CHILDHOOD ALOPECIA AND COMMON PEDIATRIC RASHES
Childhood Alopecia
TINEA CAPITIS, TRICHOTILLOMANIA, ALOPECIA AREATA, AND TELOGEN EFFLUVIUM ACCOUNT FOR >95% OF CASES OF ALOPECIA IN CHILDREN.
Normal Hair Cycle:

1st = anagen phase (80-90%)

3rd = telogen phase (5-10%)

2nd = catagen phase (1-3%)

hair is shed
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
Trichorrhexis 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 one's 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. audouini 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
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
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
complications:

- secondary bacterial infection
  - crusted exudative patches
  - usually caused by GAS or S. aureas
  - 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
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 pruritus 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
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. Hypocromic 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, repeat in 7 days
- patient, entire family, and other 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 pruritus
- 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 reinfection 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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