اغلب المحاضره صور
:
☺ما في خوف صديق

Pigment and Hair disorders

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المصادر :

تيم 428 + نوت الدكتور

Pigment disorders

- Classification:

  Hypo , hyper or depigmentation
- Vitiligo
- Albinism
- Melasma

NB :
Hypo : decrease color
Hyper : increase color
Depigmentation : totally disappearance of the color

Histology :

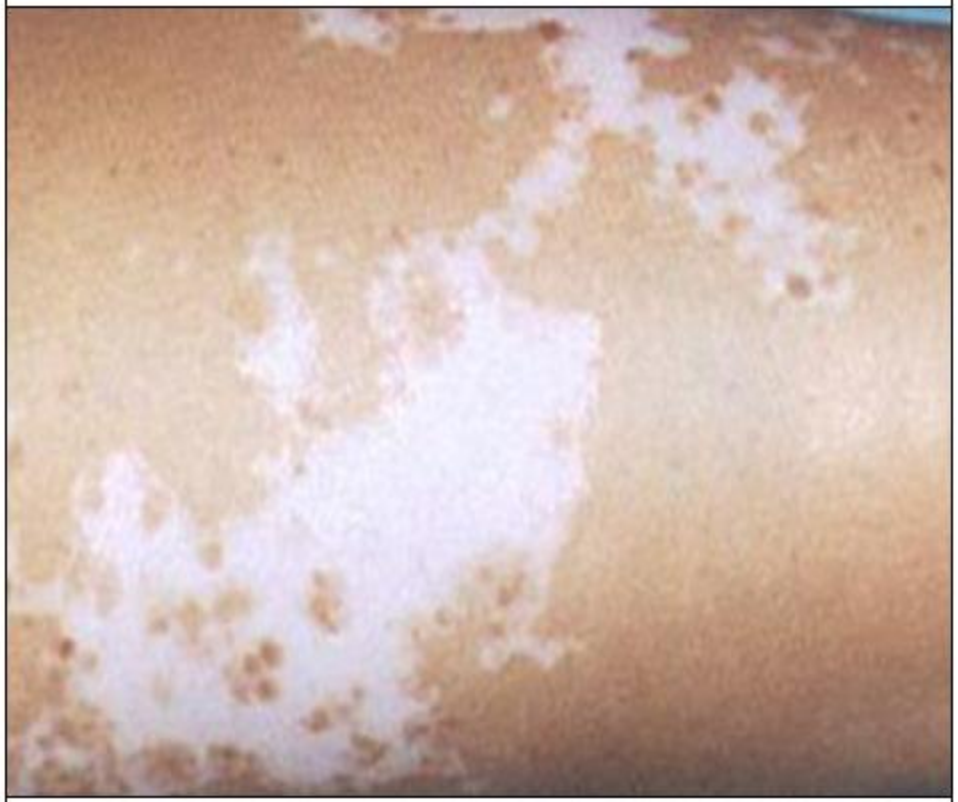
✓Introduction to skin pigment

The skin gets it color from the (melanin cells) , these cells located in the Basal layer of the epidermis مهمه جدا

So why some people have White color and others have black color ?

It depend on the activity of the Melanin cells

اولا : Vitiligo



VITILIGO:

- Acquired coetaneous **depigmentation due to loss of normal** melanocytes.
- Notice that it's **depigmentation (no pigmentation) not hypo pigmentation (decreased pigmentation) because there is a** difference.
- **Common site :** area with fracture
- → Keobner phenomena: Specifically on the leg, elbow and knee because mostly these are the trauma sites.

- **Why does it occur?** Loss of normal melanocytes. (NO more melanocytes) → due to autoimmune activity مهمه

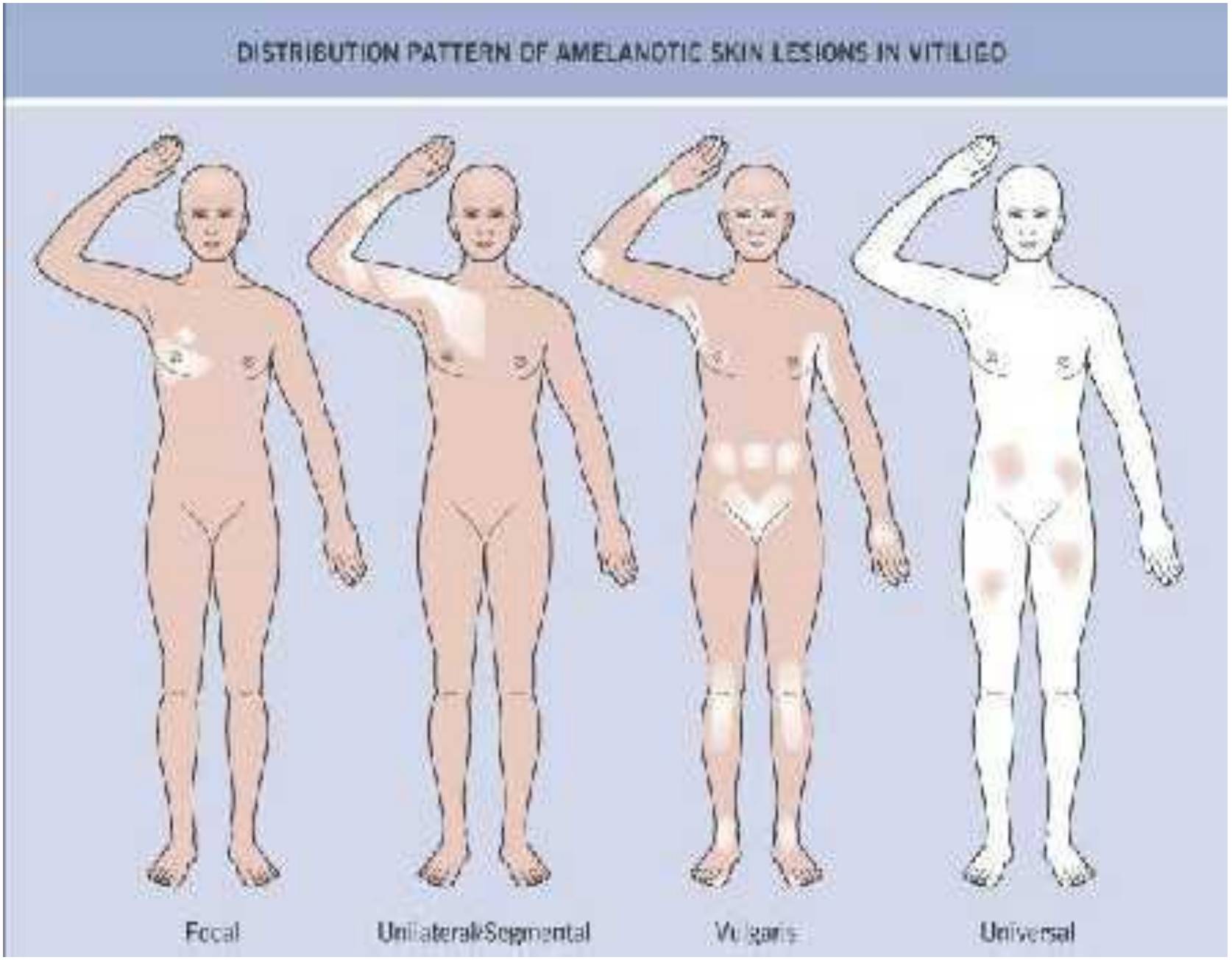
- To diagnose Vitiligo:
  - Woods lamp
  - Dopa stain: the stain will show NO melanocytes, it is the most commonly used stain

Causes and **Pathogenesis: (multifactorial):**

1. Genetic background.
   - A high proportion have positive family history
2. Autoimmune diseases. (the main cause)
   - Anti-melanocyte antibody can be detected in a proportion of patients suggesting
   - There is association with other autoimmune disease such as:
     - Pernicious anemia
     - Thyroid disease
     - Addison's disease
3. Neural (related to nerves).

- As it mentioned before the Autoimmune pathway is the most likely so in Vitiligo patient, it is IMP to check other Autoimmune Disease like (DM I and thyroid disease)

- **Natural course:**
  - It varies: could be localized, then becomes systemic, could be extensive or not.
- There is no rule.
- There is no definite treatment for Vitiligo but it can be well controlled.


DISTRIBUTION PATTERN OF AMELANOTIC SKIN LESIONS IN VITILIGO
Focal
Unilateral/Segmental
Vulgaris
Universal

•Note in the Picture there are some areas with hyper pigmentation, this is called **Perifollicular repigmentation, and it is the commonest in Vitiligo, because of** presence of melanocytes in the hair matrix.

•This is the commonest form of repigmentation. مهمه جدا – البقع البنيه

Distribution pattern (Types) :

- **Focal: 2 or 3 patches.**
- **Unilateral/segmented:**
  - **This is what raised the neural theory.**
  - **We say it's segmental** or unilateral if it involved a nerve area.
- **Vulgaris:**
  - **In the trauma sites (as we mentioned)**
  - **in the figure before the armpit and** the groin are included because they are sites of infection.
- **Universal:**
  - **When 90% or more of the body is involved.**
  - **In this case we don't treat,** we remove the normal area.

Why do we divide the Disease into the previous types :

- **To determine the management :** تفصل بشكل افضل لاحقا – هنا خذ فكره عامه :
- Focal and unilateral/segmental → Topical treatment.
- Vulgaris → Phototherapy.
- Universal → De-pigment the small normal areas (bleach).

Prognosis

- The prognosis of Vitiligo is very varied:
  - In some individuals only a small area of skin is affected and spontaneous repigmentation occurs
  - In others there is continuing and extensive loss
- There some cases have bad prognosis :
  - Widely separated → it is difficult to treat
  - if the tip of the Lip is affected → it is difficult to treat

Psychosocial effects

- Pt, with this Disease have many **Psychosocial problems like :**
  - the Society think that their disease is Contagious
  - the society think that their disease is due to poor hygiene
  - It can cause great distress in the community if the patient is dark skinned

- **These effects is more among the females**

Special studies to be done for the patient: مهمه جدا

Because most probably it is associated with autoimmune diseases we screen for the following:

1. T4, TSH, FBS → In case the patient has thyroiditis or diabetes.
2. ANA/Ro/La :
   A. prior to PUVA
   B. Because we need to check if he has photosensitivity or not
   C. If he has photosensitivity and we applied phototherapy on the patient, it will cause burn.
   D. For e.g. If the patient had SLE and Vitiligo (Due to SLE s/he is photosensitive so, we can't give her/him phototherapy).

Treatment:

1. Sunscreen:
   A. because their skin is sensitive and has no melanocytes
   B. Therefore no protection against sun light.
   C. To prevent sunburn, koebnerization and tanning. It is usually seen with universal type

2. **Skin camouflage** **مكياج ليوحد لون البشره – يوضع عند المناسبات**

3. Topical (limited treatment)

A. **Corticosteroids: Class 3 topical GlucoCorticoid (steroid) → The drug of choice.**

B. **Immunomodulators:**

   i. Topical Tacrolimus

   ii. not as effective as the steroid but it's safer.

C. **Outdoor topical psoralen (**Topical PUVA)

   i. **تضع هذا الكريم وتخرج للشمس لكي يشجع خلايا الميلانين على العمل**

4. Phototherapy
    A. UVA+ Psoralen= PUVA (not used anymore)
    B. NBUVA
    C. Excimer laser: narrow band, limited and on specific areas
    D. Phototherapy should be used for Generalized type IMP!!
5. Systemic treatment
    A. Usually Corticosteroids
    B. Used in Rapidly progressing disease
    C. Remember the Side effects:
        i. Osteoporosis
        ii. Liver disease
        iii. Psychosis
        iv. HTN
        v. Neurosis
        vi. DM
        vii. Infections
        viii. Cushing syndrome

6. Surgical

A. Done for resistant cases and the patient is stable for 2 years (to avoid Keobner phenomenon)

B. types:

I. Tissue graft : take a whole piece normal skin and implant it over the affected site

II. cellular : implant the melanin cells to the affected site

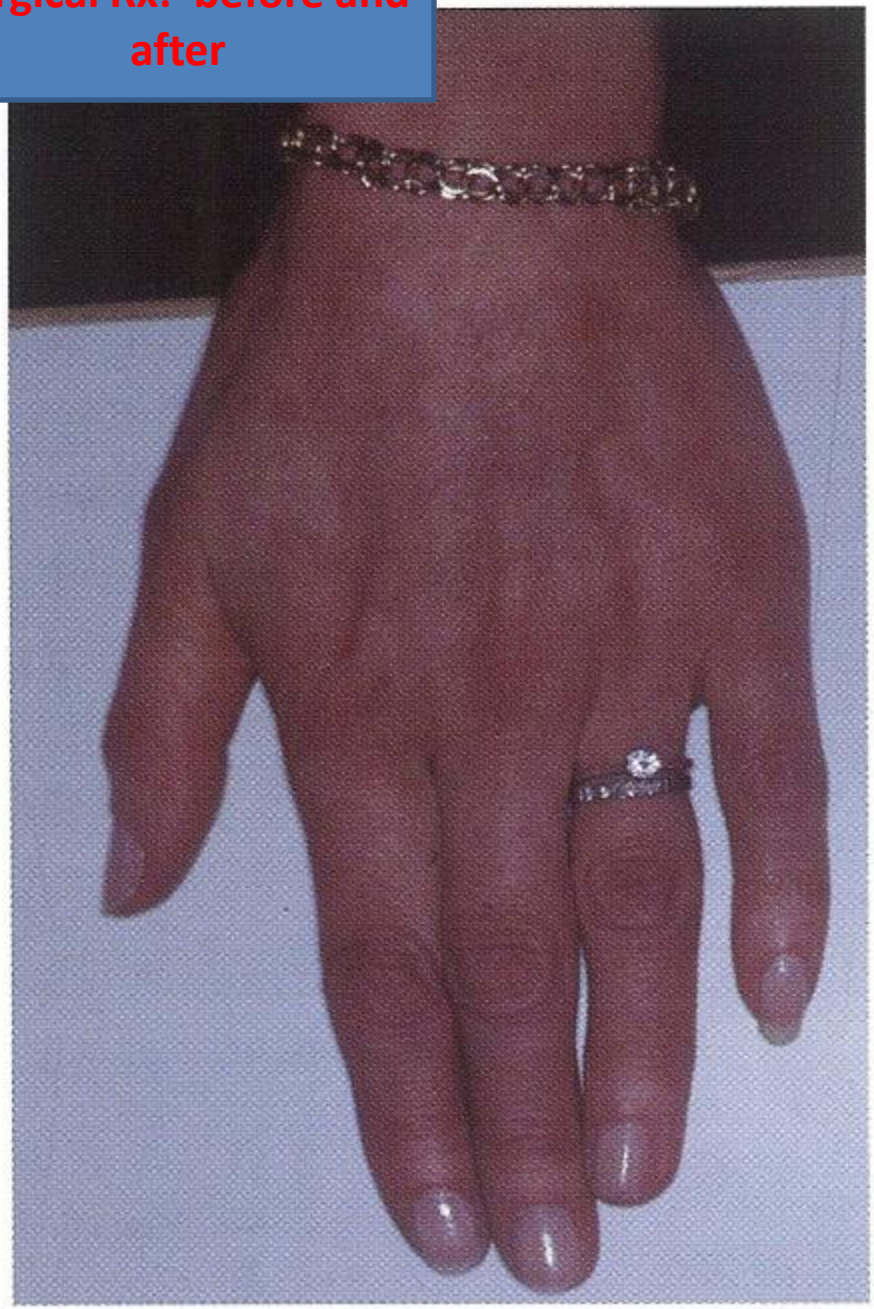
C. Conditions :

I. Disease should be stable for at least 2 year

II. the disease should cover only small area

III. put in your mind that some pt response better than others

Surgical Rx: before and after



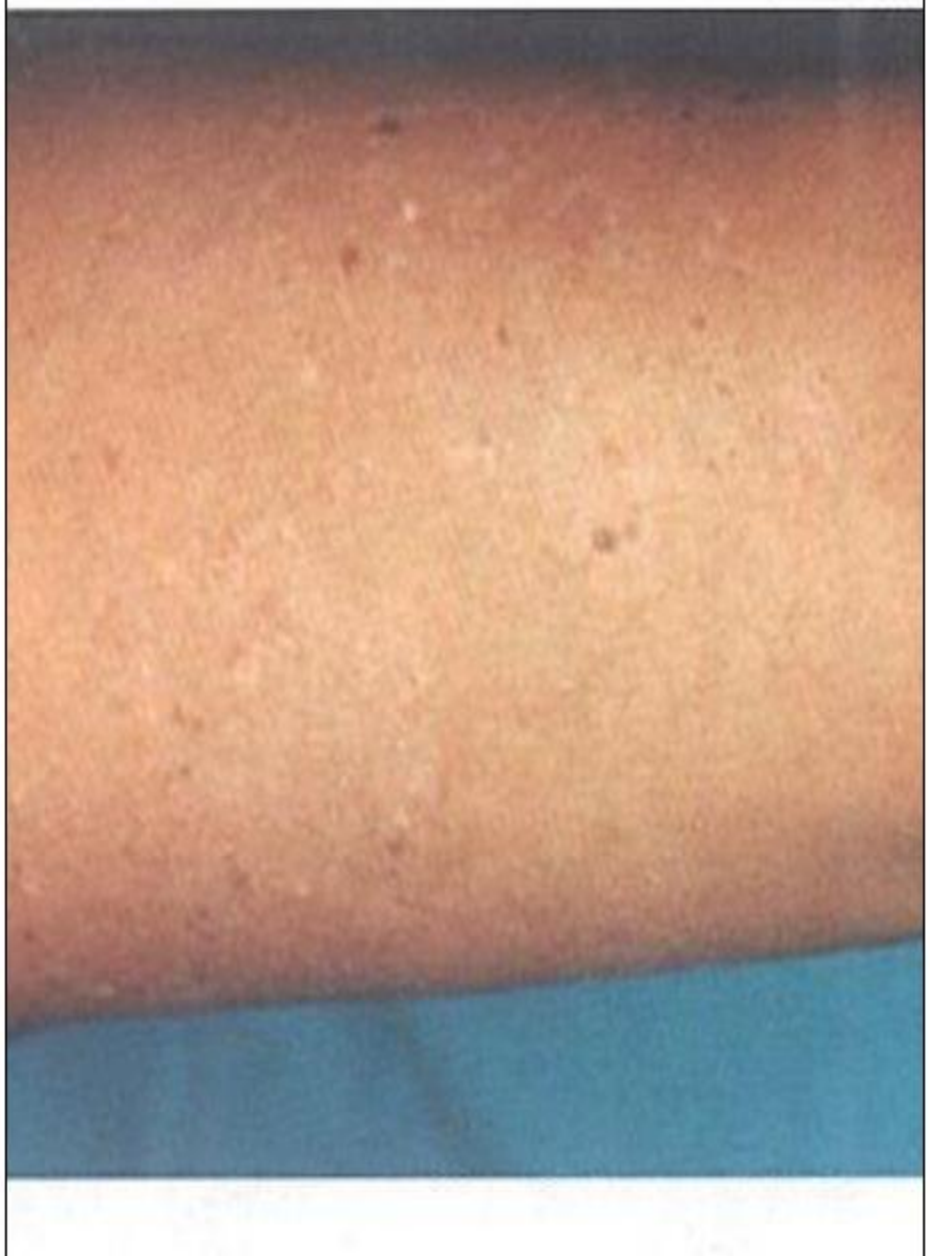
Surgical Rx: before and after

Steps of surgery

1

2

3



7. Depigmentation (bleaching agent)

A. Conditions : widely separated (Universal, over 50 % of the body)

B. Side effects :

   A. sensitivity to the sun

   B. osteoporosis

→ Universal

ثانيا : Albinism المهق

- autosomal recessive disorder

-lack of pigment production by melanocytes in the epidermis, hair bulb, and eye.

-skin is white or pink

- the hair is pale blonde

- iris is translucent.

Albinism

-Sunlight is very poorly tolerated, and sunburn and photophobia are common symptoms.

-Nystagmus

-Albinism is a serious condition.WHY?

-skin ages prematurely

- high incidence of malignant skin tumors.

Notes:

- So regular follow up With dermatologist is Required مهمه
- If the country is tropical or subtropical the main problem would be skin cancer due to sunrays
- If the country has temperate climate the main problem would be ocular due to poor vision

Albinism pt has : Squamous cell carcinoma

Albinism pathology

Melanocytes are present in the basal layer of the epidermis

-tyrosinase negative albinism

-tyrosinase positive albinism

Pathology: notes

- Melanocytes are present in the basal layer but the function is abnormal IMP!
- The melanocytes can be:
  - Tyrosinase positive: enzyme is present but poorly functioning
  - Tyrosinase negative: enzyme is completely missing
- Can be differentiated by incubating plucked hair bulbs in vitro with tyrosine or DOPA
- Tyrosinase : is the enzyme which controls the synthesis of melanin in melanocytes

management

Sun Avoidance

Total Sunblock

Regular clinical review for early diagnosis of skin tumours.

Children with ocular albanism may have learning difficulties due to poor vision and require specialist opthalmological supervision. مهمه

ثالثا : Chloasma (Melasma)

- hormonally stimulated increase in melanogenesis

-mainly affects the face

-seen in pregnant women and those on the contraceptive pill.

-Sunlight and Oral contraceptive will aggrevate the situation

Treatment:

Sunscreen

Bleaching creams

Chemical peeling

- **Hormonally-stimulated increase in melanogenesis.**
- **The** melanocytes number is the same but there is increase in the pigmentation, melanogenesis.
- Mainly affect the : Face, checks, eye.
- **Predisposing factor:**
  - Pregnancy: Usually it spontaneously improved after pregnancy in most cases.)
  - OCP حبوب منع الحمل – مهمه.
  - Darkly pigmented skin (rarely found in fair skinned)
  - Sun exposure.
- **Treatment:**
  - **sun block**
  - **bleaching**
  - **stop the Predisposing factors (OCP for ex)**

Elsevier 2004. Habif: Clinical Dermatology 4E - www.clinderm.com

الجزء الثاني من الدرس

HAIR DISORDERS

- HAIR LOSS(alopecia)

  alopecia areata

  androgenetic alopecia

  telogen eff.

  anagen eff.

  trichotillomania

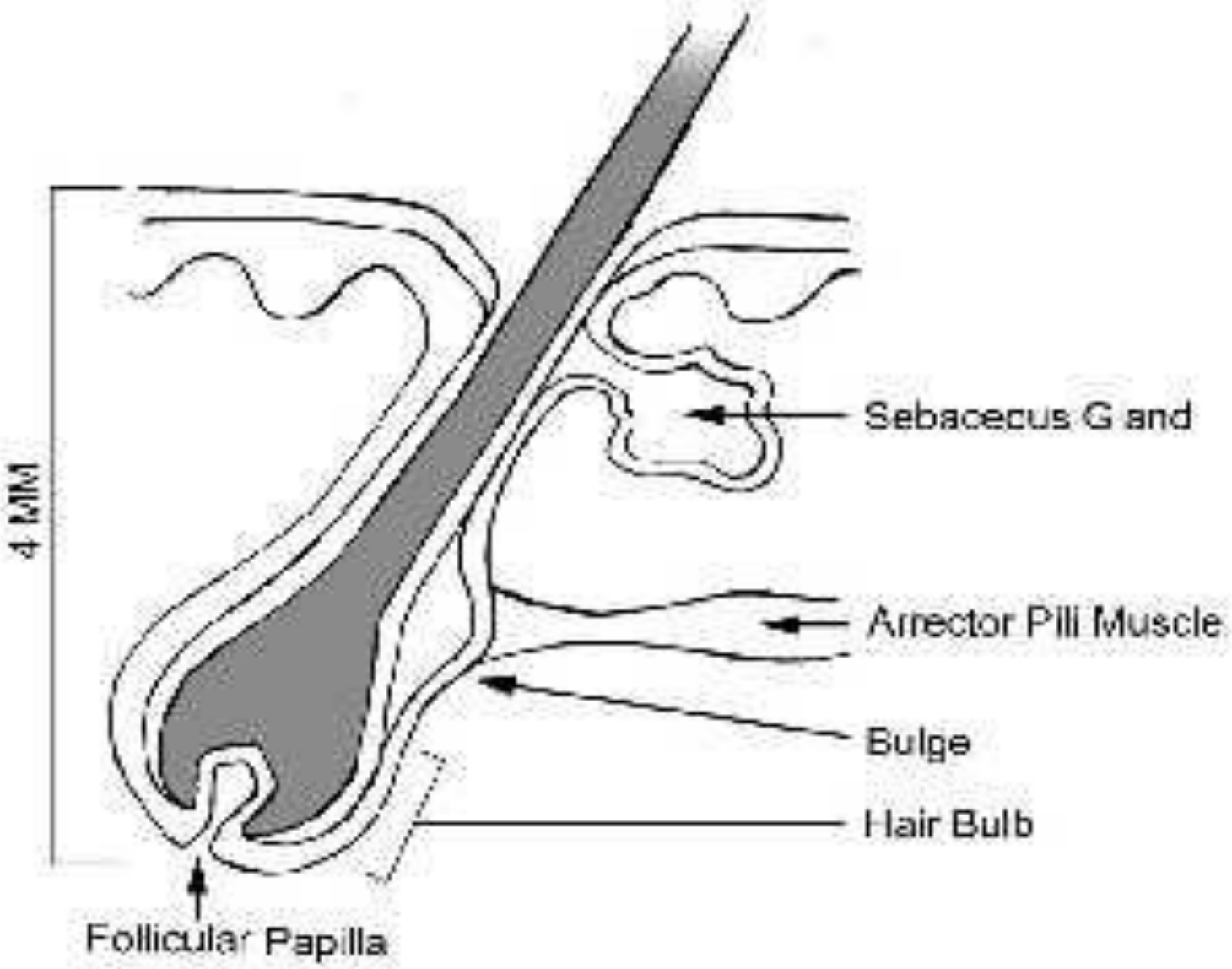
- HAIR EXCESS

  hirsutism

  hypertrichosis

Hair Structure:

- The hair matrix is in the hair bulge. It contains the melanocytes that determine the hair color.
- When the arrector pilli muscle contracts the hair becomes straighter and the hair follicle becomes perpendicular to the skin. (The hair stands up).
- **The hair is divided in 4 sections:**
  - The **infundibulum extends until the opening of** the sebaceous gland.
  - The **isthmus is from the gland to the bulge**
  - The **bulge is our target in laser hair removal,** because it's the *main regeneration site.*
  - The **lower follicle.**
  - The hair **bulb is also responsible for the** regeneration of the hair. If it was destroyed there won't be any more hair. (also a target in hair removal)


4 MM
Sebacecus G and
Arrector Pili Muscle
Bulge
Hair Bulb
Follicular Papilla

Hair Types: مهمه – لفهم الامراض

1. **Vellous:**
   A. in the whole body
   B. bright in color
   C. thin, and short.
2. **Terminal:**
   A. Thick hair like the hair on the:
   B. scalp, eyebrow, axilla.
3. **Androgen dependant terminal:**
   A. In the androgen dependant areas
   B. axilla, groin.

- The vellous hair is thin. The terminal & the androgen dependant terminal hair are both thick, dark & can be long. The terminal hair & the androgen dependant hair only differ in their site.

- ✓ **How many hairs in the body?**

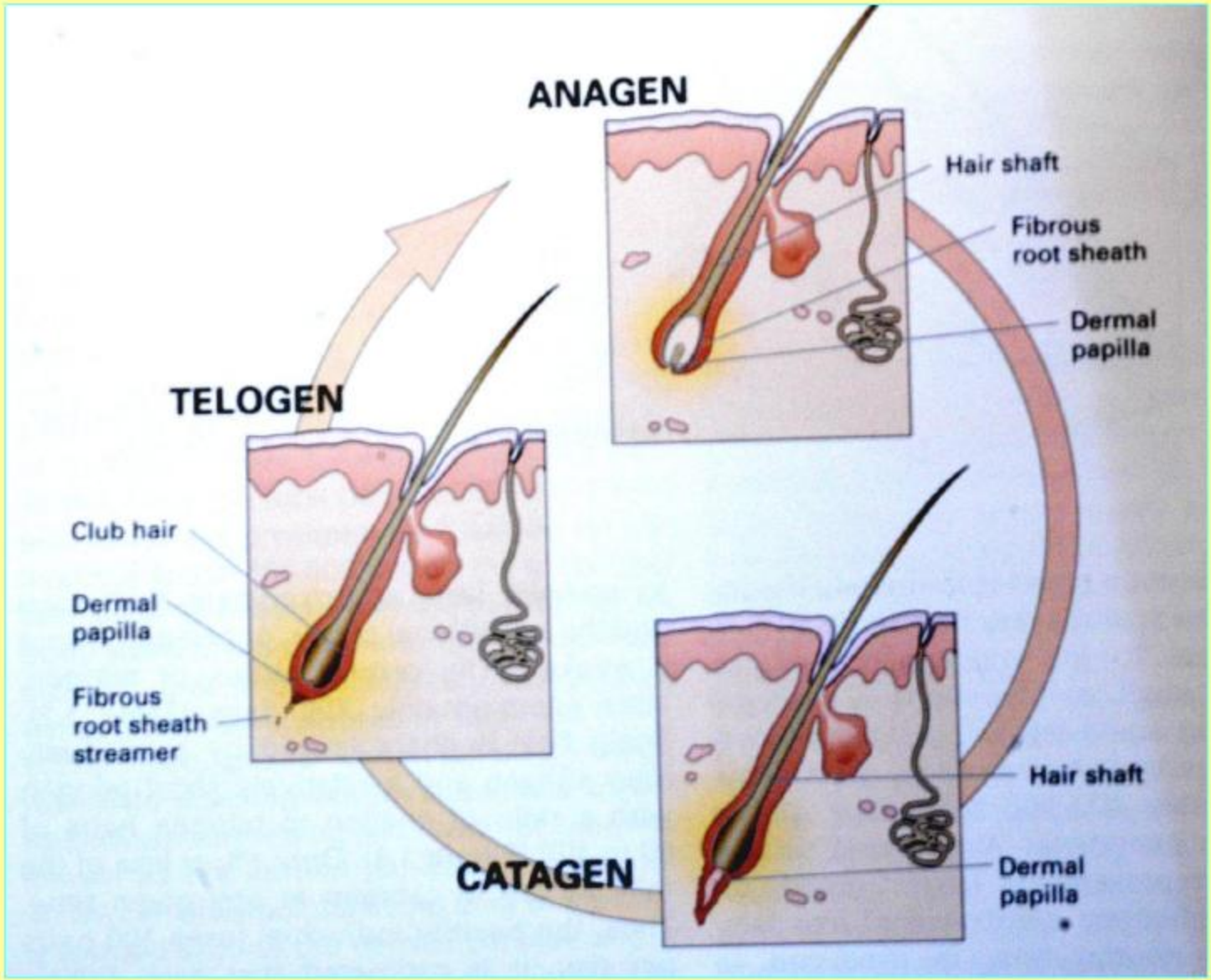
  5 millions hairs;

  100,000 in the scalp

- ✓ **Growth rate?**

0.3mm/day for scalp hair

The hair cycle مهمه جدا لفهم الامراض



The Hair Cycle:

- The hair cycle is divided into 3 stages:

1. **Anagen (growth phase):**
   - **70-80% of the hair follicle is in this phase.**
   - The length of this phase determines the length of the hair
   - If this phase is long.. let's say 4-5 years the hair will be long.. while if it was short.. let's say 1 year it will be short
   - **the average is 3 years and it differ from area to area but here we are talking about the scalp .**

2. **Catagen (resting phase):**
   - **5% of the hair is present in this phase (the least).**
   - **It last for 3 weeks**
3. **Telogen (shedding phase):**
   - **15% of the hair is in this phase**
   - **shortening of the hair &** the epidermal papillea atrophies.
   - And then the hair starts to grow in the Anagen phase.
   - It last for 3 months and normally the person loss 150 hair a day

So remember 3 years(anagen), 3 weeks(catagen), 3 months(telogen)

- **Note: When the hair falls you can see that its clubbed in the end, this is normal shedding,** If there was no clubbing, then this is immature shedding

- → So any disturbance in this cycle will result in the hair disease

Alopecia

Non-Scarring (Reversible)

Scarring (Irreversible)

ALOPECIA:

1. **Non-scarring alopecia (reversible):**
   - **The skin is normal, so there is hope for the hair to** grow again and it is treatable.
   - It is called non-scarring because there is no fibrosis.
   - In this type the hair follicles are Intact

هذا النوع هو اللي سيفصل بالمحاضره

2. **Scarring alopecia (irreversible):**
   - **There is fibrosis and sever inflammation,**
   - **there is no** hope for hair re-growth
   - In this type the hair follicles are damaged

NB : How to differentiate between them clinically ?

By looking for (hair ostia مسام الشعر) by the scope → if they intact then the alopecia is Non scaring and vise versa

مهمه

causes

Nonscarring alopecia	Scarring alopecia
Telogen effluvium	Developmental defects (e.g., Aplasia cutis)
Anagen effluvium	Infections (bacterial, viral, fungal)
Alopecia areata	Trauma (irradiation, thermal or caustic burns)
Androgenetic alopecia	Neoplastic disorders
Hair shaft abnormalities	Lichen planus (lichen planopilaris), lupus erythematosus, morphea, scleroderma, sarcoidosis
Trauma (e.g., traction)	Keratosis pilaris atrophicans
Infectious disorders (e.g., dermatophyte, syphilis)	Folliculitis decalvans
Systemic diseases (e.g., thyroid, systemic lupus erythematosus, iron-deficiency anemia)	Dissecting cellulitis of the scalp
Intoxications (e.g., vitamin A, Bismuth)	Acne keloidals
Nutritional deficiencies (e.g., zinc, biotin)	Pseudopelade
Medications	Alopecia mucinosa

Cont, the causes

Common causes of Non-scarring alopecia: كلها ستفصل لاحقا

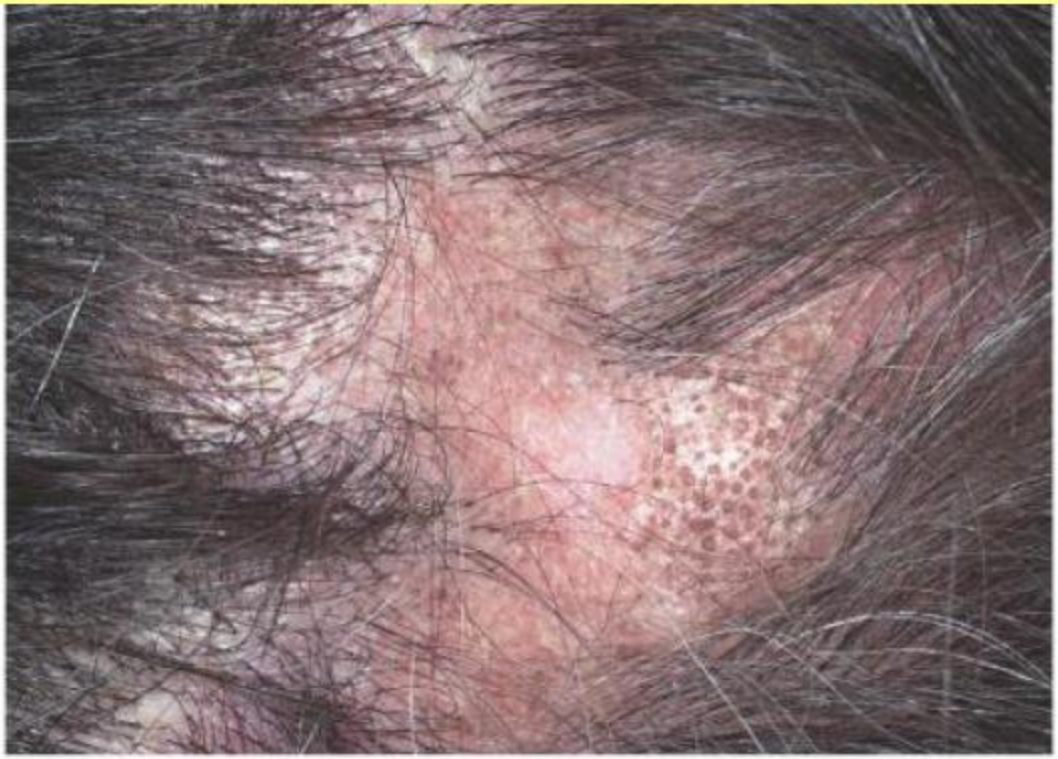
- **Telogen effluvium تساقط الشعر في مرحله التساقط من دوره الشعر: post-pregnancy, post-medication.**
- **Anagen effluvium تساقط الشعر في مرحله النمو من دوره الشعر: It occurs after chemotherapy.**
- **Alopecia Areata: Loss of hair in a well defined area (الثعلبه)**
- **Androgenetic alopecia: more in male.**
- **Hair shaft abnormality: Due to a genetic disease.**
- **Trauma: Some people when they are in stress they pull their hairs.**
- **Infectious disorder: e.g. Tinea pedis could be scarring or non-scaring (more usually). If there is** inflammation, then there is scarring.
- **Infections: Severe infections.**

Cont,

Common causes of scarring alopecia:

- **Neoplastic disorders: Basal Cell Carcinoma.**
- **Trauma: 2nd & 3rd degree burns.**
- **Lichen planus: It's a common cause of scarring hair loss.**
- **In lupus erythromatosus: 1st** there is generalized hair loss.
- In discoid lupus there is scarring alopecia.
- Regarding Morphea, it's a C.T disease. It resembles scleroderma (localized scleroderma).

scarring



اولا

Androgenetic Alopecia

(Male and Female Pattern Hair Loss)

Features

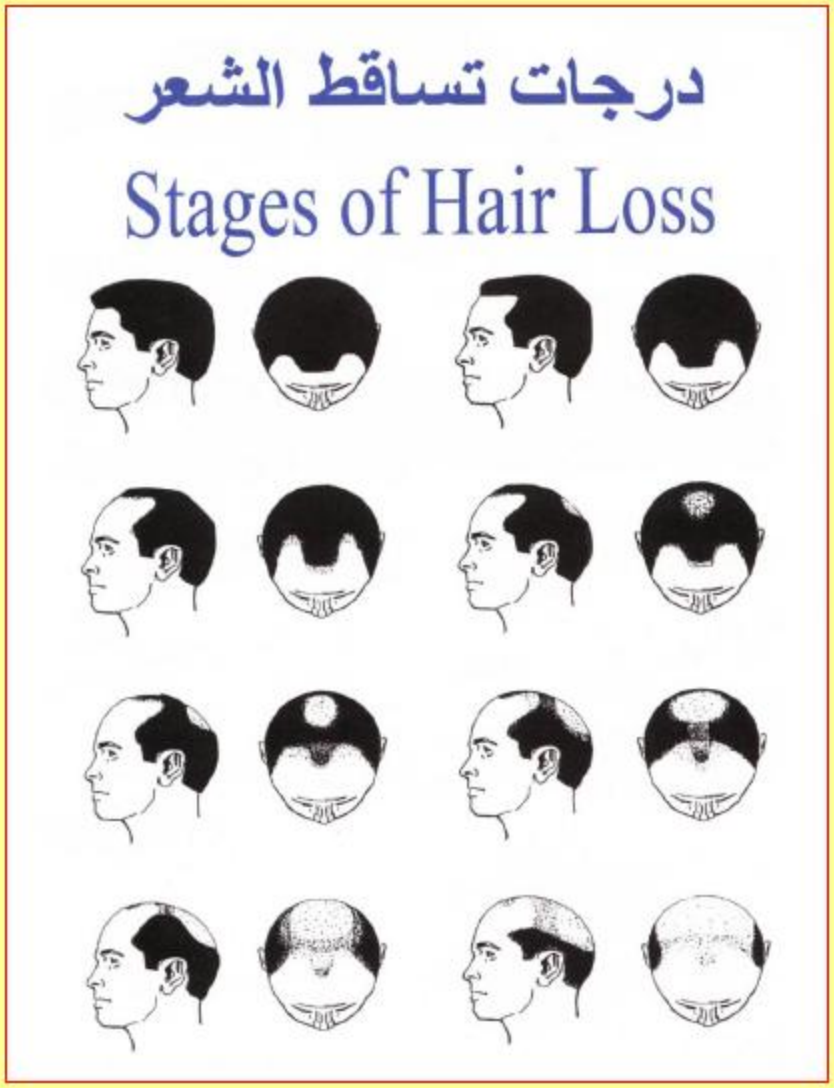
- It’s an androgen dependant hair loss.
- Androgen dependent loss of scalp hair, for e.g. the crown area.
- Androgenetic Alopecia affects up to 50% of males (and they end with baldness) and 40% of females (they rarely end with boldness; instead they end up with thinning).
- Autosomal dominant with variable penetrance.
- 85%: +ve family history

Male pattern hair loss:

- It starts with thinning, it's called fronto-parietal recession and then it goes upwards.
- Frontal recession may occur too.
- it usually spares the Temporal and occipital areas
- In males: the last stage is complete baldness

Male Pattern Hair Loss

It has 7 stages

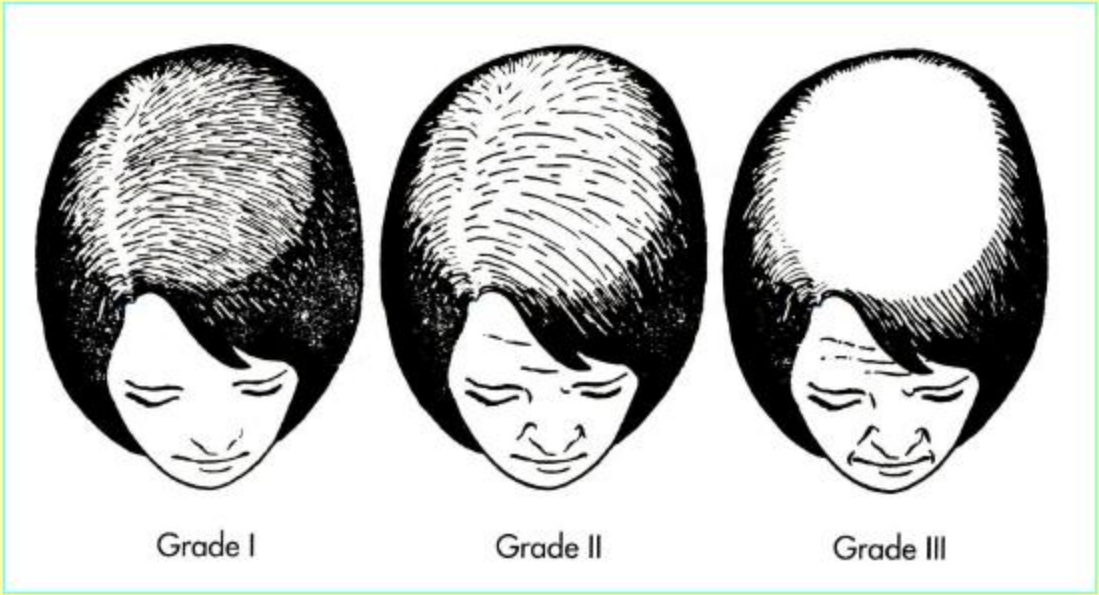



Male Pattern Alopecia

Female pattern hair loss:

- There is no fronto-parietal recession and no frontal recession, so the frontal hair line الشعر بين الجبهه وبدايه فروه الراس is preserved.
- In the female: There is never complete boldness, there is thinning.
- It more common in post menopausal women

Female Pattern Hair Loss



Female Pattern Alopecia

Pathophysiology :

- **The androgen is the key pathogen in this disease as following :**

Testosterone $\xrightarrow{\text{5 ALPHA Reductase}}$ Dihydrotestosterone (active)

- The Dihydrotestosterone causes Miniaturization of Terminal Hairs. it becomes thin and then falls
- There is localized hypersensitivity to the normal circulating hormones.
- In this condition the hair in the androgen dependant area becomes very thin.
- You can't see it but if you come closer you will find thin tiny hair.
- To treat this condition we have to block dihydrotestosterone, by using antiandrogens; e.g. Minoxidil. We either block it locally or systematically

Treatment

- Lifelong treatment unlike Alopecia Areata

A. Topical:
  - Local blockage
  - Neoxidil (monoxidil; drug for HTN)
  - 2% for females and 5% for males
  - Sometimes in females if 2% isn't working we use for them 5%.
  - It may cause hair growth in the face if it was applied on it, so we instruct the patient to be cautious and apply close to the scalp only. Used twice per day.
  - Regarding females it shouldn't be used during pregnancy
  - Side effects:
    - excess hair in unwanted areas
    - irritation on hair sites

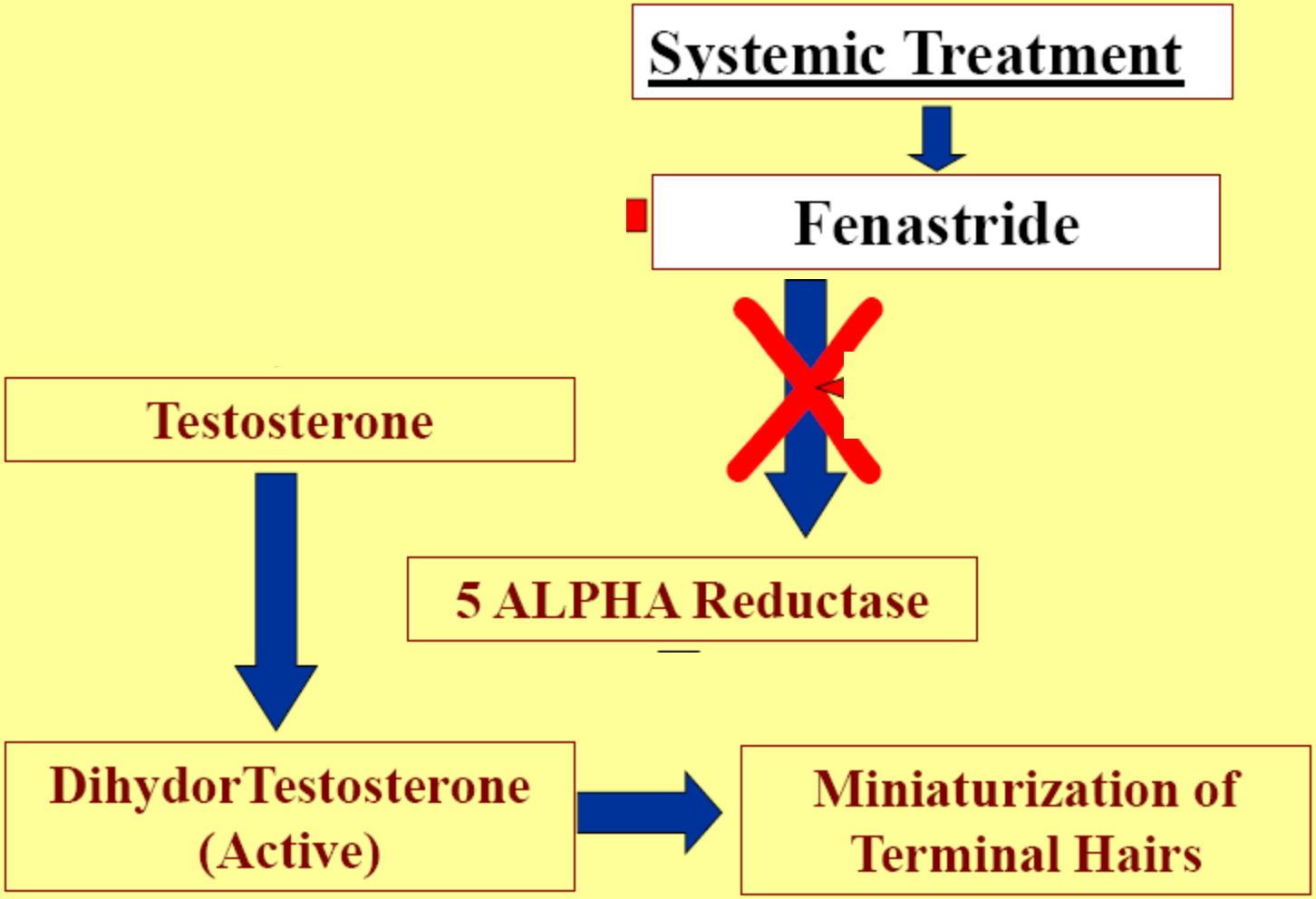
Minoxidil 5%

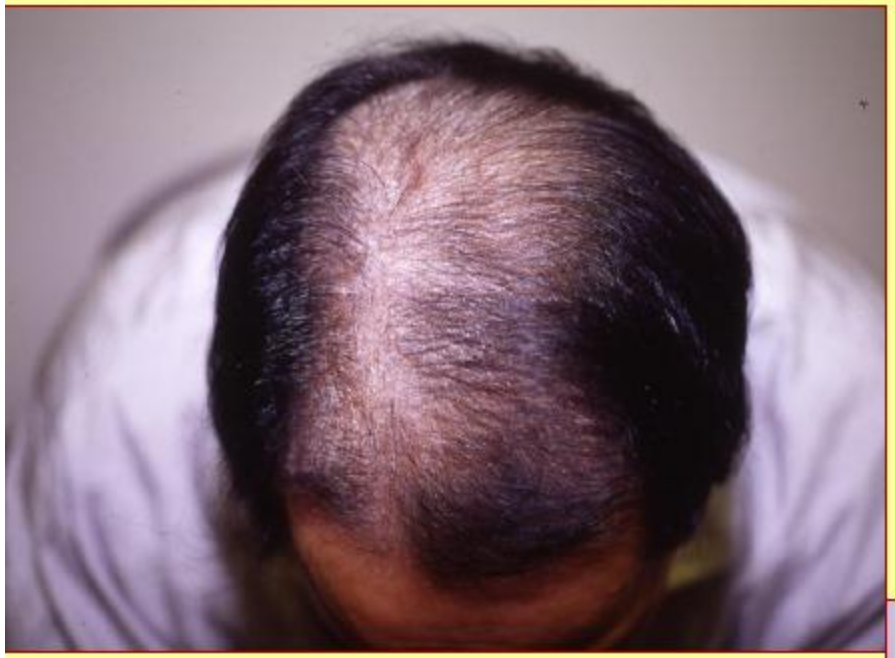
Minoxidil 2% and 5%

Side effects

Treatment: continued

B. Systemic:
   - Fenestride:
     - It is a 5-alpha reductase inhibitor
     - We can use is it as adjunct to other drugs
     - it makes the hair thicker and makes it grow faster
     - **This drug is more effective in the parietal area – less in frontal مهمه**
     - Side effects:
       - **in male : feminization**
       - **in female : the pregnant with male infant only → (it affect the gonads of the infant) مهمه**
   - Spironolactone: Has an anti-adrenergic effect.


Systemic Treatment
Fenastride
Testosterone
5 ALPHA Reductase
DihydorTestosterone
(Active)
Miniaturization of
Terminal Hairs



Fenastride

Treatment: continued

C. Surgical:
   - Hair transplant which is done under local anaesthesia
   - Procedure : take the hair from the (Occipital area) and then implant it on the (frontal area)
   - It is time consuming
   - Before doing it check :
     - density of the hair in the donor area
     - laxivity of the scalp
   - There are 2 major methods of hair transplant : توضح بالصور
     - Taking long (25 cm) with (1cm) thin hair line from the donor area and then send it to the lap to be cut then implant it on the receiver area
     - Take single hair – one by one - and then implant it on the affected site (you can take the hair from any site in the body)

1- Taking long (25 cm) with (1cm) thin hair line from the donor area and then send it to the lap to be cut then implant it on the receiver area

هذه الطريقه تتضمن شق وخياطه

Cut In the lab

2- take single hair – one by one - and then implant it on the affected site (you can take the hair from any site in the body)

مهمه جدا :
اثناء نقل الشعر تزرع شعره الى شعرتين في المنطقه الاماميه وثلاث الى اربع في المنطقه الخلفيه

قبل وبعد

ثانيا :

Alopecia Areata

Alopecia Areata:

- A condition characterized by either generalized or localized sudden hair loss from the scalp or other body sites
- Common: It affects up to 2% of the population.
- 75%: Spontaneous self recovery (usually in 2-6 months).
- It can affect any age, but most cases are children or young adults

Causes (multifactorial):

- Psychological stress can be a predisposing factor.
- 30%: +ve Family history (genetic background).
- Autoimmune مهمه:
  - The strongest
  - because there is inflammation and the immune system will attack the hair follicle

Clinical findings: مهمه

- Well defined and demarcated alopecic patches.
- Exclamation point:
  - characteristic around the hair follicle.
  - Normally the hair is thick near the follicle and it becomes thinner as we go up, in this case the hair is thin near the follicle resembling an exclamation point → **!)**
- Normal scalp: no inflammation or scarring. مهمه
- Nail: pitting, ridges. مهمه

فيه التهاب لذلك هذه ليست ثعلبه

Types of Alopecia Areata:

1. Localized partial:
   - 1-2 patches.
   - complete recovery
   - most common type
2. Localized extensive:
   - 5-6 patches and could reach 10.
3. Alopecia ophiasis:
   - At the periphery of the scalp, for e.g. in the occipital region
   - Having this type of alopecia is a bad prognostic factor
4. Alopecia totalis: total hair loss in the scalp.
5. Alopecia universalis:
   - The whole body is affected (e.g. no eyelashes, actually NO hair at all, all over the body).

Bad prognostic factors: مهمه

- Young age.
- Atopy. (**very important)**
- Alopecia totalis, universalis, ophiasis.
- Nail changes: because it indicates that the effect of the disease is very bad.
- Loss of eyebrows and eyelashes
- These are very bad prognostic factors but it doesn’t mean a patient with one of these factors or more than one that their hair won’t grow again.

Diagnosis:

- Clinically: spot diagnosis, an area with no hair and the skin under it is normal.
- History and examination (pathology):
  - swarm bees; collection of lymphocytes around the hair follicle.
  - Very characteristic. In alopecia areata.
  - Woods lamp is used to exclude fungal infections

Treatment:

- The aim of the treatment is to enhance the hair growth.
- We either give steroids (to reduce the inflammation and suppress the immunity because it is an autoimmune disease) or skin sensitizers (it introduces inflammation to enhance the hair to grow).
- N.B/ If there are 1 or 2 patches, we don't need to treat it because it will resolve spontaneously.
- The method that **we should not use is: Hair Transplant, because the immune** system will attack the new hair and it'll fall

1. Observation:
   - because in most cases the hair will re grow spontaneously.
   - do it if the lesion small
2. Intralesional corticosteroid:
   - It's the drug of choice in adults.
   - We can't give it to children b/c it's painful. So, we give them topical corticosteroids coated under occlusion.
3. Skin Sensitiziers: Anthraline, Diphencyclopropenone (DPCP) and others. مهيجات للجلد تسبب نمو الشعر
4. Topical Steroids: under occlusion
5. Systemic Steroids
   - If the case was acute or an extensive case.
   - We only give it for a short period of time
6. Cytotoxic treatment: immunosupressant
7. Phototherapy: Narrow band – UVB – PUVA
8. Minoxidil: acts through hormonal web, it makes the hair thicker and the hair growth faster, it is used as an adjuvant treatment.

ثالثا :

Anagen effluvium:

Anagen effluvium

- Always related to cytotoxic chemotherapy.
- The hair falls prematurely and all of it.
- Acute and severe alopecia.
- Mostly reversible but not always.

رابعا :

Telogen effluvium:

Telogen effluvium

- Chronic alopecia.
- Any stress whether it's physiological or pychological can cause it-anemia-chronic diseases.
- There won't be complete loss of hair.
- Reversible (but may be become chronic)
- It takes 3-4 months for the hair to fall & may take months to re grow again. For e.g. a pregnant lady delivered her baby…If her hair is going to fall, it won't fall instantly, it will take about 3-4 months then it will fall (it occurs due to the drop in the estrogen level).

- To differentiate between telogen effluvium and androgenetic alopecia you part the hair form the midline equally, if the hair loss is the same in the frontal and in the occipital area this is Telogen effluvium, if the hair loss is more in the occipital area than the frontal area this is androgenetic alopecia. → TE does not follow regular pattern and it affect the whole scalp equally

Causes:

- Physiologic:
  - Physiologic effluvium of the newborn
  - Postpartum effluvium مهمه
- Injury or stress:
  - High fever
  - Sever infection
  - Sever chronic illness.
  - Major surgery
  - Hypo- or hyperthyroidism
  - Crash diets, precipitous decrees of calories or protein.
  - Iron deficiency.
  - Essential fatty acid deficiency.
  - Biotin deficiency.
  - Drugs.

Postpartum effluvium

- During pregnancy blood oestrogen levels are high
- This causes the percentage of hair in Anagen phase to rise and hold the hair in anagen phase
- After delivery the oestrogen drops and the hair enters the telogen phase which causes it to shed
- Seen 4-9 months after delivery and is self limiting

Drugs associated with Telogen Effluvium:

ACE inhibitors e.g., captopril,[215] enalapril[216]
Albendazole[217]
Amphetamine[218]
Aminosalicylic acid[219]
Anticoagulants e.g., heparin,[220] warfarin[221]
Antiepileptics e.g., carbamazepine,[222] valproic acid[214,220]
Beta blockers e.g., metoprolol,[223] propranolol[224]
Bromocriptine[225]
Cimetidine[226]
Danazol[227]
Interferons[228] e.g., interferon α
Levodopa[229]
Lithium[230]
Oral contraceptive pills – during or after discontinuation[214]
Retinoids e.g., etretinate[231] and excess vitamin A[214]
Pyridostigmine[232]

Treatment:

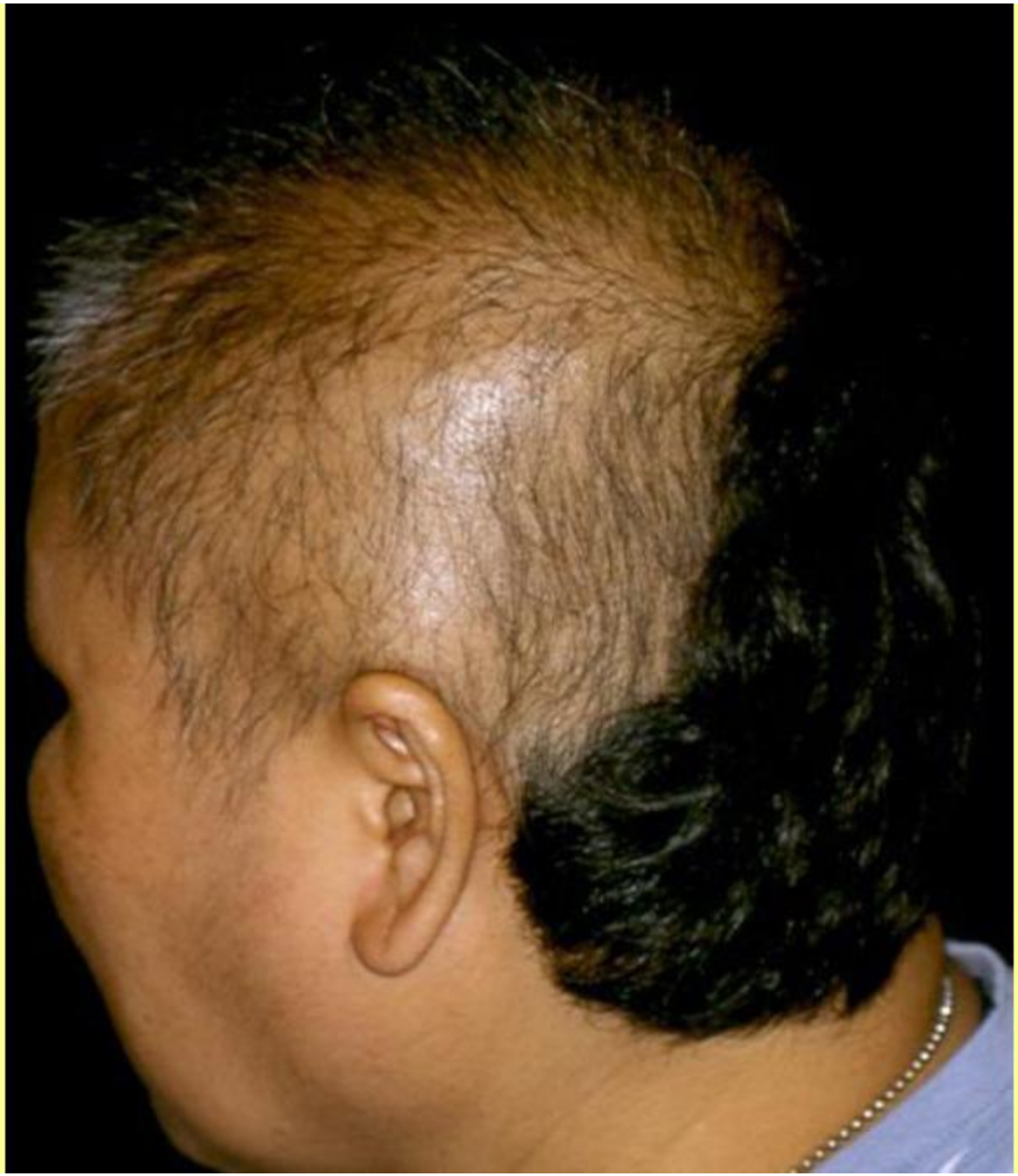
- Remove or treat the cause: For e.g. if the patient had anemia and you didn’t treat it, her hair will keep falling.
- Minoxidil 2% solution to enhance hair growth.

خامسا Trichotillomania

- Obsessive compulsive disoder
- Pulling of hair
- Asymmetrical areas And usually follow bizarre and irregular pattern
- Treatment:

  Behavioural therapy

  SSRI



Diagnosis :

At clinical ex : The hair will be with different lengths as shown in the pic

Biopsy : Will show hematomas around the hair follicles

سادسا :

Fungal Scalp Infections(tinea capitis)

Child had contact with animals.

-Scaly or inflammed alopecia or Kerion

- siblings may also be affected.

-Wood's Light

-Scalp scrapings and hairs should be examined microscopically, and cultured to demonstrate the fungus.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96 9 9
UBC Dermatology. http://www.derm.ubc.ca

Fungal scalp infections

Treatment: Only SYSTIMIC treatment مهمه جدا

-Oral antifungal(griseofulvin or terbinafine or itraconazole)

-Hair regrow normally unless a kerion has led to scarring.

سابعا :

Chronic Discoud lupus

-patchy and scaly alopecia ,

- follicles plugged by scales and erythematous advancing margin.

- scaring process => permanent alopecia

مهمه جدا : اذا تقرحت تكون دائمه

-Treatment of early lesions

الجزء الاخير من الدرس

Hirsutism

-Hirsutism is excess growth of androgen-dependent hair in a male pattern

- Hypertrichosis is excess growth of hair in non androgenic pattern.

Seen in both sexes but hirsutism is restricted to females. مهمه جدا

The different between Hirsutism and Hypertrichosis is So imp

Facial hair in female

Hirsutism

In female pt hair grows in the beard area, around the nipples, and in a male pattern on the abdomen.

-Hirsutism can cause great distress to a healthy woman and lead to psychological disturbances with depression.

Causes مهمه

Adrenal: Cushing's syndrome, virilizing tumours, congenital adrenal hyperplasia

Pituitary: acromegaly

Ovarian: Plycystic ovaries, virilizing tumours, gonadal dysgenesis

Iatrogenic: due to androgenic drugs

Idiopathic: Target or end-organ hypersensitivity(the commonest cause) t

Investigation

To exclude the underlying, treatable causes

-Although many tests of adrenal, ovarian, and pituitary function are available, the young woman who is menstruating regularly, and has had one or more successful pregnancies, requires little endocrine assessment.

Full endocrine assessment is, however essential in patients with amenorrhoas, scanty, irregular periods, or with signs of excess androgen stimulation.

Signs of virilization?

treatment

Laser Hair removal

Diane Oral contraceptive that has antiandroginic effect

Hypertrichosis

-Either congenital or acquired

-Congenital associated with melanocytic naevi, while lumbosacral hypertrichosis (faun tail) should alert the pediatrician or obstetrician to the possibility of spina bifida occulta.

مهمه جدا : يجيك سؤال ويقولك طفل عمره شهرين وعنده اعراض المشكله هذي

-Acquired drug induced

Endocrine disorder like thyroid dysfunction

Anorexia nervosa

Drugs

Hypertrichosis

Drug causing hypertrichosis include : diazoxide diphenylhydantoin, penicillamine, and the psoralens.

If the offending drug is withdrawn, the excessive hair growth will cease.