بسم الله الرحمن الرحيم ..

نضع بين أيديكم تلخيصا لراوندات مادة الجلدية مع الدكتورة ختام الرفوع والدكتور عوض الطراونة راجين من الله القبول وأن يجعل فيه النفع والفائدة ..

ولأن من لايشكر الناس لا يشكر الله فكل الشكر لأفراد الفريق الأكاديمي للجنة الطب والجراحة على جهودهم العظيمة في تفريغ وطباعة وتدقيق هذا العمل ونخص بالذكرالطالبة أسماء قاسم والطالب حمزة وادي نسأل الله لهم ولكل من ساهم التوفيق والنجاح.

normal skin & principle

** primary lesions :

- papule
- pastule
- nodule
- plaque
- macule
- patch
- vesicle
- bullous
- comedon
- burrow
- wheal
- cyst

** secondary lesions :

- erosion
- ulcer
- atrophy
- scar
- lechinification
- scratch / excoriation
- fissure
- crust / scale

** Plaque : change of texture

Ex: psoriasis

_____

** Macule : discoloration , less than 0.5 cm

No changes in texture

_____

** Patch : same as macule
But
More than 0.5 cm

_____

** Wheal :
Transient, Erythematous, edematous.

Ex: urticaria

_____

** Burrow: charactarestic of scabies

Primary lesion of scabies

_____

** Skin atrophy : Decreased skin marking
V.S
Lichenification : exaggerated skin marking , indicates chronic eczema

…….

-Ulcer : all thickness ,with scar

Erosion : superficial , no scar

- Keratenization: 28 day from basal to horny layer

-configuration :::

*disc shape lesion = discoid lupus

*target lesion = erythema multiforme

*annular lesion = tenia

+ biopsy :

-diagnosis of inflammatory diseases : psoriasis

-neoplastic disease

- for treatment plan

**** Wood light**
- T.capitis: green
- T.versicolor : yellow (ill defined margins)
- Erythrasma: coral red colour
- Vitiligo: milky white (well defined)
- Melasma : - if it is superficial (epidermal : dark brown)
  - If it deep dermal .... ???
- Pityriasis alba : negtive

acne vulgaris

** Acne vulgaris
• Age : adolescent
• Site : face , upper back and upper chest
• Asymptomatic (no pain)
• Characterized by comedons :if comedon white head >> new lesion ,if black head >> old lesion

** Commedons are primary lesion of acne vulgaris

** Factors related to the ACNE formation:
- hyperkornification (means increased keratin production) of the pylosebaceous duct
- increased sebum production due to increased androgen (testosterone)

- hormonal factor
- propriobacterial acne that forms the pus

** acne:
-the presence of commedons is nessesary if there is no commedons we think about folliculitis

** Don't leave it to be treated alone ,it will leave scar of pigmentation

**Acne is related to :
-PCO (poly cystic ovary)
-vit B12

-steroids : the main drug
-vit D -omega3

** Azelaic acid (used to treat acni) : tyrosinase inhibitor cause hypopigmentation

** white head comedon (not black one) : can progress to papule , pastule and other inflammatory lesion

** monomorphic acni occur in drug induced acne : steroid and B12

** most common cause of late onset acni (mature acni) :
* drugs 👆
* PCO

** skin manifestations of PCO : Acni , hairsutism , hair loss

** T - shaped involvement of face by acni occur before menses

** pseudofolliculitis is a DDx of acni (important)

** **roaccutane (vit. A derivative) for acni** :

* not for pts less than 18 years : إلا مع مرافق

* vitamins are contraindicated with it
* exercises are contraindicated ,rabdomyolysis
* blood giving is contraindicated

** side effects :

* dry skin , lips and eyes

* dry nasal mucosa , epistaxis

* mild alopecia

* general arthralgia and myalgia

* hyperlipidemia : may cause pencreatitis

* alter liver function test : pt , ptt and albumin

* teratogenic : the major most important one

* drug interaction with vit.A derivatives
* over dose cause folliculitis
* depression

** any married female take roaccutane ----> she have to have two methods to prevent the pregnancy

** If you want to give roaccutane you have to check ---> KFT, LFT, CBC , lipid profile

** Treatment :

1. topical :

* antibiotic for anti-inflammatory role : tetracycline , erythromycin , clindamycin

* retinoid : the best

* benzoyl peroxide

2. systemic :

* antibiotic : doxycycline

* retinoid : roaccutane

* cyproterone acetate

**Treatment :

1) mild(commedons only) : topical treatment
2) moderate(papulopatular) : topical + oral antibiotic
3) sever(nodulocystic) : oral retinoid

*best systemic treatment is roaccutane ,but not the first choice.

** total dose of roaccutane = 120 * body weight

** Systemic Treatment of acne with retinoids as contraindicated in pregnancy as it may cause teratogenicity

** Acne variants:

- late onset acne (the patient needs to do investigations as at may be due to polycystic ovary , you can also ask this patient about hirsutism and other symptoms of polycystic ovary)
- infantile acne

- acne fulminans:

usually associated with internal systemic disease, male,young,need systemic treatment ,associated with systemic symptoms

- acne-cosmotica
- drug-induced acne

-indication for roaccutane :

#more than 3 months not responding

#gram -ve

#scar tendency

#acne fulmenent

#psychological

#nodulocystic acne

#other areas than face

** Rosacea : is not a variant of acne it’s a separate disease
Its risk factors include :
-female
-fair skin
-age (between 40-50)
Rosacea shows erythema but without commedons

** Facial erythema differential diagnosis:
1-lupus (upon exposure to sun)
2-rosacea
3-photosensitivity rash
4-seborrheic dermatitis

** Steroids are prevented in all variants of acne and rosacea

** side effects of topical steroid
- atrophy
- fungal infection
- telengectasia
- acne

bacterial infection

** Types of cotanous TB:
1.scrofuloderma
2.lupus vulgaris
3.warty tuberculosis
4.tuberculides

** Impetigo
- is a bacterial infection and it is contagious and classified into bollus infection and non-bolous infection
- There is Crust lesion(mixture of scale and fluid) which is acute (for 1 week)

** Impetigo : honeycomb crust (characteristic)
** the difference between cellulitis and erysipelas:
- Erysipelas : superficial streptococcus infection \well defined \localized .

-while cellulitis is deeper /ill defined /spreading

*M.c.c of bacterial folliculitis >> staph and psuedomonas.
*only Non-bolus impetigo is associated with PSGN .bzc bolus is caused by staph .
*M.c site for carboncle >> occipital area
*Scarlet fever : sand paper rash , strawberry tongue , perioral rash , pastia lines

** Impitigo in children less than 9 months may progress to bullous impitigo then to 4S syndrome (staphylococcus scalded skin syndrome)

For impitigo : -antibiotic :amoclan -fucidic acid

** erythrasma:
- on wood's light appear as cordal red colour

*DDX of axilla infection: -erythrasma -eczema -..

-DDX of 3rd&4th nail infection : -fungal T.pedis -psoriasis -erythrasma -psodomonas

** in patient with cellulitis > check for tinea pedis or insect bite

fungal infection

** tinea pedis
_ is the most common
_it is the source of tinea cruris and the other types because it expands

** tinea have active margin and more common in male

** annular lesion (clear from inside)
*causes
_ tinea cruris
_ annular psoriasis

_erythema marginatum

** tinea unguium
_affect on nail then transmit to the adjacent nails
_but it's not necessarily to affect all nails

** Candida : present normaly

Risk factor :
1) immunocompromized
2) diabetic
3)use systemic antibiotic and steroids
4)sweating

*manifestation of mucocutanaes Candida
1)oral thrush
2)angular chelitis
3)chronic paronychia
4)intertigo (satellite lesion of Candida is important for diagnosis)

*Candida diagnosis .... KOH its fungi

*Wood light .... geenish fluresance for diagnosis some types of tenia capitis and no rule in dx of Candida

*hypopigmented of area post infection

** Tenia versicolour Not infectious

** floconazole taken after sweating for tratment of tinea vercicolor

*Tinea versicolor is the only type of tinea not caused by dermatophyte , its caused by malassezia .

** Dx of tinea vercicolor :
* stretch test , appear scaly
* KOH : it will disolve keratocyte so I can see the fungus
* woods light
* may do culture but it need long time

** no role for topical steroid in tinea , bacterial , viral infection

** indiciation for systemic antifungal treatment in tinea :
* nail involvement : tinea angiuum
* hair involvement : tinea capitis
* tinea incognito

** tinea is one of DDx of pytrias alba

** Tenia versicolor : treated because this fungus produce azeliac acid which is tyrosinase inhibitor and this lead to depigmentation

** Fungel infection expanding
** Tenia cruris in groin (koh test) chrecitrised by erythematous scaly lesion with expanding margin
** Teni pedis , tinea angiuum (onycho mycosis)fungl infection
** Koh to prove fungel infection

** Intertrigo inflammation of any skin folds (axillary \ breast skin folds) (presence of satellite lesions)

** Acute paronychia --> causative organisms --> bacteria
** Chronic paronychia---> causative organisms --> candida (fungi)

-most common risk factor of chromic paronychia is moist environment.

** Tinea incognito : is the tinea that result from maltreated tinea

** Tinea Capitis : more common in children
Hair loss
erythematous sclap

Dx by -hair pulling >6 , woods light ,KOH

Kerion : type of tinea capitis that caused by cattle ringworm:
antifungal+ antibiotic

** Tinea unguium :
-proximal type (start from proximal nail fold then spread distally)
- distal type (start from distal part of the nail and spread proximaly)

** tinea capitis >>> scaly scalp
- inflammatory type with red scalp
- non inflammatory type without erythema

viral infection

-HSV1 carrier 85% ,sub-clinical

- HSV1 vs. HSV2 by serology

-we treat HSV1 at the first day with antiviral(valaciclovir) , if the patient came at the next day we treat him with antibiotic

aciclovir	valaciclovir
High dose	Low dose
nephrotoxicity	- no nephrotoxicity
Low absorption	Absorption 90%

- dose of oral valaciclovir = dose of IV aciclovir

-If with HZV was more than one dermatome & hemorrhagic = think of malignancy

** Presence of black dots on the warts represent thrombosed vessels this indicates regression of warts.

** Liner distrubition grouping vesicles dermatomale distribution : herpes zoster
** Herpes simplex : genitleis , lebialis

** herpes simplex best treated by valacyclovir than acyclovir

** erythema multiformis ass. with HSV
** in zoster pain precede rash and still after it

- molloscum contagiosm in genitalia :think of child abuse!

- male with warts ,if he is engaged we should give his fiancee(خطيبته) vaccine and if he is married that means his wife has the virus also.

** Plane warts best treatment is no treatment

_____

** Planter warts, when pared away"_اكشط الجلد فوقها": pin point bleeding.

.

** Calluses and corns when pared away no bleeding.

** we do always PAP smear in women with HSV , HPV (important)

** Koebner phenomenon occur in :

1_psoraisis
2_lichen planus
3_plane warts

** Orf : due to parapox virus : infected fresh meat by this virus cause disease in person

Abscess presentation on one finger

** Thrombosed blood vessel indicates warts

** Vesicle on palm and soles , no vesicles on the abdomen or back >> Hand foot mouth disease

** reactivation of herpes simplex may be anywhere

** we differentiate between type 1 and 2 herpes simplex in genitalia by serology as IgG in 1 and IgM in 2 (type 1 may occur in genitalia)

** if she is affected , pregnant women has type 1 usually

** if zoster infect other human it cause chickenpox

** addmession for pts of zoster if :

* occur in neck or above

* involve multiple dermatome : usually haemorrhagic and ass. with underling malignancy in elderly

** if high fever (may be with seizure) followed by rash after few days >> it is roseola infantum, so roseola infantum rash is not associated with fever.

Vesicles if group ➡(HSV , HZV)

All over the body ➡ chicken pox

on Palm sole ➡ hand foot mouth

** each virus and its disease
it is a Q in miniosce :

* zoster : varicella zoster virus

* warts : human papilloma virus

* Molluscum contagiosum (MC) : MCV type of poxvirus

* orf : parapox virus

* erythema infectiosum : parvovirus B19

* Roseola infantum : human herpes virus type 6

* Gianotti–Crosti syndrome : Epstein–Barr virus (EBV) & hepatitis B

* Hand, foot and mouth disease : Coxsackie virus A

** Group of vesicles at the angle of mouth >>herpes simplex

** warts vs corn:
-Warts --->have rough sarface which contain black dots that indicate thrombosed blood vessls and present at any site

-corn---> have smooth suface, skin lines are still present over the lesion(its only thick area of skin) and at pressure points

**herpes zooster
-group of vesicls
-painful
-on dermatomal distribution(linear distribution)
-there is a history of previous chikenpox infection
-it will infect others in form of chikenpox because the virus of both is varicellazoster virus
-sometimes it will cause post-herpitic neuralgia and prevented by earliar treatment

** calosity (corn) atropy poins
- central corn (dead tissue)
- skin line present (not wart)
- no pointed heamorrhage after scraping

** Chickenpox can cause :

* interstitial pneumonitis ,

* nephritis,

* encephalitis

* meningitis

scabies , pediculosis , leishmania

** Scabies:
*Itching Precedes rash ,itching at night

-burrow is the primary skin lesion : diagnostic

-genital scabitic nodules : diagnostic
*rash sites in adult: interdigital , wrist, umbilical area , nipple , axilla , genitalia.
In children: palm and sole .
* In first time of infection rash needs 4 weeks to appear while in second time it appears immediately.
* 3 agents for treatment:
1-permethrin cream for 3 days
2-benzylbenzoate emulsion
3-crotamiton
** It is important to ask about family history of itching >>scabies

-in scabies neck &back are spared.

-(11-17) mites needed to have scabies

Why to leave 1 week between the 2 doses ?

- to treat family members

- to allow eggs to hatch from the skin .

Pediculosis :

1-common :hair

2-body: poor hygiene

3-crab :STD

-White lyce then suck blood and become brown .

DDx of occipital itching : -pediculosis -psoriasis - seborrhic dermatitis .

-most common cause of scalp impitigo in childrin :pediculosis

Leishmania : -painless - imidiate rash -non itching

-papule_nodule_ulcer -no allergy

Psoriasis

** psoriasis spare central face

** nail changes in psoriasis :
* pitting : large and irregular
* onycholysis
* oil spot : yellow discoloration
* thickening
* splitting haemorrhage

** psoriasis does not respect hair line in scalp

** most dangerious types : pastular , erythrodermic

** guttate : young pt and bacterial infection characterize it

-- rain drop sign of guttate psoriasis

-acute pustular psoriasis = sterile culture .

-erythrodemic psoriasis is a serious condition.

*no way for systemic steroid in treatment of psoriasis.

-Guttate psoriasis : good prognosis , treat with antibiotic .

** psoriasis is not contagious nor inherited , it has just genetic predisposion

** all of these increase risk for psoriasis :
* infection
* stress , pregnancy ,trauma
* drugs ,antimalarial
* irritation
*hypocalcemia

** all of these provoked by psoriasis :
* cvs diseases
* renal diseases
* obesity
* hyperlipidemia

** just topical steroid in psoriasis , no need for systemic ,Because

1- Tern to pustular psoriasis

2- Tachyphylaxis ; get back

** treatment :
topical :
* emolient
* keratolytic : salisalic acid
* vit D
* vit E : retinoid
* tar
systemic : more than 10% of body surface
* phototherapy : uva , uvb , puva
* immunosuppressor , cytotoxoc
* retinoid
* biological agents

** we shift to systemic treatment when
_ there is more than 10% of total body area are involved
_or when there is failure of topical treatment ,not responding

_sever form : acute pustular &erythmodermic

_psoriatic arthropathy

** biological treatment is the last line of treatment of psoriasis

** we can treat psoriasis but it is not curative

** Describe psoriasis lesion :
Well_demarkated scaly erythematous plaque

** Koebner phenomenon occur in :

1_psoraisis
2_lichen planus
3_plane warts

** DDx of knee lesion :
1.Psoriasis
2.Frictional dermatitis

** Salmoon erythema —>in psoriasis

** Annular lesion +active margin —->tenia

** psoriasis : may cause hair loss but usually it doesnot

** psoriasis vulgaris is the most common type of psoriasis

** psoriasis more common on extensor surface

** scalp psoriasis :
-you can see and feel the scales (as a rule of thumb)
-silvery whitish dry scale on scalp
- little itching
- thicker

we have to differentiate it from :
* seborrhoic dermatitis :
- yellowish green scale
- more itchy
-lesion respect the hair line

** guttate psoriasis :
-may follow streptococcal sore throat
-particularly in young adults

** erythrodermic psoriasis :
_erythema involve more than 90% of total body skin surface (except face)
-causes : drugs , eczema ,and lymphoma.

** palmo pustular psoriasis :
- symmetrical involvement
- may be starts on one hand and after period of time appears on another hand in symmetrical form

** DDX of psoriatic arthropathy is rheumatoid arthritis

** It's not contagious but if the patient have HIV or other viruses
the virus may transmit to other people

** skin biopsy is important

** typical psoriasis: in elbow and knee

** Auspitz sign : for differentiation of psoriasis
If there is blood indicate blood supply that area and this is positive for Psoriasis.

eczema & pruritus

** atopic eczema appear in children on extensors (لما يزحف البيبي)but in young adult on flexurors

-atopy diagnosis: at least 3 major criteria

-major criteria: 1.pruritis 2.chronic 3.family hx 4.typical morphological distribution of age > children :flexures adult:extensor

Characterestics for atopic dermatitis:

-itchy -scaly -red patches -lichenification -dry skin -flexural area

** seborrheic eczema involve nasolabial folds

** Causative of sebohrreic dermatitis is (bacteriosporum ovale)…….. normal commensal on skin ,, but linked to malassezia

* in seborrhic eczema :nasolabial fold involved

- ddx for frictional ezcema is psoriasis

** hyperpigmintation in static eczema is due to haemosidrin

** hydrocortisone (low concentration)for contact dermatitis in children

** seborreic dermatitis respect hair line in scalp (in contrast to psoriasis)

** generalized itching due to skin disease : scabies , urticaria , dermatitis herpitiformis

** no stellite lesions in irritant napkin dermatitis but in candidial napkin dermatitis there are

** Patch test for contact allergic dermatitis

** pompholyx eczema : Appear in places of no sweating (palm & sole)

pompholyx eczema = dyshidrotic eczema

** Generalized puriritis
A:with primary skin lesion the cause is skin disease like cutaneous t – cell lymphoma ,licken planus ,drug induced
B : WITH no skin primary lesion or with secondary lesion the cause is systemic disease

** C fibers transmit the itching and thermal sensation so decrease in the temperature will increase the threshold of itching 10% of these fiber are histamine dependant so anti histamine not always work

** Sever itching with Brown discoloration ddx: exclude scabies

** photosensitive dermatitis:
-Type of eczema with rash called photosensetive rash
-photosensitive rash: not from birth

** irritant contact dermatitis treated with steroid for 2 weeks then stop treatment to prevent tolerance then continue

** allergic contact dermatitis
- patient has to be sensitized
- spread
- need 24 hour to appear
- patient is genetically susceptible
- amount of irritant not important

** irritant contact dermatitis
- not spread
- patient not be sensitized
- localized
- depends on the amount of irritant material

bullous dermatosis

Pemphigus vulgaris	Bullous pemphigoid
Younger patients affected	Elderly are affected
Mucosal involvement is common (Oral lesions present)	Mucosal involvement is rare (Oral lesions absent)
Antibodies against desmoglein 3 (desmosomes)	Antibodies against hemidesmosomes
Intraepidermal (superficial) blisters	Subepidermal (deep) blisters
Blisters are flaccid and rupture easily	Blisters are tense and firm
Nikolsky's sign is positive	Nikolsky's sign is negative
Acantholysis on Tzanck smear	No acantholysis on Tzanck smear
Immunoflourescence shows net like IgG	Immunoflourescence shows linear IgG
Most patients die without treatment	Prognosis is good, most patients do well
Tombstone appearance of basal layer	Eosinophilic infiltration seen on histology

** Treatment
High dose of prednisolon (60_120)mg
Immune suppressive agent

** pemphigus valgaris may ass. with malignancy

** bullous pemphigoid preceded by generalized itching

** bullous pemphigoid surrounded by erythema (characteristic)

*Bollus pimphigoid on extremities , bollus vulgaris on trunk .

skin manifestation of internal diseases

** DM: تعداد النقاط في السلايد حفظ
** DM: cause immunodeficiency which cause candidiasis (opportunistic: it is normal flora but with the low immunity in DM it grow and flare up and cause candidiasis)
** DM: granuloma annulare (is an anuular lesion "peeded lesion(peeded nodules)

** Erythema abigne: in hypothyriodism the patient has cold intolerance which will cause him to sit near a heat source(chronic exposuer to heat), which will cause the lesion,
it is also seen in DM(because of the nuropathy) so the patient does not feel the high heat

** lupus pernio: when found on the nose, ear, cheeks it's most likely from sarcoidosis with lung affection (which is known to be affected with sarcoidosis)

-lupus pernio: associated with lung disease -sarcoidosis (not lupus).

-systemic amyloidosis :needs further investigation .

- paniculitis : subcutanous tissue inflammation .

** second file, slide #11, point (7): those two diseases have high association with each other 90%

** skin manifestations of internal malignancy : مهم جدا
there is skin lesion with high risk of malignancy transformation (like the mentioned in point 7)
and there is lesion tells us that there is an internal malignancy

** patient of DM
- candida infection interdigital
- on neck >>> skin tags , not warts

** Linear diribution with gaping of lesions (insect bite)

** malignancy can suspected in :

- lanogo hypertrichosis

- malignant acanthosis nigricans

25-30% of dermatomyocitis associated with malignancy.

Tripe palms=100% malignancy .

20% of sweets syndrome = acute myelocytic lukemia

Pyoderma gangrinosum :

1-maignancy

2-inflammatory bowel disease

3-connictive tissue disease

-erythema nodosum:

-deep seated

-bilateral

-tender

-erythema

pigmentary disorders

** Melasma : hyperpigmented patches comes with pregnancy and aging .
-Treated by hydroquinone (don't use it more than 3 months) rare side effect of this drug : Vitiligo => can be treated by tacrolimus

** Lintigo ➡ senil spot come with age & sun exposure

** spitz navous diff. from melanoma by histology (biopsy)

** pityriasis albae occur due to dryness and sun exposure and need not more emollients

** Vitilligo
It is autoimmune disease so it can associated with :

- DM

- thyroid disorders

- pernicious anemia

- SLE

** vetiligo:
-Depigmented area(not hypopeigmented) white in color, no scales, inflamatory disease comes in symmetrical distribution
-D.DX:
1)pityriasis alba: in children comes after sun exposure , ill defined margins
2)post inflamatory hypopegmented area: after eczema and psoriasis

** For Caucasian sun cause :

1. Aging (early wrinkles)

2. Liable to develop skin cancer (not protected by melanocytes)

** Africans liable for melasma and not liable for cancer or aging (protected by melanocytes).

** investigations of vitiligo :

1. fasting blood sugar

2. ANA

3. Anti TPO antibodies

4. B12

5. CBC

** acanthosis nigrican :

- Familial

- Obesity

- DM : insulin resistance

- Increase androgen : cushing , PCO

- Addison's , hypothyroidism

- why postpsoriasis hypopigmentation? Because keratenocyte turnover is very rapid(4days) that there is no time for melanocyte for pigmentation .

Freckles treatment : -reassurence -sun block

Hairy nivus :30% risk of vitiligo .treatment:topical tacrolimus

Best TTT for vitiligo : narrow band

Woods light is a type of UVA and not harmful for pregnant.

hair & nail

** Alopecia areata:

*Oval or round area of hair loss with normal scalp

*presence of "exclamation mark hair" !!!!! At the edge of the patch indicates active process of alopecia areata.

*multiple lesions and marginal scalp involvement = poor prognosis.

** treatment of alopecia areata : intra-lesional steroid

** Cuticle of hair : determine hair direction

** minoxidil : drug convert villous hair to terminal : used to stop baldness

** Anagen period determined by genetic factor : differ from population to another.

-male pattern balding :PCOS, genetic,hormonal .

Treatment : minoxidil,spiranolactone , PRP

** Telogen effluvium :
Diffuse hair loss and reversible .
occurs in patients with stress, hemorrhage and weight loss

** telogen effluvium : it is transformation of hair from anagen phase to catagen one due to stress in acute one (fever , W.loss , surgery , hemorrhage , postpartum) and IDA in chronic type

** each hair grew independently

** anagen phase of scalp hair differ from these hair in other areas , so each site in body has specific anagen phase period (Eyelashes , Eyebrows , ….)

** estrogen maintain hair in anagen phase in pregnancy , so no hair loss during pregnancy but immediately after delivery there is for 3 or 4 months due to delivery stress and hemorrhage

** Estrogen = anti-androgenic and androgen has role in hair loss

** Androgenic alopecia :
common in polycystic ovary
Early use of minoxidil is useful in androgenic alopecia

** Loss of eyebrow :
1. Congenital
2. hypothyroidism
3. trichotillomania

*Trichotillomania >> psycological disorder in which patient pull his own hair leading to alopecia .

** Regrowing of hair in alopecia areata —>vellous hair or white hair not black hair at beginning then black terminal

** Trichotillomania—->patchy hair loss

** hypertrichosis caused by : topical steroid , cyclosporine , tumor , friction

** netherton syndrome :

- ichthiosiform skin changes

- trichorrhhexis invaginata (bamboo hair)

- atopic dermatitis

** Tinea capitis is a DDX of allopeacia areata

** Laboratory investigations are requested for hair loss :

- CBC
- Ferritin
- TFTs
- Vit. B12
- Vit. D
- Zinc

** In Patchy hair Loss , skin :

Normal ➡ alopecia areata

Scaly & erythmtous ➡T. Capitis

Other DD ➡ TT (trichotillomania)

** deficiency of : zinc , iron , V.D , B12 cause hair loss

** cuticle is important to protect the nail matrix
** Lunula is visible part of nail matrix
** Trauma to nail matrix lead to abnormal growth of the nail

** melanonychia : not shifted by time (differ from trauma that does)

urticaria & erythema multiforme

** Target lesion in erythema multiforme , erythematous outer zone ,pale middle , central dead tissue (necrosis)

-- urticaria : dermal edema. -weal -itching -erythema -recurrent -no scar
-less than 24H

** Pin-pointed erythematous papule ——->characteristic of cholinergic urticarial

** Urticaria —>may come with angioedema (40% of cases)

** Angioedema alone —->think of hereditory angioedema

** Urticaria —>drug of choice anti-histamine

** Urticaria —>maybe induced by stress

** Urticaria
Ordinary urticaria >>the individual wheal does not last longer than 24 hours
If it lasts more than 24hours > urticaria vasculitis , painful , leave pigment

** Acute urticaia >>less than 6 weeks
Chronic urticaria >>more than 6weeks

** Urticaria for >6 months >>>the patient needs full assessment to rule out systemic disease (KFT,LFT,patch test,...) ,HCV also

** adrenergic urticaria >>> papule and pallor around it "vasoconstriction"
** cholenergic urticaria >>> papule and erythema around it "vasodilatation"

** causes of urticaria
infection , drug , food , and 60% is idiopathic

- if with urticaria angioedema or at larynx :: adrenaline/ hydrocortison

STD

** any patient with STD check for anther STDs
** gonococcrrhia is a complication of gonorrhea (toxemia)(complicates gonorrhea)

** Chancroid: multiple, painful

** chancre : single, painless, firm(as it is painless the patient may not seek treatment and it will heal, but if the disease is not treated the pateint may become in scondrey syphilis stage)

*both are ulcers

** secondary syphilis skin manifestations :
1-papulosquamous rash that affect the palms and the soles
2-chondylomata lata
3-hair loss
4-white mucous patches

** condylomata lata is highly infectious
*Condylomata lata >> **non itching** rash of syphlis , contagious .

** tertiary syphilis : Point of no return

lichen planus , pityriasis rosea & icthyosis

** pityriasis rosea
-not infectious
-no scars
-no recurrence

- herald patches

-self resolving

-lesion can mimic tinea

-parallel peripherally scaling lesion : Christmas tree lesion

** Licken simplex chronica:
-Typical sites:
1-neck posteriorly
2-knee
-Like psoriasis but psoriasis is silvery dry scales

** we use sometimes biopsy to differentiate between lechin planus and psoriasis

** we can give systemic steroid in lechin plnus in contrast to psoriasis

** Hypertrophic licken
- still long time

- May show resistance to treatment
- May show malignant transformation to squamous cell carcinoma

** DDX. for oral licken planus (atrophic type) is oral candidiasis

** Any type of licken planus May associated with nail affection but it is rare

** Licken planus tt. Is anti inflammatory .... topical or systemic (steroid)

*Tx for lichen planus >> systemic steroid .
*Indications of systemic steroid :
wide spread disease , disfiguring , nail involvement , errosive mucos membrane lesions .

*Follicular lichen planus causes scaring alopecia .

** If not response to tt. May leave post inflammatory hyperpigmentation lesions

** Pityriasis rosea is common
* Its differential diagnosis includes:
- Guttate psoriasis
- lichen planus (but Pityriasis rosea comes with no itching like lichen planus)
- rash of secondary syphilis
- mycosis fungoidus
- drug-induced Pityriasis rosea

** Pityriasis rubra pylaris means :
- Pityriasis means قشور

- Rubra means red
- Pylaris means hair follicle

Icthyosis: is hereditory disorder -there is cong. Icthyosis

Icthyosis vulgaris :spare fluxures -onlu extensors.

Icthyosis treatment : -topical antibiotic -emollient -nersery -fluids

Nepherton syndrome: icthyosis + bamboo sign

……

** Investigation for dermatology (general)
1. Woods light
2. Potassium peroxidase (KOH) operation
3. Hair microscopy
4. diascopy test (like a slide put on the lesion)
5. Tzanck smear (for viral infection)
6. Gram stain (for gonorrhea)
7. Patch test
8. Skin biopsy for :
--any disease come with atypical presentation
--to confirm differentiation between similar diseases
--malignancy
9. Lab test
10. Imaging study

** most common cause of immunodeficiency nowadays is recurrent steroid injections

** symmetrical involvement of skin lesions :

* inflammatory skin lesion : psoriasis , vetiligo , lechin planus

** unsymmetrical involvement of skin lesions :

* neoplastic

* infection

--heliotrope rash : dermatomyocitis

-if scar over burn : marjolen ulcer -- squamous cell carcinoma.

-cryotherapy : using cryogun :liquid nitrogen

-miliaria &milia :retention cyst.

-after chickenpox virus == pox scars.

Scales in :

-contact dermatitis :biobsy

-psoriasis : auzpits sign

-fungal : KOH

*child with cystic fibrosis will have salt tasting skin