Dermatology Team 441

MED441
KING SAUD UNIVERSITY

BH
Bassam Alhubaysh
REVIEWED BY

Pigmentary & Hair disorders

Objectives:

- Common hair disorders, physiology, presentation, investigation, and management
- Common cutaneous pigments disorders, physiology, presentation, investigation, and management

Color index:

- Main text
- Important
- Dr's explanation
- Golden notes
- Extra

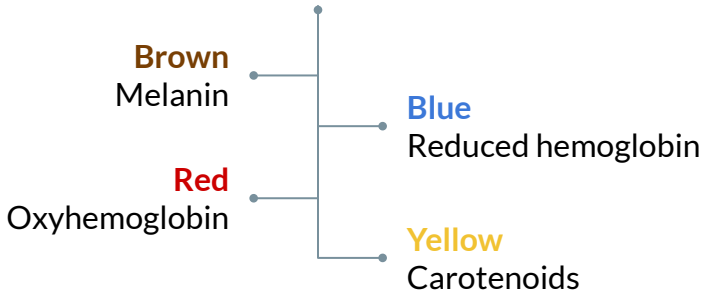
Dermatology Team 439

DERMATOLOGY TEAM 438

This lecture was originally done by both 438 & 439 teams.
So great thanks to them

Pigment Disorders

Skin Pigment:



THERE ARE 6 DIFFERENT SKIN TYPES				
PHOTOTYPE	HAIR	SKIN	TENDENCY TO BURN	TANNED
I	Red Hair	Milky	Constant high	Null
II	Blonde Hair	Light	Constant medium	Mild
III	Brown	Light	Frequent	Clear
IV	Dark Brown	Matt	Infrequent	Dark
V	Very Dark Brown	Matt	Exceptional	Very dark
VI	Black	Black	No	Black

Human skin color is classified according to Fitzpatrick skin phototype

Pigment disorders are divided into:

1. **Hyperpigmentation:** increase of melanin in the epidermis.
2. **Hypopigmentation:** decrease of melanin in the epidermis.
3. **Depigmentation:** loss of melanin in the epidermis.

Definitions:	
Melanocytic hypermelanosis:	An increase in the number of melanocytes in the epidermis (melanin) (an example Is lentigo)
Melanotic hypermelanosis	No increase of melanocytes but an increase in the production of melanin only (an example Is melasma)
Melanopenic hypomelanosis:	a decrease of the production of melanin only (an example is albinism).
melanocytopenic hypomelanosis:	a decrease in the number or absence of melanocytes in the epidermis producing no or decreased levels of melanin (an example is vitiligo).

Pigment Disorders

Hyperpigmentation

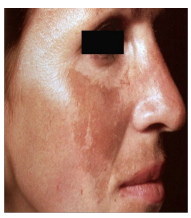
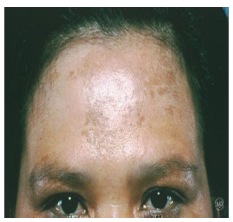
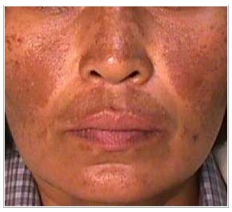
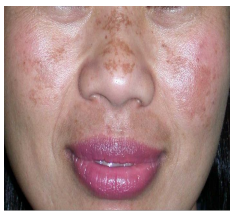
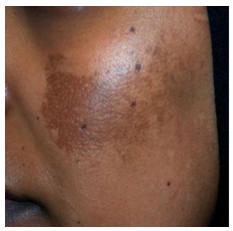
Melasma
كلف

- Common acquired skin disorder Presents as a **bilateral, brownish facial pigmentation.**
- Acquired symmetrical blotchy hyperpigmentation mostly on face mostly on cheeks
- More common in women than male, Mostly in young females(20-40), only 10% males
- Overactivity of an increased number of melanocytes.
- Epidermal, dermal, mixed (most common) only melanin no increase of melanocyte
- Present as **sharply marginated macules and patches with irregular borders on cheeks and forehead.**

Risk factors: Genetic predisposition (Indian), excessive sun exposure, pregnancy, oral contraceptives can trigger the disease.

Treatment:

- Sun protection
- Kligman's formula: Hydroquinone whitening + Tretinoin peeling + corticosteroid to decrease inflammation but steroid could cause hyperpigmentation
- Hydroquinone 4% cream
- Glycolic acid, azelaic acid, kojic acid
- Chemical peels: glycolic acid, TCA, phenol, resorcinol faster better
- Fractional laser

Post inflammatory hyperpigmentation (PIH)

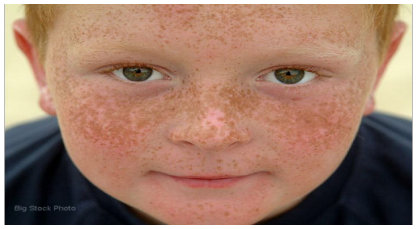
- Any inflammatory disease can cause it, e.g. Acne, eczema, psoriasis (more severe ,deep with lichen planus) , trauma, laser hair removal, burns, etc. some time after trauma you get the burn hypopigmented and border hyperpigmented
- Improve with time but may persist for years.
- Treatment as melasma.

Freckle (lentigo)
نمش

- Overactivity of an increased no. of melanocytes.
- Common in **fair-skinned people,** especially in children.
- Sun exposure in genetically predisposed individuals.

Treatment:

- **Sun block**
- **Pigmented laser**
- bleaching cream usually not useful because lentigo is increase in cell number

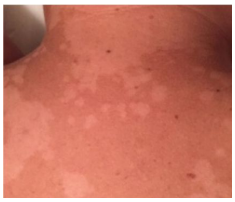



Pigment Disorders

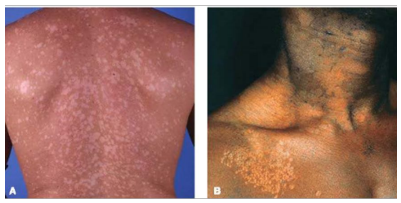
Hypopigmentation

Post Inflammatory Hypopigmentation

- Could happen after any inflammatory dermatosis.
- Pityriasis versicolor hypopigmentation (fungal infection).
- Post chemical peel or laser
- Post intralesional corticosteroid injection common



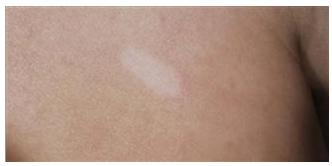
Treatment:
Make up, Tattoo, Excimer laser, NB-UVB to Simulate melanin production



Depigmentation

Nevus Depigmentosus

- Congenital, solitary depigmented patch
- Cutaneous mosaicism with altered clones of melanocytes with decreased ability to produce melanin
- Stable not going to increase in size
- no risk of koebner phenomenon
- Mostly in trunk and extremities

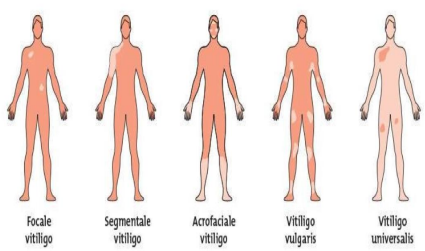


Treatment: make up, tattoo, melanocyte transfer excimer laser not useful because no melanocyte to stimulate it

Vitiligo البهاق

Clinical Features

- A chronic autoimmune disease with genetic predisposition.
- Complete absence of melanocytes. Immune system attack melanocyte
- Incidence 1%.
- Early onset (50% before the age of 20, 80% before the age of 30).
- Rarely could be associated with:
  - alopecia areata
  - thyroid disease
  - pernicious anemia
  - diabetes mellitus.
  - Repigmentation in hair follicle
- Ivory white macules and patches with sharp convex margins.
- Could affect skin, hair, retina, but Iris color no change.
- Koebner phenomenon. (If you scratch the skin in the active phase you will get new lesion, happens in psoriasis and lichen planus).
- Types:
  - Focal. Topical treatment
  - Segmental. dermatomal distribution
  - Generalized (commonest).
  - Trichrome: light brown, hypopigmentation.
  - Acral.
  - Vulgaris it is common around eye ,knee ,elbowe
  - Poliosis: white hair. زي الشيب
  - Universal (>90%). We treat it by inducing more hypopigmentation
  - Affect the eye (retina) need ophthalmologist to see it iris color is normal



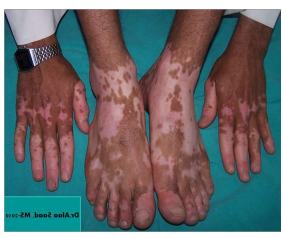
Pigment Disorders

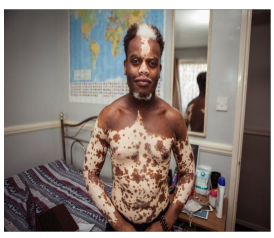
Depigmentation

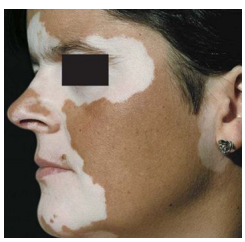
Vitiligo البهاق

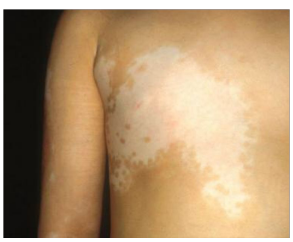
Diagnosis	• Diagnosis usually clinically. • Wood's lamp for early vitiligo & white people. • Skin biopsy? Pathology shows normal skin with no melanocytes.

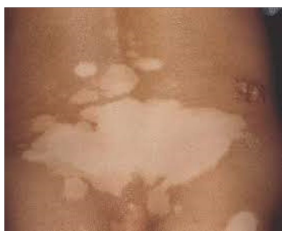
Management	• General measures:
	  ○ Sun protection: sun-avoidance, clothes, hats, sunscreens..etc. because hypopigmented skin is more sensitive to sunlight
	  ○ Make up. It is a must
	  ○ Tattoo.
	  ○ Psychological Support. We have to educate the patient and community that it is not always genetically transfer to their children
	• Focal disease:
	  ○ Topical corticosteroids.
	  ○ Topical calcineurin inhibitors e.g. tacrolimus.
	  ○ 8-MOP topical phototherapy.
	  ○ Excimer laser.
	  ○ Localized NB-UVB.
	  ○ Surgical (stable disease for 2 years) : melanocytes transfer, blister graft, punch graft.
	• Generalized:
	  ○ NB-UVB.
	  ○ Oral PUVA.
	  ○ Systemic therapy: oral corticosteroids, methotrexate, cyclosporine, mycophenolate mofetil, azathioprine.
	  ○ Depigmentation e.g. with 20% monobenzylether of hydroquinone cream If more than 90% of skin affected Induce hypopigmentation by destroying melanocyte only in universal
	  ○ Depigmentation with Q-switched laser and cryotherapy

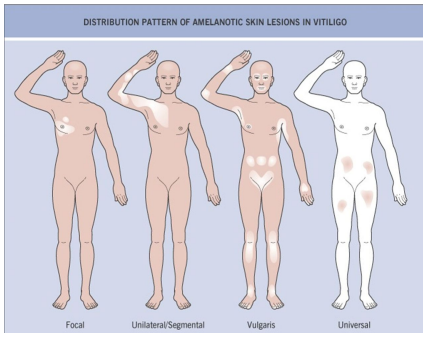












Disorders of hair follicle

Hair follicle cycle

Anagen

- Growth phase. Hair length depend on the age genetically related
- Determines the length of the hair (e.g. scalp hair: 2-6y, arms: 2-3 m, eyelashes: 1-6)
- 85% of hair .
- 2-6 years.
- Hair length increase 1 cm every month
- Chemotherapy affect this stage and cause baldness

Catagen

- Transition phase between anagen and telogen (apoptosis driven).
- 1% of hair.
- 1-3 weeks.

Telogen

- Resting phase.
- 5-15%.
- 3-4 months.
- Affected in post partum does not cause baldness

Exogen

- Active shedding hair.
- Hair follicle not lost with every hair loss

Types of Hair:

Lanugo hair

Fetus, shed before birth

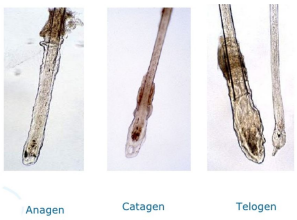
وبر.

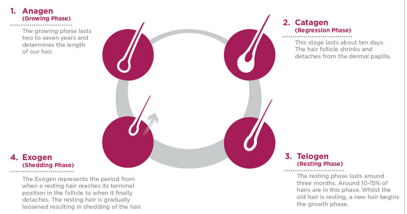
Vellus hair

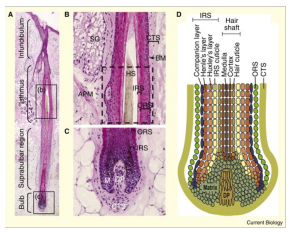
Fine, non-pigmented hair.
On hand and legs

Terminal hair

Thick pigmented hair found on scalp, eyebrows/eyelashes,beard, axilla, pupic area and its growth is affected by hormones. Scalp is affected by hormone

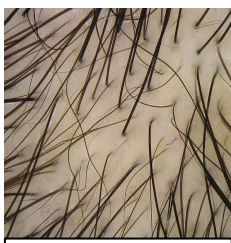






Diagnosis
(Hair evaluation methods)

- Hair pull test: + 6 is positive. If you pull 60 hair and 5% of them fall .positive test
- Trichogram: 50 hair pull for anagen/telogen hair ratio (painful procedure not used now).
- Trichoscopy (Dermatoscope): we see hair follicle and scalp , this is what is used now for the diagnosis.
- Scalp biopsy.
- Scanning Electron Microscopy.
- 120 hair loss daily is normal



Androgenetic alopecia

Alopecia Classification:

Non cicatricial (No scarring)/ alopecia

- No clinical sign of tissue inflammation, scarring, or atrophy of skin.
- Examples: Male/Female Pattern hair loss, Alopecia Areata, Telogen Effluvium, Anagen Effluvium, Trichotillomania.

VS

Cicatricial (scarring)/ alopecia

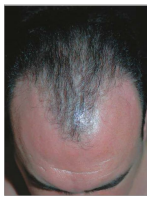
- Evidence of tissue destruction such as inflammation, atrophy, and scarring may be apparent. Ex: lupus, lichen planus

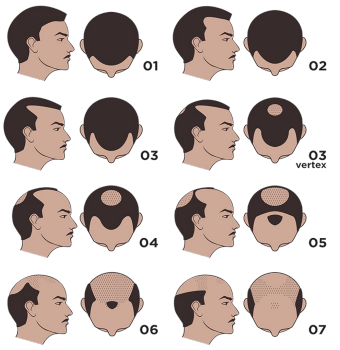
Hair Disorders

Non cicatricial alopecia

Male Pattern Hair Loss

- Most common type in adult men.
- Genetic predisposition (Autosomal dominant) and Androgen hormones (Androgenic alopecia).
- Susceptibility genes inherited from both mother and father.
- Genetic sensitivity of hair follicle receptors to Dihydrotestosterone (DHT).
- DHT decrease anagen phase from years to months or weeks.
- DHT is regulated by 5 alpha reductase.
- Testosterone → (5alpha reductase I&II) → DHT.
- Type II alpha reductase present in Scalp & beard hair, seminal vesicle, prostate, epididymis, scrotum.
- Present as receding hairline and hair loss on frontal area frontoparietal recession ,temporal recession .thinning of vertex. With time will be completely bald.
- Hamilton Classification (used in diagnosis and treatment, We start hair transplant at stage 5, before that we use Drugs).
  - Anagen lost, hair space on vertex is bigger
  - No scar no atrophy
  - Air age decrease .thin hair it is not loss مو تساقط





Management:

- Minoxidil solution 2% and 5% liquid or foam (better) S.E: Itching, Irritation.
- Finasteride 1mg/d (type II 5 alpha reductase inhibitor)
- Dutasteride 0.5mg/d (type I & II) Not FDA approved more potent and more side effects.
- Hair transplant (in severe cases). Redistribution take hair from back to front
- Hair piece wig , Tattoo, powder.
- Others: PRP, Low-level laser therapy.. etc

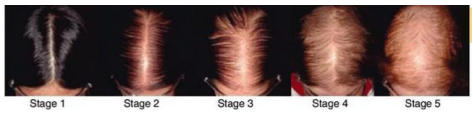
Female Pattern Hair Loss

- 40% of women ages 50 has some hair loss.
- Diffuse thinning of hair due to shedding and decrease volume.
- Begins at the vertex mainly over the crown but NEVER goes bald, usually with preservation of the frontal hair.
- Genetic predisposition, polygenic, either parent.
- Usually Normal androgen level.
- More common after menopause, ? Estrogen stimulate hair growth
- Polycystic Ovarian Syndrome (PCOS) , Congenital Adrenal Hyperplasia (CAH)

Investigation: Trichogram, DHEAS, Prolactin, Free testosterone, LH/FSH, CBC, iron, Ferritin, TIBC, thyroid function test, Scalp biopsy.

Management:

- Minoxidil 2%, 5% (may cause hypertrichosis on face and neck).
- Finasteride
- Dutasteride.
- Spironolactone
- Flutamide.
- Cyproterone acetate ,lasix (furosemid)
- hair transplant
- Others: PRP, Low-level laser therapy.. Etc
- Cosmetics: Hair piece,hair spray, tattoo, powder.
- Anti androgen drug is contraindicated in pregnancy (category X)should be stopped one month before pregnancy



Hair Disorders

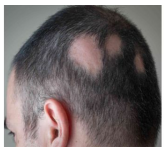
Alopecia areata (الثعلبة)

Pathogenesis

- Autoimmune disorder, with T-cells around hair follicles
- Genetic predisposition, 10-20% positive family history.
- Affect males and females at any age (80% before age of 40).young population
- Lifetime risk is 1–2%.
- It is histologically characterised by T cells around the hair follicles.
- Association with vitiligo, thyroid disease, atopic dermatitis and Down syndrome.
- Triggers could be viral infection, trauma, hormonal changes, severe emotional stress.
- You have to differentiate between associations and triggers.

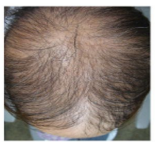
Types

- Localized alopecia areata.
- Alopecia totalis: all scalp hair.
- Alopecia universalis: whole body.
- Ophiasis: occipital and lateral scalp. Bad prognosis
- Diffuse alopecia areata. Diffuse thinning not lost in specific area
- Patchy Alopecia Areata (Most common type):
  - Patchy hair loss of scalp, beard, eyebrow, eyelash hair.
  - Sudden onset.
  - Regrowth of white hair then pigment comes back.
  - Nail pitting and ridging in 10-50% of patients.
  - Exclamation marks are 2-3 mm broken hair with distal end broader than proximal at the margin of the hairless patch.
  - Scalp is healthy no inflammation

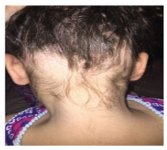


Localized AA

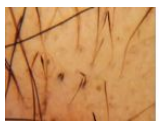
Totalis or universalis



Diffuse AA



Ophiasis



Management

- Intralesional / topical corticosteroids. Because it is autoimmune inflammation
- Minoxidil.
- Anthralin.
- Diphencyprone (DPCP). Immuno sensitiser fake inflammation to bring T-cell away from hair follicle
- Phototherapy.
- Systemic corticosteroids, pulse therapy.
- Immunosuppressants: e.g. Methotrexate, azathioprine..
- JAK inhibitors (Tofacetinib, Ruxolitinib).
- Cosmetics: Artificial eyelashes, eyebrow tattoo, hair piece.. etc

Prognosis

- Single patch: 80% resolution in 1 year.
- Poor prognostic factors: **(Super important)**
  - Extensive disease. Totalis universalis
  - Duration >1 year. After treatment
  - Ophiasis pattern.
  - Nail involvement.
  - Childhood onset.
  - Positive family history.
  - Other concomitant autoimmune diseases.
  - Atopy.
  - Down syndrome.

Hair Disorders

Telogen Effluvium (تساقط الشعر الكربي)

Clinical features

- Temporary hair loss of telogen hair. Diffuse
- System shock: change anagen hair to telogen.
- Vit D, iron deficiency
- Diffuse hair fall, but in pattern hair loss it is more on the crown.
- Might take 2-4 months after shock to start losing hair. Not sudden
- Usually last for 6-9 months with incomplete recovery.
- Could be chronic, but doesn't cause complete baldness Because telogen hair represent only 5-15% of total hair.
- Hair pull test is positive in opposites to hair pattern loss

Causes

- Postpartum
- Fever, surgery with general anesthesia, childbirth, severe emotional trauma, **severe weight loss (bariatric surgery)**, high fever (Covid-19), Vit D, iron deficiency
- Drugs: Heparin, warfarin, B-blockers, ACE-inhibitors, lithium, anticonvulsants (especially valproic acid).

Diagnosis

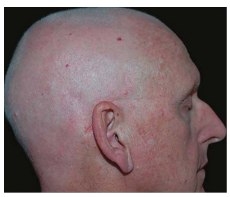
- HAIR PULL: +ve with reduced percentage of anagen hair.
- CBC, Serum iron, iron-binding capacity and Ferritin.
- TSH to Rule out thyroid disease.
- Vitamin D level
- Zinc
- B 12
- Histology: swarm of bees
- Even if she is postpartum we have to look for other reasons.

Treatment

- Treat the cause
- Minoxidil 2%

Anagen Effluvium

INFO



- Onset is usually rapid and extensive. Could be a week or less

Etiology:

- Chemotherapy with alkylating agents.
- Radiation therapy to head.
- Intoxications.

Pathogenesis: occurs after any insult to the hair follicle that impairs its mitotic/metabolic activity.

- Regrowth is usually rapid after Discontinuation of chemotherapy.

Scarring Alopecia

- Lichen planopilaris LPP.
- Frontal fibrosing alopecia: post menopausal women. european
- Central centrifugal cicatricial alopecia. in african american rare in ksa
- Discoid lupus erythematosus of scalp.hypo\hyper pigmentation and no hair follicle found
- Traction alopecia from hair style
- Trichotillomania when repeated
- Acne keloidalis nuchae.
- Kerion (tinea capitis). Fungi have pus in it

Quiz!

1- A 25-year old male presented to the dermatology clinic complaining of hair loss. On examination, there were 2 well-defined hairless non-scarring smooth patches over the occipital area of his scalp. What is the most likely diagnosis?

A) Alopecia areata
B) Anagen effluvium
C) Telogen effluvium
D) Androgenic alopecia

2- which of following hair phases is affected the most by chemotherapy?

A) Anagen
B) Telogen
C) Catagen
D) Exogen

3- What's the best initial therapy for a localized vitiligo?

A) Methotrexate
B) Phototherapy
C) Topical steroid
D) Tacrolimus

4- A 45 year old female presented with decreased hair density over the vertex in non-scarring alopecia - The anterior hairline was preserved- Which one of the following is the most likely diagnosis ?

A) Telogen effluvium
B) Traction alopecia
C) Female pattern hair loss
D) Alopecia areata

5- which of the following is not poor prognostic factor in Alopecia Areata?

A) Family History
B) Adolescent onset
C) Duration >1 year
D) Nail involvement

Answers:
1:A, 2: A, 3: C, 4: C 5: B

Thanks!!

DERMATOLOGY TEAM 438

Dermatology Team 439

438 Team leader:
Mohsen Almutairi
Lama Alyahya

438 Done by:
Abdullah Alnuwaybit
Mohammed Alqahtani
Rema AlMutawa
Sarah Maghribi

439 Team leader:
Mohammed Albabtain

439 Done by:
Mohammed Akresh
Saud Alrsheed

438 Academic leader
Saud Bin Queid

439 Academic leader
Hamad Almousa

This lecture was updated by: 441 Academic Leader *Bassam Al Hubaysh*

Dermatology Team 441

MED441
KING SAUD UNIVERSITY

BH
Bassam Alhubaysh
REVIEWED BY