Dermatosis due to physical factors

Dermatosis due to physical factors:

- Cold injuries.
- Actinic injuries.
- Miliaria
- Erythema ab igne

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Cold injuries: includes

1- Perniosis (chilblains).

2- Frostbite.

3- Acrocyanosis

Perniosis (chilblains)

Local inflammatory reaction induced by abnormal response to cold above the freezing point. Thus, it is seen during the cold months of winter.

There are vascular instability, with prolong vasoconstriction(of the small arteriol & venule) when expose to cold. Associated with reactive hyperemia (larger vessels dilation).

The spontaneous remission is common when spring arrives, and relapse is frequent during the following winters.

Chilblain lupus is a distinct disease and is similar to discoid lupus erythematosus.

Clinical features

-Female > male, age at 20 years

-Single or multiple, erythematous or purplish swellings.

-In severe cases, blisters, pustules, and ulceration may occur.

-Patients may complain of itching &/or burning pain.

-Sites: include the fingers, toes, dorsal and plantar surfaces of the toes, heels also the nose and ears.

Treatment

1- protection of the affected parts against further exposure to cold.

2- antipruritus (antihistamine, topical calamina).

3- Peripheral vasodilators (nifedipine, nicotinamid, pentoxyphylline and topical minoxidil

4- Systmic steroid, if perniosis associated with SLE.

Frostbite

Occurs when tissue freezes after exposure to extremely cold air, liquids, or metals.

The clinical effects of accidental injury that leads to the death of tissues are similar to those caused by burn.

Clinical features:

Usually affect the cheeks, ears, nose, fingers, and toes.

Painless, pale- erythema, waxy, edema, vesicles& bullae.

Destruction may reach to muscles, tendons, bone and nerves.

Sometime superficial and deep gangrene.

Treatment:

1- Rapid rewarming
2- Analgesia
3- Bed rest
4- High colorie & protein diet.
5- Wound care and antibiotics.

Acrocyanosis

Rare form of cold injuries with unknown cause.

Women > man

Clinical features:

Persistent blue discoloration of the entire hand or foot worsened by cold exposure.

Persistent coldness of fingers & toes with hyperhidrosis.

Acrocyanosis is distinguished from Raynaud syndrome by its persistent (rather than episodic) nature and lack of tissue damage (ulceration, distal fingertip resorption).

Treatment: difficult, and Patients should avoid cold exposure, smoking, coffee and tea.

Actinic injuries:

1- Suntan & sunburn

2- Photosensitivity

Suntan

It is a physiological process to protect the skin from sun damage after moderate sun exposure, has 2 stages:

1- Immediate tanning (pigment darkening)

Caused by UVA,

Hours after sun exposure,

Due to photochemical activation & changes of existing melanin.

2- Delayed tanning

Caused by UVB,
start 2-3 days after sun exposure,
there is synthesis of new melanin,
Last for about 30 days.

Sun burn(Solar erythema)

After hours of extensive exposure to UVB,

Consider as a first degree burn,

C/F: Erythema, pain & fever; in severe cases blisters, continue for 2-3 days.

Heal with desquamation & pigmentation.

Treatment of sunburn: symptomatic

1- Topical cooling agents (callamina)

2- NSAID for pain (ponstan, indocide)

3- In early sunburn: Topical and systemic steroid/ 1week.

Ultraviolet Sun light

UVA—320-400nm (long wave)

Penetrate glass

Responsible for immediate tanning of the skin

Little role in delayed tanning & skin aging

It is abundance in sunlight about 100 time than UVB

UVB—290-320nm (middle wave)

Absorbed by glass

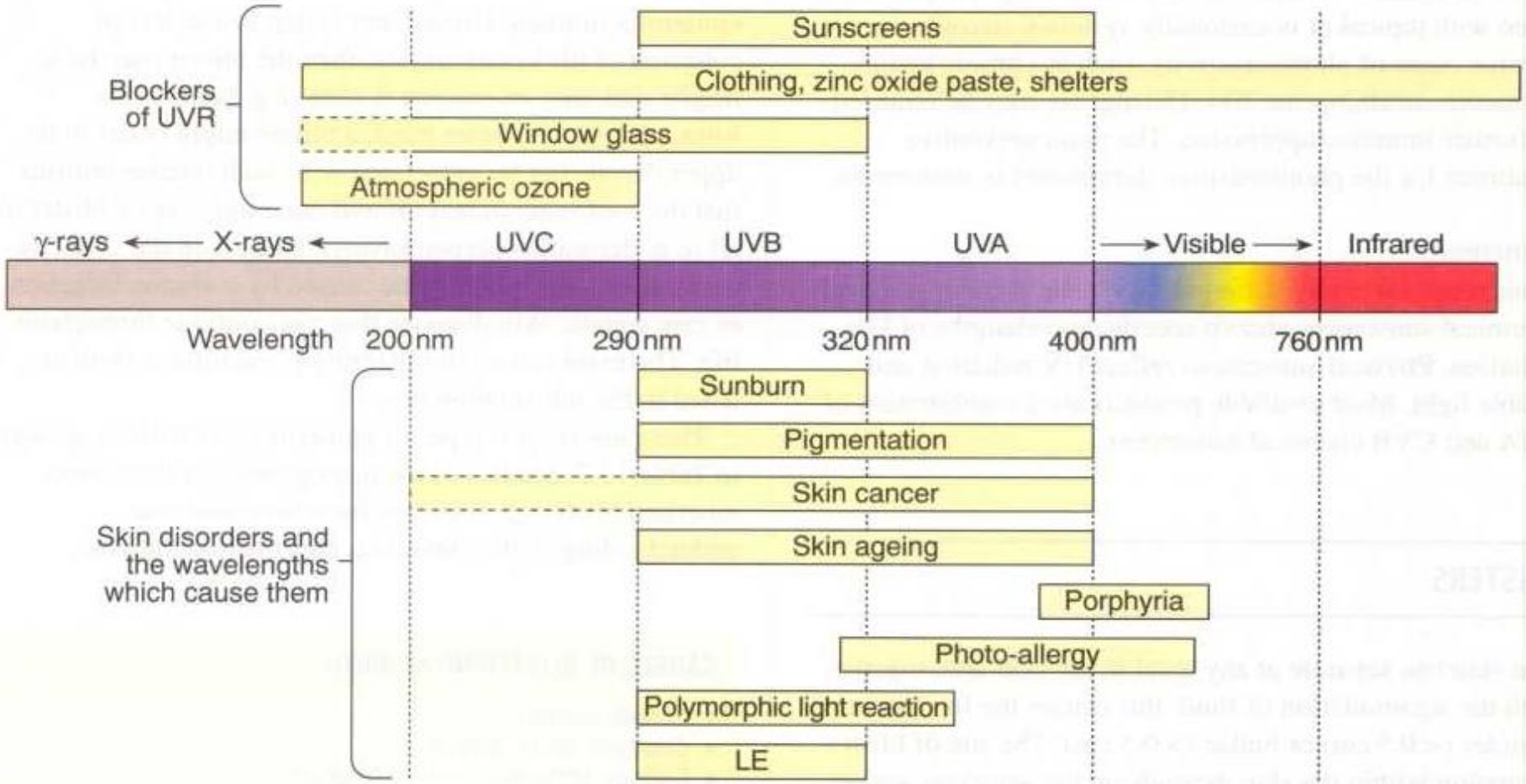
Responsible for delayed tanning, sunburn, aging,

It is important in vitamin D synthesis.

UVC----200-290nm(short wave)

Absorbed by ozone layer.

It is germicidal.


Blockers of UVR
Sunscreens
Clothing, zinc oxide paste, shelters
Window glass
Atmospheric ozone
γ-rays
X-rays
UVC
UVB
UVA
Visible
Infrared
Wavelength
200 nm
290 nm
320 nm
400 nm
760 nm
Skin disorders and the wavelengths which cause them
Sunburn
Pigmentation
Skin cancer
Skin ageing
Porphyria
Photo-allergy
Polymorphic light reaction
LE

Photosensitive dermatosis (photodermatosis) Classified to 4 major groups:

1- Metabolic(Endogenous) induced photosensitivity:

As porphia cutanea tarda, xeroderma pigmentosa, pellagra.

2- Chemically(Exogenous) induced photosensitivity:

Include phototoxic(sunburn) & photoallergic(eczema) reaction.

Causes are (topical, systemic drugs or plants)+ sun light.

3- Idiopathic diseases

Without cause, as in polymorphic light eruption, solar urticaria.

4- Diseases exacerbated by sun light:

As example in herpes simplex, systemic lupus erythematosus.

phototoxic

* Non immunological skin reaction.
* Occurs minutes to hours after exposure to agent (topical drug, systemic drugs(as in phenothiazin, tetracyclines & amiodaron
* or plants) and light.
* Appears as an exaggerated sunburn reaction (reddening and swelling)
* Vesicles, blisters and bullae may occur in severe reactions.
* May or may not be itchy.

phototoxic

- Less commonly, skin may change color, e.g. blue-green pigmentation is associated with amiodarone.
- Reaction is limited to sun-exposed skin.
- In dark-skinned individuals… pigmentation & Photo-onycholysis (separation of the distal nail plate from the nail bed) may arise with many oral photosensitizing medications and may be the only sign of phototoxicity.

Photopatch test

Photoallergic

- Immunological reactions
- Eczema either acute or chronic +itchy .
- Occurs 1-3 days after exposure to agent and light (drug+ sun).
- As in thiazin, antimalaria drugs, phenothiazine & enalpril
- May spread to areas that have not be sun-exposed.
- Treatment: Stop the drug and sun exposure, topical & systemic steroid.

polymorphic light eruption

-- Most common form of photosensitivity

-- After exclude all other causes of photosensitivity.

-- Age of onset before 30 years of life, female > male.

-- Onset at spring time of the year.

-- Different clinical presentation(papules most common).

-- Sites: face, V-shape of chest, neck & forearms.

-- Treatment: avoid sun, topical steroid, antihistamine, systemic steroid, PUVA, antimalarial drugs or azathioprin.

Sun protection method

1- Wear cloths against sun.

2- Avoid peak sunlight density between (10am-3pm).

3- Antioxidant agents(vitamines C, E, A…)

4- Topical sunscreens include:

- Chemical sunscreen: absorb UVA, UVB or both.
- Physical sunscreen: Zinc oxide, titanium dioxide

Miliaria

Cause, the retention of sweat as a result of occlusion of eccrine sweat ducts.

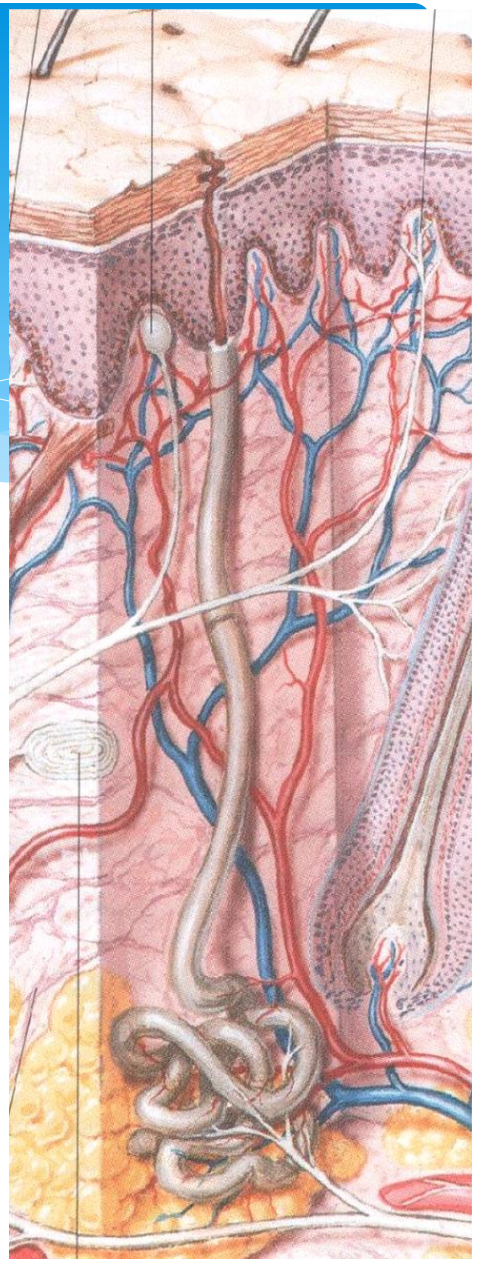
Common in hot, humid climates, such as in the tropics and during the hot summer months in temperate climates.

Staphylococcus epidermidis, anormal flora, which produces an extracellular polysaccharide substance, induces miliaria in an experimental setting. This polysaccharide substance may obstruct the delivery of sweat to the skin surface..

Miliaria

The occlusion prevents normal secretion from the sweat glands, and eventually pressure causes rupture of the sweat gland or duct at different levels.

The escape of sweat in to the adjacent tissue produces miliaria.



Depending on the level of the injury to the sweat gland or duct, several different forms are recognized

1- **Miliaria crystallina (sudamina)**

2- **Miliaria rubra (prickly heat)**

3- **Tropical anhidrotic asthenia**

4- **Miliaria pustulosa**

5- **Postmiliarial hypohidrosis**

6- **Miliaria profunda**

1- Miliaria crystallina (sudamina)

* Is characterized by small, clear, superficial vesicles with no inflammatory reaction.
* It appears in bedridden patients whose fever produces increased perspiration or when clothing prevents dissipation of heat and moisture, as in bundled children.
* The lesions are generally asymptomatic, and their duration is short-lived because they tend to rupture at the slightest touch.
* The lesions are self-limited; no treatment is required.

2- Miliaria rubra (prickly heat)

* The lesions of miliaria rubra appear as discrete, extremely pruritic, erythematous papulovesicles , accompanied by a sensation of prickling, burning, or tingling. They may become confluent on a bed of erythema.
* The sites: the antecubital and popliteal fossae, trunk, infra-mammary areas, abdomen (especially at the waistline), and inguinal regions.
* Exercise-induced itching or that of atopic dermatitis may also be caused by miliaria rubra.
* Sweat escape in the prickle cell layer.

Erythema ab igne

* Erythema ab igne, is a persistent erythema &/or the reticulated residual pigmentation resulting from it.
* Produced by long exposure to excessive heat without the production of a burn.
* Its mottling pigment caused by local hemostasis.

Sites of Erythema ab igne

* Erythema ab igne on the legs results from habitually warming them in front of open fireplaces, space heaters, or car heaters.
* On the lower back or at hands or forearms due to an electric heating pad application.
* On the upper thighs with laptop computers,
* On the posterior thighs from heated car seats.
* The condition occurs also in cooks and others who exposed over long periods to direct moderate heat.

Treatment of Erythema ab igne:

- Avoid the cause
- Emollients containing α-hydroxy acids
- Cream containing fluocinolone acetonide 0.01%,+ hydroquinone 4%,+ tretinoin 0.05%
- The Q-switched neodymium-doped yttrium-aluminum-garnet (Nd:YAG) laser.