infections and infestations	Fungal infections and infestations of the skin
Case 75 — Psoriasis and the papulosquamous diseases	Eczema and papulosquamous disease
Case 76 — Blistering disease and the cutaneous drug emergencies	Vesiculobullous disease; drug eruptions and cutaneous emergencies
Case 77 — Skin tumours and the pigmented lesion	Benign and malignant skin tumours
Case 78 — Acne, rosacea, and disorders of pigmentation, hair and nails	Acne, rosacea and adnexal disorders; pigmentation, hair and nail disorders
Case 79 — Sexually transmitted infections	Sexually transmitted diseases

Purpura, the erythemas and urticarial disorders are distributed across the book rather than gathered into one case: purpura in Case 52 and Case 61, erythema nodosum in Case 70, erythema multiforme and urticaria in Case 76.

System Opener · Describing the Skin

Dermatology is learned as a vocabulary before it is learned as a set of diseases. A student who can describe a rash accurately can be helped by a dermatologist over the telephone; a student who says "a red rash on the leg" cannot.

The history

- **Time course:** when it started, where it started, how it spread, and whether it is continuous or episodic.
- **Symptoms:** itch — and **how much it disturbs sleep**, which is the best measure of severity — pain, burning, or nothing at all.
- **Provoking factors:** sunlight, heat and sweat, water, friction, occupation, cosmetics, contacts, and foods.
- **Drugs.** Ask about everything started in the preceding two months, including over-the-counter and traditional preparations. **A morbilliform drug eruption typically appears 7–14 days after a new drug**, and sooner on re-exposure — so the culprit is often not the most recently started medicine.
- **Previous treatment — and specifically how much and for how long.** "I tried a steroid cream" usually means a small tube used for two days, which is not a treatment failure.
- **Personal and family history** of atopy, psoriasis and autoimmune disease; systemic symptoms; and **impact on sleep, work, school and mood**, which is substantial and routinely unasked.

The examination

- **Examine the whole skin in good light** — including the scalp, behind the ears, the nails, the mouth, the genitals, the palms and soles, and between the toes. Rashes are diagnosed by their distribution, and the diagnostic part is frequently the part left covered.
- **Then describe in a fixed order:** site and distribution → configuration → number → **primary lesion morphology** → secondary change → colour → size → surface → margin.
- **Palpate** — temperature, tenderness, texture, depth — and press with a glass slide to test whether the lesion **blanches**.

The primary lesions

Term	Definition
Macule	Flat area of altered colour, under 1 cm
Patch	Flat area of altered colour, over 1 cm
Papule	Raised solid lesion, under 1 cm
Plaque	Raised flat-topped lesion, over 1 cm
Nodule	Raised solid lesion over 1 cm, extending deeper into dermis or subcutis
Vesicle / bulla	Fluid-filled lesion — vesicle small, bulla over 1 cm
Pustule	Pus-filled lesion — which does not necessarily mean infection
Wheal	Transient oedematous papule or plaque, resolving within 24 hours
Purpura	Non-blanching extravasated blood. Petechiae are pinpoint; ecchymoses are larger
Telangiectasia	Visible permanently dilated small vessel

Secondary change and configuration

- **Secondary change:** scale, crust, excoriation, **lichenification** (thickened skin with exaggerated markings from chronic rubbing), fissure, **erosion** (loss of epidermis only, heals without scarring), **ulcer** (extends into dermis, heals with a scar), atrophy, scar, sclerosis.
- **Configuration:** annular, linear, grouped or herpetiform, target, reticulate, serpiginous.
- **Distribution:** flexural, extensor, photodistributed, dermatomal, acral, symmetrical — each of which narrows the differential sharply.

THE DERMATOLOGICAL EMERGENCIES

Erythroderma — erythema over more than 90% of the body surface, with fluid loss, heat loss and high-output cardiac failure.

Stevens–Johnson syndrome and toxic epidermal necrolysis — painful skin, **mucosal involvement at two or more sites**, and a positive Nikolsky sign, days to weeks after a new drug. Stop the drug and transfer.

Meningococcal purpura — non-blanching rash with fever. Antibiotics immediately.

Necrotising fasciitis — **pain out of proportion to the visible signs**, rapid progression, systemic toxicity, crepitus. A surgical emergency.

Eczema herpeticum — monomorphic punched-out erosions in a patient with eczema, who is unwell. Aciclovir urgently.

Drug reaction with eosinophilia and systemic symptoms — rash, fever, facial oedema, eosinophilia and organ involvement, typically 2–8 weeks after the drug.

Angioedema involving the airway.

Case 70 · Cutaneous Manifestations of Systemic Disease

CLINICAL VIGNETTE

A **34-year-old woman** is referred with three weeks of **tender red raised lesions on both shins**. They are painful to touch, have not ulcerated, and the older ones are fading through the colours of a bruise. Over the same period she has had a dry cough, mild breathlessness on exertion, intermittent fever and painful swollen ankles.

On examination: multiple tender erythematous **nodules** over both shins, poorly demarcated, warm, none ulcerated. Both ankles are swollen and tender. Temperature 37.8 °C. Chest is clear.

Chest radiograph: bilateral hilar lymphadenopathy.

READING THE SKIN AS A SYSTEMIC SIGN

The skin lesions are **erythema nodosum** — a septal panniculitis that is a **reaction pattern, not a diagnosis**. The task is always to find what provoked it.

Here, erythema nodosum with **bilateral hilar lymphadenopathy, ankle arthritis and fever** constitutes **Löfgren syndrome**, an acute presentation of sarcoidosis with an excellent prognosis.

1. Erythema Nodosum — Find the Cause

Cause	How to pursue it
Sarcoidosis	Chest radiograph, serum calcium, angiotensin-converting enzyme, ophthalmology review
Streptococcal infection	Throat swab and antistreptolysin O titre — the commonest identifiable cause in younger patients
Tuberculosis	Chest radiograph, interferon-gamma release assay or tuberculin test, sputum — and it must be excluded before corticosteroids
Brucellosis	Serology and blood cultures — a realistic cause in this region, and easily missed
Inflammatory bowel disease	Bowel symptoms, faecal calprotectin, endoscopy
Drugs	Sulfonamides, penicillins, the combined oral contraceptive
Pregnancy, Behçet disease, fungal infection, malignancy	Pregnancy test; oral and genital ulceration; exposure history
Idiopathic	Up to half of all cases, after the above have been excluded

2. Reading the Skin in Systemic Disease

The rest of this case is a reference table. Learn to recognise these, because in each of them **the skin sign appears before the diagnosis is made**.

Endocrine and metabolic

Sign	Disease
Necrobiosis lipoidica — shiny yellow-brown atrophic plaques with telangiectasia on the shins	Diabetes mellitus
Acanthosis nigricans — velvety hyperpigmented thickening of the neck, axillae and groin	Insulin resistance and type 2 diabetes. Sudden, extensive or mucosal involvement suggests underlying malignancy, classically gastric adenocarcinoma
Granuloma annulare, eruptive xanthomas, diabetic dermopathy, recurrent candidiasis	Diabetes mellitus
Pretibial myxoedema and thyroid acropachy	Graves disease
Dry coarse skin, hair loss, carotenaemia	Hypothyroidism
Pigmentation of buccal mucosa, palmar creases and scars	Adrenal insufficiency
Thin skin, easy bruising, purple striae, proximal myopathy	Cushing syndrome
Xanthelasma and tendon xanthomata	Hyperlipidaemia — particularly familial hypercholesterolaemia

Gastrointestinal, hepatic and renal

Sign	Disease
Spider naevi, palmar erythema, jaundice, scratch marks, leuconychia	Chronic liver disease
Pyoderma gangrenosum — a rapidly enlarging painful ulcer with a violaceous undermined border	Inflammatory bowel disease, rheumatoid arthritis, haematological malignancy. Do not debride it — surgery worsens it through pathergy. It is treated with immunosuppression, not with a scalpel
Erythema nodosum, aphthous ulceration	Inflammatory bowel disease
Generalised pruritus, half-and-half nails, calciphylaxis	Chronic kidney disease

Connective tissue disease and malignancy

Sign	Disease
Malar rash sparing the nasolabial folds; discoid lesions that scar	Systemic lupus erythematosus
Heliotrope rash of the eyelids and Gottron papules over the knuckles	Dermatomyositis — and in an adult this mandates a search for underlying malignancy
Sclerodactyly, calcinosis, Raynaud phenomenon, telangiectasia	Systemic sclerosis
Palpable purpura on dependent areas	Small vessel vasculitis — check urine and renal function
Acquired ichthyosis, generalised pruritus, erythema gyratum repens, sudden eruption of seborrhoeic keratoses	Underlying malignancy — the paraneoplastic dermatoses

Infection

Sign	Disease
Non-blanching purpura with fever	Meningococcal sepsis — antibiotics now
Splinter haemorrhages, Osler nodes, Janeway lesions	Infective endocarditis
Rash involving the palms and soles	Secondary syphilis, rickettsial infection, hand-foot-and-mouth disease
A chronic painless ulcer with a raised indurated border on an exposed site	Cutaneous leishmaniasis — endemic in parts of this region and frequently treated as a bacterial ulcer for months before the diagnosis is considered

3. Investigation and Management of This Patient

- **Chest radiograph** — done, and diagnostic of the syndrome in this context. Serum **calcium** (hypercalcaemia occurs in sarcoidosis), angiotensin-converting enzyme, inflammatory markers, complete blood count, liver and renal function, **urinalysis**.
- **Exclude the alternative causes:** throat swab and antistreptolysin O titre, **tuberculosis screening**, and **brucella serology**.
- **Ophthalmology review for uveitis** and an electrocardiogram for cardiac involvement — both silent and both important in sarcoidosis.
- **Biopsy of the nodules is not usually necessary** where the clinical picture is characteristic, and biopsy of erythema nodosum shows only a septal panniculitis without identifying the cause.
- **Löfgren syndrome resolves spontaneously in most patients over weeks to months.** Treat with rest, leg elevation, compression and anti-inflammatory drugs.
- **Corticosteroids are reserved for organ-threatening disease** — ocular, cardiac or neurological involvement, hypercalcaemia, or progressive pulmonary disease. **Exclude tuberculosis before starting them.**

4. Teaching Points and Viva Questions

- Erythema nodosum is a reaction pattern. The diagnosis is whatever caused it.
- In this region, tuberculosis and brucellosis belong at the top of that list, not the bottom.
- Never debride pyoderma gangrenosum.
- Adult dermatomyositis requires a malignancy screen.
- Sudden extensive acanthosis nigricans is a warning sign, not a metabolic curiosity.
- A chronic ulcer with a raised border on an exposed site here is leishmaniasis until proved otherwise.

Questions you should be able to answer:

- Name six causes of erythema nodosum and the test you would use for each.

- Why must tuberculosis be excluded before you prescribe prednisolone here?
- A surgical colleague proposes debriding a rapidly enlarging leg ulcer with a purple undermined edge. What do you say?
- A 62-year-old man has a heliotrope rash and proximal weakness. What is your next step?
- What is the prognosis of this syndrome, and what will you tell her?

Case 71 · Eczema

CLINICAL VIGNETTE

A **22-year-old woman** has had an itchy rash since infancy, currently at its worst for years. She scratches through the night, sleeps about four hours, and has taken three days off work. She has asthma and allergic rhinitis.

She was given "a steroid cream" but stopped it after two days because she had read that it thins the skin. She washes with soap and takes hot showers, which relieve the itch briefly.

On examination: thickened, **lichenified**, excoriated plaques in both antecubital and popliteal fossae, on the neck and on the backs of the hands, with generalised dry scaly skin. Over the left forearm there is **golden-yellow crusting**. There are infra-orbital folds beneath both eyes.

1. Focused History

- **Age of onset and how the distribution has changed** — facial and extensor in infancy, flexural in older children and adults, and often predominantly on the hands in adults.
- **Itch severity measured by sleep loss and time off work or school**, which is the most honest measure of how bad the disease is.
- **Triggers:** heat and sweat, soaps and detergents, wool, house dust mite, stress, infection, and — in a minority of young children only — foods.
- **Occupation and hobbies**, particularly wet work and contact with irritants, which drive adult hand eczema.
- **Current treatment, quantified.** How large a tube, how often, over how many days, and to which areas. Then ask directly what worries her about using it.

2. Differential Diagnosis

Diagnosis	The feature that discriminates it
Scabies	Intense nocturnal itch, burrows in finger web spaces and wrists, involvement of genitals and axillae, and other members of the household itching. Commonly missed and treated as eczema for months.
Tinea	Annular with a raised scaly advancing edge and central clearing, often asymmetrical. Topical steroid makes it spread while reducing the inflammation — tinea incognito. Scrape it before you treat it.
Psoriasis	Well-demarcated plaques with silvery scale on extensor surfaces, scalp and natal cleft, with nail pitting and onycholysis.
Contact dermatitis	Distribution follows the exposure — under a watch strap, at the hairline, on the hands. Patch testing distinguishes allergic from irritant.
Seborrhoeic dermatitis	Greasy scale in the scalp, eyebrows, nasolabial folds and behind the ears.
Cutaneous T-cell lymphoma	An older patient with persistent, atypical, fixed patches that do not respond to adequate treatment. Biopsy.

3. Investigations

- **The diagnosis is clinical.** Investigations are directed at complications and at the mimics.

- Bacterial swab where there is crusting or weeping, as here; **viral swab and polymerase chain reaction if eczema herpeticum is suspected**; **skin scrapings for fungal microscopy and culture** where the rash is asymmetrical or atypical.
- **Patch testing** where allergic contact dermatitis is suspected. Total and specific immunoglobulin E rarely change management in adults.

4. Management

THE CENTRAL PROBLEM IN ECZEMA CARE

Eczema is under-treated far more often than it is over-treated.

The usual pattern is a small tube of a weak steroid, used for two days, applied to a fraction of the affected skin, stopped early through fear — and then recorded as treatment failure. **Almost all of what follows is about giving enough, of the right strength, for long enough.**

Emollients — prescribe by the kilogram

- **Apply liberally several times daily, and continue when the skin is clear.** For widespread disease an adult needs something of the order of 500 g per month; a prescription for a 50 g tube is not treatment.
- **Use a soap substitute and stop soap and shower gel entirely.** Warm rather than hot water — hot water relieves itch for minutes and worsens it for hours.
- Apply emollient in the direction of hair growth to avoid folliculitis, and leave a gap of around 20–30 minutes between emollient and topical steroid.

Topical corticosteroids — match potency to site and severity

- **Mild or moderate potency for the face, flexures and genitals; potent preparations for the body and limbs; very potent short courses for thickened lichenified areas** such as this patient's.
- **Teach the fingertip unit** — the amount squeezed from the tip of an adult index finger to the first crease treats an area equal to two adult palms. Give her a written quantity, not "apply sparingly", which is the phrase that causes most under-treatment.
- **Proactive maintenance:** once controlled, applying topical steroid or a calcineurin inhibitor twice weekly to sites that habitually flare substantially reduces relapse.
- **Topical calcineurin inhibitors** — tacrolimus and pimecrolimus — for the face, eyelids and flexures, and as a steroid-sparing agent in areas needing long-term treatment.

STEROID PHOBIA

She stopped an appropriate treatment because of a fear she had read about. **Address it directly rather than repeating the prescription.**

Explain the distinction that matters: **short courses of appropriately matched potency, supervised, are safe** — whereas long-term unsupervised use of potent steroid on the face and flexures does cause atrophy. Then explain the harms of under-treatment: sleeplessness, infection, lichenification, time off work, and the mood consequences of years of itch.

A patient who understands why she is using it will use enough of it.

The rest

- **Treat the infection.** Golden crusting is impetiginised eczema — oral flucloxacillin, and consider antiseptic or dilute bleach baths where infection recurs.
- **Sedating antihistamines may help sleep during a bad flare.** Non-sedating antihistamines do little for the itch of eczema, although they help coexisting urticaria and rhinitis.
- Bandaging and wet wraps for severe lichenified disease; treat coexisting asthma and rhinitis.
- **Refractory disease:** phototherapy, ciclosporin, methotrexate, azathioprine, and the newer targeted agents — **dupilumab and Janus kinase inhibitors** — which have changed outcomes in severe atopic dermatitis.
- **Give a written treatment plan** setting out what to use where, how much, and what to do in a flare. Arrange follow-up rather than a single consultation.

ECZEMA HERPETICUM — THE COMPLICATION TO RECOGNISE

Rapidly worsening, painful eczema with clusters of monomorphic punched-out erosions and vesicles, often with fever and malaise, in a patient with atopic dermatitis. It is disseminated herpes simplex infection of eczematous skin.

Start aciclovir urgently. If it involves the skin around the eye, arrange same-day ophthalmology review. It can be fatal, and it is regularly mistaken for bacterial infection and treated with antibiotics alone while it spreads.

5. Teaching Points and Viva Questions

- Prescribe emollient by the kilogram and steroid by the fingertip unit. Never write "apply sparingly".
- Ask what worries the patient about steroids, then answer it.
- Scabies and tinea are the two mimics that get worse when you treat them as eczema.
- Golden crust means bacterial infection; punched-out erosions mean herpes and an urgent antiviral.
- Measure severity by sleep loss and days lost, not by the appearance alone.

Questions you should be able to answer:

- How much emollient should she be using in a month, and how would you write the prescription?
- Which potency would you choose for her face, and which for her lichenified forearms? Why the difference?
- She asks whether steroids will thin her skin. Answer her.
- Her rash is asymmetrical, annular and spreading despite treatment. What has happened?
- She returns febrile with painful clustered erosions. What is your diagnosis and immediate action?

Case 72 · Snake, Scorpion and Spider Envenomation

CLINICAL VIGNETTE

A **28-year-old man** was bitten on the back of the right hand two hours ago while moving rocks on farmland. He saw a snake but cannot describe it. A companion tied a cord tightly around his forearm and made two small cuts over the bite.

The hand and forearm are now painfully swollen to just below the elbow. He has noticed **bleeding from his gums** and oozing from the cuts.

On examination: heart rate 108/min, blood pressure 108/70 mmHg, alert. Tense swelling of the hand and forearm with bruising around two fang marks. **Bleeding from the gums and from the venepuncture site.** No ptosis, normal eye movements, normal single-breath count.

Twenty-minute whole blood clotting test: the blood has not clotted. Platelets 78 × 10^9/L · INR unrecordable · fibrinogen low · creatinine rising.

FIRST AID — AND THE FIRST AID THAT CAUSES HARM

The companion applied a **tight tourniquet** and **incised the wound**. Both are harmful and both are still widely practised.

Do not: apply a tourniquet or tight band · cut or incise · suck the wound · apply ice, heat, electric shock or chemicals · use traditional or herbal applications · attempt to capture or kill the snake.

Do: reassure the patient, **immobilise the limb in a neutral position with a splint**, remove rings, watches and anything constrictive, keep the patient still, and transport urgently to hospital.

Releasing a long-standing tight tourniquet can release a bolus of venom, so do so **with resuscitation facilities to hand**.

1. Focused History

- **Time of the bite** and the interval since; the site; and what the patient was doing.
- **Any description of the snake** — but **do not delay treatment to identify it, and never ask anyone to bring in the animal.** Treatment is guided by the clinical syndrome.
- **First aid already applied**, including traditional remedies.
- **Symptoms of systemic envenoming:**
  - **Haemotoxic** — bleeding from gums, nose, wounds or venepuncture sites, haematuria, bruising. Characteristic of viper envenoming, which predominates in this region.
  - **Neurotoxic** — **ptosis first**, then diplopia, difficulty swallowing and speaking, then limb and respiratory muscle weakness. Characteristic of cobra and related elapid envenoming.
  - **Myotoxic** — severe muscle pain and dark urine.
  - **Systemic** — vomiting, abdominal pain, collapse, shock.
- **Comorbidity, anticoagulant use, previous antivenom exposure** — which increases reaction risk — and tetanus immunisation status.

2. Physical Examination

THE SIMPLEST USEFUL THING YOU WILL DO

Draw a line at the leading edge of the swelling and write the time beside it. Repeat hourly.

Progression of swelling is the most useful bedside measure of ongoing envenoming, and it is entirely lost if nobody marks the starting point. Measure and record the limb circumference at a fixed landmark at the same time.

- **Bleeding:** inspect gums, nose, the bite site, venepuncture sites and the urine.
- **Neurological: ptosis is the earliest sign of neurotoxicity** — look for it deliberately and repeatedly. Then eye movements, bulbar function, neck flexion strength, **single-breath count and forced vital capacity**.
- Vital signs, perfusion, tender regional lymphadenopathy, local blistering and necrosis, and assessment for compartment syndrome.

3. Investigations

THE 20-MINUTE WHOLE BLOOD CLOTTING TEST

Place **2 mL of fresh venous blood in a clean, dry glass tube**, leave it completely undisturbed for **20 minutes**, then tip it gently.

If the blood has not clotted, there is a coagulopathy indicating systemic envenoming by a viper, and antivenom is indicated.

It requires no laboratory, no reagents and no electricity, and it remains one of the most useful bedside tests in medicine wherever snakebite is common. The tube must be glass and must not be shaken.

- Complete blood count, coagulation screen with **fibrinogen** and D-dimer, urea, electrolytes and creatinine, **creatine kinase**, urinalysis for blood and myoglobin, electrocardiogram, group and save, blood gas and lactate.
- **Repeat the clotting test and the bloods every 6 hours, and 6 hours after each dose of antivenom**, since coagulopathy can recur as venom continues to be absorbed from the bite site.

4. Management

- Airway, breathing, circulation. **Intravenous access in an unaffected limb.** Continuous monitoring.

Antivenom — the only specific treatment

	Detail
Indications	Systemic envenoming — coagulopathy or spontaneous bleeding, neurotoxicity, cardiovascular instability, acute kidney injury, rhabdomyolysis — or severe local envenoming with rapidly progressive swelling involving more than half the bitten limb.
Not indicated	A bite with no evidence of envenoming. Dry bites are common. Observe for at least 24 hours with serial clotting tests rather than treating pre-emptively.
Administration	Intravenously, according to the product instructions. Draw up adrenaline before you start and have resuscitation equipment at the bedside — anaphylaxis is a real and immediate risk.
Repeat dosing	If coagulopathy persists on repeat testing after 6 hours, or if neurotoxicity progresses. The dose is the same for children as for adults — the amount of venom is the same.

Supportive care

- **Analgesia with paracetamol or opioids. Avoid non-steroidal anti-inflammatory drugs and aspirin**, which worsen bleeding.
- **Avoid intramuscular injections** entirely while coagulopathy persists. Tetanus prophylaxis once clotting has been restored.
- **Antibiotics are not routine** — give them only where there is evidence of infection. Clean and dress the wound; elevate the limb once antivenom has been given.
- **Blood products only for active bleeding, and only after antivenom** — giving clotting factors while venom is still circulating simply consumes them.
- **Neurotoxic envenoming:** monitor the vital capacity as in the neurology block, prepare for intubation, and consider a trial of an anticholinesterase in cobra envenoming.
- **Compartment syndrome is over-diagnosed** in swollen envenomed limbs. Measure the pressure rather than assuming. **Fasciotomy in an uncorrected coagulopathy causes catastrophic bleeding**, and antivenom rather than surgery is the treatment for the swelling itself.
- Support renal function; watch for **serum sickness 5–10 days after antivenom**; and give prevention advice — footwear, lighting, care when moving rocks and firewood.

SCORPION ENVENOMATION

Scorpion sting is far commoner than snakebite in this region, and children are at greatest risk.

The picture: severe local pain with strikingly little to see, followed in significant envenoming by an **autonomic storm** — sweating, salivation, vomiting, agitation, hypertension then shock, pulmonary oedema and myocarditis.

Management: analgesia including local anaesthetic infiltration; close cardiorespiratory monitoring in children; treatment of the autonomic and cardiac effects with critical care support; and scorpion antivenom according to local protocol. **A child with a scorpion sting and systemic features needs monitoring, not reassurance and discharge.**

Spider bites

- **Widow spiders** (*Latrodectus*) — severe generalised muscle cramps and **abdominal rigidity that mimics peritonitis**, with sweating and hypertension. Treat with analgesia, benzodiazepines and antivenom where available.
- **Recluse spiders** (*Loxosceles*) — a progressive necrotic ulcer, occasionally with systemic haemolysis. Management is supportive wound care; **early surgical excision is not helpful** and should be deferred until the lesion has demarcated.
- Most reported "spider bites" are not spider bites. **A necrotic skin lesion attributed to a spider is more often a staphylococcal abscess**, and should be managed as one unless the spider was actually seen.

5. Teaching Points and Viva Questions

- No tourniquets, no incisions, no suction. Immobilise, splint and transport.
- Mark the swelling edge and the time. Repeat hourly.
- The 20-minute whole blood clotting test needs a glass tube and nothing else.

- Antivenom treats systemic envenoming, not the bite. Dry bites are observed, not treated.
- Adrenaline drawn up before the antivenom runs.
- Ptosis is the first sign of neurotoxicity, and the vital capacity is the observation that follows it.
- No anti-inflammatory drugs, no intramuscular injections, and no fasciotomy in an uncorrected coagulopathy.

Questions you should be able to answer:

- The tourniquet has been on for two hours. How do you remove it and why the caution?
- Describe how you would perform the 20-minute whole blood clotting test.
- He has swelling to the elbow but a normal clotting test and no systemic features. Do you give antivenom?
- The limb is tense and painful. A colleague suggests fasciotomy. What is your response?
- A 6-year-old is brought in an hour after a scorpion sting, sweating and vomiting. What is your plan?

Case 73 · Bacterial and Viral Infections of the Skin

CLINICAL VIGNETTE

A **58-year-old man** with type 2 diabetes has had three days of a hot, red, painful, swollen left lower leg which is spreading upwards. There is a small fissure between the fourth and fifth toes.

But: the pain is **far worse than the appearance suggests**, the skin over the shin has become **dusky purple with two haemorrhagic blisters**, there is **crepitus** on palpation, and the induration extends well beyond the visible erythema.

On examination: temperature 38.4 °C, heart rate 124/min, blood pressure 94/56 mmHg, confused. **Lactate 4.2 mmol/L.**

NECROTISING FASCIITIS — RECOGNISE IT IN THE FIRST MINUTE

This is **not** cellulitis. The features that give it away are the ones the examination was designed to find:

Pain out of proportion to the appearance · rapid progression over hours · systemic toxicity out of proportion to the skin · dusky or purple discolouration · **haemorrhagic bullae · crepitus ·** anaesthesia of the overlying skin · woody induration extending beyond the erythema.

The diagnosis is clinical and surgical. Do not wait for imaging and do not use a score to rule it out — the LRINEC score supports the diagnosis but does not exclude it.

Immediate surgical exploration and debridement · broad-spectrum antibiotics with clindamycin added for toxin suppression · aggressive resuscitation · critical care. Survival is measured against the time to the first operation.

1. Cellulitis and Its Mimics

- **Cellulitis** is unilateral, warm, tender, spreading, with an ill-defined edge. **Erysipelas** is sharply demarcated, raised and bright red, often on the face, and usually streptococcal.

TWO THINGS ROUTINELY GOT WRONG

Bilateral "cellulitis" is almost never cellulitis. Consider venous eczema, stasis dermatitis, lymphoedema, and acute lipodermatosclerosis — none of which need antibiotics.

And always look for the portal of entry: interdigital fissures and tinea pedis, an ulcer, eczema, lymphoedema, an insect bite, or trauma.

Treating the tinea pedis is what prevents the next episode, and it is the step most often omitted in a patient with recurrent cellulitis.

- Other mimics: deep vein thrombosis, acute gout, contact dermatitis, erythema nodosum (Case 70).
- **Practical management:** mark the edge of the erythema with the date and time and review it; elevate the limb; treat the portal of entry; choose the antibiotic by severity and local resistance, **considering resistant staphylococcal cover where prevalent**; look for an abscess, because **an abscess is drained and antibiotics are the adjunct**; and consider osteomyelitis in a diabetic foot (Case 25).

2. The Other Bacterial Infections

- **Impetigo** — golden crust, staphylococcal or streptococcal; topical antiseptic or antibiotic for limited disease, oral therapy if extensive. **Bullous impetigo** in children.
- **Folliculitis, furuncle and carbuncle**; **abscess — incision and drainage**; and **hidradenitis suppurativa** (Case 78), which is repeatedly misdiagnosed as recurrent boils.
- **Staphylococcal scalded skin syndrome** and **toxic shock syndrome** — toxin-mediated, with fever, rash, desquamation and shock; supportive care with antitoxin-suppressing antibiotics.

3. The Viral Infections

Infection	Points that matter
Herpes simplex	Primary gingivostomatitis; recurrent cold sores; herpetic whitlow; eczema herpeticum (Case 71); a common trigger of erythema multiforme. Aciclovir, with suppression for frequent recurrence.
Varicella	More severe in adults — varicella pneumonia, hepatitis, encephalitis. Dangerous in pregnancy, the immunosuppressed and neonates. Treat adults and at-risk patients with aciclovir; consider immunoglobulin for exposed at-risk contacts.
Herpes zoster	Dermatomal, with pain often preceding the rash by days. Treat within 72 hours to reduce post-herpetic neuralgia. Ophthalmic zoster with a lesion on the nose — Hutchinson sign — needs same-day ophthalmology. Ramsay Hunt syndrome with facial palsy and ear vesicles. Multidermatomal or disseminated zoster means immunosuppression — test for HIV (Case 45). Vaccination in older adults.
Warts and molluscum contagiosum	Self-limiting; destructive or topical therapy. Extensive molluscum in an adult should prompt an HIV test.
Hand-foot-and-mouth disease; measles	Measles — prodrome, Koplik spots, cephalocaudal rash, with pneumonia, otitis and encephalitis as complications. Notify, and give vitamin A where indicated.

4. Teaching Points and Viva Questions

- Pain out of proportion to the appearance means the operating theatre, not a scan.
- Bilateral leg redness is almost never cellulitis.
- Find and treat the portal of entry, or the cellulitis will recur.
- Drain the abscess.
- Treat zoster within 72 hours, and refer ophthalmic zoster the same day.
- Disseminated zoster or florid molluscum in an adult means test for HIV.

Questions you should be able to answer:

- Which six features told you this was not simple cellulitis?
- A patient has red, scaly, itchy skin on both lower legs and no fever. What is the diagnosis and what would you not prescribe?
- He has had four episodes of cellulitis in the same leg this year. What have you missed?
- A 40-year-old has zoster in three separate dermatomes. What do you test for?
- Why does the timing of aciclovir matter in shingles?

Case 74 • Fungal Infections and Infestations

CLINICAL VIGNETTE

A **30-year-old woman** has had an itchy rash on the trunk for three months. It began as a small scaly ring with a raised edge, and a pharmacist supplied a potent steroid cream. **The itch improved for a few days, and then the rash spread widely** and lost its scaly border. She now has extensive, poorly demarcated erythema across the trunk and both flanks.

Her six-year-old son has a scaly patch on the scalp with hair loss.

Skin scrapings: fungal hyphae seen on microscopy.

TINEA INCOGNITO

A topical corticosteroid suppresses the inflammation while the fungus spreads. The itch settles briefly, the diagnostic scaly advancing edge disappears, the rash becomes widespread and poorly demarcated — and it now looks like eczema, so more steroid is applied.

Scrape before you steroid. Any rash that is **annular, asymmetrical or unilateral**, or that **worsens or spreads on a topical steroid**, is tinea until scrapings prove otherwise.

And scalp tinea in a child needs oral therapy — topical treatment will not cure it.

1. The Dermatophyte Infections

- **The classical appearance:** an annular scaly plaque with a **raised advancing edge and central clearing**, usually asymmetrical or unilateral.
- By site: **corporis, cruris, pedis, manuum, faciei, capitis** (children, needs oral therapy) and **unguium**.
- **Diagnosis: skin scrapings, nail clippings or plucked hairs for microscopy and culture.** It is cheap, and it prevents months of the wrong treatment. Confirm before committing a patient to a long oral course.
- **Treatment:** topical azole or terbinafine for limited disease; **oral terbinafine or itraconazole for scalp, nail, extensive or steroid-modified disease** — nails require three to six months, with liver function monitoring and attention to drug interactions.

2. Yeasts

- **Candidiasis** — intertrigo in skin folds, oral, and genital. **Recurrent or extensive candidiasis is a reason to check the glucose and consider immunodeficiency, including HIV** (Case 45). Ask about antibiotic and inhaled steroid use and denture hygiene.
- **Pityriasis versicolor** — finely scaly hypo- or hyperpigmented macules on the trunk, common in hot humid climates; microscopy shows the "spaghetti and meatballs" appearance. Treated with topical ketoconazole. **Warn the patient that the pigment change takes months to normalise after the fungus has gone**, or the treatment will be judged a failure.
- Deep and systemic mycoses in the immunosuppressed (Case 46); and **mycetoma**, which is endemic in parts of this region and presents as a chronic swelling with discharging sinuses and grains.

3. Infestations

SCABIES

Suspect it from: itch that is worse at night, burrows in the finger web spaces, wrists, axillae and genitals — and other members of the household itching.

Treatment: topical permethrin 5% or oral ivermectin — and treat every household and close contact simultaneously, whether or not they itch. Wash bedding and clothing at high temperature. Treating the patient alone guarantees reinfestation.

Then warn that the itch persists for two to four weeks after successful treatment — it is an immune response to dead mites, not treatment failure. Without that warning the patient will re-treat repeatedly and develop an irritant dermatitis.

Crusted (Norwegian) scabies in the elderly and immunosuppressed — hyperkeratotic, sometimes barely itchy, **enormously contagious**, and regularly misdiagnosed as eczema or psoriasis for months while it spreads through a ward or care home.

- **Head and body lice**; **cutaneous larva migrans** — a serpiginous migrating track after beach or soil exposure, treated with ivermectin or albendazole; and **cutaneous leishmaniasis**, covered in Case 70.

4. Teaching Points and Viva Questions

- Scrape before you steroid. Annular, asymmetrical or steroid-worsened means tinea.
- Scalp and nail disease need oral treatment.
- Recurrent candidiasis means check the glucose and think about immunodeficiency.
- Treat all the household contacts for scabies at the same time.
- Warn that scabies itch lasts weeks after cure, and that versicolor pigment takes months.
- Crusted scabies masquerades as eczema and infects whole wards.

Questions you should be able to answer:

- Explain what the steroid cream did to her rash and to your ability to diagnose it.
- Her son has scalp involvement. Why will a cream not work?
- A whole family itches at night. What do you prescribe, and for whom?
- A treated patient returns at three weeks still itching. Has the treatment failed?
- An elderly nursing home resident has thick scaly plaques thought to be psoriasis, and three staff members are now itching. What is the diagnosis?

Case 75 · Psoriasis and the Papulosquamous Diseases

CLINICAL VIGNETTE

A **32-year-old man** has had scaly plaques on his elbows, knees and scalp for six years, worse in winter. Two weeks ago his skin became far more extensive and inflamed **shortly after finishing a course of oral prednisolone** given for back pain. He has also developed pain and swelling of the right index finger and pitting of several nails.

On examination: well-demarcated salmon-pink plaques with **silvery scale** over both elbows, knees, the scalp margin, the natal cleft and the umbilicus. **Nail pitting and onycholysis.** The right index finger is uniformly swollen — **dactylitis**. Removing scale from a plaque produces pinpoint bleeding.

THE ERROR IN THIS VIGNETTE

Do not treat psoriasis with systemic corticosteroids.

They work briefly, and **withdrawal can precipitate generalised pustular or erythrodermic psoriasis**, which is a medical emergency. This patient's flare was caused by his treatment.

Other triggers to ask about: streptococcal throat infection (guttate psoriasis), stress, skin trauma (the **Koebner phenomenon**), alcohol, smoking, and **drugs — beta blockers, lithium, antimalarials and non-steroidal anti-inflammatory drugs.**

1. Recognising Psoriasis

- **Signs:** well-demarcated plaques with silvery scale on **extensor** surfaces; **Auspitz sign** (pinpoint bleeding after scale removal); **Koebner phenomenon** (lesions at sites of trauma); and involvement of the **scalp margin, natal cleft, umbilicus and nails** — the places to look when the diagnosis is uncertain.
- **Nail changes:** pitting, onycholysis, subungual hyperkeratosis, and the oil-drop sign.
- **Types:** chronic plaque; **guttate**, following streptococcal infection in a young person; flexural or inverse; **generalised pustular psoriasis and erythrodermic psoriasis, both emergencies with fever and systemic upset**; scalp; and nail disease alone.

WHAT TO SCREEN FOR AT EVERY REVIEW

Psoriatic arthritis affects up to a third of patients and is often missed — ask about inflammatory joint pain, dactylitis, heel pain and back pain at every review, and examine the joints (Case 60).

And psoriasis is a cardiovascular and metabolic risk state — metabolic syndrome, fatty liver, cardiovascular disease, depression and alcohol misuse are all more common. Check blood pressure, lipids, glucose and mood, and ask about alcohol. The skin is the visible part of a systemic inflammatory disease.

2. Treatment

- **Topical:** generous emollient, with a **vitamin D analogue and a potent corticosteroid** as the mainstay, plus coal tar and salicylic acid preparations, and specific scalp formulations. Quantity and site-appropriate potency as in Case 71.
- **Phototherapy:** narrowband ultraviolet B for extensive disease.
- **Systemic:** methotrexate; **acitretin — teratogenic, with pregnancy avoidance required for three years afterwards**; ciclosporin for short-term control. **Then biologics — tumour necrosis factor, interleukin-17 and interleukin-23 inhibitors — which have transformed severe disease**, with tuberculosis and hepatitis screening beforehand.

- **Never oral corticosteroids.** Address smoking, alcohol and weight, all of which worsen the disease and reduce treatment response.

3. The Papulosquamous Differential

Condition	What identifies it
Psoriasis	Extensor, well-demarcated, silvery scale, nail and scalp involvement
Lichen planus	Violaceous flat-topped polygonal papules with Wickham striae, on the wrists and shins; oral involvement is common; scarring alopecia and nail destruction. Associated with hepatitis C — and a lichenoid drug eruption is the mimic
Pityriasis rosea	A herald patch, then oval scaly lesions along the lines of cleavage on the trunk — the "Christmas tree" pattern. Self-limiting over six to eight weeks; reassurance is the treatment
Seborrhoeic dermatitis	Greasy scale in the scalp, eyebrows, nasolabial folds and behind the ears. Florid, extensive disease should prompt an HIV test
Pityriasis versicolor	Fine scale with pigment change on the trunk (Case 74)
Secondary syphilis	A non-itchy coppery papulosquamous rash involving the palms and soles. Always in the differential, and diagnosed by serology (Case 79)
Cutaneous T-cell lymphoma	Persistent, fixed, atypical patches in an older patient that do not respond to adequate treatment. Biopsy rather than escalate the steroid
Discoid lupus and subacute cutaneous lupus	Scarring, photodistribution, follicular plugging (Case 59)

4. Teaching Points and Viva Questions

- Never give oral steroids for psoriasis — withdrawal can precipitate pustular or erythrodermic disease.
- Look at the scalp margin, natal cleft, umbilicus and nails.
- Ask about joints and screen the cardiovascular risk at every visit.
- A scaly rash involving the palms and soles should prompt syphilis serology.
- A fixed atypical patch in an older patient that resists treatment needs a biopsy.

Questions you should be able to answer:

- What caused his flare, and what will you tell the doctor who prescribed the prednisolone?
- What does the swollen finger signify and what does it change?
- Name four drugs that worsen psoriasis.
- Distinguish psoriasis from lichen planus at the bedside.
- A young man has an itchy scaly trunk rash six weeks after a sore throat. What is the likely diagnosis?

Case 76 · Blistering Disease and the Cutaneous Drug Emergencies

CLINICAL VIGNETTE

A **68-year-old man** has had three weeks of intensely itchy urticarial plaques on the trunk and limbs. Over the past week **tense blisters** have appeared on the plaques; they are difficult to rupture and leave intact skin around them. **The mouth and other mucosae are unaffected.** No new drugs. Nikolsky sign negative.

Biopsy with direct immunofluorescence: linear IgG and C3 at the basement membrane.

1. The Two Autoimmune Blistering Diseases

	Bullous pemphigoid	Pemphigus vulgaris
Age	Elderly	Middle-aged
Blister	Tense and subepidermal, difficult to rupture	Flaccid and intraepidermal, rupturing to leave painful erosions
Preceding phase	Weeks of intensely itchy urticarial plaques before any blister appears	Often begins with erosions rather than blisters
Mucosa	Usually spared	Prominently involved, and frequently the first site — painful mouth erosions
Nikolsky sign	Negative	Positive
Antibody	Anti-BP180 and BP230	Anti-desmoglein 1 and 3
Treatment	Potent topical corticosteroid, oral corticosteroid, doxycycline, immunosuppressants	Systemic corticosteroid with rituximab — historically a fatal disease

- **Diagnosis: biopsy of a fresh lesion for histology, plus a perilesional biopsy for direct immunofluorescence**, with serum indirect immunofluorescence or enzyme immunoassay. **Two biopsies, from two different places** — an important practical point.
- **Dermatitis herpetiformis** — intensely itchy grouped vesicles on the elbows, knees and buttocks. **It is coeliac disease of the skin:** check coeliac serology, prescribe a gluten-free diet, and use dapsone for rapid relief — **checking glucose-6-phosphate dehydrogenase status first**.
- Other causes of blisters: bullous impetigo, herpes simplex and zoster, insect bites, bullous erythema multiforme, porphyria cutanea tarda, bullosis diabeticorum, friction and burns.

2. Drug Eruptions — Benign or Dangerous?

- **The common, benign one** is the **morbilliform drug eruption**: a symmetrical itchy maculopapular rash appearing **7 to 14 days after a new drug**, with no mucosal involvement and no systemic upset. Stop the drug; it settles.

THE FEATURES THAT SEPARATE A RASH FROM AN EMERGENCY

Skin pain rather than itch · **mucosal involvement — mouth, eyes or genitals** · **blistering or skin detachment** · **a positive Nikolsky sign** · **facial oedema** · fever · lymphadenopathy · **eosinophilia** · deranged liver or renal function.

Any one of these turns a drug rash into a medical emergency. Stop the drug, escalate, and do not wait to see whether it settles.

Syndrome	Recognition and management
Stevens–Johnson syndrome and toxic epidermal necrolysis	One to four weeks after a new drug. Prodrome, then painful dusky macules, blistering and detachment, with mucosal erosions at two or more sites. Graded by the area of detachment. Culprits: allopurinol, lamotrigine, carbamazepine, phenytoin, sulfonamides including co-trimoxazole, nevirapine, NSAIDs. Management: stop the drug, transfer to a burns or specialist unit, meticulous supportive care — fluids, temperature control, analgesia, wound care, nutrition, and same-day ophthalmology because ocular scarring blinds. Consider ciclosporin or other immunomodulation; prognosticate with SCORTEN. Record the drug as an absolute lifelong contraindication and tell the patient in writing.
Drug reaction with eosinophilia and systemic symptoms	Later — two to eight weeks after the drug. Fever, widespread rash, facial oedema, lymphadenopathy, eosinophilia, and organ involvement: hepatitis, nephritis, myocarditis, and thyroiditis which may appear months later. Culprits: allopurinol, anticonvulsants, sulfonamides, vancomycin, minocycline. Stop the drug, give systemic corticosteroid with a slow taper, and monitor organ function — including thyroid function — for months.
Acute generalised exanthematous pustulosis	Rapid onset after an antibiotic: sterile pustules on a background of erythema, with fever and neutrophilia. Resolves on withdrawal.

ERYTHRODERMA

Erythema over more than 90% of the body surface, from any cause — psoriasis, eczema, a drug reaction, cutaneous T-cell lymphoma, or pityriasis rubra pilaris.

It is a medical emergency because the skin has stopped working: fluid loss and dehydration, heat loss and hypothermia, high-output cardiac failure, hypoalbuminaemia, electrolyte disturbance, and infection through a breached barrier.

Admit · keep the patient warm · rehydrate and monitor electrolytes and albumin · bland emollient and no irritants · stop every non-essential drug · treat infection · then treat the underlying cause.

- **Urticaria and angioedema:** acute versus chronic spontaneous urticaria; **non-sedating antihistamines up-titrated to several times the standard dose** rather than long-term corticosteroid; omalizumab in refractory chronic disease. **Angioedema involving the airway is an anaphylaxis emergency. Urticarial vasculitis** — individual lesions lasting more than 24 hours, bruising as they fade, with systemic features — needs a biopsy.

3. Teaching Points and Viva Questions

- Tense blisters and spared mucosa in an elderly patient is pemphigoid; flaccid blisters with mouth erosions is pemphigus.
- Two biopsies — one lesional for histology, one perilesional for immunofluorescence.
- Dermatitis herpetiformis is coeliac disease; check the serology and the G6PD.
- Skin pain, mucosal involvement, blistering, facial oedema, fever or eosinophilia turn a drug rash into an emergency.
- Severe drug reactions come one to four weeks later, and DRESS as late as eight.
- Erythroderma kills through the failure of the skin as an organ.

Questions you should be able to answer:

- Which two features of this man's blisters distinguish his diagnosis from pemphigus vulgaris?

- Which biopsies do you take, and from where?
- A patient develops a rash 12 days after starting allopurinol, with a swollen face, fever and eosinophils of 2.4. What is the diagnosis and what do you monitor for months?
- Name five drugs that commonly cause Stevens–Johnson syndrome.
- Why is an erythrodermic patient at risk of cardiac failure and hypothermia?

Case 77 · Skin Tumours and the Pigmented Lesion

CLINICAL VIGNETTE

A **62-year-old farm worker** with decades of outdoor sun exposure presents with three lesions.

On the nose: a slowly growing **pearly nodule with a rolled edge, surface telangiectasia and central ulceration**, present for about eighteen months and occasionally bleeding.

On the ear: a firm, tender, **keratotic nodule that has grown over three months**.

On the back: a pigmented lesion that his wife says has changed. It is **9 mm across, asymmetrical, with an irregular border and two shades of brown and black**.

1. The Three Common Malignancies

	Basal cell carcinoma	Squamous cell carcinoma	Melanoma
Appearance	Pearly nodule, rolled edge, telangiectasia, central ulcer	Keratotic or ulcerated nodule, often tender	Pigmented lesion that is changing
Growth	Slow, over months to years	Faster, over weeks to months	Variable; nodular type grows fast
Metastasis	Essentially never — but locally destructive	Yes — higher risk on ear, lip and scalp, in scars, and in the immunosuppressed	Yes, and early
Management	Excision, or Mohs surgery at high-risk sites; topical or destructive therapy for superficial lesions	Excision with margins, and assessment of regional nodes	Excisional biopsy, then wide excision by Breslow thickness, sentinel node in selected cases

- **Premalignant and in situ disease: actinic keratoses** — rough scaly macules on sun-damaged skin, treated as a field rather than lesion by lesion; **Bowen disease** — squamous carcinoma in situ, a persistent scaly plaque often on the lower leg; and **keratoacanthoma**, which grows rapidly and may regress but is managed as a squamous carcinoma.

2. The Pigmented Lesion

ASSESSING A PIGMENTED LESION

A — **A**symmetry · **B** — **B**order irregularity · **C** — **C**olour variation · **D** — **D**iameter over 6 mm · **E** — **E**volution or change.

Plus the ugly duckling sign: the lesion that looks unlike all the patient's others. **And the most important single feature is change**, which is why the history from the patient or their family matters more than the photograph.

Do not shave or punch a suspected melanoma. Perform an excisional biopsy with a narrow margin, so that the Breslow thickness — which determines the definitive excision margin, the staging and the prognosis — can be measured.

- **Subtypes:** superficial spreading; **nodular — fast-growing and sometimes amelanotic, so a rapidly growing pink nodule is not automatically benign**; lentigo maligna; and **acral lentiginous and subungual melanoma**.

THE LESIONS THAT GET MISSED

Melanoma is less common in darker skin — and it presents later and at sites that are not routinely examined.

Acral and subungual melanoma occur on the soles, palms and nail beds. So **examine the soles, the palms and the nails**, and treat **pigment spreading onto the nail fold — Hutchinson sign — as melanoma until proved otherwise** rather than as a bruise or a fungal nail.

The consequence of the assumption that pigmented skin does not get melanoma is that when it does, it is found late.

- **Modern systemic therapy — immune checkpoint inhibitors and BRAF/MEK inhibitors — has transformed advanced melanoma**, which is a further reason to biopsy properly and stage accurately rather than treating a suspicious lesion destructively.

3. Benign Lesions Worth Recognising

- **Seborrhoeic keratosis** — "stuck-on", warty, greasy; **naevi**; dermatofibroma; skin tag; cherry angioma; epidermoid cyst; **pyogenic granuloma** — a rapidly growing friable bleeding nodule, which must be distinguished from amelanotic melanoma by histology.

- **And one sign to know: the sudden eruption of multiple seborrhoeic keratoses — the sign of Leser–Trélat — suggests an internal malignancy** (Case 70).

4. Assessment and Prevention

- **Examine the whole skin, not only the lesion the patient points at** — including scalp, behind the ears, soles, between the toes and the nails. Measure and photograph; use dermoscopy where available; **palpate the regional lymph nodes.**

- **Prevention, which matters particularly in this climate:** avoid the midday sun, cover with clothing and a hat, use broad-spectrum sunscreen, avoid sunbeds, and protect children — cumulative childhood exposure drives adult risk.

- **Surveillance of organ transplant recipients and other chronically immunosuppressed patients**, who develop multiple and aggressive squamous cell carcinomas.

5. Teaching Points and Viva Questions

- Pearly with a rolled edge and telangiectasia is basal cell; keratotic, tender and fast-growing is squamous.
- Basal cell carcinoma does not metastasise but destroys locally.
- Change is the most important feature of a pigmented lesion.
- Excisional biopsy, never a shave, for suspected melanoma.
- Examine the soles, palms and nails — that is where melanoma is missed in darker skin.
- Examine all the skin, not just the lesion shown to you.

Questions you should be able to answer:

- Describe each of his three lesions and give your diagnosis for each.

- Why is the lesion on the ear of more concern than the one on the nose?
- The surgeon offers a shave biopsy of the pigmented lesion today. What do you say and why?
- A dark-skinned patient has a pigmented streak in the thumbnail extending onto the cuticle. What is your concern?
- A renal transplant recipient has four scaly nodules on the forearms. What is the diagnosis and what does he need?

Case 78 · Acne, Rosacea, and Disorders of Pigmentation, Hair and Nails

CLINICAL VIGNETTE

A **24-year-old woman** has had acne for four years. It now involves painful deep nodules on both cheeks and along the jawline, and she has developed pitted scars. It is worse before each period. Her periods are irregular, she has noticed coarse hair on the chin and upper lip, and she has gained weight.

She has used several creams and **two long courses of oral antibiotics bought from a pharmacy**.

She also reports **patches of complete pigment loss on the backs of both hands and around the mouth** appearing over six months, and **diffuse hair shedding for the past three months**, which began about two months after a febrile illness.

1. Acne

- **Lesions:** comedones, papules, pustules, nodules and cysts. **Grade by severity and, crucially, by the presence of scarring.**
- **Associations to look for: polycystic ovary syndrome — investigate any woman with acne plus hirsutism and menstrual irregularity, as here**; congenital adrenal hyperplasia; Cushing syndrome; and **drugs — corticosteroids, anabolic steroids, lithium, phenytoin, some progestogens and epidermal growth factor receptor inhibitors.**

THE PRINCIPLE THAT GOVERNS ACNE TREATMENT

Scarring is permanent and preventable, so escalate early rather than repeating antibiotic courses.

Foundation: a topical retinoid with benzoyl peroxide. **Topical antibiotics are used only in combination, never alone**, because of resistance. **Oral antibiotics for no more than about three months** — not repeated courses over years, which is what this patient has had.

In women: a combined oral contraceptive or spironolactone. **For severe, nodular, scarring or refractory acne: isotretinoin** — with the pregnancy prevention programme, and monitoring of lipids, liver function and mood.

And ask about the psychological impact, which is substantial and correlates poorly with the clinical severity. A patient with mild acne may be seriously distressed by it.

2. Rosacea and the Adnexal Disorders

- **Rosacea:** central facial erythema and flushing, telangiectasia, papules and pustules **without comedones** — the feature that separates it from acne. **Ocular rosacea with blepharitis and gritty eyes is common and needs ophthalmology referral.** Rhinophyma in longstanding disease.
- Triggers: heat, sunlight, alcohol, spicy food, hot drinks. Treatment: **sun protection**, topical ivermectin, metronidazole or azelaic acid, oral doxycycline, brimonidine for erythema, and vascular laser.
- **Hidradenitis suppurativa** — painful recurrent nodules, sinus tracts and scarring in the axillae, groin and inframammary folds, associated with obesity and smoking. **It is repeatedly managed as recurrent boils with repeated antibiotics and incisions.** Treatment: antiseptic washes, doxycycline, clindamycin with rifampicin, anti-tumour-necrosis-factor therapy, and surgery — with weight loss and smoking cessation.

3. Pigmentation

- **Vitiligo** — symmetrical, sharply demarcated depigmented macules, characteristically acral and periorificial, accentuated under a Wood's lamp. **Screen for associated autoimmune disease: thyroid function above all, and consider diabetes, pernicious anaemia and adrenal insufficiency.** Treatment: topical corticosteroid or calcineurin inhibitor, phototherapy, and camouflage.

> DO NOT CALL IT COSMETIC
> **The psychological and social impact of vitiligo is severe, and it is greatest in patients with darker skin, where the contrast is most visible.**
> It affects marriage prospects, employment and mood, and patients are frequently told it is "only cosmetic". **Ask about it, treat the distress as part of the disease, and offer camouflage and psychological support alongside any topical treatment.**

- **Melasma** — symmetrical facial hyperpigmentation associated with pregnancy, oral contraception and sun exposure. **Rigorous sun protection is the treatment**, with topical depigmenting agents; it recurs readily.

- **Post-inflammatory hyperpigmentation and hypopigmentation** — very common in darker skin and **slow to resolve, often over many months.** Explain the timescale explicitly, or the patient will assume the treatment failed and seek harmful skin-lightening products.

- Also: drug-induced pigmentation, acanthosis nigricans and Addison pigmentation (Cases 26 and 70).

4. Hair

Pattern	Diagnosis
Diffuse shedding 2–4 months after a trigger — fever, surgery, childbirth, rapid weight loss, severe illness	Telogen effluvium — self-limiting, with full recovery. This patient. Reassure, and check ferritin and thyroid function
Gradual patterned recession or vertex thinning	Androgenetic alopecia — topical minoxidil, finasteride in men
Well-circumscribed smooth bald patches with exclamation-mark hairs, sometimes with nail pitting	Alopecia areata — autoimmune; screen thyroid; topical or intralesional corticosteroid, and newer targeted therapy in severe disease
Loss of follicular openings, scarring	Scarring alopecia — irreversible, so it must be recognised early: lichen planopilaris, discoid lupus, and traction alopecia and central centrifugal cicatricial alopecia related to hairstyling and chemical practices

- **Investigate diffuse hair loss with ferritin and thyroid function**, and with androgen studies where there is hirsutism or virilisation.

5. Nails

- **Pitting and onycholysis** — psoriasis; **onychomycosis** — confirm with clippings before a long oral course (Case 74); **clubbing**; **koilonychia**; **splinter haemorrhages**; **Beau lines** after a systemic illness; leuconychia; half-and-half nails of renal failure; **melanonychia with Hutchinson sign — subungual melanoma** (Case 77); and ingrowing and pincer nails.

6. Teaching Points and Viva Questions

- Acne with hirsutism and irregular periods means investigate for polycystic ovary syndrome.
- Scarring is permanent — escalate early rather than repeating antibiotics.
- Papules and pustules without comedones is rosacea, and the eyes are often involved.
- Hidradenitis is not recurrent boils.
- Screen thyroid function in vitiligo and alopecia areata, and never call vitiligo cosmetic.
- Telogen effluvium follows a trigger by two to four months and recovers.
- Scarring alopecia is irreversible — recognise it before the follicles are gone.

Questions you should be able to answer:

- Which features of her history should prompt endocrine investigation, and what would you send?
- Why is repeating a third course of oral antibiotics the wrong answer?
- What do you check in a patient presenting with vitiligo?
- Explain her hair loss and its prognosis.
- How would you distinguish rosacea from acne on examination?

Case 79 · Sexually Transmitted Infections

CLINICAL VIGNETTE

A **27-year-old man** presents with a widespread rash. Three weeks ago he noticed a **single painless ulcer on the penis**, which healed without treatment. For the past week he has had a **non-itchy rash of coppery-red scaly papules over the trunk and limbs, including the palms and the soles**.

On examination: generalised non-tender lymphadenopathy, **greyish mucous patches on the buccal mucosa**, and patchy "moth-eaten" loss of scalp hair. He feels mildly feverish and tired.

Treponemal antibody test positive; rapid plasma reagin titre 1:128.

BEFORE THE EXAMINATION

Take the sexual history plainly, without assumptions and without embarrassment, because the patient's willingness to answer determines whether the diagnosis is made.

Cover: partners in the last three months and their gender · the sites of exposure · condom use · symptoms in partners · previous infections · HIV status and last test · vaccination · and, in women, contraception and pregnancy.

Confidentiality must be explicit and it must be real. In this setting the social and legal consequences of disclosure can be serious, and a patient who fears them will not return — so explain what will and will not be recorded and who will and will not be told, before you ask the questions.

1. Think in Syndromes, Not Organisms

Syndrome	Causes and management
Urethritis and discharge	Chlamydia — commonest, and frequently asymptomatic; gonorrhoea — purulent, short incubation; *Mycoplasma genitalium*; *Trichomonas*. Diagnose by nucleic acid amplification testing from first-void urine or the relevant site — including throat and rectum where exposure occurred. Treat gonorrhoea with ceftriaxone according to local resistance data — quinolone resistance is widespread and cephalosporin resistance is emerging — and chlamydia with doxycycline. Complications: epididymo-orchitis, pelvic inflammatory disease and tubal infertility, reactive arthritis (Case 60), disseminated gonococcal infection (Seminar 6).
Genital ulceration	Herpes simplex — the commonest cause: multiple painful shallow ulcers, with systemic symptoms in a primary episode. Aciclovir, with suppressive therapy for frequent recurrence, and counselling about transmission and about pregnancy. Syphilis — a single painless indurated chancre. Also chancroid, lymphogranuloma venereum, Behçet disease (Case 61), trauma and malignancy.
Genital lumps	Anogenital warts — human papillomavirus; treated destructively or with topical agents. Human papillomavirus vaccination prevents them, and prevents cervical, anal and oropharyngeal cancer. Molluscum contagiosum.
Vaginal discharge	Bacterial vaginosis, candidiasis and trichomoniasis — not all of which are sexually transmitted, which matters for how the conversation is handled.

2. Syphilis — the Great Imitator

Stage	Features
Primary	A single painless indurated ulcer at the site of inoculation, with regional lymphadenopathy. It heals spontaneously, which is why patients present later.
Secondary (6–12 weeks)	A generalised non-itchy papulosquamous rash characteristically involving the palms and soles, mucous patches, condylomata lata, moth-eaten alopecia, lymphadenopathy and fever. Highly infectious — and it also resolves spontaneously. This patient.
Latent	Asymptomatic, detected only serologically.
Tertiary (years later)	Gummata; cardiovascular syphilis with aortitis and aortic regurgitation (Case 16); neurosyphilis — tabes dorsalis, general paralysis, Argyll Robertson pupils, meningovascular disease.
Congenital	Prevented by antenatal screening and treatment — which is why the screening programme exists.

- **Serology: a treponemal test (enzyme immunoassay or TPPA) remains positive for life and tells you the patient has had syphilis. A non-treponemal test (RPR or VDRL) titre reflects disease activity and is used to confirm the response to treatment.** Dark-ground microscopy or polymerase chain reaction from a lesion where available.

- **Treatment: benzathine penicillin — a single dose in early disease, a longer course in late or neurosyphilis. Warn about the Jarisch–Herxheimer reaction** in the first 24 hours, and follow the RPR titre to confirm a fall.

WHAT MUST HAPPEN FOR EVERY DIAGNOSIS

Every patient with one sexually transmitted infection is tested for the others: HIV, syphilis, hepatitis B and C, gonorrhoea and chlamydia. A diagnosis of one is a marker of exposure risk, not a complete assessment (Case 45).

Then: partner notification and treatment · abstinence until treatment is complete and partners are treated · follow-up serology or a test of cure · vaccination against hepatitis A and B and human papillomavirus · and consideration of HIV pre-exposure prophylaxis where the risk continues.

Partner notification is a clinical duty and a skill. It is done with the patient's cooperation, and handled badly it prevents future presentation — by them and by everyone they talk to.

3. Teaching Points and Viva Questions

- A non-itchy papulosquamous rash involving the palms and soles is secondary syphilis until serology says otherwise.
- Both the primary ulcer and the secondary rash resolve without treatment — spontaneous resolution is not cure.
- The treponemal test stays positive for life; the RPR titre tracks activity and treatment response.
- Test for all the others when you find one.
- Painless single ulcer is syphilis; multiple painful ulcers are herpes.
- Explain confidentiality before you take the history, not after.

Questions you should be able to answer:

- What was the painless ulcer three weeks ago, and why did he not present then?
- Which single feature of the rash makes you think of this diagnosis?
- Explain the difference between the two serological tests and what each one tells you.
- What else must be tested for, and what happens about his partners?
- He becomes febrile with rigors four hours after his injection. What has happened?