

## Paediatric Dermatological Conditions Introduction & Neonatal Skin Disorders

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### Principles of Diagnosis

#### Ten Basic Questions for a History

##### Special History

- When, where and how did it start?
- Has it occurred anywhere else?
- How does it behave?
- What affects it?
- Does it itch, hurt, burn, or anything else?

##### General History

- Do you/your child suffer from any symptoms not related to the skin?
- Have you/your child been ill recently or previously?
- Have your child been treated topically or systemically, and if so how?
- Are there or have there been similar problems in other family members?
- What are your thoughts about the cause of the problem?

### Description & Terminology of Skin Diseases

Basic vocabulary of well defined terms is indispensable for a proper description of dermatological conditions

#### Primary Skin lesions

(These are fundamental morphological changes that appear first on formerly unchanged skin.) They include:

- Macule, Patch, Papule, Plaque, Nodule, Tumour (Mass), Wheal, Vesicle (Small blister), Bulla (large blister)

#### Secondary Skin Lesions

(These usually do not develop on uninvolved skin, but mostly arise from alteration of primary lesions)

- Scale & Keratosis, Erosion & Excoriation, Fissure & Ulcer, Crust & Scar,

#### Primary Skin Lesions

**MACULE** – circumscribed, flat area of skin different in colour or texture from the surrounding, normal skin. A macule does not exceed 1 cm in greatest diameter. Multiple small hyper pigmentations on nose & cheeks (freckles)

**PATCH** – A large macule more than 1 cm in diameter. A macule or patch can result from: Deposition of endogenous (hemosiderin) or exogenous products (tattooing, topical agents, ingrained dirt, systemic drugs). Extravasation of blood (petechiae, purpura, ecchymoses, haematoma). Changes in melanin content of the epidermis or dermis (hyper- and hypopigmentation or depigmentation, melanoderma and leukoderma). Active erythema and passive hyperaemia (cyanosis). Diminished blood supply and vasoconstriction. Occasionally, macules and patches may be slightly depressed below the skin surface or show minor surface changes, such as scaling.**PAPULE** – a circumscribed solid elevation of the skin up to 1 cm in diameter,

mostly caused by tissue proliferation or cell infiltration. The following papules can be distinguished: An epidermal papule is composed of localized thickening of the epidermis or of the stratum corneum. A dermal papule is composed of localized, solid thickening of the upper dermis produced by hyperplasia of dermal structures, deposition of metabolic products, concentration of cells, or pathologic changes. Dermo-epidermal papules are formed by both epidermal and dermal abnormalities. Caused by an abundance of different pathological processes, papules may show a considerable variety of shapes, colours and surface qualities.

**PLAQUE** – a circumscribed, superficial, solid elevation of the skin greater than 1 cm in diameter. Plaques may occur as primary lesions but may also result from coalescence of papules and then strictly speaking represent secondary lesions.

**NODULE** – a circumscribed solid lesion of the skin up to 1 cm in size with depth. Nodules may not be absolutely present as an elevation, but can always be palpated. The distinction between papule and nodule depends essentially on extension to the depth. Nodules may be located deep dermally, dermal-subdermal or solely in the sub-cutaneous fat.

**TUMOUR** (mass) – a solid lesion of the skin greater than 1 cm in diameter with more than superficial height or palpable depth. Tumours differ from papules and nodules by size, from plaques by endophytic and exophytic extension. A tumour may be inflammatory or non-inflammatory, benign or malignant.

**WHEAL** – a transient dermal oedema, varied in size. Wheals are characteristically evanescent, disappearing within up to 24 hours. The changeability is due to the fact that they are mostly made up of circumscribed, rapidly resorbed accumulation of interstitial fluid in the upper dermis and hardly of cellular components. The colour is pale red if the capillaries are dilated, or whitish if the dermal oedema is heavy enough to compress the blood vessels. Wheals typically cause itching which is answered by rubbing. They are the characteristic lesion of urticaria. Whether a wheal should be regarded as a basic dermatologic lesion or better described as a type of plaque or papule is disputed. Multiple coalescing marginated lesions on the trunk (acute urticaria)

**VESICLE** (small blister) – a circumscribed elevation of the skin up to 1 cm in diameter and containing fluid.

**BULLA** (large blister) - a circumscribed elevation of the skin greater than 1 cm in diameter containing a fluid. The distinction between vesicle and bulla depends on size only. The term blister unites vesicle and bullae.

Types of vesicles and bullae: A subcorneal vesicle or bulla is formed by exudate beneath the stratum corneum as in bullous impetigo. Intra-epidermal vesicles or bullae are located within the epidermis. Subepidermal vesicles or bullae – the roof of the bulla is composed of the entire epidermis.

A dermal vesicle or bulla is caused by separation of tissue components of the dermis. Blisters may house serum, blood, lymph or a mixture of these fluids.

Depending on their pathogenesis they may consist of unilocular or multilocular blisters. Vesicles are almost always tense, bullae may be tense or flaccid.

**PUSTULES** – a circumscribed superficial elevation of the skin filled with pus.

### Common Terms in Clinical Dermatology

Abscess

Aphtha –small ulcer of mucous membranes

Cyst – any closed cavity with an epidermal, endothelial or membranous lining containing fluid or soft material

Erythema – transient redness

Erythroderma – generalized redness associated with infiltration and desquamation of the skin

Exanthem – widespread rash of similar skin lesions

Gangrene – necrotizing process due to arterial occlusion or infection

Impetiginization

Lichenification - thickening of the skin with accentuation and coarsening of the skin markings

Milium – tiny white cyst containing keratin

Petechia – isolated punctate haemorrhagic spot

Purpura – eruption of many petechiae

Scab – devitalized portion of the skin due to necrosis

Telangiectasia – visible dilation of small cutaneous blood vessels

### Morphological characteristics

#### Size & Colour

**Shape** - guttate (drop shaped), nummular (coin shaped), discoid (coin shaped), polygonal (several sides), annular (ring-like), serpiginous (wavy, snake-like), target lesion (iris lesion), linear, whorled, arcuate (arc-like)

#### Contour

**Surface characteristics** – roughness, smoothness, shine, dullness, verrucous, papillomatous

Margins – regular, irregular, sharply or indistinctly marginated

Consistency – soft, pasty, firm, hard, fluctuating, compressible, lobulated

**Number** – low number of lesions up to 10 should be counted, higher numbers may be called numerous, multiple or countless

**Arrangement**- how the lesions are arranged

**Köbner Phenomenon** - Psoriatic lesions around the neck provoked by a necklace

**Nikolski's sign** - When exerting tangential pressure on apparently normal skin, particularly near vesicles, the epidermis or parts of it may be detached. (TEN, EB)  
Dermographism - This term refers to the cutaneous response to a firm stroke applied with a wooden spatula to the skin on the back. A bright red non-raised line due to vasodilatation occurs after 3-15 seconds (red dermographism).

In patients with atopic dermatitis the response is paradoxically anaemic (white dermographism)

### Neonatal Skin and Skin Disorders

**Cutis Marmorata** -A transient, benign, reticulate, mottled, bluish discolouration of the skin that may last minutes to hours. Both full term and preterm infants may be affected

**Skin Fragility** (fissures & skin fragility in premature) - Neonatal skin exhibits a fragility that is not present in mature skin Weakened attachments between epidermis & dermis that are easily severed by physical or chemical trauma. This leads to iatrogenic abrasion from adhesive tape and other mild forms of trauma. Fragility is pronounced in preterm infants.

**Peeling** - Desquamation of neonatal skin is present in most term neonates in the first few days of life. Most pronounced in infants born 40-42 weeks gestation. Physiological peeling – hands, ankles and feet. Post mature peeling – extremities and trunk

**Lanugo** - Hair Prominent in premature infants, first coat of hair usually shed in utero during last trimester and replaced with second coat of shorter lanugo hair

**Epstein's Pearls** - Benign cystic lesions occur along the median palatal raphe, most commonly at the junction of the hard & soft palate. Occurs in 60-85% of neonates. Epidermal inclusion cysts - contain desquamated keratin. Counterpart of milia. No therapy indicated, rupture spontaneously.

**Sucking Calluses** - Sucking calluses develop on the lips as solitary, oval thickenings or extensive vermillion. When these lesions are congenital, they are indicative of vigorous sucking in utero. Presentation after birth is more common in breast fed, black infants. Sucking calluses involute spontaneously within a few days to weeks after birth or upon cessation of breast-feeding.

**Sebaceous Gland Hyperplasia** - Sebaceous gland hyperplasia occurs in > 50% term infants. Multiple, pinpoint, yellowish papules seen at the opening of each pilosebaceous follicle in areas where sebaceous glands are abundant, such as the nose. Sebaceous hyperplasia results from the influence of maternal androgens on the pilosebaceous follicle. Lesions resolve spontaneously

**Milia** - Tiny epidermal inclusion cysts. White, pearly, firm 1-2mm globular papules. Occur on the nose, cheeks, chin and foreheads in 40% of term neonates. Larger solitary lesions can be seen on the foreskin, scrotum, areolae and labia majora. Usually appear and disappear spontaneously during first month of life.

**Background:** Milia are very common, benign, keratin-filled cysts that occur in persons of all ages, from infants to elderly persons. Primary milia are typically seen in infants but also may occur in children and adults.

**Pathophysiology:** Milia are tiny epidermoid cysts. The cysts may be derived from the pilosebaceous follicle. Primary milia arise on facial skin bearing vellus hair follicles.

**Frequency:** Primary milia in newborns are so common that they can be considered normal. **Race:** No racial predilection is recognized. **Sex:** Sexual prevalence is equal. Eruptive milia and milia en plaque occur more frequently in women. **Age:** Milia occur in persons of all ages.

**History:** Milia are asymptomatic lesions. In children and adults, they usually arise around the eye.

**Physical:**

- Skin lesions: Milia are superficial, uniform, pearly white to yellowish, domed lesions measuring 1-2 mm in diameter.
- Skin distribution: Primary milia, in term infants, occur on the face, especially the nose. They also may be found on the mucosa and palate. Palatal lesions are known as Epstein pearls.

**Causes:** Primary milia are believed to arise in sebaceous glands that are not fully developed, explaining the high prevalence in newborn infants.

**Lab Studies:** No investigations are needed for simple milia. The clinical appearance is diagnostic.

**Procedures:** Performing a skin biopsy is necessary only if the diagnosis is in doubt.

**Histologic Findings:** The histological features are identical to those of epidermoid cysts, but the cysts are much smaller. The milium is usually located in the superficial dermis and has a complete epithelial lining (with a granular cell layer). It contains a variable amount of lamellated keratin. The common primary milia in infants and children are found in the undifferentiated sebaceous hair collar surrounding vellus hair follicles.

**Medical Care:** No topical or systemic medications are effective on primary milia.

**Complications:** No systemic complications have been reported.

**Prognosis:** Milia seen in infancy tend to spontaneously disappear within the first few weeks of life.

**Neonatal Mastitis** - Maternal and placental hormonal effects on the neonate.

Palpable breast tissue hypertrophy is typical of term infants. Secretion of colostrum like substance called “witches milk” late during first week of life. Infrequently, stagnant milk can become infected leading to mastitis and abscess formation.

**Mongolian Spots** - Brownish, blue-gray or blue-black patch usually located over the sacro-gluteal area. Most common of all birthmarks in pigmented races 80% in blacks but only 10% in whites. Increases in size over first year and then fades. No treatment necessary.

**Salmon Patch/ Stork Bite Nevus** - Occipital and nuchal telangiectatic changes

**Vesicopustular & Bullous disease of the Newborn**

**Erythema Toxicum**

**Background:** Erythema toxicum neonatorum (ETN) is a benign self-limited eruption occurring primarily in healthy newborns in the early neonatal period. ETN is characterized by macular erythema, papules, vesicles, and pustules, and it resolves without permanent sequelae.

**Pathophysiology:** Increased levels of immunological and inflammatory mediators (eg, interleukins 1 and 8, eotaxin, the adhesion molecule E-selectin, the water channel proteins aquaporin 1 and aquaporin 3, the chemotactic factor psoriasin, nitric oxide and its isoforms, and the antimicrobial peptide LL-37) suggest that ETN may be an immune system reaction. Its location to primarily hair-bearing areas suggests that the hair follicle may be involved.

The eosinophilic infiltrate of ETN suggests an allergic-related or hypersensitivity-related etiology, but no allergens have been identified. Similarly, contactants and mechanical irritation have been considered and rejected as etiologies. Newborn skin appears to respond to any injury with an eosinophilic infiltrate. Because ETN rarely is seen in premature infants, it is believed that mature newborn skin is required to produce this reaction pattern.

**Frequency: Internationally:** ETN occurs in one third to one half of full-term infants and in 5% of premature infants.

**Mortality/Morbidity:** ETN is a self-limited eruption that resolves spontaneously. Although one study found that infants with ETN had an increased risk of atopy, subsequent studies have failed to support this finding.

**Race:** No racial or ethnic predisposition is known. **Sex:** The prevalence is higher in males (55%) than in females (30%), except among females born of first pregnancies, who have a higher rate than males of first pregnancies.

**Age:** Erythema toxicum neonatorum presents within the first 4 days of life in full-term infants, with the peak onset occurring within the first 48 hours following birth. Rare cases have been reported at birth. Incidence rises with increasing gestational age and birth weight. Delayed onset rarely may occur in full-term and preterm infants up to age 14 days.

**History:** Focus the history on age at onset of the eruption, absence of systemic signs (eg, fever, irritability, lethargy, mucocutaneous involvement), or maternal history of herpes simplex/varicella viral infection, bacterial pyoderma, or candidiasis. Infants with ETN otherwise are healthy and lack systemic symptoms. The eruption is self-limited with most cases resolving within 5-14 days without residual sequelae. Recurrences are uncommon but have been reported up to the sixth week of life. They tend to be mild in severity.

**Physical:** Focus the physical examination on location, size, and distribution of macules, wheals, papules, and pustules on the skin. Note the absence of mucosal, palmar, or plantar involvement. Signs of systemic toxicity, including hypothermia or hyperthermia, lethargy, and irritability, are not associated with ETN.

- ETN most commonly presents with a blotchy, evanescent, macular erythema, often on the face or trunk. The macules are irregular, blanchable, and vary in size.
  - In more severe cases, pale yellow or white wheals or papules on an erythematous base may follow. In approximately 10% of patients, 2-4 mm pustules develop. Numbers and distribution of lesions vary from a few and widely scattered to numerous and extensive.
  - Sites of predilection include the forehead, face, trunk, and proximal extremities, but lesions may occur anywhere, including the genitalia. Involvement of the mucous membranes and palms and soles rarely occurs.
- Causes:** The cause of ETN is unknown. Multiple theories have been proposed to explain this common disorder.
- Neonates have an increased number of hair follicles compared with adults, and the occurrence of ETN in non-hair-bearing areas such as palms and soles is rare. Inflammatory cells tend to concentrate around hair follicles, and coccilike microbes have been demonstrated in the follicular epithelium and inside the inflammatory cells. This suggests that ETN may be a response to microbes that have penetrated the hair follicle. This process may possibly be integral in developing the new immune system.
  - The high frequency of eosinophilia suggests an allergic basis, leading some authors to suggest that ETN may be an immediate hypersensitivity reaction to a

substance passed from the mother transplacentally; however, convincing support is lacking for this theory.

- No responsible exotoxin, allergen, component of sebum, or infectious agent has been linked credibly to ETN.
- Medications administered to newborns and the mode of feeding have no effect on incidence.
- Other proposed theories include a transient adjustment reaction of the skin to mechanical or thermal stimulation or an acute graft-versus-host reaction induced by the maternal-fetal transfer of lymphocytes before or during delivery.

**Risk factors** include birth in hot, wet climates, being fed on a mixed diet or milk powder substitute, and being born via vaginal delivery. A positive correlation has been recognized between the length of labor and both the incidence of ETN and the duration of the cutaneous manifestations.

#### **Transient Neonatal Pustular Melanosis -**

**Background:** Transient neonatal pustular melanosis is a benign skin condition with distinctive features characterized by vesicles, superficial pustules, and pigmented macules. The lesions are commonly present at birth and are most likely to appear on the chin, neck, forehead, chest, and back. Although less common, lesions may be seen on palms and soles. The vesicles and pustules usually resolve within 48 hours, while the brown macules may persist for several months.

**Frequency:** In the US: The rate of transient neonatal pustular melanosis is estimated to be 0.1-0.35% in white infants and 4-5% in black infants. The overall rate has been reported to be as high as 2.2%.

**Mortality/Morbidity:** Transient neonatal pustular melanosis is a benign, asymptomatic, and self-limiting skin eruption with no associated mortality or morbidity.

**Race:** Transient neonatal pustular melanosis occurs in as many as 5% of African American newborns and in less than 0.4% of white infants. **Sex:** This condition occurs equally in both sexes. **Age:** Transient neonatal pustular melanosis is present at birth.

**History:** Often, only pigmented macules are present at birth, in which case the pustular phase may have occurred in utero. Skin findings can be correlated with gestational age at birth. Postterm infants are more likely to have pigmented macules. No systemic symptoms are associated with the skin lesions.

**Physical:** Transient neonatal pustular melanosis is characterized by vesicles, superficial pustules, and pigmented macules. Because of the fragile nature of the superficial pustules, most of them are broken in the initial drying or cleansing of newborns. Intact lesions may remain in more protected areas such as beneath the chin, in the axillae, or in the groin. The vesicles and pustules may desquamate with the neonate's first bath, leaving a white collarette of scale and brownish macules. Therefore, depending on the time of the examination in the neonatal period, the vesicles, pustules, and pigmented macules may be found predominantly on the chin, neck, or forehead; behind the ears; or on the trunk, palms, and soles. The lesions are 2-10 mm in diameter. Vesicular eruptions are usually 2-4 mm and are often filled with milky fluid. No systemic signs are associated with the skin eruptions. Papules are not seen in transient neonatal pustular melanosis, but they may be seen in neonates with

erythema toxicum neonatorum, acne neonatorum, or miliaria. The vesiculopustular lesions may be similar to lesions seen in acropustulosis. However, patients with acropustulosis have lesions that cluster on the palms and soles.

**Causes:** The etiology is unknown. No familial predisposition has been identified.

**Lab Studies:** The diagnosis is usually made at clinical examination. A Tzanck smear may be performed. With a cellular stain (eg, Wright-Giemsa stain), a Tzanck smear reveals a predominance of neutrophils without evidence of bacteria, yeast, or viroplasmic changes. Gram stain preparations for bacteria are negative. Blood and skin culture results are negative.

**Histologic Findings:** Vesicles and pustules show intracorneal and subcorneal collections of neutrophils with some eosinophils and, occasionally, fragmented hairs. The dermis has an infiltrate of neutrophils and scattered eosinophils. The brown macules show epidermal basal cell melanosis.

**Medical Care:** No specific therapy is indicated.

**Deterrence/Prevention:** Contagious isolation is unnecessary.

**Prognosis:** The prognosis for this benign condition is good. The vesicles and pustules usually resolve within 48 hours, while the brown macules may persist for several months.

**Glands of the Skin -** Eccrine gland ducts exit directly on the skin surface independent of the hair follicle.

#### **Miliaria**

**Background:** Miliaria is a common disorder of the eccrine sweat glands that often occurs in conditions of increased heat and humidity. It is thought to be caused by blockage of the sweat ducts, which results in the leakage of eccrine sweat into the epidermis or dermis.

The 3 types of miliaria are classified according to the level at which obstruction of the sweat duct occurs. In **miliaria crystallina**, ductal obstruction is most superficial, occurring in the stratum corneum. Clinically, this form of the disease produces tiny, fragile, clear vesicles. In **miliaria rubra (prickly heat)**, obstruction occurs deeper within the epidermis and results in extremely pruritic erythematous papules. In **miliaria profunda**, ductal obstruction occurs at the dermal-epidermal junction. Sweat leaks into the papillary dermis and produces subtle asymptomatic flesh-colored papules. When pustules develop in lesions of miliaria rubra, the term miliaria pustulosa is used.

**Pathophysiology:** The primary stimuli for the development of miliaria are conditions of high heat and humidity that lead to excessive sweating. Occlusion of the skin due to clothing, bandages, or plastic sheets (in an experimental setting) can further contribute to pooling of sweat on the skin surface and overhydration of the stratum corneum. In susceptible persons, including infants, who have relatively immature eccrine glands, overhydration of the stratum corneum is thought to be sufficient to cause transient blockage of the acrosyringium.

If hot humid conditions persist, the individual continues to produce excessive sweat, but he or she is unable to secrete the sweat onto the skin surface because of ductal blockage. This blockage results in the leakage of sweat en route to the skin surface, either in the dermis or epidermis, with relative anhidrosis.

When the point of leakage is in the stratum corneum or just below it, as in miliaria crystallina, little accompanying inflammation is present, and the lesions are

asymptomatic. In contrast, in miliaria rubra, the leakage of sweat into the subcorneal layers produces spongiotic vesicles and a chronic periductal inflammatory cell infiltrate in the papillary dermis and lower epidermis. In miliaria profunda, the escape of sweat into the papillary dermis generates a substantial, periductal lymphocytic infiltrate and spongiosis of the intra-epidermal duct.

Resident skin bacteria, such as *Staphylococcus epidermidis* and *Staphylococcus aureus*, are thought to play a role in the pathogenesis of miliaria. Patients with miliaria have 3 times as many bacteria per unit area of skin as healthy control subjects. Antimicrobial agents are effective in suppressing experimentally induced miliaria. In an experimental setting, only the strains of *S epidermidis* that produce EPS can induce miliaria.

**Frequency:**

**In the US:** Miliaria crystallina is a common condition that occurs in neonates, with a peak in those aged 1 week, and in individuals who are febrile or those who recently moved to a hot, humid climate. Miliaria rubra also is common in infants and adults who move to a tropical environment; this form occurs in as many as 30% of persons exposed to such conditions. Miliaria profunda is a rarer condition that occurs in only a minority of those who have repeated bouts of miliaria rubra.

**Internationally:** The best data about the incidence of miliaria in newborns are from a Japanese survey of more than 5000 infants. This survey revealed that miliaria crystallina was present in 4.5% of the neonates, with a mean age of 1 week. Miliaria rubra was present in 4% of the neonates, with a mean age of 11-14 days.

Worldwide, miliaria is most common in tropical environments, especially among people who recently moved to such environments from more temperate zones.

**Mortality/Morbidity:** The complications of miliaria are altered heat regulation and secondary infection.

**Miliaria crystallina** is generally an asymptomatic self-limited condition that resolves without complications over a period of days. It may recur if hot, humid conditions persist.

**Miliaria rubra** also tends to resolve spontaneously when patients are moved to a cooler environment. Unlike patients with miliaria crystallina, however, those with miliaria rubra tend to be symptomatic; they may report itching and stinging. Anhidrosis develops in the affected sites and may last weeks. If generalized, anhidrosis can lead to hyperpyrexia and heat exhaustion. Secondary infection is another possible complication of miliaria rubra; this appears as either impetigo or multiple discrete abscesses known as perioritis staphylogenes.

**Miliaria profunda** is itself a complication of repeated episodes of miliaria rubra. The lesions of miliaria profunda are asymptomatic, but compensatory facial hyperhidrosis and axillary and inguinal adenopathy may develop. The widespread inability to sweat, the result of eccrine ductal rupture, is known as tropical anhidrotic asthenia; this condition predisposes patients to heat exhaustion during exertion in warm climates.

**Race:** Miliaria occurs in individuals of all races, although some studies show that Asians, who produce less sweat than whites, are less likely to have miliaria rubra.

**Sex:** No sex predilection is recognized.

**Age:** Miliaria crystallina and miliaria rubra can occur in persons of any age, but the diseases are most common in infants. Three cases of congenital miliaria crystallina are reported. Miliaria profunda is more common in adults than in infants and children.

**History:**

Miliaria crystallina

- This form usually affects neonates younger than 2 weeks and adults who are febrile or those who recently moved to a tropical climate.
- Lesions appear in crops within days to weeks of exposure to hot weather and disappear within hours to days.
- Lesions are generally asymptomatic.

Miliaria rubra

- This form usually affects neonates aged 1-3 weeks and adults who live in hot, humid environments.
- Lesions may occur within days of exposure to hot conditions, but they tend to appear after months of exposure.
- Lesions resolve within days after the patient is removed from the hot, humid environment.
- Lesions cause intense pruritus and stinging that is exacerbated by fever, heat, or exertion.
- Patients may report fatigue and heat intolerance, and they may notice decreased or absent sweating at the affected sites.

**Physical:**

Miliaria crystallina

- Lesions are clear, superficial vesicles that are 1-2 mm in diameter.
- Lesions occur in crops and are often confluent, without any surrounding erythema.
- In infants, lesions tend to occur on the head, neck, and upper part of the trunk.
- In adults, lesions occur on the trunk.
- Lesions rupture easily and resolve with superficial branny desquamation.

Miliaria rubra

- Lesions are uniform, small, erythematous papules and vesicular papules on a background of erythema.
- Lesions occur in a nonfollicular distribution and do not become confluent.
- In infants, lesions occur on the neck and in the groin and axillae.
- In adults, lesions occur on covered skin where friction occurs; these areas include the neck, scalp, upper part of the trunk, and flexures. The face and volar areas are spared.
- In late stages, anhidrosis is observed in affected skin.

Miliaria profunda

- Lesions are firm, flesh-colored, nonfollicular papules that are 1-3 mm in diameter.
- Lesions occur primarily on the trunk, but they can also appear on the extremities.
- Lesions are transiently present after exertion or other stimulus that results in sweating.
- Affected skin shows diminished or absent sweating.

- In severe cases that lead to heat exhaustion, hyperpyrexia and tachycardia may be observed.

#### Causes:

- Immaturity of the eccrine ducts: Neonates are thought to have immature eccrine ducts that easily rupture when sweating is induced; this rupture leads to miliaria.
- Lack of acclimatization: Miliaria is common in individuals who move from a temperate climate to a tropical climate. The condition usually resolves after the individual has lived in the hot, humid conditions for many months.
- Hot, humid conditions: Tropical climates, incubators in neonatal nurseries, and febrile illnesses may precipitate miliaria.
- Exertion: Any stimulus to sweat may precipitate or exacerbate miliaria.
- Bacteria: Staphylococci are associated with miliaria, and antibiotics prevent miliaria in an experimental setting.

#### Lab Studies:

- Miliaria is clinically distinctive; therefore, few laboratory tests are necessary.
- In miliaria crystallina, cytologic examination of the vesicular contents fails to reveal inflammatory cells or multinucleated giant cells (as would be expected in herpes vesicles).

**Histologic Findings:** In miliaria crystallina, intracorneal or subcorneal vesicles communicate with eccrine sweat ducts, without surrounding inflammatory cells. Obstruction of the eccrine duct may be observed in the stratum corneum. In miliaria rubra, spongiosis and spongiotic vesicles are observed in the stratum malpighian, in association with eccrine sweat ducts. Periductal inflammation is present.

#### Medical Care:

- No compelling reason to treat miliaria crystallina exists because this condition is asymptomatic and self-limited.
- Miliaria rubra can cause great discomfort, and miliaria profunda may lead to heat exhaustion. Treatment of these conditions is warranted.
- The prevention and treatment of miliaria primarily consists of controlling heat and humidity so that sweating is not stimulated. Measures may involve treating a febrile illness; removing occlusive clothing; limiting activity; providing air conditioning; or, as a last resort, having the patient move to a cooler climate.
- Antimicrobial agents are effective in suppressing experimentally induced miliaria.

#### Treatment

The goal of treating miliaria rubra and miliaria profunda is to provide symptomatic relief and prevent hyperpyrexia and heat exhaustion. The authors know of no strong evidence indicating efficacy with systemic medications; therefore, topical medications are preferred.

Drug Category: *Topical therapies* -- Anhydrous lanolin is believed to prevent ductal blockage, allowing sweat to flow to the skin surface. Calamine lotion provides cooling symptomatic relief after miliaria develops.

**Complications:** The most common complications of miliaria are secondary infection and heat intolerance.

**Prognosis:** Most patients recover uneventfully within a matter of weeks, once they move to a cooler environment.

#### Special Concerns:

Miliaria crystallina and miliaria rubra are common in infants; therefore, pediatricians must be able to distinguish these conditions from other common eruptions that affect infants.

- Miliaria crystallina can be confused with congenital herpes simplex, varicella, syphilis, candidiasis, or staphylococcal scalded skin syndrome. Cytologic findings in the blister fluid should rule out these conditions; cytologic methods may involve Tzanck preparation, Gram staining, and potassium hydroxide preparation, as well as the acquisition of a biopsy sample for histopathologic analysis.
- Miliaria rubra can be confused with erythema toxicum neonatorum, infantile acne, or folliculitis. Pustules of erythema toxicum are characteristically filled with eosinophils, unlike those of miliaria rubra. Infantile acne typically involves the face in a follicular distribution. Miliaria may involve the face, as well as the trunk and axillae. Superficial folliculitis, as its name suggests, is follicular, unlike miliaria.

**Neonatal acne (Transient Neonatal Cephalic Pustulosis)** - Onset at a few weeks of life. Multiple inflammatory, erythematous papules & pustules. Common on face, neck, chest & back. Resolves within months. Maternal & neonatal hormones may stimulate sebaceous glands and influence formation of acne.

**Epidermolysis Bullosa** - Mechanobulbous disorder. Neonatal vesicles, bullae & denuded skin, with friction and trauma induced blistering are the hallmark of EB. Various genetic etiologies. 3 Subtypes of EB – some lethal. Types: Simplex, Junctional & Dystrophic. Unable to distinguish between subtypes clinically. Have to do a skin biopsy to classify the type.

**Subcutaneous Fat Necrosis** - Idiopathic necrosis of the panniculus (subcutaneous fat) of the neonate. Indurated plaques or nodules below the skin. Term healthy infants. Also associated with perinatal asphyxia, hypoxaemia, hypothermia or other forms of trauma during labour & delivery.

#### Paediatric Dermatological Conditions -

##### Major Eczematous eruptions in childhood

- Atopic dermatitis
- Seborrhoeic dermatitis
- Nappy dermatitis
- Contact dermatitis

#### Definitions:

*Atopic dermatitis* – Chronic, Relapsing & Pruritis

Clinical features: Xerosis, Inflammation, Lichenification. Disease varies in location according to age of patient. Associated - family history, asthma, allergic rhinitis.

*Eczema* – (boiling over) Erythema, vesicles, scaling and crusts. May find eczematous lesions in conditions other than AD

*Dermatitis* - All inclusive term for inflammation of the skin.

#### Atopic Dermatitis

AD a disease of exacerbations & remissions. Most patients improve with age.

Prevalence ; 30% 4month old infants  
20% 3yr old children  
10% in adults

Major cause of morbidity in children in Western world. It affects lives of parents, affected children & secondary effects on the family. Genetic predisposition - Mode of inheritance not clear.

AD - usually first manifestation of atopic triad: AD, asthma & hay fever. Occurs in 60% during 1st yr of life.

There are three distinct clinical phases of AD and both the site & morphology of the lesions change with age. These phases may overlap or be separated by a period of remission

Infantile (0-2yrs of age)  
Childhood (2yrs-puberty)  
Adult (puberty onward)

#### **Infantile AD**

Pruritis - characteristic & most NB symptom and a major cause of morbidity.

Eruption sites; on cheeks and scalp, often involves lateral aspects of extensor surfaces of lower legs, involve creases & trunk. The nappy area are often spared

#### *Morphology of lesions*

Usually symmetric, ill-defined, scaly, erythematous patches, with or without small areas of crusting. Major feature - generalised xerosis, including dry hair & scalp

#### **Childhood AD**

Sites of predilection: flexural areas and most commonly affected are the antecubital and popliteal fossae, neck, flexures of wrist & ankles and buttock/thigh crease. These areas are particularly prone to sweating.

#### *Morphology of lesions*

Pruritic, ill-defined, erythematous scaly patches often studded with crusts & excoriations. First manifestations of lichenification appear. Lichenification most often in antecubital fossae, around knees and wrists. AD lesions in black children more papular & follicular.

#### **Adult AD (After Puberty)**

Clinical features: Face, neck, body are more diffusely affected. Erythema & more scaling. Less exudation. Xerosis & lichenification still prominent.

#### **Adult chronic AD**

Distinctive rippled, brown, macular discolouration around the neck

#### **General**

AD lesions do not scar unless there is severe secondary infection. Post inflammatory hypo- & hyperpigmentation disappear after weeks or months.

Associated findings; Hyperlinear palms and soles Crease line under lower eyelid (Dennie-Morgan fold)

#### **Complications of AD**

Tendency to develop viral & bacterial skin infections

**Eczema herpeticum** – extensive HSV infection

Unknown why AD patients are susceptible to generalized HSV infection.

Primary infection: Febrile, small vesicles appear in area of eczema. Eruption spread to normal skin. Vesicles form erosions and crusts within 24-48 hours.

Rx Acyclovir IV & systemic antibiotics.

#### **Significant Staph aureus colonisation**

93% in involved skin

76% in non-involved skin

<10% in normal population

Clinical infection occurs frequently in AD patients but seldom results in severe systemic infection

Eczemous plaques in popliteal fossae with S. aureus secondary infection.

#### **Atopic dermatitis**

**Background:** Atopic dermatitis (AD) is a pruritic disease of unknown origin that usually starts in early infancy and is typified by pruritus, eczematous lesions, xerosis (dry skin), and lichenification on the skin (thickening of the skin and increase in skin markings). AD is associated with other atopic diseases (eg, asthma, allergic rhinitis, urticaria, acute allergic reactions to foods, increased immunoglobulin E [IgE] production) in many patients. It is a disease of great morbidity, and the incidence appears to be increasing.

**Pathophysiology:** The pathophysiology of AD is poorly understood. Several cell types seem to be involved, including T lymphocytes, eosinophils, Langerhans cells, and keratinocytes. Other factors, including cytokines and IgE, are also implicated. Laboratory findings suggest a number of different pathogenetic mechanisms. One invokes an immune defect involving an abnormality of T<sub>H</sub>2 cells that interacts with Langerhans cells and results in increased production of interleukin (IL)-4, IL-5, IL-6, IL-10, and IL-13. This leads to increased IgE and decreased gamma interferon levels. The imbalance of T<sub>H</sub>2 cells occurs in the acute process, with a swing toward T<sub>H</sub>1 cells in the chronic stages of the disease. Another theory involves defective barrier function in the stratum corneum leading to the entry of antigens, which results in the production of various inflammatory cytokines.

Xerosis is known to be an associated sign in most AD patients. The xerosis is thought to involve defective lipid (particularly ceramide) production. A third mechanism involves environmental antigens from food (the gut), dust mites (the lungs), and other factors and portals of entry that react with antibodies to produce increased levels of IgE and, possibly, increased histamine reactions from mast cells. Superimposed with these mechanisms is a genetic predisposition to react to various environmental allergens.

**Frequency: In the US:** The prevalence rate is 10-12% in children and 0.9% in adults. **Internationally:** The prevalence rate is as high as 18% and is rising, especially in developed countries. In China and Iran, the prevalence rate is approximately 2-3%. The frequency is increased in patients who immigrate to developed countries from underdeveloped countries.

**Mortality/Morbidity:** Incessant itch and work loss in adult life is a great financial burden. A number of studies have reported that the financial burden to families and government is similar to that of asthma, arthritis, and diabetes mellitus. In children,

the disease causes enormous psychological burden to families and loss of school days. Mortality due to AD is unusual.

- Kaposi varicelliform eruption (eczema herpeticum) is seen with some frequency in patients with AD. It usually occurs with a primary herpes simplex infection, but it also may be seen recurrently. Vesicular lesions can begin at any location, but they are particularly common in areas of eczema. The virus spreads rapidly to involve all eczematous areas and healthy skin. Lesions may become secondarily infected. Although vaccination with the vaccinia vaccine for the prevention of small pox is now no longer mandatory, patients with AD can contract eczema vaccinatum either from the vaccination of themselves or their relatives. This condition had a high mortality rate (up to 25%). In the current climate of threats of bioterrorism, vaccination may once again become necessary and physicians should be aware of eczema vaccinatum in this setting.
- With regard to bacterial infection (eg, with *Staphylococcus aureus* or *Streptococcus pyogenes*), note that the skin of most patients with AD is colonized by *S aureus*. Clinical infection may occur and is worsened by scratching and occlusion from medications. Eczematous and bullous lesions on the palms and soles are often infected with beta-hemolytic group A *Streptococcus*.
- Urticaria and acute anaphylactic reactions to food occur with increased frequency in patients with AD. The food groups most commonly implicated include peanuts, eggs, milk, soya, fish, and seafood.
- Of patients with AD, 30% develop asthma and 35% have nasal allergies.

**Race:** AD may be more common among whites, but it affects persons of all races.

**Sex:** The male-to-female ratio is 1:1.4. **Age:** In 85% of cases, AD occurs in the first year of life; in 95% of cases, it occurs before age 5 years.

- Disease is most prevalent in early infancy and childhood. The disease may have periods of complete remission, particularly in adolescence, and may then recur in early adult life.
- In the adult population, the rate of AD frequency diminishes to 0.9%. Rarely, onset may be delayed until adulthood, when the disease is more difficult to control.

**History:** Incessant pruritus is the only symptom. Although pruritus may be present in the first few weeks of life, parents become more aware of the itch as the itch-scratch cycle matures when the patient is approximately age 3 months; children then scratch themselves uncontrollably.

**Physical:** Primary findings include xerosis, lichenification, and eczematous lesions. The eczematous changes are seen in different locations, and the morphology changes with age.

#### Infancy

- AD may be noticed soon after birth. Xerosis also occurs in the neonatal period. Xerosis involves the whole body but usually spares the diaper area.
- The earliest lesions are often evident in the creases (ie, antecubital and popliteal fossae), where the lesions consist of erythema with exudation. Over the following few weeks, lesions localize to the cheeks and forehead and extensors of the lower legs, but they may occur in any location on the body, often sparing

the diaper area. Lesions are xerotic, erythematous, and scaly (eczematous) ill-defined patches and plaques.

- The scalp is frequently involved with a pruritic scaly dermatitis.
- Lichenification is seldom seen in infancy.

#### Childhood

- Xerosis is often generalized. The skin is flaky and rough.
- Lichenification is characteristic of childhood AD. It signifies repeated rubbing of the skin and is seen mostly over the folds and bony protuberances.
- Lesions are eczematous and exudative. Pallor of the face is common; erythema and scaling occur around the eyes. Dennie-Morgan folds (increased folds below the eye) are often seen. Flexural creases, particularly the antecubital and popliteal fossae, and buttock-thigh creases are often affected.
- Excoriations and crusting are common.

#### Adulthood

- Lesions become more diffuse with an underlying background of erythema. The face is commonly involved and has a dry, scaling appearance.
- Xerosis is prominent.
- Lichenification is present.
- A brown macular ring around the neck is typical but not always present.

**Williams diagnostic criteria:** According to the criteria of Williams et al, proposed diagnostic guidelines include the following:

- Patients must have an itchy skin condition (or parental report of scratching or rubbing in children).
- Patients also must have 3 or more of the following:
  - History of involvement of the skin creases, such as folds of the elbows, behind the knees, fronts of the ankles, or neck
  - Personal history of asthma or hay fever or a history of atopic disease in a first-degree relative in patients younger than 4 years
  - History of generally dry skin in the last year
  - Visible flexural dermatitis or dermatitis involving the cheeks or forehead and outer limbs in children younger than 4 years
  - Onset younger than age 2 years (not used if child is <4 y)

#### Causes:

- A genetic abnormality is possibly related to bands 11q13 or 5q31. These findings have yet to be corroborated; a family history of AD is common.
- The skin of patients with AD is colonized by *S aureus*. Lesions flare following infection by *S aureus*, but they may occur with any type of skin or systemic infection. *S aureus* has been proposed as a cause of AD by acting as a superantigen.
- AD flares occur in extremes of climate. Heat is poorly tolerated, as is extreme cold. A dry atmosphere increases xerosis. Sun exposure improves lesions, but sweating increases pruritus. All these external factors may act as antigens, ultimately setting up an inflammatory cascade.
- The role of food antigens in the pathogenesis of AD is controversial, both in the prevention of AD and by their effect with withdrawal of certain foods in persons with established AD. Most reported research has methodologic flaws. One

article showed improvement at 1 year with continued breastfeeding. At 4 years, no difference was noted in the incidence of AD between the group that had not been exclusively breastfed and the group that had.

- A role for aeroallergens and house dust mites has been proposed, but this awaits further corroboration.

**Other problems to consider:** AD occasionally is indistinguishable from other causes of dermatitis. In infancy, the most common difficulty is distinguishing it from SD. This entity is not seen with the same frequency as a decade ago. Both AD and SD are associated with cradle cap (a scale found on the vertex of the scalp), which is greasy and yellow in individuals with SD and dry and crusted in individuals with AD. Other areas of involvement in SD are the intertriginous areas, where marked erythema and a greasy scale can be seen over the eyebrows and the sides of the nose. In AD, xerosis of the skin and severe pruritus are seen, which are not usually features of SD. Both conditions should be distinguished from psoriasis.

Scabies manifests in infancy or childhood as a pruritic eruption. Other members of the family may be itchy, and the primary sites of involvement are moist warm areas. The eruption is polymorphic with a dermatitis, nodules, urticaria, and 6-10 burrows. Pustules on the hands and feet are common in infancy. Facial involvement is rare, and xerosis does not occur.

Other causes of dermatitis, particularly contact dermatitis from nickel in infants, are sometimes difficult to distinguish from AD. A central area of dermatitis (from nickel snaps in undershirts or snaps in jeans) is helpful for making the diagnosis, although a dermatitic eruption may occur as an Id reaction in other areas, particularly the antecubital fossae. Xerosis and facial involvement are absent. AD usually starts earlier than contact dermatitis.

Children with a severe itch and generalized dermatitis in the setting of recurrent infections should be investigated for evidence of an immunodeficiency. Failure to thrive and repeated infections help distinguish the eruption from AD.

Tinea corporis usually manifests as a single lesion, but inappropriate treatment with steroids may cause a widespread dermatitis. Facial involvement, the presence of xerosis, the age of appearance, and an early onset (in AD) help distinguish between the 2 conditions.

#### **Lab Studies:**

- Laboratory testing is seldom necessary.
- Allergy and radioallergosorbent testing is of little value.
- A platelet count for thrombocytopenia helps exclude Wiskott-Aldrich syndrome, and testing to rule out other immunodeficiencies may be helpful.
- Scraping to exclude tinea corporis is occasionally helpful.

**Histologic Findings:** Biopsy shows an acute, subacute, or chronic dermatitis, but no specific findings are demonstrated.

**Medical Care:** Patients with AD do not usually require emergency therapy, but they may visit the emergency department for treatment of acute flares caused by eczema herpeticum and bacterial infections.

#### **Moisturization**

- Depending on the climate, patients may benefit from short, cool showers or baths followed by the application of a moisturizer such as white petrolatum. Another regimen includes "soaking and greasing." Frequent baths with oil (1 capful of emulsifying oil added to lukewarm bath water) for 5-10 minutes comprise this regimen. In infants, 3 times a day is not a great burden; in adults, once or twice a day is usually all that can be achieved. Leave the body wet after bathing. Oil and water are kept in solution by an emulsifier in the oil, thus preventing evaporation of water to the outside environment.
- Advise patients to apply an emollient such as petrolatum all over the body while wet, to seal in moisture and allow water to be absorbed through the stratum corneum. The ointment spreads well on wet skin.

#### **Topical steroids**

- Topical steroids are currently the mainstay of treatment. In association with moisturization, responses to this regimen are excellent.
- Ointment bases are preferred, particularly in dry environments.
- Patients with AD may develop a contact allergy to topical medications and moisturizers. The allergy may be to a preservative or the active ingredient. Allergy to hydrocortisone is recognized with increasing frequency. Preservatives are less commonly present in ointments.
- Initial therapy consists of hydrocortisone 1% powder in an ointment base applied 3 times daily to lesions on the face and in the folds.
- A midstrength steroid ointment (desonide for milder areas or higher-strength steroids such as triamcinolone or betamethasone valerate for more severe areas) is applied daily to lesions on the trunk until the eczematous lesions clear.
- Steroids are discontinued when lesions disappear and are resumed when new patches arise.
- Flares may be associated with seasonal changes, stress, activity, staphylococcal infection, or contact allergy.

#### **Other treatments, effective and ineffective**

- Probiotics have recently been explored as a therapeutic option for the treatment of AD. The rationale for their use is that bacterial products may induce an immune response of the TH1 series instead of TH2 and could therefore inhibit the development of allergic IgE antibody production. This research, although garnering fairly convincing support, has yet to be proven.
- UV-A, UV-B, a combination of both, psoralen plus UV-A (PUVA), or UV-B1 (narrow band UV-B) therapy may be used. Long-term adverse effects of skin malignancies in fair-skinned individuals should be weighed against the benefits.
- In patients with eczema herpeticum, acyclovir is effective.
- In patients with severe disease, and particularly in adults, phototherapy, methotrexate (MTX), azathioprine, and cyclosporine have been used with success.

- Both hydroxyzine and diphenhydramine hydrochloride provide a certain degree of relief from itching but are not effective without other treatments.
- Ketotifen (a calcium channel blocker) may be effective.
- Oil of evening primrose was believed to be effective, but in a randomized controlled study, it showed no benefit in children and little improvement in adults.
- Results with many other medications, such as thymopentin, gamma interferon, and Chinese herbs, have been disappointing. Many medications are not practical to use, and they can be expensive. Some Chinese herbal preparations contain prescription medications, including prednisone.
- Antibiotics are used for the treatment of clinical infection caused by *S aureus* or flares of disease. They have no effect on stable disease in the absence of infection. Laboratory evidence of *S aureus* is not evidence of clinical infection because staphylococcal organisms commonly colonize the skin of patients with AD.

#### Nonmedical efforts

- Clothing should be soft next to the skin. Cotton 100% is comfortable and can be layered in the winter.
- Cool temperatures, particularly at night, are better because sweating causes irritation and itch.
- A humidifier (cool mist) prevents excess drying and should be used in both winter, when the heating dries the atmosphere, and in the summer, when the air conditioning absorbs the moisture from the air.
- Clothes should be washed in a mild detergent with no bleach or fabric softener.

**Consultations:** Consulting an allergist may be necessary, particularly if the patient develops asthma and/or hay fever or an acute reaction to a type of food.

**Diet:** Avoid foods that provoke acute allergic reactions (hives, anaphylaxis). Most frequently, allergic reactions occur to peanuts (peanut butter), eggs, fish and seafood, milk, soya, and chocolate. Advise patients to apply a barrier of petroleum jelly around the mouth prior to eating to prevent irritation from tomatoes, oranges, and other irritating foods.

**Activity:** Advise patients to avoid activities that cause excessive sweating. Swimming in an outdoor pool (or wading pool for babies) in summer provides therapeutic benefit by exposing the person to the sun but avoiding the heat.

#### Treatment

The basis of treatment is to provide moisturization for dryness, allay pruritus, and manage inflammation of the eczematous lesions.

**Anti-inflammatory agents** -- Provide relief of inflammation of eczematous lesions. Ointment base provides moisturization. White petrolatum is useful to avoid potential sensitization to preservatives in water-based moisturizers.

**Hydrocortisone** (LactiCare HC, Cortaid, Westcort, Dermacort, DermaGel) -- Mild topical corticosteroid mixed in petrolatum for facial application. Has mineralocorticoid and glucocorticoid effects, resulting in anti-inflammatory activity. Use 1% ointment daily. Apply sparingly to affected areas bid/tid; discontinue when cleared. Caution around eyes and in stasis dermatitis, prolonged use, and application over large surface areas; occlusive dressings may increase systemic absorption of corticosteroids

**Betamethasone valerate** (Betatrex, Valisone, Luxiq) -- Medium-strength topical corticosteroid for body areas. Decreases inflammation by suppressing migration of polymorphonuclear leukocytes and reversing capillary permeability. Affects production of lymphokines and has inhibitory effect on Langerhans cells. Use 0.05% ointment in pediatrics. Apply topically bid/tid until response; discontinue when cleared. Do not use in skin with decreased circulation; can cause atrophy of groin, face, and axillae; may cause striae distensae in teenagers or rosacea-like eruption; may increase skin fragility; rarely, may suppress HPA axis; if infection is present, discontinue use until infection is under control.

**Antihistamines** -- Provide symptomatic relief of pruritus.

**Hydroxyzine hydrochloride** (Atarax) -- Antihistamine with antipruritic, anxiolytic, and mild sedative effects. Antagonizes H1 receptors in periphery. May suppress histamine activity in subcortical region of CNS. Syrup available as 10 mg/5 mL.

**Diphenhydramine** (Benadryl) -- Antihistamine used for pruritus and allergic reactions.

**Antiviral agents** -- For management of herpetic infections and to treat AD in patients who develop chickenpox.

**Acyclovir** (Zovirax) -- Inhibits activity of both HSV-1 and HSV-2. Has affinity for viral thymidine kinase and, once phosphorylated, causes DNA-chain termination when acted on by DNA polymerase. Patients experience less pain and faster resolution of cutaneous lesions when used within 48 h of rash onset. May prevent recurrent outbreaks. Early initiation of therapy is imperative. Zoster dose is 4 times higher than that for herpes simplex. Duration of therapy varies.

**Antibiotics** -- Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens in the context of the clinical setting. For the treatment of clinical infection by *S aureus*, cloxacillin or cephalexin is used. In streptococcal infections, cephalexin is preferred. If not effective, penicillin and clindamycin in combination are effective. Consider staphylococcal infection in every flare of AD.

**Further Outpatient Care:** Monitor patients frequently. Reinforce therapeutic regimens with patients.

**Deterrance/Prevention:** Moisturization is important on an ongoing basis and seems to prevent flares.

#### Complications:

- If topical corticosteroids are used inappropriately or if superpotent steroids are used in teenagers during rapid growth, striae may occur. Skin thinning can result if steroids are used inappropriately in older patients.
- Whether verrucae vulgaris and mollusca contagiosa are more frequent is difficult to assess, but certainly, they can be more widespread and difficult to eliminate.
- Tachyphylaxis to topical steroids occurs if they are not used on a stop-start basis.

**Prognosis:** Most patients improve; this can occur at any age. While the frequency of AD is as high as 20% in childhood, it is 0.9% in adults. One third of patients develop allergic rhinitis. One third of patients develop asthma.

**Patient Education:** Frequently reinforce treatment and maintenance regimens with patients.

**Medical/Legal Pitfalls:** Failure to explain the adverse effects of topical steroids to patients may result in medico-legal problems.

#### Special Concerns:

- Other children in the family may develop AD.

- Patients may develop acute food allergies (eg, to peanuts and/or eggs). EpiPen should be available.
- Patients may develop a generalized reaction to herpes simplex virus (eczema herpeticum).
- One third of patients develop asthma, and one third develops allergic rhinitis.

### Seborrhoeic Dermatitis

Seborrhoea means flow of sebum. No evidence of increased sebum production. Unknown etiology. Affects infants from age 3 weeks. Uncommon after 6 months. Usually asymptomatic, 33% itchy. Scalp first area of involvement.

Greasy scale of seborrhoeic dermatitis on scalp

Diffuse greasy yellow or white scales – “cradle cap “. Hair loss not seen. Erythema variable. Facial involvement – hairline & eyebrow area. Scale is greasy, yellow overlying erythema.

### Complications of Seborrhoeic Dermatitis

Lesions may become macerated, crusted and superinfected with *Candida albicans*. Lesions often confined to scalp & nappy area. They may spread to involve flexural creases of the axillae, retro-auricular areas & neck. Intertriginous areas involved with scaling and linear erythema.

Rx: Bathing in soothing oatmeal/maizena baths

Tar shampoo daily

Hydrocortisone cream 1% 2-3x/day to affected areas

**Background:** 09/13/05 Selden article current for 2005. AB

Seborrhoeic dermatitis is a papulosquamous disorder patterned on the sebum-rich areas of the scalp, face, and trunk. In addition to sebum, this dermatitis is linked to *Malassezia*, immunologic abnormalities, and activation of complement. It is commonly aggravated by changes in humidity, changes in seasons, trauma (eg, scratching), or emotional stress. The severity varies from mild dandruff to exfoliative erythroderma. Seborrhoeic dermatitis may worsen in Parkinson disease and in AIDS.

**Pathophysiology:** Seborrhoeic dermatitis is associated with normal levels of *Malassezia* but an abnormal immune response. Helper T cells, phytohemagglutinin and concanavalin stimulation, and antibody titers are depressed compared with those of control subjects. The contribution of *Malassezia* may come from its lipase activity—releasing inflammatory free fatty acids—and from its ability to activate the alternative complement pathway.

**Frequency: Internationally:** The prevalence rate of seborrhoeic dermatitis is 3-5%, with a worldwide distribution. Dandruff, the mildest form of this dermatitis, is probably far more common and is present in an estimated 15-20% of the population.

**Race:** Seborrhoeic dermatitis occurs in persons of all races.

**Sex:** The condition is slightly worse in males than in females.

**Age:** The usual onset occurs with puberty. In infants, it occurs as cradle cap or, uncommonly, as a flexural eruption or erythroderma.

### History:

- Intermittent, active phases manifest with burning, scaling, and itching, alternating with inactive periods. Activity is increased in winter and early spring, with remissions commonly occurring in summer.

- Active phases may be complicated by secondary infection in the intertriginous areas and on the eyelids.
- Candidal overgrowth is common in infantile napkin dermatitis. Such children may have a diaper dermatitis variant of seborrhoeic dermatitis or psoriasis.
- Generalized seborrhoeic erythroderma is rare. It occurs more often in association with AIDS, congestive heart failure, Parkinson disease, and immunosuppression in premature infants.

### Physical:

- Scalp appearance varies from mild, patchy scaling to widespread, thick, adherent crusts. Plaques are rare. From the scalp, seborrhoeic dermatitis can spread onto the forehead, the posterior part of the neck, and the postauricular skin, as in psoriasis.
- Skin lesions manifest as greasy scaling over red, inflamed skin. Hypo-pigmentation is seen in blacks. Infectious eczematoid dermatitis, with oozing and crusting, suggests secondary infection. A seborrhoeic blepharitis may occur independently.
- Distribution follows the oily and hair-bearing areas of the head and the neck, such as the scalp, the forehead, the eyebrows, the lash line, the nasolabial folds, the beard, and the postauricular skin. An extension to submental skin can occur. Presternal or interscapular involvement is more common than nonscaling intertrigo of the umbilicus, axillae, inframammary and inguinal folds, perineum, or anogenital crease, which also may be present.
- Two distinct truncal patterns can occasionally occur. An annular or geographic petaloid scaling is the most common. A rare pityriasisiform variety can be seen on the trunk and the neck, with peripheral scaling around ovoid patches, mimicking pityriasis rosea.

### Causes:

- *Malassezia* organisms are probably not the cause but are a cofactor linked to a T-cell depression, increased sebum levels, and an activation of the alternative complement pathway.
- Because seborrhoeic dermatitis is uncommon in preadolescent children, and tinea capitis is uncommon after adolescence, dandruff in a child is more likely to represent a fungal infection. A fungal culture should be completed for confirmation.
- Various medications may flare or induce seborrhoeic dermatitis. These medications include auranofin, aurothioglucose, buspirone, chlorpromazine, cimetidine, ethionamide, gold, griseofulvin, haloperidol, interferon alfa, lithium, methoxsalen, methyl dopa, phenothiazines, psoralens, stanozolol, thiothixene, and trioxsalen.

**Lab Studies:** A clinical diagnosis of seborrhoeic dermatitis is usually made based on a history of waxing and waning severity and by the distribution of involvement upon examination.

**Procedures:** A skin biopsy may be needed in persons with exfoliative erythroderma, and a fungal culture can be used to rule out tinea capitis.

**Histologic Findings:** Dermatopathologic findings of seborrhoeic dermatitis are nonspecific. Hyperkeratosis, acanthosis, accentuated rete ridges, focal spongiosis, and parakeratosis are characteristic. Psoriasis is distinguished by regular acanthosis, thinned rete ridges, exocytosis, parakeratosis, and an absence of spongiosis. Neutrophils may be seen in both diseases.

**Medical Care:** Early treatment of flares is encouraged. Behavior modification techniques in reducing excoriations are especially helpful with scalp involvement.

- Topical corticosteroids may hasten recurrences, may foster dependence because of a rebound effect, and are discouraged except for short-term use. Skin involvement responds to ketoconazole, naftifine, or ciclopirox creams and gels. Alternatives include calcineurin inhibitors (ie, pimecrolimus, tacrolimus), sulfur or sulfonamide combinations, or propylene glycol. Class IV or lower corticosteroid creams, lotions, or solutions can be used for acute flares. Systemic ketoconazole or fluconazole may help if seborrhoeic dermatitis is severe or unresponsive.
- Dandruff responds to more frequent shampooing or a longer period of lathering. Use of hair spray or hair pomades should be stopped. Shampoos containing salicylic acid, tar, selenium, sulfur, or zinc are effective and may be used in an alternating schedule. Overnight occlusion of tar, bath oil, or Baker's P&S solution may help to soften thick scalp plaques. Derma-Smoother F/S oil is especially helpful when widespread scalp plaques are present. Selenium sulfide (2.5%), ketoconazole, and ciclopirox shampoos may help by reducing *Malassezia* yeast scalp reservoirs. Shampoos may be used on truncal lesions or in beards but may cause inflammation in the intertriginous or facial areas.
- Seborrhoeic blepharitis may respond to gentle cleaning of eyelashes with baby shampoo and cotton applicators. The use of ketoconazole cream in this anatomical region is controversial.

The goals of **pharmacotherapy** are to reduce morbidity and to prevent complications.

**Antifungals** -- Mechanism of action may involve alteration of RNA and DNA metabolism or an intracellular accumulation of peroxide that is toxic to fungal cells.

**Ketoconazole creams and shampoos** (Nizoral) -- Imidazole broad-spectrum antifungal agent. Inhibits synthesis of ergosterol, causing cellular components to leak, resulting in fungal cell death.

**Corticosteroids** -- Have anti-inflammatory properties and cause profound and varied metabolic effects. Also modify body's immune response to diverse stimuli.

**Clobetasol** (Temovate, Olux foam, Clobex shampoo) -- Class I superpotent topical steroid; suppresses mitosis and increases synthesis of proteins that decrease inflammation and cause vasoconstriction. Can be used in acute flares.

**Betamethasone** (Diprolene, Betatrex) -- For inflammatory dermatosis responsive to steroids. Decreases inflammation by suppressing migration of polymorphonuclear leukocytes and reversing capillary permeability. **Keratolytics** -- Cause cornified epithelium to swell, soften, macerate, and then desquamate.

**Salicylic acid (Kerasol)** -- By dissolving intercellular cement substance, produces desquamation of horny layer of skin while not affecting structure of viable epidermis.

Coal tar (DHS Tar, MG217, Theraplex T, Psoriasis) -- Inhibits deregulated epidermal proliferation and dermal infiltration; antipruritic and antibacterial. **Urea** (Carmol UltraMide) -- Keratolytic and antimycotic. Available in 10-50% strength lotion, gels, and creams. **Immunosuppressants** -- Exert anti-inflammatory affect by inhibiting T-lymphocyte activation. Safer than topical steroids for prolonged use or in skin folds. **Tacrolimus** (Protopic) ointment 0.03% and 0.1% -- Nonsteroidal anti-inflammatory agent. Should not cause steroid-type skin atrophy. Currently indicated only for atopic dermatitis in immunocompetent patients  $\geq 2$  y.

**Pimecrolimus** (Elidel cream 1%) -- Nonsteroidal anti-inflammatory agent. Should not cause steroid-type skin atrophy. Currently indicated only for atopic dermatitis in immunocompetent patients  $\geq 2$  y. Use cream sparingly to avoid maceration in skin folds. **Special Concerns:** A severe, explosive onset of seborrhoeic dermatitis may be a marker for HIV infection, regardless of age. It may appear as a butterfly rash, similar to the acute facial eruption associated with systemic lupus erythematosus. The dermatitis appears early in persons with AIDS, affects 25-50% of persons with AIDS, and has greater involvement and greater activity in those with diminished T-cell function. The dermatitis may be treated with topical preparations, but if severe, treatment with 400 mg of oral ketoconazole daily for 2 weeks may be necessary. Letterer-Siwe disease in infants may manifest as a scaling scalp and purpura.

#### **Irritant Nappy Dermatitis**

Some children constitutionally more susceptible

Risk factors: Diarrhoea, antibiotics, occlusion, maceration, candida & bacteria. Occlusion – wet over-hydrated skin susceptible to friction and damage. Commonly occurs 3-18 months of age. Elevations of pH of the nappy area due to faeces mixed with urine - activate fecal lipases & proteases with damage to the epidermis, loss of barrier function and increased susceptibility to irritation.

Erythema of convex surface of inner upper thigh area & buttock. Creases are spared. Deeply erythematous, glistening appearance & wrinkled surface.

Rx: Frequent nappy changes

Emollients - Zinc oxide, Petrolatum. If no improvement 1% hydrocortisone may be applied under the emollient.

#### *Complications of Nappy Dermatitis*

Secondary bacterial infection, *S. aureus*

#### **Candidiasis**

Common, maybe due to frequent antibiotic use.

Clinical picture: Diffuse erythematous patch extending over the genitalia with peripheral scale and satellite red pustules. Small pink papules surmounted by a scale and coalescence in some areas. Involvement of the creases important diagnostic consideration

#### *Treatment*

Topical therapy - Nystatin, Ketoconazole, Clotrimazole 2-3x/day

Adding 1% hydrocortisone may promote more rapid healing (potent corticosteroids should be avoided)

Oral nystatin treatment did not affect the outcome more favourably than topical nystatin alone

Candida Nappy Dermatitis - Involvement of creases and a central red plaque with surrounding satellite papules. Small pink papules surmounted by scale, coalescent in some areas

#### **Contact Dermatitis**

Contact dermatitis is caused by irritation or allergic reaction.

Allergic CD seldom in children <10 yrs old. Children react most commonly to nickel and rubber. Adhesive tape reactions may occur at any age, particularly in hospitalised patients.

#### **Impetigo**

Round confluent superficial blisters which rupture early and form crusts. Caused by – Staph & Strep organisms. Rx Topical and systemic antibiotics

#### **Erysipelas**

Superficial form of cellulitis involving the dermis and upper subcutaneous tissue. Usually due to *S. pyogenes*.

#### **Staphylococcal Scalded Skin Syndrome**

**Background:** Staphylococcal scalded skin syndrome (SSSS) is a toxin-mediated type of exfoliative dermatitis. Toxin-mediated staphylococcal syndromes comprise a group of blistering skin diseases, ranging in severity from localized bullous impetigo to SSSS, in which superficial blistering and exfoliation follow widespread painful erythema.

**Pathophysiology:** The disorder is caused by toxigenic strains of *Staphylococcus aureus*, usually belonging to phage group 2 (types 3A, 3B, 3C, 55, or 71). Two exotoxins (ETs), epidermolytic toxin A (ET-A) and epidermolytic toxin B (ET-B), are responsible for the pathologic changes seen in SSSS. These toxins cause intraepidermal splitting through the granular layer by specific cleavage of desmoglein 1 (also the target protein in the autoimmune blistering dermatosis, pemphigus foliaceus), a desmosomal cadherin protein that mediates cell-to-cell adhesion of keratinocytes in the granular layer.

Specific targeting of desmoglein 1 by ETs allows *S aureus* to proliferate and spread beneath the barrier of the skin. Crystal structures and amino acid sequences indicate that these toxins act as serine proteases and cleave desmoglein 1 after glutamic acid residue 381 between extracellular domains 3 and 4. The ET-A and ET-B amino acid sequences are approximately 40% identical with each other.

Most recently, Amagai has found an additional ET family member, ET-D, by screening the genomes of *S aureus* isolated from patients with skin infections and demonstrated that ET-D specifically digested desmoglein 1. However, only ET-A and ET-B have been firmly linked to human SSSS. It has been suggested that ET-B is more frequently isolated than ET-A in children with SSSS. This link between ET-B and generalized SSSS might be due to increased virulence of ET-B or to more abundant ET-B release. Because ET-A is chromosome borne and ET-B is plasmid borne, multiple copies of the ET-B gene could possibly lead to higher ET-B production. However, levels of ET-A and ET-B are quite similar in vitro. The link between ET-B and generalized SSSS may be explained, at least in part, by lower levels of anti-ETB antibodies than anti-ETA antibodies in the general population. An asymptomatic adult carrier introduces the causative bacteria into the nursery. Asymptomatic nasal carriage of *S aureus* occurs in 20-40% of healthy individuals,

with the organism being isolated from the hands, the perineum, and the axillae in a smaller proportion of the general population.

**Frequency: In the US:** SSSS most commonly occurs in infants and in young children, and it tends to occur in outbreaks in neonatal nurseries or in day care nurseries. Large outbreaks of SSSS in neonatal nurseries have been described, but the occurrence of SSSS in adults as a nosocomial infection appears to be exceptional; epidemics have never been observed.

Epidemiologic data on strains of *S aureus* that produce ET are scarce. In a prospective clinical and bacteriologic study, 5.1% of 944 isolates of *S aureus* were identified as ET producers. SSSS in adults is an exceedingly rare disorder, with only 50 reported cases.

**Mortality/Morbidity:** Children generally do well and are not as ill as their dramatic eruptions might suggest. SSSS is usually associated with a trivial infective focus in the conjunctivae or the skin; however, severe infections, such as sepsis, do contribute to a low but appreciable fatality rate (4%).

- Morbidity in the occasional child who develops cellulitis, sepsis, and pneumonia can be significant.
- In children, the foci of staphylococcal infections are usually the nasopharynx or localized skin infections.

**Race:** Black children are less prone to SSSS than white children. **Sex:** A male-to-female predominance exists (2:1 in sporadic cases, 4:1 in epidemics).

**Age:** The disease most commonly affects children younger than 5 years, particularly neonates. A decreased ability to achieve renal clearance of toxins and a lack of specific immunity (antibody) to the toxins make neonates the group at highest risk.

#### **History:**

- SSSS originates from a focus of infection that may be a purulent conjunctivitis, otitis media, or occult nasopharyngeal infection.
- It usually begins with fever, irritability, and a generalized, faint, orange-red, macular erythema with cutaneous tenderness.
- Periorificial and flexural accentuation may be observed. A positive Nikolsky sign (slippage of the superficial layer of the epithelium on gentle pressure) can often be elicited at this stage.

#### **Physical:**

- Within 24-48 hours, the rash progresses from a scarlatiniform to a blistering eruption.
- Characteristic tissue paper-like wrinkling of the epidermis is followed by the appearance of large, flaccid bullae in the axillae, in the groin, and around the body orifices.
- Subsequent generalized involvement occurs elsewhere on the body, but infection spares the mucous membranes.
- As sheets of epidermis are shed, a moist erythematous base is revealed.
- Despite the dramatic clinical picture, the entire process usually subsides with superficial desquamation, and healing is usually complete within 5-7 days.

- Cultures obtained from intact bullae are usually sterile; this finding is consistent with hematogenous dissemination of a toxin produced at a distant focus of staphylococcal infection.

#### Lab Studies:

- The definitive diagnosis depends on culture and biopsy results. Examination of frozen sections of the lesions can easily confirm the diagnosis.
- Slide latex agglutination, double immunodiffusion, and enzyme-linked immunosorbent assay tests can identify the toxins responsible for SSSS.
- Perform culturing in all patients with suspected SSSS for identification and antibiotic sensitivities of the causative organism. *S aureus* may be cultured from the conjunctiva, nasopharynx, feces, or pyogenic foci on the skin. Blood cultures are almost always negative in children, but they may be positive in adults.
- Investigate the possibility of a staphylococcal carrier in the vicinity.

**Histologic Findings:** All forms of scalded skin syndrome are characterized by intraepidermal cleavage, with splitting that occurs beneath and within the stratum granulosum. The cleavage space may contain free-floating or partially attached acantholytic cells. The remainder of the epidermis appears unremarkable, and the dermis contains no inflammatory cells.

#### Medical Care:

Direct the therapy for scalded skin syndrome toward eradication of the staphylococcal focus of infection, which generally requires intravenous, penicillinase-resistant, antistaphylococcal antibiotics. The current treatment of choice is cloxacillin.

- Antibiotics, supportive care, and appropriate attention to fluid and electrolyte management because of disrupted epidermal barrier function usually ensure rapid recovery. Moist, denuded areas should be lubricated with a bland emollient to decrease pruritus and tenderness.

Recognizing the potential for epidemic scalded skin syndrome in neonatal care units is important.

- Identification of health care workers colonized or infected with toxigenic *S aureus* is an integral part of managing the problem.
- Apply control measures, including strict enforcement of chlorhexidine hand washing, administration of an oral antibiotic therapy for workers who are infected, and application of mupirocin ointment for eradication of persistent nasal carriage.

#### Medication

The goal of pharmacotherapy is to reduce morbidity and to prevent complications.

**Antibiotics** -- Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens .

**Further Inpatient Care:** Recent developments in the understanding of the exfoliative toxins should lead to new and improved diagnostic and therapeutic strategies, including the use of specific antitoxins to prevent exfoliation.

- A recent surge has occurred in reports of methicillin-resistant *S aureus* strains causing SSSS, often with a fatal outcome. These cases emphasize the need to develop alternative treatment strategies before multiple antibiotic resistance becomes a problem.

**Deterrence/Prevention:** Investigate the possibility of a staphylococcal carrier in the vicinity

**Complications:** Cellulitis, sepsis, and pneumonia are possible complications that may occur in children with SSSS.

**Prognosis:** The prognosis of the disease is good in children, and the mortality rate is low if they are treated.

#### Pityriasis Rosea

Acute self limited, papulo-squamous disorder. Rash is often preceded by a herald patch. Papules exhibit a collarette of scale.

#### Erythema Multiforme Simplex

Acute self-limiting vesicobullous disease with erythematous macules and papules which evolve into "target" lesions (diagnostic feature of EM). Associated with Herpes simplex infections in children. Lesions fade over a period of 1-2 weeks. Mucous membrane is frequently involved in herpes associated EM.

#### Stevens-Johnson Syndrome

Target lesions are atypical in SJS. Mucous membrane involvement is more extensive and severe.

Causative factors:

Antibiotics -Sulfonamides, Penicillin

Anticonvulsants - Phenytoin, Carbamazepine, Valproate

NSAIDs

Mycoplasma pneumoniae

#### Clinical features

Prodromal illness 1 - 2 weeks. Fever, cough, headache, malaise, arthralgia, myalgia and GI upset. Sudden onset of rash, tender erythematous eruption. Positive Nikolski's sign. Coalescence of lesions with flaccid bullous lesions. Involvement of conjunctiva and genital mucous membranes.

#### Toxic Epidermal Necrolysis

TEN and SJS are similar and differs only in severity.

Nikolski's sign demonstrated.

#### Erythema Nodosum

Panniculitis (Inflammation of the subcutaneous fat). Septal panniculitis without vasculitis. Abrupt onset of tender red subcutaneous nodules on extensor surfaces of lower legs. Peak age 10 – 14 yrs Associated with TB, Strepto-coccus, Yersinia, Histoplasma, sulfonamides & oral contraceptives.

#### Vascular reactions

Urticaria

Erythema multiforme

Stevens Johnson syndrome

Toxic epidermal necrolysis (TEN)

Henoch Schönlein purpura

#### Genetic skin conditions

Atopic Dermatitis

Psoriasis

Ichthyosis

Tuberous sclerosis

Neurofibromatosis

Epidermolysis bullosae \*

### **Connective tissue disorders**

SLE

Dermatomyositis

Juvenile idiopathic arthritis

### **Paediatric Dermatological Conditions – Infestations**

#### **Pediculosis Capitis**

Infestation of the scalp by the human head louse, *Pediculus capitis*

Pruritis is common in long term infestation, but first time infestation may produce no symptoms whatsoever

Investigating the pruritis reveals the nits adhering to the hair

The nits that are easily seen are usually empty shells of eggs that have hatched – whitish to sandy-coloured

Viable intact eggs are difficult to see due to their darker colour

The cap or operculum of the egg always faces away from the skin or scalp

Developing headlouse embryo 4 days old

Head lice prefer clean, healthy hosts

Not linked to poverty or poor living conditions

Commonly found in people with good hygiene & grooming habits

#### *Life Cycle*

1 : Adult female

2-4: egg & embryo development

5: Larva ready to expel itself

6-8: Three instar stages of nymphs

Significant problem throughout the world

High rates of infestation in developed as well as developing countries

Both in temperate & tropical regions

Head lice prevalence is rising in many developed countries

Female lice 20% larger than males

Males have brown bands traversing the abdomen

Infestation most common in children 3-11 yrs

▪ Related to head-to-head and body contact

▪ Sharing of objects to which lice cling

More common in girls than boys

Length of hair has not been shown to be a significant factor in host preference among head lice

Anecdotal evidence that head lice may prefer certain blood types. This may explain why within family or classroom, some children are more prone to infestation than others

#### *Rx*

- Mechanical removal of lice & nits with a nit comb, shampooing, blow-drying
- Most head lice products kill the adult lice but not the nits. Thus all topical treatments should be applied twice, 1 week apart

Shampoos

Gamma benzene hexachloride – Gambex

- High Rx failure rate due to tolerance and resistance

Permethrin –Lyclear, Nitagon

- Resistance also becoming a problem and treatment should also be applied twice

#### **Scabies**

Human scabies is caused by the release of toxic or antigenic secretions of the female mite *Sarcoptes scabiei* var *hominis*. Family Sarcoptidae, Class Arachnida  
Common worldwide

Earliest and most common symptom is itching, particularly at night

Most common sites

- Hands, palms, wrists, buttocks, feet, soles, neck skin folds

*Microscopic view of female scabies mite with ovum*

A female mite exudes a fluid that dissolves the skin surface, forming a well in which she sinks

In cool climates she forms a burrow of 0.5-5 mm where she remains for her life ( ± 30 days)

Females lay ± 3 eggs/day, requiring ± 4 days to hatch. Time from egg laying to adult mite is 10–14 days

Mites are not blood feeders, but are thought to feed on intercellular fluid

*The eggs of the scabies mite are enormous, about 1/3 of the body*

Scabies

Earliest physical sign – small 1-2mm papules. Scratching excoriations due to intense itching. Various degrees of crusting and scaling.

Chronic form – lichenified skin or 2-4mm granulomatous nodules

Hyperpigmentation from chronic irritation

Typical burrows often rare or absent in infants or in warm climates

Scabies lesions on the foot of an infant. In infants a nodular reaction is seen in intertriginous areas, but may be generalized. Body distribution of scabies in elderly and AIDS patients are similar to that seen in infants

#### **Scabies Systemic Manifestations**

- Tired & irritable
  - Fever & lymph-adenopathy if secondary infection present
  - Acute glomerulo-nephritis if infected with Group A Streptococcus
- Scabies lesions on the hand of an infant - erythema, nodules & crusting  
Burrows ink test on an infant's foot indicating a scabies mite burrow.

Seldom find burrows in tropical climates

#### **Scabies Complications**

Secondary bacterial infection is common

Organisms causing the infection are commonly

Streptococcus pyogenes

Staphylococcus aureus

Pyodermatous sequelae of scabies

Chronic nodular scabies with pyoderma and crusting

Secondary infection may cause crusted purulent sores (ecthyma)

#### **Scabies Therapy**

- Older children: Apply Lindane or Quellada lotion (Gamma Benzene hexachloride 1%) to the entire body below the neck. Application is left on for 8 hours and then washed off thoroughly.

- Infants and young children: Apply sulphur 2,5% ointment 3 times daily for 3 days. Tetmosol soap is used in institutions in addition to the definitive treatment. Benzyl benzoate may alternatively be applied for 24 hours. It is cheaper but burns.
- Permethrin with 70-80% of ovicidal activity has no reported adverse properties. Apply the cream rinse for 10 minutes and then rinse it off with water.

#### **Cutaneous Larva Migrans (Sandworm)**

A creeping eruption caused by *Ancylostoma braziliense* the dog and cat hookworm. The eggs of the hookworms are passed in the faeces of the dog or cat. They often defecate in sandboxes, parks or beaches. A larvae hatches that is capable of penetrating intact skin.

Larval migration cause intense pruritis as well as linear, erythaematous, raised, serpigenous tracts

- Because of the mode of infection, cutaneous larva migrans occurs in feet
- The larvae tunnels in the epidermis because it cannot penetrate the basal layer

Rx

- Topical thiabendazole 10-15% ointment apply 2x/d for 10 days
- Oral Albendazole 400 mg daily x 3 days
- Antibiotics for secondary infection

Secondary infection is common

#### **Fungal Infections**

- Major pathogens causing superficial fungal infections in children
- Dermatophytes (The term "Tinea" is used to identify dermatophyte infections)
  - Trichophyton
  - Microsporum
  - Epidermophyton
- Yeasts
  - Candida
  - Malassezia

#### **Tinea Capitis**

Tinea capitis (scalp ring-worm) is the most common fungal infection in children

*T. violaceum* is the most common cause of ringworm in black children

The lesions consist of patchy or diffuse areas of scaling & hair loss

**Background:** Tinea capitis is a disease caused by superficial fungal infection of the skin of the scalp, eyebrows, and eyelashes, with a propensity for attacking hair shafts and follicles. The disease is considered to be a form of superficial mycosis or dermatophytosis. Several synonyms are used, including ringworm of the scalp and tinea tonsurans. In the United States and other regions of the world, the incidence of tinea capitis is increasing.

Dermatophytosis includes several distinct clinical entities, depending on the anatomic site and etiologic agents involved. Clinically, the conditions include tinea capitis, tinea favosa (favus resulting from infection by *Trichophyton schoenleinii*), tinea corporis (ringworm of glabrous skin), tinea imbricata (ringworm resulting from infection by *Trichophyton concentricum*), tinea cruris (ringworm of the groin), tinea unguium or onychomycosis (ringworm of the nail), tinea pedis (ringworm of the feet), tinea barbae (ringworm of the beard), and tinea manuum (ringworm of the hand).

Clinical presentation of tinea capitis varies from a scaly noninflamed dermatosis resembling seborrheic dermatitis to an inflammatory disease with scaly erythematous lesions and hair loss or alopecia that may progress to severely inflamed deep abscesses termed kerion, with the potential for scarring and permanent alopecia. The type of disease elicited depends on interaction between the host and the etiologic agents.

The term tinea originally indicated larvae of insects that fed on clothes and books. Subsequently, it meant parasitic infestation of the skin. By the mid 16th century, the term was used to describe diseases of the hairy scalp. The term ringworm referred to skin diseases that assumed a ring form, including tinea. The causative agents of tinea infections of the beard and scalp were described first by Remak and Schönlein, then by Gruby, during the 1830s. Approximately 50 years later, in Sabouraud's dissertation, the endothrix type of tinea capitis infection was demonstrated, and it was known that multiple species of fungi cause the disease. Simple culture methods were described and treatment using x-ray epilation was reported in 1904. Effective treatment of tinea capitis by griseofulvin became available in the 1950s.

**Pathophysiology:** Tinea capitis is caused by fungi of species of genera *Trichophyton* and *Microsporum*. Tinea capitis is the most common pediatric dermatophyte infection worldwide. The age predilection is believed to result from the presence of *Pityrosporum orbiculare* (*Pityrosporum ovale*), which is part of normal flora, and from the fungistatic properties of fatty acids of short and medium chains in postpubertal sebum.

Causative agents of tinea capitis include keratinophilic fungi termed dermatophytes. These molds usually are present in nonliving cornified layers of skin and its appendages and sometimes are capable of invading the outermost layer of skin, stratum corneum, or other keratinized skin appendages derived from epidermis, such as hair and nails.

Dermatophytes cause a variety of clinical conditions. They are among the most common infectious agents of humans. Collectively, the group of diseases is termed dermatophytosis. From the site of inoculation, the fungal hyphae grow centrifugally in the stratum corneum. The fungus continues downward growth into the hair, invading keratin as it is formed. The zone of involvement extends upwards at the rate at which hair grows, and it is visible above the skin surface by days 12-14. Infected hairs are brittle, and by the third week, broken hairs are evident.

The infection continues (for 8-10 wk) to spread in the stratum corneum to involve other hairs, at which point, the infected area is approximately 3.5-7.0 cm in diameter. The spontaneous cure of naturally occurring infection at puberty is a familiar clinical observation; however, the precise mechanism is unclear.

Three types of in vivo hair invasion are recognized.

- Ectothrix invasion is characterized by the development of arthroconidia on the exterior of the hair shaft. The cuticle of the hair is destroyed, and infected hairs usually fluoresce a bright greenish-yellow color under a Wood lamp ultraviolet light. Common agents include *Microsporum canis*, *Microsporum gypseum*, *Trichophyton equinum*, and *Trichophyton verrucosum*.
- Endothrix hair invasion is characterized by the development of arthroconidia within the hair shaft only. The cuticle of the hair remains intact and infected hairs

do not fluoresce under a Wood lamp ultraviolet light. All endothrix-producing agents are anthropophilic (eg, *Trichophyton tonsurans*, *Trichophyton violaceum*).

- Favus, usually caused by *T schoenleinii*, produces favuslike crusts or scutula and corresponding hair loss.

**Frequency/Internationally:** Tinea capitis is widespread in some urban areas in North America, Central America, and South America. It is common in parts of Africa and India. In Southeast Asia, the rate of infection has been reported to have decreased dramatically from 14% (average of male and female children) to 1.2% in the last 50 years because of improved general sanitary conditions and personal hygiene. In northern Europe, the disease is sporadic.

**Mortality/Morbidity:** Classification and severity of tinea capitis depend on the site of formation of their arthroconidia.

- Ectothrix infection is defined as fragmentation of the mycelium into conidia around the hair shaft or just beneath the cuticle of the hair, with destruction of the cuticle. Inflammatory tinea related to exposure to a kitten or puppy usually is a fluorescent small spore ectothrix. Some mild ringworm or prepubertal tinea capitis infections are of the ectothrix type, also termed the gray-patch type (microsporosis). Some ectothrix infections involute during the normal course of disease without treatment. Depending on the extent of associated inflammation, lesions may heal with scarring.
- Endothrix infections are noted in which arthrospores are present within the hair shaft in both anagen and telogen phases, contributing to the chronicity of the infections. Endothrix infections tend to progress, become chronic, and may last into adult life. Lesions can be eradicated by systemic antifungal treatment. Since the organisms usually remain superficial, little potential for mortality exists. Disseminated systemic disease has been reported in patients who are severely immunocompromised.

**Sex:** Incidence of tinea capitis may vary by sex, depending on the causative fungal organism. In *Microsporum audouinii*-related tinea capitis, boys are affected much more commonly. The infection rate has been reported to be up to 5 times higher in boys than in girls; however, the reverse is true after puberty, possibly as a result of increased exposure to infected children by women and to hormonal factors. In infection by *M canis*, the ratio varies, and the infection rate usually is higher in male children. Girls and boys are affected equally by *Trichophyton* infections of the scalp, but in adults, women are infected more frequently than are men.

**Age:** Tinea capitis occurs primarily in children and occasionally in other age groups. It is seen most commonly in children younger than 10 years. Peak age range is in patients aged 3-7 years.

#### **History:**

- Infection begins as a small erythematous papule around a hair shaft on the scalp, eyebrows, or eyelashes.
- Within a few days, the red papule becomes paler and scaly, and the hairs appear discolored, lusterless, and brittle. They break off a few millimeters above the scalp skin surface.
- The lesion spreads, forming numerous papules in a typical ring form. Ring-formed lesions may coalesce with other infected areas.

- Pruritus usually is minimal but may be intense at times.
- Alopecia is common in infected areas.
- Inflammation may be mild or severe. Deep boggy red areas characterized by a severe acute inflammatory infiltrate with pustule formation are termed kerions or kerion celsi.
- Favus (also termed tinea favosa) is a severe form of tinea capitis.
  - Favus is a chronic infection and is caused most commonly by *T schoenleinii* and, occasionally, by *T violaceum* or *Microsporum gypsum*.
  - Scalp lesions are characterized by the presence of yellow cup-shaped crusts termed scutula, which surround the infected hair follicles.
  - Favus is seen predominantly in Africa, the Mediterranean, and the Middle East and, rarely, in North America and South America, usually in descendants of immigrants from endemic areas.
  - Favus usually is acquired early in life and has a tendency to cluster in families.
  - In favus, infected hairs appear yellow.

**Physical:** Pertinent physical findings are limited to the skin of scalp, eyebrows, and eyelashes.

- Primary skin lesions
  - Lesions begin as red papules with progression to grayish ring-formed patches containing perifollicular papules.
  - Pustules with inflamed crusts, exudate, matted infected hairs, and debris may be seen.
  - Black dot tinea capitis refers to an infection with fracture of the hair, leaving the infected dark stubs visible in the follicular orifices.
  - Kerion celsi may progress to a patchy or diffuse distribution and to severe hair loss with scarring alopecia (see Image 3).
- Id reaction: Dermatophyte idiosyncratic or id reactions are manifestations of the immune response to dermatophytosis.
  - Id reactions occur at a distant site, and the lesions are devoid of organisms.
  - Id reactions may be triggered by antifungal treatment.
  - The most common type of id reaction is an acute vesicular dermatitis of the hands and feet. The grouped vesicles are tense, pruritic, and sometimes painful. Id reactions are noted in patients with inflammatory ringworm of the feet, primarily resulting from infection by *Trichophyton mentagrophytes*. Similar lesions may occur on the trunk in tinea capitis.
  - Vesicular lesions may evolve into a scaly eczematoid reaction or a follicular papulovesicular eruption.
  - Other less common types of id reactions include annular erythema and erythema nodosum. These patients have a strong delayed-type hypersensitivity reaction to intradermal trichophytin.
- Distribution of lesions: Skin lesions appear on the scalp with extension to the eyebrows and/or eyelashes.
- Regional lymph nodes: Cervical lymphadenopathy may develop in patients with severe inflammation associated with kerion formation.

### Causes:

- Infection of the scalp by dermatophytes usually is the result of person-to-person transmission. The organism remains viable on combs, brushes, couches, and sheets for long periods. Certain species of dermatophytes are endemic only in particular parts of the world. Zoophilic fungal infections of the scalp are rare.
- In the United States, *T tonsurans* has replaced *M audouinii* and *M canis* as the most common cause of tinea capitis. *T tonsurans* also is the most common cause of the disease in Canada, Mexico, and Central America.
- Historically, *M audouinii* was the classic causative agent in Europe and America and *Microsporum ferrugineum* was most common in Asia. Currently, *M audouinii* and *M canis* remain prevalent in most parts of Europe, although *T violaceum* also is common in Romania, Italy, Portugal, Spain, and the former USSR, as well as in Yugoslavia. In Africa, *T violaceum*, *T schoenleinii*, and *M canis* commonly are isolated. *T violaceum* and *M canis* are prevalent agents in Asia. *T schoenleinii* is common in Iran and Turkey, while *M canis* is common in Israel. *Epidermophyton floccosum* and *T concentricum* do not invade scalp hair. *Trichophyton rubrum*, which is the most common dermatophyte isolated worldwide, is not a common cause of tinea capitis.
- Dermatophytic fungi causing tinea capitis can be divided into anthropophilic and zoophilic organisms. Anthropophilic fungi grow preferentially on humans, and the most common type forms large conidia of approximately 3-4 mm in diameter within the hair shaft. Zoophilic fungi are acquired through direct contact with infected animals. Smaller conidia of approximately 1-3 mm in diameter typically are present, extending around the exterior of the hair shaft.
- Dermatophytosis customarily is divided into endothrix (inside the hair shaft) and ectothrix (extending outside the hair shaft) infection based on the location of proliferation of pathogenic fungi and destruction of the hair structure.
- Common causes of endothrix infection include *T tonsurans*, characterized by chains of large spores and *T schoenleinii*, characterized by hyphae with air spaces. Infected hairs break off sharply at the follicular orifice, leaving a conidia-filled stub or black dot. Suppuration and kerion formation (see [Image 2](#)) commonly are associated with *T tonsurans* infection.
- In ectothrix infection, fragmentation of the mycelium into spores occurs just beneath the cuticle. In contrast to endothrix infection, destruction of the cuticle occurs. This type of infection is caused by *T verrucosum*, *T mentagrophytes*, and all *Microsporum* species.

### Lab Studies:

- Laboratory diagnosis of dermatophytosis depends on examination and culture of skin rubbings, skin or nail scrapings, hair pluckings (epilated hair), or nail clippings from lesions. Infected hairs appearing as broken stubs are best for examination. They can be removed with forceps without undue trauma or collected by gentle rubbing with a moist gauze pad or toothbrush.
  - Definitive diagnosis depends on an adequate amount of clinical material submitted for examination by direct microscopy and culture. The turn-around time for routine direct microscopy is 24 hours; however, culture may

take several weeks. Histopathology is faster than culture for the diagnosis of onychomycosis.

- In patients with suspected dermatophytosis of skin, any ointment or other local applications present should be removed with alcohol. Using the end of a glass slide or other implement, scale is scraped from the lesion. In cases of vesicular tinea pedis, the tops of any fresh vesicles should be removed because the fungus is often plentiful in the roof of the vesicle. In the case of tinea capitis, a moist gauze pad may be rubbed across the scalp. Broken, infected hairs adhere to the gauze. A toothbrush may be used in a similar fashion.
- Selected hair samples are cultured or allowed to soften in 10-20% potassium hydroxide (KOH) before examination under the microscope. Examination of KOH preparations (KOH mount) usually determines the proper diagnosis if a tinea infection exists.
- Microscopic examination of the infected hairs may provide immediate confirmation of the diagnosis of ringworm and establishes whether the fungus is small-spore or large-spore ectothrix or endothrix.
- Culture provides precise identification of the species for epidemiologic purposes. Primary isolation is carried out at room temperature, usually on Sabouraud agar containing antibiotics (penicillin/streptomycin or chloramphenicol) and cycloheximide (Acti-Dione), which is an antifungal agent that suppresses the growth of environmental contaminant fungi. In cases of tender kerion, the agar plate can be inoculated directly by pressing it gently against the lesion. Most dermatophytes can be identified within 2 weeks, although *T verrucosum* grows best at 37°C and may have formed only into small and granular colonies at this stage. Identification depends on gross colony and microscopic morphology. Specimens should be inoculated on to primary isolation media, such as Sabouraud dextrose, and incubated at 26-28°C for 4 weeks. The growth of any dermatophyte is significant.
- In some cases, other tests involving nutritional requirements and hair penetration in vitro are necessary to confirm the identification.
- Wood lamp examination: In 1925, Margarot and Deveze observed that infected hairs and some fungus cultures fluoresce in ultraviolet light. The black light commonly is termed Wood lamp. Light is filtered through a Wood nickel oxide glass (barium silicate with nickel oxide), which allows only the long ultraviolet rays to pass (peak at 365 nm).
  - Hairs infected by *M canis*, *M audouinii*, and *M ferrugineum* fluoresce a bright green to yellow-green color (see [Image 7](#)).
  - Hairs infected by *T schoenleinii* may show a dull green or blue-white color, and hyphae regress leaving spaces within the hair shaft.
  - *T verrucosum* exhibits a green fluorescence in cow hairs, but infected human hairs do not fluoresce.
  - The fluorescent substance appears to be produced by the fungus only in actively growing infected hairs.
  - Infected hairs remain fluorescent for many years after the arthroconidia have died.

- When a diagnosis of ringworm is under consideration, the scalp is examined under a Wood lamp. If fluorescent infected hairs are present, hairs are removed for light microscopic examination and culture. Infections caused by *Microsporum* species fluoresce a typical green color.
- Unfortunately, most tinea capitis infections in North America are caused by *T tonsurans* and do not demonstrate fluorescence.
- In favus, infected hairs appear yellow.

- Serology is not required for a diagnosis of dermatophytosis.

**Histologic Findings:** Only rarely is biopsy necessary. Skin biopsy with particular emphasis on examination of infected hairs with special histochemical stains aids in the identification of the causative fungus. Overall, the histologic picture is that of a subacute and chronic dermatitis with follicular inflammation and destruction. Suppurative folliculitis may be present. In the mildest form, hyperkeratosis, parakeratosis, spongiosis, slight vasodilatation, and a perivascular inflammatory infiltrate in the upper dermis are present. Fungal hyphae can be demonstrated using routine hematoxylin and eosin stain, and identification can be facilitated by using special stains. Periodic acid-Schiff stain with diastase digestion or counterstained with green dye facilitates identification of fungal elements.

Fungi are seen sparsely in the stratum corneum. Hyphae extend down the hair follicle, growing on the surface of the hair shaft. Hyphae then invade the hair, penetrate the outermost layer of hair (ie, cuticle), and proliferate downward in the subcuticular portion of the cortex, gradually penetrating deep into the hair cortex. Pronounced inflammatory tissue reaction with follicular pustule formation surrounding hair follicles is seen in patients with the clinical form of infection termed kerion celsi. In endothrix infection, spheric-to-box-like spores are found within the hair shaft. This type of infection is caused by *T tonsurans* or *T violaceum*.

In ectothrix infection, organisms form a sheath around the hair shaft. In contrast to endothrix infection, destruction of the cuticle by hyphae and spores occurs.

**Medical Care:** Choice of treatment is determined by the species of fungus concerned, the degree of inflammation, and in some cases, by the immunologic and nutritional status of the patient.

- Systemic administration of griseofulvin provided the first effective oral therapy for tinea capitis.
- Topical treatment usually is ineffective.
- Newer antifungal medications, such as ketoconazole, itraconazole, terbinafine, and fluconazole, have been reported as effective alternative therapeutic agents for tinea capitis. Of these agents, itraconazole and terbinafine are used most commonly.
- Selenium sulfide shampoo may reduce the risk of spreading the infection early in the course of therapy by reducing the number of viable spores that are shed.

#### Medication

Griseofulvin has been the treatment of choice in all ringworm infections of the scalp. The effective dosage of griseofulvin often prescribed by specialists is 20-25 mg/kg/d for 6-8 weeks. Griseofulvin accumulates in keratin of the horny layer, hair, and nails, rendering them resistant to invasion by the fungus. Treatment must continue long enough for infected keratin to be replaced by resistant keratin, usually 4-6 weeks. In

inflammatory lesions, compresses often are required to remove pus and infected scale. Therapy progress is monitored by regular clinical examination with the aid of a Wood lamp for fluorescent species such as *M audouinii* and *M canis*.

Several newer antimycotic agents, including itraconazole, terbinafine, and fluconazole, have been reported as effective and safe. Gupta et al reported the following alternative effective and safe treatment regimens for tinea capitis with endothrix species infection including *T tonsurans*: itraconazole continuous regimen (3-5 mg/kg/d with a full meal for 4-6 wk), itraconazole pulse regimen with capsules (5 mg/kg/d for 1 wk times 3 pulses 3 wk apart), and itraconazole pulse regimen with oral solution (3 mg/kg/d for 1 wk times 3 pulses, ie, 1 wk per mo). The oral solution contains cyclodextrin, which may cause diarrhea in children. Pharmacokinetics of the liquid formulation are not well established in children. In some children (weighing 20-40 kg), a single 100-mg capsule daily for 4-6 weeks has been used successfully. Because itraconazole has been associated with heart failure, it is currently not favored as a first-line therapy for tinea. An exception may be serious *M canis* infections, which are relatively insensitive to terbinafine.

Terbinafine tablets at doses of 3-6 mg/kg/d for approximately 2-4 weeks have been used successfully for *T tonsurans* infections. *M canis* is relatively resistant to this drug but has been treated effectively with longer courses of therapy. General guidelines for tinea capitis by weight include more than 40-kg body weight, 250 mg/d; 20-40 kg, 125 mg/d; and 10-20 kg, 62.5 mg/d for 2-4 weeks.

Fluconazole tablets or oral suspension (3-6 mg/kg/d) are administered for 6 weeks. In 1 trial, a dose of 6 mg/kg/d for 20 days was effective. An extra week of therapy (6 mg/kg/d) can be administered if clinically indicated at that time.

In ectothrix infection (eg, *M audouinii*, *M canis*), a longer duration of therapy may be required.

Although oral ketoconazole also is an acceptable alternative to griseofulvin, it is not considered a treatment of choice because of the risk of hepatotoxic effect and higher cost. Treatment for the deep folliculitis seen in Majocchi granuloma is systemic oral antifungal therapy.

Oral steroids may help reduce the risk for and extent of permanent alopecia in the treatment of kerion. Avoid using topical corticosteroids during treatment of dermatophyte infections.

Drug Category: *Antifungal agents* -- Mechanism of action may involve an alteration of RNA and DNA metabolism or an intracellular accumulation of peroxide that is toxic to the fungal cell.

#### Further Outpatient Care:

- Asymptomatic carriers who demonstrate neither signs nor symptoms of skin infection, including adults and siblings in the family of patients with tinea capitis and patient caretakers and playmates, require active treatment, since they may act as a continuing source of infection.
  - Shampoo and oral antimycotic therapy have been advocated for eradication of the carrier state.
  - Studies have shown that most children who received griseofulvin plus biweekly shampooing with 2.5% selenium sulfide were negative for fungi on scalp culture after 2 weeks.

- Shampoo containing povidone-iodine has been shown to be more effective in producing negative cultures than shampoos containing econazole and selenium sulfide and than Johnson's Baby Shampoo. Therapeutic shampoos are applied twice weekly for 15 minutes for 4 consecutive weeks. Both povidone-iodine and selenium shampoos require further clinical study for the control of fungal spore loads in infected children and asymptomatic carriers.
- Classrooms with young children (ie, kindergarten through second grade) must be evaluated for tinea capitis infection, since these children are most susceptible and have a greater risk of disease transmission.
- Playmates in close physical contact with patients can spread tinea capitis organisms by sharing toys or personal objects including combs and hairbrushes. These individuals need to be evaluated for the presence of infection.

**Deterrence/Prevention:**

Asymptomatic carriers should be detected and treated, since they are the continuous source of infection. Siblings and playmates of patients should avoid close physical contact and sharing of toys or other personal objects, such as combs and hairbrushes, since organisms can spread from one person to another and infectious agents can be transported to different classrooms within the same or in different schools. Shared facilities and objects also may promote spread of disease, both within the home and the classroom.

**Complications:** The causative fungal organisms of tinea capitis destroy hair and pilosebaceous structures, resulting in severe hair loss and scarring alopecia. Since tinea capitis is the most common dermatophyte infection in the pediatric population in the United States, without accurate diagnosis and proper treatment, the disease is detrimental, both physically and mentally, to children who are affected. Young patients with itchy scalp and patchy or total hair loss frequently are ridiculed, isolated, and bullied by classmates or playmates. In some cases, the disease can cause severe emotional impairment in vulnerable children and can destabilize family relationships

**Prognosis:** Continuous shedding of fungal spores may last several months despite active treatment; therefore, keeping patients with tinea capitis out of school is impractical

**Patient Education:** Patient education is paramount in eradicating tinea capitis. The current recommendations of the Committee on Infectious Diseases of the American Academy of Pediatrics state that "Children receiving treatment for tinea capitis may attend school. Haircuts, shaving of the head, wearing a cap during treatment are not necessary

**Medical/Legal Pitfalls:**

- Failure to recognize the characteristic skin lesions on the scalp, identify the causative fungal organisms on KOH preparation (with or without a Wood fluorescent lamp), and perform biopsy examination of the affected skin (with or without fungal culture) may result in destruction of hair and pilosebaceous structures with severe hair loss and scarring alopecia. Since tinea capitis is the most common dermatophyte infection in the pediatric population in the United States, lack of accurate diagnosis and proper treatment may result in serious

cosmetic impairment in young patients who are affected. The tragic detrimental effects on them are both physical and psychological. Young victims with scalp pruritus and patchy or total hair loss frequently are ridiculed and bullied by classmates or playmates. In some cases, the disease can cause emotional devastation in vulnerable uncoached children.

- Failure to identify and institute therapy in dermatophyte carriers, eg, caretakers, parents, siblings, and playmates, may hinder remedy of the disease further.

**Special Concerns:**

Public health measures regarding the source of infection should be a concern for controlling tinea capitis. As many as 14% of asymptomatic children have been found to be carriers of causative dermatophyte for tinea capitis in a primary school in Philadelphia (Williams et al). Without therapy, 4% developed symptoms of infection, 58% remained culture positive, and 38% became culture negative within an average 2.3-month follow-up period.

**Tinea corporis**

Active raised scaly margins which spread outwards

*Treatment*

Topical preparations

- Imidazoles  
Treats tinea corporis

Oral preparations

- Griseofulvin  
Specific for dermatophyte infections. Treats Tinea capitis

Tinea corporis involving the face

**Pityriasis Versicolor**

- Caused by malassezia yeast, children often have facial lesions
- Lesions on the face are usually hypopigmented, faintly scaling & ovoid
- Rx – ketoconazole shampoo or fluconazole 400mg

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