Disorders of pigmentation

Key terms

Hypopigmentation: partial loss of the skin pigment.

Depigmentation: complete loss of skin pigment.

vitiligo

Vitiligo is an acquired disorder characterized by circumscribed depigmented macules and patches that result from a progressive loss of functional melanocytes.

The incidence of vitiligo about 1% of the world's population. It can affects all races. Vitiligo can begin at any age but the majority of cases are between the age of 20 and 30 years. The prevalence is most probably the same in both sexes.

PATHOGENIC HYPOTHESES FOR VITILIGO;

1) Autoimmune destruction of melanocytes.
2) An intrinsic defect in melanocytes.
3) Defective defense against oxidative stress leading to destruction of melanocyte.
4) Genetic susceptibility.

It is un autoimmune disease in a genetically predisposed individual

Clinical feature;

The most common presentation of vitiligo is totally amelanotic (i.e. milk- or chalk-white) macules or patches surrounded by normal skin. The lesions enlarge centrifugally over time at an unpredictable rate

The most common sites of vitiligo involvement are the face, neck, forearms, feet, dorsal hand, fingers, and scalp. When found on the face, lesions may favor a periocular or perioral distribution.

Additionally, lesions may occur in regions frequently subjected to trauma, such as bony prominences, elbows, and knees. Koebner phenomenon is defined as the development of vitiligo in sites of trauma, such as a cut, burn, or abrasion. Koebnerization may occur in as many as 20-60% of vitiligo patients. Spontaneous repigmentation can sometimes be noted in sun-exposed areas, and can have a typical perifollicular appearance.

Body hair in vitiliginous macules may be depigmented, this is known as leukotrichia, and it may indicate a poor prognosis with regard to repigmentation therapy.

Vitiligo of the scalp most often presents as one or several localized patches of white or gray hair (poliosis).

Distribution of amelanotic skin lesion in vitiligo;

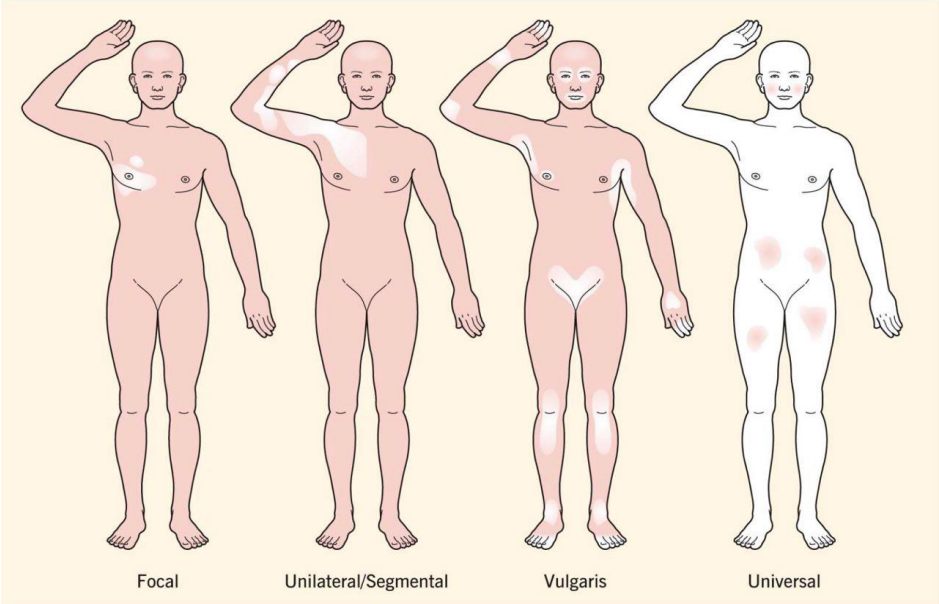
- **Localized**

Focal, segmental or mucosal

- **Generalized**

vulgaris, acrofacial or mixed

- **Universal**



Diagnosis;

WOOD'S LIGHT EXAMINATION

Examination with Wood's light in a dark room accentuates the hypopigmented areas and is useful for examining patients with light complexions.

Differential diagnosis;

1) Post inflammatory hypopigmentation
2) Pityriasis versicolor
3) Pityriasis alba
4) A nevus depigmentosus and a nevus anemicus

Treatment;

The aims of vitiligo treatment are stabilization of the depigmentation process and repigmentation.

Repigmentation usually appears in a perifollicular pattern from melanocyte in hair unit and/or from the periphery of the lesions.

And/or diffuse pattern from unaffected melanocyte within the area of depigmented epidermis

First line:

There are many topical and some oral agents that are inexpensive, easy to administer, and effective at halting disease progression and inducing repigmentation

- Psychotherapy
- Topical corticosteroid , potent topical corticosteroid preparations (e.g. 0.1% betamethasone valerate or 0.05% clobetasol propionate).

- Topical calcinurin inhibitors, (pimecrolimus cream, tacrolimus ointment)
- PUVASOL (psoralens + natural sunlight)
- Short course of systemic steroid if unstable disease.
- Antioxidants.

Second line:

- Systemic psoralen photochemotherapy PUVA
- NBUVB phototherapy with or without topical therapy like calcineurin inhibitor and potent steroid
- localized targeted phototherapy devices (excimer lamp or lasers with a peak at 308 nm)

Third line

- Surgical methods (Grafting techniques)
- Depigmenting treatment: In those patients with extensive vitiligo and only a few residual areas of pigmentation. , skin bleaching with 20% monobenzylether of hydroquinone or laser therapy.

Pityriasis alba

Pityriasis alba is a common finding (5% of children) that is probably more usual in patients with the atopic diathesis The condition appears in most instances before puberty.

The face, neck, and arms are the most common sites. The lesions begin as a nonspecific erythema and gradually become scaly and hypopigmented. The condition gradually improves after puberty.

Treatment

consists of lubrication. topical steroids, Pimecrolimus cream 1% or tacrolimus ointment 0.03%

The condition is often confused with vitiligo and tinea versicolor. Vitiligo does not scale. The potassium hydroxide preparation is positive in tinea versicolor.

Nevus anemicus

Nevus anemicus is a congenital localized pharmacologic cutaneous anomaly most often seen on the trunk.

The lesion usually consists of well defined hypopigmented macule with an irregular border, often surrounded by smaller hypopigmented macules beyond the border of the major lesion. Histologically the skin appears normal;

the pale color has been attributed to decrease blood flow through the capillaries in the dermal papillae due to local blood vessel hypersensitivity to catecholamines. Hence, it has been called a pharmacologic nevus.

. Friction, cold or heat application fails to induce erythema in the involved areas.

Treatment is not required. Camouflage make-up hides the lesion.

Nevus depigmentosus

It is a misnomer, it is hypopigmented rather than depigmented .

It is congenital defect result from decrease production of melanin with in this site.

It is appear during infancy – childhood as a well defined hypopigmented macule or patch that enlarge propotionary with the body and it does not require treatment.

Disorders of hyperpigmentatiom

▶ Melasma

Melasma is a common acquired symmetric brown hyperpigmentation involving the face and neck in genetically predisposed women and men.

The pigmentation develops slowly without signs of inflammation and may be faint or dark. Blacks, Asians, and Hispanics are the most susceptible populations.

causes include pregnancy, Genetic factors and UV radiation are the most important, oral contraceptives, estrogen-progesterone therapies, thyroid dysfunction, cosmetics, phototoxic and anti seizure drugs.

The forehead, malar eminences, upper lip, and chin are most frequently affected

Treatment

- ▶ Avoid sun exposure and daily use of broad spectrum sunscreen (SPF ≥ 30%).
- ▶ Discontinue oral contraceptive, if possible
- ▶ Triple combination of topical hydroquinone + retinoid + corticosteroid at bedtime.
- ▶ Azelaic acid 15-20%.
- ▶ L-ascorbic acid
- ▶ Glycolic acid peel
- ▶ Laser therapy

CAFÉ-AU-LAIT MACULES

Well-circumscribed, uniformly light to dark brown macules or patches that often range from 2 to 5 cm (in adults)

Usually noted during infancy or early childhood

Found in 10–20% of the normal population but can serve as a marker of an underlying genodermatosis, especially when it is multiple like in neurofibromatosis type1.

It may be located anywhere on the body, with the exception of the mucous membrane.

Post inflammatory hyperpigmentation

May appear after any inflammatory conditions affecting the skin especially skin of color

Like lichen planus, insect bite, eczema, drug reaction and others.

Freckles (Ephilides)

Appear during early childhood in patient with fair skin.

- Usually after intensive sun exposure as a small light brown macules.

It is become darker in summer and fade during winter or over time with age.
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Solar lentigines

Appear during adulthood and affect both dark and light skin.

It is larger and darker than freckles.

Appear after repeated sun exposure.

The color become darker in summer but do not fade during winter.