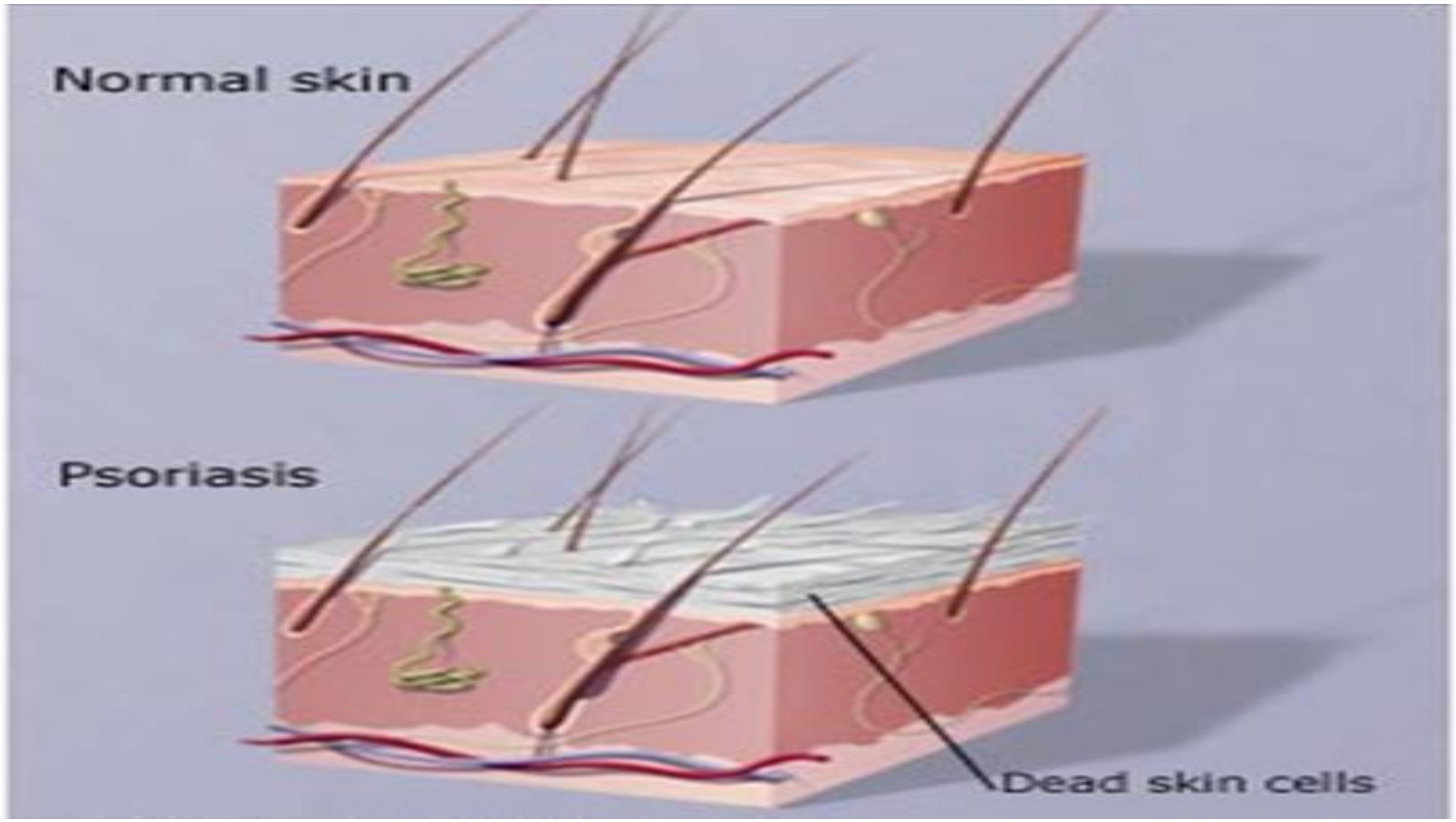
Lecture 6 Dermatology

Dr khaled sobhy



3- Vitilligo

It is genetic and autoimmune disorder

Diagnosis:

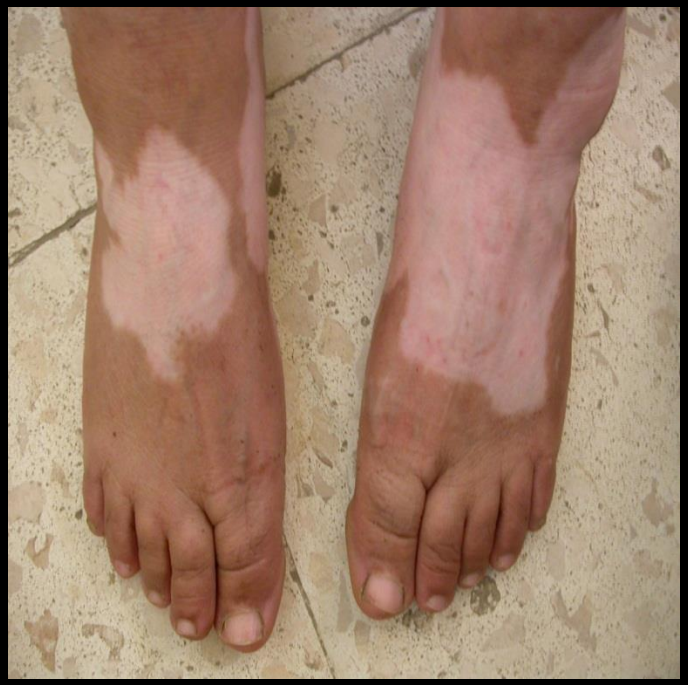
This depends on location and presentation:

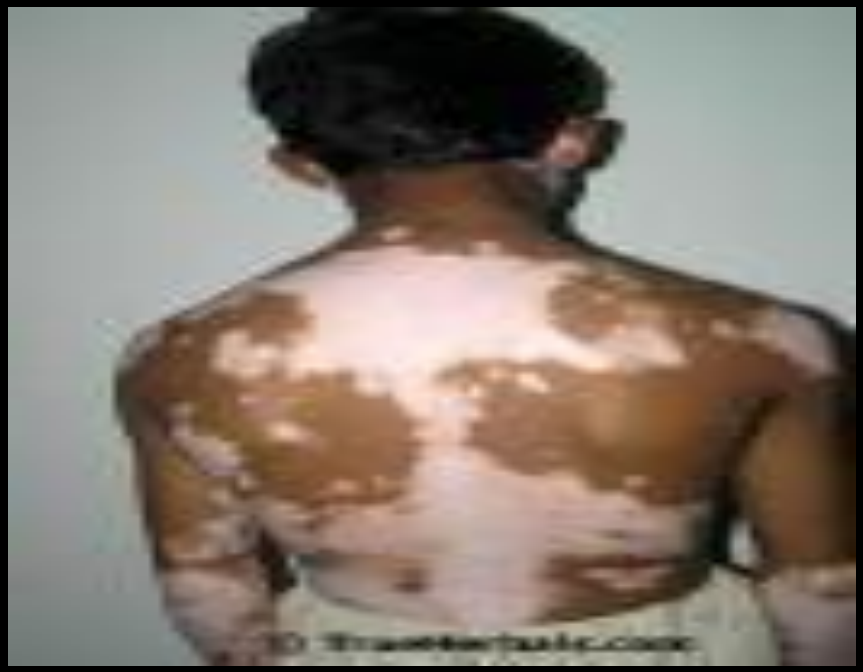
It gradually start on sun exposed area (arm , face, hand, elbow) and hyper pigmented area (genitalia)

It appears as milky white macules with sharp erythematous margin without itching or scale (difference from tinea versicolour)

Vitilligo

Vitilligo





Treatment (not OTC)

1- systemic corticosteroid:(prednisone, dexamethazone) its immunosuppressive effect reduce the progress of the lesion

2- repigmentation (Phototherapy):

-Khelline (ezaline®) or psorallen photosensitizer followed by UV exposure (stimulate melanocyte to produce melanin). Psorallen may show phototoxicity and mutagensis

- meladinine precursor (methoxsalen) followed by UV exposure (ultrameladinine®)either lotion (for small area) or tablets (for large area)

Duration of therapy for 2-4 months

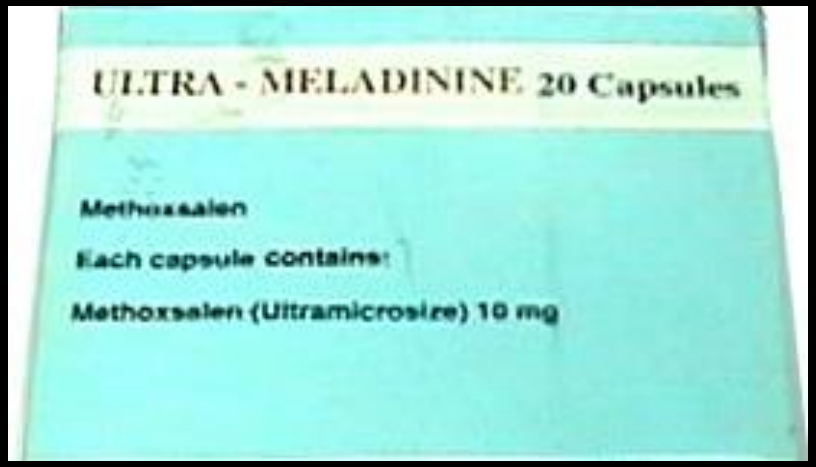
Treatment (not OTC)

3-tanning agents: Dihydroxyacetone or DHA (Vitinorm®) make coloration of the skin

4- Depigmentation (fading the rest skin of the body)

Hydroquinone cream

Vitilligo treatment



4- Pityriasis alba

common cause of hypopigmentation in children,

especially in darker skins. it is Pale patches or white spot with a slightly scaly surface appear mainly in face of children

Diagnosis: location, age, presentation

unknown cause but suspect to be caused by malnutrition (calcium and vitamin A) or fungal infection

Ptyriasis alba

Treatment(OTC)

1- corticosteroid cream

2- moisturizing cream

3-may add vitamins and minerals

4- may add antifungal topical preparation.

It shows good prognosis

Hyperhydrosis

Excessive sweating, either **generalized or focal** (e.g., palmar, axillae), and affecting 2%—3% of general population; most common in **adolescence and young adults**.

Generalized hyperhidrosis is associated with underlying systemic disorder, e.g., Endocrine(hyperthyroidism), or neurologic but **focal hyperhidrosis often idiopathic**.

Lead to a strong bad body odor (unpleasant smell) that is a social embarrassment for many people

If accompanied with:

1. inadequate attention to personal hygiene
2. in presence of Certain types of bacteria a strong odor.
3. taking certain social drugs such as marijuana, or smoking

Diagnosis

Focal, visible, **excessive sweating of at least 6 months** duration without apparent cause with at least 2 of the following characteristics:

Bilateral and relatively symmetrical sweating.

Frequency of at least 1 episode per wk.

Impairment of daily activities.

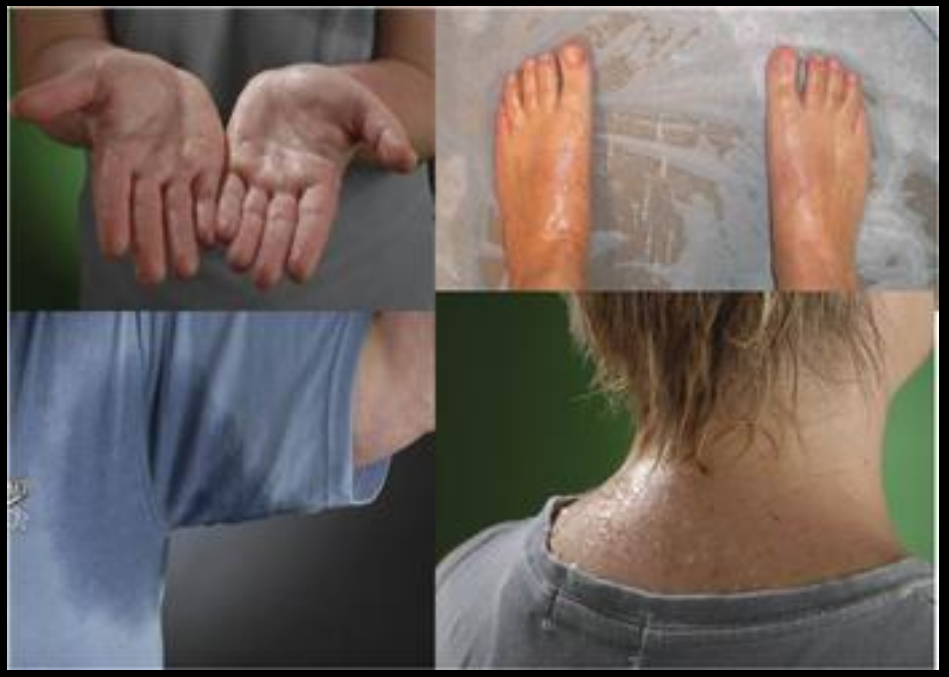
adolescence

Positive family history.

Cessation of sweating during sleep.

Differential diagnosis: **Starch iodine** test can be used to outline the area of excessive sweating.

Hyperhydrosis



Treatment

Topical antiperspirant deodorants:

Aluminum chloride hexahydrate solution in ethanol 12% (Drysol®) or in gel base instead of alcohol(Hydrosal®)

They make irritation to skin.

Systemic:

Botulinum toxin (Botox®) injections very effective; can last 6—12 months.

Surgery: Endoscopic thoracic sympathectomy, subcutaneous liposuction.

Prevention & recommendation

Showering with scrubbing the body

scrub the body, especially the armpits, with water and a deodorant soap at morning and night under the shower

Choose suitable clothes

Choose natural fabrics such as cotton and wool that absorb perspiration better than synthetics.

Keep your clothes fresh

Regular washing of clothes is important. change each day, especially in the summer months.

Dietary advice: Avoid or reduce the intake of garlic, fish, onions.

Reduce your intake of caffeine (coffee, tea and cola drinks), which stimulates sweat activity.

Avoid smoking

Shaving hair under the arms

Sun dermatologic disorders

8-Sun burns

- **UV divided to UV "A" 320 to 400 nm" & UV "B" "290 to 320 nm". The shorter the wave length, the more deeply penetration of the skin causing powerful burning**
- It is more likely to occur in people with light coloured skin

Sun burn (means skin inflammation or redness) The skin is red **with peeling** , hot, painful and swollen

there may be headache, fever, nausea and possibly delirium.

-Taking various drugs (such as some antibiotics, hormones and tranquillisers) can increase the risk of sunburn.

8-Sun burns

Types:

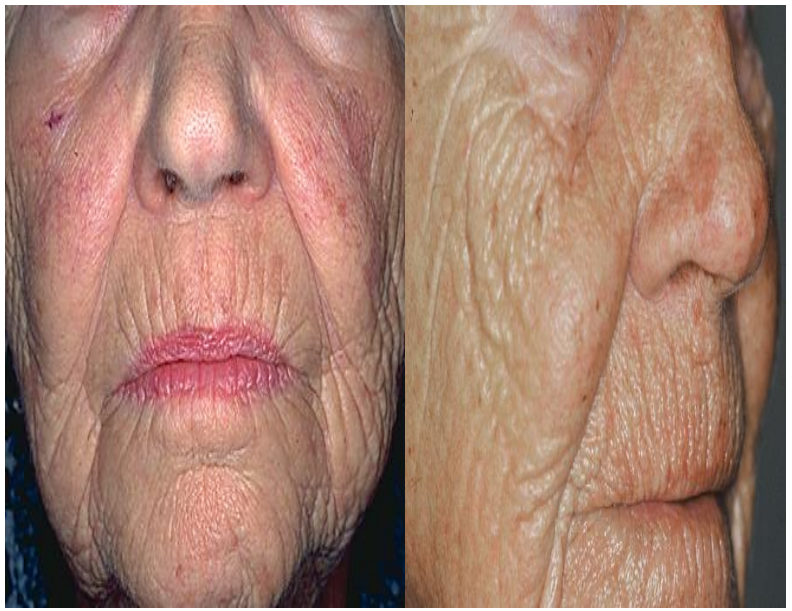
1-photoaging

Definition:

Premature aging where skin is inflamed due to **chronic exposure** most commonly with old farmers

Symptoms:

wrinkles, coarseness, scaling & dryness



8-Sun burns

2-Pigmentary changes

May be hyper pigmentation or hypo pigmentation

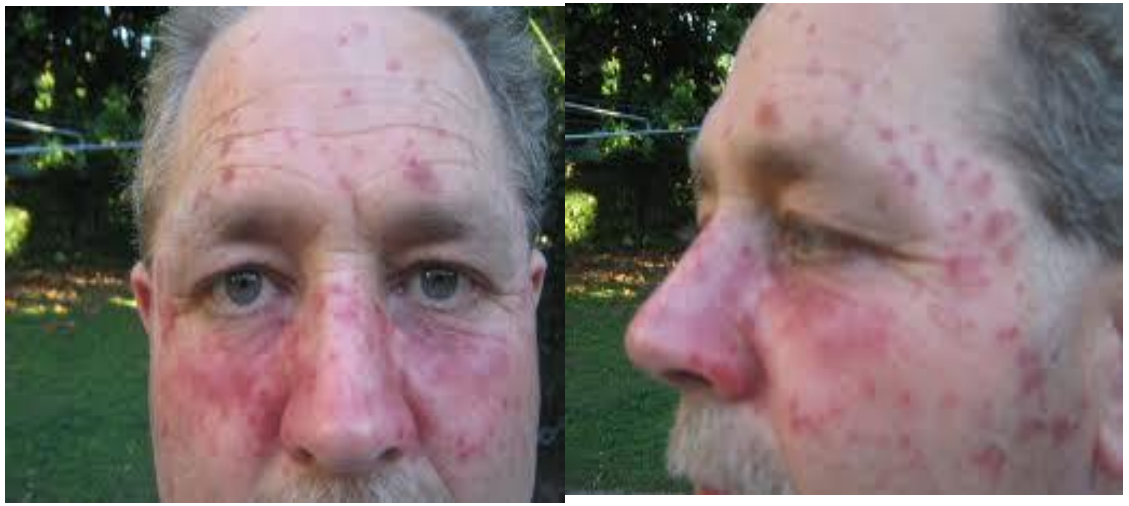
Most common after beach swimming

3- Solar keratosis

3-Solar Keratosis

Most common **precancerous lesions** in humans, and more prevalent in **fair-skinned** individual

Patches of **Slightly erythematous, rough, scaly lesion** on sun exposed areas, i.e., head, neck, backs of hands and forearms.



Prevention and treatment

*By using sun screens & sun blockers

***Infant < 6 months not need sunscreen or blocker but recommend sun avoidance.**

-Avoid the direct sun from 10 am to 3 pm.

-Use natural shade. Beware of reflected light from sand and water.

-Wear broad-brimmed hats and protective clothing.

-Use zinc oxide ointment for maximum protection (physical sun screen).

Sun screen & Sun blockers

Sun screen :

Reduce UV amount that reach the skin by either physical & chemical ways.

Physical: By reflect or scattering certain UV rays.

Chemical: by absorption of UV spectrum.

Sun blockers:

Completely block UV radiation.

SPF

The efficacy of sunscreen is indicated by **"SPF"** sun protection factor which is the ratio of time to produce skin reddening with sunscreen compared to that time without sunscreen.

SPF:

8 – 11	**"high protection, permit some tanning"**
12 – 19	**"Very high protection" doesn't permit tanning"**
20 – 30	**"Ultra protection, doesn't permit tanning"**
> 30	**"Sun blocker for highly sensitive skin"**

Composition of sunscreen

Chemical :absorb UV radiation

Benzophenone g.p.: oxybenzone (spectraban 28) ® & dioxybenzone

P-amino benzoic a (Per sun 46)®

Cinnamate ex: cinnoxate, methyl cinnamate

Salicylate: as octyl salicylate

Physical :

opaque in nature, so reflect UV radiation

- **Ex1: Titanium dioxide: reflect & scatter UV.**
- **Ex2: Zn oxide.**
- **Ex3: Avobenzone.**

Composition of sunscreen

NB:

Sunscreen should be applied <u>30 min before sun exposure</u>.

Applied as:

½ tea spoonful for face & neck

1 tea spoonful for arm & shoulder

2 tea spoonful for leg & feet

Limit sun exposure to be before 10 am & after 3 pm.

4-Skin Cancer

Skin cancer is usually found in **fair-skinned people** who are exposed to **too much sun**.

The main types of skin cancer:

Basal cell carcinoma: the commonest and least dangerous type, usually appears on the face and neck, is easily treated apear shiny, pearly skin sore

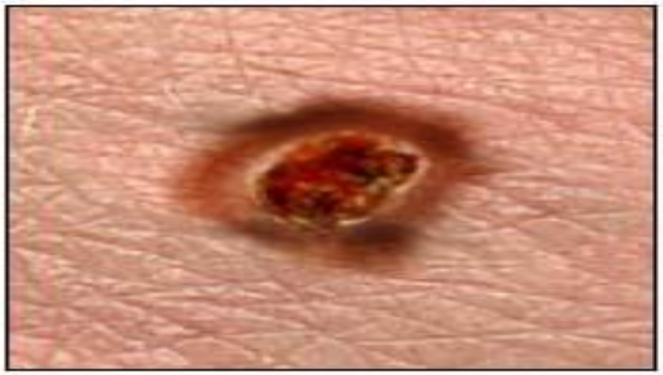
Squamous cell carcinoma: is quite dangerous, appears on hands, forearms, face and neck , can spread to other parts if left untreated too long. Appear as nodule then the center become necrotic with ulceration (sores)

Suspect if :

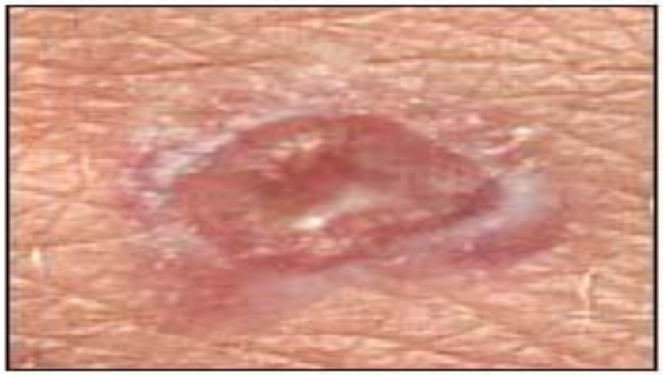
non-healing sore which require sample examination

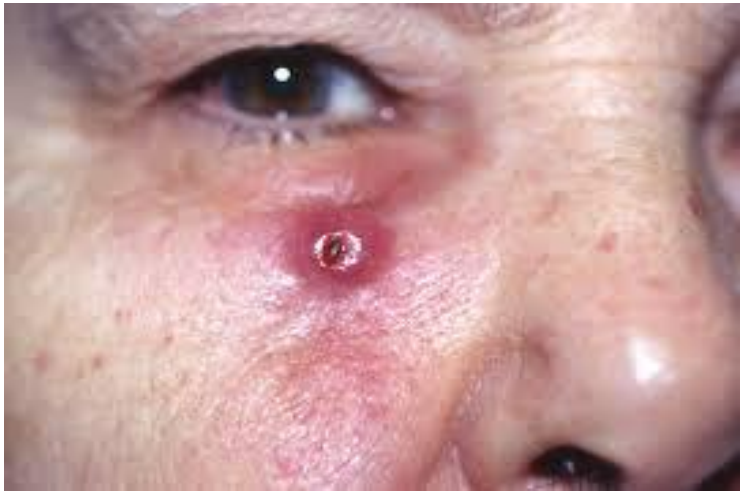
Skin Cancer

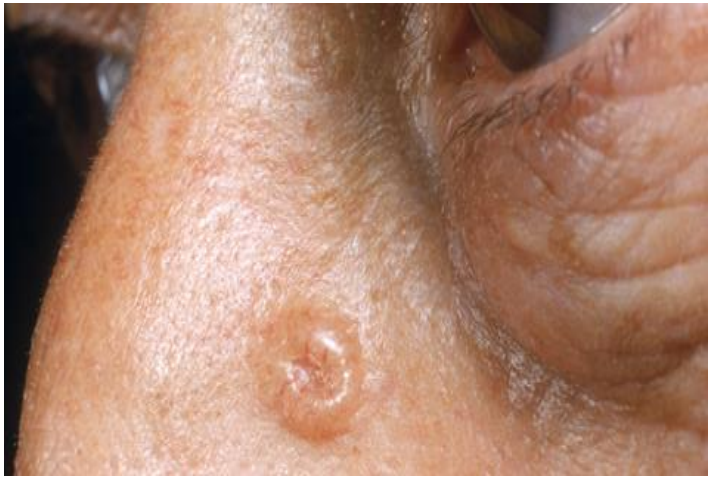
Squamous cell carcinoma



Basal cell carcinoma







Melanoma

the most dangerous type of skin cancer. It grows from special cells in the skin called *melanocytes*. A melanoma is usually **brown or blackish lump with itching and bleeding. Occur in area of sun exposed area mainly face.**

People at increased risk are those with:

- several dark moles
- freckles
- fair white skin

mole may turn to melanoma. Any changes that occur in a mole should raise suspicion. Changes may include:
any change in the colour , size, thickness, bleeding, itching or spreading of the mole.

Melanoma

- Once suspicion is raised about a mole, it should be treated by **surgery** (wide **excision**) by your doctor. The removed sample is examined with a microscope.

- melanoma can be completely cured.

Prevention is the best cure:

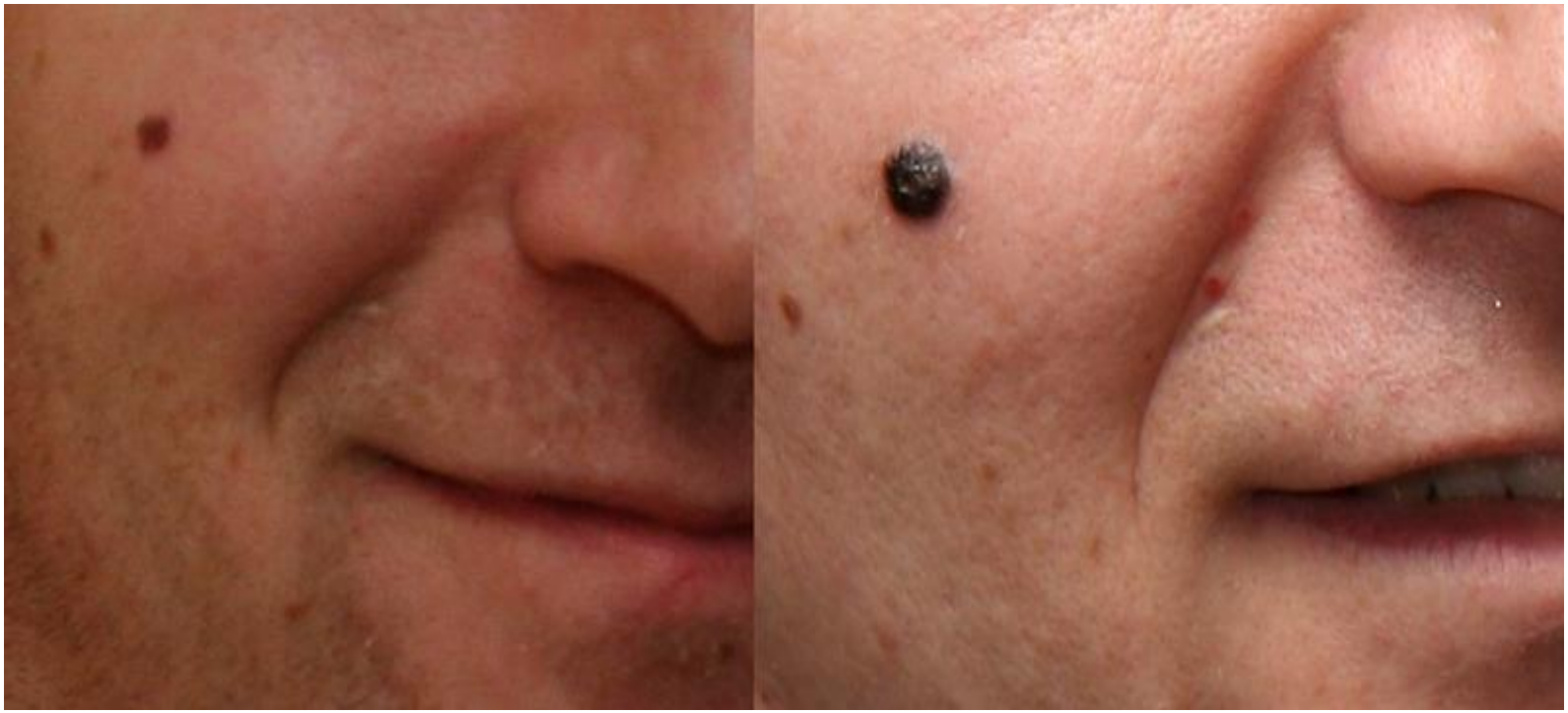
-protect yourself from the sun. These rules should be followed:

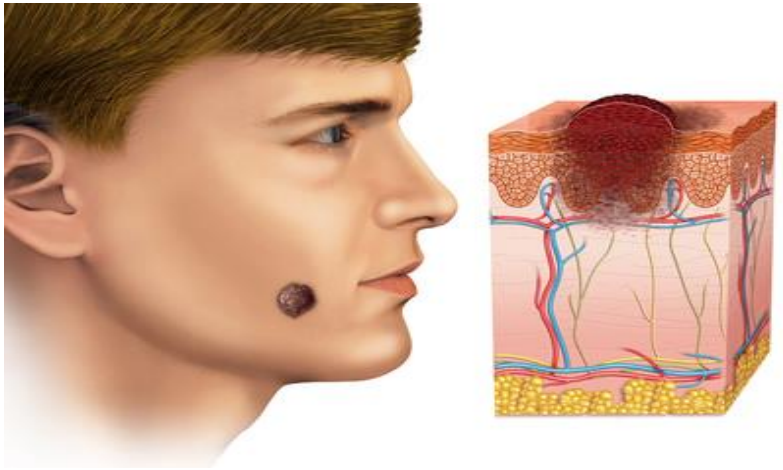
-Try to avoid direct sunlight when the sun is strongest (from 10 am to 3 pm).

-Always wear a broad-brimmed hat and T-shirt in the sun.

-Use 15+ SPF sunscreen on exposed skin and renew it regularly.

Melanoma

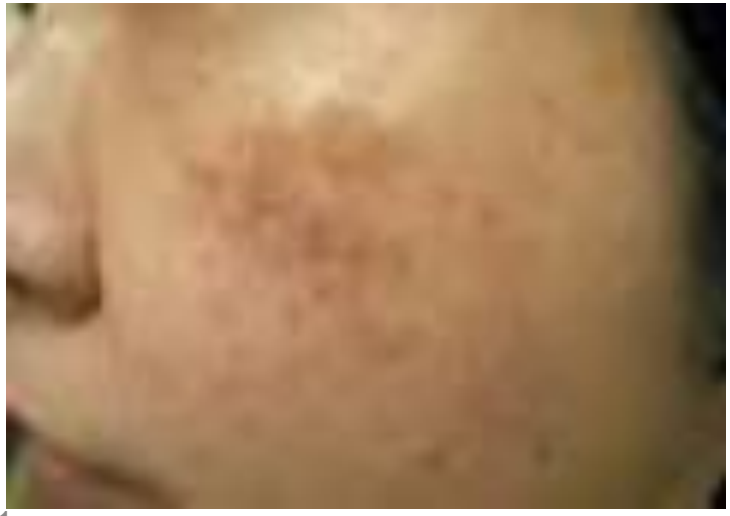


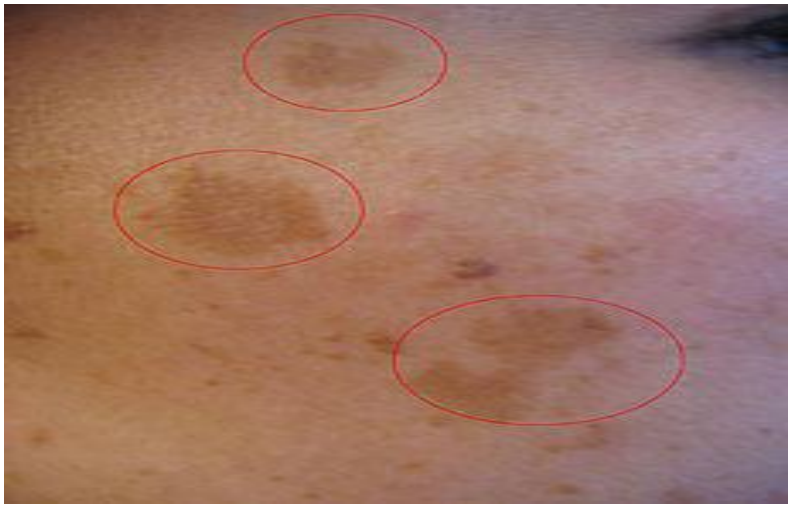


6- Melasma(OTC)

Etiology: from exposure to sunlight. Can be associated with pregnancy (“mask of pregnancy”), oral contraceptives.

Well-demarcated, brown hyperpigmented macules, mostly on Face on forehead and mandible**. asymptomatic,** often starts in pregnancy, **worsens in summer**.





Melasma treatment

Daily sun protection is essential with high SPF and broad brimmed hat; sun blocks may preferred.

■ Hydroquinone (2% OTC; 4% by physician) are most commonly Used

■ Kligman formula combines a hydroquinone with a mild (betaderm)and tretinoin.

■ Chemical peels.

■ Lasers occasionally used.

Freckles

Freckles are not a skin disorder, but people with freckles generally have a lower concentration of photo protective melanin

clusters of small patches of hyperpigmented area (concentrated melanin) which are most often visible on people with a fair skin.

The formation of freckles is triggered by **exposure to sunlight**. The exposure to UV-B radiation activates melanocytes to increase melanin production overproduce melanin granules.

Freckles are predominantly found on the **face or sun exposed area**

Freckles

People whose skin tends to freckle should **avoid overexposure to sun and use sunscreen**

They tend to become darker and **more apparent after sun exposure** and lighten in the winter months.

Freckle prevention is more effective than freckle removal. Freckle-reduction treatments are more difficult and not always satisfactory.

Freckles are more **commonly found on children before puberty** (may fade with age).

Freckles

Freckles treatment

1. **Bleaching creams**: hydroquinone in combination with sun avoidance and sun protection.

2. **Retinoids**: Sometimes used in conjunction with other bleaching creams, tretinoin (vitamin A acid, Retin-A), tazarotene, and adapalene (Differin) also may help **lighten freckles** when applied consistently over a period of several months.

3. **Laser treatment**: Multiple types of lasers may help lighten and decrease the appearance of freckles safely and effectively.

Freckles prevention

- use of sunscreens with SPF (sun protection factor) 50,
- use of wide hats (6 inches)
- use of sun-protective clothing (shirts, long sleeves, long pants),
- avoidance of the peak sun hours of 10 a.m. to 3 p.m.,
- seeking shade and staying indoors

1- Acanthosis nigricans

Etiology

1-non malignant:

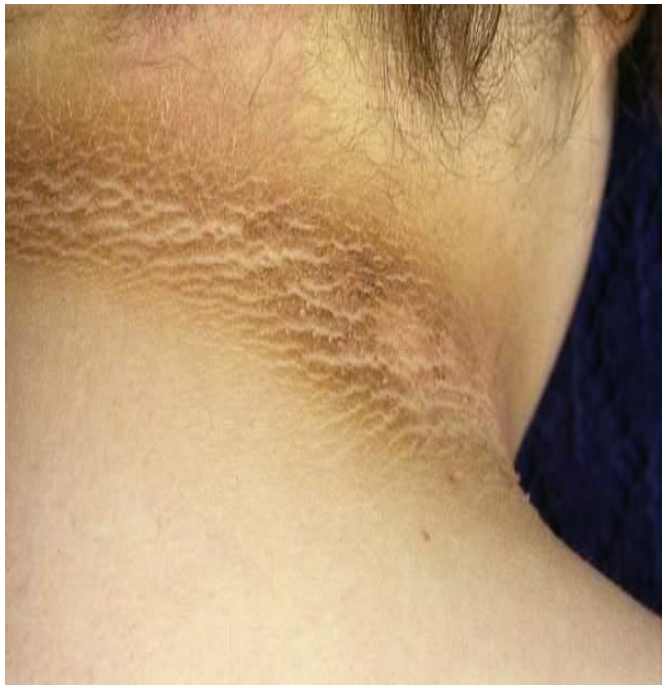
a.Heredity

b. Endocrine disorder—most commonly associated with insulin resistance (i.e., diabetes mellitus)

c. Obesity

d. Drugs—e.g., nicotinic acid,oral contraceptives, steroids

2- Malignancy, usually adenocarcinoma—e.g., gastrointestinal (60% stomach), lung, breast



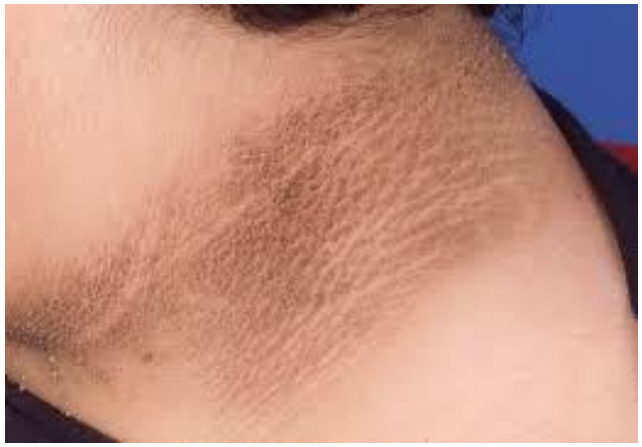
Diagnosis

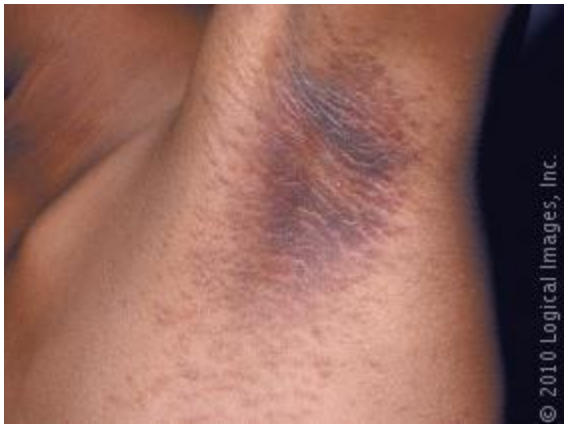
1- brown to black Hyperpigmented velvety, typically symmetric. predominantly on the neck, axillae, and armpit.

2-History of previous causes

Diagnois depend on location, presentation and cause.

Treatment: treat the cause, bleaching creams if small area





Psoriasis

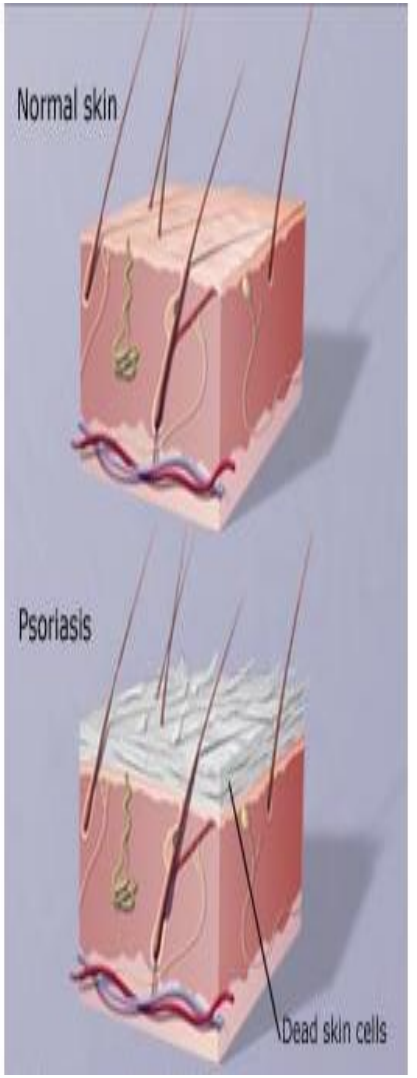
Normal human skin has **epidermal cell cycle** where outer layers of epidermis is replaced by inner most layers. This takes about 30 days

In psoriasis, the rate is 10-20 faster than normal

Caused by genetic change in cellular immunity. (progressive T-lymphocyte–mediated systemic inflammatory disease)Leading to accumulation of layers of keratins forming scales and plaque

Psoriasis is a chronic disorder shows regression and recurrence (**waxes and wanes**)

run in families (about 80%)



Psoriasis

- psoriasis is a lifelong illness (very visible and emotionally distressing) need for empathy with patients. No cure for it
- Lesion may spread to areas where trauma, cuts, burn, or surgery occur (**kobner phenomena**). So, special care is required.

Triggers (precipitating factors) include: Injury to the skin, infection, drugs (lithium and antimalaria), seasonal changes, smoking, alcohol consumption, obesity, and psychogenic stress.

Comorbide condition: Psoriatic arthritis is one of the most common and well-known extracutaneous manifestations of disease. Other include metabolic syndrome, depression

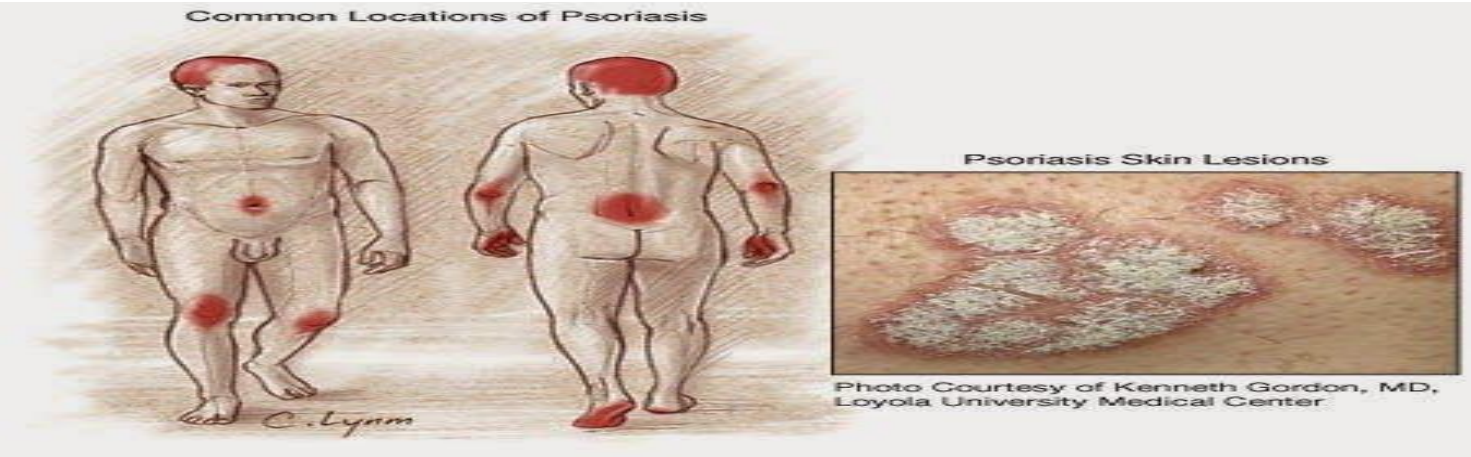
Diagnosis

Diagnosis depend on location and presentation:

It occur mainly in extensor surface (knee, elbow), sacral area, scalp, abdomen (uncommon in face)

gradual onset It appear as plaque silvery white or Red-violet on top and deep erythematous below with inflamed margin **with mild irritation but usually disfiguring**

Pinpoint bleeding may occur on removing plaque (**Auspitz sign**) from rupture of very close capillaries



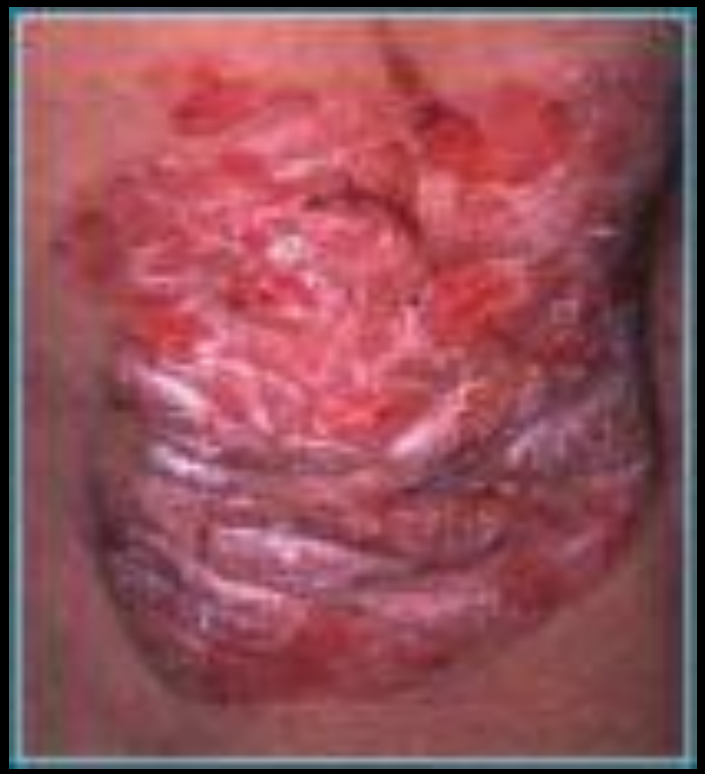
Classification:

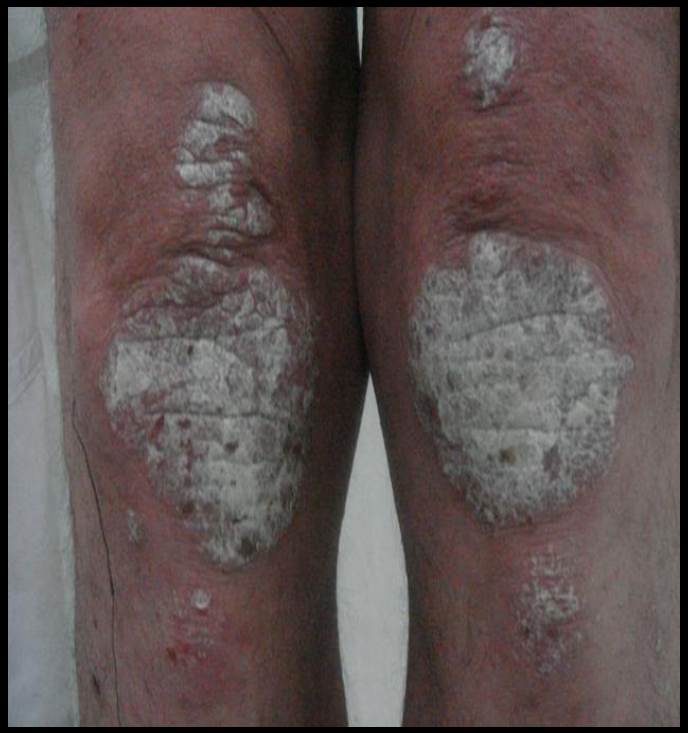
3 main categories: mild, moderate, and severe.

- Mild psoriasis: ≤10% BSA involvement
- Moderate to sever psoriasis: BSA ≥10%
- Severe: BSA ≥20%

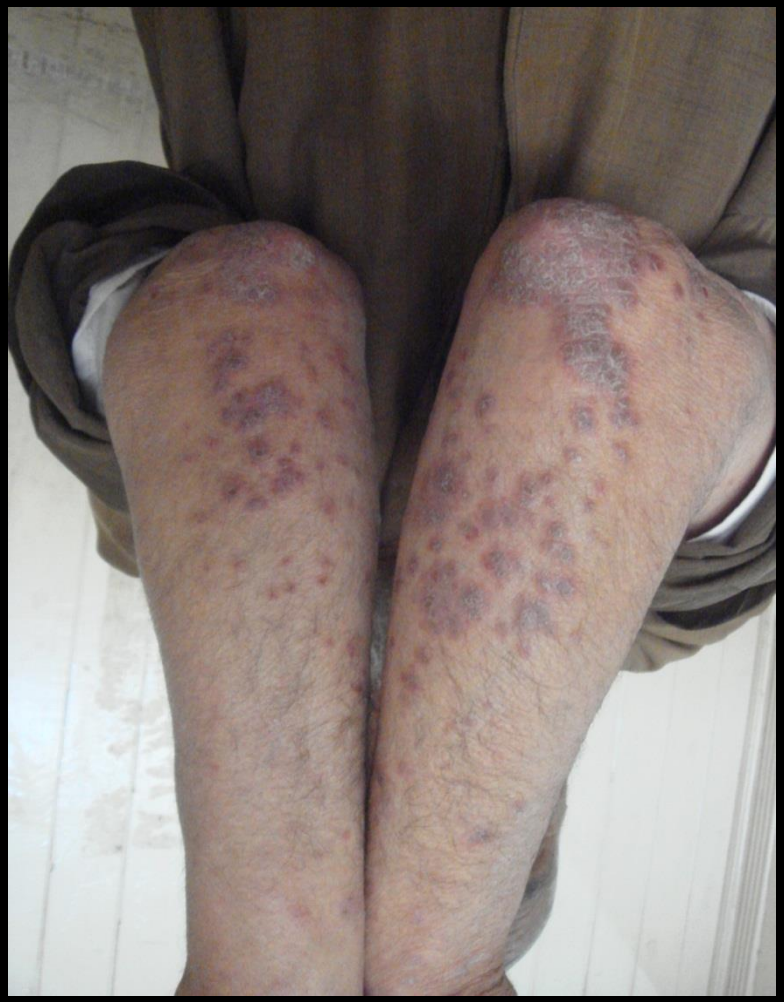
- Practically, palm size is approximately 1% BSA
- head and neck involvement is approximately 10% BSA,
- both upper limbs approximately 20% BSA,
- trunk involvement (front and back) 30% BSA
- both lower limbs approximately 40% BSA.

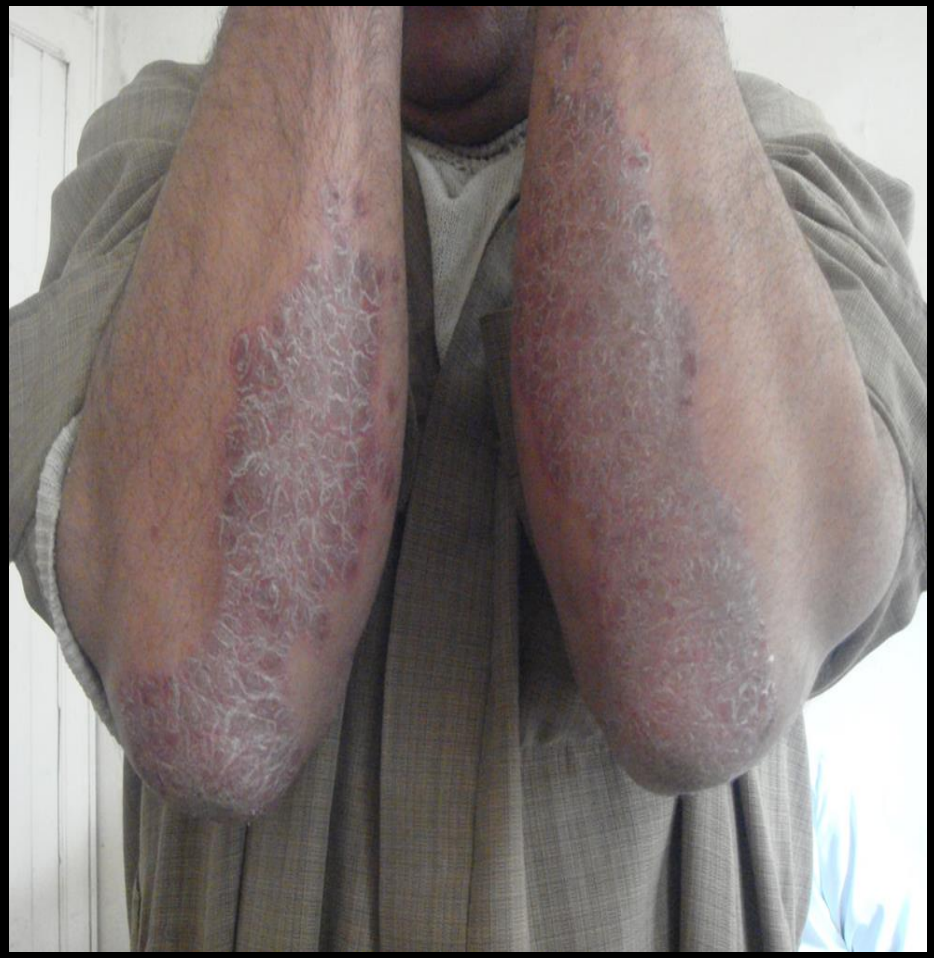
Psoriasis lesion



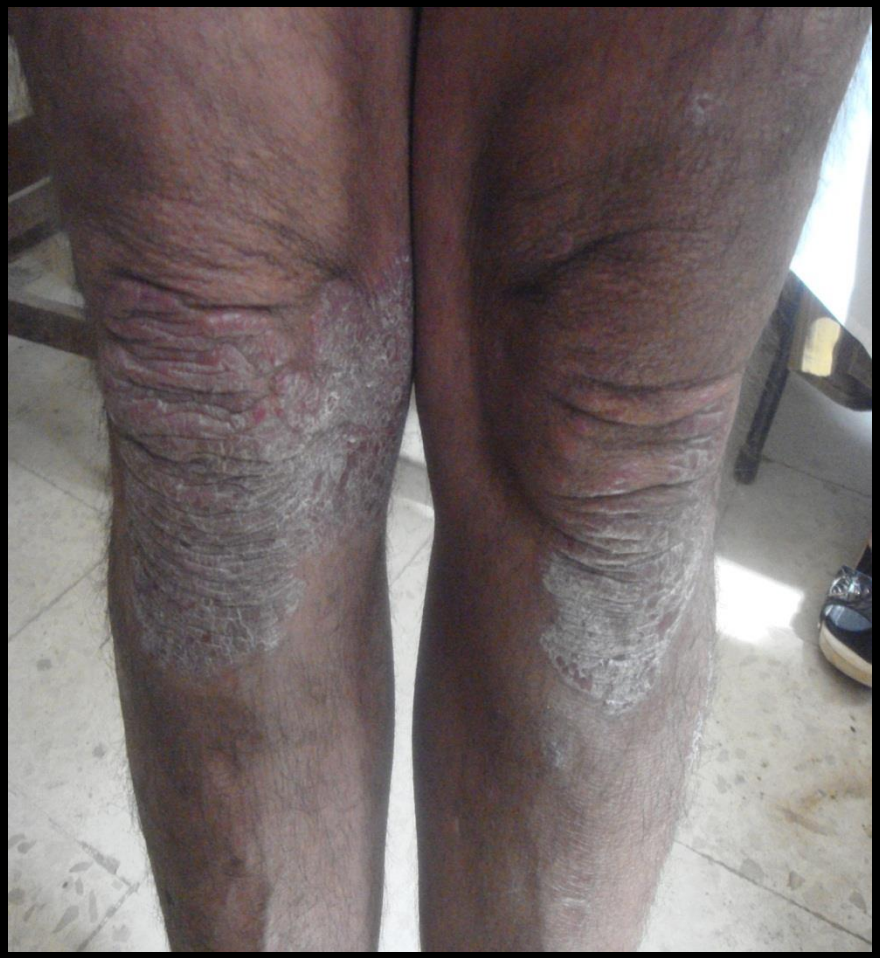


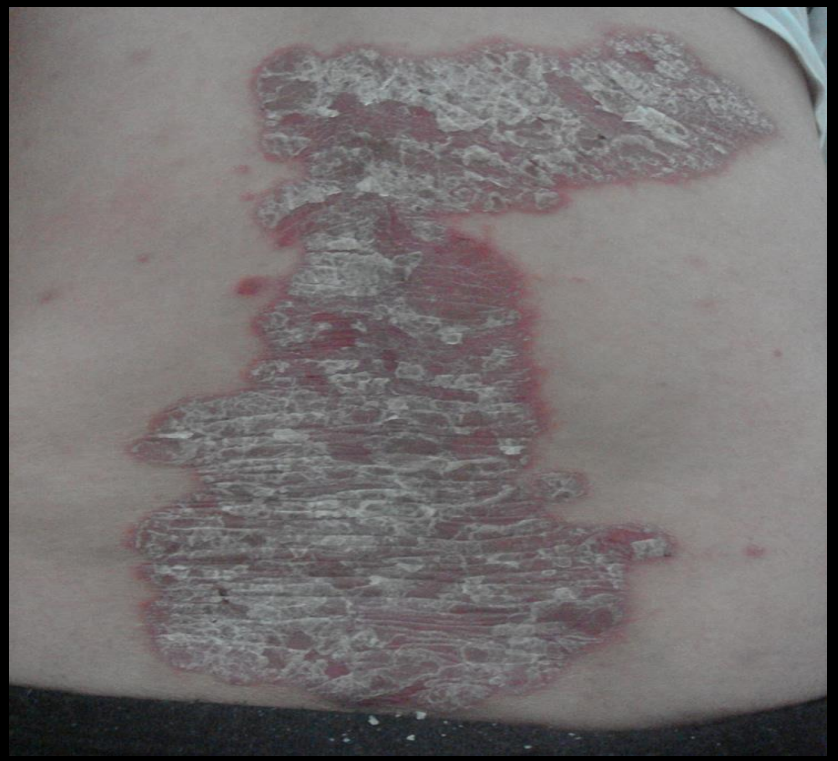
Psoriasis lesion



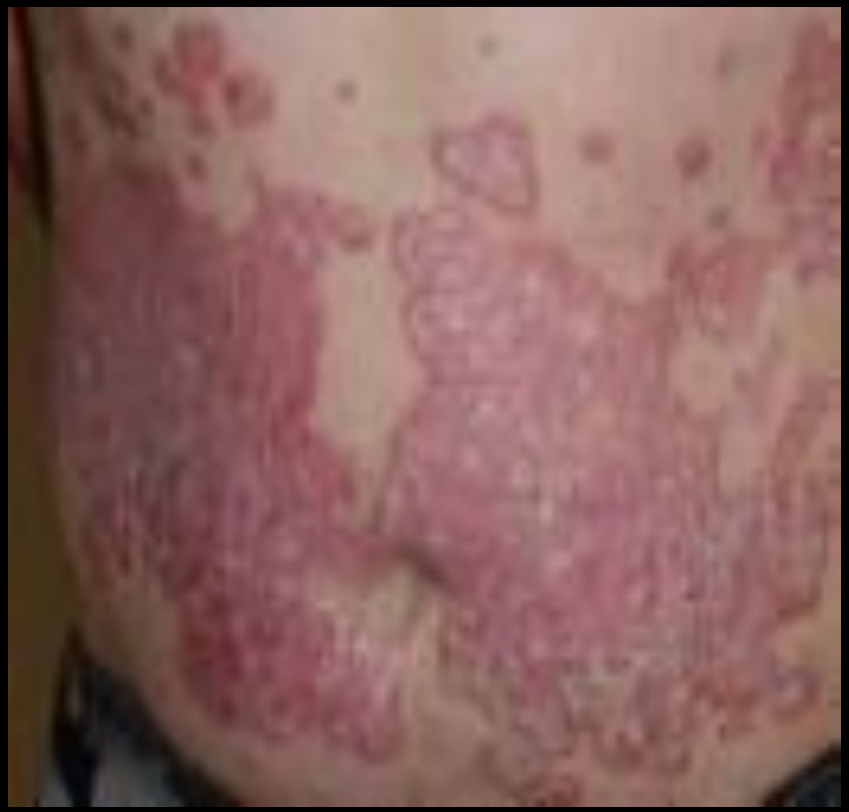


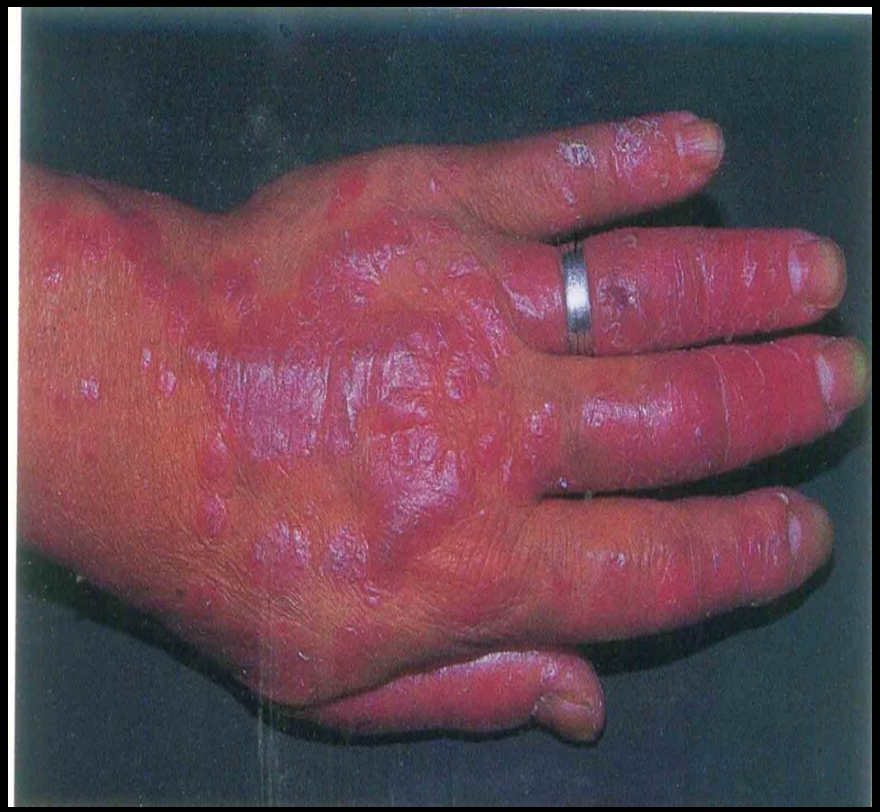
Psoriasis lesion



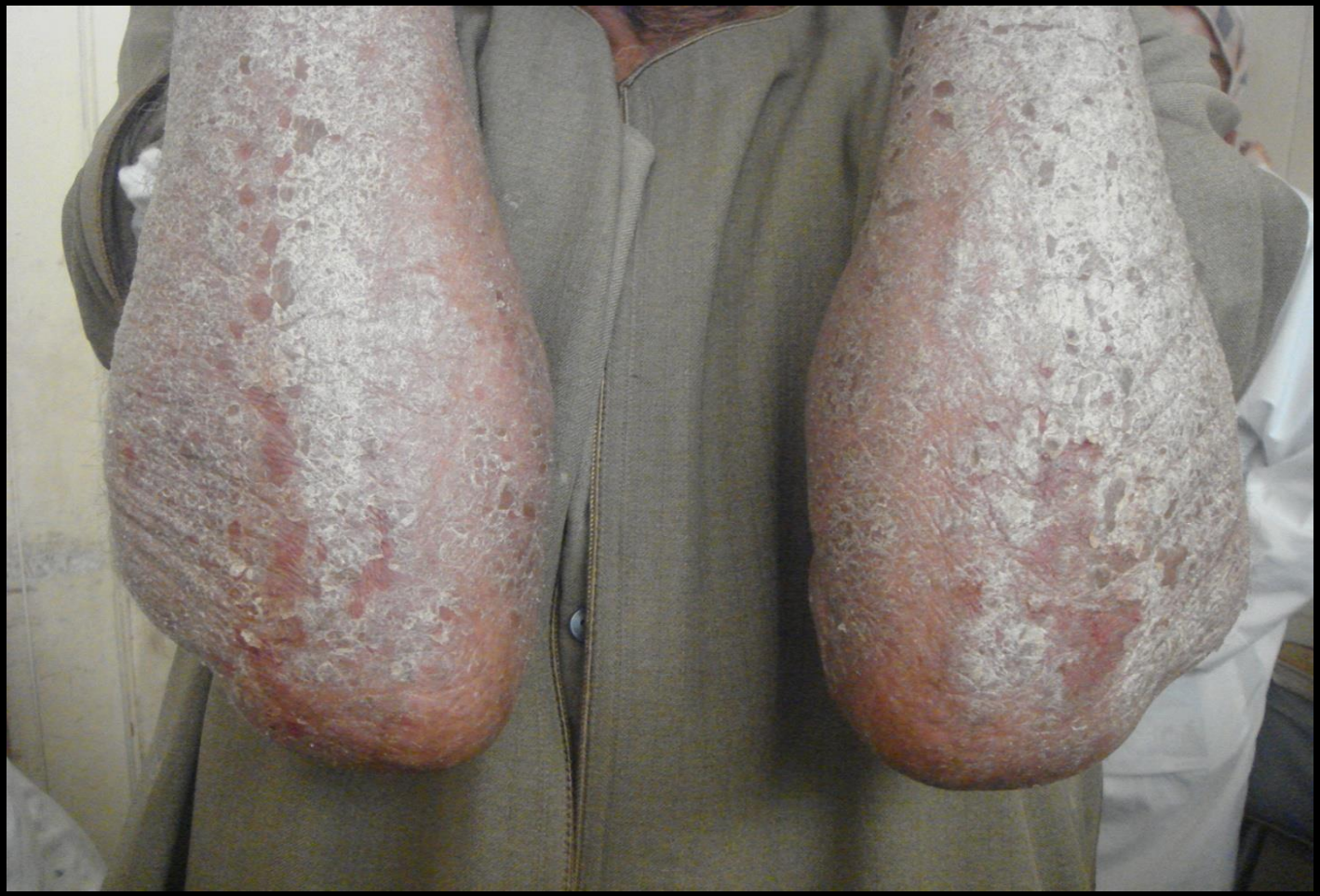


Psoriasis lesion on abdomen



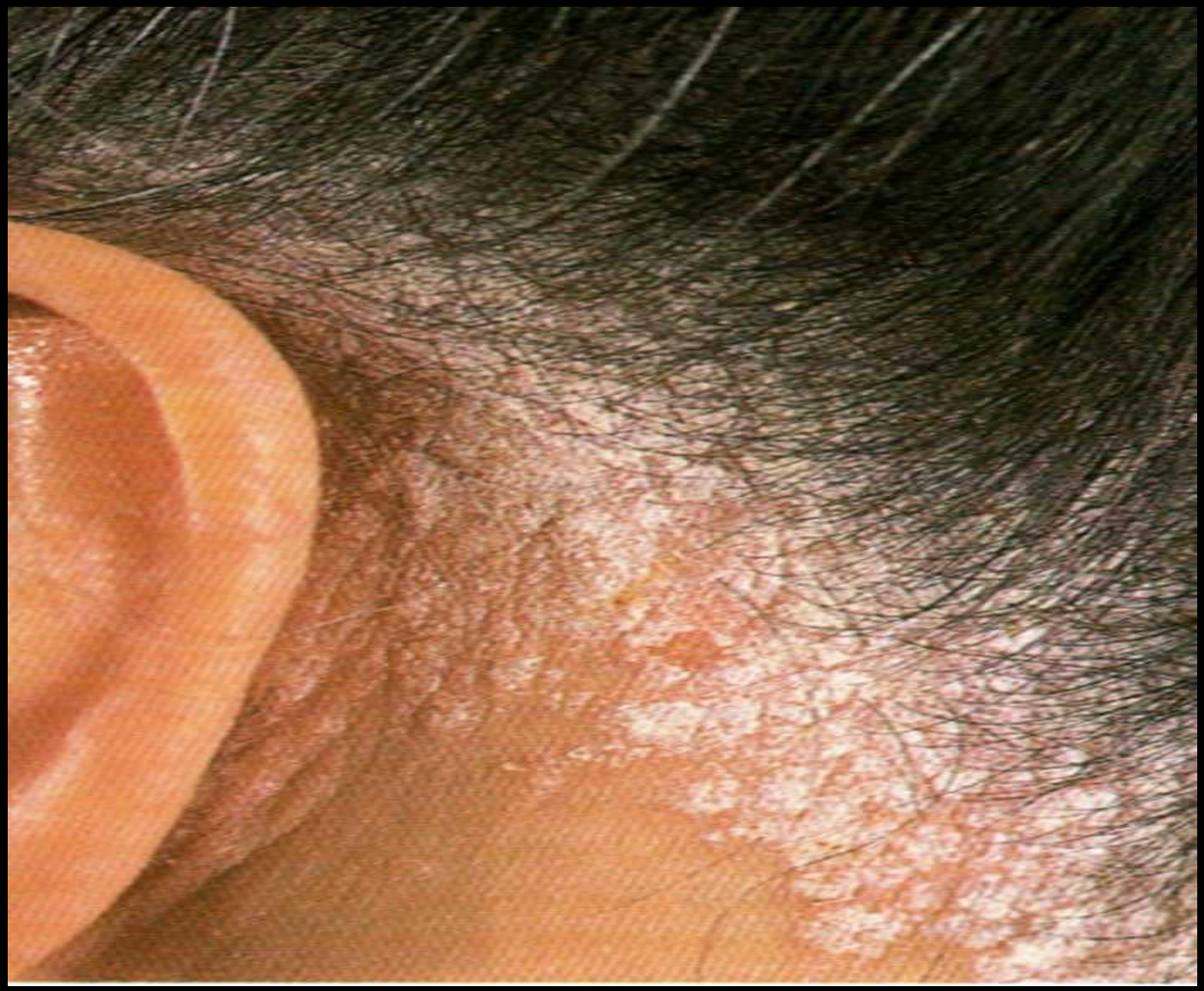


Psoriasis lesion on Elbow



Auspitz sign in psoriasis

Psoriasis lesion on scalp



Treatment Goal

1. Improve QOL Minimizing or eliminating the signs of psoriasis, such as plaques and scales (75% loss of lesion)
2. Alleviating pruritus and minimizing excoriations
3. Reducing the frequency of flare-ups (recurrence)
4. Ensuring appropriate treatment of associated comorbid conditions such as psoriatic arthritis, hypertension, dyslipidemia, diabetes, or clinical depression.
5. Avoiding triggers factors or precipitating factors.

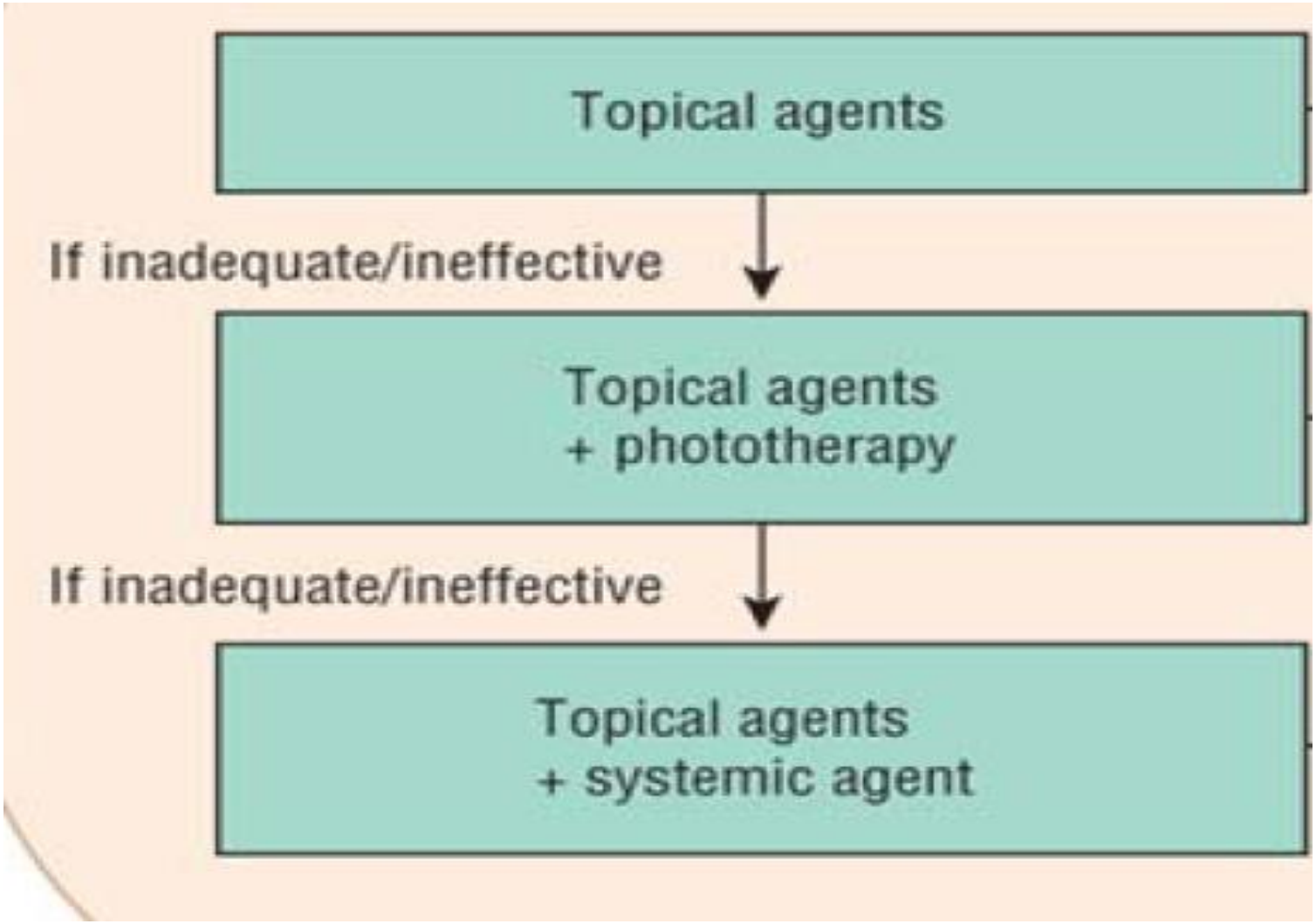
The 2011 European consensus defined **induction (first 16-24 weeks) and maintenance phases till remission**.

Monitoring: Treatment goals should be assessed at **10 to 16 weeks and then every 8 weeks thereafter**.

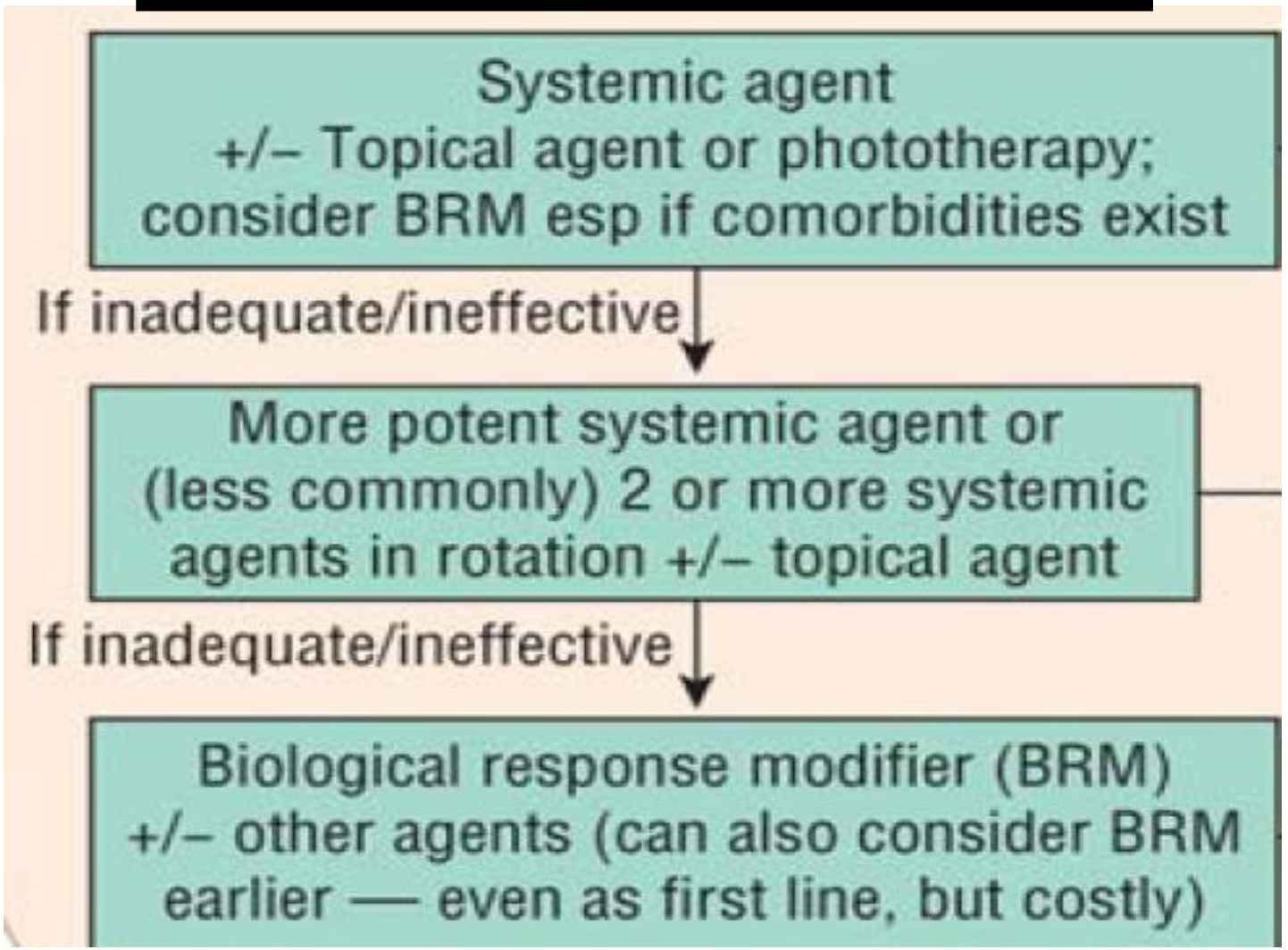
Non pharmacological treatment

1. stress-reduction strategies
2. moisturizers: **carbamide (urea):** promote hydration and remove excess keratin in dry skin and increase the action of corticosteroid. other emollient like **glycerin, Vaseline** may be used to reduce irritation
3. oatmeal baths
4. skin protection using **sunscreens**. sunburns can trigger an exacerbation of psoriasis.
5. **Balenotherapy** means treatment using bath in waters containing higher concentration of certain salts, combined with natural exposure to sun)
6. For patients with **comorbidities** such as dyslipidemia, obesity, or cardiovascular disease, cessation of nicotine and alcohol consumption, diet control, and increasing physical activity are all important interventions

Treatment for mild



Treatment for moderate-severe cases



Topical therapy

- 1- **corticosteroid topically**: it suppress the rate of cell turn over (clobetasol) Dermovate®
- The use of ultrapotent corticosteroids should be limited to a duration **of 2 to 4 weeks**.
- Cutaneous adverse effects include striae, skin atrophy, acne, contact dermatitis, folliculitis
- Systemic adverse include hypothalamic–pituitary–adrenal (HPA) axis suppression and less commonly Cushing's syndrome, osteoporosis, cataracts, and glaucoma

Topical drugs

2- Coal Tar (Polytar shampoo®):

-it act by reducing the number and the size of produced epidermal cell

-It is less frequently used now due to **many side effects** which are:

- increase tendency to sunburn (phototoxicity)
- cancer risk in groin and genital area
- make coloration to skin, cloth, and hair

-cannot be used in inflamed area or before UV exposure

3-Vitamin D3 Analogs

- Topical vitamin D3 analogs include calcipotriol (calcipotriene), calcitriol (the active metabolite of vitamin D). Only calcipotriol is currently available in USA

- Their mechanisms include binding to vitamin D receptors, which results in inhibition of keratinocyte proliferation and enhancement of keratinocyte differentiation. They also inhibit T-lymphocyte activity.

- Topical SE: mild irritant contact dermatitis; others include burning, pruritus, peeling, dryness, and erythema.

- systemic adverse effects, including **hypercalcemia and parathyroid hormone suppression but rare**

Topical

- 4- **salicylic acid 3%** : it has keraotlytic action (remove . scales from epidermal cell). it enhances steroid penetration thus increasing efficacy. diprosalic® (betamethasone + salisylic acid).

- 5-Tazarotene is a topical retinoid that acts through the following mechanisms: normalizing abnormal keratinocyte differentiation, diminishing keratinocyte hyperproliferation.

- It is effective in clearing psoriatic plaque lesions and achieving remission.

Topical

- Anthralin :
- Anthralin is not as commonly used
- Short-contact anthralin therapy (SCAT) is usually the preferred regimen, where the anthralin ointment is applied only to the thick plaque lesions for 2 hours or less and then wiped off.
- Because lesions are generally well demarcated, zinc oxide ointment or a nonmedicated stiff paste should be applied to the surrounding normal skin to protect it from irritation and burning.
- Anthralin should be used with caution, if at all, on the face and intertriginous areas because of the risk of severe skin irritation.

Systemic Therapy

1-Acitretin:

Acitretin (Soriatane®): oral retinoid, act on retinoid receptors in the keratinocyte to correct abnormal cell differentiation

- Currently, acitretin is more commonly used in combination with topical calcipotriol or phototherapy. Its efficacy appears to be dose dependent but low-dose acitretin (25 mg/day) is safer and better tolerated than higher-dose (50 mg/day) therapy.

- **SE:** hypertriglyceridemia and mucocutaneous adverse effects such as dryness of the eyes, nasal and oral mucosa, chapped lips, cheilitis, epistaxis, brittle nails, and burning or sticky skin.

Systemic Therapy

2-Cyclosporine:

- Cyclosporine is a systemic calcineurin inhibitor. The original formulation was first approved as a post transplant immunosuppressant to prevent organ rejection.
- The more bioavailable **microemulsion formulation**, Neoral, was approved by the FDA in 1997 for the treatment of psoriasis and rheumatoid arthritis.
- Cyclosporine is efficacious for both inducing remission and maintenance therapy for patients with moderate to severe plaque psoriasis.
- SE: cumulative renal toxicity, hypertension, and hypertriglyceridemia.

Systemic Therapy

3-Methotraxate:

- Methotraxate has direct anti-inflammatory benefits due to its effects on T-cell gene expression and cytostatic effects.
- It is more efficacious than acitretin and similar or slightly less efficacious than cyclosporine
- Initial doses of 7.5 to 15 mg/week may be increased to 20 to 25 mg/week if the response is inadequate at 8 to 12 weeks, with appropriate adverse effect monitoring.
- Main SE is liver toxicity and folate deficiency.

- (**Cyclic therapy**):**Sequential therapy and rotational therapy** may minimize drug-associated toxicities; however, continuous treatment is now the standard of care for many dermatologists.

Biologic Response Modifiers (BRM)

- Currently, BRMs are often considered for patients with moderate to severe psoriasis when other systemic agents are **inadequate or relatively contraindicated**.

- BRMs are sometimes recommended for first-line therapy, alongside conventional systemic agents, for patients with moderate to severe psoriasis; however, in practice, **cost** considerations may be a limiting factor.

- BRMs may be appropriate as first-line therapy if **comorbidities** exist like active psoriatic arthiritis.

- BRMs currently available for treatment of psoriasis include adalimumab, alefacept, etanercept, infliximab, and ustekinumab.

Biologic Response Modifiers (BRM)

- The biologic agents adalimumab,etanercept, and infliximab are TNF-α inhibitors. **more rapid disease control** than other BRMs.
- **Adalimumab (Humira®)is given as 80 mg subcutaneously in the first week, then 40 mg the following week, and thereafter 40 mg every other week continuously**
- Ustekinumab is an IL-12/23 monoclonal antibody approved for the treatment of psoriasis in adults 18 years or. It selectively targets IL-12 and IL-23 (two cytokines that play a role in the pathogenesis of psoriasis).
- Alefacept was the first BRM to receive approval for the treatment of psoriasis, CD4 T lymphocytes can be depleted (less potent)
- **Main SE**: increased risk of infection and development of autoimmune diseases such as multiple sclerosis, and skin cancer

Phototherapy

- an **old important treatment** modality for psoriasis

- UVB is given alone. UVA is generally given with a photosensitizer, such as an oral psoralens, PUVA (psoralen and ultraviolet A light). PUVA is very effective in the majority of patients, with the potential for long remissions.

- UVB interferes with protein and nucleic acid synthesis, decreasing proliferation of epidermal keratinocytes.
- UVA has similar effects on epidermal keratinocytes with deeper penetration into the dermis.

- SE: Long-term use can lead to photoaging, mutagensis

Remarks

Before applying topical therapy, **wet the lesion with water for about 5** min to hydrate epidermal cells

Duration of therapy is until clinical improvement, then as frequently needed to control the condition.

In general, to determine the **quantity of topical agents** required, the fingertip unit can be used. One approximately 500 mg which is sufficient to cover one hand (front and back) or about 2% BSA. The trunk (front and back) is about 30% BSA; to cover the entire trunk once, about 15 fingertip units, or 7500 mg (7.5 g), would be required.

Prevention:

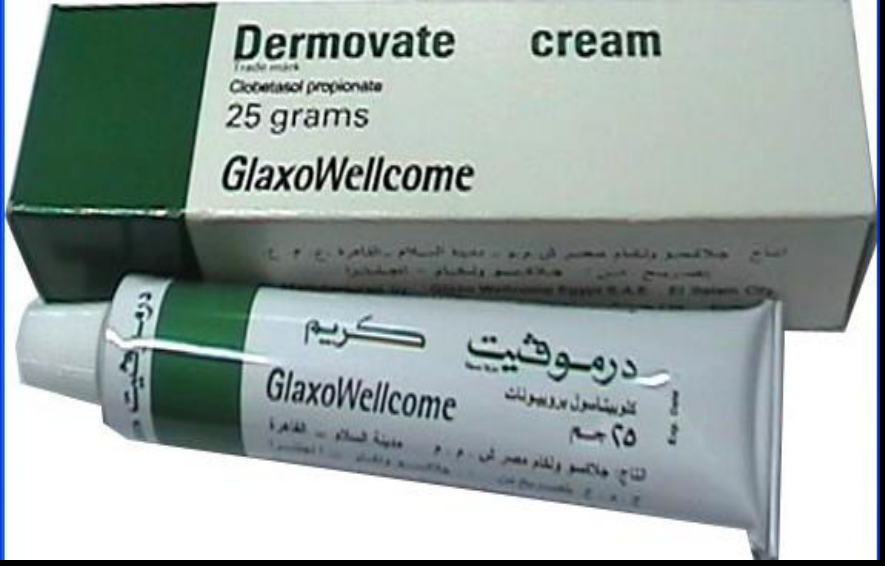
- avoid trauma

-avoid **stress,** outbreak is often triggered by mental stress. As, it is most unlikely to appear on the face. It should not prevent you from enjoying a normal life.

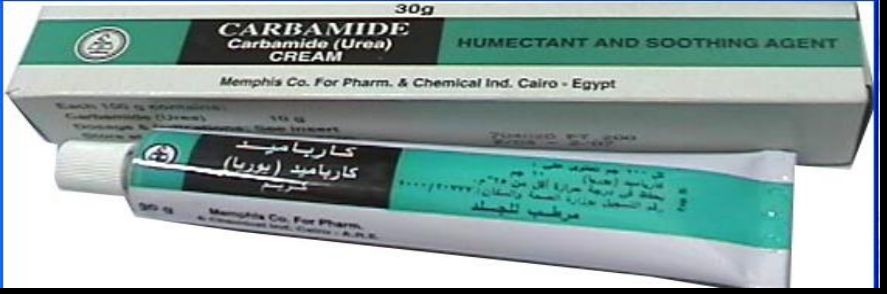
-Avoid sunburn.

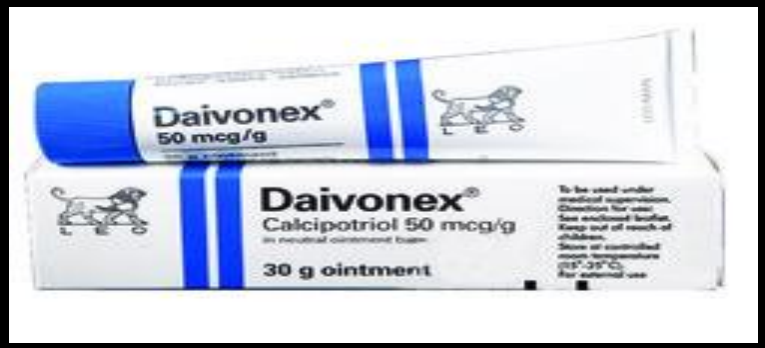
-It is worse in winter, due to the relative lack of sunlight.

-avoid scratching (bleeding risk)



Topical Corticosteroids





Topical treatment

Systemic treatment

Seborrheic dermatitis

Etiology:

Hyper proliferative disorders caused by **excessive sebum** production linked to **Malassezia** furfur, a common yeast which produce free F.A. from sebum which in turn irritate the skin.

Symptoms:

- **Excessive** Yellowish, greasy(oily), scaly patches (flaking) of skin with pruiritis on the scalp, face, ears and chest

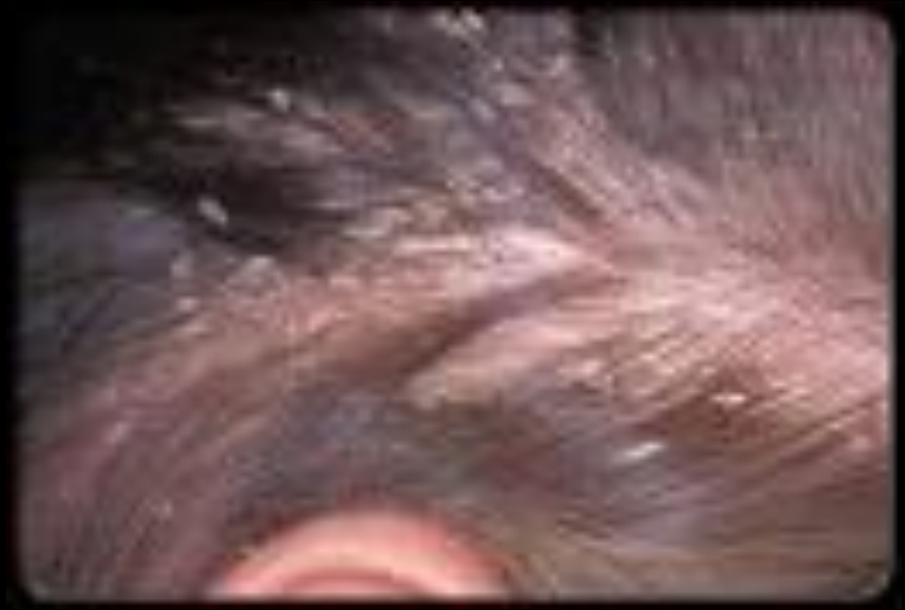
Common in infants, Affects **3%—5% of population**; two peaks, one in infancy and the other post puberty; improves in summer.

Diagnosis: location, presentation, age

Differentiate from dandruff (only to hairy area)

7-Seborrheic dermatitis

Dandruff



Treatment(OTC)

Like dandruff, in addition to CS, coal tar, salicylic acid

1-:Ketoconazole antifungal (Nizoral, Nizapex) ®

daily then taper with improvement

Oral antifungals in more resistant cases: ketoconazole 200 mg daily x 10 d.

Ex2: Sulfur by anti fungal "1.8 – 3%"

Ex3: Mild topical steroids (often combined with topical antifungals) or steroid-sparing immunomodulators (tacrolimus)

Treatment

Ex4: Coal tar (Poly tar) ®

Act by reducing the No. & size of epidermal cell produced by body.

Avoid exposing to sunlight after application to avoid sunburn

Ex5: Zn pyrithrin (Zincopan) ®: Antifungal.

Ex6: Selenium sulfide (selsun blue) ®

Reduce cell turnover rate.

Ex7: Salicylic acid:

Lower PH tissue, so increase H_2O conc. in epidermal cell which soften & destroy the stratum corneum also acts as antifungal **ex (Tonoscalpine)®**

Seborrheic Dermatitis

Common dermatological lesions by locations

Face	Acne, Impetigo, Melasma, Seborrheic dermatitis, Vitiligo, Pityriasis alba Infantile eczema, Sun burns
Mouth	Canker sore, Mouth thrush (oral cadidiasis), Herpes simplex, Angular Cheilitis
Groin	Candidal intertrigo, Erythrasma, Tinea cruris, Diaper rash(contact dermatitis)
Scalp	Dandruff, Alopecia areata, Androgenetic alopecia, Pediculosis (lice) Tinea capitis, Trichotillomania
Foot	Corn and callus , Onychomycosis, Plantar wart, Tinea pedis, dry skin
Hands	Atopic eczema, Contact dermatitis, Hyperhidrosis, Psoriasis, Scabies, Common Warts, Onychomycosis, Syphilis
Limb	Atopic eczema, Cellulitis, Psoriasis, Vitilligo, uricaria, Tinea corporis
Axilla	Acnthosis nigricans, Allergic contact dermatitis, Erythrasma, Hyperhydrosis,
Trunk & abdomen	Psoriasis, Striae, Tinea corporis, Tinea versicolor, Urticaria, Scabies , Chickenpox (Varicella)
Genitalia	Herpes simplex, Psoriasis, Scabies, Syphilis (chancre), Wart, candidal infection

Finally, dermatology is the science that must be seen by naked eye, this presentation can not replace attending pharmacies and hospital to see patients