International Journal of Medical Science and Clinical Research Studies

ISSN(print): 2767-8326, ISSN(online): 2767-8342

Volume 02 Issue 12 December 2022

Page No: 1425-1428

DOI: https://doi.org/10.47191/ijmscrs/v2-i12-11, Impact Factor: 5.365

Polymorphous Solar Eruption and its Association with Systemic Lupus Erythematosus

Martha Paulina Ornelas-Villalobos[1], Diana Laura Núñez Arriaga[2], José Oswaldo Pérez Ruelas[3], Mariana Rueda Ruiz-Velasco[4], Esmeralda Hernández-Lerma[5], María Fernanda Hernández Carrillo[6], Paola Denisse Osorio Casimiro[7]

[1]Universidad Autónoma de Guadalajara. Guadalajara, Jalisco, México.

[2,3]Instituto Politécnico Nacional, Campus Celaya. Celaya, Guanajuato, México

[4]Escuela Nacional de Medicina y Homeopatía, Instituto Politécnico Nacional. Ciudad de México, México

[5]Universidad Autónoma del Estado de Hidalgo. Pachuca, Hidalgo, México

[6]Universidad Nacional Autónoma de México. Ciudad de México, México

[7]Escuela Superior de Medicina del Instituto Politécnico Nacional. México

ABSTRACT

Polymorphic Light Eruption (PLE) is a dermatosis related to ultraviolet radiation (UV), which is why it occurs mainly in anatomical sites exposed to the sun. It can vary in morphology from one subject to another, however, in the same patient it is usually monomorphic and the most common form of presentation is the papular variant. There is a higher prevalence of this disorder in fair-skinned people and in women, while climate and latitude are also contributing factors. Its relationship with systemic lupus erythematosus is not very clear, but its histopathology and symptoms may be the key to understanding this phenomenon.

ARTICLE DETAILS

Published On:
06 December 2022

Available on:
https://ijmscr.org/

EPIDERMIOLOGY

The polymorphous solar flare (PLE) represents the third part of all idiopathic photodermatoses, which becomes the most common of these entities. Its prevalence is estimated to be between 10-20% in Europe and North America. Although it has a wide geographical distribution, it is observed more frequently in temperate climates than in tropical areas and its frequency increases as the increases the distance from the equator. This is attributed to the variation in the proportions of UVA and UVB radiation in the different regions of the globe. In the areas where there are seasons it is more frequent during the spring and autumn months, probably for a higher ratio of UVA to UVB radiation in these seasons.[1]

The polymorphic solar eruption can affect all races and skin types. Although it is more common in individuals fair-skinned, also seen in mixed race patients black, Oriental, and Native American. [2]

However, it seems that men are affected later than women and usually have a more serious illness. The picture is also described during childhood, mainly in children from 5 to 12 years old and is called of juvenile spring eruption. It is considered a form localized polymorphic solar eruption, given the clinicopathological similarity with this entity, such as the delayed onset of the lesions once sun exposure ceases, the caracter transitory and recurrent of the same and the familiar affectation; but it differs in that it mainly affects children and young men, in the form of itchy papules located on the helix of the ears, which evolve into vesicles and then to scabs, which resolve without leaving a scar. [3,4]

GENETIC FACTORS

Family aggregation cases support the theory of a genetic susceptibility, since up to 50% of they have a positive family history; although bliss familial aggregation could result from environmental factors common, shared, without necessarily involving genetic factors.Some authors suggest that the polymorphic solar rash is inherited as an autosomal dominant character with variable penetrance. [5]

Others attribute a multifactorial inheritance, with several posible genes involved in the abnormal response to radiation UV; according to this hypothesis, there is a major or main gene, which has a high frequency in the general population (which makes the 72% of the general population susceptible

to PLE), and some polygenes that would determine which individuals will develop the disease. [6]

IMMUNOLOGICAL ASPECTS

Due to the latency time of the lesions, a delayed hypersensitivity reaction to a induced or overexpressed antigen after sun exposure. In addition, an infiltrate of T lymphocytes has been found. CD4+, observed in early lesions, which changes to a CD8+ pattern after 72 hours, and the expression of ICAM-1 (ligand for antigen associated with leukocyte function), probably secondary to IFN release range, which are findings compatible with dermatitis contact allergic and tuberculin reaction; both of them delayed hypersensitivity processes. After sun exposure, patients with PLE have abnormal depletion of Langerhans cells; in addition, they overexpress heat stroke proteins, the which could act as chromophores and are thus implicated in a possible autoimmune pathogenesis. [7,8]

The cytokine profile of PLE is unknown, but an increase in the activity of IL-6, IL-8, and possibly IL-1. In general, it is considered that the immunological defect consists of a decreased capacity for immunosuppression induced by UV radiation, which allows patients with PLE, recognize an antigen that can be expressed in all individuals. In studies where skin infiltration by neutrophils is measured, in response to UVB stimulation, a marked decrease in these cells and their products, disrupting the production of cytokines that mediate the immunosuppressive effect of UVR in normal subjects, such as IL-4, IL-1O and TNF alpha, and consequently there is a decrease in the migration of Langerhans cells out of the skin and a little change to a Th2 response pattern. [9,10]

Another mechanism proposed in the presentation of the PLE is a disturbance in tryptophan metabolism, which leads to an accumulation of kynurenic acid. But not has been proven, this theory forms the basis for the treatment with nicotinic acid. [11]

A metabolic disorder is also involved of arachidonic acid, since the topical application of indomethacin in certain patients with PLE fails to inhibit the erythema associated with UVB radiation, as occurs in normal subjects or with very mild PLE. [12]

CLINICAL MANIFESTATIONS

Despite the wide range of presentations of the PLE, it is a monomorphic eruption on the same individual, frequently composed of erythematous and pruritic papules, with symmetrical distribution. In sometimes vesicles, bullae or type forms can be seen erythema multiforme. The papular form is the most common. Followed by the plaque type and the papulo-vesicular type. It also there are vesicobullous forms, prurigo type, erythema type multiform, maculo-nodular, urticarial and hemorrhagic. [13]

The sites most frequently involved are the presternal region and the extensor surface of the upper limbs. The face is generally spared, which is attributed to a desensitization process, since is continually exposed to some degree of UV radiation. [14]

This desensitization involves the suppression of immune mechanisms, although the increase in melanization and the thickening of the stratum corneum are important factors. Although in the vast majority of cases the areas exposed, in patients with long-standing Disease lesions may appear in covered areas. [15]

Its onset takes between 30 minutes to 5 days after the sun exposure and may be preceded by sensation burning or itching. If there is no additional sun exposure, disappears in a few days to weeks (1 to 6 days), leaving no scar. [16]

It follows a recurring nature, and the distribution and the morphology of the lesions can change through the weather. On rare occasions, itching may be the only symptom. [17]

Systemic symptoms, such as headache, fever, chills and nausea are rare but can occur radiation type UVA light appears to be more effective than UVB in initiating the lesions. This claim is based on the possibility of induction of lesions when UVR is received through a glass. However, there are ambiguous results in which lesions cannot be induced by broad-spectrum UVA, and are developed in private geographical locations of UVA of short wave, which suggests an inhibitory effect of this type of vibe. Abnormal reactions to the UVB and visible radiation and it has been shown that the spectrum of action changes in an individual over time and is related to the variation of the disease according to the season weather at the time of testing. [18,19]

For induction of lesions during evaluation clinic of the disease, solar radiation is more effective simulated. This is due to a single chromophore that can be activated by different UVR spectra and give as the diversity of the lesions resulted, either through different pathogenic mechanisms or through a wide range of range of antigens activated by UVR. [20,21]

The phototest to determine MED is usually normal, although values in the lower limit are frequently observed normal. Photoprovocation tests are positive in 50% to 90% of patients18 and make it possible to establish a forecast. When they are negative, the disease presents at a younger age and tends to remit. Yes are positive, the disease follows a more chronic course and persistent. [21]

In those who undergo photopatch tests, finds many contact and photocontact allergies, mainly to sunscreens and products for daily skin care. The differential diagnosis should include: lupus erythematosus, subacute cutaneous disease, which is generally less pruritic and may be ANAS, anti-Ro, and anti-La positive. The Jessner's lymphocytic infiltration is very similar to PLE in plaques, it is persistent, but its histology is distinctive. Erythropoietic protoporphyria, phototoxic reactions and photoallergic in forms of extensive eczema as well may resemble an PLE. Finally, it should be considered photosensitive erythema multiforme. [21]

HISTOLOGY

Biopsy reveals an inflammatory infiltrate in the superficial and deep dermis, with perivascular predominance, composed mainly of T lymphocytes, and with less frequently neutrophils and eosinophils. In addition, there is edema of the papillary, perivascular, and endothelial dermis in the epidermis it is possible to find spongiosis and parakeratosis. These findings are appreciated in the papular form; in the others clinical presentations there are discrete variations, such as spongiotic vesicles and subepidermal blistering in the vesico-bullous form; and lichenoid infiltrate with great spongiosis in plaque forms. Immunofluorescence is usually negative. [22]

ASSOCIATION WITH SYSTEMIC LUPUS ERYTHEMATOSUS

It has recently been suggested that PLE and systemic lupus erythematosus (SLE) share a pathogenic mechanism common. Approximately 50-75% of patients with some of the forms of lupus are photosensitive and sometimes the lesions are indistinguishable from those seen in PLE. Up to 10% of PLE patients may have positive ANAs in the absence of lupus symptoms; sayings patients tend to have a longer duration of illness and sometimes they can develop some form of lupus. [23]

In addition, a higher prevalence of PLE has been reported in relatives of patients with lupus, which supports a genetic mechanism for the two diseases. Even for some authors, patients with PLE represent a population suffering from SLE whose manifestation on the skin it is in the form of PLE .In a study of 94 patients with PLE, a increased risk three times compared to the general population, of suffering autoimmune diseases. Come in hypothyroidism or nontoxic goiter and some patients they were diagnosed with SLE. PLE is a predisposing factor for the development of SLE, LEO and PA, although it has not been possible to demonstrate a specific gene responsible for the association between the three diseases. The specific disease phenotype can develop in individuals with PLE, in association with HLA specific, which shows that there are distinctive reactions of hypersensitivity to UV light directed by the HLA. [23]

TREATMENT

Treatment varies depending on the severity of the disease. In mildly affected people, it may be enough a behavior that avoids sun exposure, and the sunscreen application. Blockers are generally ineffective, as they primarily protect against UVB, while allowing more radiation UVA (responsible for the disease), before it appears sunburn. [24]

Those who suffer from a moderate to severe presentation also require the application of topical steroids and in sometimes a short course of oral prednisolone, especially during the desensitization process with phototherapy, to avoid thus triggering the rash. has also been Cyclosporine was used as an immunosuppressive agent. [24]

Other systemic therapies with uncertain effectiveness have been tried, including hydroxychloroquine, thalidomide, beta-carotene and nicotinamide. They can also be used antioxidant substances such as omega-3 polyunsaturated fatty acids, ferulic acid and tocopheryl acetate, but in general, there is a lack of well-designed studies to verify its effectiveness. [24]

Taking into account the adaptation and tolerance that shows the skin with repeated exposure to UVR, it is have used phototherapy. Both PUVA and UVB have been shown to be effective in controlling the disease. [25]

Narrowband UVB is preferred because it is better tolerated, and some studies have found a effectiveness comparable to PUVA. For the scheme of desensitization the minimum erythema dose is calculated (MEO) and therapy is started with 50% of the MEO; progressively increases of 10% to 20% are made in each session. Once symptom control is achieved, recommends frequent sun exposure to reinforce the desensitization scheme. It is important to remember that the remission of the disease is temporary; usually lasts between 4 to 6 weeks, which requires new sessions of phototherapy, every year approximately. During phototherapy it is not necessary to expose uncompromised areas by polymorphic solar eruption, such as the face and back of the hands. [25]

CONCLUSION

In summary, this is a dermatosis that can vary in its presentation and severity from person to person, which is related to the different immunological and pathogenic factors related to it. The differential diagnoses of other photodermatoses should be taken into account when considering this disorder, mainly due to the distribution of the lesions. Treatment may vary depending on the severity, however the importance of prevention as the first line of management is emphasized.

REFERENCES

I. Morison WL, Stern RS. Polymorphous light eruption: a commonreaction uncommonly recognized. Acta Derm Venereol (Stockholm)1982; 62: 237–240.
II. Ros AM, Wennersten G. Current aspects of polymorphous lighteruption in Sweden. Photodermatology 1986; 3: 298–302.
III. Pao C, Norris PG, Corbett M, Hawk JL. Polymorphic lighteruption: prevalence in Australia and England. Br J Dermatol1994; 130: 62–64.
IV. Khoo SW, Tay YK, Tham SN. Photodermatoses in a Singaporeskin referral center. Clin Exp Dermatol 1996; 21: 263–268.
V. Norris PG, Hawk JLM. The idiopathic photodermatoses: poly-morphic light eruption, actinic prurigo and hydroa vaccini-forme. In: Hawk JLM, ed. Photodermatology. London: Arnold, 1999;178–190

VI. Jansen CT. The natural history of polymorphous light eruptions.Arch Dermatol 1979; 115: 165–169.

VII. Kerr HA, Lim HL. Photodermatoses in African Americans: aretrospective analysis of 135 patients over a 7-year period. JAmAcad Dermatol 2007; 57: 638–643.

VIII. Epstein S. Studies in abnormal human sensitivity to light. IV.Photoallergic concept of prurigo aestivalis. J Invest Dermatol 1942;5: 289–298.

IX. Norris PG, Morris J, McGibbon DM, et al. Polymorphic lighteruption: an immunopathological study of evolving lesions. Br JDermatol 1989; 120: 173–183.

X. Norris PG, Barker JNWN, Allen M, et al. Adhesion moleculeexpression in polymorphic light eruption. J Invest Dermatol 1992;99: 504–508.

XI. K¨olgen W, Van Weelden H, Den Hengst S, et al. CD11b1cellsand ultraviolet-B-resistant CD1a1cells in skin of patients withpolymorphous light eruption. J Invest Dermatol 1999; 113: 4–10.

XII. K¨olgen W, van Meurs M, Jongsma M, et al. Differential expres-sion of cytokines in UV-B-exposed skin of patients with poly-morphous light eruption: correlation with Langerhans cellmigration and immunosuppression. Arch Dermatol 2004; 140:295–302.

XIII. Palmer RA, Friedmann PS. Ultraviolet radiation causes lessimmunosuppression in patients with polymorphic light erup-tion than in controls. J Invest Dermatol 2004; 122: 291–294.

XIV. van de Pas CB, Kelly DA, Seed PT, et al. Ultraviolet-radiation-induced erythema and suppression of contact hypersensitivityresponses in patients with polymorphic light eruption. J InvestDermatol 2004; 122: 295–299.

XV. Schornagel IJ, Sigurdsson V, Nijhuis EHJ, et al. Decreased neutro-phil skin infiltration after UVB exposure in patients with poly-morphous light eruption. Invest Dermatol 2004; 123: 202–206.

XVI. Palmer RA, Hawk JL, Young AR, et al. The effect of solar-simulated radiation on the elicitation phase of contact hyper-sensitivity does not differ between controls and patients withpolymorphic light eruption. J Invest Dermatol 2005; 124:1308–1312.

XVII. Millard TP, Bataille V, Snieder H, et al. The heritability ofpolymorphic light eruption. J Invest Dermatol 2000; 115:467–470.

XVIII. McGregor JM, Grabczynska S, Vaughan RW, et al. Geneticmodeling of abnormal photosensitivity in families with poly-morphic light eruption and actinic prurigo. J Invest Dermatol 2000;115: 471–476.

XIX. McFadden JP, Norris PG, Cerio R, et al. Heat shock protein 65immunoreactivity in experimentally induced polymorphic lighteruption. Acta Derm Venereol 1994; 74: 283–285.

XX. Tegner E, Bradin AM Polymorphous light eruption in hypopig-mented pressure areas with a UVA sunbed. Acta Derm Venereol1986; 66: 446–448.

XXI. H¨olzle E, Plewig G, Hofmann C, et al. Polymorphous lighteruption: experimental reproduction of skin lesions. J Am AcadDermatol 1982; 7: 111–125.

XXII. Ortel B, Tanew A, Wolff K, et al. Polymorphous light eruption:action spectrum and photoprotection. J Am Acad Dermatol 1986;14 (5 Pt 1): 748–753.

XXIII. Van Praag MC, Boom BW, Vermeer BJ. Diagnosis and treatmentof polymorphous light eruption. Int J Dermatol 1994; 33:233–239.

XXIV. Farr PM, Diffey BL. Adverse effects of sunscreens in photosensi-tive patients. Lancet 1989; 1: 429–431.

XXV. Baadsgaard O, Cooper KD, Lisby S, et al. Dose response and timecourse for induction of T6-DR1; human epidermal antigen-presenting cells by in vivo ultraviolet A, B, and C irradiation. JAmAcad Dermatol 1987; 17 (5 Pt 1): 792–800