Pigmentary Skin Disorders

DR. SANAA ALHUSSEINY

Pigmentary Skin Disorders

- There are group of disorders affecting the skin pigmentation
- either in: * increase pigmentation

  OR * decrease pigmentation

- Skin colour is due to a mixture of the pigment (melanin) & oxyhemoglobin (in blood) & carotene (in the stratum corneum & subcutaneous fat).

Decreased Pigmentation

- It is either partial (i.e. hypopigmentation) or complete (depigment).
- **Causes:**

1. Vitiligo
2. Halo nevus: a variant of vitiligo
3. Post-inflammatory hypopigmentation: many diseases tends to have this e.g. psoriasis, HSV, seborrheic dermatitis
4. Pity riasis alba: it is common among children, usually in the face, also the trunk, it is characterized by 2 stages : A. Mild erythematous stage: red macules.
   B. White stage: hypopigmented.

   It is caused by dryness, especially with excessive wash with soap & exposure to sunlight

Causes

5. Chemical Leukoderma: usually in persons dealing with phenolic compounds or it is derivatives in industries or else where.
6. Nevus depigmentosus: white patch with irregular border, may be 2 patches, present at birth & remains for life.
7. Post. Burn leukoderma: it looks like vitiligo (Hx is important).

Causes

8. Idiopathic guttate hypomelanosis:

- Presented with small whitish macules (2-3 mm) similar to vitiligo on sun-exposed areas (forearms, leg).
- common in middle aged person.
- Rx: similar to vitiligo (poor response).

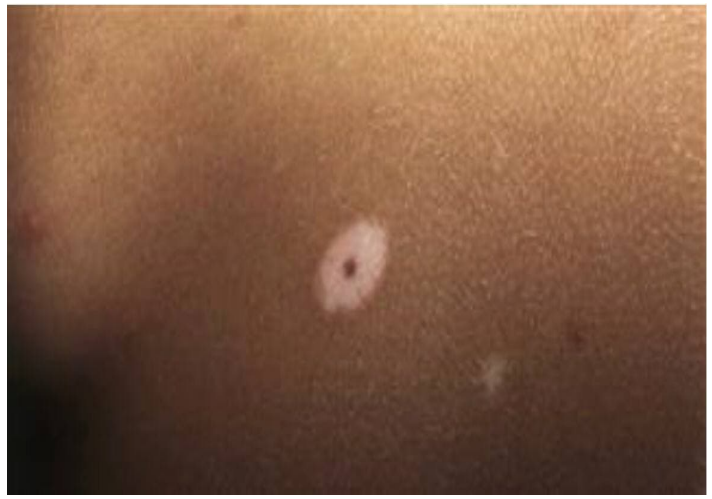
9. Steroid Leukoderma: it appears following I.M., I.V. or even intra-articular injection of steroids e.g. (triamcinolone) after which there will be areas of whiteness at the injection site & s.t. spread along lymphatics… Rx. as vitiligo

Causes

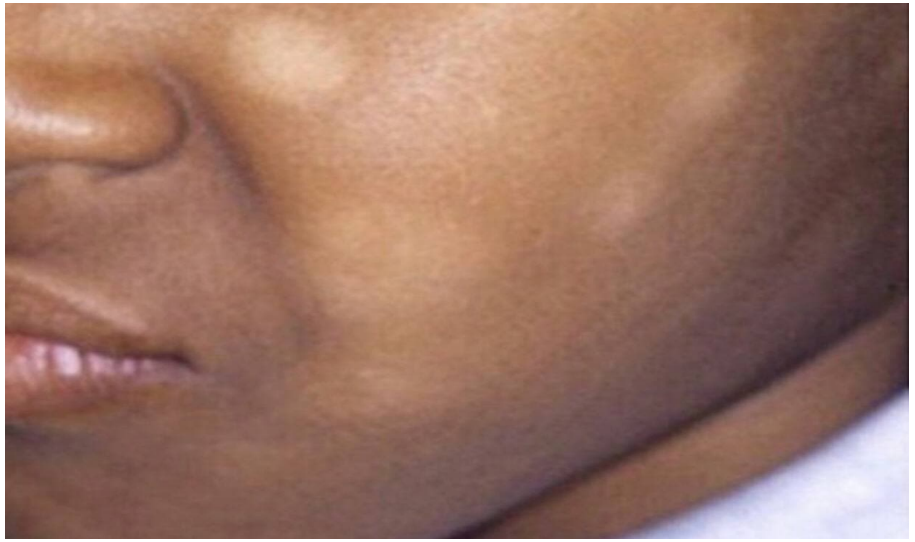
10. Tuberous sclerosis (epiloia): there will be white spots (macule or patch) = ash-leaf white patch in addition to the other criteria of epiloliale., i.e. epilepsy, Low-mentalitiy, adenoma sebacaum

11. Albinism: autosomal recessive disorder in which melanocyte fail to form pigment in the skin & hair bulb & eye due to partial or complete loss of enzyme = tyrosinase.

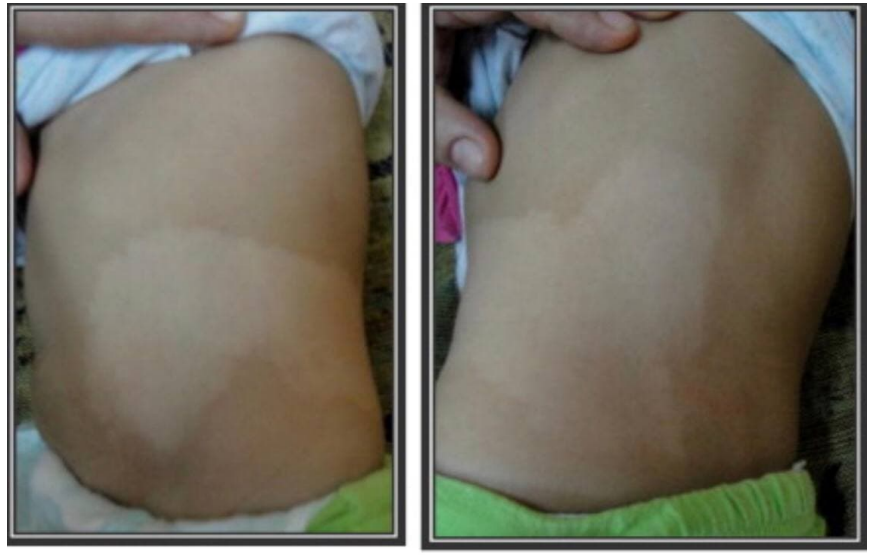
It may associated with head nodding, errors of refraction, photo-phobia & skin malignancies.



Halo nevus

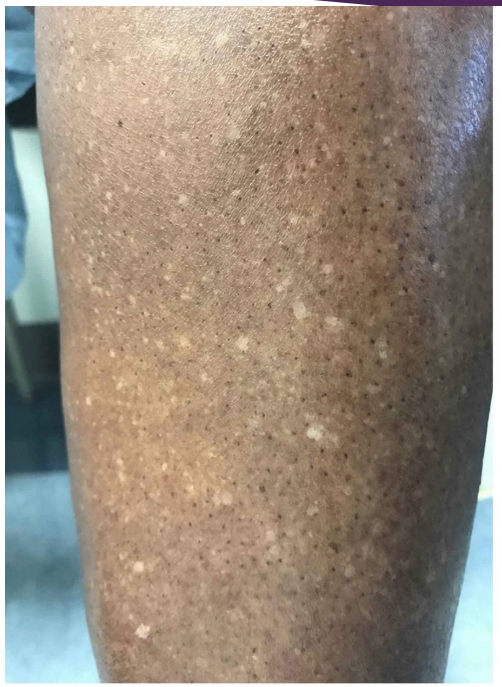


Pity riasis alba

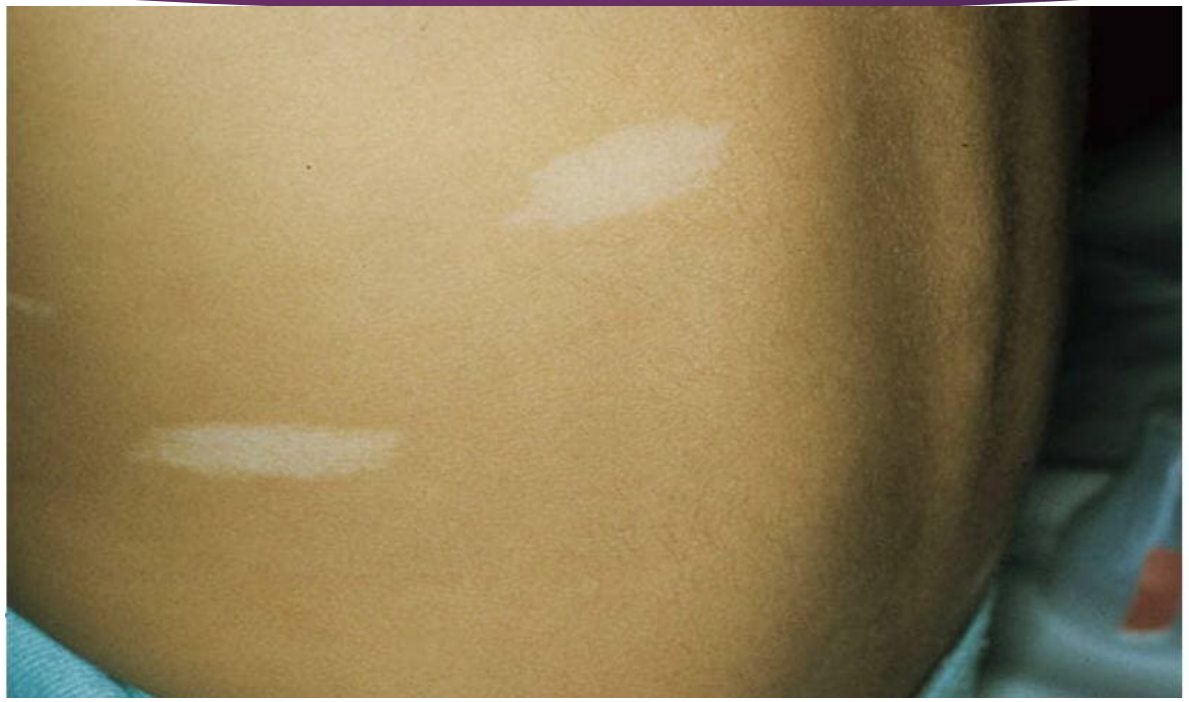


A B

Nevus depigmentosus



. Idiopathic guttate hypomelanosis



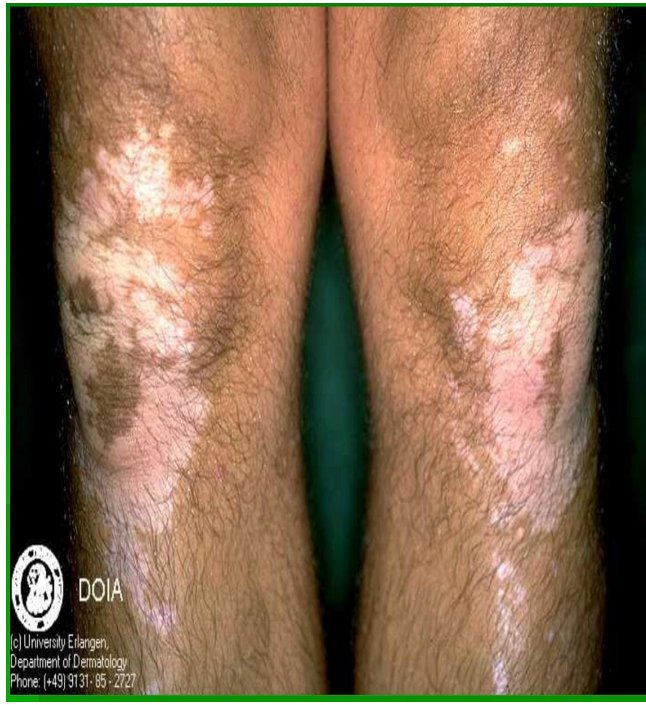
- ash-leaf white patch

Vitiligo

- It's a major common problem all over the world affecting 1% of the population & all races are equally affected.
- It is an acquired pigmentary disorder with genetic susceptibility, characterized by whitish area usurally in symmetrical pattern.
- usually there is +ve family Hx (30-40%)

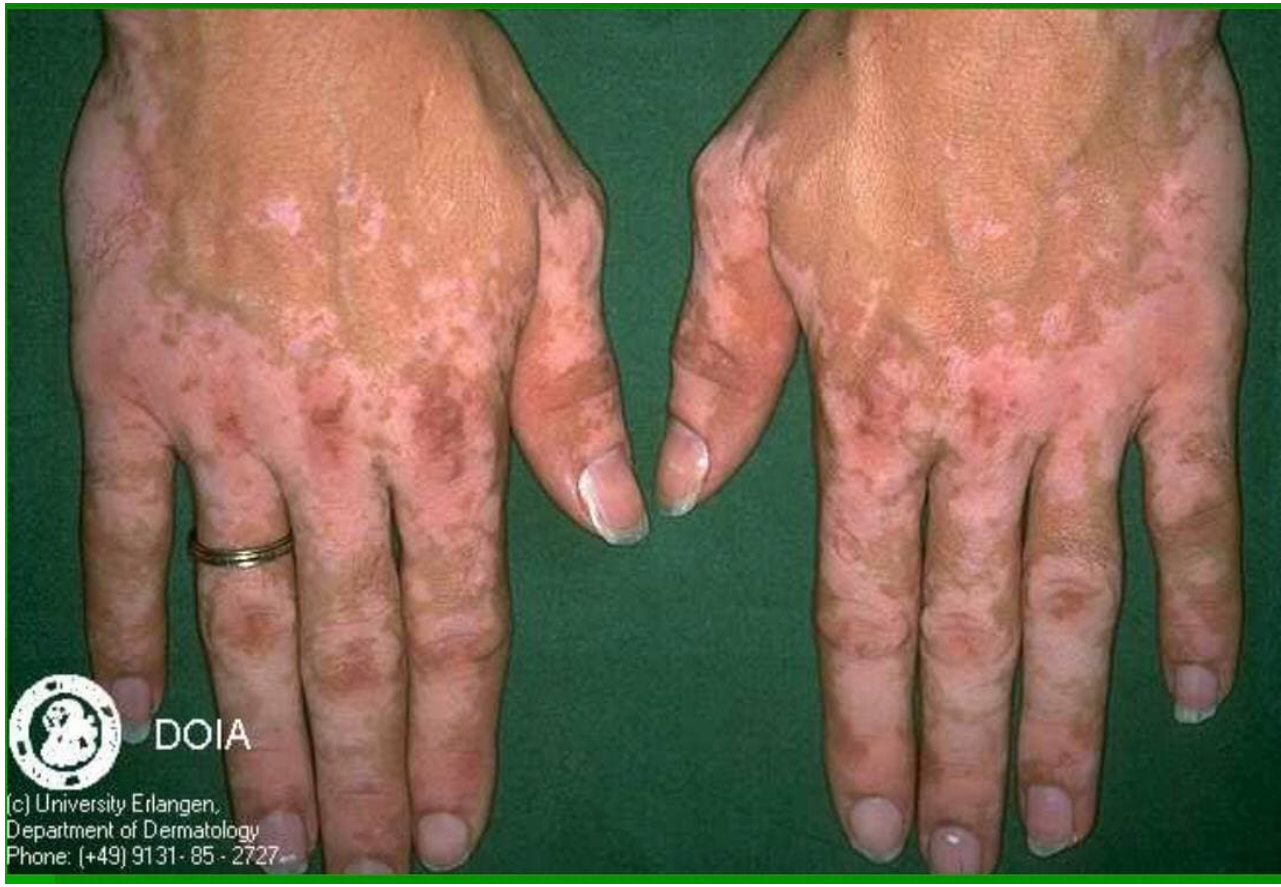
Clinical Features

- It a disease of all ages, but mainly it is a disease of young age group.
- Areas of predilection are:
    1. Peri-orificial: like mouth, ears, nose, eyes & genitalia.
    2. frictional site: hands, feet, elbow & knee.
    3. areas of trauma (Köebners phenemenon) which is also seen in: psoriasis, lichen planus.
    4. flexural: axilla, genitalia



Stages Of Pigmentation

- Stage I : whitish brown (ie. partial pig. loss).
- Stage II : milky white (i.e. complete loss).



Types Of Vitiligo

- **Focal type:** multi-focal areas of involvement on the body, usually symmetrical.
- **generalized type:** disseminated with few areas of normal skin.
- **Segmental vitiligo:** semi dermatomal distribution i.e. localized in one side e.g. face, trunk, limb & remain stationary.
- **Halo nevus;** vitiligo around a nevus, which get Lighter in color graudually till complete loss of pig.
- **Grayness of the hair:** pre-mature grayness before the age of (40) ; either patchy or diffuse
- **Chemical vitiligo:** due to exposure to chemical agents or drugs, rubber industry, or phenolic compounds which affect or toxic to melanocyte ----- vitiligo.

Etiopathogenesis

- It is an auto-immune disease which is cell-mediated with humoral disorder; evidences for that are; 1. Association with other auto – immune diseases e.g. AA, pancreatitis, thyroidits,

  Diabetes mellitus …. etc.

  2. presence of organ specific auto antibodies as: antipancreatic, antiparietal,

  antithyroid …. Also anti-melanocytic antibodies.

  3. Histopathologically: there is inflammary changes with T-cell cytotoxic

  infiltration surrounding the melanocyte.

Treatment

- There will be response to Rx in 60% of patients & the response could be partial or complete & could be rapid or slow. It is s.t. take several months or even years.

1. **Psoralin compounds:** psoralen is an extract from a plant called Amni magus. Psoralin is used either with sunlight or artificial uv light (UVA) that is why it called PuvA (p+ UVA). This could be topical PUVA or systemic PuVA.
2. **Systemic steroids:** especially in early & evolving disease.
3. **Topical steroids:** also effective
4. **Vit. C & Vit. B12 & Ca** are important elements with some benefit.
5. If vitiligo is near-universal: monobenzyle ether of hydroquinon (= Benoquin) is considered to depigment the remaining normal skin area.

Prognosis

- unpredictable

  Disease duration >5 yr

  Early onset

  +ve Köebner

  Diffuse disease

  → all carry bad prognosis

Hyperpegmentation

- Here there will be an increase in the melanin in the epidermis (basal layer) & in the dermis.
- When melaninis produced, it will be taken by keratinocyte, but in the dermis it is take by macrophage (melanophage)

Causes:

1. Melasma, Freckling, lentingines.
2. Post –inflammatory eg: Lichen plans, trauma
3. Lifa disease
4. Becker's nevus
5. Phytophoto dermatosis: common in children & females after eating fruits (which contain photo sensitizers) around the mouth or in the hand hyperpig.

Causes

6. Frictional melanosis: occurs in flexures & area of friction

7. Berloque dermatitis: due to the use of some type of perfumes (contain psoralin) --- pig. on sides of neck

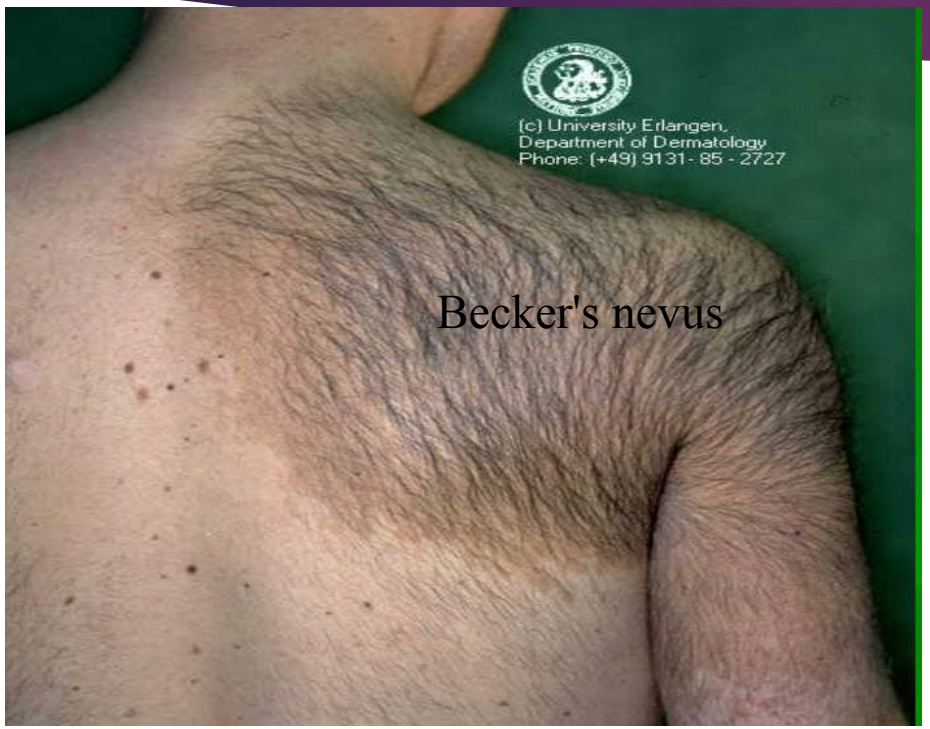
8. Reihls melanosis: dermal melanosis, usu. due to cosmetics

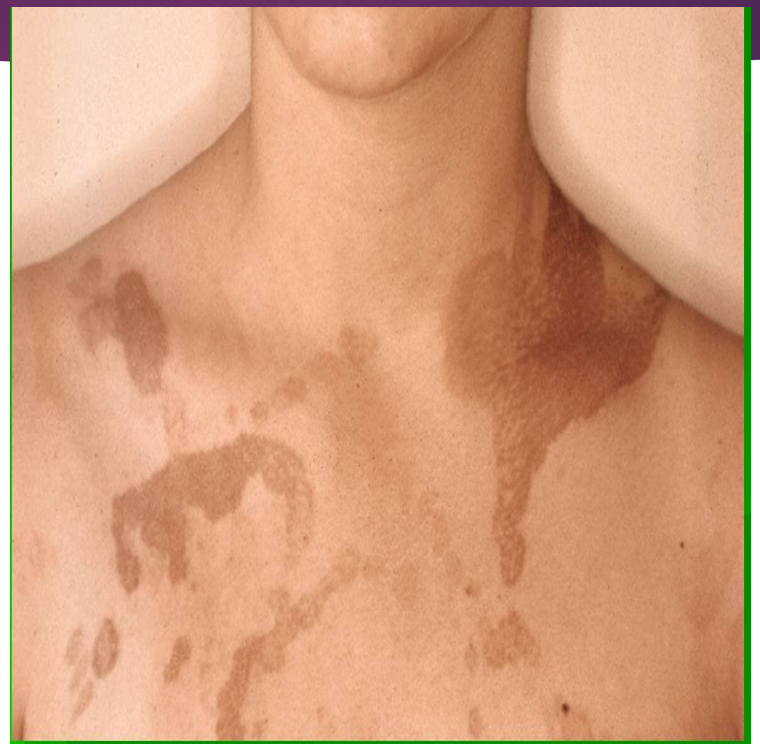
9. Endocrine problem : generalized hyperpig.

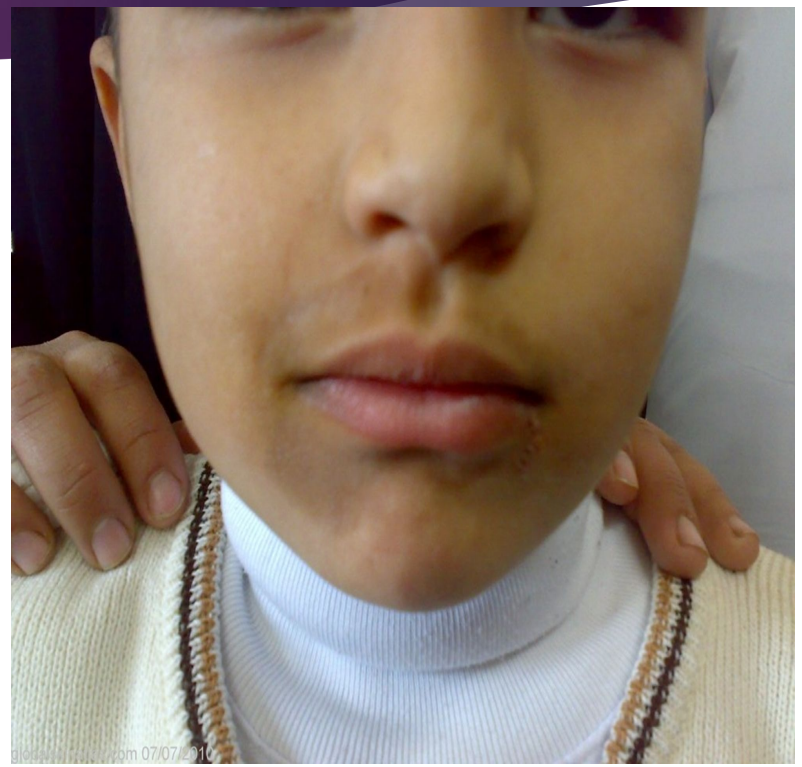
- Addison's ------ with oral involvement
- Cushing syndrome ---- without oral involvement
- Thyrotoxicosis

Causes

10. Neoplasm: pheochromocytoma, carcinoid syndrome -----
11. Chronic illness: e.g. chronic infection, CA, chronic renal failure
12. Metabolic: haemochromatosis, porphyria
13. Nutritional: malabsorption, malnutrition, vit-deficiency A, C, folic, pellagra
14. Drugs: phenothiazines, amiodarone, anti-epileptic, M. leprase has a predilection.


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727
Becker's nevus





Lifa Disease

- It is commin in Iraq among slim people especially, female
- It occurs over bony prominence : elbow, shin, extensor surface of forarms, clavicles … etc
- It is caused by trauma by using lifa with stong washings ----- sequeezing the skin against the bone leading to the basal layer ------melanin will go downward ------ ---- taken by macrophage (melanophange) ----- dermal melanosis.
- The area will be dirty looking, encourging the fernale to increase forceful washing by lifa ----- more dermal melanosis.
- Rx is difficults, stop using lifa for 6-12 month, topical steroids, topical bleaching.

Melasma (Chloasma)

- It is light to dark brown pigmentary disorder of the face.
- It develops slowly & symmetrically.
- It ís usually seen in females but could happen in males .

Causes:

1. Genetic susciptibility.

2. It is a disease of reproductive life . Neither seen in children nor in elderly i.e. there may be a hormonaI role in the disease

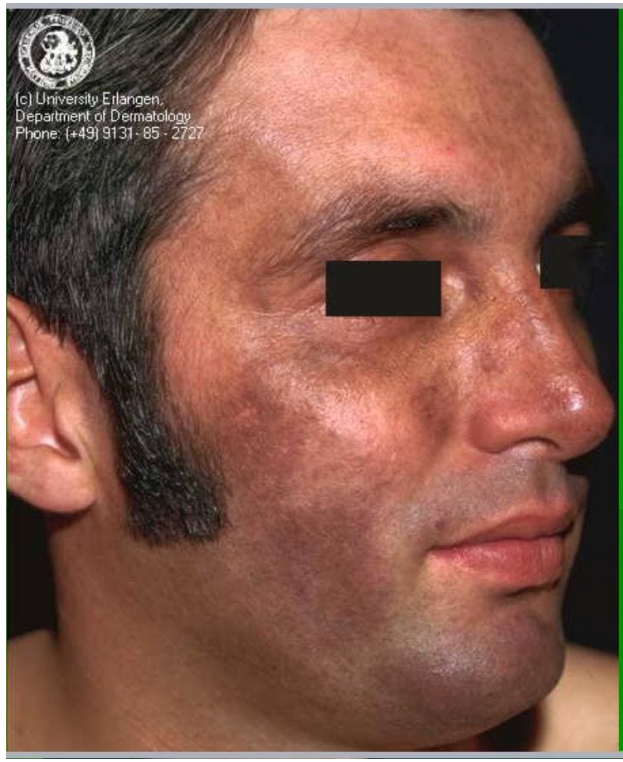
3. Their may be a role for sunlight, pregnancy, ocp & cosmetics

Exacerbating factors

- Sunlight
- Premenstrual
- Hair epilation
- Pregnancy
- Friction
- OCP
- Cosmetic
- Emotional
- Idiopathie

Clinical Features

- It is light or dark brown pigmentation of the face ------epidermal type
- If it is dark bluish pigmented lesion ---- dermal
- It may affect forehead, cheeks, nose, upper lip
- Partial or diffuse (all of the face)



Mangement

1. Avoidance of sunlight exposure or sunscreen
2. Bleaching agents:
   * Temporary bleaching - Hydroquinone 2% or 4%
     - Retinoic acid
     - Kojic acid
     - Azealic acid
     - Glycolic acid
   * Perminant bleaching – vitiligo like by:
     monobenzone = monobenzyle either of hydroquinone (Benoquin 20%)
3. Chemical peeling (less rewarding)
4. Surgical peeling (dermoabrasion) esp. in dermal m.
5. Laser Rx (also there is failure & repigmant).


Thank you!!!!!!