Vitiligo

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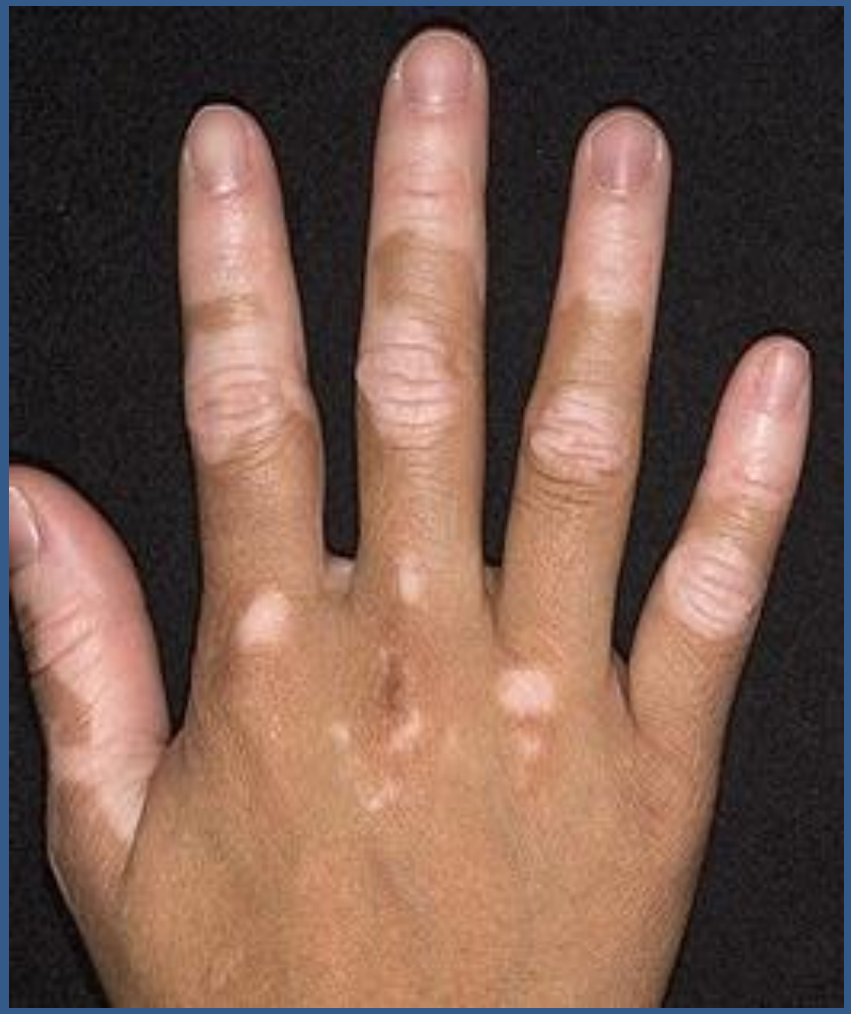
Vitiligo

- Acquired pigmentary disorder
- Depigmented macules and patches

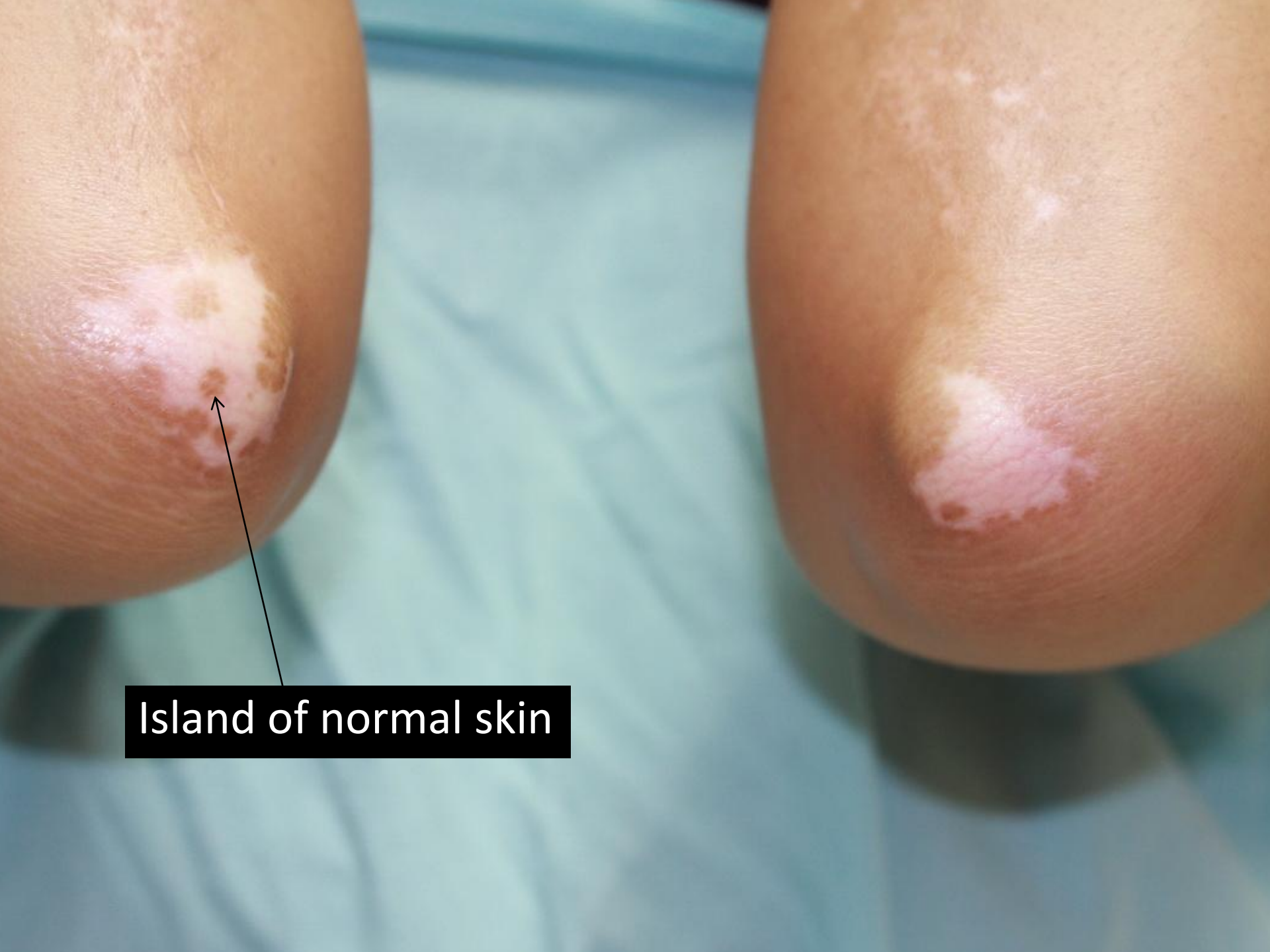


Prevalence

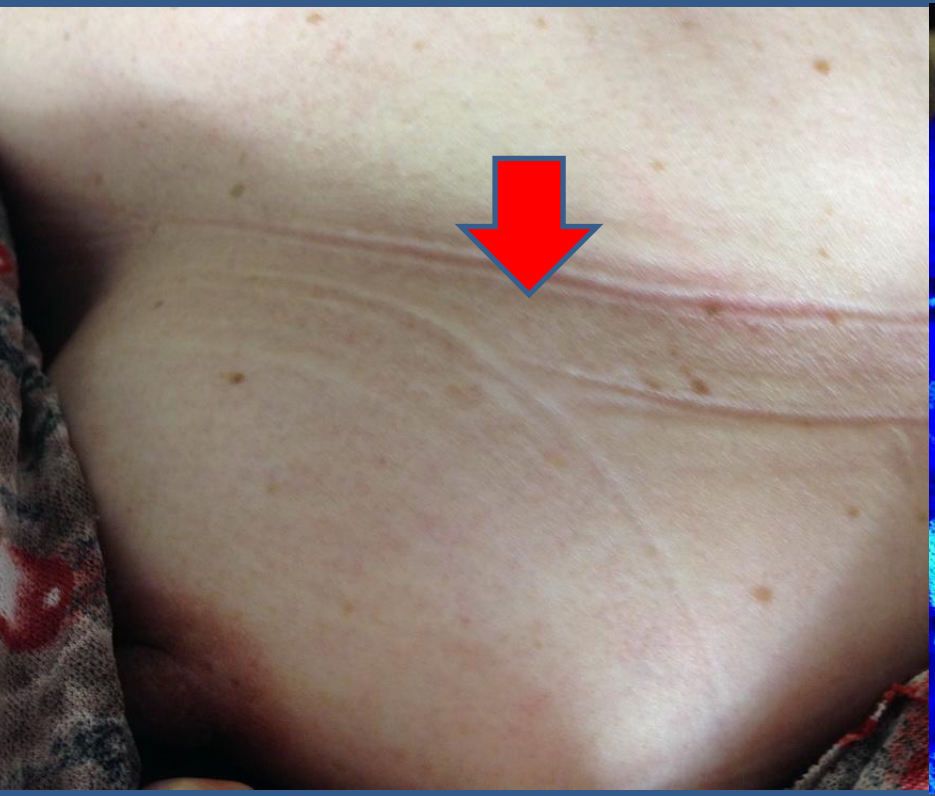
- The worldwide prevalence of vitiligo is up to ~2%

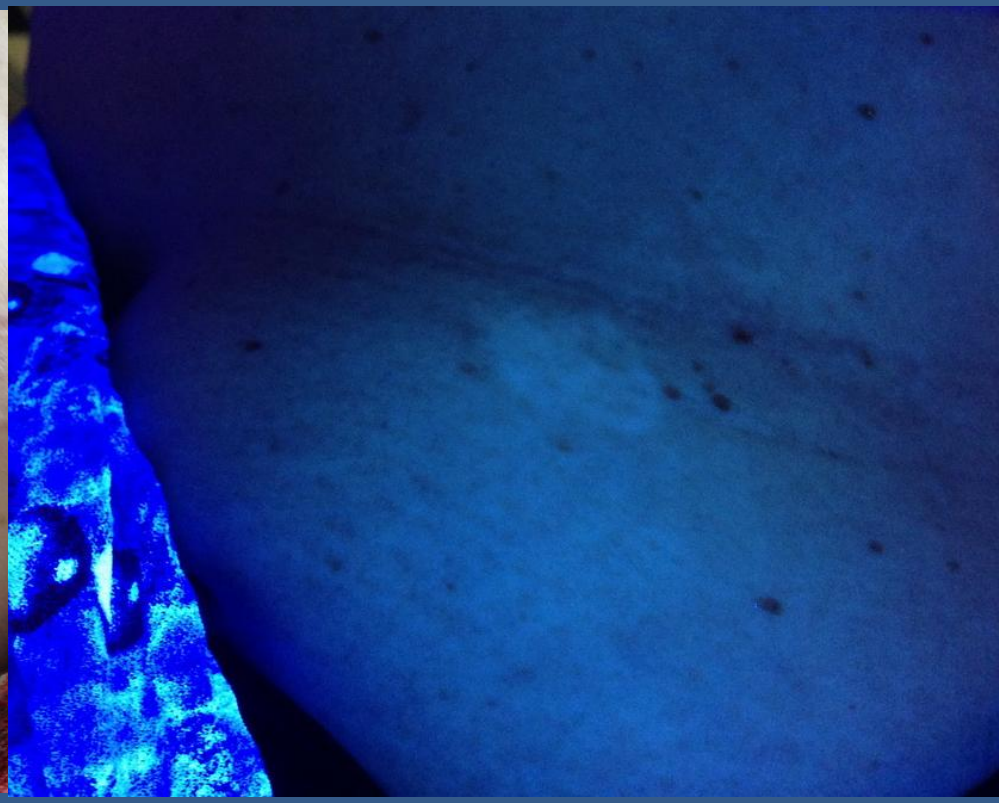
Clinical manifestations

- Asymptomatic depigmented patches and macules


Island of normal skin

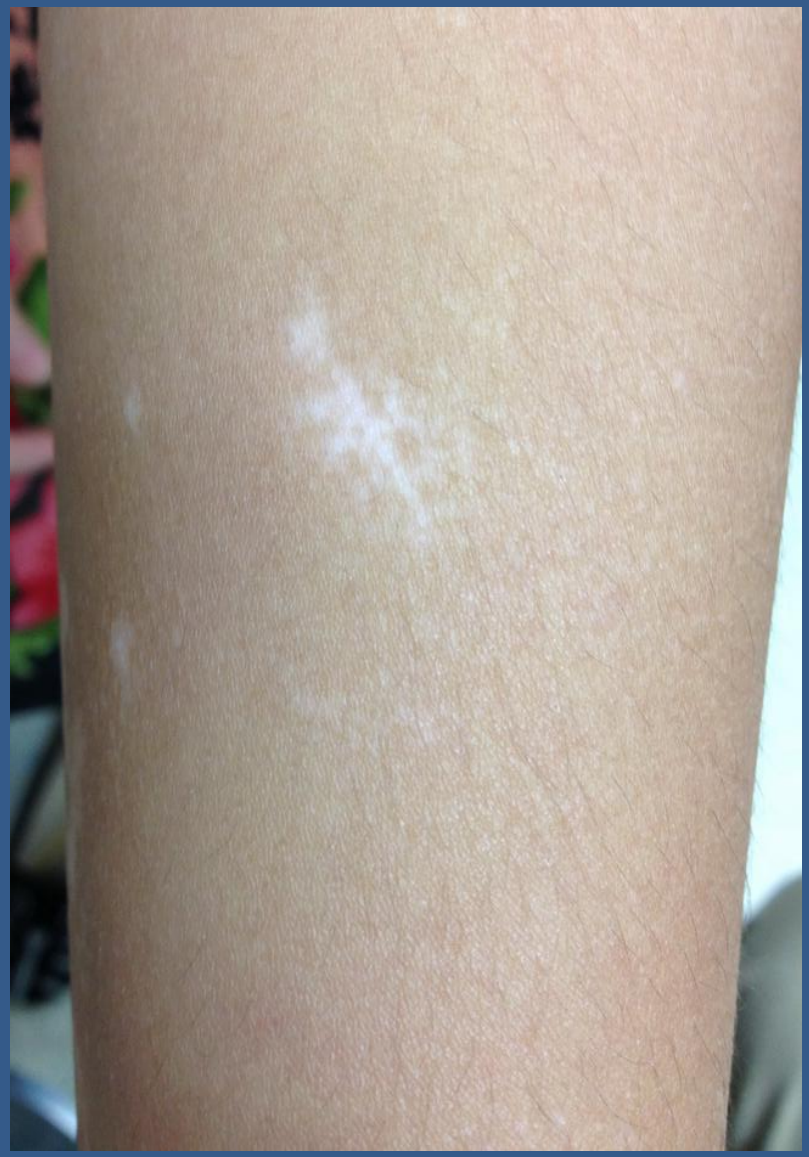
Wood's light





Clinical manifestations

- Koebner's phenomenon (the development of lesions at sites of specifically traumatized uninvolved skin of patients with cutaneous diseases)

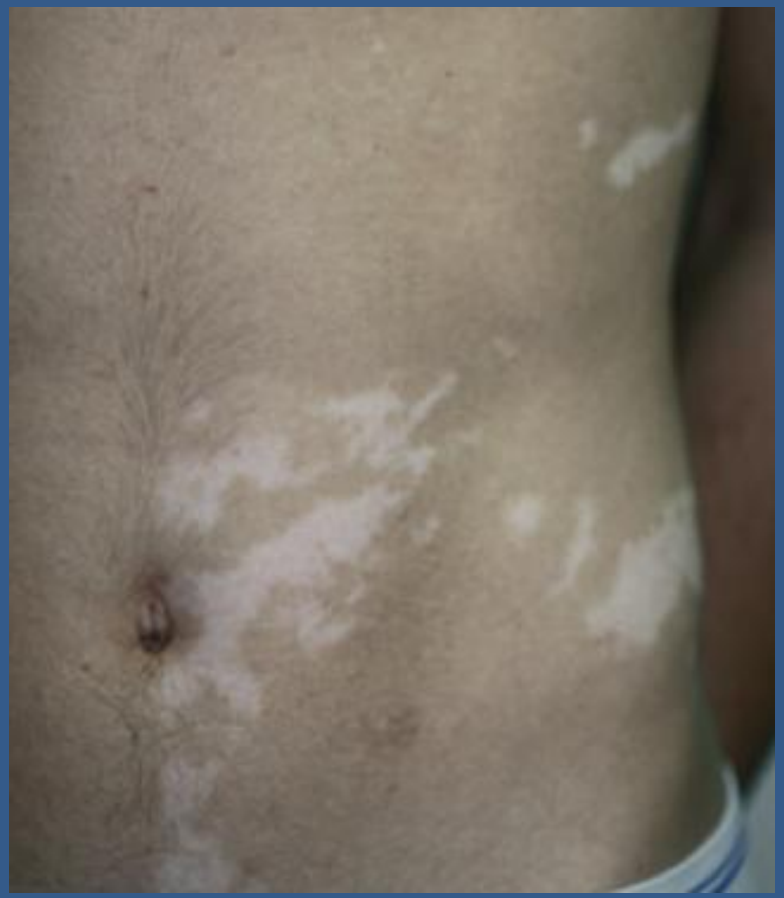


Classification of vitiligo

- Segmental vitiligo
- Non-segmental vitiligo
- Unclassified: mucosal, focal

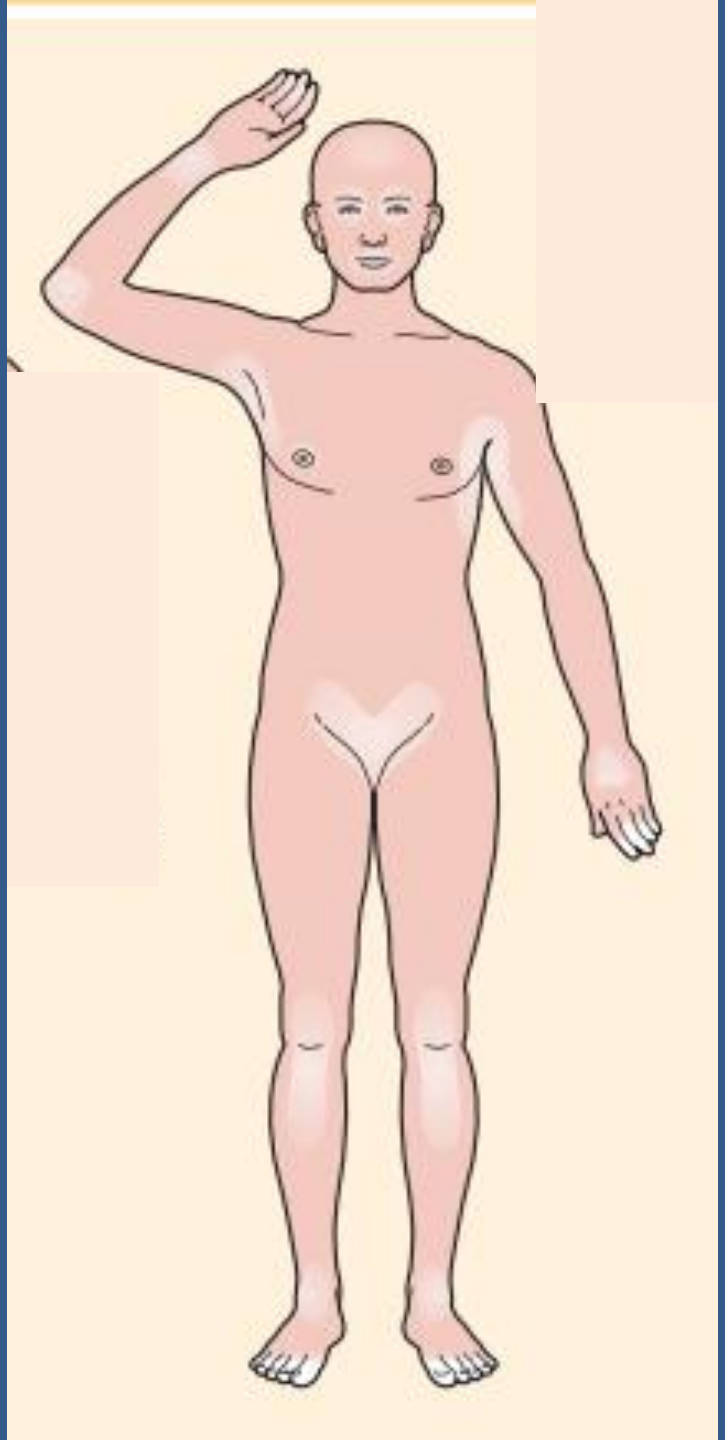
Segmental vitiligo

- Mono-segmental vitiligo: most common
- Bi-segmental vitiligo
- Plurisegmental vitiligo



Non-segmental vitiligo

- Typically evolves over time (distribution, extension) often involving both sides of the body with tendency toward symmetrical distribution
  - -acrofacial (face, head, hands, feet)
  - -generalized
  - -universal: 80-90% of BSA
  - -mixed vitiligo: initial SV followed by bilateral NSV patches



NSV
(Generalized vitiligo)

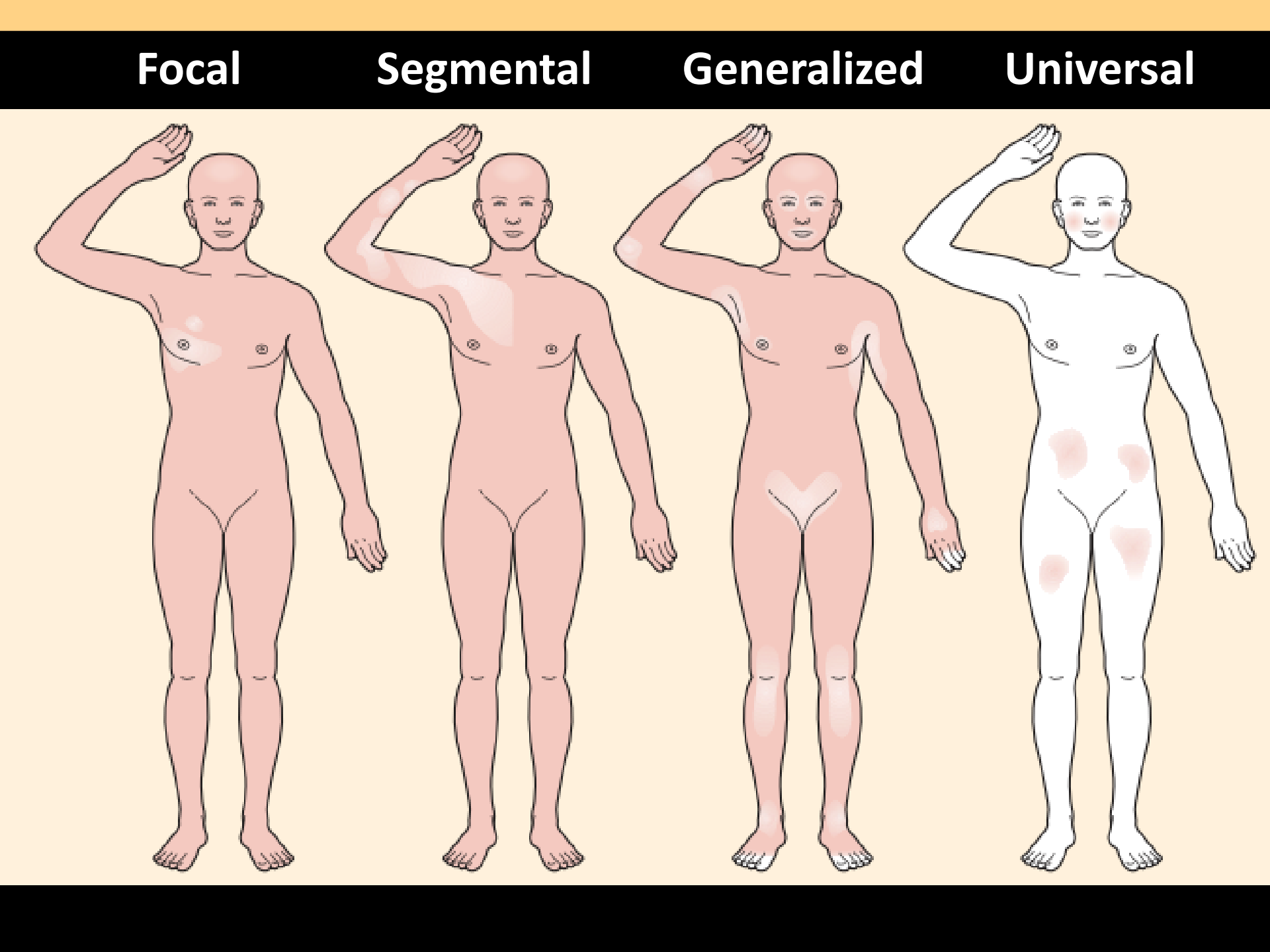
- Face: periorbital, perioral
- Trunk, axilla, groin, umbilicus
- Extremity: elbow, wrist, hand, feet

Unclassified: mucosal vitiligo

- An isolate involvement of oral and/ or genital mucosa for at least 2 years F/U
- When mucosal vitiligo occurs in the context of NSV, it is classified as NSV
- Differential diagnosis: lichen sclerosus

Unclassified: focal vitiligo

- Acquired, small, isolated depigmented lesion that does not fit a typical segmental distribution and has not evolved into NSV after a period of 2 yr
- The diagnosis should be considered only after having ruled out all other diagnoses, and a biopsy may be helpful


Focal
Segmental
Generalized
Universal

Pathogenesis

- Autoimmune: best supported theory
- Neurohumoral: segmental vitiligo
- Oxidative stress
- Melanocytorrhagy

Vitiligo and autoimmune diseases

- Patients with generalized vitiligo, especially when familial, are more likely to have autoimmune disorders than those with SV

Common associations

More common associations

- Addison disease
- Alopecia areata
- Atopic dermatitis
- Autoimmune thyroid disease
- Chronic urticaria
- Diabetes mellitus
- Halo nevi
- Hypoacusis
- Hypoparathyroidism
- Ichthyosis
- Ocular abnormalities
- Pernicious anemia
- Psoriasis
- Rheumatoid arthritis

J Am Acad Dermatol 2011; 65: 473-91.

Autoimmune thyroid disease (ATD)

- Median prevalence of ATD in vitiligo
  -children: 6.89% (5.79-12.7%)
  -adult: 18.6% (13.7-22.9%)
- The risk of ATD in vitiligo patients seems to increase with age

Br J Dermatol 2012; 167: 1224-35.

Less common associations

Less common associations

- Acrokeratosis paraneoplastica Bazex
- Alezzandrini syndrome
- [illegible]
- Dysgammaglobulinemia
- HIV
- Inflammatory bowel disease
- Kabuki syndrome
- Kaposi sarcoma
- Melanoma
- MELAS syndrome
- Morphea
- [illegible]
- [illegible]
- Schmidt syndrome
- Systemic lupus erythematosus
- Turner syndrome
- Twenty-nail dystrophy
- Vogt-Koyanagi-Harada syndrome

ANA is positive in up to 12.4% of patients

J Am Acad Dermatol 2011; 65: 473-91.

Recommendations

- TSH
- ANA
- Thyroid antibodies: can present up to 7 years before clinical diagnosis of autoimmune thyroid diseases

J Am Acad Dermatol 2011; 65: 473-91.

Neurohumoral hypothesis

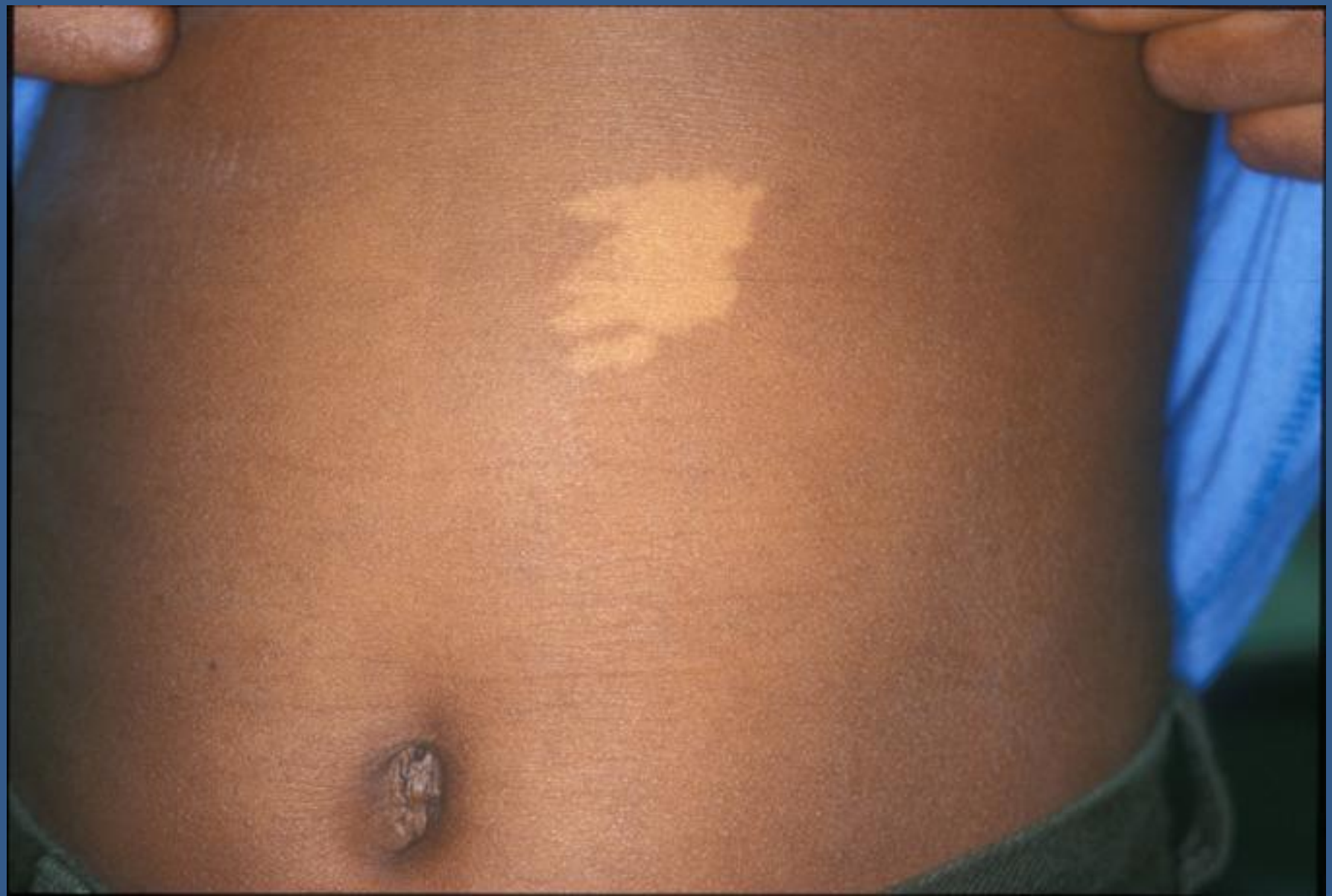
- Melanocytes and nerves arise from neural crest cells
- Lesions may also exhibit increased levels of NE and decrease AchE
- Alteration in neurotransmitters may cause
  - -melanocyte cytotoxicity
  - -vasoconstriction, cell hypoxia

Arch Dermatol Res 1996; 288: 14-8.
Acta Anat 1989; 136: 139-41.

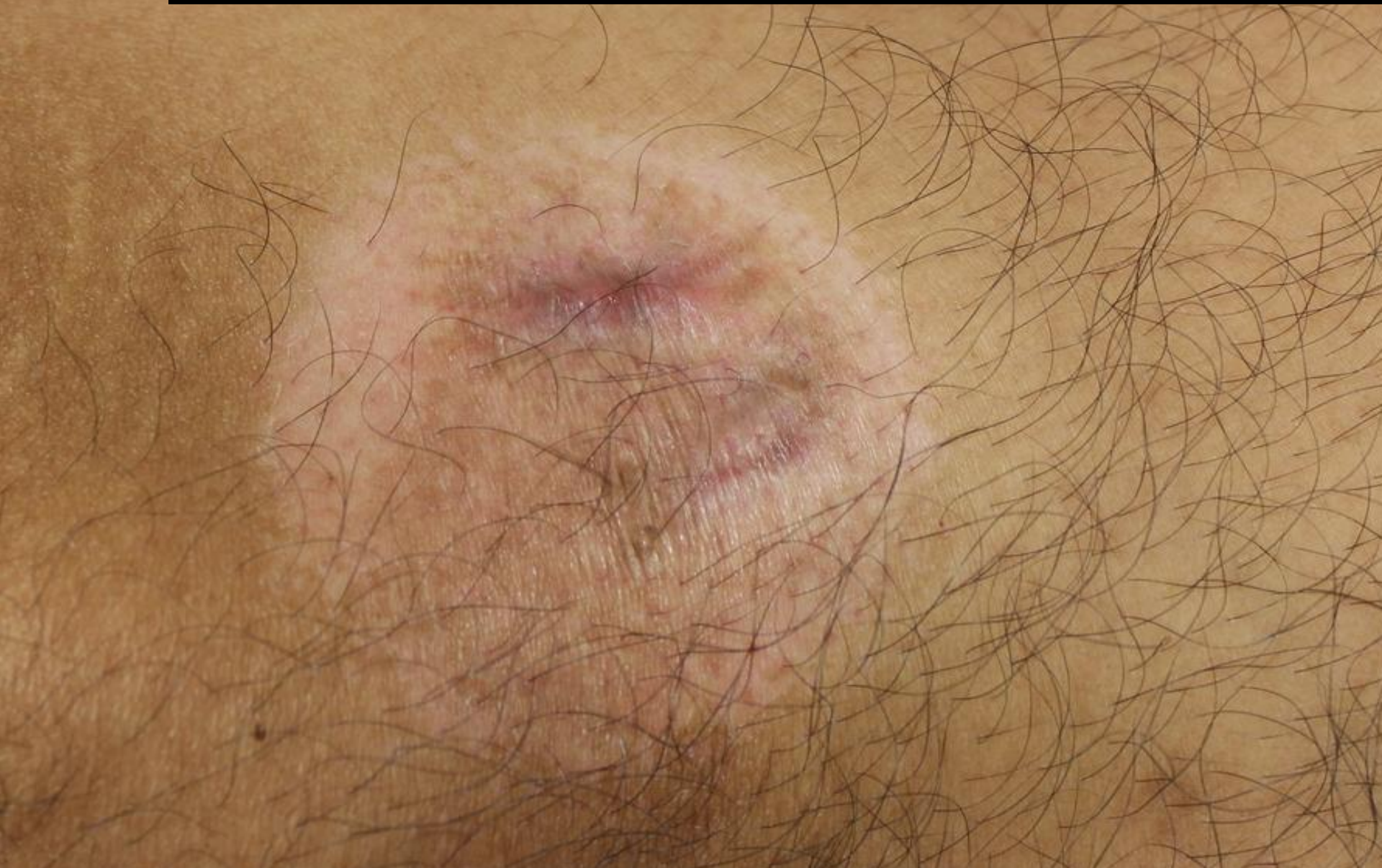
Differential diagnosis

- Depigmented lesion
  - -nevus depigmentosus
  - -chemical leukoderma
  - -postinflammatory depigmentation
  - -lichen sclerosus
  - -idiopathic guttate hypomelanosis
  - -vitiligo-like DLE

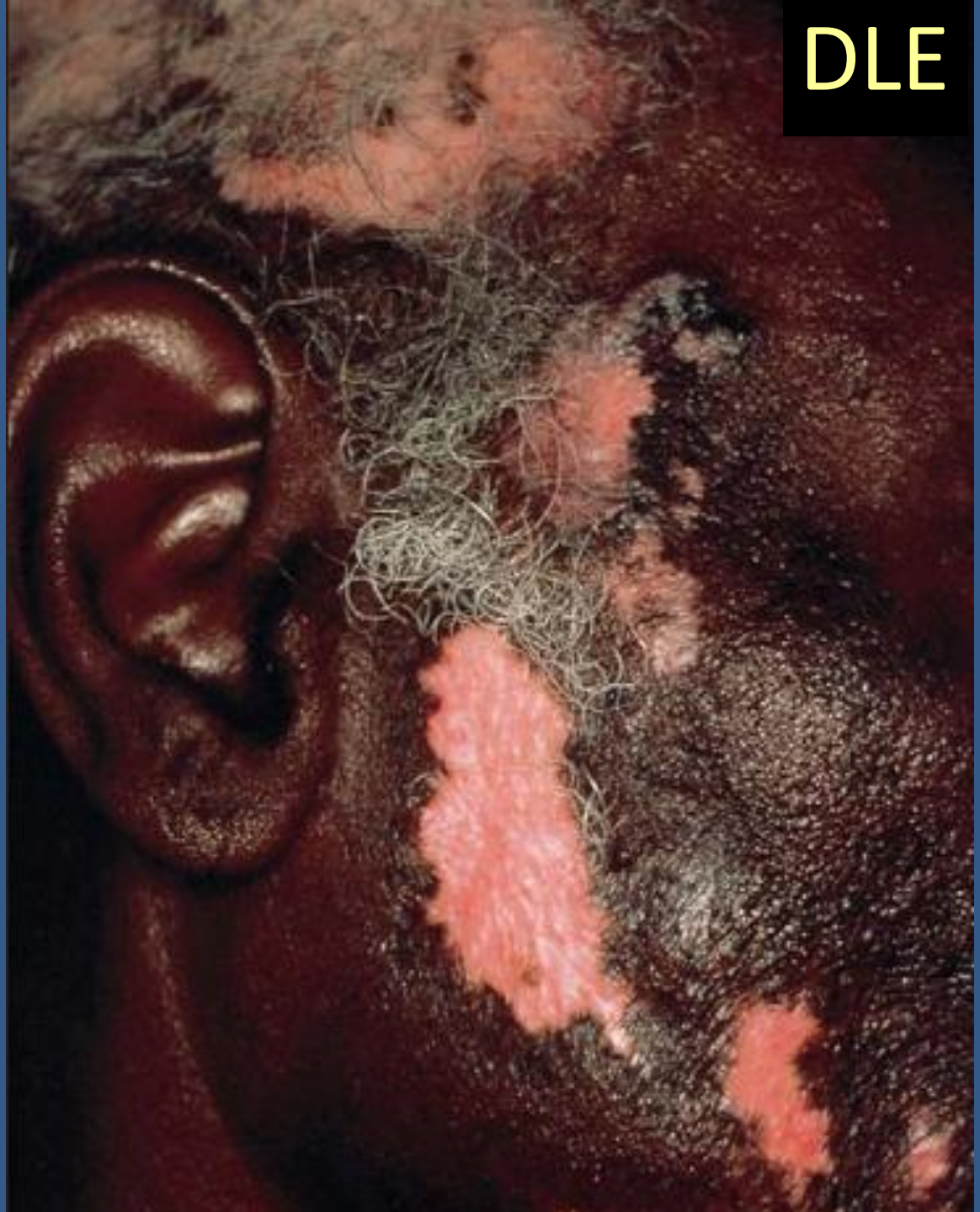
Nevus depigmentosus

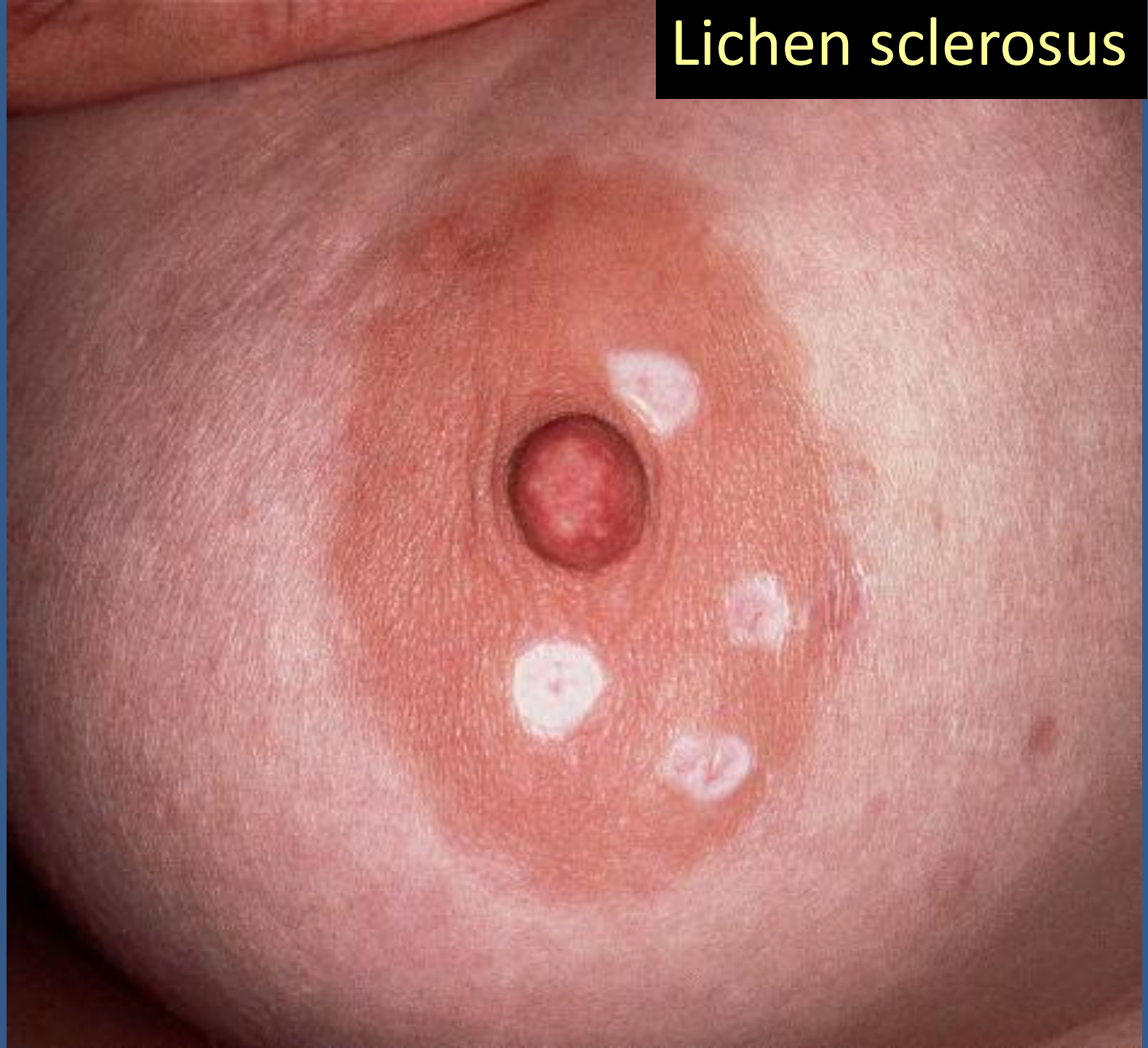


Chemical leukoderma: hydroquinone

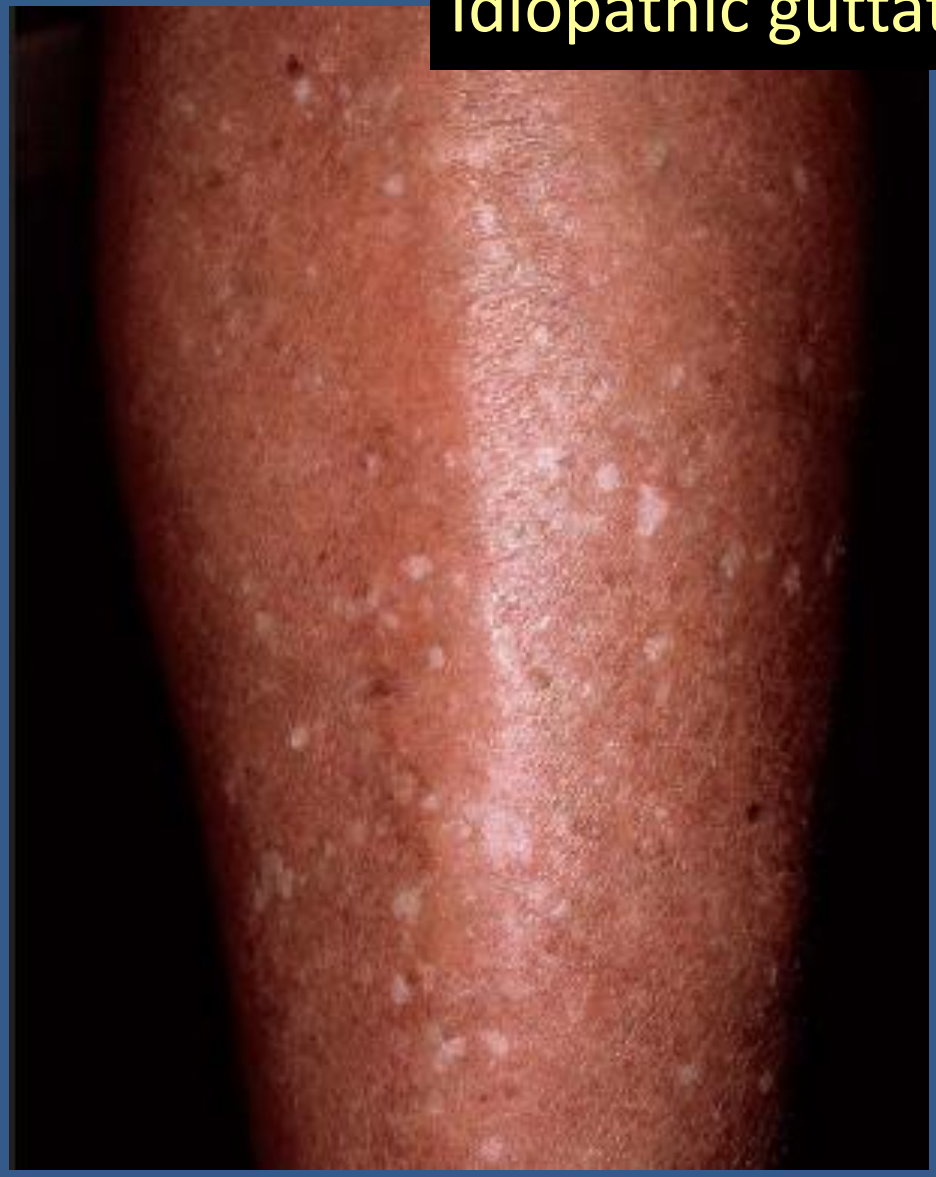


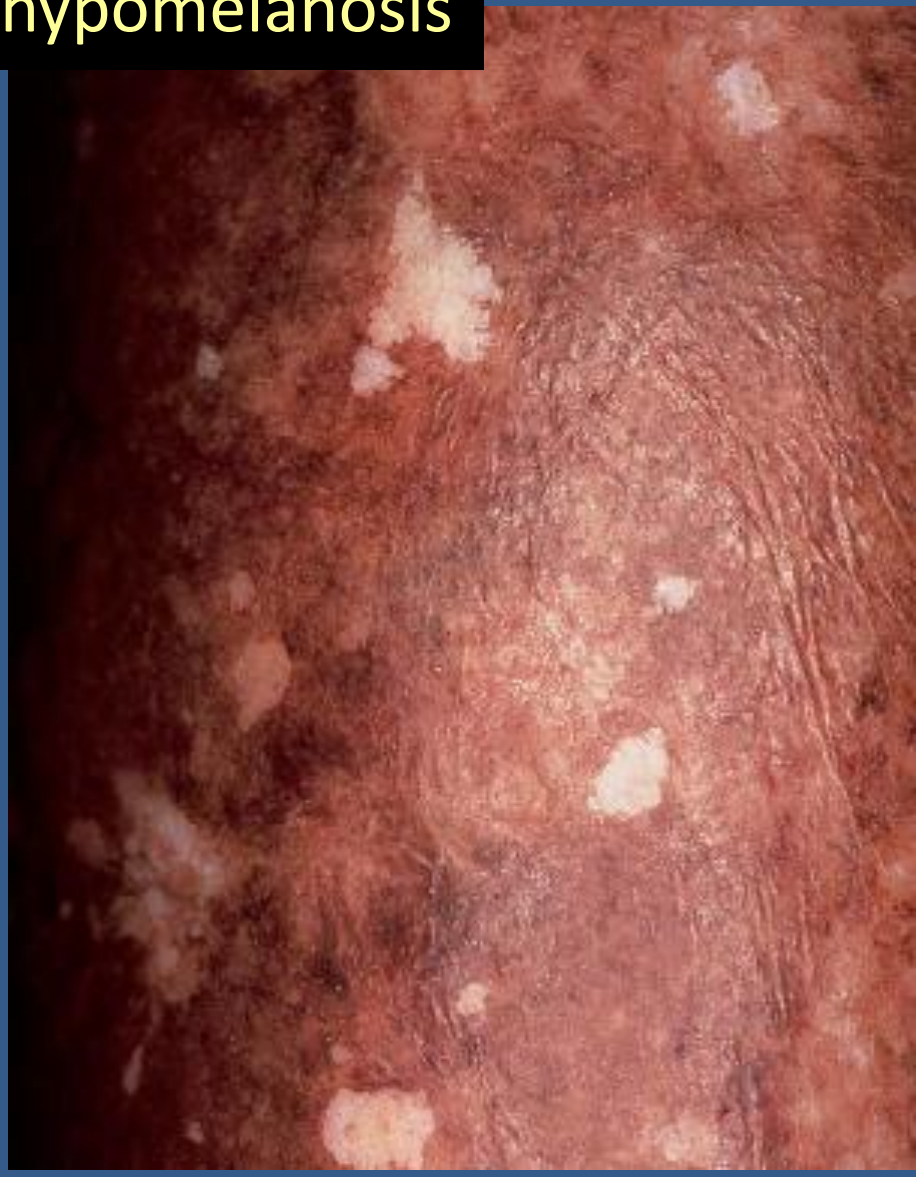
Postinflammatory depigmentation in severe atopic dermatitis

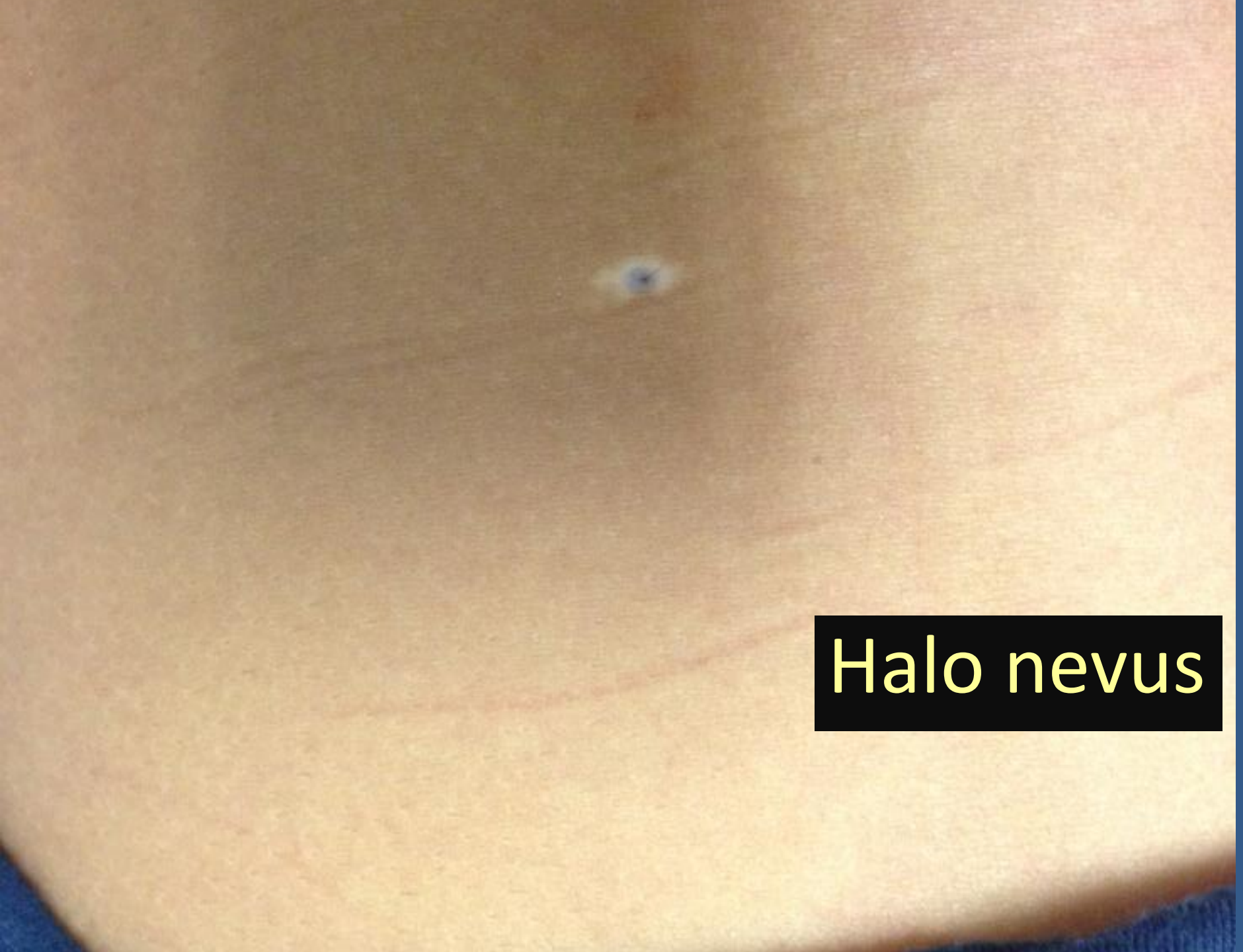

DLE

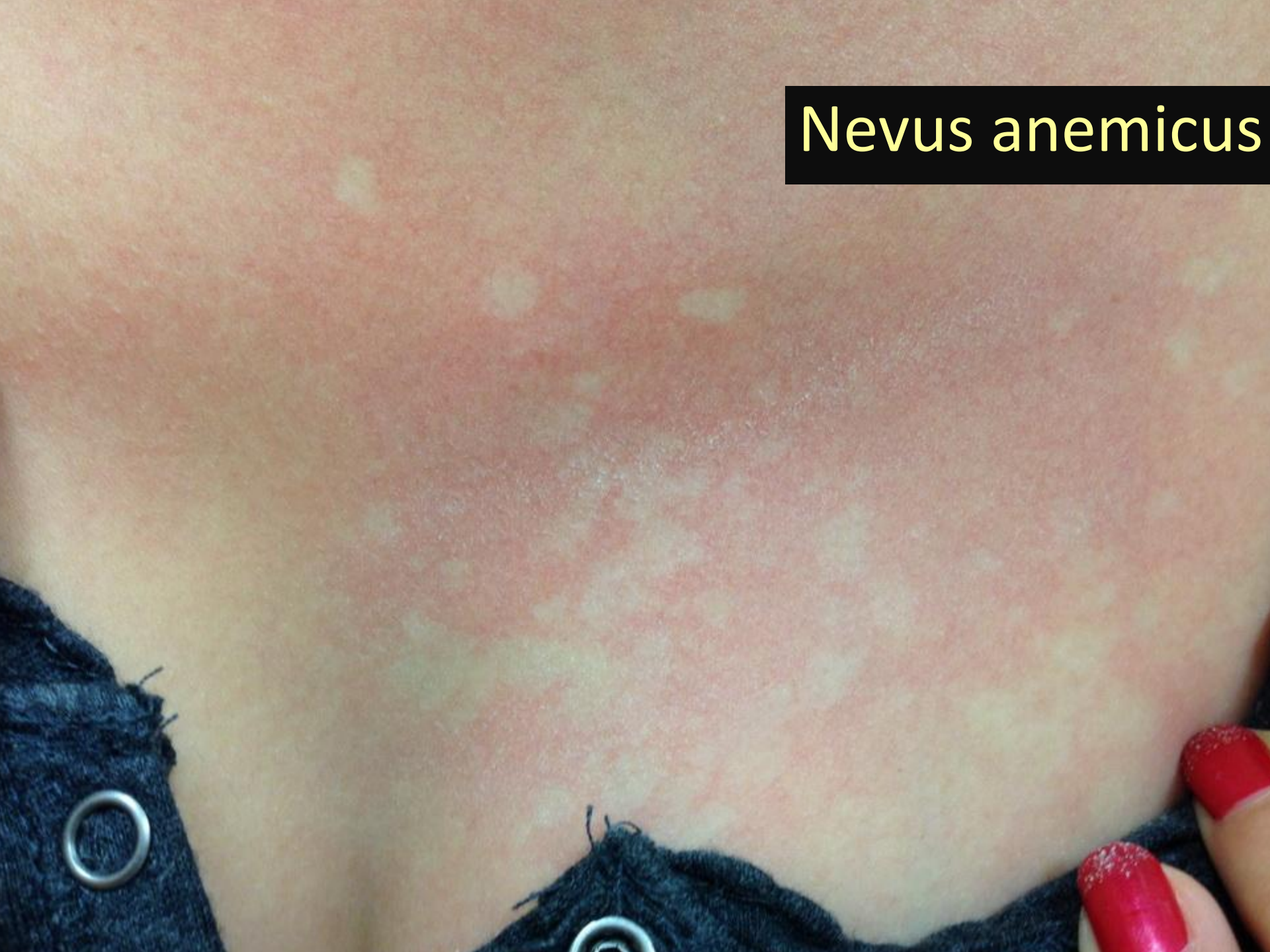

Lichen sclerosus

Idiopathic guttate hypomelanosis



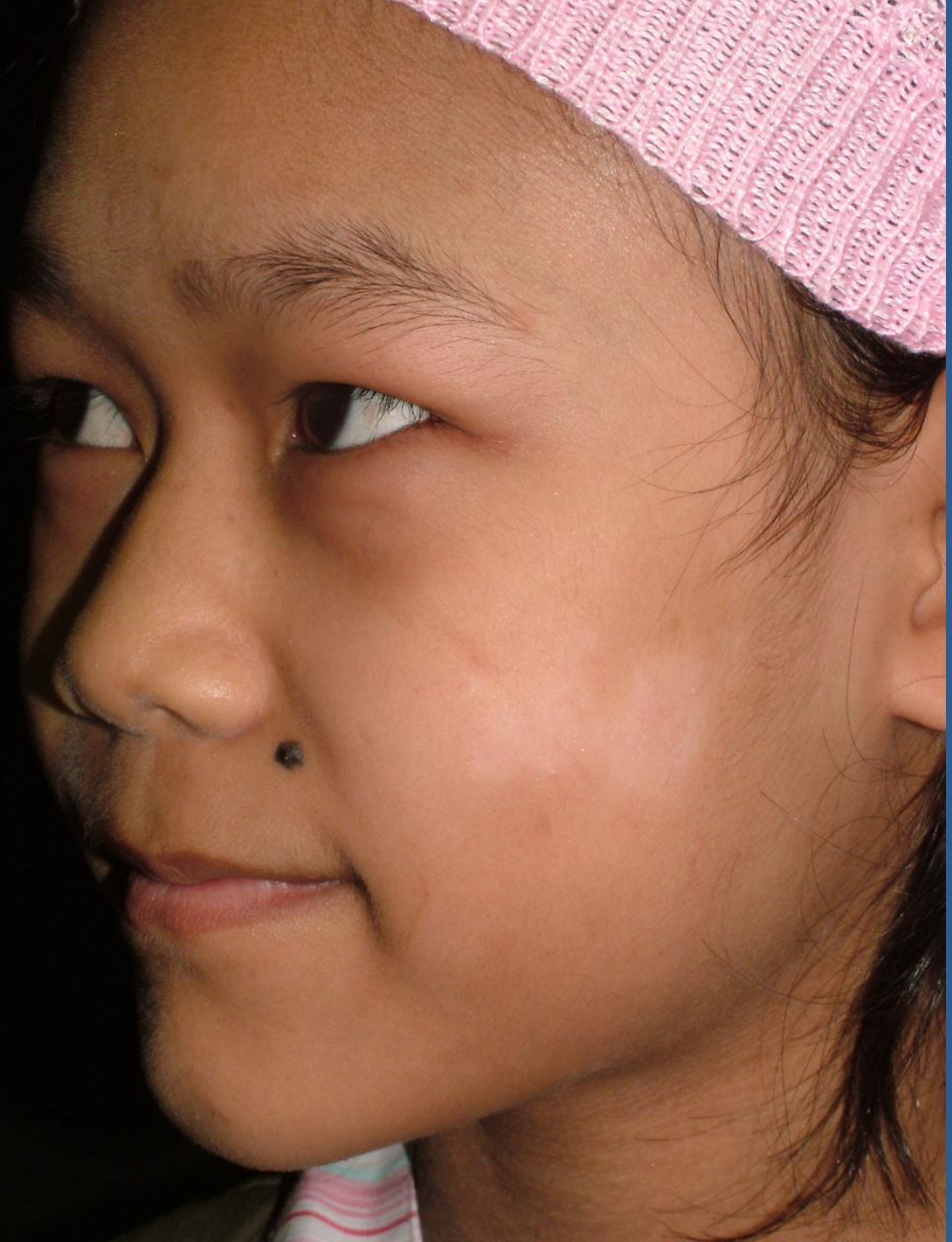



Halo nevus


Nevus anemicus

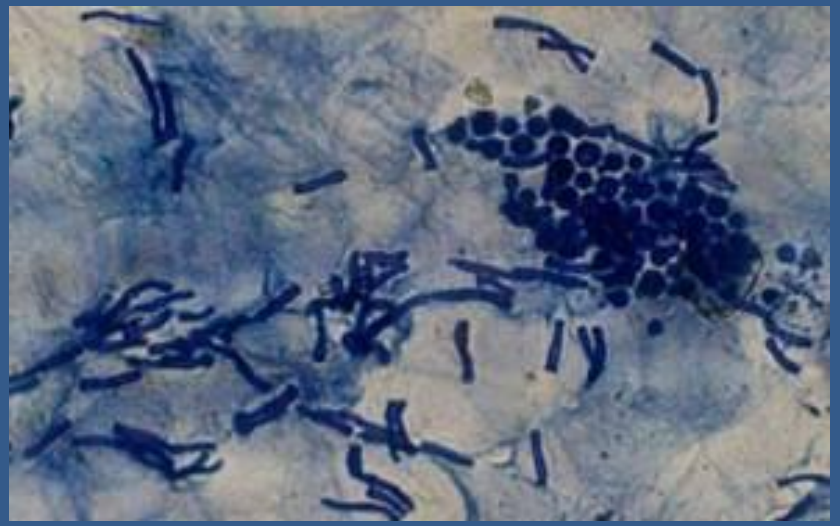
Differential diagnosis

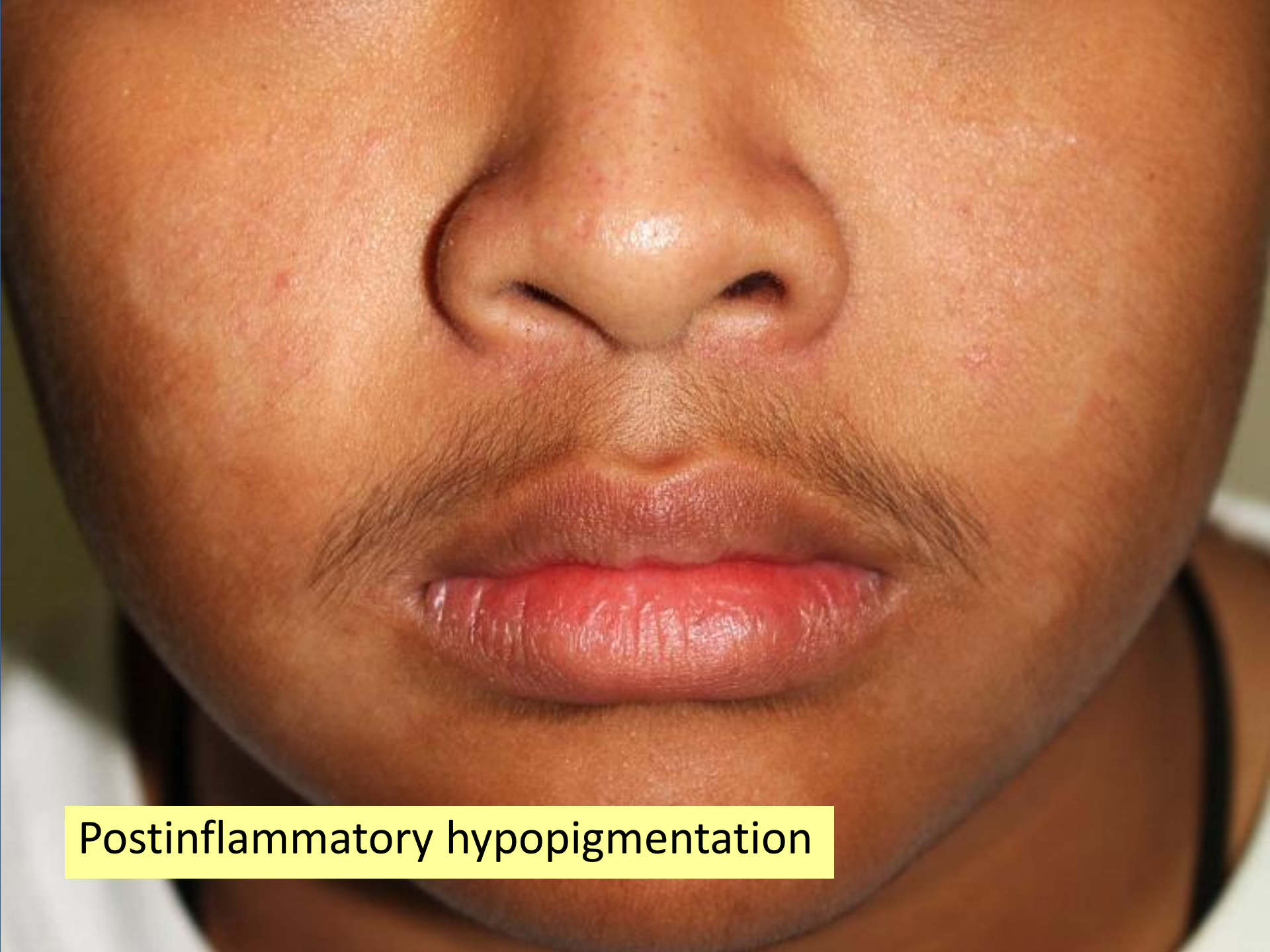
- Hypopigmented lesion
  - -pityriasis alba
  - -pityriasis versicolor
  - -postinflammatory hypopigmentation
  - -hypopigmented mycosis fungoides
  - -progressive macular hypomelanosis
  - -tuberculoid leprosy
  - -Ash-leaf hypomelanotic macule (tuberous sclerosis)



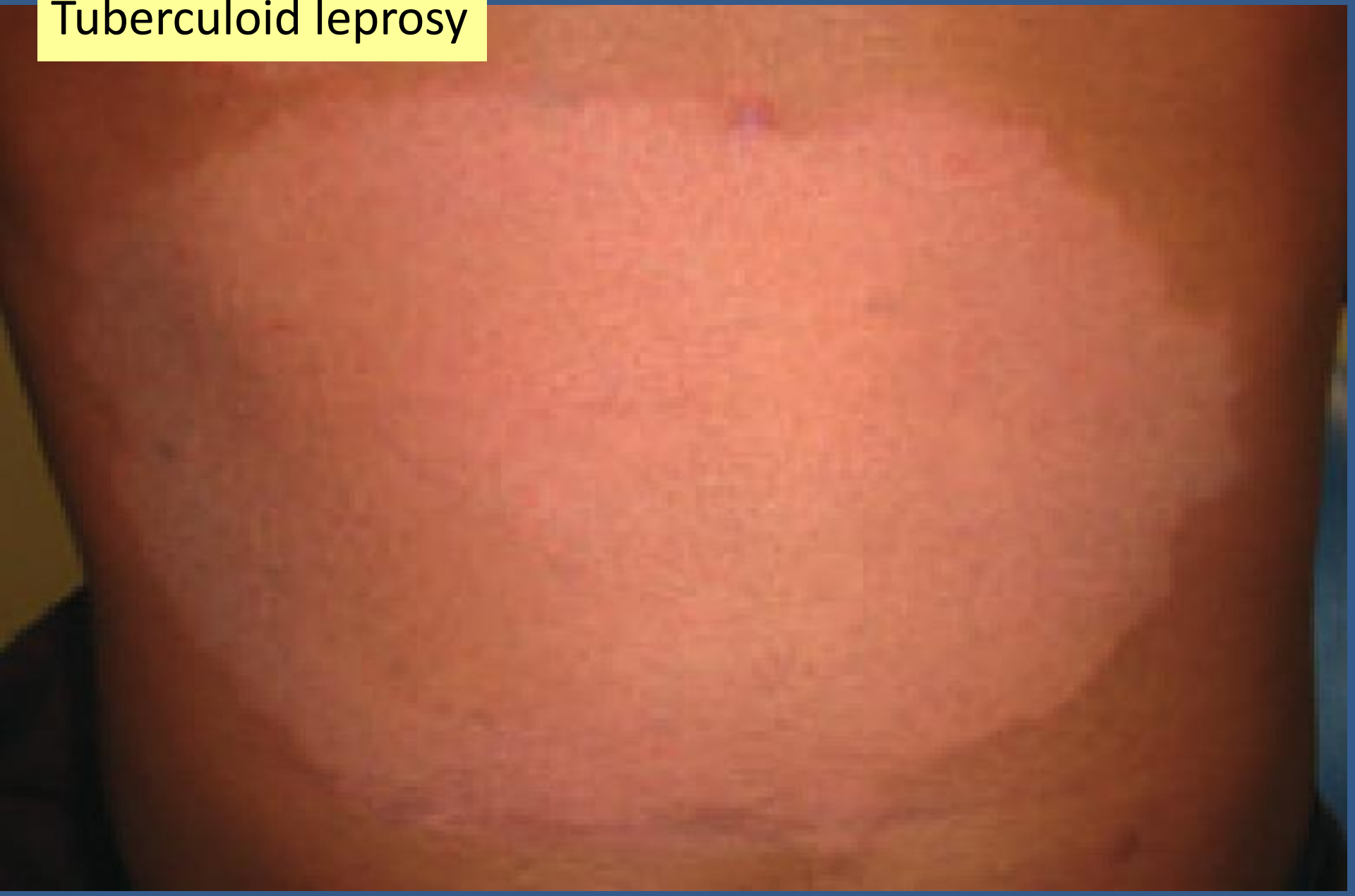
Pityriasis alba

Pityriasis versicolor

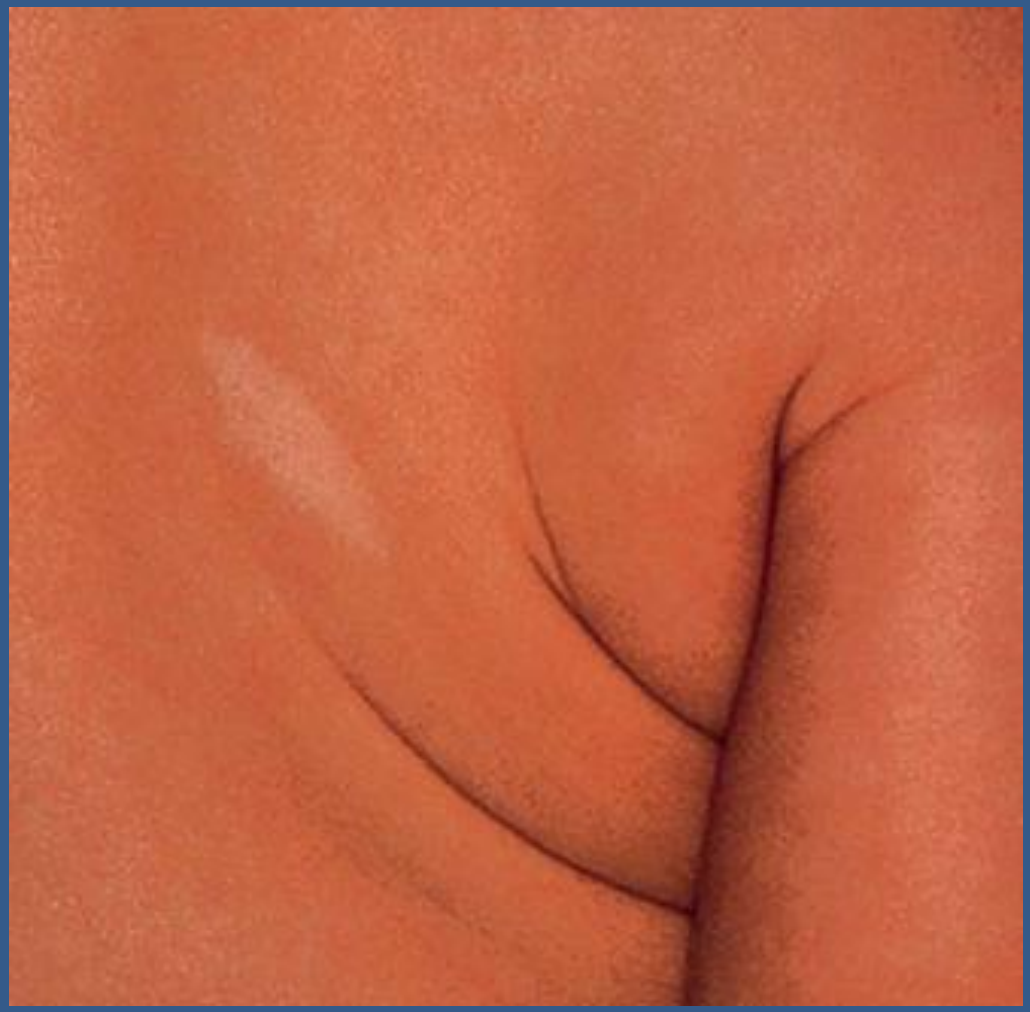



Postinflammatory hypopigmentation

Tuberculoid leprosy



Ash leaf macule



Management

Topical corticosteroids (TCS)

- Up to 75% repigmentation on face and neck, in dark skin, and recent lesions

Adverse effects of topical steroids

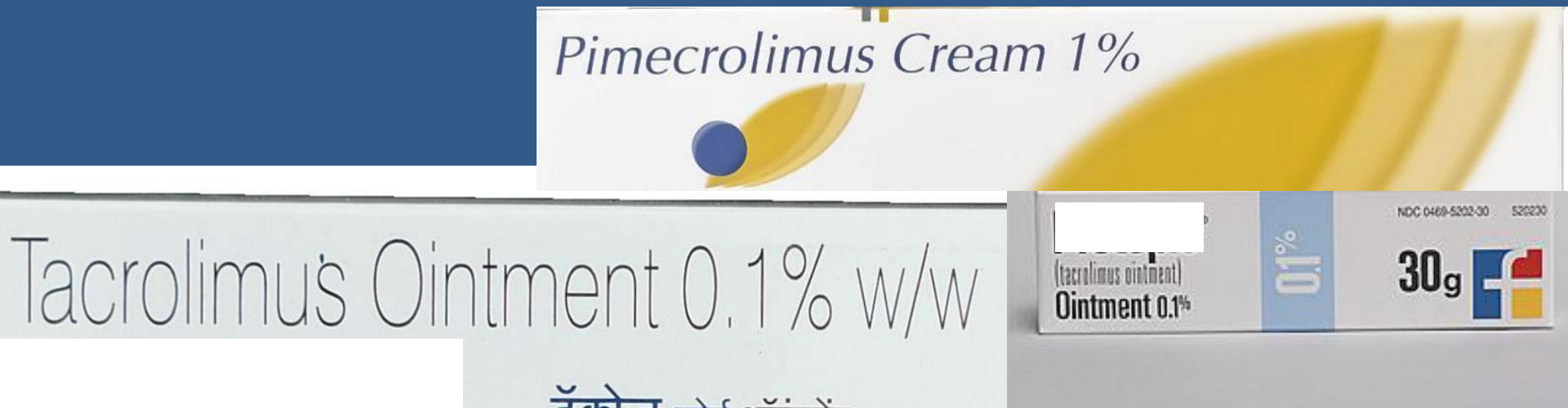
- Atrophy
- Telangiectasia
- Purpura, easy bruising
- Striae
- Acne
- Hypertrichosis
- Glaucoma
- Cataract
- Etc.

TCS: recommendations

- Application of potent TCS is advised to limited, extra-facial lesion for
  - -3 months (everyday) or
  - -6 months (15 days/month)
- Large area of skin, thin skin, children: momethasone furoate is preferred

Topical immunomodulators (TIM)

- Tacrolimus, pimecrolimus
- Alternative to TCS for lesions on thin skin
- Results similar to TCS with fewer side effects
- Occlusion enhance the effect
- TIM enhance the efficacy of phototherapy



TIM: recommendations

- TIM should be restricted to face and neck region
- Twice daily applications are recommended
- The treatment should be prescribed initially for 6 months. If effective, treatment longer than 12 months may be proposed
- During the period of treatment, moderate but daily sun exposure is recommended

Narrowband UVB and targeted phototherapy

- NUVB
  - -mean repigmentation is 41-68% from 3-6 mths
  - -a gold standard for the treatment of vitiligo
- Targeted phototherapy
  - -for small/ localized lesion
  - -2-3 times/week

NUVB and targeted phototherapy: recommendations

- Total NUVB is indicated for generalized NSV (>15-20% BSA involvement)

- Targeted phototherapy is indicated for
  - -small lesion
  - -all cases where C/I exist for total NUVB

NUVB and skin cancer

- NUVB does **NOT** significantly increase risk of NMSC compared with the general population

J Am Acad Dermatol 2012; 66: 326-7.

Other systemic treatments

- Current data do not provide enough evidence to recommend systemic corticosteroids, immunosuppressants or biologics in vitiligo

Br J Dermatol 2013; 168: 5-19.

Surgery: recommendations

- Surgery should be preserved for SV, localized vitiligo, after failure of other treatments
- For NSV, stable disease and KP negative are eligible

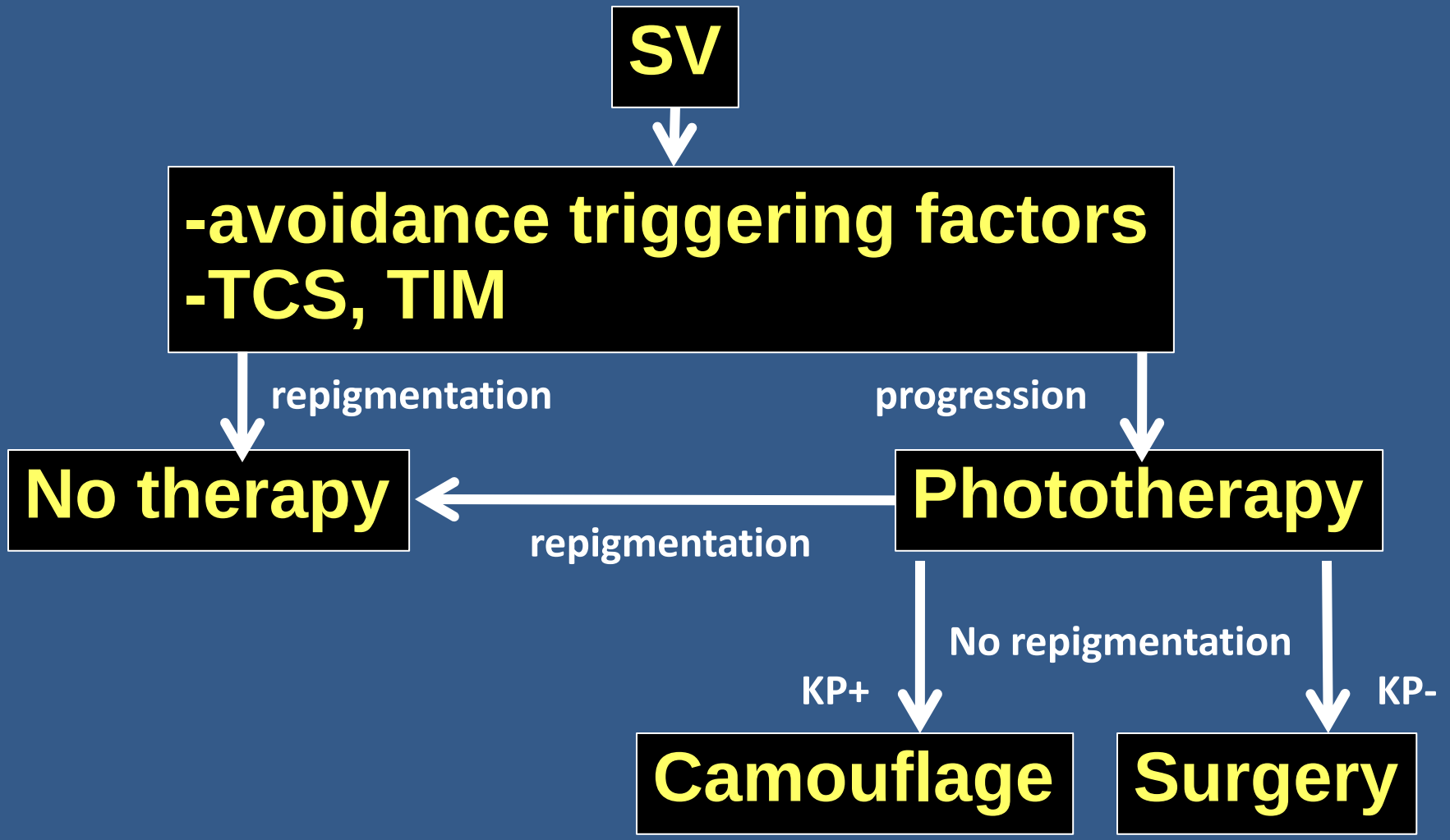
Br J Dermatol 2013; 168: 5-19.

Vitiligo surgery

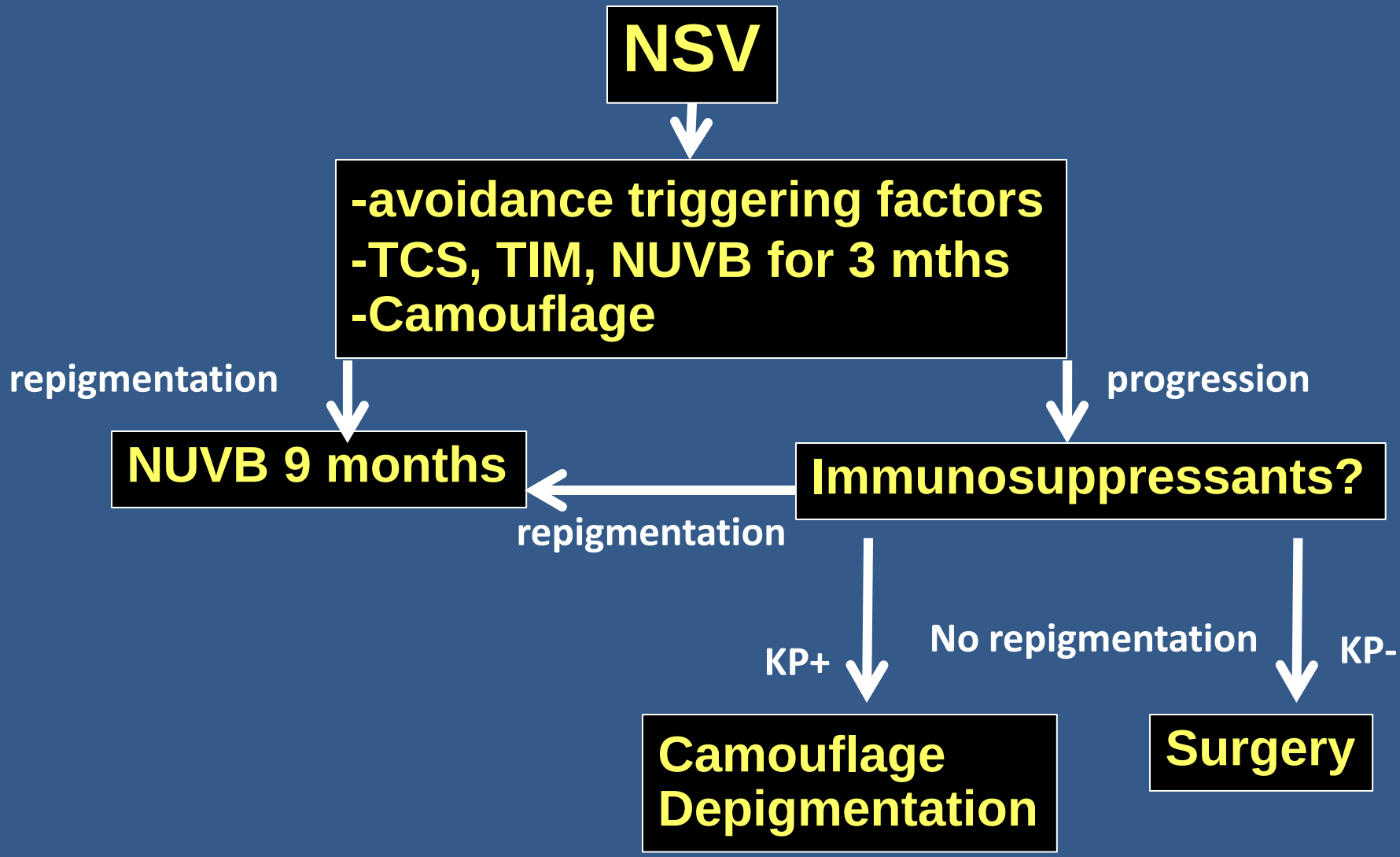
- Tissue graft
  - -punch graft
  - -suction blister graft

- Cellular graft
  - -non-cultured epidermal cell suspension
  - -melanocyte culture

Treatment algorithm



Br J Dermatol 2013; 168: 5-19.



Br J Dermatol 2013; 168: 5-19.

Q & A

Melasma

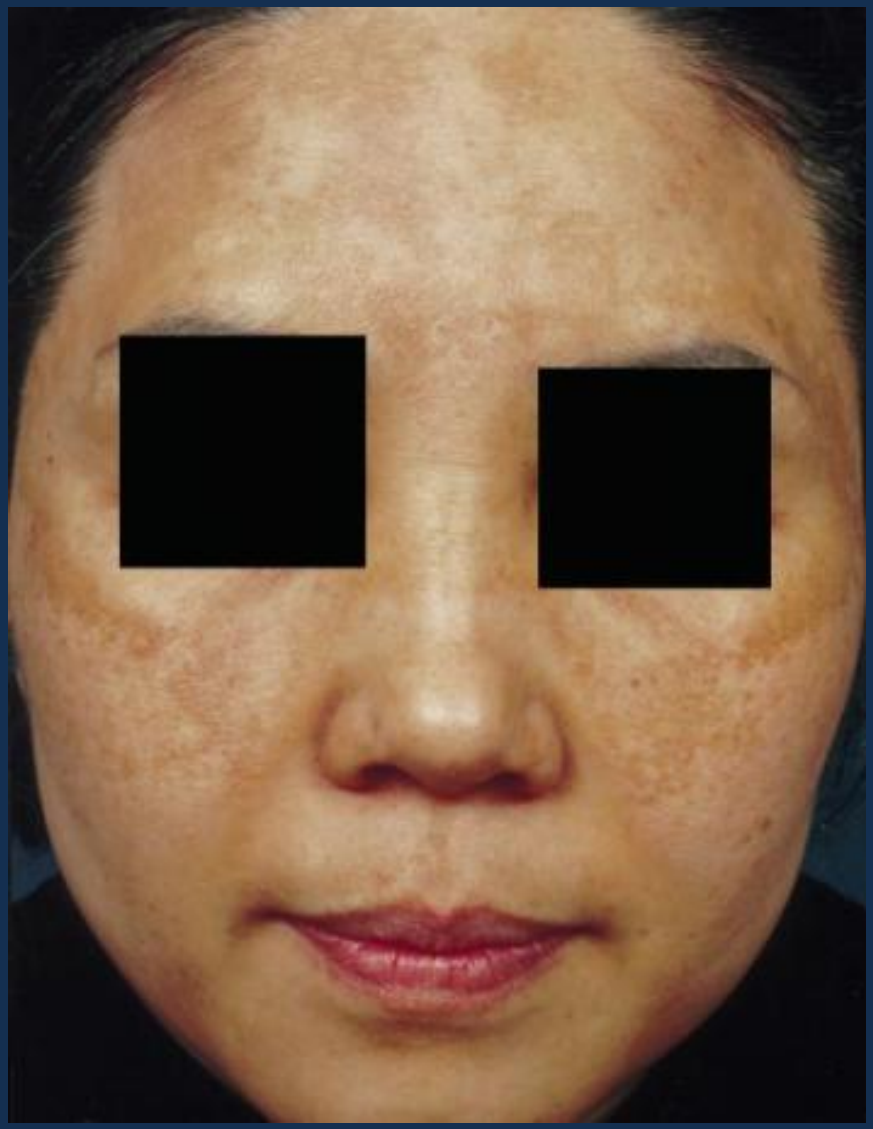
ผศ.นพ.วาสนภ วชิรมน

หน่วยโรคผิวหนัง ภาควิชาอายุรศาสตร์

คณะแพทยศาสตร์โรงพยาบาลรามาธิบดี

Melasma

- Acquired pigmentary disorder
- Symmetrical hyperpigmented patches and macules, especially the forehead, malar area, and chin



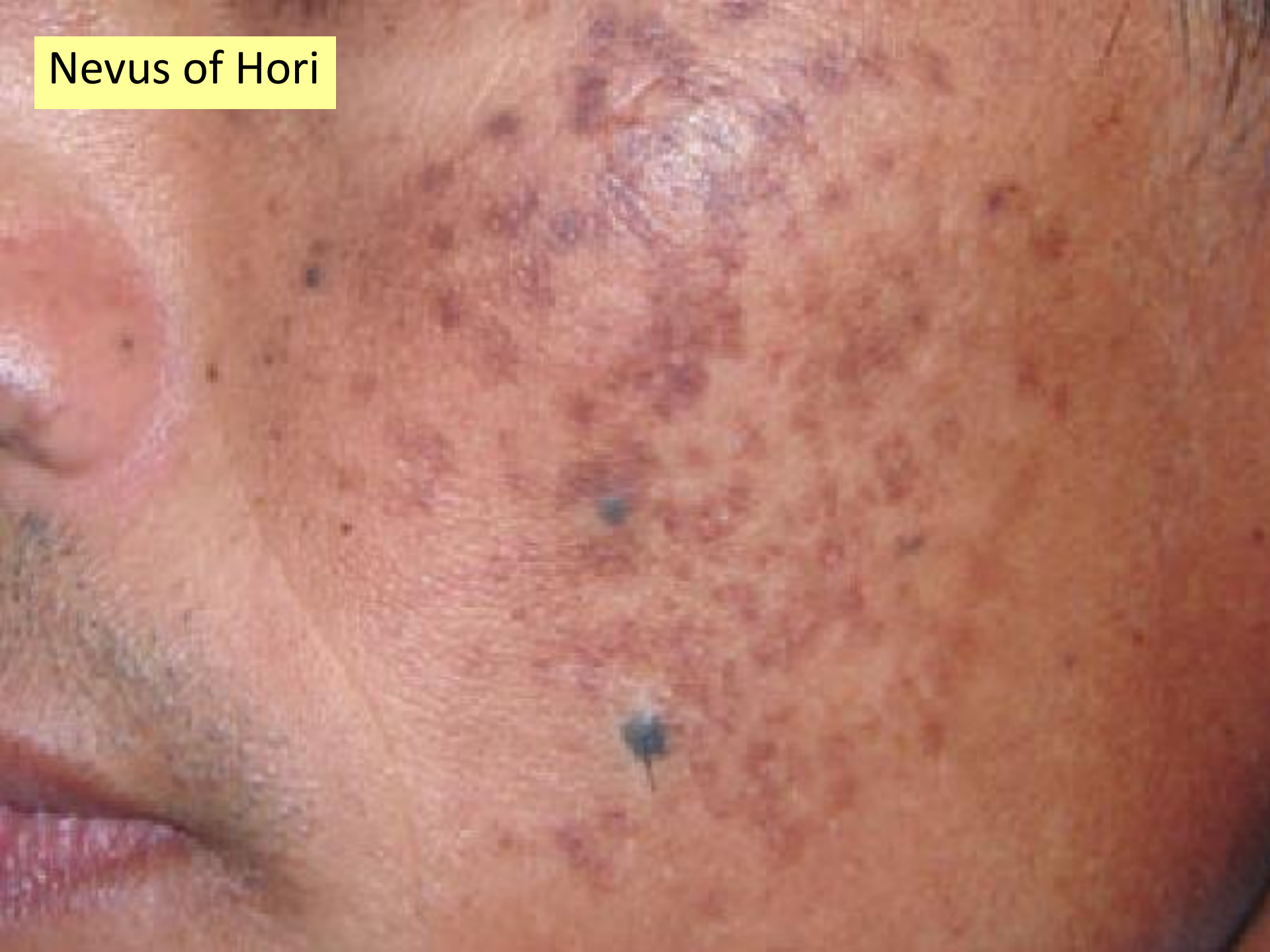
Epidemiology

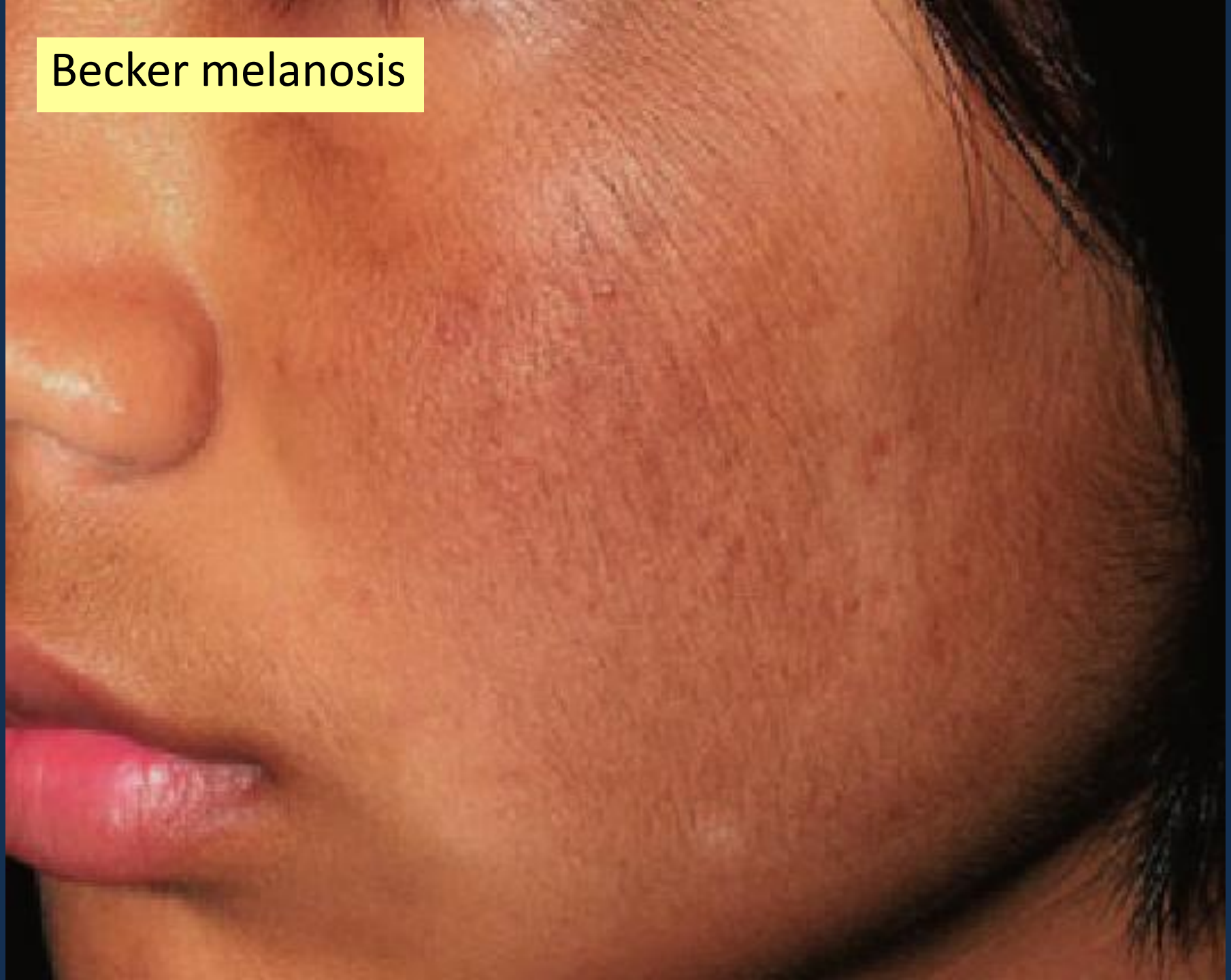
- The reported prevalence of melasma ranges from 8.8% among latino females to 40% in SE populations
- Onset: 20^{+}-40^{+} YO

Differential diagnosis

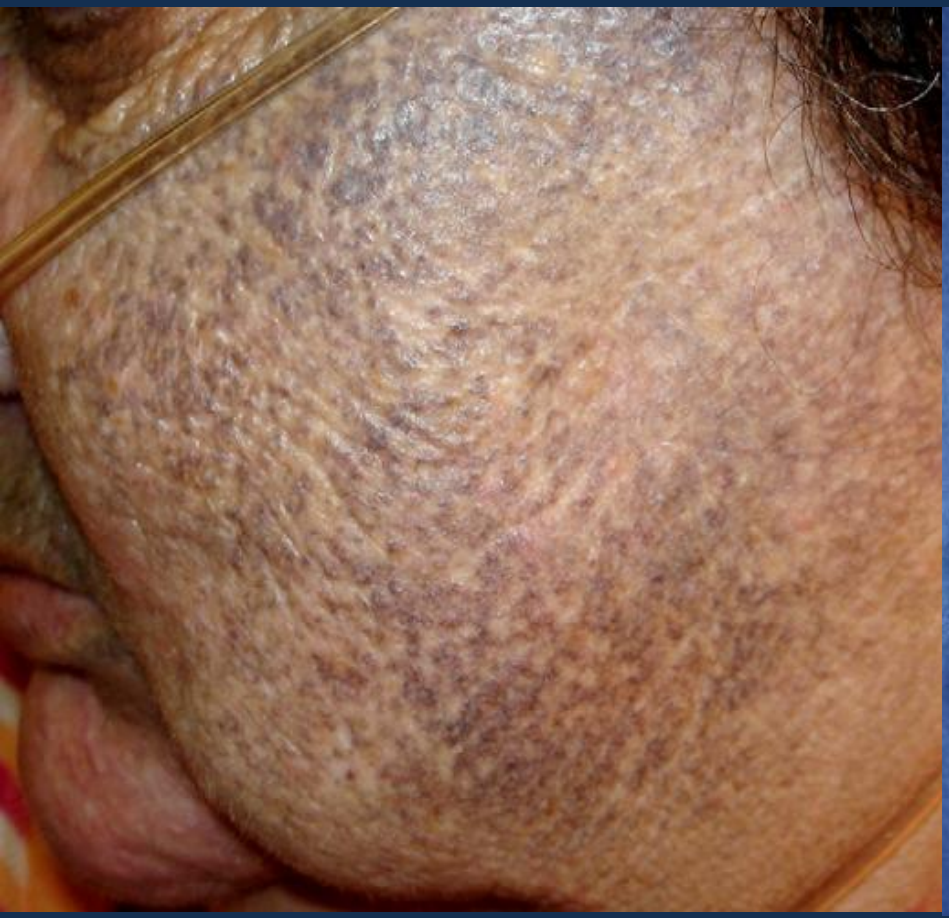
- Postinflammatory hyperpigmentation
- Nevus of Hori
- Becker melanosis
- Drug induced hyperpigmentation: minocycline, phenytoin, clofazimine
- Solar lentigo
- Acanthosis nigricans
- Lichen planus actinicus

Postinflammatory hyperpigmentation 2° to acute cutaneous LE

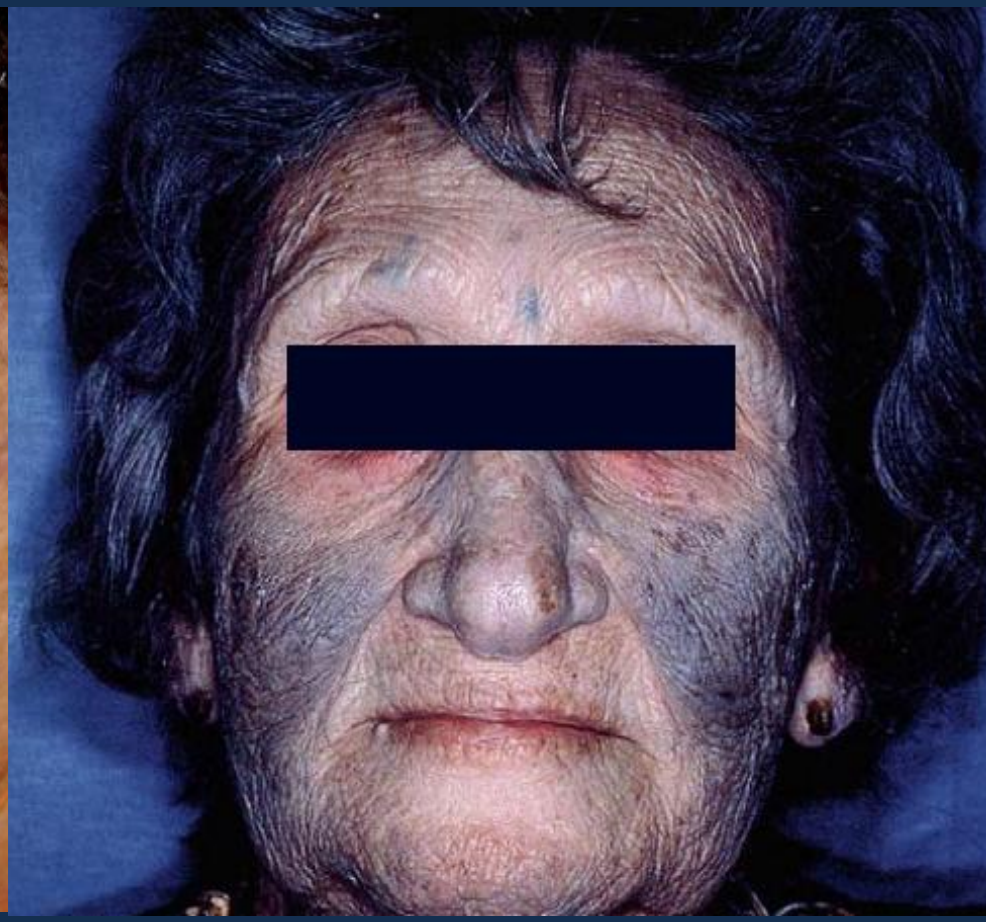

Nevus of Hori


Becker melanosis

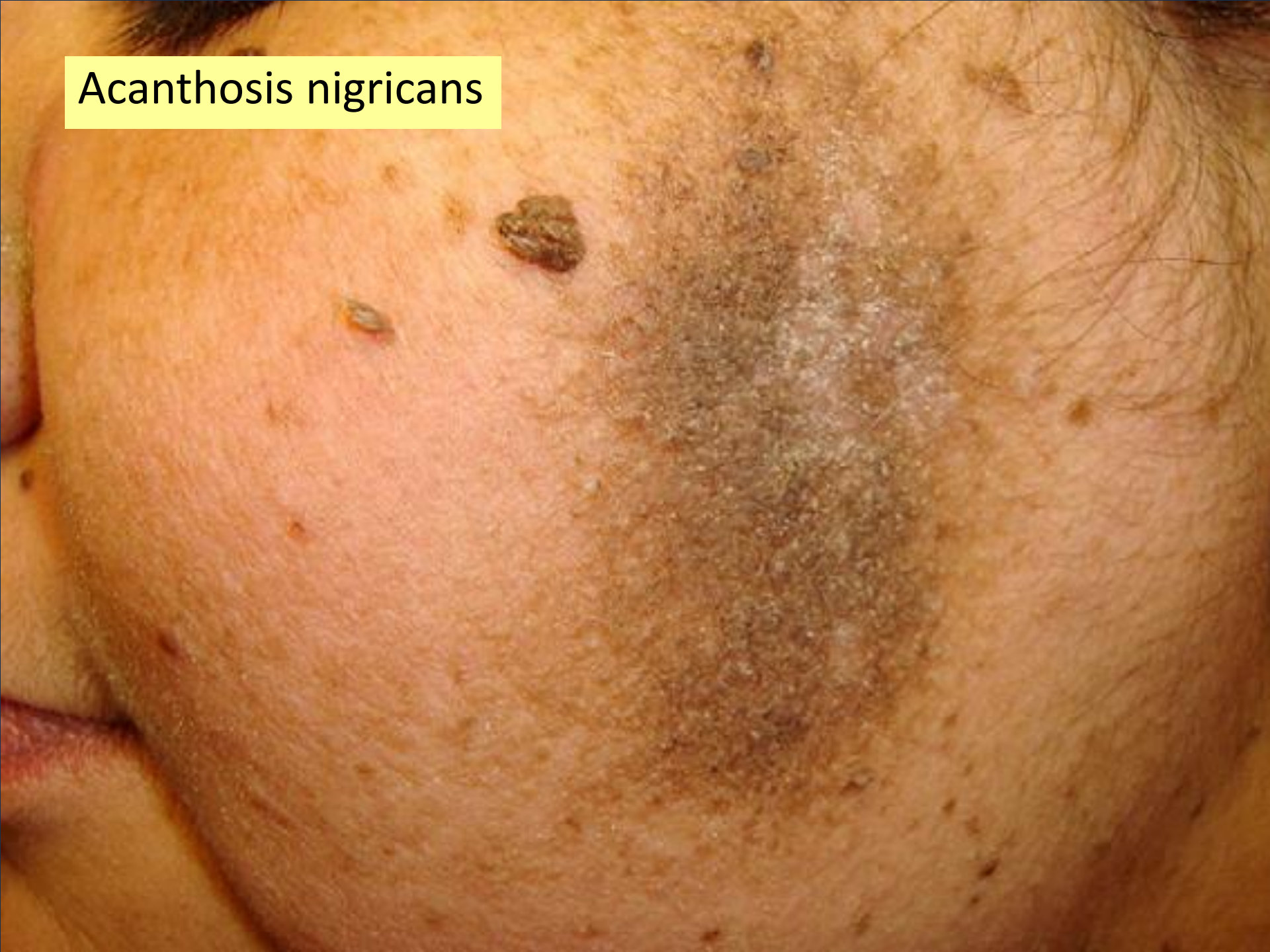
Drug-induced hyperpigmentation



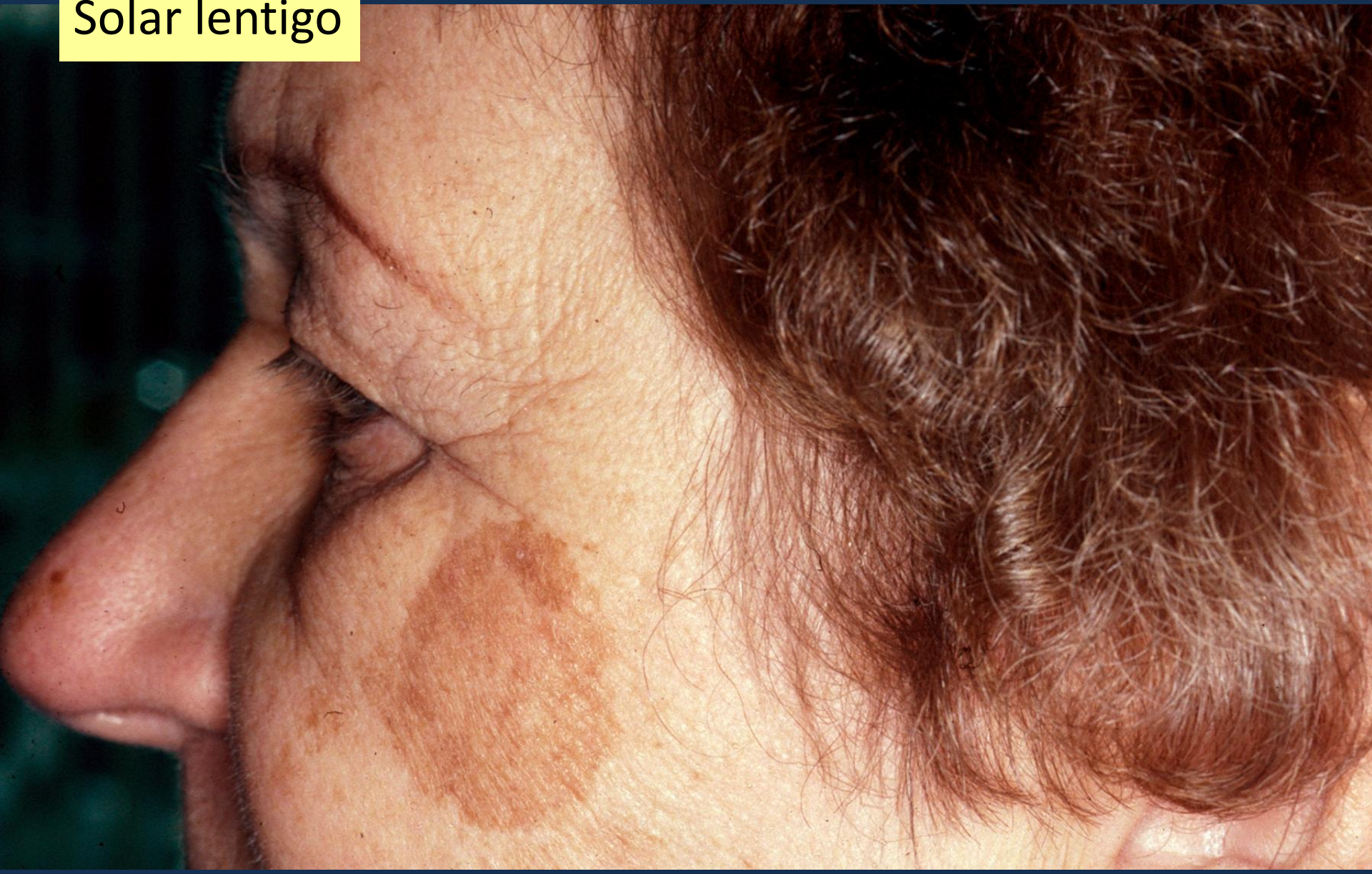
Minocycline



Clofazimine


Acanthosis nigricans

Solar lentigo



Pathogenesis

Genetic predisposition

- A positive family history of melasma were found in 10% -70% of study subjects

Hormone

- Many patients note the onset or worsening of disease with pregnancy or OCP use: estrogen, progesterone
- Thyroid hormone??
- LH??
- ACTH, MSH??

UV light

- UV radiation stimulate the production of multiple cytokines (e.g., IL-1, ET-1, α-MSH, ACTH, SCF, GRO-α, GM-CSF, PGE_2) from keratinocytes which upregulate melanocyte proliferation and melanogenesis

Treatment

- Before melanin synthesis e.g., UV block, cytokine inhibitors, receptor blocking agents, tyrosinase transcription
- During melanin synthesis e.g., enzyme inhibition (e.g., tyrosinase)
- After melanin synthesis e.g., inhibition of melanosome transfer, increase skin turnover

Patient education

- Sun avoidance
- Patients who develop melasma while using hormonal contraception *should stop* the medication

Sunscreen

- A regular use of broad spectrum sunscreen is effective both in preventing melasma and in enhancing the efficacy of topical therapies for melasma
- A broad spectrum UVA- and UVB-protective sunscreen with an SPF of at least 30 along with a physical block (e.g., titanium dioxide or zinc oxide) should be used and reapplied frequently

Topical treatment: first line

- Hydroquinone: tyrosinase inhibition
- Retinoids: inhibit tyrosinase transcription, ↑cell turnover, ↓melanosome transfer
- Triple combination: hydroquinone, retinoids, steroids

Topical treatment: adjunctive

- Azelaic acid
- Kojic acid
- Arbutin
- Ascorbic acid
- Licorice extract
- Soy

Chemical peels

- Glycolic acid may be the most efficacious peeling agent for melasma, but it should be used cautiously
- Glycolic acid peels should be used in conjunction with a depigmenting agent for maximal benefit and to minimize the risk of postinflammatory hyperpigmentation

Laser and light

- Laser and light therapy (e.g., fractional laser, IPL) may also provide modest benefit as an adjunctive treatment in a select population of patients, but larger studies are needed before this therapy can be widely recommended

Q & A