

Lichenoid Dermatitis-Treatment With Pulsed Dye Laser: A Case Study

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Background and Objectives: Both the diagnosis and the treatment of lichenoid dermatosis are often difficult and can be time-consuming. There are now more and more publications about the use of laser systems—especially the flashlamp-pumped pulsed dye laser—in the treatment of inflammatory dermatoses, although the laser's exact mechanism of action in these cases is not yet clear.

Study Design/Patients and Methods: We report on a female patient with lichenoid dermatitis that was presumably drug-induced (roxatidine, H₂ receptor antagonists). After a 10-month treatment with local corticosteroids, without significant clearance the drug was discontinued and the pulsed dye laser was used (wavelength 585 nm, pulse duration 450 microseconds).

Results: Six laser treatments resulted in complete clearance of the lesions. No recurrence occurred during the follow-up period of 54 months. Scars were not observed. A post-operative biopsy showed no evidence of lichenoid dermatitis.

Conclusions: The pulsed dye laser seems to accelerate the clearance of presumably drug induced corticosteroid-resistant lichenoid dermatoses. No permanent pigmental changes or scarring were observed. *Lasers Surg. Med.* 31:23–26, 2002. © 2002 Wiley-Liss, Inc.

Key words: lichenoid dermatitis; laser; dye laser

INTRODUCTION

Lichenoid tissue reactions occur in a number of inflammatory skin diseases, including pityriasis lichenoides et varioliformis, lupus erythematosus, chronic graft-versus-host disease, and lichenoid drug-related exanthem [1–4]. Lichen planus is considered to be the classic example of papulous, lichenoid-inflammatory dermatitis of unknown etiology [5].

We report about a female patient with lichenoid dermatitis of presumably drug-induced (roxatidine, H₂-receptor antagonist) on her back. After discontinuation of the drug and six-treatment sessions with the pulsed dye laser, the efflorescences had resolved completely. No recurrence was observed in the follow-up period of 54 months. A definitive histological, clinical, and etiological classification of the clinical picture was not possible. In spite of this, however, the potential etiology and the differential diagnosis of the skin changes are to be discussed here, especially given the good response to laser therapy.

CASE REPORT

Anamnesis

The female patient, who is now 72-year-old, first presented in 8/1994. Since 5/1994, there had been erythematous, pruritic skin changes on her back which had continuously increased in size and had barely responded to 10 months of local treatment with 0.1% triamcinolone acetate ointment (8/94 to 6/95). The anamnesis included a pulmonary embolism with deep venous thrombosis (1991). Due to reflux esophagitis, systemic medication with Roxit[®] (= roxatidine acetate, H₂ blocker) was administered from 4/94 to 5/95. After 1 month of roxatidine treatment, the exanthem occurred. Concomitant treatment with Talcid[®] (= hydrotalcid, antacid) began in 4/94 and continues to date.

Dermatological Findings

Between the scapulae on the back there were many 7–10 cm polycyclic, erythematous-livid skin changes; some were well-defined, and some had irregular margins. In parts of these areas, the skin appeared to be atrophied and/or lichenified (Fig. 2a). There was no involvement of oral and genital mucous membranes or the remaining integument.

Laboratory Findings

- 8/1994: Normal laboratory findings except for low leukocyte levels (3,500/nl)
- 4/1995: Positive ANA screening (titer 1:320, homogenous pattern), other autoantibodies negative. Other laboratory findings normal
- 4/2000: ANA titer 1:80 (nucleolar), other autoantibodies negative
- 12/2000: Scratch test: roxatidine, hydrotalcid, and omeprazole all negative

Histological Findings

Histological examination, 8/1994 (Fig. 1a,b), 4/1995, 5/1995. Epidermis with hypergranulosis and

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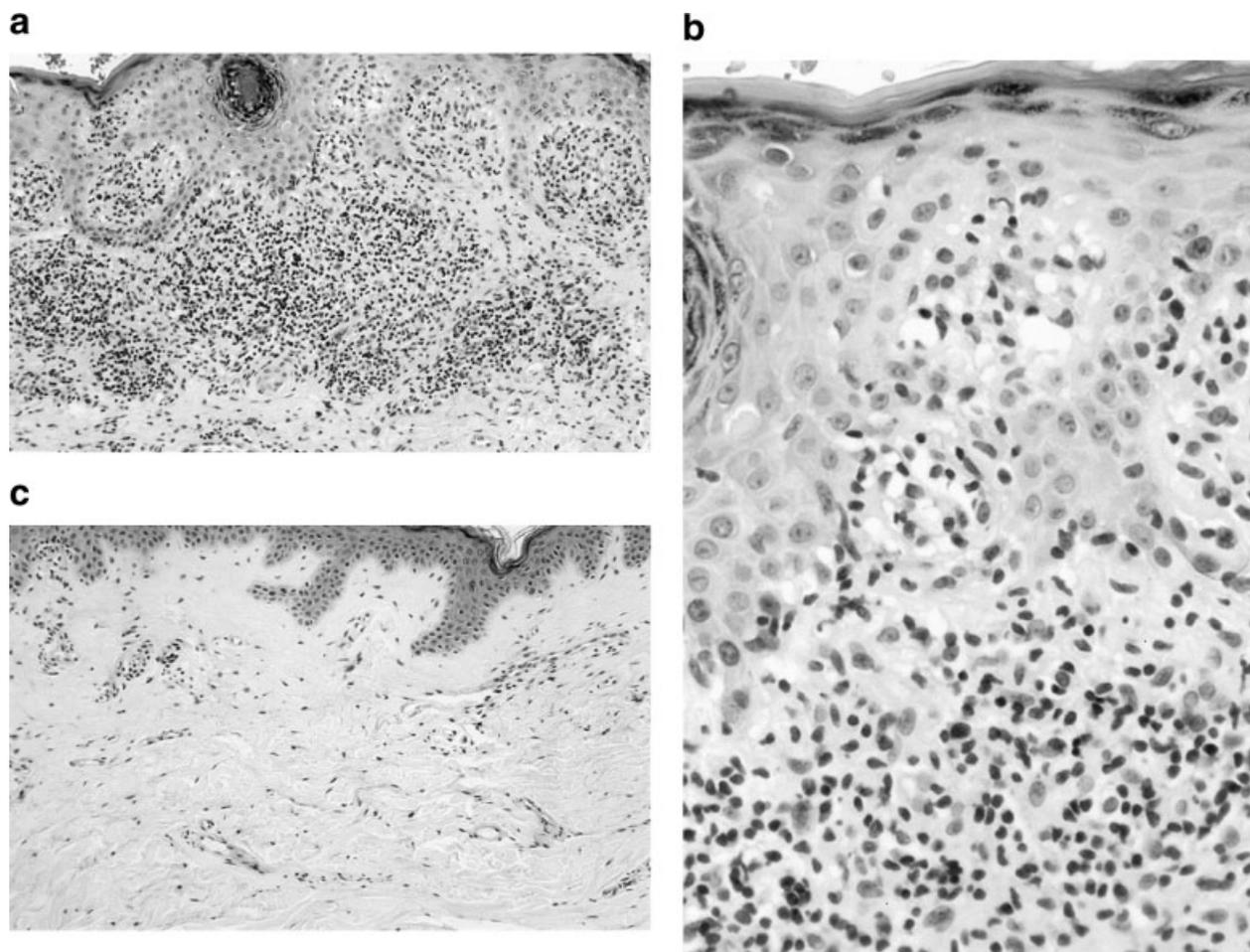


Fig. 1. **a**: Overview: histological image before laser treatment: largely superficial, clearly lichenoid dermatitis with hypergranulosis and compact orthohyperkeratosis (dorsum). H & E, 20. **b**: Magnified image. Individual cytotoid bodies in the vacuolated basal cell membrane and subepidermal. H&E, 40. **c**: Histological image after laser treatment: slight fibrosis with very discrete foci, only slight evidence of superficial lichenoid dermatitis.

compact orthokeratosis. Band-like round-cell infiltration in the stratum papillare. Junctional colloid bodies. Evidence of the perifollicular infiltrates extending into mid-lower dermis, in some cases surrounding the sweat glands and blood vessels. Focal follicle-aggressive pattern (Fig. 1c).

Immunohistological examination, 5/1995. No immunoglobulin deposits around the epidermal basal membrane zone, i.e., no evidence of discoid or systemic lupus erythematosus.

Final assessment. Lichenoid, partly granulomatous dermatitis; no conclusive evidence of lupus erythematosus.

Follow-up biopsy, 2/2000—44-month after the final laser treatment. No conclusive findings; nearly complete resolution of the inflammatory lichenoid components (Fig. 1c).

Therapy and Course of Disease

Treatment with omeprazole instead of roxatidine was begun in May 1995 because a 10-month local treatment

(8/94 to 6/95) with corticosteroids had only slightly improved the condition of the skin, and an etiological interaction with Roxit could not be ruled out. Talcid was continued. At nearly the same time, a treatment with a flashlamp-pumped pulsed dye laser at a wavelength of 585 nm and a pulse length of 0.3–0.45 milliseconds (PhotoGenica V, Cynosure, Inc.) was begun. The fluence used in the six treatment sessions (6/95–4/96) was between 5.5 and 6.0 J/cm² at an impulse spot size of 7 mm. Local anesthetic measures were not necessary.

Within 6 weeks after each treatment session successive lightening occurred; by 6/1996 the inflammatory skin changes had cleared completely. No scarring occurred.

A follow-up examination in 2/2000 (Fig. 2c) and most recently in 12/2000 revealed no evidence of recurrence. Individual hypopigmentations occurred in the otherwise healthy skin. The follow-up biopsy on 2/2000, showed no further evidence of lichenoid dermatitis.

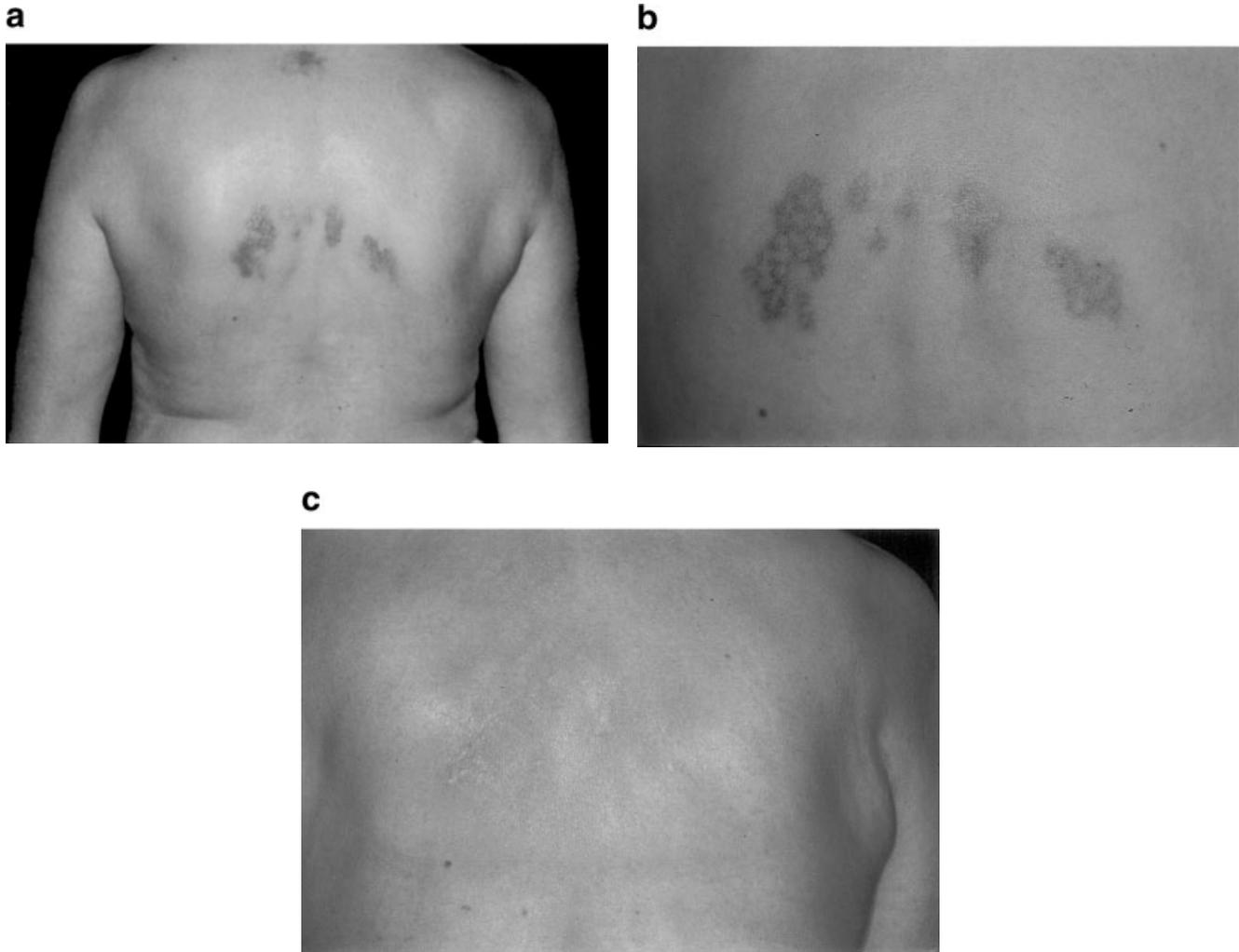


Fig. 2. **a:** Erythematous-livid plaques on the back of a 72-year-old female patient (02/1995). **b:** Status 2 months after the first laser treatment (09/1995). Marked skin changes visible exclusively in the areas treated with the laser. **c:** Status 44 months after the final dye laser treatment (02/2000).

DISCUSSION

In this article, we report on a female patient with localized inflammatory skin changes of presumably drug-induced etiology which were histologically determined to be lichenoid dermatitis.

Lichenoid reactions have light-microscopic traits that resemble lichen planus, in spite of etiological differences between the two [6]. In addition to hypergranulosis of the epidermis, junctionally located colloid bodies (hyaline bodies, Civatte bodies) can be identified, as can basal cell vacuolation and band-like round-cell infiltrate in the upper dermis [5].

Distinguishing between lupus erythematosus and lichenoid dermatitis is often difficult in terms of differential diagnostics. Colloid bodies occur often in both clinical pictures but are more frequent and deeper in lichen planus, occasionally even reaching the stratum reticulare [5]. In

the direct immunofluorescence of lupus erythematosus, immunoglobulin, and complement deposits are present as a granular, linear band along the dermoepidermal junction, whereas this sort of immunodeposit in lichen planus is usually restricted to colloid bodies [5]. The clinical, histological, and immunohistological assessment of our patient yielded no evidence of lupus erythematosus. An increase in ANA titer is not pathognomonic in and of itself; increased ANA titers can also occur in many other diseases. Schrallhammer-Benkler et al. [7] were among those who reported about the induction of antinuclear antibodies via acute mercury poisoning in a lichenoid skin reaction.

In addition to the association of lichenoid skin changes with diseases of the gastrointestinal tract (liver disease, colitis ulcerosa) and malignancy [5], it is also relevant that lichenoid dermatitis can be triggered by medication [1–4].

In our example, the first lichenoid foci developed shortly after treatment with the H₂-antagonist roxatidine and an

antacid began. In a current case report, Bong et al. [2] discuss recurrent dermal reactions of this kind on the torso and arms of an 81-year-old female patient with esophagitis after administration of three different proton pump inhibitors (omeprazole, lansoprazole, pantoprazole). On the other hand, Ohtsuka et al. [8] report the successful treatment of a corticoid-resistant pruritic lichenoid dermatitis using omeprazole in a patient with ulcer ventriculi and a concomitant *Helicobacter pylori* infection.

In our patient's case, the gastric medication was changed to omeprazole after a year of unsuccessful local treatment of the skin changes; about 1-month later, laser treatment began. In spite of the fact that the drug was discontinued at nearly the same time that laser treatment began, Figure 2b (2 months after the first treatment) supports the conclusion that dye laser treatment aided a more expedient clearance of the lesion. The discontinuation of the H₂ blocker is certainly responsible for the long recurrence-free period as well. By contrast, a study by Halevy and Shai [3] indicates that even when a (potentially) causative medication is discontinued, a resolution of lichenoid skin changes cannot always be expected until months or even years have passed.

The mechanism of action of the pulsed dye laser in treating inflammatory skin diseases (e.g., psoriasis lichen sclerosus et atrophicus, lupus erythematosus, granuloma faciale) is still unknown [9–13]. We are currently conducting histological, immunohistological, and immunochemical examinations as part of dye laser therapy of lupus erythematosus; this may help clarify the issue.

In conclusion, here the pulsed dye laser seems to have accelerated the clearance of presumably drug-induced

corticosteroid-resistant lichenoid dermatoses without causing adverse side effects.

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