

## Acrosyringial Lichen planus

ANDERS ENHAMRE and BJÖRN LAGERHOLM

*Department of Dermatology, Karolinska sjukhuset, Stockholm, Sweden*

Enhamre A, Lagerholm B. Acrosyringial lichen planus. *Acta Derm Venereol (Stockh)* 1987; 67: 346-350.

Six patients with lichen planus or lichenoid toxicodermia are described. A dermal lymphocytic infiltrate in juxtaposition to the acrosyringium and liquefaction degeneration of the acrosyringial basal cell layer were prominent findings. Three patients had no medication. In two there was an association with  $\beta$ -adrenoceptor blocking drugs. The term acrosyringial lichen planus is proposed for this histological picture and some explanations for its pathogenesis are suggested. *Key words: Acrosyringium; Eccrine sweat duct; Lichenoid toxicodermia; Drug eruptions; Adrenergic beta receptor blockers.* (Received September 12, 1986.)

A. Enhamre, Department of Dermatology, Karolinska sjukhuset, S-10401 Stockholm, Sweden.

Although previous reports exist on localization of lichenoid histological changes around the acrosyringium (the intraepidermal eccrine sweat duct) in lichen planus (LP) (1, 2) this phenomenon has not attracted much attention in the literature.

We report here six cases of LP and lichenoid toxicodermia (LT) with a marked localization of histological changes to the acrosyringium.

### CASE REPORTS

*Case 1.* In August 1983, a 63-year-old woman developed red, itching and scaling maculae with a bluish hue on the extremities and later on the trunk. Due to hypertension she had been treated with alprenolol (Aptin<sup>®</sup>, Hässle) for several years until March 1983. In October she was prescribed atenolol 50 mg daily (Tenormin<sup>®</sup>, ICI) and a week later the lesions became more intense. The clinical diagnosis was LT. Prednisolone treatment was begun with 25 mg daily, an initial attempt to lower the dose resulted in exacerbation of lichenoid papules on the extremities and blue-red scaling maculae mainly on the abdomen and thighs. Atenolol was discontinued and the prednisolone dose could slowly be diminished over a three-month period. The rash had disappeared completely by February 1984.

Punch biopsies taken from the thighs after exacerbation in October showed a somewhat hyperplastic epidermis with orthokeratosis, normogranulosis, and numerous colloid bodies. A band-like subepidermal lymphocytic infiltrate with a moderate occurrence of eosinophilic granulocytes was seen and there was a marked accentuation around the acrosyringium. A multifocal liquefaction degeneration with acrosyringial accentuation was also seen (Fig. 1).

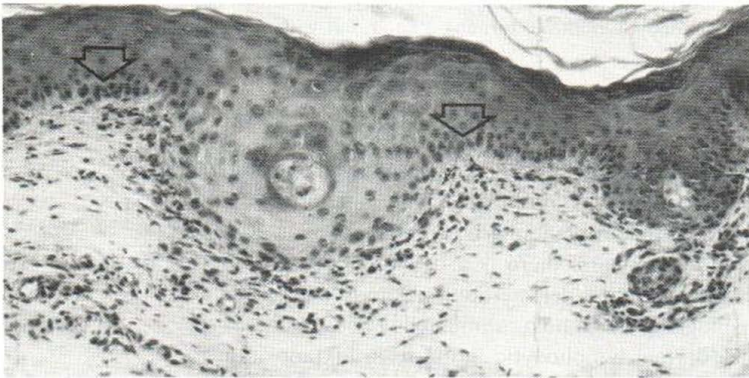
*Case 2.* In August 1983 a 51-year-old woman, who had previously had sarcoidosis and hepatitis, developed a violaceous rash on her trunk. Her medication was 800 mg labetalol (Trandate<sup>®</sup>, Glaxo), 2.5 mg bendroflumethiazide and 570 mg potassium chloride (KCl) (Salures-K<sup>®</sup>, Ferrosan), and 0.1 mg belladonna alkaloids, 0.3 mg ergotamine tartrate, and 20 mg phenobarbital (Bellergal<sup>®</sup>, Sandoz) daily. The clinical diagnosis was LT. She continued to take labetalol and the rash did not disappear until October 1984. In August 1986 she is still taking 800 mg labetalol daily and has no skin problems.

Histologic examination revealed a hyperplastic epidermis with acrosyringial accentuation, in these areas orthohyperkeratosis, hypergranulosis, colloid bodies, and liquefaction degeneration of the basal layer were seen. In juxtaposition to the acrosyringium there was a subepidermal lymphocytic infiltrate without occurrence of eosinophilic granulocytes.

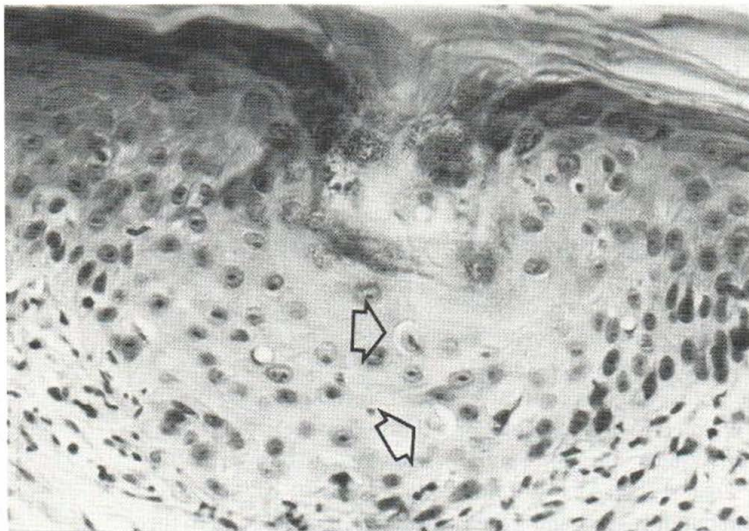
*Case 3.* An 80-year-old man with a stage III A IgA myeloma discovered in March 1983 developed an itching lichenoid rash on his trunk and extremities in July 1983. Treatment with interferon had been started in July. Due to nausea and fatigue the dose was later reduced to 10 MIE i.m. daily. He was also taking oral furosemide, KCl, theophylline, bromhexine, acetaminophen, chlormezanone, and nitrazepam. The clinical diagnosis was LT. The rash disappeared within 5 months without change of medication and only topical corticosteroids were given.



*Fig. 1.* Case 1. Liquefaction degeneration and a band-like mainly lymphocytic infiltrate around the acrosyringium. Note the intact basal cell layer in the surrounding epidermis (arrow). Hematoxylin-eosin,  $\times 350$ .



*Fig. 2.* Case 3. Two acrosyringeal structures surrounded by lichenoid changes. The left sweat duct is dilated. Note the distinct point at which the basal cell layer in the surrounding epidermis is intact and beneath which there is no band-like infiltrate (arrows). Hematoxylin-eosin,  $\times 350$ .



*Fig. 3.* Case 3. In addition to the acrosyringeal lichenoid changes marked hypergranulosis and colloid bodies (arrows) are seen. Hematoxylin-eosin,  $\times 700$ .

A biopsy taken from an arm in October showed orthokeratosis, hypergranulosis, and minor acrosyringeal edema. In the acrosyringeal areas there were colloid bodies and a subepidermal lymphocytic infiltrate. A moderate liquefaction degeneration was also seen near the acrosyringium (Figs. 2 and 3).

*Case 4.* A 57-year-old woman who had had similar skin eruptions in 1976 and 1979 again developed an itching rash on her extremities in 1983, consisting of violaceous shiny papules, sometimes appearing in groups. The clinical diagnosis was "characteristic" LP. There were no mucous membrane lesions. She had no medication. The lesions gradually disappeared within six months, but since then a few hypertrophic LP lesions have persisted on her lower legs, still existing in August 1986.

Histologic examination revealed hypergranulosis, orthohyperkeratosis, marked liquefaction degeneration of the basal cell layer, numerous colloid bodies, and a dermal lymphocytic infiltrate. The changes were general but accentuated around the acrosyringium and the acrotichium. There was no infundibular plugging. A moderate pigment incontinence was seen.

*Case 5.* A 63-year-old woman developed an itching violaceous rash on the front of her wrists, on her legs and later on the trunk. Polygonal papules with Wickham's striae were seen and the clinical diagnosis was LP. There were some white streaks on the buccal mucosa. She took no drugs. She healed spontaneously within 10 months.

A punch biopsy specimen from her left wrist showed an acrosyringeal edema with colloid bodies, orthohyperkeratosis, hypergranulosis, liquefaction degeneration, and a subepidermal lymphocytic infiltrate. These changes were localized to the acrosyringium exclusively and did not affect the epidermis or dermis between the sweat glands.

*Case 6.* In April 1984 a 55-year-old man developed a slowly spreading itching rash on his back and lower legs. Red or blue-red papules were seen and there were some buccal mucosa streaks. The clinical diagnosis was LP. He had no medication. The lesions cleared within 9 months.

A biopsy taken from the back in September showed hypergranulosis, orthohyperkeratosis, liquefaction degeneration and a subepidermal lymphocytic infiltrate forming a band, but with acrosyringeal accentuation.

## DISCUSSION

Lichen planus has a distinctive histologic picture, consisting of widespread liquefaction degeneration, colloid bodies, and in the dermis a confluent, mainly lymphocytic infiltrate (3). Some variations exist, among them lichen planopilaris, where follicular lesions predominate over typical LP. In lichen planopilaris there is a dense lymphocytic infiltrate along the length of the hair follicle, liquefaction degeneration at the follicular epidermal-dermal junction, and infundibular plugging by orthokeratotic cells (4).

All our cases have in common a dermal lymphocytic infiltrate in juxtaposition to the acrosyringium and liquefaction degeneration of the acrosyringeal basal cell layer (Figs. 1 and 2). In cases 1, 4, and 6 there were general changes with a marked acrosyringeal accentuation, in cases 2, 3, and 5 the lichenoid changes were localized exclusively to the acrosyringium (Fig. 2). The clinical diagnosis in our cases was LP or LT. We propose the term "acrosyringeal lichen planus" (ALP) for cases of LP or LT presenting this histological picture.

In case 4 there was an engagement also of the acrotichium. However, this patient differs from cases of lichen planopilaris in three ways; 1) the clinical picture was that of LP; 2) there was no infundibular plugging; 3) there was an acrosyringeal engagement hitherto not described in lichen planopilaris.

Our observations are in agreement with those of Fischer (1), who found that the subepidermal infiltrate was localized around the acrosyringeal sweat duct in early LP lesions. His findings have been confirmed by Steigleder & Gans (2). These reports (1, 2) suggest that LP actually starts around the acrosyringium, but later spread, forming the confluent picture usually attributed to the 'characteristic' histology of LP (3). However, the lesions examined in our cases were not initial (mean duration 3 months) and instead of interpreting our findings as early changes we suggest that ALP is a histologic variety of LP and LT.

Three patients had no medication but in cases 1 and 2 the rashes had some relation to the

$\beta$ -adrenoceptor blocking drugs ( $\beta$ -blockers) alprenolol, atenolol, and labetalol.  $\beta$ -Blockers are common drugs and the simultaneous administration in these cases might merely be a co-occurrence. However, in case 1 introduction of atenolol resulted in an exacerbation and the rash did not subside until this drug was discontinued. In case 2  $\beta$ -blocker administration was continued and the rash persisted for 14 months. However, in spite of continuous medication the LT eventually disappeared.

Lichenoid rashes due to  $\beta$ -blockers have been reported before (5). Earlier descriptions of the histopathology of the  $\beta$ -blocker induced LT are consistent with that of the previously described lichenoid drug eruptions (6), and do not include the localized acrosyringial changes observed in our cases.

When speculating upon the pathogenesis of ALP several explanations could be proposed; 1) the acrosyringium is a morphologic and functional entity that is distinct from the epidermis in general (7, 8). Furthermore, antigens reactive with human sera have been demonstrated in human sweat (9). The acrosyringium could possess antigenic properties different from the epidermis in general, perhaps with interindividual variations. Autoimmunity is one of the current theories for the etiology of LP (3); 2)  $\beta$ -blockers or other drugs could affect the acrosyringium directly by secretion through the sweat duct. It has been shown that drugs may be excreted in sweat (10); 3)  $\beta$ -blockers could influence the physiology of the sweat duct to produce the acrosyringial changes. The eccrine sweat gland has both cholinergic and adrenergic innervation (11). Both  $\beta$ -adrenergic and  $\alpha$ -adrenergic agonists stimulate sweat secretion, and the  $\beta$ -blocker propranolol drastically inhibits the  $\beta$ -agonist-induced secretory response (12).

However, most of our patients had no  $\beta$ -blocker medication and we therefore suggest that ALP is a histologic variety seen in some cases of LP and LT, and that this, in the latter case, could possibly be caused by  $\beta$ -blockers. We hope future research will show if and how  $\beta$ -blockers are involved in this phenomenon.

#### ACKNOWLEDGEMENT

We thank Dr Casper Tarras-Wahlberg at the Department of Dermatology, Nässjö Hospital, for providing us with case 2.

#### REFERENCES

1. Fischer H. Zur Frage der Bedeutung abnormer Hautdrüsensekrete für das pathologische Geschehen auf der Haut. *Zentralbl Hautkr* 1927; 398-399.
2. Steigleder GK, Gans O. Pathologische Reaktionen in der Epidermis. In: Steigleder GK, Gans O, eds. *Handbuch der Haut- und Geschlechtskrankheiten. Normale und pathologische Anatomie der Haut II*. Berlin: Springer 1964: 274.
3. Samman PD. Lichen planus and lichenoid eruptions. In: Rook A, Wilkinson DS, Ebling FJG eds. *Textbook of dermatology*. Oxford: Blackwell Scientific Publications. 1979: 1483-1502.
4. Ackerman AB. *Histologic diagnosis of inflammatory skin diseases*. Philadelphia: Lea & Febiger, 1978: 651-652.
5. Felix RH, Ive FA, Dahl MGC. Cutaneous and ocular reactions to practolol. *Br Med J* 1974; iv: 321-324.
6. Wilson DJ. Eczematous and pigmentary lichenoid dermatitis. *Arch Dermatol Syph* 1946; 54: 377-396.
7. Pinkus H, Rogin JR, Goldman P. Eccrine poroma. Tumors exhibiting features of the epidermal sweat duct unit. *Arch Dermatol* 1956; 74: 511-521.
8. Christophers E, Plewig G. Formation of the acrosyringium. *Arch Dermatol* 1973; 107: 378-382.
9. Wilkinson RD, Elie JD, Miot MJ, Wakkary JA. Autoimmunity to human sweat. I. Demonstration of antigens in sweat reactive with autologous and homologous sera. *J Invest Dermatol* 1971; 57: 401-406.

10. Comaish JS, Shelley WB. Fluorometric determination of quinine and fluorescein excretion in human sweat. *J Invest Dermatol* 1965; 44: 279-281.
11. Uno H. Sympathetic innervation of the sweat glands and piloerector muscles of macaques and human beings. *J Invest Dermatol* 1977; 69: 112-120.
12. Sato K. The physiology and pharmacology of the eccrine sweat gland. In: Goldsmith LA, ed. *Biochemistry and physiology of the skin*. New York: Oxford University Press, 1983: 596-641.