RIEHL MELANOSIS TRIGGERED BY CLEANING AGENTS

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Summary

Hypermelanases are defined as hyperpigmentative skin disorders resulting from quantitative abnormalities (melanin excess) or qualitative melanin pigments. Hypermelanosis can be determined by genetic and acquired factors. Some melanosis occur by increasing melanogenesis under the influence of genetic factors. Other melanosis occur by increasing the number of melanocytes (Mongoloida patch, Ota nevita, Ito nevul). Other melanomas occur under the influence of hormonal factors, UV, chemical compounds and cosmetics. Riehl's melanosis (cosmetic pigmentary dermatitis or facial melanoma of women) is a skin disorder that is manifested by the appearance of a pigmentation in the cephalic extremity encountered especially in women and in the appearance of which cosmetics, detergents and sunlight appear to play a role important. We present the case of a 43-year-old patient presenting in our clinic for a dramatic facial melanoma with a major negative impact on the emotional state of the patient. Diagnosis based on clinical examination, history, challenge tests and histological examination is Riehl's melanosis.

***Keywords**: melanosis, Riehl, cosmetics, detergents, hyperpigmentation.*

Received: 11.09.2017 *Accepted: 3.11.2017*

Introduction

Melanosis is a common disorder in dermatological medical practice and in dermatocosmetics, with a major impact in the vast majority of cases, with a negative impact on social life, especially for women. We speak of hypermelaninases when melanocyte activity is increased and hypermelanocytosis when melanocytes are excessively elevated [1].

The etiology of melanosis is still quite unclear, and there are a number of factors that can trigger the condition. Melanosis can be related to the nature of the triggering process: genetic, metabolic, endocrine, deficiency, physical, toxic, parasitic, infectious, tumor, neurological, systemic, post-inflammatory, cosmetic or idiopathic [1].

It is well known in hyperpigmentation that the major trigger factor is exposure to ultraviolet. Ultraviolet have the ability to trigger and aggravate the evolution of any melanosis [2]. Ultraviolet stimulates the proliferation and migration of melanocytes but also melanogenesis. The influence of the sun is proven by the fact that the onset of the disease is usually summer and later it is always aggravated during the warm season. But in the case of Riehl's melanosis there are other factors that are more prominent in triggering hyperpigmentation, namely cosmetics and detergents [3].

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Initially, the pigmentation is punctual, but later it extends, including the whole of the cephalic extremity as well as the limbs and the chest.

Pigmentation has different shades, so it can be yellow-brown, dark-brown and even black, with the net limit of the banner. The skin is generally normal, rarely slightly atrophic, and occasionally there are lichenoid papules and rarely follicular hyperkeratosis.

Skin color can be constitutional (native, influenced by genetic factors), or induced (under the action of solar rays or other factors). Melanosis occurs more frequently in certain ethnic groups, such as hispanic, oriental or asian individuals [2,4].

Skin pigmentation is fairly common in both males and females, irrespective of race, and is characterized by brown or black, circumscribed lesions (in which case the skin between the healthy and diseased skin is clearly visible), diffuse or generalized.

Women are more affected, especially during the reproductive period, with a peak between 20-30 years [2,3,4]. It has been noticed that melanoses are much rare before puberty. Skin hyperpigmentation is common in both men and women, but women are those who, through the perceptions that define „beauty, perfect skin," are more emotionally affected, with a negative impact on the quality of life. No matter how small the affected or intense area is pigmentation, most affected people feel the disease intense and have a relatively high degree of frustration over this aesthetic problem. Melanoses can be limited, circumscribed, and become broad, diffuse in evolution.

There are varying degrees of pigmentation in various regions of the body but the most unpleasant aspect experienced by the patient is when the extremity of the head and the decollete are affected areas.

Melanosis is in most cases a problem of aesthetics, more of a „social visiting card" felt by the affected individual, within well-defined disorders that cause skin hyperpigmentation.

Material and methods: case presentation

We present the case of a 43-year-old smoker from the urban environment presenting in our clinic for the appearance of a pigmentation in the cephalic extremity, limbs and thorax with evolution over two months without significant pathological personal history. Also, the patient accuses facial pruritus and especially at the level of the decolletage. Initially, before hyperpigmentation, the patient said that face and neckline areas were pruritic and erythematous.

The clinical examination describes an eruption consisting of hyperpigmented plaques located at the face, neckline, neck and superior upper limb area, which began about two months ago. The entire face and the decollete are covered by a diffuse stretched, discreetly cross-linked, melanodermic plaque, with a significant difference in color from normal skin (the patient has constitutively hyperpigmented skin, phenotype IV). Hyperpigmentation is also evident in the upper limbs, in the upper 1/3 and the dorsal faces of the hand.

The patient is employed by a company that provides cleanliness by being in contact with a variety of detergents and cleaning agents, and cleaning products are often changed during the cleaning process. He states that the exposure to the cleaning products he works with generates a generalized pruritus, respiratory discomfort, even the simple odor causes a state of illness. All these accusations disappear when they leave their jobs. This awareness appears to have occurred more than 10 years ago when working in this environment. The introduction of new detergents a few months ago triggered this state of affairs, which is why he presented himself to the dermatologist.

Insisting on the anamnesis, we found out that she does not use facial cosmetics (creams, lotions, serums), only soap with which she washes twice a day on the face. Decorative cosmetics are rarely used occasionally. She also uses deodorant spray, a deodorant that has no contact with the face.

She was not particularly exposed to the sun, her work being mostly at night, and rests during the day. The period in which she presents is the late autumn at the end of October, but we are still thinking about an unintended sun exposure during the summer. She says she never used sunscreen creams.

The patient is not pregnant and does not use estro-progestative contraceptives or any other

medication taken as a chronic medication. She also does not suffer from osteoporosis, she does not use hormonal therapies that would be indicated in the therapy of this disease. We have insisted in this direction because the immediate diagnosis was melasma. This is all the more so since melasma is more common in people with a hyperpigmented native constitution, our patient fits into this phenotype IV.

It seems that estrogen induces melasma by stimulating the estrogen receptor present on melanocytes, resulting in increased melanin production. Also, pelvic inflammatory diseases in women, dysfunction and inflammation of the ovary and its annexes, strongly suggest involvement in the appearance of melasma / facial hyperpigmentation. [Kim and Sawhney]. Therefore, the patient was investigated on this line and no pelvic inflammation was found, the echography showing normal appearance and the inflammatory specimens (VSH, C-reactive protein, fibrinogen) being normal.

Another direction that was followed was that of endocrine hormonal investigations, performing tests to detect thyroid and adrenal dysfunction. Thyroid hormones TSH, T3, T4 as well as ATPOs were in normal limits. Also, there was neither Addison's disease nor Cushing's syndrome, with ACTH being normal.

The UV test was negative, both immediate reading and 24 hours.

Epicutan standard tests and environmental antigen testing were performed. The patient was negative in all environmental antigen tests but the epicutaneous tests at 24 and 72 hours were positive for mercapto mix, fragrance mix II and potassium dichromate. The components in these mixes, like potassium dichromate, are used industrially in the manufacture of cleaning agents, detergents and cosmetics.

These positive tests have greatly focused our attention on awareness of detergents and / or cosmetics. Because the patient says she uses very little cosmetics but uses various detergents and workplace cleaners daily, we tend to think of an awareness of detergents or their components.

The objective exam identify a asthenic normoponderal patient with a TA of 110/80 mmHg, compensated hemodynamic and respiratory. The ECG is in normal limits, as is the pneumological examination. The local exam reveals dark-brown macules, with a clear gray tendency, with a tendency to confluence in plaques, located on the face, chest, upper limbs and thighs, moderately pruritic, painless. The entire face and neckline are covered with a stretched, melanodermic diffuse plaque, finely cored, with extremely significant color difference to the healthy skin. On the periphery of melantic plaques there are hyperpigmentation spots that stand out from the normally colored skin around. We mention that the patient has a hyperpigmented constitutional skin (fig. 1, fig. 2, fig. 3).

Excisional biopsy was performed with a histopathological examination of epidermal skin fragments with moderate hyperorthokeratosis and epithelial basal hyperpigmentation that interest the malpighian inferior portion, with no cavities, discrete infiltrated focal perivascular lymphocytic infiltration and moderate macrophages loaded with brown pigment present. Histopathological aspects support the diagnosis of melanoderma. Histologically, the first stages describe a lichenification of the basal layer of the epidermis and the appearance of a perivascular dermal infiltrate. These define pigment incontinence. Afterwards the epidermis appears to be normal, but more melanophores appear in the upper dermis. The histological aspects observed by us correspond to those described in the speciality literature.

Ultrastructural studies show intra and intercellular edema of keratinocytes and multilateralisation of basal lamina, as well as migraine melanoma in the dermis (Fig. 4, Fig. 5).

Corroborating all the aspects described, the diagnosis of Riehl melanosis triggered by contact with detergents or their components is established and melasma is excluded as the main diagnosis.

Topical depigmented treatment is initiated, applied oo the lesions presented. During hospitalization, there is a slight attenuation of the lesions.

The therapeutic indication is to definitely avoid contact with substances that could trigger melanosis. The patient is also rigorously instructed to use depigmenting and photoprotective creams. Dermatocosmetic therapies (laser, peeling, etc.) had no place to be indicated,

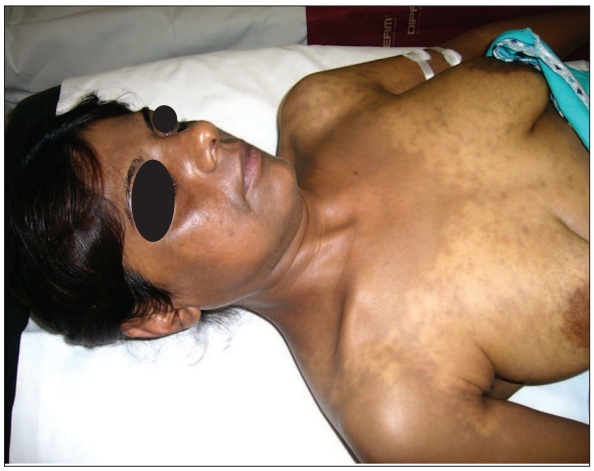

Fig. 1. Facial hypergimentation, cleavage and arms

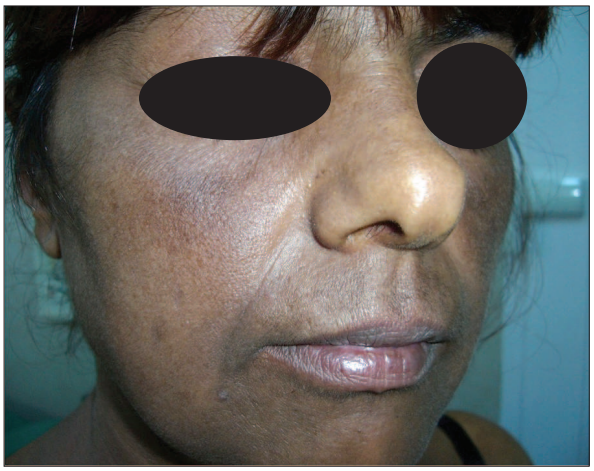

Fig. 2. Facial grey-brown accentuated hyperpigmentation

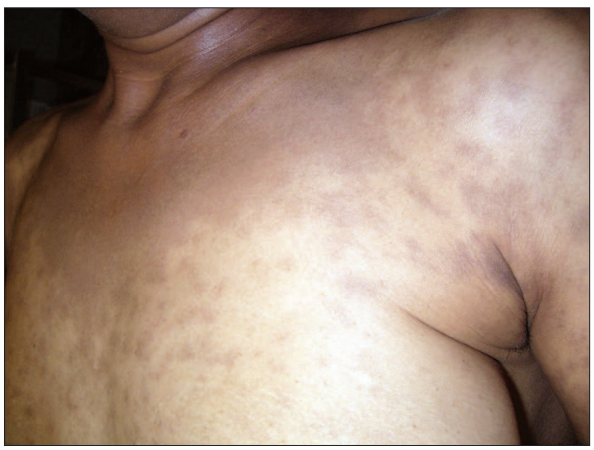

Fig. 3. Spotted and diffuse pigmentation

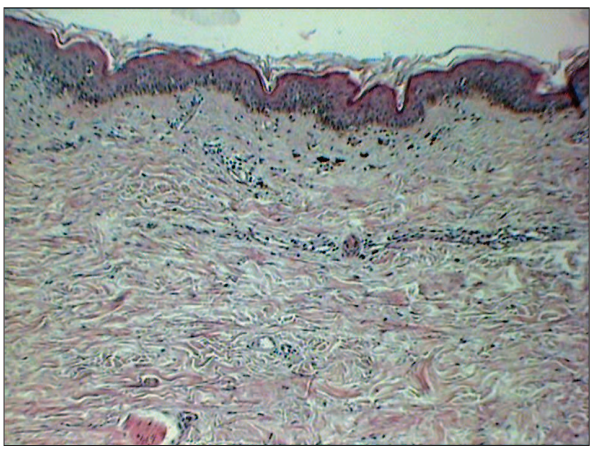

Fig. 4. Atrophic epidermis, hyperpigmentation of the basal layer. Derm - moderately inflamed and melanogenic pigment (4x col.HE)

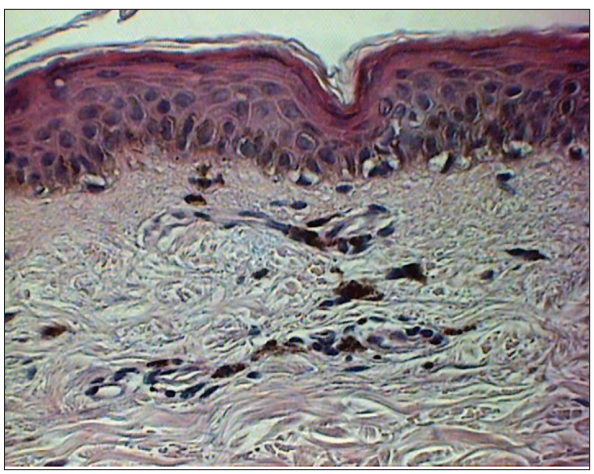

Fig. 5. Atrophy of the epidermis. Pigmentation of basal cell cells. derm - moderate lymphocytic infiltration, melanic pigment disposed granular (10x col.HE)

reported strictly to the patient's material condition, although it would probably have led to the improvement of the present hyperpigmentation. In addition, these dermatocosmetic therapies should have been carefully followed by the specialist to avoid some adverse effects such as exacerbation of pigmentation if the patient did not strictly follow the post-therapeutic protocols of the procedures.

Discussions

Skin pigmentation is a process that involves melanocyte melanogenesis, followed by the transfer of melanosomes (containing melanin resulting from tyrosinase action) by cytokine

mechanism to the neighboring keratinocytes. In humans pigmentation of the skin involves a double mechanism: 1) the production of pigment by melanocytes- Melanogenesis and 2) the distribution and transfer of the pigment to surrounding epidermal keratinocytes. Each epidermal melanocyte is surrounded by a group of keratiocytes, with which it maintains close functional contact, constituting the melanic unit. Although the number of melanic units varies depending on the topographic cutaneous disposition in the various regions of the body, the number of keratinocytes in the melanic unit is constant, around 36 [5,6].

The main fact that is involved in the color of the skin is definitely the epidermal distribution of melanin and its quantity. Variation in the number, distribution and size of melanosomes, along with melanocytes and keratinocytes, determines differences in race and ethnicity, relative to skin color.

But it seems that along with melanin there is a mixture of biopolymers located in the basal layer of the epidermis, biopolymers synthesized by melanocytes. It is known that melanin is classified according to the chemical composition in eumelanin and feomelanin. It is also known that darker-skinned individuals have more total melanin and eumelanine is found in a higher proportion than lighter skin [5].

Melanocytes form at the basal layer a dendritic network that has the role of transferring melanosomes through cytoplasmic filaments (more obvious to albinos and those with white and efelid skin) to the neighboring keratinocytes [3,7]. The neighboring melanocyte-keratinocyte complex (averaging 36) is the melanic epidermal unit with a structural and functional role, varying in different areas of the body. Melanocytes occupy a volume of 1.0-1.5 cm cubic, having a higher density in the face and the genital area than in the chest[5,6]. Their number increases after exposure to the sun and decreases with age, decreasing by about 6-8% in every decade of life.

There are also melanocytes that make a transfer of melanosomes from the perinuclear area to dendrite and vice versa, they are called melanophores. Melanin from melanosomes causes an increase in their electrons. It is recirculated in the body. Because melanin is phagocytized by leukocytes, it can circulate in the body. Thus, melanin due to the ability to circulate through the body can influence cellular metabolism. Starting with this premise, melanin may even be considered a hormone [3].

The skin's color varies according to race and ethnicity, as we mentioned above, being determined by the melanosome characteristics produced by melanocytes and then transferred to neighboring keratinocytes (their number, shape, size, and way of organizing). In the white race, melanosomes are small and are organized into membrane-adhering complexes containing three or more keratinocytes, being degraded from the basal layer of the epidermis under the action of lysosomal enzymes, while in the black race or arboriens, the melanosomes are large, having 1 micrometer length, not organized into aggregates, being intact in the stratum corneum. Melanin concentration in melanosomes in the basal layer of the epidermis is double in the black race. In addition, melanosome degradation, along with keratinocytes, is slower in darker-haired races [6].

Women are usually less pigmented than men. Pigmentation is non-homogeneous in different areas of the body, with a variable distribution depending on race (in the white race, the upper portion of the thigh is the most pigmented and the most hypopigmented lumbar area, while in the black race the abdomen is much more pigmented than the lumbar region).

In some breeds (Japanese or African-Americans, there is a clear delimitation between the shades of different anatomical regions (for example, the lateral side of the arm (darker) and the medial (lighter)). Skin pigmentation is also influenced by the geographic region, being more intense in people living in the tropics than those in the temperate zone. Hyperpigmentation offers many advantages, such as protection against intense solar radiation, elimination of phototoxic degraded cancer cells, protection against malaria or other parasites as well as malnutrition. Melanin appears to have an important role in reducing free radicals by absorbing and diffusing sunlight absorbed into the skin. As a disadvantage, people with pigmented skin absorb more heat in warmer climates and lose more heat than those with white skin in cold

climates. Also, hyperpigmentation inhibits the synthesis of vitamin D, thus favoring the development of rickets.

Pathology of melanie disorders

Pigmentation disorders occur through mechanisms that may involve: melanosomes in melanocyte melanosis of melanosomes, melanosomal secretion in keratinocytes or their transport at this level with or without their degradation.

Three mechanisms appear to be involved in determining the skin's color:

a) cellular pigment can act as an independent executor and is stimulated to act directly by light radiation;
b) melanic pigment movement within the melanophore may be under nervous control;
c) melanic pigment activity can be influenced by hormones.

From here, the interesting idea that melanin pigment acts itself as a hormone but at the same time is influenced by other hormones in the body [3].

Melanogenesis is controlled by alpha MSH (melanocyte stimulating hormone alpha), having the proopiocortin precursor. Some endocrine disorders may be associated with hyperpigmentation (Addison syndrome, Nelson's syndrome) [7]. Also, estrogens can amplify skin pigmentation.

Increasing the quantity of melanin in the epidermis is expressed by the inconsistent appearance of a brown color, whereas the accumulation of melanin in the dermis causes a gray or blue color. Etiology of this pathology involves genetic, hormonal and environmental factors (UV radiation and chemical factors). It can be generalized (malignant melanoma with metastases, including mucous), diffuse, localized (segmental) or circumscribed. Large pigment granules are observed in the skin of patients with lentigineosis, spilus nev and neurofibromatosis. Some white patients with diffuse hypermelanosis may have unaggregated melanosomes within the keratinocytes, similar to those of the black race or those treated with trimethylsoralen and subjected to UVA radiation.

Types of facial hyperpigmentation *(adapted after Vashi NA, Kundu RV. Facial hyperpigmentation: causes and treatment. British Journal of Dermatology 2013, 169 (suppl. 3) pp 41-56)* [2]

Melasma	Symmetrical, centrofacial pigmentation, color variation from light brown to dark. Historic use of anticonvulsants / pregnancy and exposure to sunlight
Ephelides	Small, hyperpigmented round, oval macules, accentuated after exposure to the sun
Lentigines	Well defined, round, oval or irregular lesions, tan to dark brown appearance
Hyperpigmentation post inflammatory	A history of inflammation with erythema and / or scales
Maturity Dyschromia (maturational dyschromia)	More frequent in black race, dark color of cheekbones and forehead to mature skin
Periorbicular hyperpigmentation	Hyperpigmentation around the eyes
Melanoza Riehl	Favored by contact with cosmetics, brown-gray color, pruritus and erythema before pigmentation in the network, diffuse
Brocq pigmented peribucal erythema	The variant of Riehl melanoma triggered by cosmetics
Exogenous ochronosis (exogenous ochronosis)	The use of hydroquinone
Acantoza nigricans	Symmetrical hyperpigmentation, soft neck and shoulder plates
Dermatosis papuloasa nigra	Pigment papules in the cheekbones and forehead
Nevul Ota	Congenital hyperpigmented, unilateral, black-blue congenital macula on the first branch of the trigeminal
Nevul Hori	Especially in Asian women, brown-gray or gray-blue macula, on the zygomatic area, rarely on forehead, tample, eyelids, nose wing
Lichen plan pigment	Oval macules, irregular gray-brown spots in sun-exposed areas
Eritema discromicum perstans (erythema dyscromicum perstans)	Gray, brownish-blue macules, in the inflammatory phase with erythematous ring

Lichen plan actinic (actinic lichen planus)	The network is fine over the violet lesions, on exposed photo areas
Follicular erythromelanosis of the face and neck (erythromelanosis folliculis faciei et colli)	Erythema, hyperpigmentative follicular papules, fever association
Post-chikungunya pigmentation	Small brownish-black macules or slices in the central area of the face, history of fever, polyarthritis
Pokilodermia Civatte	Brown-brown macules, cross-linked paternity, atrophy, telangiectasia

Treatment of melanosis

Treatment still continues to be a challenge for melanosis. The treatment includes the elimination of trigger factors when they are discovered, mandatory photoprotection, the topical application of various formulations to ensure the depigmentation of the lesions, the decorative cosmetic to camouflage the pigmentation, if there is no major contraindication, as is the case with Riehl's melanosis [8].

In the case of identifying the triggering agent, removing it results in a net improvement in hyperpigmentation. Re-exposure to the causative agent, however, leads to the recurrence of melanosis. This therapeutically method is the most convenient, with minimal costs for the success of the treatment of the condition [9].

Also, photoprotection is indicated in all cases of hyperpigmentation, as it is known that exposure to UV triggers/accentuates facial hyperpigmentation.

Hydroquinone (1,4-dihydroxybenzene) was the gold standard for the treatment of facial hyperpigmentation. However, undesirable side effects have made it banned in Europe. Hydroquinone inhibits tyrosinase activity, thereby reducing the production of melanin by melanosomes. It seems that even melanosome destruction and even inhibition of AND synthesis and RNA occurs. The hydroquinone efficiency depends on the location of the melanin pigment, the more superficial epidermal localization responding better to therapy than a profound dermal localization of melanin. Also, the concentration and vehicle of hydroxychloroquine is important in therapeutic success, and the hydroalcoholic solution is more effective than cream [9]. In order to obtain results, the therapy should last for 4-12 months, with results averaging about 6 weeks on average. The adverse reactions that occur (irritation, erythema, confetilike depigmentation, oc- nosis) are directly dependent on the duration and concentration of the product with hydroquinone.

Current topical therapies include the use of retinoids, Kojic acid, azelaic acid, ascorbic acid (Vit C), soy extracts, niacinamide, mequinol (a hydroxychloroquine derivative), glycolic acid [10,11].

The physical, dermatocosmetic therapies that can be used for melanos are laser therapy, chemical peeling or dermabrasion but with great care, among the frequent side effects, even the accentuation of post-procedure hyperpigmentation [12].

Proposed Treatments for Facial Melanosis (adapted after Damevska Katerina. New aspects of melasma. *Serbian Journal of Dermatology and Venereology*, 2014;6 (1):5-18) [5]).

Phenolic components	hydroquinone 4-hydroxyanisole (Mequinol) N acetyl 4 scistaminylphenol
Non-phenolic components	Azelaic acid Topical retinoids Kojic acid Ascorbic acid (Vit C) adapalene
Chemical peeling	Alpha hydroxy acids (AHA) Beta hydroxy acids (BHA) Jessner solution Trichloroacetic acid Glycolic acid
Physical therapies	Intense pulse light lasers microdermabrasion
Plant extracts	Arbutin, licorice extract, soy, green tea, oregonin, orchid extract, comoric acid, gentisic acid, niacinamide, arbutin
Others	Indomethacin, mercury, ZnSO4, topical corticoids, lignin peroxidase

Conclusions

Melanoses are hyperpigmentative skin disorders with incompletely elucidated etiology, but with many trigger factors: solar radiation, food deficiency, environmental and occupational factors.

Riehl melanosis is also called cosmetic pigment dermatitis or facial melanosis of women. Little is known in the literature about this condition. It seems to be more common in women than there are men affected. Initially, it appears to have been described in Vienna during the First World War, when the incriminating trigger factor was its coal and derivatives, and women were affected because the situation required them to handle the procurement and handling of coal. Nowadays, Affection occurs almost exclusively in women aged 30-50 years. It often debuts suddenly in the form of an erythema accompanied by pruritus, which is then followed by pigmentation in a brown-gray network, in many cases pigmentation being the only manifestation of the disease.

Evolution of the disease is extended for many years and it is recommended to avoid sun exposure as well as the application of photosensitising substances, the mandatory application of SPF 50 photoprotective cream.

The main trigger factor appears to be exposure to cosmetics or some ingredients in the detergent industry. Also, professional exposure to cleaning products and bituminous derivatives and tar seems very likely to trigger melanosis. However, other factors, including food, may be involved, as cases of Riehl melanosis are noted in children and people who have denied any contact with cosmetics, tar or detergents. Where there is contact with a trigger agent, its removal and interruption of exposure to the allergen led to the improvement of melanosis, after a slow evolution, for several months.

Wherever possible, the patches of incriminating agents, cosmetics, detergents, specific personal use products used by the patient, tend to orientate the diagnosis relatively easily. Even photopatch can be considered as a diagnostic test. Suspected allergen challenge tests or repeated applications with the incriminated agent can be used to clearly identify the etiology of hyperpigmentation.

Riehl melanosis is not very common and the trigger agent's detection remains a challenge.

At the same time treatment is difficult, with aesthetic results inconsistent, with high costs in many cases.

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Conflict of interest
NONE DECLARED

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