ACTINIC RETICULOID

MONICA COSTESCU*,**, DIANA ANA-MARIA NIȚESCU**, IULIA IOANA ANDRONACHE**, SIMONA-ROXANA GEORGESCU*,**, OANA-ANDREIA COMAN*, **

Summary

Actinic reticuloid is a rare, persistent, sometimes debilitating dermatosis of the sun exposed skin areas, which usually affects persons with chronic sun exposure. The particular feature of this pathology derives from the similarity with cutaneous lymphoma both clinically and histologically, yet presenting a small risk of malignant transformation.[1]Clinically, the eruption of chronic actinic dermatitis is pruritic, confluent, or in separated plaques, mainly in sun exposed areas, affecting normal skin, in patients with a history of dermatitis or rarely after chronic consumption of photosensitizing drugs.

We report a case of actinic reticuloid, the severe form of chronic actinic dermatitis, in a 49-year-old female patient with a history of moderate sun exposure. The diagnosis was supported by the clinical aspect and subsequently confirmed by the histopathological aspect.

Key words: *chronic actinic dermatitis, actinic reticuloid, phototherapy.*

Received: 13.07.2017 *Accepted: 29.08.2017*

Introduction

The first references to a suggestive pathology for chronic actinic dermatitis date back to 1933, when it was called persistent light reaction. It was not until 1969 that its severe form, named actinic reticuloid, and then the moderate variant, called photosensitising dermatitis, were introduced into skin rashes induced by sun exposure category.[1]

Actinic reticuloid is the most severe variant of the chronic actinic dermatitis spectrum. Etiology is still unknown, but is likely multifactorial, involving contact allergy, photoallergy, phototoxicity, immunological and metabolic factors.

Three criteria are required for the diagnosis of reticuloid syndrome:

1. Persistent papules and plaques on sun exposed areas with extension on covered areas or generalized erythroderma,

2. Photosensitivity to a wide range of ultraviolet, including UV-B, UV-A and some part of the visible spectrum

3. Histopathological examination with dermal infiltration and lymphoid atypical cells.

Case presentation

We report the case of a 49-year-old woman presenting for a poor defined eruption consisting of brown-erythematous papules on the neck, anterior and posterior thorax, accompanied by a erythematous, finely squamous eruption located on the cheeks and forehead, pruritic, with an evolution of about 2 years. Blood tests were within normal limits and immunological tests revealed anti-vascular antibodies as well as anti-scl-70 antibodies. Biopsy of the lesion from the posterior thorax was performed. The histopathological

* ''Carol Davila'' University of Medicine and Pharmacy Bucharest, Romania.
** Clinical Hospital of Infectious and Tropical diseases''Prof. Dr. Victor Babeș'', Department of Dermato-Venereology, Bucharest.

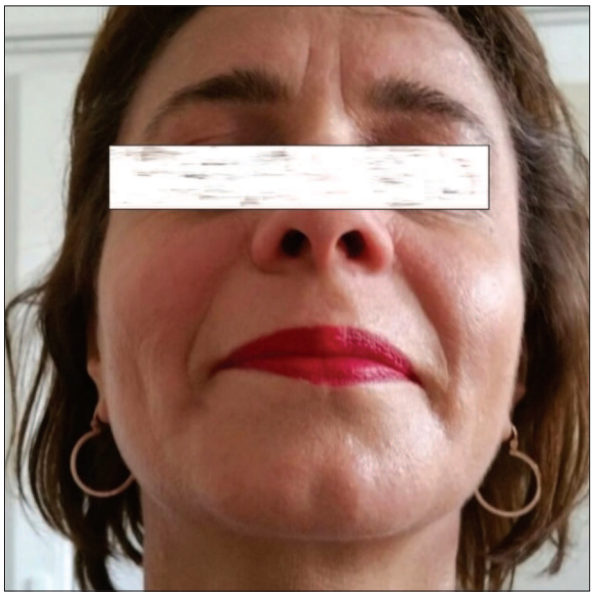

Fig. 1. Actinic reticuloid, facial involvement

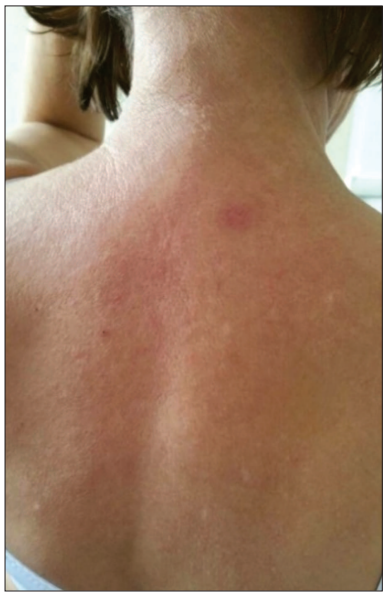

Fig. 2. Actinic reticuloid, posterior thorax involvement

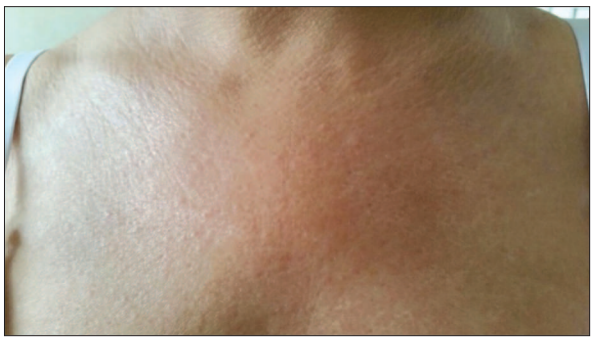

Fig. 3. Actinic reticuloid, anterior thorax involvement

examination revealed ortokeratosis, irregular epidermic acanthosis, focal hyper-granulosis, minimal spongiosis and exocitosis; centrally with cystic dilation of the follicular ostium, covered by parakeratine and crust with serofibrin. In the dermis, dilated vessels appear, surrounded by dense infiltration of lymphocytes, histiocytes and rare eosinophils, disposed perianexially and perivascularly. Histopathological diagnosis confirms the clinical diagnosis of reticuloid syndrome or chronic actinic dermatitis.

It should be noted that the patient had received prednisone at moderate doses (30mg / day), over a period of one month, about 6 months prior to admission, with the disappearance of the eruption, but with a gradual recurrence after systemic corticotherapy was stopped. The

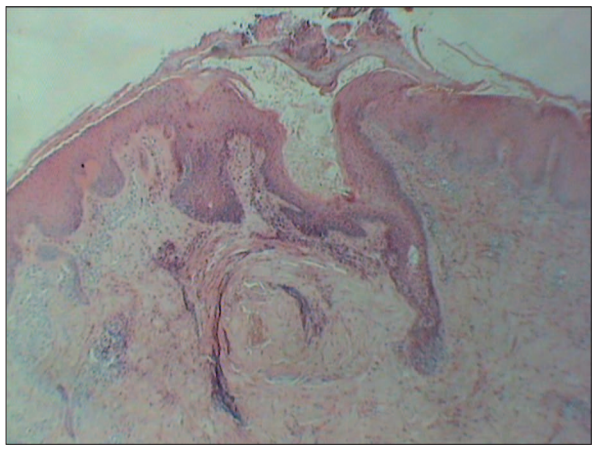

Fig. 4. Overall image, 4x lens, hemalaun-eosin staining. Cystic dilation of the ostium covered by a crust, irregular acanthosis and parakeratosis are observed

evolution was favorable under systemic treatment with antihistamines, sedatives, vitamin supplements and under topical treatment with high potency dermatocorticoids(class III a) and emollients. The patient was advised to avoid sunlight exposure and the use of sun screens (SPF 50+) with periodic follow-up control.

Discussions

Chronic actinic dermatitis spectrum comprises persistent light reaction, actinic reticuloid, photosensitizing eczema and

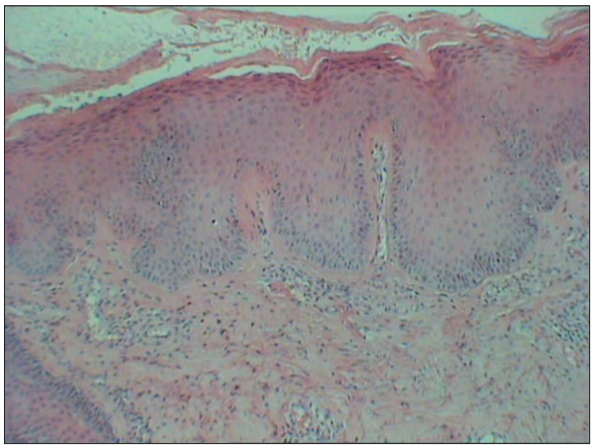

Fig. 5. 10x lens, detail. Irregular acanthosis, hypergranulosis, minimal exocytosis, minimal spongiosis and degraded dermal collagen are observed

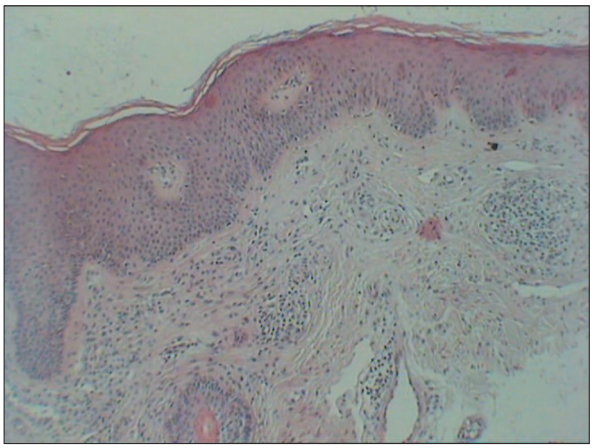

Fig. 6. 40 x Objective. The type of lymphocytic infiltrate and degraded collagen are observed

photosensitising atopic dermatitis. Persistent light reaction has been reported since 1960 in individuals with photo-allergic dermatitis associated with exposure to halogenated salicylates, persisting even after the exposure was stopped [2]. Actinic reticuloid presents an identical pattern as the persistent light reaction with the particularity that at histopathological examination atypical lymphocytes are described. The photosensitising eczema is similar to the persistent light reaction except for the absence of a history of photoallergy [2].

The solar spectrum involved in triggering chronic actinic dermatitis is represented by UVA and UVB. The theory that chronic actinic dermatitis is a delayed hypersensitivity response common to allergic contact dermatitis is confirmed by the dermal infiltrate with CD8 positive lymphocytes and by the pattern of adhesion molecules involved in this process.

Chronic actinic dermatitis typically affects sun exposed people, whether by their job nature or not, having a pre-existing photo-allergic contact dermatitis to exogenous sensitizers such as compositae or sunscreen creams. It is thus possible that chronic photo-destruction to diminish normal skin immunosuppression in these individuals and on the other hand, exogenous contact dermatitis to increase the immune response against a weak, physiologically non-immunogenic endogenous photo-antigen.

The harmful effects of ultraviolet rays derive from their ability to be absorbed into the skin and, implicitly, by the changes they can cause. Thus, UVA rays (400-315nm) are absorbed as far as the profound dermis, UVB (315-290 nm) in epidermal layer and UVC (290-200nm) in the stratum corneum and superficial layers of the epidermis. Once absorbed by biomolecules, they trigger a chain of photochemical and photo-biological reactions. Chromophores, defined as light-absorbing molecules, are transformed either directly by photochemical reactions or indirectly through an intermediate molecule in a photosensitizing reaction. The affected cell reacts by clinically visible changes. Other layers of the skin may be affected as a result of the pro-inflammatory cytokines involved in this process.

The effects of ultraviolet light on the immune system are both pro-inflammatory and immunosuppressive. Photoreceptors that trigger the activation of the immune response signalling pathways are: DNA (by degradation products), urocanic acid (by isomerization from the trans form to the cis form) of the stratum corneum, and membrane lipids (by altering the redox membrane potential).[1] According to recent studies, the cis form of urocanic acid induces gene transcription and the synthesis of immunomodulatory mediators in human keratinocytes in vitro.[3] Also, other studies have shown that ultraviolet rays activate the innate immune system by stimulating the production of

antimicrobial peptides in the human skin, as is the case of psoriasin, thereby deriving beneficial effects on immunity.[3] In fact, a controlled degree of induced immunosuppression of adaptive immunity may be beneficial in preventing immune response to photo-antigens, as in the case of polymorphic light rash.

From a clinical point of view, actinic reticuloid generally occurs in middle-aged men, especially during summer, and appears in the form of pruritic eczema (papules, nodules or plaques) in the exposed areas (cheeks, ears, throat, posterior forearms), sparing the upper eyelids and skin folds. Occasionally, unexposed areas may also be affected, phenomenon explained either by extreme light sensitivity,with penetration through the clothes fabric, or by the immunological reactions propagated in the unexposed skin. In severe cases, generalized erythroderma can occur [4]. The tegument is thickened and sometimes covered with sheds, even with leonin facies, in more severe forms.

The differential diagnosis of actinic reticuloid is done with photoaggravated dermatoses, with photosensitization on certain drugs, with T-cell lymphoma, with erythroderma of other causes. Negative results of phototests indicate cutaneous lymphoma while infiltrates with CD8 positive cells and the prompt response to treatment indicates chronic actinic dermatitis with its forms. Also, immunohistochemistry and gene rearrangement assays are required to confirm the diagnosis of T-cell lymphoma.[5]

Photo-aggravated dermatoses can lead to a drastic decrease in the quality of life of patients[6] , therefore, besides medication it is necessary to learn a particular lifestyle that supposes avoiding sun exposure, wearing appropriate clothing, using periodically photoprotective creams and avoiding exposure to allergens (mention on the important role of paraphenylendiamine as a contact allergen). [7]

First-line treatment involves: strict photoprotection and avoidance of contact allergens, topical corticosteroids and emollients. Second line therapy includes: azathioprine, cyclosporine, mycophenolate, hydroxycarbamide, UVA photochemotherapy and narrowband UVB phototherapy as well as topical calcineurin inhibitors. [8] Third line treatment includes: hydroxychloroquine, etretinate, danazol, thalidomide, interferon and infliximab.[9]

It is interesting to note the positive effect achieved by systemic therapy with UVA at lower doses than those that actually trigger the disease.[10] The association at this therapy of corticosteroids or azathioprine, during the induction period, is very important. According to literature studies, despite the clear benefits of this type of therapy, maintaining remission over time is a possible achievement in a relatively limited number of patients.[10],[11] Azathioprine is often the rescue drug in many refractory cases following clinical trial experience.[12]

The positive evolution of the disease with the adoption of the right treatment measures is clearly admitted by most studies but per se healing is practically difficult to predict.[13]

Conclusions

We have reported an atypical actinic reticuloid case in a 49-year-old woman, with a moderate history of sun exposure during the year, who presented for an exacerbation of the disease in the winter months, suggesting increased sensitivity to ultraviolet rays. Although the known malignant transformation risk is low, periodic monitoring of the patient is recommended.

Bibliography

1. Bologna L. Jean , Jorizzo L. Joseph, Schaffer F. Julie, Dermatology 3rd Edition, Saunders Elsevier, 2012.
2. O. Braun Falco, Braun Falco's Dermatology, 3rd edition, Springer, 2009.
3. Tony Burns, Stephen Breathnach, Neil Cox, Christopher Griffiths, Rook's Textbook of Dermatology, Wiley Blackwell, Cutaneous Photobiology, Chapter 29.4.
4. Neena Khanna, Saurabh Singh, Bhutani's Color atlas of Dermatology, sixth edition, Health Sciences Publisher, 2015.

5. V Bakels, J W van Oostveen, A H Preesman, C J Meijer, and R Willemze, Differentiation between actinic reticuloid and cutaneous T cell lymphoma by T cell receptor gamma gene rearrangement analysis and immunophenotyping. J Clin Pathol. 1998 Feb; 51(2): 154–158.
6. The quality of life of 790 patients with photodermatoses. Jong CT, Finlay AY, Pearse AD, Kerr AC, Ferguson J, Benton EC, et al. Br J Dermatol 2008; 159: 192–7.
7. Contact and photocontact sensitization in chronic actinic dermatitis: a changing picture. Chew AL, Bashir SJ, Hawk JLM, Palmer R, White I, McFadden JP. Contact Dermatitis 2010; 62: 42–6.
8. Treatment with topical tacrolimus favours chronic actinic dermatitis: a clinical and immunopathological study. Ma Y, Lu Z. J Dermatolog Treat 2010; 21: 171–7.
9. Mark G. Lebwohl, Warren R. Heymann, John Berth-Jones, Ian Coulson, Treatment Of Skin Disease, Comprehensive therapeutic strategies, 4th edition, Elsevier Saunders, 2014
10. PUVA therapy of chronic actinic dermatitis. Hindson C, Spiro J, Downey A. Br J Dermatol 1985; 113: 157–60.
11. Masood Q, Sameem F, Hassan I, Khan D, Majid I, Bhat T, Singh G. PUVA therapy in chronic actinic dermatitis: A preliminary study. Indian J Dermatol 2005;50:212-5
12. LEIGH, I.M. and HAWK, J.L.M. (1984), Treatment of chronic actinic dermatitis with azathioprine. British Journal of Dermatology, 110: 691–695. doi: 10.1111/j.1365-2133.1984.tb04706.x
13. Robert S. Dawe, MRCP; Iain K. Crombie, PhD; James Ferguson, FRCP, The Natural History of Chronic Actinic Dermatitis, Arch Dermatol. 2000;136(10):1215-1220.

Conflict of interest
NONE DECLARED

Correspondance address: Diana Ana-Maria Nițescu
Clinical Hospital of Infectious and Tropical diseases " Prof. Dr. Victor Babeș"
Bucharest, Mihai Bravu Street, no 281
E-mail: drnitescudiana@gmail.com