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ACTINIC RETICULOID : A STUDY OF 12 CASES

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Clinical study of 12 Libyan male patients of actinic reticuloid (AR) treated since 1982 is presented. 9 patients had wheatish complexion and 3 were black. Median age at onset of symptoms was 47 years. All the patients had pruritic infiltrated plaques and papules on the exposed sites with involvement of covered parts in 4 patients. 2 patients had significant dermopathic lymphadenopathy. All the patients showed partial improvement with the use of local sun-screens in day and steroid ointment at night. 5 patients showed significant improvement with systemic steroid therapy and 1 with chloroquine. Azathioprine therapy tried in 1 patient was ineffective. Later the same patient was given oral cyclosporine 4-5 mg/kg/day which resulted in almost complete clearence of the lesions at first but later failed to respond. Complete clearence of lesions could not be atained in any of the patients.

Key Words : Actinic reticuloid (AR), Prednisolone, Chloroquine, Azathioprine, Cyclosporine

Introduction

Actinic reticuloid (AR) is a chronic persistent photosensitive dermatosis of elderly men, described first by Ive and co-workers in 1969.[1] It is clinically characterized by pruritic infiltrated papules and plaques on exposed parts with occasional extension to covered areas and rarely associated with episodes of erythroderma. Action spectrum involves UVB, UVA and visible light beyond 400 nm.[2,3] Histologically it resembles lymphoma.[1] Immunohistochemical analysis of the cutaneous infiltrate done by Toonstra et al[4] revealed presence of activated T cells, numerous histiocytes, macrophages and B cells. In majority of their patients, most of the lymphocytes expressed the phenotype of suppressor cells; the number of Leu 8^+ cells was inversely proportional to HLA-DR expression by the dermal infiltrate which suggested a negative correlation between a state of activation and the concentration of Leu 8^+ cells. They also found many IgE bearing dendritic cells in the dermis associated with elevated serum IgE levels.

We have treated 12 Libyan cases of AR in last 11 years. The date of their clinical features, histopathologic picture, response to various treatment regimes and follow-up are presented.

Materials and Methods

All the 12 patients were admitted to the ward for initial assessment, investigations and treatment. Details of history, clinical features, results of investigations, treatment and follow-up were recorded in a special protocol. The diagnosis was based on typical clinical features and histopathology.

Results

All the patients were Libyan males. 9 patients had wheatish complexion and 3 were black. Age at onset of symptoms ranged between 39 years to 55 years (median age 47 years) and the age at diagnosis between 47 years to 70 years (median age 52.5 years). 6 patients were farmers, 2 drivers, 2 shop-keepers and 2 masons. In 6 patients with job of farming, lesions started appearing while they were in active job and remaind persistently progressive even after retirement.[2] Two patients were masons and were in

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frequent contact with cement. Initially they developed lesions like contact dermatitis on hands, feet and face. They later gave up the job but even after that the lesions were persistly progressive.

All the patients initially had recurrent erythematous eczematous lesions, more severe during summer but gradually the lesions became persistent and increased in thickness. At the time of diagnosis all the patients had infiltrated papules and plaques. In adddition 2 patients had erythematous scaly lesions. Distribution of the lesions is shown in Table 1. Significant lymphadenopathy of cervical and axillary region was observed in patients No. 6 and that of cervical in patients No. 8. All the patients complained of moderate to severe itching and burning sensation on exposure to sun light.

Table I. Distribution of lesions :

Sites	No. of Patients
Face	12
Back	10
V of neck	8
Dorsum of hands	10
Palms	3
Forearms	7
Dorsum of feet & ankles	4
Soles	1
Trunk & other unexposed parts	4

Patient No. 6,8,9 and 11 were suffering from moderate hypertension and were on antihypertensive drugs. Patient No. 8 developed diabetes while on systemic steroid therapy and patients No. 11 was a known diabetic for last 10 years.

Routine investigations on blood, urine and stool did not reveal any significant abnormality.

Histopathological studies were performed in all the patients. Biopsies were repeated at intervals in some patients.

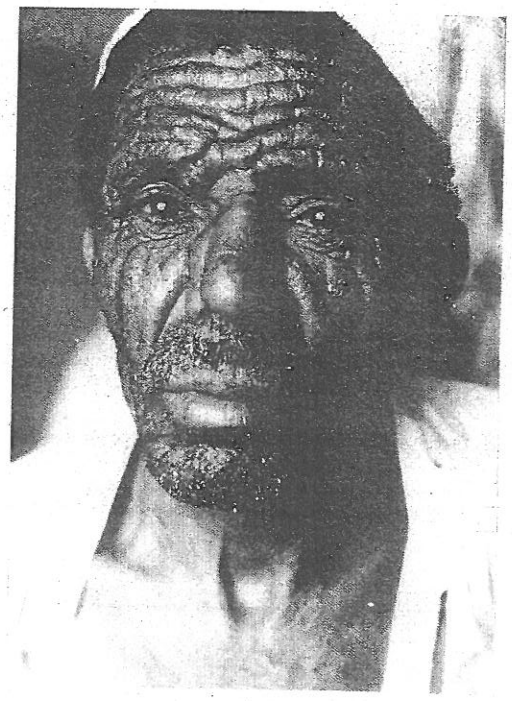

Fig. 1. Case 6 - Infiltrated lesions of AR all over the face.

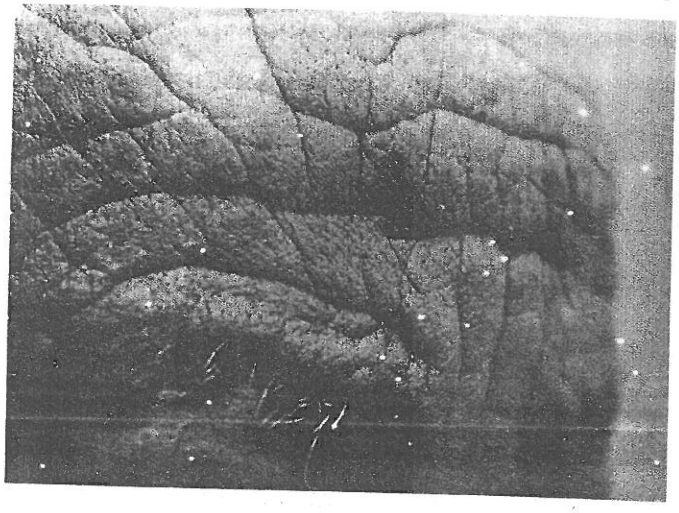

Fig. 2. Case 8 - Thick Infiltrated lesions on forehead.

Biopsies from thickened lesions revealed infiltration of round cells formed mainly of lymphocytes and macrophages along with a few eosinophils and plasma cells in the dermis. The infiltration was reaching upto the dermal papillae. The infiltrate was quite dense and mainly perivascular. Many of the lymphocytes showed convoluted nuclei. Epidermal changes revealed hyperkeratosis with patchy parakeratosis and acanthosis. In some sections minimal spongiosis was seen with exocytosis

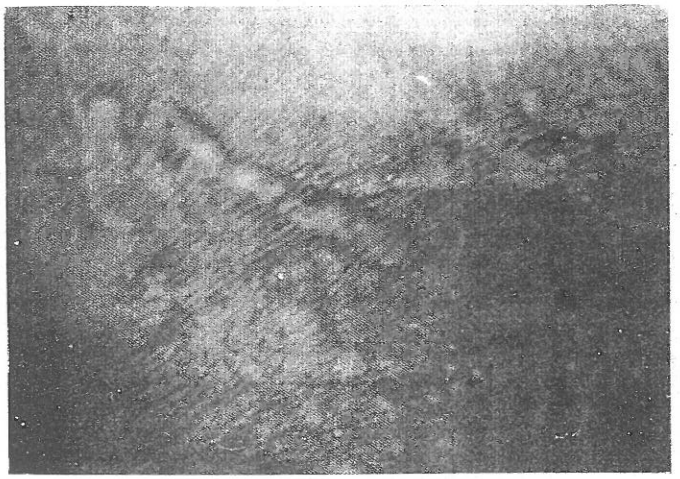

Fig. 3. Case 8 - Infiltrated papules and plaques on back of the neck.

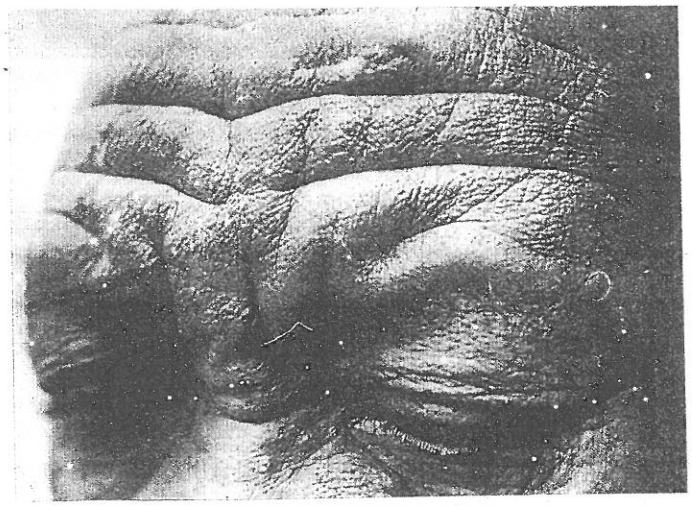

Fig. 4. Case 11 - Infiltrated lesions on forehead.

of lymphocytes. Pautrier's microabscesses were not seen in any of the sections. Biopsies from enlarged lymph nodes (patient No. 6 and 8) revealed histopathological changes consistent with dermopathic lymphadenopathy.

Patients were advised to avoid exposure to sun light and fluorescent tube lights as far as possible. All the patients were given local treatment in the form of zinc oxide cream in day time as sun screen and moderate steroid ointment during night. In addition 6 patients were given interrupted courses of tapering doses of oral prednisolone starting with 40 mg/day. 2 of the hypertensive patients were given chloroquine therapy starting with 150 mg twice daily, gradually tapering upto 150 mg alternate days. All the patients improved partially with local treatment alone a significantly well after oral prednisolone except patient No 8. Of the 2 patents on chloroquine therapy 1 responded well but the other did not show any improvement.

As patient No 8 had very thick lesions and did not show satisfactory response to prednisolone alone so he was suplemented with azathioprine 150 mg/day for 2 months without any additional benifit. Later this patient was given oral cyclosporine 4 mg/kg/day in addition to tapering does of prednisolone upto 15 mg/day. He showed significant improvement within 1 month but not complete remission. The dose of cyclosporine was increased to 5 mg/Kg/day and prednisolone was reduced to 10 mg/day. After 2 months we observed almost complete remission of his lesions. Prednisolone was discontinued at this time as he developed diabetes. After a month the dose of cyclosporine was reduced to 4.5 mg/Kg/day. He was well under control for 4 months but later he developed exacerbation and the dose of cyclosporine was again raised to 5 mg/kg/day. This time he did not improve so the treatment was again suplemented with prednisolone and cyclosporine was gradually withdrawn. While on cyclosporine he developed mycotic keratitis. No other adverse effect of cyclosporine was encountered.

Regular and up to date folllow-up was possible in 7 patients. They were admitted several times with exacerbations. They improved well during hospitalization but none of them showed complete remission of the lesions. In other 5 patients follow-up was possible from 5 months to 2 years only.

Comments

The clinical features in our patients were

almost similar to that of previously reported cases.[1,5,6] Toonstra et al[5] reported persistent erythroderma in 10 out of their 16 patients, we did not observe such a change in any of our patients.

Two of our patients were masons and 6 were farmers. All of them had onset of the lesions on the exposed parts during their active professional life although the lesions remained persistently progressive after retirement. Although we could not perform patch tests and photo- patch tests, we presume that some of these patients might have developed photoallergic dermatitis to start with and gradually progressed to AR. The role of different photoallergens in AR has been suspected by previous workers.[5-10]

Two of our patients had significant dermopathic lymphadenopathy. Similar changes are recorded in previously reported cases.[5] Few cases of AR developing lymphoma have been recorded in previous literature.[10] We could not reveal such a change in any of our patients.

Local and systemic steroid were partially effective in most of our patients as also noticed in previous studies.[5] Azathioprine therapy was ineffective in patients. Similar results were recorded by Norris et al,[11] but variable results are reported in previous studies.[5] Cyclosporine was initially very effective in patient No. 8 but later it did not work. Similar results were observed by Toonstra et al[5] in one of their patients while Norris et al[11] reported complete clearance of lesions with cyclosporine in their 2 patients.

Toonstra et al[5] have treated patients of AR with tolerance induction therapy with UVB alone or in combination with prednisolone, azathioprine, B-carotene, hydroxychlorquine and cyclosporine and observed good to excellent results in majority of their patients.

References

1. Ive FA, Magnus IA, Warin RP, et al. 'Actinic reticuloid;' a chronic dermatosis associated with severe photosensitivity and histological resemblance to lymphoma. Br J Dermatol 1969; 81 : 469-85.
2. Magnus IA. Actinic reticuloid In : Dermatological Photobiology. Oxford : Blackwell 1976 : 190-4.
3. Frain-Bell W. Photosensitivity dermatitis and actinic retuculoid syndrome. In : Cutaneous photobiology. Oxford : Oxford University Press 1985 : 106-24.
4. Toonstra J, Van Der Putte SCJ, Van Wichen DF, et al. Actinic reticuloid : immunohistochemical analysis of the cutaneous infiltrate in 13 patients. Br J Dermatol 1989; 120 : 779-86.
5. Toonstra J, Henquet CJM, Weelden H, et al. Actinic reticuloid : A clinical, photobiologic, histopathologic, and follow-up study of 16 patients. J Am Acad Dermatol 1989; 21 : 205-14.
6. Frain-Bell W, Lakshmipathi T, Rogers J, et al. The syndrome of chronic photosensitivity dermatitis and actinic reticuloid. Br J Dermatol 1974; 91 : 617-34.
7. Frain-Bell W, Hetherington A, Johnson BE. Contact allergic sensitivity to chrysanthemum and the photosensitivity dermatitis and actinic reticuloid syndrome. Br J Dermatol 1979; 101: 491-501.
8. Frain-Bell W, Johnson BE. Contact sensitivity to plants and the photosensitive dermatitis and actinic reticuloid syndrome. Br J Dermatol 1979; 101 : 503-12.
9. Horio T. Actinic reticuloid via persistant light reaction from photoallergic contact dermatitis. Arch Dermatol 1982; 118 : 339-42.
10. Ashinoff R, Buchness MR, Lim HW. Lymphoma in a black patient with actinic reticuloid treated with PUVA : Possible etiologic considerations. J Am Acad Dermatol 1989; 21 : 1134-7.
11. Norris PG, Camp RDR, Hawk JLM. Actinic reticuloid : Response to cyclosporine. J Am Acad Dermatol 1989; 21 : 307-9.