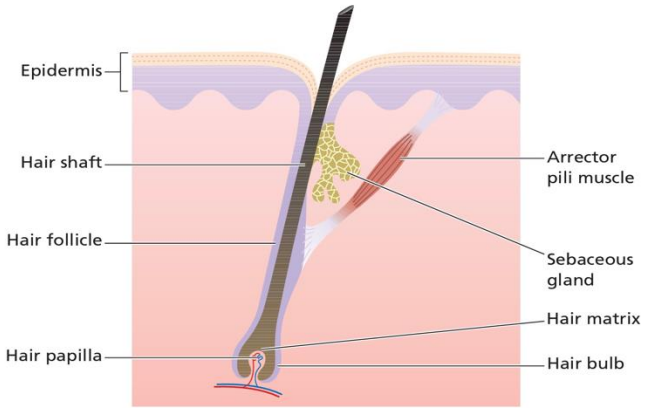
Fifth stage
Dermatology **Lec 8**
Dr. Intisar Ahmed

Hair Disorders

*Hair is human plumage.
*Hair follicles form before the **ninth week** of fetal life.
*The sebaceous gland is an outgrowth at the side of the hair, forming pilosebaceous unit.
*Hair **matrix**, is the germinative part of the follicle, equivalent to the basal cells of the epidermis.
*Adjacent to the sebaceous gland is the region of insertion of the arrector pili muscle called the **bulge**, This area contains hair follicle stem cells which can regenerate the entire hair follicle and sebaceous gland, damage to this area will cause permanent hair loss.
*Melanocytes migrate into the matrix and are responsible for the different colors of hair (**eumelanin**, brown and black; **phaeomelanin**, red).
*Hairs are classified into three main types.
1. Lanugo hairs: fine long hairs covering the fetus, but shed about 1 month before birth.
2. Vellus hairs: fine short unmedullated hairs covering much of the body surface, they replace the lanugo hairs just before birth.
3. Terminal hairs: long coarse medullated hairs seen, for example, in the scalp or pubic regions, their growth is often influenced by circulating androgen levels.

Note: lips, glans penis, labia minora, palms and soles are free of hair follicles.

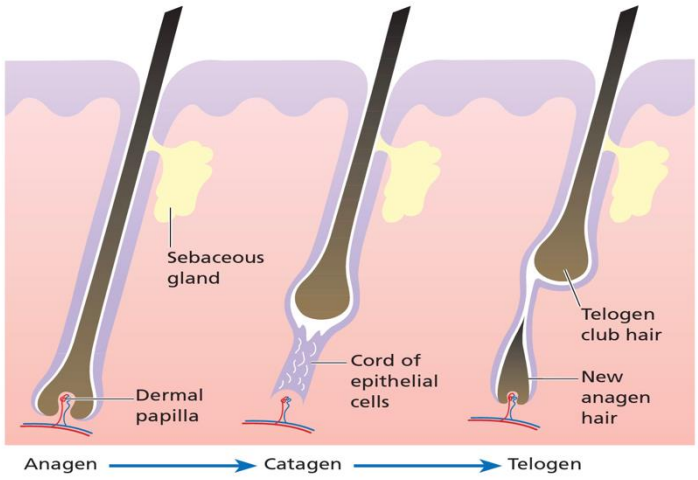


***The hair cycle:**

Each follicle passes, **independently of its neighbours,** through regular cycles of growth and shedding.The three phases of follicular activity are:

1. **Anagen**: the active phase of hair production.
2. **Catagen**: a short phase of conversion from active growth to the resting phase. Growth stops, and the end of the hair becomes club-shaped.
3. **Telogen**: a resting phase at the end of which the club hair is shed.

*The duration of each of these stages varies from region to region, eg: on scalp, anagen lasts up to **5 years**,
catagen **2 weeks,**
telogen **3 months.**
***85%** are normally in anagen and **15%** in the telogen phase.
*The scalp contain an average of **100 000** hairs.
***100** hairs may be shed from the normal scalp every day as a normal consequence of cycling.
*The length of hair is determined by the duration of anagen (e.g. the hairs of the eyebrows have shorter cycles than those of the scalp).



Alopecia

*Loss of hair and has many causes and patterns. Divided into **localized and diffuse** types.

*****Localized alopecia**

Causes of localized alopecia

1.Non-scarring:
-Alopecia areata
-Androgenetic
-Scalp ringworm (human)
-Hair-pulling habit
-Traction alopecia
2.Scarring:
-Burns, radiodermatitis
-Aplasia cutis
-Kerion
-Carbuncle
-Cicatricial basal cell carcinoma
-Lichen planus
-Lupus erythematosus
-Necrobiosis
-Sarcoidosis
-Central centrifugal cicatricial alopecia

Alopecia areata:

*localized area of hair loss.
*lifetime risk is about 2%.

Cause:

1. immunological: association with autoimmune diseases such as Hashimoto's thyroiditis, atopy, vitiligo, inflammatory bowel disease and other.
2. Genetic.
3. 10% of patients with Down's syndrome.
4. Environmental factors in the genetically predisposed.

Presentation:

*A typical patch is uninflamed, with no scaling, with empty hair follicles.
*Pathognomonic **'exclamation-mark'** hairs seen around the edge, are broken off hair about 4 mm from the scalp, with the proximal end more narrowed and less pigmented.
*Lose of hair from all heads, **alopecia totalis** and from whole skin surface, **alopecia universalis.**
*Common in scalp and beard areas, can occur on eyelashes and eyebrows.
*Up to 50% of patients show fine pitting or wrinkling of the nails.

Course:

*Unpredictable.
*In a first attack, regrowth is usual within a few months.

*50% resolve spontaneously without treatment in 1 year
*Only 10% develop severe chronic disease.
****A poor prognosis:**
1 Onset before puberty.
2 Association with atopy or Down's syndrome.
3 Unusually widespread alopecia.
4 Involvement of the scalp margin (ophiasiform type), especially at the nape of the neck.
Investigations:
*Histology: T lymphocytes cluster like a swarm of bees around affected hair bulbs.
Treatment:
-Topical corticosteroid creams of high potency.
-Intradermal injection steroid.
-Mild irritants, dithranol.
-PUVA for extensive cases.
-Topical immunotherapy, diphencyprone and squaric acid dibutyl ester.
-Tacrolimus.
-Wigs and cosmetics.

Androgenetic alopecia (male and female-pattern hair loss):

Cause:
1.Familial.
2.Hormonal: androgen hormones are implicated in the pathogenesis.
*In men high levels of dihyrotestosterone DHT (Testosterone is converted to DHT by the enzyme 5αreductase).
*DHT is responsible for temporal scalp hair recession, terminal hair growth in the beard, external ears, nostrils and limbs, and acne.
*In female-pattern hair loss, increased sensitivity to circulating androgen, as androgen levels are usually within normal limits.
Presentation:
*In men loss of hair first from the temples, then from the crown.
*In women the hair loss may be much more diffuse particularly over the crown.
*In bald areas, terminal hairs are replaced by finer vellus ones.
Complications:
1.Anxiety.
2.Bald scalps burn easily in the sun, and may develop multiple actinic keratoses.
3.Bald men are more likely to have a heart attack and prostate cancer.
Investigations:
-None.
-In women virilization may have to be excluded.

Treatment:
-Scalp surgery.
-Hair transplants and wigs.
-Topical minoxidil.
-Women may benefit from suppressing ovarian androgen production with oral contraceptives or anti androgen such as spironolactone.
-Finasteride an inhibitor of human type II 5α-reductase (not indicated in women or children). Side effects decreased libido, erectile dysfunction and altered prostate-specific antigen levels.

Traction alopecia:

Cause:
*Hair pulled out by several procedures including hot-combing to straighten kinky hair, tight hair styles such as a ponytail.
Presentation:
*Seen in girls and young women.
*Marginal alopecia is common pattern in which hair loss is mainly around the edge of the scalp.
*The bald areas show short broken hairs, folliculitis and, sometimes, scarring.
Clinical course:
*Even if the pt alter their cosmetic practices, regrowth is incomplete.
Treatment:
-Patients have to stop doing whatever is causing their hair loss.
-Rollers that tug can be replaced by those that only heat.

Central centrifugal cicatricial alopecia:

Cause:
*Hair loss with **scarring** over the vertex of the scalp.
*Hair styling practices including chemical hair relaxer and hot comb use reported.
Presentation:
*Most common form of scarring alopecia among black women.
*Beginning at the vertex of the scalp, gradually spread centrifugally, causing permanent loss of the hair follicles.
*Pruritus, tenderness and tufted hairs are common findings.
Some patients may note pustules and crusting.
Clinical course:
*Chronic and progressive.

Treatment:
-Topical and intralesional corticosteroids in combination with oral antibiotics such as doxycycline for their anti-inflammatory properties.

*****Diffuse hair loss**
*Causes of diffuse hair loss:
-Telogen effluvium.
-Endocrine:
*hypopituitarism.
*hypo-or hyperthyroidism.
*hypoparathyroidism.
*high androgenic states.
-Drug-induced:
*antimitotic agents (anagen effluvium)
*retinoids.
*anticoagulants.
*vitamin A excess.
*oral contraceptives.
-Androgenetic.
-Iron deficiency.
-Severe chronic illness.
-Malnutrition.
-Diffuse type of alopecia areata.

Telogen effluvium:
Cause:
*Triggered by any severe illness, particularly those with bouts of fever or haemorrhage, by childbirth and by severe dieting.
*All of these synchronize catagen so later on, large numbers of hairs are lost at the same time in the telogen phase.
Presentation and course:
*Hair fall, 2–3 months after the provoking illness, can be mild or severe.
*Beau’slines may be seen on the nails.
*Regrowth, not always complete, usually occurs within a few months.
Differential diagnosis:
*In androgenetic alopecia in female, onset is **gradual** in **mid adulthood**, and hairs remain rather **firmly** anchored to the scalp.
*In telogen effluvium the onset is **abrupt** and **follows** acute illness and **lightly** pulling on scalp hairs dislodge many.

Treatment:
-This condition is unaffected by therapy, but patients can be reassured that their hair loss will be temporary.

Note
Checking for haemoglobin, ESR, antinuclear antibody, serum iron, ferritin, thyroxine and TSH levels. Also checking the serum free testosterone and dihydroepiandrosterone sulfate levels in women with menstrual irregularities or hirsutism.

Rare genetic causes of hypotrichosis:
-Hypohidrotic ectodermal dysplasias.
-Monilethrix.
-Menkes' syndrome.
-Netherton's syndrome.

Hirsutism and hypertrichosis

*Hirsutism is the growth of terminal hair in a woman which is distributed in the pattern normally seen in a man.
*Hypertrichosis is an excessive growth of terminal hair that does not follow an androgen-induced pattern.

Hirsutism:

Cause:
1. Constitutional (dermatologic): normal or slightly increased circulating levels of testosterone.
-Familial.
-Adrenal (adrenal SAHA syndrome).
-Ovarian (ovarian SAHA syndrome).
-Hyperprolactinemic(SAHA syndrome with hyperprolactinemia).
2. Endocrine:
-Congenital adrenal hyperplasia.
-Cushing's syndrome.
-Polycystic ovary syndrome.
-Ovarian tumors.
-Prolactin-secreting pituitary adenoma.
3. Ectopic hormone production:
-Carcinoids.
-Choriocarcinomas.

4. Drugs:
-Anabolic steroids.

Presentation:
*Excessive growth of hair in beard area, on the chest, shoulder-tips, around the nipples and in the male pattern of pubic hair.
*Androgenetic alopecia may complete the picture.
Complications:
1. Infertility.
2. Psychological disturbances.
Investigations:
-Serum testosterone,
-Sex hormone binding globulin,
-Dehydroepiandrosterone sulfate,
-Androstenedione and
-Prolactin.
-Ovarian ultrasound is useful if polycystic ovaries are suspected.
Treatment:
-Any underlying disorder must be treated.
-Home remedies: commercial depilatory creams, waxing or shaving, or making the appearance less obvious by bleaching.
-Electrolysis.
-Laser.
-Topical therapy with eflornithine, an inhibitor of ornithine decarboxylase, can slow regrowth.
-Oral antiandrogens (e.g. cyproterone acetate; Dianette, or spironolactone): Pregnancy must be avoided during such treatment as it carries the risk of feminizing a male fetus.

Hypertrichosis:
1. localized type is seen in:
-Over melanocytic naevi including Becker's naevi.
-The sacral area –as a 'satyr's tuft' patients with spina bifida.
-Near chronically inflamed joints or under plaster casts.
-Occupational pressure (e.g. from carrying weights on the shoulder).

1.Generalized hypertrichosis caused by:
-Malnutrition: anorexia nervosa, starvation.
-Drug-induced (minoxidil, diazoxide, ciclosporin, phenytoin).
-Cutaneous porphyrias.

-Fetal alcohol and fetal phenytoin syndromes.
-Hypertrichosis lanuginosa (both congenital type and acquired).
-Some rare syndromes: Cornelia de Lange syndrome and Hurler's syndrome.