Chapter 13:

DERMATOLOGICAL CONDITIONS

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This chapter covers the following topics:

- Describing skin lesions
- Eczema/dermatitis
- Autoimmune skin conditions
- Infections
- Congenital skin conditions
- Hair conditions
- Trauma, drug reactions and bite reactions
- Dermatology cheat sheet

DESCRIBING SKIN LESIONS

Skin lesions may be primary or secondary. They may be further categorised according to the arrangement or distribution e.g. sun-exposed areas.

Primary Skin Lesions

They may be:

- Flat or raised
- Solid or fluid-filled (if raised)

Flat Lesions

Macules are well-circumscribed, flat, non-palpable lesions that are <1 cm in diameter. Patches are well-circumscribed, flat, non-palpable lesions that are >1 cm in diameter, see related image here.

Raised Lesions

They may be:

- Solid lesions (well-circumscribed, raised, palpable lesions which extend into the dermis or subcutaneous tissue):
    - Papules – solid lesions <1 cm in diameter
    - Plaques – flat-topped lesions >1cm diameter (see related image here)
    - Nodules – dome-shaped lesions >1cm diameter (see related image here)
- Fluid-filled:
    - Vesicles – well-circumscribed, raised lesions <1cm diameter which are filled with clear fluid (see related images here)
    - Bullae – well circumscribed, raised lesions >1cm diameter which are filled with clear fluid
    - Pustules – well-circumscribed, raised lesions <1cm diameter which are filled with pus

Secondary Lesions

They include:

- Scale – accumulated fragments of stratum corneum produced from shed skin
- Crust – dried serum, blood or pus

- Erosion – open area of the skin which is the result epidermal loss
- Fissure – linear, open area of the skin which is the result epidermal loss
- Ulcer – irregular, open area of the skin which is the result epidermal loss and at least partial dermal loss
- Excoriation – superficial skin abrasion that usually results from scratching.
- Lichenification – dry, thickened, dark (hyperpigmented) skin with accentuated skin markings

Arrangement of Lesions

Lesions may be:

- Annular (ring-shaped) e.g. tinea corporis (ringworm), pityriasis rosea, syphilis, urticaria, lichen planus, psoriasis, seborrheic eczema
- Grouped e.g. herpes simplex (see related image here), insect bites, warts, molluscum contagiosum
- Linear e.g. scratch marks, scar, keloid, scabies, psoriasis, lichen planus, warts

ECZEMA/DERMATITIS

Eczema is a chronic inflammatory skin disease which is common in children. It mainly affects the epidermis and may be complicated by secondary infection by bacteria (*Staphylococcus aureus, Streptococcus pyogenes*), viruses (HSV) or fungi (*Candida albicans)*. Children may present with:

- Atopic eczema (see related image here and here)
- Nummular eczema
- Seborrhoeic eczema
- Napkin eczema
- Perioral eczema

Atopic/Allergic Eczema

This is a chronic condition that has acute exacerbations and which is often associated with asthma, allergic rhinitis and allergic conjunctivitis (see related image here).

Pathophysiology

The pathophysiology is poorly defined, but there are two hypotheses:

- Primary immune dysfunction leads to IgE sensitisation, allergic inflammation and secondary epithelial barrier disturbance.
- A primary epithelial barrier defect leads to secondary immunologic dysregulation and inflammation as bacteria are able to enter through the skin.

It is associated with elevated serum IgE levels and there is usually a family history of atopy. Symptoms generally start at 3 months old, fluctuate and remit by school-going age in 50% of children. See images related to children with atopic eczema on this web page.

Clinical Features

The child will present with:

- Acute eczema – red and swollen skin, weeping/oozing vesicles (clear, serous fluid), crusting (when the fluid dries up)
- Subacute eczema – mild erythema with or without oozing, scaling, early thickening
- Chronic eczema – lichenification (leathery, thickened skin, hyper/hypopigmentation, prominent skin markings)

The child's presentation may vary depending on his/her age. Infants present with a red, wet and/or scaling eruption which involves the head, extensor areas and trunk. There is nasal sparing.

Older children usually have localized lesions in the flexures. They may have other signs which are pathognomonic of atopy (atopic diathesis):

- Eyes
    - Allergic shiners (dark circles under the eyes which are the result of nasal or sinus congestion)
    - Dennie-Morgan folds/infraorbital folds (lines on the skin below the lower eyelid)
    - Muddy sclera (brown discolouration of the sclera)
- Ears

    - Infra-auricular fissures (fissures beneath the earlobes, which may be weeping)
- Nose
    - Headlamp sign (nasal/central face-sparing when there is eczema of the face).
    - Nasal crease (produced because of chronic upward rubbing of the nose)
    - Salute sign (action of rubbing the nose)
- Lips
    - Lip hyperlinearity
    - Angular cheilitis (erythematous, swollen, and painful patches in the corners of the mouth
- Limbs
    - Hyperlinearity of the palms and soles
    - Keratosis pilaris (dry and rough patches on the skin of the upper arms, thighs, and cheeks)

Diagnosis

The UK Working Party Diagnostic Criteria is used to diagnose atopic eczema. For the diagnosis to be made, the child must have 1 major and 3 minor criteria.

- Major criterion – pruritis
- Minor criteria:
    - Family/personal history of atopy
    - Onset <2 years old
    - History of dry skin
    - History of flexural dermatitis
    - Visible flexural dermatitis

Investigations

Atopic eczema is a clinical diagnosis, therefore no investigations are necessary. However, it may be helpful to do skin prick testing (SPT) to identify triggers.

Management

The mnemonic ASSEBLIEF can be used to remember the approach to management for the child with atopic eczema (see related image here).

Table 13.1: ASSEBLIEF Mnemonic for the Management of Atopic Eczema

A - Antihistamines	Oral antihistamines may be given to sedate the child and avoid nocturnal scratching. The child <2 years old may be given chlorphenamine (0.1 mg/kg/dose at night) and the child >2 years old may be given cetirizine (5-10 mg at night).
S – Steroids	Use potent topical steroids daily (initially and for flares). Use hydrocortisone 1% on the face or skin folds and use beclomethasone 0.1% on the rest of the body.
S - Scratching	An important aspect of treating eczema is avoiding scratching by keeping the skin moist (liberally use moisturisers). Explain the itch-scratch-itch cycle to the parents and child.
E - Education	Inform the parent and child that eczema is a chronic condition caused by barrier dysfunction. Provide support and counselling.
B – Bacteria	Eczema is vulnerable to bacterial/viral infection, especially *S. aureus* (impetiginised eczema) and HSV (eczema herpeticum). Keep the infected skin clean and moist. Treat with antibiotics if necessary.
L – Look	Look for associated atopic conditions and avoid triggers e.g. soaps, bubble bath, wool and other irritating fibres in clothing. Children with atopic eczema are more likely to have atopic rhinitis, conjunctivitis or asthma.
I – If	If severe, use wet wraps unless there is a secondary infection of that area.
E - Emollients	Use emollients as a soap substitutes (help remove crusts but is gentle on the skin) and moisturisers e.g. emulsifying ointment, cetomacrogol.
F – Forever	This condition is lifelong.

Occlusive, wet dressings are to be used for severe or thick, lichenified skin. However, they must not be used if there is a sign of infection as it will promote spread of the infection.

Table 13.2: Impetiginised Eczema

Impetiginised Eczema
It is superficial infection of eczematous skin with *S. pyogenes* or *S. aureus*. The affected skin will have yellow-brown crusting. If severe, there may be systemic signs (fever, malaise, lymphadenopathy). Impetiginised eczema is managed by:

- Removing crusts with wet dressings soaked in saline
- Giving antibiotics – with antibiotic-steroid ointment/cream (for small areas) and systemic erythromycin or cloxacillin (for severe or widespread infection).

Table 13.3: Drugs Used in Dermatology

Drugs Commonly Used in Dermatology

Weak topical steroids (typically used on the face)	● Hydrocortisone 0.5% (Cutaderm®, Dilucort®) ● Hydrocortisone 1% (Mylocort ®, Procutan ®)
Moderate topical steroids	● Alclometasone (Aclosone®) ● Clobetasone (Eumovate®)
Potent topical steroids (typically used on the body)	● Beclomethasone (Propaderm®) ● Fluticasone (Cutivate®) ● Methylprednisolone (Advantan®) ● Mometasone (Elocon®)
Very potent topical steroids	● Clobetasol (Dovate®)

Nummular/Discoid Eczema

Aetiology Pathophysiology

The cause and pathophysiology of nummular eczema are not known, but it may be associated with atopic eczema. Stress and bacterial infection can aggravate the condition.

Clinical Features

The child will have well-rounded (discoid), discrete patches of eczema, usually with a crusted or weeping surface. They are most common on the limbs (especially on the legs) but may occur anywhere on the body.

Management

Patients are managed with potent topical steroids (clioquinol where possible) and antibiotics (e.g. flucloxacillin 250 mg 6 hourly), if infected.

Seborrhoeic Eczema (‘Greasy Eczema’)

It is most common in children.

Aetiology and Pathophysiology

The cause and pathophysiology are unknown but it is associated with large sebaceous glands and is common in immunocompromised individuals. The condition may be aggravated by stress and yeast infection of the hair follicles.

Clinical Features

The child will have erythematous, ill-defined lesions with greasy scale. Lesions are usually found in areas with many sebaceous glands e.g. flexural areas, scalp, face, scalp and nappy area.

Management

The condition is usually self-limiting in infants. Skin lesions should be treated with hydrocortisone 1% cream, and scalp lesions treated with detergent shampoo (tar, detergent, selenium sulphide, ketoconazole or zinc pyrithione) and hydrocortisone 1% (if the scalp is inflamed). Oral antibiotics should be prescribed if the skin is infected.

Napkin Eczema

This is a common form of irritant, contact eczema which may be due to soaps or prolonged exposure to urine and faeces in the diaper. Candidal infection may aggravate the condition.

Clinical Features

The eczema ranges in severity from mild to severe:

- Mild napkin eczema:
    - It is usually asymptomatic but the child may have some erythematous patches and papules. There is minimal maceration.
- Moderate napkin eczema:
    - The child may have pain or discomfort.
    - The skin will be shiny/glazed, red and macerated with superficial erosions.
- Severe napkin eczema:
    - The child will have pain.

- There will be significant erythema, and glossy erosions, papules and nodules).

Management

One must advise the parents to regularly change the child's diapers and leave the child nappy-free for a few hours daily. Nappy cream should be applied (acts as a barrier). One should also apply a weak topical steroid (hydrocortisone 1%) mixed with aqueous cream twice daily. If candida is suspected, the child should be prescribed steroid 10% and nystatin 20% in zinc cream.

Table 13.4: Contact Eczema

Contact Eczema
It is due to a skin irritation (irritant contact eczema) or an allergic reaction to substances in contact with the skin (allergic contact eczema). Common causes include cosmetics, creams, jewellery and detergents. The child will, therefore, present with a localised rash and compatible history. *Irritant Contact Eczema* There are two main types – napkin eczema (see above) and dry skin eczema. Dry skin dermatitis is usually due to excessive moisture exposure, such as soap or sweating. The child might experience burning, stinging and discomfort. On examination, the skin is dry and cracked with macular erythema. The mainstay of treatment is emollient therapy. *Allergic Contact Dermatitis* It is an inflammatory reaction of the skin secondary to exposure to an absorbed allergen or antigen. The child will present with itching and a rash. If acute, there will be oedema, erythema and vesicles that often rupture and form crusts. If subacute or chronic, there will be lichenification, erythema and scaling.

Perioral Eczema

Its pathophysiology is not well understood but it is associated with prolonged topical corticosteroid use.

It most commonly affects young females. They present with small, inflammatory papules around the mouth and nose.

Management includes:

- Discontinuing topical corticosteroids
- Avoiding skin irritants

- Using topical calcineurin inhibitors
- Using oral tetracyclines

Pityriasis Sicca Alba

This is regarded as a mild phenotype of eczema due to unknown cause. It is thought to be caused by a genetic predisposition to impaired skin barrier function.

Clinical Features

The child will have hypopigmented macules and patches with a fine scale (most common on the face). Central hyperpigmentation may also occur.

Management

This is self-limiting condition but management may include:

- Allergen identification and avoidance
- Topical treatment
    - Steroids (hydrocortisone 1%/LPC 5% in aqueous cream for the face, 10% steroid/ LPC 5% in emulsifying ointment for the body)
    - Calcineurin inhibitors e.g. tacrolimus, pimecrolimus).

Antihistamines and wet dressings may also be employed.

AUTOIMMUNE SKIN CONDITIONS

Albinism

This is a congenital skin condition.

Clinical Features

The child will have diffuse depigmentation of the skin, hair and eyes. Chronic sun exposure can lead to premature aging, solar keratoses and sun-associated skin cancers.

Management

Supportive measures include:

- Providing genetic counselling and emotional support
- Preventing the complications associated with sun exposure by encouraging sun avoidance, sunscreen use and wearing modest clothing

- Closely monitoring the child to detect skin cancers early

Vitiligo

Pathophysiology

It is an autoimmune disorder in which there is melanocyte destruction, leading to depigmentation. Re-pigmentation may spontaneously occur and is often triggered by sun exposure.

Clinical Features

The child will present with macular depigmentation that is often symmetrical but may be focal. Secondary sun damage (redness, thickening and scaling) and hair loss may also occur.

Re-pigmentation tends to start at the hair follicles. Acral areas (hands and feet), lips and genitalia seldom regain pigment.

Management

Non-pharmacological management includes

- Counselling the child and family (must be informed that vitiligo is an autoimmune, lifelong condition with no definitive cure), see related image here.
- Encouraging moderate sun exposure but discouraging burning
- Camouflage cosmetics

Pharmacological management:

- Vitamin C
- Oral antihistamines
- Potent topical steroids

Lichen Planus

Pathophysiology

This is a cell-mediated immune response of unknown origin. It is sometimes seen in sun-exposed areas and may be a response to drugs e.g. thiazide diuretics, hypoglycaemic agents, methyldopa and anti-TB treatment. Mucosal surfaces and skin are involved (wrists, forearms, palms, soles and nail folds).

Clinical Features

Lichen planus is described using the six Ps – pruritic, purple, planar (flat-topped), polygonal, papules/plaques which can be polymorphous. Lesions may also have Wickham's striae (white lacy network on the surface). The Koebner phenomenon may be present (appearance of new lesions at an area of injury).

Management

Non-pharmacological management includes sunscreen use (if the rash is photo-distributed) and adjusting medication (if there is suspicion that it is drug-induced). Pharmacological management is with steroids (topical or systemic). Oral lesions may be treated with a steroid spray or gel.

Psoriasis

This is an inflammatory immune-mediated condition which results in hyperkeratinisation.

Pathophysiology

Psoriasis usually starts in early adult life, see related image here. The patient has a genetic predisposition but the exact trigger is unknown.

An immune response is activated by the presence of antigenic stimuli in the skin and results in T cell differentiation. These T cells release cytokines which cause keratinocyte hyperproliferation.

Clinical Features

The child may present with:

- Skin involvement:
    - There will be pink-to-red, well-demarcated, pruritic plaques with a silver scale. Pustules are occasionally seen.
    - Scratching the lesions may be associated with the Koebner phenomenon.
    - Lesions are mostly seen on the scalp (crossing the hairline) and extensor surfaces (elbows and knees), but can occur anywhere on the body e.g. flexural areas, perineum.
- Nail signs e.g. pitting, onycholysis, opaque, deformed and crumbling nails

- Joint involvement (psoriatic arthritis)

Management

Non-pharmacological management includes:

- Counselling and educating the patient and family
- Exposure to sunlight
- Avoidance of triggers like stress and streptococcal infection

Pharmacological management includes:

- For the skin:
    - LPC 10% or salicylic acid (to remove scales)
    - Emollients
    - Weak steroids for the face and potent steroids for the body
- For the scalp:
    - LPC 10% or salicylic acid overnight
    - Shampoo in the morning with a detergent or tar preparation
    - Tar/dilute steroid lotion
- Systemic therapy (dermatologist-initiated)

INFECTIONS

Molluscum Contagiosum

It is caused by infection with a poxvirus.

Pathophysiology

The virus can be transmitted via direct skin contact or via contact with fomites e.g. bath towels, sponges, gymnasium equipment, shared bathwater. It may be inoculated into sites of minor skin trauma.

The lesions usually undergo spontaneous resolution in 6 months to a year, but may take longer.

Clinical Features

The child will have one or more round, dome-shaped, skin-coloured or pearly papule(s) which has an umbilicated centre and contains a caseous plug. Lesions can

be anywhere in the body, but typically occur on the trunk, arms, legs, face or genitalia. See related image here.

Management

There is no universally effective treatment. However, whatever the treatment, one must try to avoid scarring.

The lesions may just be observed if they do not bother the child. Otherwise they may be treated with:

- Benzoyl peroxide cream (applied daily)
- Wart paint (applied to individual lesions)
- Liquid nitrogen (applied to individual lesions every 2-3 weeks)
- Tretinoin (applied to individual lesions)
- Imiquimod cream
- Surgical removal under local anaesthetic

Tinea

Pathophysiology

Tinea is caused by fungal infection with dermatophytes. These fungi can be found on humans, animals or in the soil.

Tinea of the nails may lead to recurrence.

Classification and Clinical Features

The child may have:

- Tinea capitis (scalp) – pruritic lesions with central scaling and patchy alopecia
- Tinea corporis/faciei (body/face) – erythematous, pruritic, scaly lesions (papules/pustules) with central clearing and raised, active edges (from which extension occurs)
- Tinea cruris (groin) – pruritic, inflamed, scaly lesions with well-defined edges and central clearing
- Tinea pedis/manuum (foot/hand) – grouped, pruritic blisters or scaly patches along the plantar line, on the sole, on the instep of the foot and/or involving the web spaces

Investigations

The scale may be scraped and specimens sent for light microscopy or fungal culture.

Management

Topical antifungals may be given e.g. Whitfield's ointment, imidazole cream (such as clotrimazole 1%). However, these agents alone are not effective in tinea capitis. Oral antifungal agents (e.g. fluconazole) are given for tinea capitis or any other form of tinea which is extensive. A different diagnosis (e.g. psoriasis, eczema) must be considered if the rash is not resolving with adequate treatment.

Tinea Versicolor (Pityriasis Versicolor)

It is caused by infection with a yeast commensal (*Pityrosporum orbiculare)*. Predisposing factors include high humidity and excessive sweating.

Clinical Features

The child will have depigmented macules with or without fine scale, usually on the trunk. The presence of scale is indicative of active infection. The rash is sometimes hyperpigmented and may be pruritic.

Investigations

One may examine skin scrapings under the microscope (will show short, unbranched hyphae and spores).

Management

Topical treatment is with selenium sulphide shampoo (apply to the whole body once weekly for 3 weeks) or imidazole/terbinafine cream. The absence of scale means that there has been adequate treatment.

Pityriasis Rosea

Its pathophysiology is unknown, but it is thought to be a response to viral infections.

Clinical Features

The child will have a herald patch (single lesion with an active edge) that usually precedes other lesions by a few days. It is often mistaken for tinea until the generalised eruption occurs.

Lesions are oval, have a fringe of scale and are confined to the trunk. They can be pruritic.

Management

The condition is self-limiting and resolves in 4-8 weeks. The child and family should be counselled and informed that the condition is not infectious and will spontaneously resolve. However, antihistamines can be given to help control itch and sedate e.g. chlorphenamine, cetirizine.

Folliculitis

Pathophysiology and Aetiology

It is inflammation of the hair follicle and may be superficial or deep. The most common infective cause is *S. aureus* but may be due to other bacteria or fungi. The non-infective causes of folliculitis include follicular trauma and occlusion.

Clinical Features

The child will have papules or pustules with an erythematous halo. These may occur in clusters.

Management

Non-pharmacological management involves keeping the area dry and clean, removing predisposing factors and using antiseptic agents. Pharmacological management is with antibiotics (given if there is severe folliculitis e.g., flucloxacillin). The patient should be referred if there is no response to treatment.

Furunculosis (Boils)

Aetiology and Pathophysiology

It is caused by *S. aureus* infection of the hair follicle. The organism is carried in the nose or under the fingernails. Individuals are prone to recurrent attacks unless eradication therapy is given.

Clinical Features

The patient will have a painful, firm, red swelling which eventually develops a necrotic centre and releases a pustular discharge. It is particularly common on the thighs or buttocks.

Management

Treatment may include:

- Bathing with a povidone-iodine wash and shampoo
- Cutting the nails short and regularly scrubbing them
- Antibiotics:
    - Topical (apply antiseptic cream to the nostrils to eradicate *S. aureus*)
    - Systemic (flucloxacillin)
- Analgesia

Plane Warts

They are caused by human papilloma virus (HPV) infection (spread via direct contact or inoculation).

Clinical Features

The child will have flat, skin-coloured papules with smooth surfaces. The most common sites are the finger (especially around the nailfold), hands, arms and face.

Management

The warts usually spontaneously resolve. They should not be excised. Keratolytics should be used instead e.g. tretinoin, salicylic acid, instead. Cryotherapy (with liquid nitrogen) is another option.

Herpes Simplex

Pathophysiology

It is caused by infection with HSV 1 or HSV 2, which is transmitted by close physical contact with an infected individual. The virus remains latent in nearby nerve root ganglia and can reactivate and cause further active infections. See images related to herpes simplex here.

Clinical Features

The child will have grouped, painful vesicles on an erythematous base. There may also be systemic signs.

Management

The lesions resolve after 10 days. Thus, treatment is with analgesia. Oral acyclovir may be given for moderate or severe disease.

The child and family must be counselled on how to prevent the spread of cold sores.

Scabies

Pathophysiology

It is caused by mites, which are spread through close contact. The mites burrow into the upper layer of skin (see related images here).

Clinical Features

Many members of the household may be affected. The child will present with an intensely pruritic, non-specific rash (papules, pustules, eczematous changes). Burrows help make the diagnosis of scabies. They are small, scaly, linear lesions with a white/black dot at the end (mite). Burrows are usually found in web spaces, and on the wrist, feet, axillae, umbilicus and palms.

Management

All members of the household must be treated at the same time to prevent re-infection. All linen, towels and clothes should be washed in hot water and exposed to direct sunlight (to kill the mites). Children >2 years are treated with benzyl benzoate 25% lotion and those <2 years are treated with permethrin 5% lotion or diluted benzyl benzoate.

Erythematous Rash with Fever

Chicken Pox (Varicella Infection)

It is a highly infectious viral disease. The child will present with mild headache, fever, malaise and generalised, vesicular rash. There is permanent immunity once infected. It is treated with acyclovir, analgesia and calamine lotion.

Erythema Infectiosum

It is caused by parvovirus B19 infection. The child will have arthropathy, “slapped cheek” appearance, perioral pallor, fever, coryza, headache and a lacy rash on the trunk. It is a self-limiting condition and does not require treatment.

Erythema Nodosum

The most important causes of erythema nodosum are *S. pyogenes* and TB. The child will have tender, red nodules, commonly on the anterior lower leg.
The child should be investigated with tuberculin skin testing, ASOT and a CXR. The underlying cause must then be treated.

Hand, Foot and Mouth Disease

It is a viral illness caused by coxsackie virus. The child will present with:

- Fever
- Malaise
- Abdominal pain
- Painful oral vesicles and erosions
- Sparse, small, grey vesicles on the hands or feet (see related image here)

Symptomatic treatment is given.

Kawasaki Disease

It is characterised by:

- Fever for >4 days
- Bilateral conjunctival injection
- Lip or oral cavity changes (strawberry tongue)
- Rash (mostly affecting the trunk)
- Lymphadenopathy

Investigations should include:

- Blood tests – CRP, ESR and FBC
- Urinalysis
- Imaging – ECG, CXR and echocardiogram

Treatment is with aspirin and IVIG. Specialist referral is necessary.

Measles

It is a highly contagious virus which is characterised by:

- Koplik spots
- Conjunctivitis
- Cough
- Fever
- Coryza

Supportive treatment is given.

Roseola Infantum

It is most commonly caused by HHV6. The patient will have a high fever, facial oedema and blanching rash. It is a self-limiting condition and is managed with supportive treatment.

Rubella

It is a viral infection which causes skin disease that is less severe than measles. The child will have a discrete rash, palatal petechiae and occipital lymphadenopathy. Management is symptomatic, see related image here.

Streptococcal/Staphylococcal Infection

The child may have:

- Impetigo (characterised by crusting)
- Cellulitis (characterised by indistinct borders)
- Erysipelas (tender, warm, bright red, well-demarcated rash and swollen skin)

These infections are treated with penicillin.

CONGENITAL SKIN CONDITIONS

Port-Wine Stain

Aetiology

It is a congenital vascular malformation which may be part of a syndrome:

- Sturge-Weber syndrome – characterised by eye manifestations (glaucoma), skin manifestations (port-wine stain) and CNS manifestations (seizures)

- Klippel-Trenaunay syndrome – characterised by port-wine stain, varicose veins, and bony and soft tissue hypertrophy of the affected limb, see related image here.

Clinical Features

The segmental, blanchable, erythematous patches are present from birth and persist for the rest of the child's life.

Management

The patient may need to be referred to the relevant specialist, depending on the site(s) involved. If the lesion involves the eye or upper third of face, refer to a neurologist.

Haemangioma (Strawberry Naevus)

This is a benign vascular tumour that is present at birth or develops in the first few weeks of life.

Pathophysiology

The naevus undergoes marked proliferation in the first year of life. This is followed by spontaneous involution/regression of the vascular component with replacement by fibrofatty tissue. Its exact cause is unknown but evidence suggests that hypoxia may play a key role in its development.

Clinical Features

The child will have a red nodule or plaque which has been present from birth or developed in the first few weeks of life. Lesions may be multiple and can occur on internal organs.

Management

One must reassure the patient and parents that treatment is not necessary, unless:

- The tumour obstructs a vital organ
- The tumour has the potential to impair function
- The tumour is on a site where life may be threatened, e.g. epiglottis (may obstruct the airway)

- There are multiple lesions (>5) on the skin (as that may imply internal organ involvement)

In these cases, the patient should be referred to a dermatologist for possible topical corticosteroids, oral propranolol or laser treatment.

Congenital Syphilis

Pathophysiology

Treponema pallidum can cross the placenta in an infected and untreated mother, and can infect the foetus. Foetal infection can occur at any stage of pregnancy. *T. pallidum* can also be transmitted to exposed neonates. Clinical manifestations may appear within the first 2 years of life (early disease) or after 2 years of life (late disease – not infectious).

Untreated syphilis during pregnancy (especially early syphilis) can lead to miscarriage.

Congenital syphilis has no primary stage (see images related to secondary syphilis here.

Clinical Features

Early congenital syphilis can present with only a rash, which may delay diagnosis. The child may have vesicles and bullae or a maculopapular rash on the palms and soles. There may be associated desquamation.

Other signs and symptoms include:

- Fever
- Lymphadenopathy
- FTT
- Hepatosplenomegaly
- Jaundice
- Meningitis
- Osteochondritis
- Pneumonitis
- Rhinitis
- Prematurity/low birth weight
- Deafness
- Neurological impairment

- Bone deformities

Investigations

One should order:

- Maternal TPHA/RPR
- FBC (to identify anaemia and thrombocytopaenia)
- LFTs
- Chest X-ray
- Full body X-ray

Management

One must try and prevent vertical transmission. Therefore, all pregnant patients with syphilis should be treated with 2.4 MU benzathine penicillin IM weekly for three weeks.

The symptomatic child with a positive syphilis test should be treated with penicillin G (50 000 IU/kg IVI twice daily for 10 days). Treatment must not be interrupted. The asymptomatic child with a positive test should be treated with penicillin G (50 000 IU IMI stat).

Pigmented Naevus

Its cause is unknown but it is the result of benign proliferation of melanocytes. One should be suspicious of malignancy if:

- A – there is asymmetry
- B – borders are poorly defined
- C – the colour is not uniform
- D – the lesion has a diameter >6 mm
- E – the lesion is evolving (changes in shape, size or colour)

Clinical Features

The child will have a well-circumscribed, brown/black papule or macule.

Management

The benign acquired naevus does not require excision. The large congenital naevus (see related images here) should be regularly monitored. The child with an atypical naevus should be referred to a dermatologist.

Mongolian Spot

It is common in Asian and Black neonates and less common in Caucasian neonates.

Pathophysiology

This lesion is benign and often fades. It is the result of the delayed disappearance of dermal melanocytes.

Clinical Features

The child will have congenital, blue-grey patches with indefinite borders. These patches are most common in the sacral area.

Café-au-Lait Spot

It is a localised area of increased melanogenesis of unknown cause.

Clinical Features

The child will have pigmented macules or patches which are present from birth or appear during early childhood. The colour of these lesions ranges from tan to dark brown.

Management

Café-au-lait spots are associated with neurofibromatosis (the presence of six or more spots is a diagnostic criterion). Patients who meet this criterion should be referred to neurology.

HAIR CONDITIONS

Alopecia Areata

Pathophysiology

The hair loss is possibly due to an autoimmune process. However, the exact mechanism remains unknown. The hair loss is sudden and occurs at any age

without preceding inflammation. Spontaneous regrowth is expected in ~6-18 months in patients with mild disease. The more the hair loss, the worse the prognosis.

Clinical Features

The child will have well-defined, patchy hair loss with normal underlying scalp.

Management

The child and parents should be reassured if there is only a single patch. However, a potent topical steroid gel or lotion may still be applied.

Patients with extensive or recurrent hair loss should receive emotional support and can be encouraged to purchase a wig, as treatment might not work.

Traction Alopecia

Pathophysiology

This hair-loss (reflecting on images here) is as the result of bad hair-grooming practices which cause breaking of the hair e.g. trichotillomania (the patient pulls out his/her own hair. In children, this may signify family stress. There may be permanent hair loss in patients with traction alopecia if the damage is severe.

Clinical Features

The child will present with hair loss at traction sites, commonly the scalp borders or vertex. At the site of alopecia, there will be damaged, brittle and broken off hair.

Management

One must explain the likely cause to the patient and parents and give advice on hairdressing techniques.

Scarring Alopecia

Pathophysiology

This type of hair loss can follow a traumatic, inflammatory or neoplastic process. The loss of hair is usually permanent.

Clinical Features

Affected scalp is bound down by fibrosis (see related images here). There is also a change in pigmentation.

Management

One must explain that the hair loss is permanent. Chloroquine should be given if the child has discoid lupus. A dermatology referral should be made if the cause is unknown. Referral to a plastic surgeon may be considered.

Pediculosis (Lice Infestation)

It is common in children and is spread by close contacts. Therefore, there are often outbreaks at schools.

Clinical Features

The child will present with intense scalp pruritus and white nits attached to the hairs. The constant scratching can lead to impetigo and secondary eczema.

Management

Apply permethrin 5% topical lotion to kill the lice. The nits may be removed by going through the freshly-washed hair with a fine-toothed comb or shaving the head. Patients with lice-infested eyelashes should be referred as there might have been sexual abuse.

TRAUMA, DRUG REACTIONS AND BITE REACTIONS

Keloid

Pathophysiology

It is an abnormal growth of scar tissue in individuals who are genetically predisposed. It may grow in response to trauma or spontaneously appear (see related images here).

Clinical Features

The child will present with firm, rubbery nodules that vary in size. Keloid lesions tend to cross the scar borders and are less painful.

Management

A zinc oxide (pressure) plaster and intralesional steroids should be applied. The patient should be referred to a plastic surgeon if the lesion is unresponsive or severe.

Hypertrophic Scar

Pathophysiology

An excessive amount of collagen is deposited, producing a raised scar. It is similar to a keloid but the lesion is within the margins of the injury.

Clinical Features

These lesions stay within the scar border, are inflamed and are very painful.

Management

The patient should be treated with intralesional steroid injections or silicone gel compression.

Fixed Drug Eruption

Pathophysiology

This is a skin reaction that tends to appear following ingestion of certain medications or substances. Any drug has a potential to cause a fixed drug eruption, but it is most commonly seen with laxative, analgesic or sulphonamides use.

Clinical Features

The patient will have round, sharply-demarcated, hyperpigmented macules. They are initially erythematous but go on to blister and then resolve (purple-grey colour). These lesions recur at the same spot every time the patient takes the offending medication/substance (see related images here).

Management

Treat the patient with an alternative drug and order a MedicAlert bracelet.

Pyogenic Granuloma

It is a small, benign vascular tumour that develops in response to trauma (see related images here).

Clinical Features

Pyogenic granulomata are common on the fingers. The child will have a nodule with a glistening, smooth and eroded surface. It easily bleeds on contact.

Management

Treatment may be with one of the options below:

- Excision or curettage removal followed by electrocautery of the base
- Application of very potent steroids
- Chemical cauterisation with a silver nitrate stick

Papular Urticaria

It is a common, chronic pruritic condition which is caused by allergy to insect bites. It usually affects young children.

Clinical Features

The lesions/bites occur in a line (“breakfast, lunch, dinner” distribution) or a group.

Management

It includes:

- Informing the patient and parents of the cause of the allergy
- Educating the family about chronicity
- Treating pets for fleas
- Checking mattresses for bed bugs
- Fumigating the home
- Applying topical corticosteroids
- Treating infected lesions with antibiotics
- Prescribing antihistamines for itch control and sedation

DERMATOLOGY SUMMARY (CHEAT SHEET)

Table 13.5: Brief Summary of Dermatological Conditions in Children

Eczema	Acute	
	Chronic	
	Face or flexures	Atopic eczema
	Sun-exposed areas	Photosensitivity
	Area of contact	Contact eczema
	Perineum or thighs in infants	Napkin eczema
	Round lesions	Nummular eczema
	Scalp, face, chest or napkin area	Seborrhoeic eczema
Scaly papules and plaques	Pink or red plaques with scale	Psoriasis
	Pink or red plaques on the trunk	Pityriasis rosea
	Purple/brown/silver papules	Lichen planus
Erythematous and purple lesions	Single red area	Erysipelas
	Tender pustule	Boil
	Flat and congenital	Port-wine stain
	Raised and congenital	Haemangioma
	Several, raised and itchy	Urticaria
	Raised, red and non-itchy	Fixed drug eruption
	Tender nodules	Erythema nodosum
	Widespread, erythematous rash	Viral infection, streptococcal infection or drug reaction
Papules and nodules	Red or pink	Erythema nodosum, boil or acne
	Purple	Lichen planus
	Brown or black	Pigmented naevus or wart
	Skin-coloured	Wart, molluscum contagiosum or keloid
Blistering diseases	Vesicles on erythematous skin	Herpes simplex, chickenpox or acute eczema
	Vesicles on normal skin	Papular urticaria
	Round, large and transparent	Bullous impetigo
	Concentric circles with a bullous centre	Erythema multiforme, fixed drug eruption
	Pustular	Scabies, acne, boil or folliculitis
Altered pigmentation	Smooth, depigmented skin	Albinism or vitiligo
	Scaly, hypopigmented skin	Pityriasis sicca alba or tinea versicolor

	Smooth, hyperpigmented skin	Mongolian spot or café-au-lait spot (see related images here)
	Scaly, hyperpigmented skin	Tinea versicolor