

CHILDHOOD ALOPECIA AND COMMON PEDIATRIC RASHES

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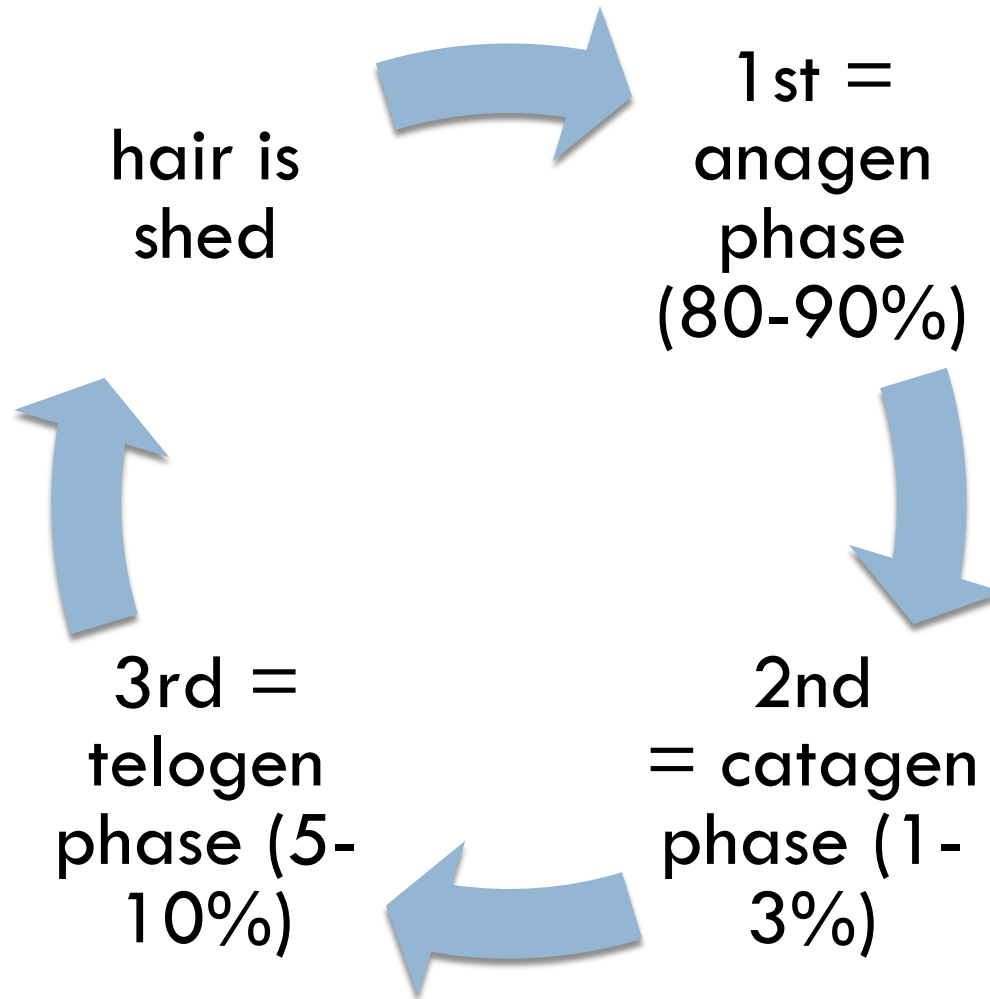
Childhood Alopecia



TINEA CAPITIS,
TRICHOTILLOMANIA,
ALOPECIA AREATA, AND
TELOGEN EFFLUVIUM
ACCOUNT FOR >95% OF
CASES OF ALOPECIA IN
CHILDREN.



Normal Hair Cycle:



What is normal hair loss?

- Normal hair loss averages 75 to 100 hairs per day.
- Hair loss is clinically apparent when a person has lost 25%-50% of hair.

Evaluation of Alopecia:

History

time of onset, associated stressors, unusual behaviors/habits, medications/exposures, ROS



Physical

Physical: weight/general appearance, distribution of hair loss, presence of scale, presence of broken hairs, nail findings, teeth abnormalities, rashes



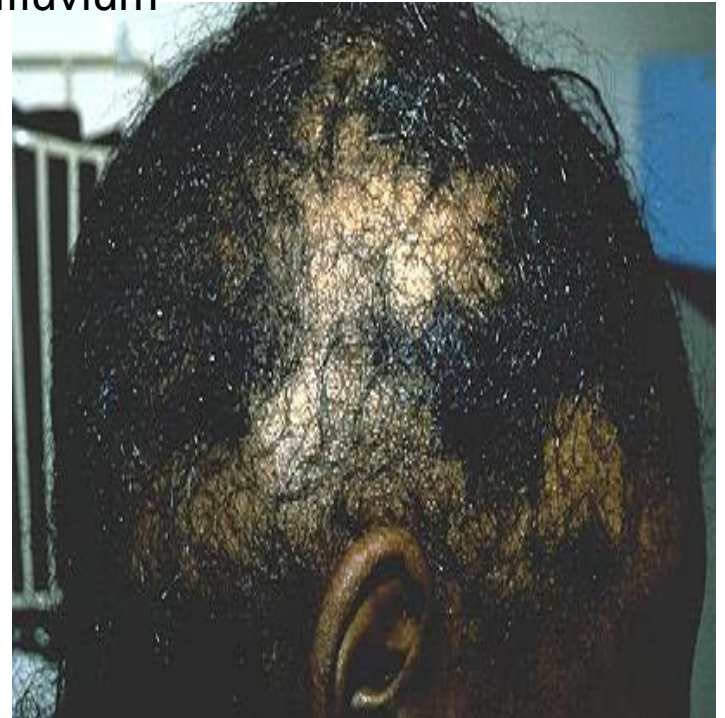
Labs

KOH prep, fungal culture, hair pluck/pull test, morphological exam of hair shaft

Differential Diagnosis

- **Toxic** - cytotoxic agents, radiation, anticonvulsants, hypervitaminosis A, anticoagulants
- **Neoplastic** - histiocytosis
- **Traumatic** - trichotillomania, traction alopecia, friction alopecia
- **Infectious** - tinea capitis, secondary syphilis
- **Congenital** - aplasia cutis congenita, nevus sebaceous, epidermal nevus, hemangioma, loose anagen syndrome, ectodermal dysplasia, hair shaft defects
- **Metabolic or Genetic Causes** - androgenic alopecia, acrodermatitis enteropathica, anorexia nervosa, malnutrition, thyroid disease, hypopituitarism, DM

- **Inflammatory** - alopecia areata, SLE, scleroderma
- **Misc** - atopic dermatitis, seborrheic dermatitis, psoriasis, telogen effluvium, anagen effluvium



The Alopecias

Non-Scarring

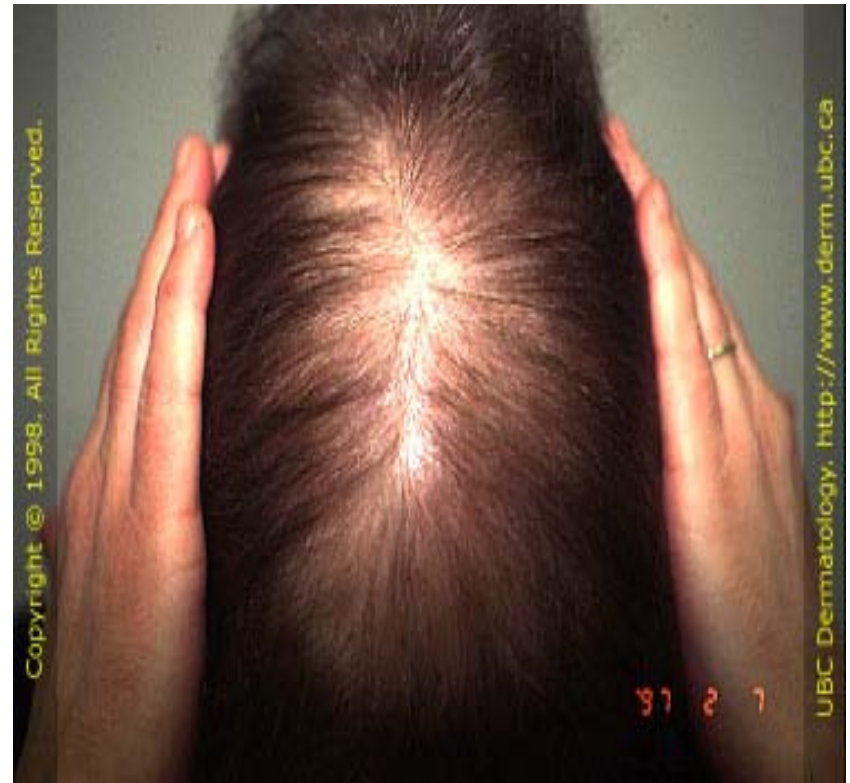
- Alopecia Caused by Systemic Insult:
 - ▣ **Telogen Effluvium**
 - ▣ Anagen Effluvium
 - ▣ **Alopecia Areata**
- Trauma-Induced Alopecia:
 - ▣ Trichorrhexis Nodosa
 - ▣ Friction Alopecia
 - ▣ Traction Alopecia
 - ▣ **Trichotillomania**

Scarring

- Aplasia Cutis Congenita
- **Tinea Capitis**

Telogen Effluvium

- the most common cause of diffuse hair loss
- partial, temporary alopecia that is seen a few months after a severe illness, major surgery, or high fever
- the initial systemic insult induces more than the usual 20% of hairs to enter the telogen phase, and 3 months later these hairs are shed simultaneously
- spontaneously resolves over several months



Anagen Effluvium

- sudden loss of the growing hairs (80% of normal scalp hairs)
- caused by abnormal cessation of anagen phase
- hair shafts taper and lose adhesion to the follicle
- most common after systemic chemotherapy

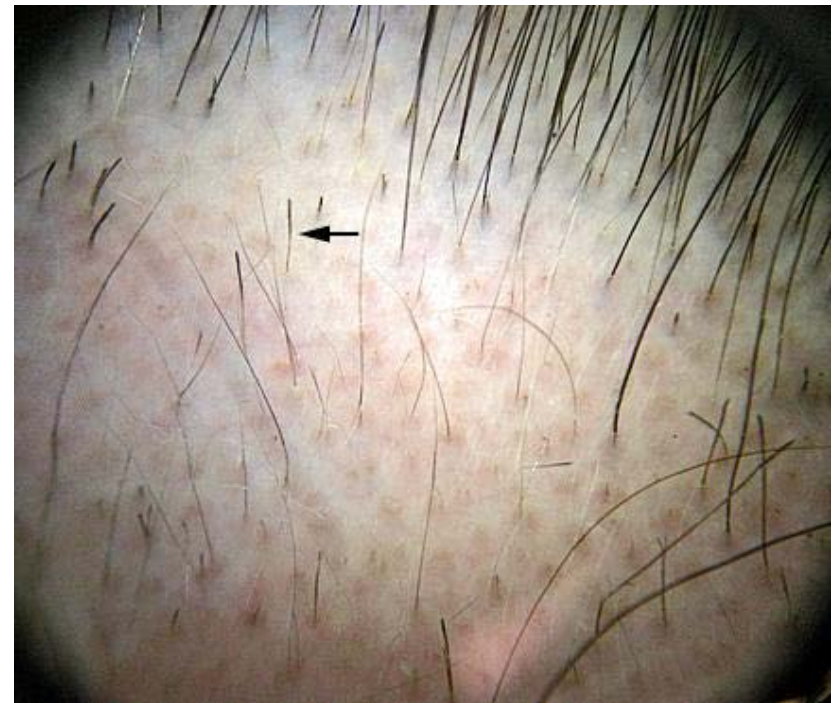
Alopecia Areata

- second most common cause of alopecia in children
- form of localized anagen effluvium
- round smooth patches of alopecia that can be located anywhere
- cause thought to be multifactorial:
immunologic, genetic, environmental



Alopecia Areata

- clues to diagnosis
 - ▣ absence of inflammation and scaling in involved areas
 - ▣ presence of short 3-6mm easily epilated hairs at the margins of the patch
 - ▣ Scotch-plaid pitting of the nails
- * Biopsy is usually not necessary to confirm the diagnosis, but may be needed in cases where the diagnosis is uncertain



Alopecia Areata

- 1/3 regress spontaneously within 6 months
- almost all will experience more than one episode of the disease
- can progress to alopecia totalis
- can progress to alopecia universalis
- eye abnormalities may occur
- poor prognosis = young age, severe disease, duration of >1 year, nail disease, atopy, involvement of peripheral scalp



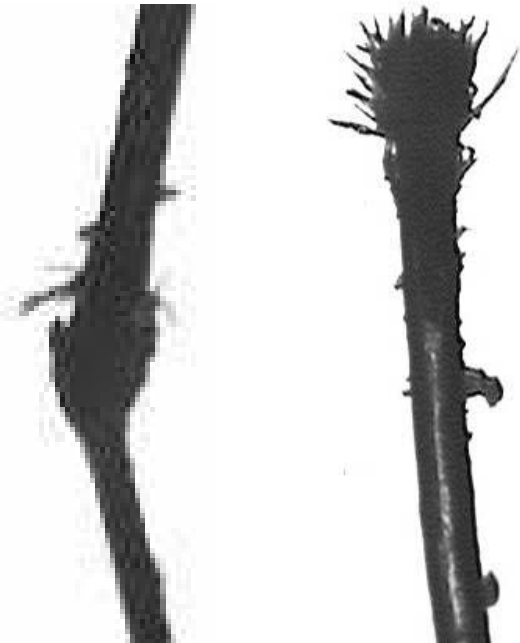
Alopecia Areata

- not all patients require treatment
- up to 80 percent of patients with alopecia areata that is limited and of less than one year's duration may expect spontaneous re-growth of hair
- intralesional/topical/systemic steroids
- minoxidil
- anthralin
- methotrexate
- topical immunotherapy



Trichorrhhexis Nodosa

- alopecia caused by hair shaft breakage due to damage to outer cortex of hair shaft and loss in structural support
- usually caused by physical trauma or chemical trauma
- diagnosed under microscope: distal ends of hairs are frayed like a broom or hairs may have nodules like two brooms stuck together
- presents at any age as brittle, short hairs that are perceived as non-growing, hairs are easily broken on gentle pull
- self-limited process
- hair re-grows when the source of the damage is eliminated



Friction Alopecia

- common on posterior scalp of infants where head rubs on pillow
- self limited
- when severe/long standing, think neglect



Traction Alopecia

- ❑ common in young girls whose hairstyles maintain a tight pull on hair shafts
- ❑ causes shaft fractures and follicular damage
- ❑ can cause permanent scarring alopecia if prolonged



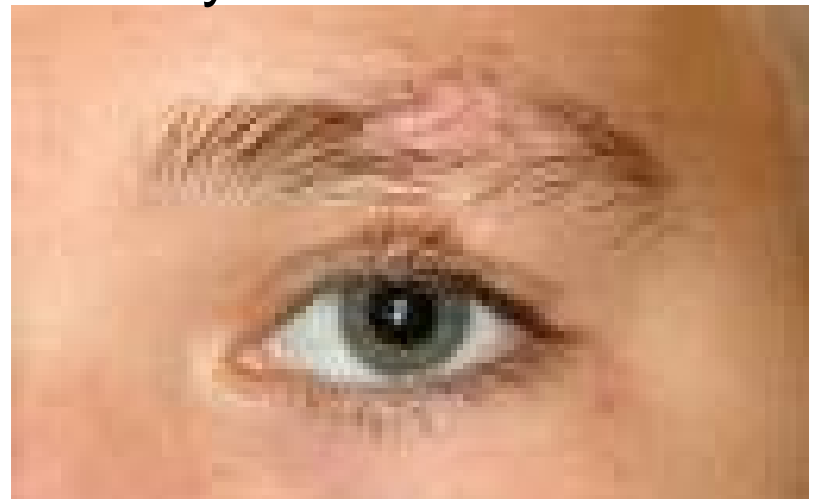
Trichotillomania

- uncontrollable urge to pull out ones own hair
- seen in school aged children and adolescents, mostly in adolescent females, but more common in boys under 6 y/o
- often associated with other compulsive behaviors
- bizarre patterns of hair loss
- rarely the scalp, eyebrows, and eyelashes are involved



Trichotillomania

- diagnosis: hair pluck, scalp biopsy
- diagnostic clues: short, broken-off hairs along the scalp with stubs of different lengths
- differentiating from alopecia areata: patches of hair loss, hair shafts are anagen hairs that are difficult to remove, no nail abnormalities
- should be distinguished from habitual hair pulling, twisting, twirling, which usually occur at bedtimes/naptimes, and habit resolves by early school years



Trichotillomania

- can occur in those with severe psychiatric disease
- most cases are associated with situational stress
- treatment = referral to psychiatry, behavior modification +/- clomipramine or fluoxetine
- prognosis = initially reversible but may become permanent if the habit persists

Aplasia Cutis Congenita

- congenital condition with absence or failure of formation of a localized area of scalp or skin
- rarely, lesions may be multiple or may involve the trunk or extremities, and may be associated with limb defects or other anomalies
- majority involve only the dermis and epidermis



Aplasia Cutis Congenita

- at birth, lesion consists of sharply circumscribed open weeping ulceration, or may be covered by thin hemorrhagic membrane or crust
- conservative treatment to prevent infection and injury
- healing occurs over weeks to months, leaving smooth atrophic and hairless scar



Tinea Capitis

- responsible for >50% of cases of hair loss in children
 - fungal infection weakens hair shaft causing breakage and results in multiple patches of partial alopecia
 - *Trichophyton tonsurans* is responsible for over 95% of scalp ringworm in US
 - unknown reasons, but infection is endemic among black school children
- *Microsporum canis* (dog/cat ringworm) can cause a few cases also, but there is no racial predilection



Tinea Capitis

Variable presentations -

- mild erythema and scaling of scalp with partial alopecia
- widespread breakage at the scalp creating a salt and pepper appearance
- annular like tinea corporis
- erythema/edema/pustule formation, as the pustule ruptures the area weeps and golden crusts form like impetigo
- heaped up scale
- less common, kerions = intense inflammation causes formation of raised tender boggy plaques or masses studded with pustules that simulate abscesses



Tinea Capitis

- ❑ dx with KOH examination of infected hairs
- ❑ fungal culture of hair and scale
- ❑ woods lamp = *M. audouinii* and *M. canis* fluoresce, but not *T. tonsurans*



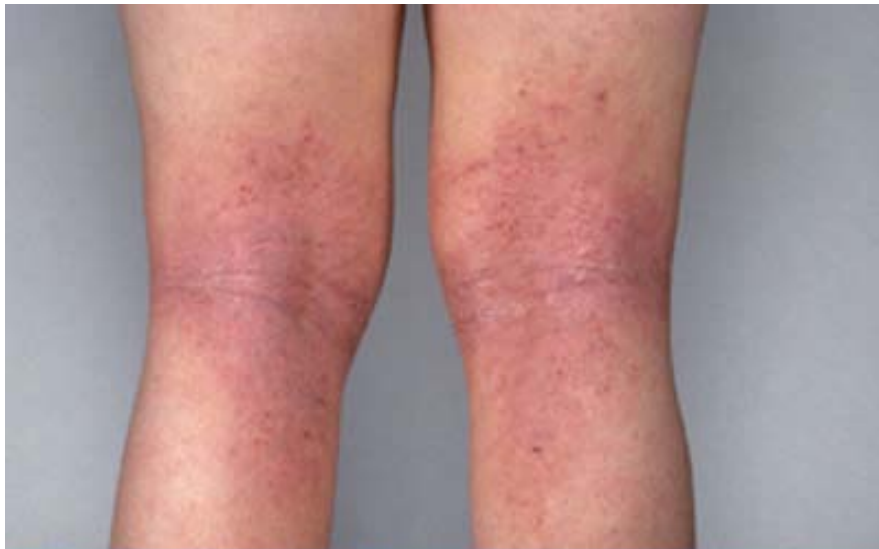
Tinea Capitis

- Trt with oral antifungals
- griseofulvin 20mg/kg once daily x 6-8 weeks
- Ketoconazole alternative
- Newer antifungals: terbinafine, itraconazole, fluconazole
- concurrent use of selenium sulfide shampoo (2.5%) reduces spore formation and shedding, which can help minimize spread
- recurrence is high





Common Pediatric Rashes



Atopic Dermatitis

- also known as eczema
- chronically recurrent, genetically influenced skin disorder
- prevalence is highest among children
- in families with a history of allergic rhinitis or asthma, ~1/3 of the children are expected to develop atopic dermatitis
- in patients with atopic dermatitis, 1/3 are expected to have a personal history of allergic rhinitis or asthma
- inherited as an autosomal trait with multifactorial influences:
 - weather - atopic dermatitis improves with warm and humid weather, worsens with cold and dry weather
 - other external factors - dry skin, soaps, wool fabrics, foods, infectious agents produce pruritus in susceptible patients

Atopic Dermatitis



the scratching leads to acute and chronic changes:

- acutely -> erythema, scaling, vesicles, crusting
- chronically -> lichenification and pigmentary changes

Atopic Dermatitis

- distribution of the rash changes with age:
 - infantile phase (birth - 3 years) – symmetrically distributed over scalp, forehead, cheeks, trunk, and extensor surfaces; spares diaper area
 - childhood phase (4 - 10 years) – distributed over wrists, ankles, flexural surfaces of the extremities, ear creases, back of neck
 - adolescent/adult phase – distributed over flexural creases of the neck and extremities, hands and feet

Atopic Dermatitis

management:

- avoid environmental irritants
- avoid scratching with:
 - loose-fitting cotton clothing; long sleeves and foot coverings may help in infants
 - antihistamines, especially at bedtime
 - emollients to prevent dry skin, liberal application at least BID
 - keep nails trimmed to prevent excoriations

Atopic Dermatitis

- for increased disease activity:
 - low and medium –potency topical corticosteroids, BID application to worst areas and tapered ASAP, overuse – causes atrophy, loss of pigment, telangiectasias, striae
 - face/groin – HC1% and 2.5%, desonide if severe
 - body – triamcinolone 0.1%, use only on thick plaques for kids <1yr
 - topical nonsteroidal calcineurin inhibitors - tacrolimus and pimecrolimus

Atopic Dermatitis

- types of atopic dermatitis:
 - nummular eczema – coin shaped, red patches made up of tiny papules and vesicles located on extremities; difficult to treat
 - follicular eczema – follicular papules on trunk and extremities, usually occurs early in flares



Atopic Dermatitis

- complications:
 - secondary bacterial infection
 - crusted exudative patches
 - usually caused by GAS or *S. aureus*
 - culture and treat with oral antibiotics, warm compresses, and emollients
 - topical mupirocin or bacitracin for localized, small, impetigo-like lesions
 - IV if failed oral therapy or widespread infection
 - eczema herpeticum
 - multiple grouped 2-3mm diameter vesicles or crusts/ulcerations associated with high fever and worsening pruritis
 - dx with viral culture, PCR, or DFA
 - admit and start IV Acyclovir immediately if suspected
 - an infection unresponsive to antibiotics should raise suspicion for eczema herpeticum





Keratosis Pilaris

- results from retention of keratin in the follicular infundibulum
- benign skin condition, but cosmetically displeasing
- often + FH, AD inheritance with variable penetrance
- females more frequently affected than males
- often improves with age, but usually never goes away
- manifests as horny follicular papules and erythema on the upper arms, medial thighs, and cheeks
- commonly associated with atopic dermatitis, ichthyosis vulgaris, xerosis
- moisturize with emollients
- try combination of emollient and exfoliant



Contact Dermatitis



- group of conditions in which an inflammatory reaction in the skin is triggered by direct contact with environmental agents

Contact Dermatitis

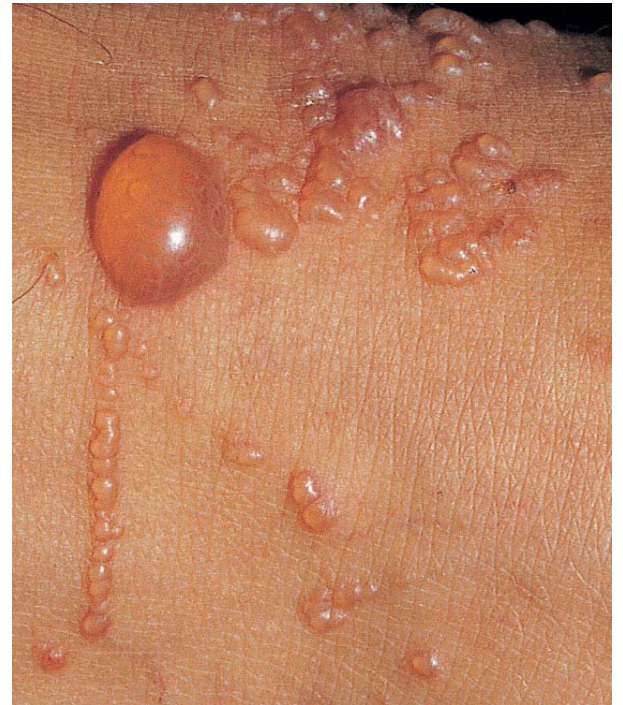
- irritant vs. allergic forms:
 - irritant is the most common form; changes in the skin induced by caustic agents (i.e. acids, alkali, hydrocarbons, etc.)
 - rash is usually occurs within minutes: well-demarcated erythema, blistering, edema, and/or crust formation
 - itching/burning sensation
 - allergic contact dermatitis is a Type IV delayed-hypersensitivity response
 - allergic response is less severe and often delayed upon initial exposure, then more rapid and severe responses occur on subsequent exposure to the allergen
 - most common allergic contact dermatitis in the US is poison ivy or rhus dermatitis



Poison Oak



Poison Ivy



Contact Dermatitis

- poison ivy, oak, and sumac dermatitis causes a rash consisting of linear streaks of erythematous papules and vesicles
- when involved in more sensitive areas such as the face or genitals, impressive swelling can occur
- thorough washing within minutes of exposure may prevent or reduce the eruption, barrier creams (Ivy Guard) applied before exposure may provide some protection
- other common contact allergens include nickel, rubber, latex, glues, dyes, neomycin, and topical anesthetics

Contact Dermatitis

- photosensitizers are allergens that require sunlight to become activated and cause a photocontact dermatitis when the patient is exposed to sunlight;
- the rash erupts in a symmetric distribution on the face, the “V” of the neck, and the arms below the shirt sleeves
- topical photosensitizers produce localized patches of dermatitis when applied to sun-exposed areas
- id reaction: severe local reaction in a contact dermatitis induces an immunologically mediated secondary eczematous dermatitis

Contact Dermatitis

treatment:

- small areas of contact dermatitis: topical corticosteroids and avoiding further contact with the inciting agent
- widespread reactions or severe local reactions in the face/genital/hands: 2-3 week tapering course of systemic corticosteroids
 - ▣ a shorter course may cause the rash to rebound
 - ▣ most respond within 48 hours



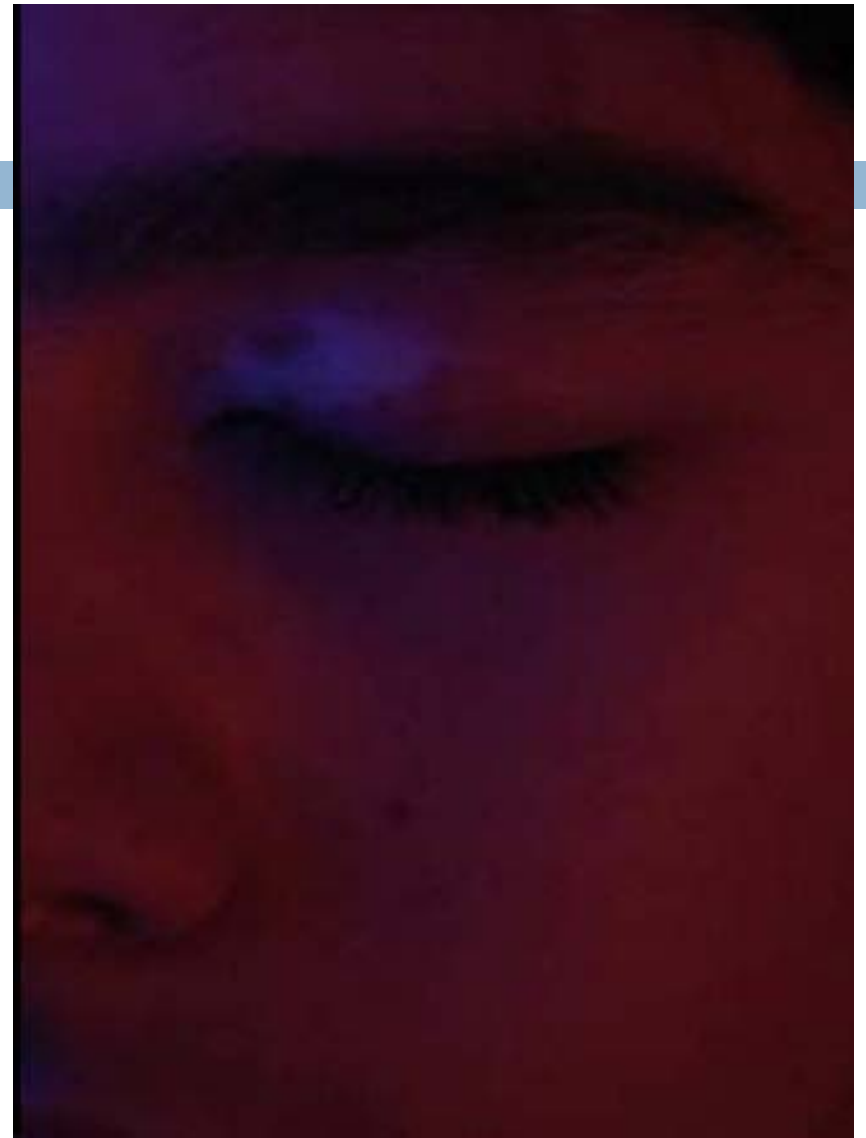
Seborrheic Dermatitis

- characterized by symmetric, red, scaling eruptions
- occurs predominantly on hair-bearing and intertriginous areas
- in infants, scalp lesions called “cradle cap” are greasy, salmon-colored, scaly; severe form is more generalized
- in adolescents, the dermatitis manifests as dandruff or flaking of the eyebrows, postauricular areas, nasolabial folds, and/or flexural areas
- pathogenesis is unknown
- usually non-pruritic, some clear spontaneously



Seborrheic Dermatitis

- management:
 - low potency topical corticosteroids
 - anti-seborrheic shampoos
- secondary bacterial infection usually caused by GAS and/or S. aureus
 - occurs commonly in the neck, axillary, and groin creases of infants
 - should be cultured and treated with antibiotics
- can differentiate from atopic dermatitis by asking about severity of pruritis and checking diaper area
- if thick white scales, or persistent diaper dermatitis and cradle cap, may be difficult to differentiate from psoriasis without a skin biopsy



Vitiligo

- acquired disorder of pigmentation in which there is complete loss of pigment in involved areas
- lesions are macular and appear progressively around the eyes, mouth, genitals, elbows, hands, and feet
- spontaneous but slow repigmentation may occur from the edges of active lesions and the hair follicles within, which can give a speckled appearance
- transient hyperpigmentation of the contiguous normal skin or hypopigmentation of the advancing edge may produce a trichrome
- rarely, the pigment in the eye may become involved
- histologically, melanocytes are completely absent in areas of vitiligo
- melanocytes are destroyed by an autoimmune mechanism

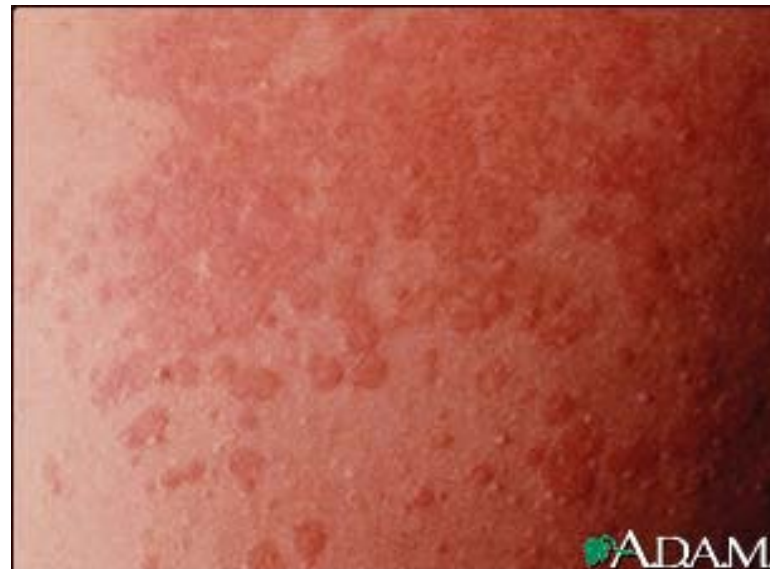
Vitiligo

management:

- protect skin from sun damage
- BID application of medium to high potency topical corticosteroids x 2-4 weeks
- light therapy with PUVA or narrow band UVB
- temporary camouflage with cosmetics and topical dyes may help hide lesions
- the well defined edges of vitiligo differentiates it from postinflammatory hypopigmentation and pityriasis alba
- the lack of scaling in vitiligo differentiates it from tinea versicolor
- by woods lamp, a blue-white sharply demarcated fluorescence is seen from the lesions



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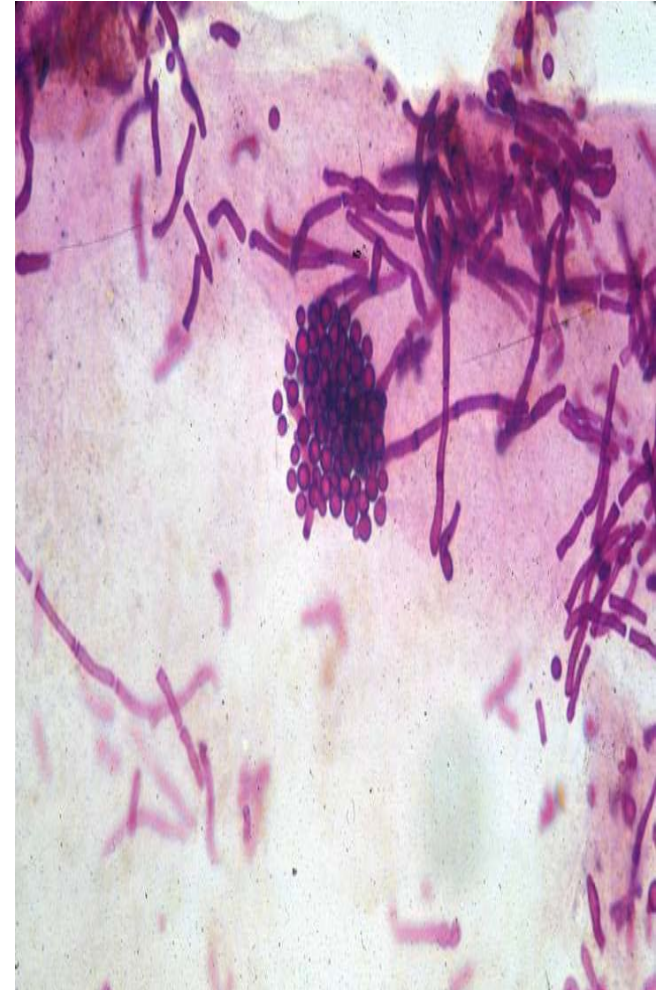
ADAM.

Tinea Versicolor

- characterized by multiple, small, oval, scaly patches that measure 1-3cm in diameter
- usually located in raindrop pattern on upper chest, back, and proximal portions of the upper extremities, facial lesions seen occasionally
- lesions may be light tan, reddish, or white in color
- usually asymptomatic but may cause some mild pruritus
- occurs more often in adolescents, but can affect children of any age
- caused by the yeast, *Malassezia furfur*, which commonly colonizes the skin by 4-6 months
- warm and moist climates, pregnancy, immunodeficiency states, and genetic factors predispose to the development of these lesions

Tinea Versicolor

- dx confirmed by KOH prep of surface scale or fungal culture
- by woods lamp, a yellow-green fluorescence is seen from the lesions
- treatment:
 - topical clotrimazole BID x 2 weeks
 - desquamating agents such as selenium sulfide x 15min daily x 2 weeks
 - for recalcitrant cases: try oral ketoconazole, itraconazole, or fluconazole
- educate patient and family that there is a high rate of recurrence and pigmentary changes may take months to clear, even after eradication of the fungus
- can try selsun blue shampoo once a month to scalp and trunk to decrease recurrence





Pityriasis Alba

- subtle and poorly demarcated areas of hypopigmentation in the face, neck, and upper extremities
- lesions may progress through 3 stages:
 - 1. Erythematous scaling papules
 - 2. Hypochromic scaling papules
 - 3. Smooth hypochromic patch
- usually occurs in atopic patients
- usually asymptomatic except for mild pruritus during stages 1&2
- occurs in people of all races, more prevalent in males
- more noticeable in summer months when rest of skin tans, and in darker skinned individuals
- re-pigmentation occurs slowly, cases can last from several months to 10 years, but the average duration is a year or more

Pityriasis Alba

- educate patient and family on sun protection and gentle skin care to prevent dry skin
- severe cases:
 - treat with topical corticosteroids
 - referral to derm for light therapy to help accelerate repigmentation
- rule out other causes of hypopigmentation by taking a good history
- rule out tinea versicolor by KOH prep of scrapings from skin lesions or fungal culture
- by woods lamp, a white-blue fluorescence may be seen like vitiligo, but not as bright and the borders are not as well defined



Pityriasis Rosea

- benign, self limited disorder
- can occur at any age, but most common in school-age children and adolescents
- prodrome of malaise, headache, and mild constitutional symptoms occasionally precedes the rash
- 1/2 of the cases begin with the appearance of a “herald patch”
- within 1-2 weeks, numerous smaller round to oval patches appear on the body, usually concentrated on the trunk and proximal extremities, forms a “Christmas tree” pattern on the back and thorax
- rash peaks in several weeks and slowly fades over 6-12 weeks
- cause unknown, viral etiology?
- UV light and oral erythromycin may hasten the disappearance of the eruption, but post-inflammatory hyperpigmentation may persist for months



Scabies

- caused by the *Sarcoptes scabiei* mite
- pruritic rash characterized by linear burrows, papules, nodules on the finger webs, wrists, elbows, feet, ankles, belt lines, areola, scrotum, and penis
- in infants, burrows are widespread on the trunk, scalp, extremities, including the palms and soles

Scabies

■ treatment:

- permethrin 5% cream can be used safely for children as young as 2 months
 - apply head to toe x 8-14 hrs, rinse off, and repeat in 7 days
- patient, entire family, and others who have had close contact to the patient should be treated simultaneously
- topical lubricants are necessary to counteract the drying and irritation produced by the scabicide
- oral or topical medications to prevent pruritis
- wash all clothing, sheets, towels, or place in sealed bag x 1 week
- educate family that pruritus can last for 2-4 weeks after treatment, but if see new lesions on skin that suggests reinfestation or inadequate therapy



Molluscum

- caused by poxvirus
- endemic in young children
- contagious by direct contact or indirect contact through fomites
- characterized by sharply circumscribed, single or multiple, superficial pearly, dome shaped, papules with umbilicated centers
- commonly distributed in the trunk, axillae, face, and diaper area
- lesions are spread by scratching and frequently appear in a linear arrangement
- in teens, molluscum occurs frequently in the genital area as a sexually transmitted disease

Molluscum

- most cases undergo spontaneous remission, but recurrences are common
- treatment directed against symptomatic lesions only
 - liquid nitrogen
 - application of a blistering agent (cantharidin) and plastic tape, peeled off in 1-3 d
 - destruction of lesions by curetting their cores
- patients with widespread, recalcitrant molluscum should be screened for congenital and acquired immunodeficiency

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