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Idiopathic Solar Dermatitis: A Literature Review

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ABSTRACT

Idiopathic solar dermatoses represent a group of diseases in which a chromophore endogenous is modified by the action of ultraviolet radiation and triggers clinical pictures characteristic.

The most common of these entities is the polymorphic solar eruption, which represents one third of all photodermatosis. The study of idiopathic solar dermatoses is important, since they are frequent entities, have a negative in the quality of life of the patient and show characteristics similar to other skin diseases.

An adequate diagnosis is crucial to determine the therapeutic approach, although on some occasions it may have spontaneous resolution. Photodermatoses are skin diseases produced by the action of electromagnetic radiation on the skin, either in the ultraviolet spectrum or in the range of visible light.

A guest is required for your presentation immunologically susceptible.

ARTICLE DETAILS

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INTRODUCTION

Electromagnetic Radiation and its Action on the Skin.

Electromagnetic radiation is divided, according to the wavelength, into:

Ultraviolet A (UVA) light: which includes wavelengths of wave between 320 and 400 nm. It is subdivided into UVA-1 light (340nm to 400nm) and UVA-2 light (320nm to 340nm).[1]

Ultraviolet B (UVB) light: located between 290nm and 320nm. It is a short wave, also known as radiation of sunburn because it produces erythema. [1]

Ultraviolet C (UVC) light, which is completely filtered by the ozone layer. Covers the range between 200 to 290nm. [1]

The visible light spectrum is between 400nm and 760nm. Although UVB is more erythematogenic than UVA, more photons in the UVA region reach the Surface terrestrial (between 10 and 100 times). Thus, a lower dose is required to produce erythema in the UVA spectrum. [1]

UVA reaches the dermis, since it does not undergo dispersión in the horny layer as it happens with UVB; besides, it is implicated in most photoallergic reactions and in idiopathic photodermatoses. [2]

The skin has multiple substrates capable of absorbing UV radiation, called chromophores, such as the keratin, hemoglobin, porphyrins, carotenes, nucleic acids, melanin, lipoproteins, peptide bonds and amino acids in proteins and urocanic acid. [2]

The effects of UV radiation on the skin include: alteration in the function of Langerhans cells, change in the proportion of lymphocyte subtypes, decrease in antigen presentation, altered production of cytokines and on the viability of monocytes. [3]

The main effectors of the immunomodulatory action of UV radiation (RUV) on the skin are cytokines, that trigger entry into and exit from the skin of various cell types. After absorbing the UVR, the cutaneous chromophore goes into an excited state, which, although it lasts just a fraction of a second is enough to produce a photoproduct, among which are oxidation of lipid membranes and the formation of dimers of pyrimidine. These photoproducts cause biochemical reactions and signals, such as the addition or removal of groups phosphate from proteins, including growth factors, and also activate transcription factors such as activator protein-1 (AP-1); the nuclear factor-KB, which in turn

initiates the synthesis of melanin, cytokines, cyclooxygenase and extracellular matrix proteins. [3,4]

IL-1 beta, TNF-alpha, and IL-8 released by the keratinocytes can induce the exit of cells from Langerhans' cells of the skin; likewise, IL-10 is associated with the migratory capacity of these cells. Parallel at the exit of the Langerhans cells, the macrophages they expand and produce their main immunosuppressive cytokine, IL-10, which counteracts the production of IL-12 by monocytes and macrophages and the production of IFN-gamma from T lymphocytes. Thus, IL-10, which also is produced by keratinocytes, TNF-alpha produced by dermal mast cells, and IL-4 favor the effect immunosuppressant in skin exposed to UVR. The generation of the Th2 lymphocyte pattern is given thanks to IL-4 and IL-10. [5,6]

The pathophysiological mechanism of photodermatoses is fully elucidated, but it is accepted that they have an immunological basis, given the clinical behavior of these diseases and histopathological findings. [7,8]

They can be classified into five categories:

1. Idiopathic photodermatoses, where the polymorphous solar eruption, actinic prurigo, dermatitis chronic actinic, solar urticaria and hydroa vacciniforme.
2. Photodermatoses due to an exogenous agent, including phototoxic and photoallergic reactions to various agents.
3. Photodermatoses due to an endogenous agent, mainly porphyrias (erythropoietic porphyrias, porphyria variegata and hereditary coproporphyria) and pellagra.
4. Photoaggravated dermatoses, such as lupus, dermatomyositis, Darier's disease, pemphigus, and bullous pemphigoid.
5. Genodermatosis with photosensitivity, among which xeroderma pigmentosum, síndrome Bloom's syndrome, Coyckayne's syndrome, Rothmund-Thomson and ataxia-telangiectasia, among others.

IDIOPATHIC PHOTODERMATOSIS

Idiopathic photodermatoses represent a group of diseases in which an unknown endogenous chromophore interacts with a specific spectrum of radiation UV and visible light and produce clinicopathological entities features. Its importance lies in the frequency, wide distribution and clinical characteristics that overlap with other diseases such as lupus erythematosus,various eczematous and urticarial dermatoses, as well as cutaneous T-cell lymphoma. [9]

For an adequate diagnosis, the evaluation of the patients with photodermatosis should include:

• Detailed clinical history: age of onset, use of photosensitizing substances, interval between the sun exposure and the onset of the rash, type of injury, duration of the same; symptoms reported in others organs, such as visceral pain, history of Disease autoimmune, and family history. [10]

• Complete physical examination: with special attention to the demarcation between exposed and covered areas. The areas that generally respect photodermatoses are the postauricular folds, the nasolabial folds, and the submental, as well as the area under the chin. [11]

• Phototest: used to determine the dose of erythema minimum (MEO), defined as the minimum dose necessary to induce discernible erythema without demarcated borders. [11]

The selected area is irradiated, generally the back, with the different wavelengths and are carried out reaction readings at 8 and 24 hours after the exposition. [12]

• Photoprovocation tests: in which the repeated exposures, with UVA, UVB and in some cases with visible light. Increasing doses are administered progressive, beginning with 3 to 4 MEO in small áreas and 2 MEO in large areas. The test ends when a pathological reaction is triggered, or after six exposures, in which case it is considered a negative result. [13]

• Photopatch tests: used to identify posible allergens implicated in a photosensitive rash. I know place the most common allergens or those presumably involved on a case-by-case basis. Two or more identical series, which are withdrawn after 48 hours and later irradiated with 0.5 to 0.7 MEO. The readings are made at 24, 48 and 96 hours. The result it is positive if there is erythema and infiltration in the área harbored the antigen. The diagnosis of Allergy photocontact is established when there is only response positive in the irradiated patch, and that of contact allergy, when there is a response in both the irradiated patch as in the one that did not receive UV light. [13]

• Laboratory tests: according to the individual clinical picture and in order to exclude an autoimmune disease, antinuclear antibodies (ANAS) should be requested, particularly anti-Ro and anti-La. Also, okay with the clinic, serum porphyrins can be ordered, stool and urine tests, such as blood tests screening to rule out cutaneous porphyrias. [13]

• Biopsy: can provide specific elements to establish the diagnosis, but its main value lies in ruling out other causes of rash. [13]

ACTINIC PRURIGO

Epidermiology

Actinic prurigo is an idiopathic photodermatosis frequent in the mestizo population of Latin America, which due to its geographical location, it receives large amounts of UV radiation during most of the year. I also know reported in indigenous peoples of the United States and Canada. [14]

This photodermatosis affects inhabitants of altitudes higher than 1000 m above sea level, 26 although in Santa Marta, Colombia, which is 1000m above sea level, up to 8% of the population suffers from prurigo actinic. [14]

The disease appears at an early age, generally in the first decade of life, around 5 to 8 years;26 it follows a chronic course and in some cases remits before puberty. It affects more frequently to women, in a 2:1 male-female ratio and in in early-onset cases, this relationship can reach be up to 4:1.28. [14]

A positive family history appears in 15% to 50% of patients with PA18 and a history of atopy in 10% of cases.The disease occurs throughout the year and worsens in the summer season. [15]

Pathogenesis

The rash can be triggered by UVA light and light UVB. MEO measurements are usually normal,however, an increase in induced erythema is reported by UVA, after topical application of indomethacin, which suggests an immunological mechanism, probably shared with the EPS. [16]

It is considered that the PA and the EPS, although they are entities clinically different, they are related. An increased risk of EPS has been shown in relatives in the first degree of consanguinity of patients with diagnosis of AP, when compared to the population general; in addition, a typical history of EPS was reported in some patients before the development of PA or simultaneously with it. The high association of PA with certain HLA molecules suggests that PA may be a form immunologically mediated EPS. There is a genetic predisposition associated with the presence of certain molecules of HLA types I and ll, depending on the geographic region where it appears the illness. In Colombia, among the Chimila indigenous people of La Sierra Nevada de Santa Marta a high occurrence is observed of HLA 840, Cw3 and Cw4. HLA-DR4 (present in 90% of patients with PA}, particularly the DRB1 *0407 subtype (present in 60% of AP cases), is considered a risk factor for conversion of EPS to PA. [17]

Especulates that these HLA molecules determine the type of radiation-induced response to a peptide antigen solar, responsible for initiating the eruption. The chromophores involved are unknown. but they could be the same ones that cause EPS, given the similarity in UV spectrum (preferably UVA) and overlap in clinical features in the two diseases; It is even considered that the hereditary EPS described in Native Americans corresponds to a PA shape.[18]

Immunology

The cells that infiltrate the skin of PA patients are T cells (LT} helpers (CD4+}, mainly memory (CD45RO). This is explained by the antigenic stimulus chronic TL, through Langerhans cells, the which have impaired ability to migrate out of the skin and therefore are appreciated in large quantities. The LTh1 produce IL-2. This cytokine has autocrine activity and paracrine, capable of stimulating the production of cytokines by TL and antibody synthesis by B lymphocytes. It is likely that IL-2 is involved by stimulating LTs to proliferate and produce cytokines that mediate pruritus, typical of PA. There is also an increase in the expression of adhesion molecules such as LFA-1, ICAM-1 and ELAM-1, a fact that implies an activation of the inmune system. [19]

TNF-alpha plays a key role in the development of actinic prurigo. Its production, triggered by the UVR, results in necrosis, induction of adhesion molecules, and infiltration by LT; In addition, it stimulates the proliferation of fibroblasts and capillaries. This is the big explanation effectiveness of thalidomide (TNF inhibitor) in the treatment of this entity. [18]

Clinical Manifestations

There are a variety of injuries. Erythematous, excoriated papulo-nodules and plaques are generally observed lichenified due to chronic scratching, which are located in exposed areas. The face, unlike the EPS, is the first to be affected; typically on the nasal dorsum, where the lesions resolve leaving small scars depressed. Other frequent locations are the V of the neck, the extensor surface of the upper extremities and the back of the hands.On some occasions the eruption spreads to covered areas, especially to the sacral region and gluteal. [18]

Treatment

Sun exposure should be restricted and instruction on the use of sunscreens; however, these measures are often insufficient to achieve control of the illness.

Antihistamines help relieve itching; are combinations of antiH1 and antiH2 are useful, especially with cimetidine, which also has an immunomodulatory effect. [18]

Thalidomide is the most effective medication in most patients. The results are as dramatic as those seen in leprosy reactions, with clearance of the lesions in two weeks. They should monitored for early adverse effects, including drowsiness, increased appetite, morbilliform rash and late ones, such as neuropathy.

CHRONIC ACTINIC DERMATITIS

Chronic actinic dermatitis (CAD) represents a wide group of skin diseases, with response abnormal to UVB. Includes photosensitive eczema, chronic photosensitive dermatitis, persistent reaction to light and the actinic reticuloid; formerly known as entities independent. Now they are recognized as part of the spectrum of the same disease. [18]

Epidemiology

In 90% of cases, CAD attacks patients boys, which have outdoor activities. The reason for the higher frequency in this age group is that aging skin has a impaired barrier function and is less effective in clearing antigens. Presentation before the age of 50 is rare with the exception of individuals suffering from dermatitis coexisting atopic. [18]

Although patients rarely realize it, the disease worsens in the summer and after exposure solar follows a persistent course. [18]

CAD occurs anywhere in the world and in all the ethnic groups The phototype does not offer special protection against the rash, as the disease is triggered by small doses of UVR. Even in some reports reports a higher frequency in phototypes V and Vl. [19]

Pathogeny

DAC is induced and maintained by exposure to small UVR doses, mainly UVB, and with les UVA frequency and visible

light. It has the clinical, histological and molecular (adhesion molecules) characteristics of allergic contact dermatitis, which is why it is considered an acquired delayed hypersensitivity response to a photoinduced endogenous antigen. [19]

The persistence of the photoallergen in the skin is considered important in the development of the allergic reaction; of that's where the name persistent light reactor comes from, however, few patients have a previous diagnosis of photoallergy. [19]

The mechanism by which a photoallergy is transforms into a DAC remains dark. It is possible that during the initial response a normal skin constituent is altered and becomes antigenic. Said initial local response, apparently induced by the covalent binding of antigen to an endogenous carrier protein, triggers the localized eczematous response. Once it transforms in DAC, the antigen is no longer necessary but the UVR evokes the response anywhere only from endogenous carrier protein, which also undergoes RUV-induced modification, thus becoming a weak antigen. One of the reactions involved in the antigen generation is the oxidation of antigen molecules histidine. [19]

Because the activating spectrum of DAC is similar to that of sunburn, in which the main target is the DNA, it is suggested that in the DAC the DNA or a molecule related acts as an antigen. [20]

The variant of CAD known as actinic reticuloid is usually a more severe dermatosis in which it does not appear a prior photoallergen is evident. On histopathology it tends to mimic cutaneous T-cell lymphoma, which can be reflex of marked and repetitive antigenic stimulation. [20]

Allergic contact dermatitis can coexist with DAC in up to 75% of cases, frequently preceding the disease. The allergen implicated with the most common is the sesquiterpene lactone (extracted from Compositae plants); others involved are the mix of fragrances, rosin, rubber and blockers solar cells. Generally, photopatch tests at these substances are negative; however, in tests vitro phototoxic activity (capacity to oxidize histidine), which contributes to the conversion of proteins in allergens. [20]

Clinical Manifestations

It is characterized by persistent eczema (at least one year duration), pruritic, with marked lichenification, which mainly affects exposed areas such as the back of the hands, the face, the scalp and the upper thorax. In severely affected patients there are erythematous, infiltrated papules or confluent plaques, which they leave islands of healthy skin. May progress to erythroderma in a significant number of patients, and sometimes presents a pseudolymphomatous character, even with cells of Sézary. [20]

Although the association has been reported of CAD with cutaneous lymphomas, this association is rare and for some authors it is a coincidence, because it is not demonstrates clonality in the infiltrate. In a study on the incidence of lymphoma in 231 patients with dermatitis chronic actinic no difference was found with respect to the general population; it is concluded then that this entity does not constitute by itself a premalignant condition. [20]

Diagnosis

In the phototest decreased MED is observed, with reproduction of the lesions in response to UVB, on occasions to UVA and in a minority to visible light. This test is useful in determining how low the dose needed to trigger the lesions is, compared to the dose that produces a sunburn, and thus measure the severity of the picture. [20]

In addition, patch and photopatch tests must be performed that include, in addition to the standard antigens, derivatives of the Compositae family, medications topical and sunscreens. They must be repeated with regularly, to detect the development of allergies to new antigens. [20]

Traditionally, steroids have been used in the topical treatment of the disease. In recent years it reports the usefulness of topical calcineurin inhibitors, such as pimecrolimus and tacrolimus, given their effectiveness in inflammatory processes mediated by LT. [20]

Azathioprine in doses of 50-150mg/d induces remission of the disease for several months; during exacerbations it is necessary to repeat the medication. Due to its high and selective capacity to inhibit the activation of T lymphocytes, cyclosporine is attributed a excellent result in the treatment of DAC. [20]

For its part, mycophenolate mofetil, with its selective action on stimulated T lymphocytes, is also very effective and should be considered as an alternative to treatment conventional in refractory cases. The exact dose of this medication is unknown, but is usually much les than that used in non-dermatological processes. The main adverse effects are gastrointestinal intolerance and leukopenia, for which follow-up with blood count and liver tests. It can even be combined mycophenolate mofetil with other therapeutic schemes, as desensitization with PUVA and oral steroids at low dose, obtaining a good response. [20]

VACCINIFORM HYDROA

Epidemiology

First described by Bazin in 1862, the hydroa vacciniforme is a very rare photodermatosis of etiology unknown.

The picture begins in childhood (around the age of six), follows a recurrent course and tends to improve spontaneously at puberty, although some patients report photosensitivity for life. In Singapore there is a report of two cases of the appearance of hydroa vacciniforme (HV) in patients around 20 years of age, which coincides with entry into military service. This late submission probably due to the fact that the children remain protected from the sun most of the time, given the hot and humid climatic conditions. [21]

The entity usually presents sporadically,although there are reports of familial cases. Men have a later onset, greater duration and severity of the disease. In a study carried out in the United Kingdom, it was estimated an incidence of

0.34/100,000 inhabitants, with a distribution similar between both sexes. [21]

Pathogeny

The action spectrum is located in UVA radiation. The lesions reproduce when exposed to UVA of wide spectrum. Some authors suggest that the absence of reproduction is a marker of good prognosis The chromophores involved and the underlying mechanism for the development of the disease are unknown; Some studies suggest damage to DNA repair. The familial aggregation cases presume a genetic defect underlying predisposition to disease. [21]

Because the course, distribution of lesions, and histology sometimes resemble those of EPS could be considered a pathophysiological mechanism Similary. The association of HV with latent EBV infection has also been reported. [21]

Clinical manifestations

The disease is characterized by vesicles and papulovesicles, which appear on the face, mainly on the cheeks, ears, nose, and on the back of the hands, although other unexposed areas can also be seen affected. These injuries resolve leaving a scar varicelliform. Between 15 minutes and a few hours after sun exposure, erythema, edema, and a burning sensation or pruritus appear. During the next 24 hours, isolated painful papules, which later become vesicular, umbilicated, crusted and sometimes hemorrhagic. In a few weeks the scab falls off, leaving a depressed, hypopigmented scar (similar to the vaccinia). Occasionally systemic symptoms occur, such as fever, general malaise, headache. [21]

Some rare presentations of the disease include ocular symptoms such as injection conjunctiva, photophobia, lacrimation, corneal ulceration that leaves a scar; and others such as deformities of the nose, ears and finger contractures. [22]

Histology

The histopathological findings of HV are characteristic of the disease. Initially a vesicle is observed intraepidermal that subsequently exhibits necrosis of the keratinocytes and spongiosis, accompanied by an infiltrate perivascular neutrophils and lymphocytes. Vasculitic changes are also reported in some case biopsy of old lesions is not diagnostic, since it is only shows an inflammatory pattern and non-specific fibrosis. Immunofluorescence is normal. [23]

Treatment

Treatment consists primarily of avoiding sun exposure and using broad-based sunscreens spectrum.

Antimalarials and beta-carotenes show limited utility. Other systemic agents such as azathioprine, thalidomide and cyclosporine have variable results and should be risk/benefit ratio should be assessed, taking into account the age group affected by the disease. [24]

In those who do not respond to intense sun Protection desensitization phototherapy with UVB can be used narrow band or PUVA, carefully monitoring a possible exacerbation, long chain polyunsaturated fatty acids (omega 3), present in fish oil, have shown a photoprotective effect in several studies. Later after oral administration, these acids are incorporated to epidermal lipids and exert an anti-inflammatory effect by competing with omega 6 acids (such as arachidonic acid) by cyclooxygenase and lipoxygenase, which results in the production of less active prostanoids. They also inhibit the production of IL-1 and TNF-alpha and due to their instability, they act as oxidizable buffers, protecting cells from free radicals. They have the additional advantage of not being toxic. [24]

CONCLUSION

There are many situations in which the skin reacts abnormally to sun exposure, and photodermatoses occupy an important place in dermatology. Idiopathic lucitis are a group of well individualized entities at present; its intimate mechanism is still incompletely elucidated, but it is beginning to be better understood. Among them, the polymorphous light eruption, particularly its minor form (benign summer lucitis), is the most frequent.

The relative frequency of photodermatoses does not seem to be unequivocal depending on the population. A study carried out in the United States in four hospital services on 1,080 patients with confirmed photodermatosis shows statistically significant differences between black and white skin: polymorphic light eruption is more frequent in black skin, contact photoallergies, and phototoxicity from medications , phytophotodermatoses, porphyria, and solar urticaria are more frequent in fair skin.

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