Diseases of the hair

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Basic aspects of hairs

Types of hairs

Lanugo hairs – newborns → nonmedullated, fine nonpigmented

Vellus hairs – have some pigment, during childhood → gradual transition of the scalp hairs from vellus → intermediate → terminal hairs

Terminal hairs – medullated, thicker, longer, more pigmented
Basic aspects of hairs

Puberty - hormonally sensitive regions (axillae, genital area, beard, trunk, extremities: vellus → replaced by terminal hairs

Hormonal influences → response to androgens
Sexual hairs – androgen concentration only in men → beard, pubic triangle, umbilicus, external ear
Ambisexual hairs – androgen concentration in women as well in men → axillae, pubic area
Nonsexual hairs – no response to androgens → eyebrows, eyelashes
Hair growth
(hair growth averages cca 0.35 mm/day, 1.0 cm/month)

**Anagen phase**
- or growing phase (3 to 6 years)

**Catagen phase**
- or transitional phase (1-2 weeks)

**Telogen phase**
- or resting phase (2 to 3 months)
Hairlessness: Alopecia

Reversible alopecia

- Alopecia areata
- Telogen effluvium
- Anagen effluvium
- Trichotillomania
- Mechanical alopecia

Irreversible alopecia

- Pseudopelade Brocq
- Androgenetic alopecia
- Mechanical alopecia
Reversible alopecia
Alopecia areata

Etiopathogenesis

- Great mystery, an immunologic origin, neither the common triggers nor the pathogenic mechanism are clear,
- Hair follicle keratinocytes → abnormally sensitive to some endogenous or exogenous stimulus
- The inflammatory response in the hair follicle causes damage → ranges from dystrophic changes and immediate hair loss → conversion to telogen hairs with hair loss.
- After inflammation the hair follicles begin to function normally again.
Alopecia areata

Clinical findings

one or more round or oval patches of sudden and complete hair loss;
the hairless region is often slightly sunken because of the loss of the mass of the hair bulbs;
minimal erythema, most often ivory white colored lesion
hair follicle opening are preserved
any atrophy
Alopecia areata

Signs of progress

**Exclamation point hairs**
0.2-0.7 cm long, minimally pigmented, often split on their distal end; pointed on their proximal end

**Cadaverized hairs**
resemble comedones, contain necrotic matrix remnants
Alopecia areata

Typical localisation

**Capillitium**: occipital and temporal regions – are most often involved;

**Other body areas**: beard, eyebrows, eyelashes,

20% of patients → variety of nail changes → pitted nails, spotted lunulae, red lunulae
Alopecia areata

Special types of alopecia areata

**Ophiasis** snake-like → along the side of the head, especially in the temporal region and nape, many patients have atopic dermatitis; difficult to treat
Alopecia areata

Special types of alopecia areata

Alopecia areata diffusa

Alopecia totalis and universalis

A. totalis → the entire scalp hairs are lost
A. universalis → the entire scalp and body hairs are lost including pubic, axillary hairs, eyelids, eyelashes
Alopecia areata

Course and prognosis

Very unpredictable, regrowth → hairs of a different color, lighter than normal, ⅓ regrowth in 6 months, ⅓ in 12 months, less than ½ of patients have effective regrowth, any recurrence in 5 years.
Poor prognostic factors → age (worse when younger), severity, speed of spread and duration, atopic patients tend to have chronic disease
poor prognosis → ophiasis, alopecia totalis and universalis

Differential diagnosis

syphilis → alopecia areolaris (II. stage of syphilis), multiple lesions, serological testing, lymphadenopathy
tinea capitis, trichotillomania, pseudopelade (biopsy)
Alopecia areata

Systemic therapy
Progressive and widespread disease
Corticosteroids: Prednison 60 mg daily → taper to 20 mg daily over 3 weeks → low-dose maintenance therapy or alternate-day therapy for 8 weeks 20% patients have satisfactory results.
Oral zinc for 6-8 months, in ⅔ patients the hairs regrows in years; high-dose cyclosporine.

Topical therapy
High-potency corticosteroids – lotions, gels → 1 cm into the normal hair, intralesional triamcinolone acetonide mixed with topical anesthetics, minoxidil solution, PUVA therapy
Iiereversible alopecia
Pseudopelade

Etiopathogenesis

- Two types
  - ½ end-point of number of scarring alopecia → lichen planus, discoid lupus erythematosus, folliculitis decalvans
  - Uncommon cause → sarcoidosis, metastases, favus, granuloma annulare
  - Separate disease idiopathic with unknown pathophysiology
  - Stem cell failure, follicular stem cells (essential for hair growth) and sebaceous gland destruction
- Slowly progressive over periods of months to years
- Middle –aged women
Irreversible alopecia
Pseudopelade

Clinical findings
Onset – one or several small patches of alopecia in the parietal region; any symptoms; slowly expanding;
Irregular lesions → may coalesce, sharp border.
Skin → shiny, yellow-white, porcelain-white, thinned, depressed, atrophic (cigarette paper-like), lack of follicular orifices.
Small bundles of hairs remain; no sings of inflammation or follicular plugging; no pruritus.
Irreversible alopecia  
**Scaring alopecia**

All scaring processes are capable of damaging the hair follicle → hair loss → irreversible alopecia

**Cause** → extensive inflammation, infections, tumors and trauma

**Histopathology** → dermal scaring, relatively deep along with absent or reduced hair follicles, reduced number of arrector pili muscles

**Lichen planus**

⅓ of patients with scaring alopecia, most patients have lichen planus on the skin or mucosa, some of them only scalp; Patchy alopecia, individual follicular papules or hyperkeratotic plugs; frontal hairline in postmenopausal women
Irreversible alopecia
Scaring alopecia

Lupus erythematosus → Lesion with erythematous border and central white scared area
Systemic LE → diffuse alopecia

Morphea → Coup de sabre form
Systemic scleroderma → diffuse alopecia
Irreversible scaring alopecia
Pseudopelade

**Course and prognosis**
Unpredictable → diffuse alopecia or focal hair loss for many years

**Differential diagnosis**
alopecia areata → regrowth

**Therapy**
Corticosteroids, topical therapy high-potency corticosteroids, intralesional corticosteroids
Progressive non-scaring alopecia
Androgenetic alopecia in men

Epidemiology
The most common alopecia of all, comprising 95% of cases; almost every white man winds up with some degree of male pattern baldness.
Progressive non-scaring alopecia
Androgenetic alopecia in men

Pathogenesis

3 major factors

- 1. genetic predisposition (dominant inheritance with variable penetrance and expression; may be polygenic inheritance)
- 2. age
- 3. androgens

Gradual conversion of terminal hair into indeterminate hair and finally to vellus hair. Hair follicles receptors are sensitive to androgens; enzyme 5-alfa-reductase converts testosterone to dihydrotestosterone influencing the hair production.
Androgenetic alopecia in men

Clinical findings
Onset → fronto-temporal area → well-defined patterns,
Hair loss is gradual, rarely shows sharp borders as it progresses.
Advanced stages → a sharp division between remained hairs and the smooth bald skin with vellus hairs;
Bald skin is not atrophic, it may feel thinned – few hair bulbs are present;
Sebaceous glands are retained → appear prominent as yellow bulbs → an oily scalp.
Androgenetic alopecia in men

Course and prognosis

Impossible to predict the rapidity of hair loss or final extent of baldness

Therapy

Systemic finasteride → inhibitor of type II 5-alfa reductase (it is not antiandrogen); topical minoxidil 2%, surgical-hair plug transplantation; micrografting.
Progressive non-scaring alopecia
Androgenetic alopecia in women

**Etiopathogenesis**

Association with underlying endocrine abnormalities—ovaries and adrenal glands produced androgens

The same genetic factors as to men, androgens in women tend to cause hirsutism and virilization, hair loss on the scalp;

Selected scalp hair follicles have exquisite androgen sensitivity, no elevated serum androgen levels.

¼ of women with virilization and elevated androgen levels have androgenetic alopecia;
Progressive non-scaring alopecia
Androgenetic alopecia in women

Clinical findings
Some patients have overlapping features

- Female pattern
  More common before menopause, 20-40 years old women, no or minimal elevations in androgens, diffuse hair loss

- Male pattern
  After menopause ⅓ have at least some frontotemporal loss, other signs of virilization
Androgenetic alopecia of women

Laboratory findings
Free testosterone and DHEA-S ↑ ratio
Testosteron/estrogen,
Less sex hormone-binding globulin (SHBG) is available and more active or free androgen is circulating.
3-α-17-αandrostendione/SHBG ratio is raised, measurement of prolactin, luteinizing hormone, follicle stimulating-hormone
Androgenetic alopecia in women

Course and prognosis
Development of complete baldness is uncommon;
No matter what is the age of onset;
Progress after menopause as the counteraction of estrogens is lost.

Therapy
Systemic oral contraceptives and antiandrogens;
Topical minoxidil solution (forbidden in pregnancy and nursing)
Reversible alopecia
Telogen effluvium

**Epidemiology** almost exclusively in women; clinical hair loss is apparently not noticeable for men with generally shorter hair styles

**Etiopathogenesis** relatively minor damage to anagen hairs leads to a premature ending of anagen phase → telogen hairs; the time 2-5 months;
Triggers → high fever, emotional or traumatic shock, delivery of a baby, after a major surgery.
→ **infection** with high fever (typhoid fever, malaria, viral infection, erysipelas)
Reversible alopecia
Telogen effluvium

• Medications and chemicals
  In low doses

• Hormonal changes
  Postpartum alopecia 2-4 months after delivery, resolves after few months (pregnancy → anagen phase is prolonged);

Starting or stopping of oral contraceptives (first 4-6 cycles) → resolve even the medication is continued;

Hypo- and hyperthyroidism;
Ovarian, pituitary adrenal tumors;
Reversible alopecia
Telogen effluvium

- Acute and chronic illnesses
  - malignancies,
  - connective tissue disease,
  - malabsorption,
  - HIV/AIDS,
  - erythroderma

- Iron deficiency
- Acute stress situation
- Physiologic neonatal hair loss
Telogen effluvium

**Clinical findings** dropout about 25% of scalp hairs before others can notice thinning; more than the average individual lose 50-100 hairs daily.

**Laboratory findings** trichological examination – more than 20% of telogen hairs

**Therapy** no systemic treatment available to halt hair loss or speed regrowth; oral gelatin, biotin, multivitamin preparations, documented hormonal problems should be corrected; topical hormonal tinctures and lotions
Reversible alopecia
Anagen effluvium

Sudden loss of dystrophic hairs secondary to acute toxic effect on the follicles (e.g. cancer therapy).

**Etiopathogenesis** difference between anagen and telogen effluvium is of the degree of follicle damage;

the same agent

- in high doses → anagen effluvium
- in low doses → telogen effluvium

Severe damage to the growing anagen hairs leads to the development over a period of days of dystrophic hairs → thin at its base, breaks and falls out → rapid diffuse loss of hair

Immediately associated with the traumatic event → **damage of the hair matrix** (inhibition of mitotic spindle, arrest cell growth)
Anagen effluvium

Hair break off just above the zone of keratinization; the necrotic matrix forms plugs (melanin, keratin, inner root sheet) → are extruded through the follicular opening (comedo-like plugs) – **cadaverized hair**

- Cholchicine, thallium, insecticides and pesticides, heavy metals – arsenic, boric acid, some plant toxins, radiation (3-10Gy)
Anagen effluvium

Clinical findings
hair loss is sudden, rapid, may be very extensive; loss of 90% of hairs in a matter of weeks, abundance of hairs on pillow (chemotherapy)

Course and prognosis
agent is removed, Withdroven of toxins \(\rightarrow\) regrowth is complete
Some chemotherapy \(\rightarrow\) regrowth of hairs in different colors and texture (curls \(\Leftrightarrow\) straight)

Therapy any effective therapy
Mechanical alopecia
Non-scaring alopecia

Acute and chronic pressure and traction → hair loss

**Chronic pressure** – localized area- occupational stigma (nun –caps, people caring heavy loads with the aid of a head strap)

**Chronic traction** alopecia – occipital region where the hair is pulled up in to a bun; old regional costumes required the hair to be pulled up tightly; braids or pony tails – the hair is pulled back tightly → hair loss along anterior marginal hair line; extremely tight hair curlers can cause alopecia.
Mechanical alopecia

Prognosis uncertain, soon interruption of mechanical forces → regrowth can be expected.
If not → regressive follicular changes will occur, leading to either cessation of hair production or production of thin, short vellus-like hair.
Reversible alopecia
Trichotillomania

Self-induced traction alopecia
The hairs are either purposely or unknowingly removed;
Relatively common, 6-times ↑ in women,
⅓ of patients are children ↓ 10 years old, ⅓ teenagers, ⅓ adults;
A sign of underlying emotional disturbances.
Trichotillomania

Clinical findings
Localized area of hair loss in the frontotemporal region
Right handed patient → lesion on the left side; the hairs are twisted around the fingers and then pulled;
Bald area → irregularly shaped, shows new hairs of varying length; can be removed when they reach a certain graspable length;
specific clinical sign → fresh hemorrhage in the follicles
Trichoclogical exam → no telogen hairs, many catagen hairs;
clinically normal regions → normal trichogram

Therapy
Psychological or psychiatric help
Excessive hair growth
Hypertrichoses

Pale vellus hairs are transformed into dark, thick terminal hairs

**Congenital hypertrichosis lanuginosa**

Very rare condition excessive lanugo hairs → long, fine, usually lack of pigment, under microscope lack of a medulla. Born variant or become apparent during the first weeks of life, sometimes terminal characteristics (pigment) „monkey men“, “dog-faced men“.
Hypertrichoses

Aquired hypertrichosis lanuginosa
Almost invariable marker for an internal malignancy (carcinoma, often metastatic)
Hypertrichoses

Aquired circumscribed hypertrichosis
chronic low-grade trauma, radiation therapy, heat, topical corticosteroid

Hypertrichosis as a sign of internal disease
Porphyria (temples and cheeks), postencephalitis hypertrichosis, multiple sclerosis, endocrine abnormalities (acromegaly, hypothyroidism), malnutrition, anorexia nervosa, dermatomyositis

Drug-induced hypertrichosis
(may convert vellus to terminal hairs) antihypertensive minoxidil, psoralens, cyclosporine, interferons, phenytoin
Hirsutism

Increased hair growth of a male pattern in women or children; caused by abnormal hormone levels or by increased end organ sensitivity to hormones.
Hirsutism

Etiopathogenesis

Endocrine – polycystic ovary syndrome (Stein-Leventhal sy); ovarian tumors; Cushing sy and disease; adrenal tumors, acromegaly, hyperprolactinemia, hypogonadism sy

Medications – ACTH, anabolic steroids, androgens, Danazol, progesterons, spironolactone

Therapy underlying cause