

A Case of Actinic Reticuloid

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Actinic reticuloid as a manifestation of chronic actinic dermatitis (CAD) is a rare dermatosis whose clinical and histologic features resemble other types of pseudolymphomas including mycosis fungoides and Jessner's lymphocytic infiltration, and it is regarded as an eventual stage of various photodermatoses like photosensitive eczema or persistent light reaction or chronic photoallergic contact dermatitis and so on. Phototests in the patients with actinic reticuloid usually reveal hypersensitivity to UVB, UVA, and sometimes to visible light.

We present a case of actinic reticuloid in a 65-year-old male, whose skin lesions developed as erythematous lichenified infiltrating plaques on the face at first, and then spread themselves onto the upper trunk later. Histologically those skin lesions showed the aggregation of atypical lymphocytes and photobiologically the results of phototests revealed photosensitivity to UVB and UVA. (*Ann Dermatol* 11(4) 240~243, 1999).

Key Words : Actinic reticuloid, Pseudolymphoma, Cross-photosensitivity.

Actinic reticuloid is an uncommon, chronic photosensitivity dermatosis with clinical and histologic features resembling lymphoma and is observed mostly in middle-aged and elderly men.^{1,2}

Clinically, extremely pruritic, infiltrated patches or plaques are present on sun-exposed skin areas, and often extend to covered areas and may transform to erythroderma later.^{3,4,5} The histologic features resemble those of cutaneous T cell lymphoma, Jessner's lymphocytic infiltration, and lymphomatoid contact dermatitis, etc.^{6,7}

In spite of the lymphomatoid histologic appearance and some frustrating intractable clinical course, actinic reticuloid is regarded as one of pseudolymphomas because it does not transform to true lymphoma in life although there were some controversies in early publications. The action spectra of photosensitivity include UVB, UVA, and usually the visible light.³ Although no specific therapy is re-

wardable for actinic reticuloid, treatment modalities with proven longstanding effectiveness are PU-VA⁸, UVB⁹ phototherapy, azathioprine^{10,11} and recently cyclosporine.^{9,12}

REPORT OF A CASE

A 65-year-old man developed severely pruritic skin lesions confined to the face and dorsal surfaces of the hands in September, 1993. He had suffered from nephrotic syndrome and received systemic corticosteroids for 9 months. On the initial examination, the eruption consisted of erythematous patches in the sun-exposed areas aforementioned sparing the covered areas. He had been treated with topical steroids and oral antihistamines but his disease showed an intractable course. In June, 1995, multiple tumors developed on the face abruptly and a skin biopsy from a lesion revealed perivascular and periappendageal aggregation of lymphoid cells mimicking a histologic finding of Jessner's lymphocytic infiltration. The laboratory findings of the following items were negative or within normal limits; complete blood cell count, urinalysis, LFT, ANA, immunoglobulin series,

Received August 21, 1998.

Accepted for publication January 14, 1998.

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Fig.1. Multiple, erythematous plaques and nodules on the face.

BUN / Creatinine. In spite of 5 months' treatment with prednisolone and hydroxychloroquine, the lesions progressively increased in number as their sizes enlarged (Fig. 1). In November, 1995, the patient was received azathioprine (150mg/day) for 3 weeks with some response but it was discontinued due to telogen effluvium. His disease showed cyclic pattern afterwards ; some improvement in the winter season but aggravation in the following spring. At that time a second biopsy was performed with systemic survey by X-ray and CT scan and bone scan to exclude lymphoma and its metastasis. A phototest and photopatch test were also done to detect any photosensitivity. The biopsy specimen showed aggregation of atypical lymphocytes around the blood vessels and the adnexal structures with some eosinophils (Fig.2 A,B). The immunohistochemical studies revealed that most of the infiltrated cells consisted of pan T cells and more than 50% of the infiltrated lymphoid cells were CD8+ cells (Fig.3). The systemic survey showed no evidence of metastasis. The phototest showed a slightly decreased MED-UVB, 30mJ/cm² (normal, >50mJ/cm²) and a normal ranged MED-UVA, 25J/cm² and no sensitivity to visible light. The photopatch test showed a positive reaction to promethazine, which had no clinical relevance such as a history of the application of topical antihistamines. He was placed on 400mg of hydroxychloroquine each day and antihistamines with topical corticosteroid ointment as well as instructed to avoid sunlight. Despite the treatment, his skin lesions were aggravated in early summer, 1996, spreading to the covered areas; chest, shoulder, and upper back area. After this, his skin lesions

Fig. 2. A,B. A. A dense dermal mononuclear cell infiltrate in the dermis. (×40, H&E stain) B. Aggregation of atypical lymphocytes with some eosinophils around the blood vessels and adnexal structures. (×200, H&E stain)

Fig. 3. Most of infiltrated cells consist of CD8+ lymphocytes (×100, CD8 stain).

improved in late autumn, keeping a seasonal fluctuation of improvement and worsening afterwards till now.

DISCUSSION

Actinic reticuloid can be regarded as the most severe variant in the spectrum of chronic actinic dermatitis (CAD). The exact etiology is still unknown, but is probably multifactorial, involving contact allergic, photoallergic, phototoxic, immunologic, and metabolic factors^{13,14,15,16}.

A diagnosis of actinic reticuloid should only be made when the following criteria are present; (1) persistent infiltrated papules and plaques on sun-exposed skin, often with the extension to covered areas or generalized infiltrated erythroderma; (2) photosensitivity to broad spectrum of wavelengths, including UVB, UVA, and visible light; (3) a dermal infiltrate with presence of atypical lymphoid cells on histologic examination⁶.

When one or two criteria are lacking, one could prefer the more general term chronic actinic dermatitis (CAD) which includes photosensitive eczema and persistent light reaction⁶. These show the clinical features sometimes similar to actinic reticuloid.

The essential difference was that the photosensitivity in photosensitive eczema was confined only to the UVB range¹⁷. Moreover, skin biopsy specimens showed changes suggestive of chronic lichenified eczema. The histologic appearance did not mimic mycosis fungoides, and frankly atypical cells were never seen.

Persistent light reaction occurs in elderly men and is characterized by a long-standing photodermatitis. Eczematous lesions and a positive photopatch test for a photocontact sensitizer are present. Although the sensitivity is mainly in the UVB region, others have found abnormal reactions to UVA as well. The histologic features are those of an eczematous dermatitis. Progression from photoallergic contact dermatitis by means of persistent light reaction to actinic reticuloid has been reported^{18,19}. Our patient revealed a positive result for promethazine on a photopatch test. But we could not find any related history with this drug, which was one of offending agents for persistent light reaction when they applied it topically in the 1950s and 1960s. We guess that such a positive photopatch result can be related to the cross-photosensitization between other phenothiazine antihistamines and promethazine itself in test allergen²⁰.

Actinic reticuloid, as a variant of chronic actinic dermatitis (CAD), may result from the dysregulation of suppressor mechanisms, enabling the development of hypersensitivity to UV-induced neoantigen or the altered self proteins²¹. It is not known why preferentially CD8+ lymphocytes occur in actinic reticuloid. Similarly, there is also an increase of CD8+ cells in late lesions of polymorphic light eruption as compared with early lesions, perhaps contributing to local suppression of the inflammatory response²². The exposure of relatively higher doses of UVB radiation to the susceptible victims would reduce the ability of antigen presenting cells (APC) to initiate contact hypersensitivity responses and results in the induction of hapten-specific suppressor T cells²³.

Several authors have stated that the histopathologic features of skin and blood specimens in actinic reticuloid can closely resemble mycosis fungoides or Sezary syndrome. But there are some important differences between them. In several cases of actinic reticuloid, dermal fibrosis and multinucleated fibroblasts can be seen in the papillary dermis. This is absent or inconspicuous in cases of cutaneous T cell lymphomas⁶. In addition, definite features of Pautrier's microabscesses are absent in actinic reticuloid, even if the exocytosis of lymphocytes into the epidermis sometimes may mimic them⁶. An important diagnostic feature in actinic reticuloid is that the occurrence of a dermal infiltrate composed mainly of suppressor T cells (CD8+ cells), in contrast to cutaneous T cell lymphoma, in which a helper T cell (CD4+ cells) proliferation is virtually always seen^{24,25,26,27}.

In erythrodermic patients, the presence of a CD8+ dominated in dermal infiltrate and, especially, a reversed CD4+ to CD8+ ratio in the peripheral blood are highly characteristic for a definite diagnosis of actinic reticuloid⁶. And we can hypothesize a chronic reactive immunoregulatory disorder is involved in actinic reticuloid, as has been hypothesized for other T cell chronic proliferations, by the fact that the polyclonal expansion of circulating T lymphocytes expresses the suppressor/cytotoxic phenotype in peripheral bloods of the patients.

REFERENCES

1. Horio T : Actinic reticuloid via persistent light re-

- action from photoallergic contact dermatitis. *Arch Dermatol* 118: 339-342, 1982.
2. Healy E and Rogers S : Photosensitivity Dermatitis/Actinic Reticuloid Syndrome in an Irish Population: A Review and Some Unusual Features. *Acta Derm Venereol*(Stockh) 75: 72-74, 1995.
 3. Norris PG and Hawk JLM : Chronic Actinic Dermatitis. *Arch Dermatol* 126: 376-378, 1990.
 4. Oh JG, Chun HS, Youn JI : A Case of Actinic Reticuloid. *Kor J Dermatol* 33 : 534-539, 1995.
 5. Bakels V, van Oostveen JW, Freesman AH, et al. : Differentiation between actinic reticuloid and cutaneous T cell lymphoma by T cell receptor gamma gene rearrangement analysis and immunophenotyping. *Clin Pathol* 51: 154-158, 1998.
 6. Toonstra J : Actinic Reticuloid. *Seminars in Diagnostic Pathology* 8: 109-116, 1991.
 7. Ploysangam T, Breneman DL, Mutasim DF : Cutaneous pseudolymphomas. *J Am Acad Dermatol* 38: 877-895, 1998.
 8. Hindson C, Spiro J, Downey A : PUVA-therapy of chronic actinic dermatitis. *Br J Dermatol* 113: 157-160, 1985.
 9. Toonstra J, Henquet CJM, van Weelden H, et al. : Actinic reticuloid. A clinical, photobiologic and follow-up study of 16 patients. *J Am Acad Dermatol* 21: 205-214, 1989.
 10. Leigh IM and Hawk JLM : Treatment of chronic actinic dermatitis with azathioprine. *Br J Dermatol* 110: 691-695, 1984.
 11. Murphy CM, Maurice PDL, Norris PG, et al. : Azathioprine treatment in chronic actinic dermatitis: A double-blind controlled trial with monitoring of exposure to ultraviolet radiation. *Br J Dermatol* 121:639-646, 1989.
 12. Norris DG, Camp RDR, Hawk JLM : Actinic reticuloid: Response to cyclosporine. *J Am Acad Dermatol* 21:307-308, 1989.
 13. Frain-Bell W : Photosensitivity dermatitis and actinic reticuloid. *Semin Dermatol* 1: 161-163, 1982.
 14. Vandermaesen J, Roelandts R, Degreef H : Light on the persistent light reaction-photosensitivity dermatitis-actinic reticuloid syndrome. *J Am Acad Dermatol* 15: 685-692, 1986.
 15. Toonstra J, Henquet CJM, van Weelden H, et al. : Actinic reticuloid. *J Am Acad Dermatol* 21: 205-214, 1989.
 16. Roelandts R : Chronic actinic dermatitis. *J Am Acad Dermatol* 28: 240-249, 1993.
 17. Ramsay CA and Black A : Photosensitivity eczema. *Trans St John's Hosp Dermatol Soc.* 59: 152-158, 1973.
 18. Ive FA, Magnus IA, Warin RP, et al. : Actinic reticuloid: a chronic dermatosis associated with severe photosensitivity and the histological resemblance to lymphoma. *Br J Dermatol* 81: 469-485, 1969.
 19. Hawk JLM : Resolution of actinic reticuloid with transition to photosensitive eczema. *J Roy Soci Med* 71: 608-610, 1978.
 20. Rietschel R, Fowler, JR JF : Fisher's CONTACT DERMATITIS. 4th ed. Williams & Wilkins, Baltimore , 1995. pp 121-122.
 21. Kim KH, Nam JT, Joh GY : A Case of Chronic Actinic Dermatitis. *Kor J Dermatol* 30: 906-912, 1992.
 22. Norris PG, Morris J, McGibbon DM, et al. : Polymorphic light eruption: An immunopathological study of evolving lesions. *Br J Dermatol* 120:173-183, 1989.
 23. Daynes RA, Spellman CW : Evidence for the generation of suppressor cells by ultraviolet radiation. *Cell Immunol* 31: 182-187, 1977.
 24. Ralfksier E, Wantzin GL, Stein H, et al. : Photosensitive dermatitis with actinic reticuloid syndrome: an immunohistological study of the cutaneous infiltrate. *Br J Dermatol* 114: 47-56, 1986.
 25. Takigawa M, Tokura Y, Shirahama S, et al. : Actinic reticuloid: an immunohistochemical study. *Arch Dermatol* 123: 296-297, 1987.
 26. Toonstra J, van der Putte SCJ, van Wichen DF, et al. : Actinic reticuloid: immunohistochemical analysis of the cutaneous infiltrates in 13 patients. *Br J Dermatol* 120: 779-786, 1989.
 27. Rujita M, Miyachi Y, Horio T, et al. : Immunohistochemical comparison of actinic reticuloid with allergic contact dermatitis. *J Dermatol Sci* 1: 289-296, 1990.