CLINICAL APPLICATION OF GaAlAs 830 nm DIODE LASER FOR ATOPIC DERMATITIS

Hideki Morita, Junro Kohno, Sachiyo Tanaka, Yukio Kitano and Seichiro Sagami

Department of Dermatology, Hyogo College of Medicine, Hyogo, 663 Japan

Patients with atopic dermatitis (AD) were treated with diode low reactive level laser therapy (LLLT), and the following results were obtained. (1) Itchy sensation decreased in 63 of 81 cases (79%) after this therapy. (2) Skin eruption improved in 57 of 81 cases (71%). (3) There were no side-effects during and after LLLT. (4) Major histocompatibility complex (MHC) class II antigen and inter-cellular adhesion molecule (ICAM)-1 expression on epidermal cells decreased after the therapy. (5) The number of CD1 positive epidermal dendritic cells did not significantly change before and after LLLT.

KEY WORDS GaAlAs diode laser therapy Atopic dermatitis

Introduction

Atopic dermatitis (AD) is one of the common chronic skin diseases in which a variety of immunological disturbances have been described. Type 1 allergy plays an important role in the pathological mechanism of AD.[1] Alternatively, cell mediated immune responses have been considered to be involved in pathological development in the skin of AD.[2] New treatments, for example cyclosporin A,[3] gamma interferon[4] and interleukin 2[5] were recently suggested for the management of the disease. They all act on some component of the immune mechanism which provoke the eczematous reactions. But because of side-effects we cannot use these kinds of therapy as our first choice. Steroid ointment is still widely used for the treatment of AD. Steroid rosacea sometimes appears during the prolonged application of steroids. In the present study, we used a CaAlAs 830 nm diode laser for treatment of patients with atopic dermatitis. Immunohistological examination was also performed before and after LLLT.

Patients and Methods

Patients

A diode laser system (Luketron) was used for treatment of AD patients who visited our clinic. The system emits a 60 mW continuous wave beam at a wavelength of 830 nm. From November 1991 to December 1992, 81 AD patients with the mean age of 19 years (6 to 45) were treated. We treated the patients with the Luketron for 120 s per 100 cm^2 of skin lesion once a week. Use of internal or external medicines were continued without change before and after LLLT.

Evaluation of Skin Symptoms Before and After LLLT

The evaluation of skin symptoms, before and after LLLT, was conducted according to the evaluation list compiled by our allergy clinic (Table 1). The symptoms included were dry skin, follicular keratosis, pityriasic scale, excoriation marks, erythema, papules, and lichenification. Severe symptoms were graded as 3 points, moderated symptoms as 2 points, mild symptoms as 1 point, and no symptoms as 0 points. Evaluation of the itchy sensation were graded as follows: strongest itching with impossibility to sleep, 3 points; strong itching but able to sleep, 2 points; slightly itching as 1 point; and no symptom as 0 points.

Immunohistological Examination of MHC Class II, ICAM-1 and CD 1

Skin biopsies were performed before and five times after LLLT. Three AD patients agreed to cooperate in the present study. In order to avoid the influence of MHC class II, ICAM-1 and CD1 expression, no internal and external medicines except LLLT were used for the treatment of these three AD patients. Immunohistochemical stainings were performed on 6-μ m cryostat sections with monoclonal antibodies. Anti-MHC class II (HLA-DR) and anit-CD1 monoclonal antibodies were purchased from Becton-Dickinson. Anti-ICAM-1 monoclonal antibody was purchased from Immunotech. Reactivity was visualized using a standard biotin-avidin immunoperoxidase technique from a commercially available kit (Becton-Dickinson).

0898-5901/93/020075-04$07.00

Accepted 6 May 1993

Results

Skin Symptoms Before and After LLLT

The clinical score values of skin symptoms before and after LLLT were calculated from the evaluation table used in our allergy clinic (Table 1). In 57 cases out of 81 (71%), the skin symptom scores decreased more than 5 points after LLLT. In 63 cases out of 81 (79%), the itchy sensation scores decreased more than 1 point after LLLT.

HLA-DR, ICAM-1 and CD1 Expression on Epidermal Cells Before and After LLLT

Before LLLT, HLA-DR positive dendritic cells were detected in the epidermis. The majority of keratinocytes were negative for HLA-DR staining. Almost all dermal infiltrating cells were HLA-DR positive (Figure 1). After LLLT, HLA-DR positive dendritic cells decreased remarkably in number compared with before LLLT. HLA-DR positive dermal infiltrating cells also decreased after LLLT (Figure 2). ICAM-1 expression was detected focally on keratinocytes before LLLT. The majority of dermal infiltrating cells expressed ICAM-1 (Figure 3). ICAM-1 expression on keratinocytes and dermal infiltrating cells almost disappeared after LLLT (Figure 4). The numbers of CD1 positive dendritic epidermal cells showed no difference before (Figure 5) and after (Figure 6) the therapy.

Discussion

Diode lasers were at first restricted to gallium arsenide (GaAs) systems, which usually produced a 904 nm beam. GaAs systems were typically difficult to run for long periods because of the propensity of the chip to overheat. Japan Medical Laser Laboratory (JMLL), together with Matsushita Electrical Company worked on developing a new gallium aluminium arsenide (GAAlAs) system for medical application, producing an 830 nm beam with 15 mW. The GaAlAs chip could run in continuous wave without overheating.[6] This therapy is generally referred to as low reactive-level laser therapy (LLLT). The reports of LLLT for clinical application have been increasing.[7, 8] In the present study we used LLLT on the patients with AD.

Table 1. Evaluation table used in our clinic

Estimate of dermatitis (clinical score)	
Dry skin	3 – 0
Follicular keratosis	3 – 0
Pityriasic scale	3 – 0
Excoriation	3 – 0
Erythema	3 – 0
Papule	3 – 0
Lichenification	3 – 0
Total	21 – 0

AD is a chronic inflammatory skin disease. The onset of AD usually occurs during infancy or early childhood. Infantile AD is typically characterized by scaly erythematous lesions on the face and the scalp. In more severe cases, generalized skin involvement with weeping and impetiginous lesions are often seen. Childhood AD is characterized by scaly erythematous lesions on the flexor aspects of the extremities as well as the face and neck. Adult AD especially involved the face and the hands with lichenified lesions. At all phases of illness, AD patients suffer from marked pruritus that is exacerbated by multiple triggers including allergens, reduced humidity, excessive sweating, and irritants such as wools, acrylics, soaps, or detergents. Recent studies suggest that allergen-triggered IgE-mediated mechanisms and delayed hypersensitivity reaction may contribute to the pathogenesis of AD.[9, 10] The cardinal feature of AD is pruritus. Pruritus may be related to mediator release from ongoing inflammation or allergen exposure. Thus, the control of pruritus is important for the treatment of AD. Topical corticosteroid is commonly used for the treatment on the inflammatory skin lesions. Corticosteroids should not be used for prolonged periods especially on the face, because of the side-effects. A second approach in the treatment of AD is the use of agents that counteract mediators released by inflammatory cells, such as antihistamines, platelet-activating factor antagonists and leukotriene antagonists. These are routinely prescribed for the treatment of pruritus but their efficacy is limited. Recently, PUVA therapy[11] has been tried to treat patients with chronic severe AD and who have failed to respond to ordinary treatments. The mechanism of action of PUVA therapy is thought to be anti-inflammatory by reducing the function of antigen-presenting cells. However, there is a risk of development of skin cancer. Therefore, the dosage and duration of PUVA therapy should be kept to a minimum. The mechanism of action of LLLT seems similar to the PUVA therapy, because the expression of MHC class II antigen and I CAM-1 on the epidermal cells decreased after LLLT. The number of CD1 positive cells did not change before and after LLLT. From these findings we consider that the effect of the mechanism of LLLT is anti-inflamatory, without any tissue damage. There is no report about the development of skin cancer after LLLT. We therefore consider that LLLT is safer than PUVA therapy. The patients with AD manifest abnormalities in immune regulation. Based on these findings, immunomodulator (interleukin-

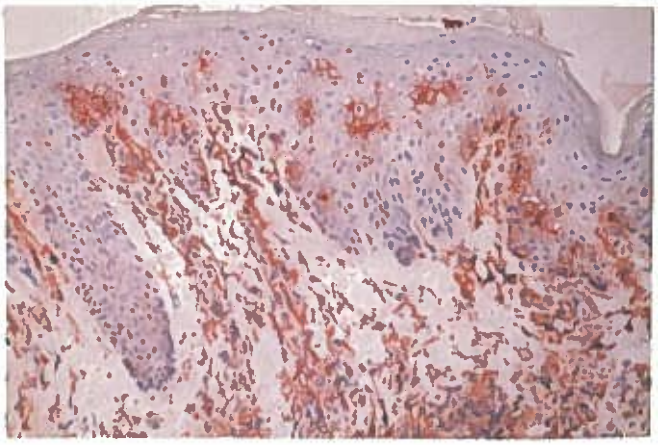

Figure 1. Before LLLT, HLA-DR positive dendritic cells were detected in the epidermis (immunoperoxidase stain, × 200)

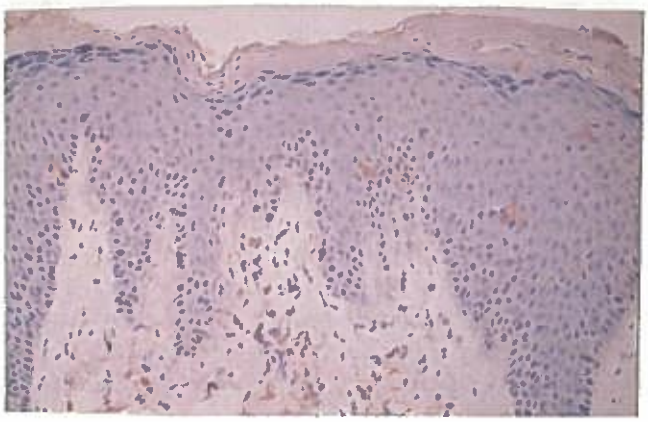

Figure 2. After LLLT, HLA-DR positive dendritic cells in the epidermis decreased remarkably in number compared with before LLLT (immunoperoxidase stain, × 200)

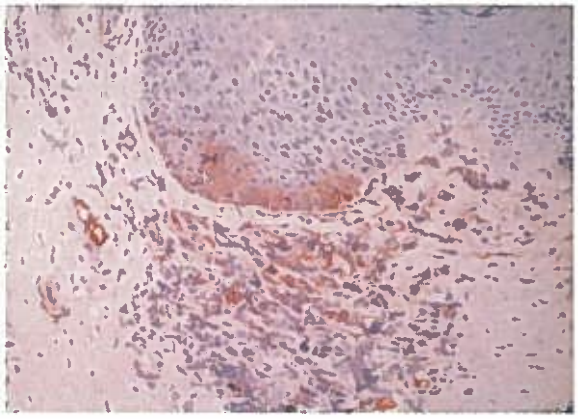

Figure 3. ICAM-1 expression was detected focally on keratinocytes before LLLT (immunoperoxidase stain, × 200)

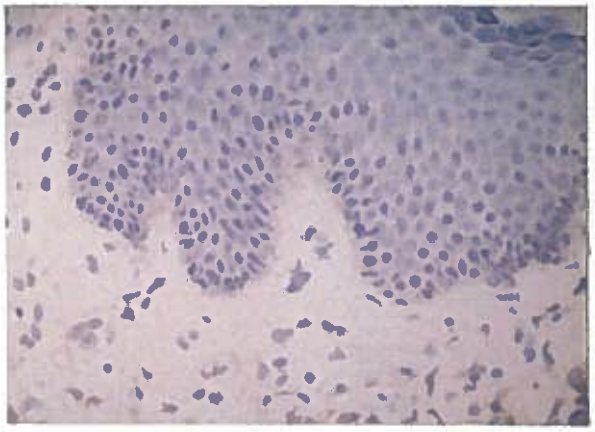

Figure 4. ICAM-1 expression on keratinocytes almost disappeared after LLLT (immunoperoxidase stain, × 200)

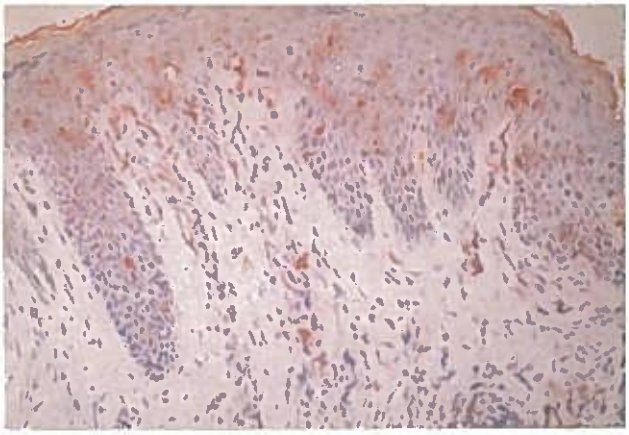

Figure 5. CD1 positive dendritic epidermal cells before LLLT (immunoperoxidase stain, × 200)

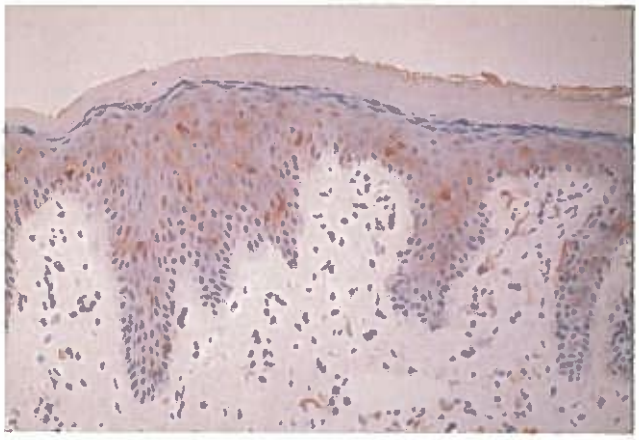

Figure 6. CD1 positive dendritic epidermal cells after LLLT. The numbers of CD1 positive dendritic epidermal cells showed no difference between before and after LLLT (immunoperoxidase stain, × 200)

2, interferon-gamma) therapy have recently been reported.[4] As the therapeutic effect is transient and immunomodulator therapy is rather toxic to the pateients (chills, malaise, hepatomegaly, edema, pleural effusion), the potential harm of immunomodulator therapy should be seriously considered in a non-fatal disease such as AD. In the present study, we treated 81 AD patients with LLLT. There was no side-effect during and after LLLT. The treatment was effective for the itchy sensation in 79% of the cases. Skin eruptions improved in 71% of the cases. Based on this evidence we consider that LLLT may become the new therapy of choice for the treatment of AD.

References

1. Gurevitch, A.W., Heiner, D.C., Reisner, R.M., *et al.* (1973). IgE in atopic dermatitis and other common dermatoses. *Archives of Dermatology* **107**, 712–715.
2. Uehara, M. and Ofuji, S. (1976). Patch test reaction to human dander in atopic dermatitis. *Archives of Dermatology* **112**, 951–954.
3. Van Joost, T., Stolz, E. and Mucle, F. (1987). Efficacy of low dose cyclosporin in severe atopic disease. *Archives of Dermatology* **123**, 166–167.
4. Boguniewicz, M., Jaffe, H.S., Izv, A., *et al.* (1990). Recombinant gamma interferon in treatment of patients with atopic dermatitis and elevated IgE levels. *American Journal of Medicine* **88**, 365–370.
5. Kue-Hsiung, H., Chen-Cheng, C. and Hiu-Feng, H. (1991). Interleukin 2 therapy in severe atopic dermatitis. *Journal of Clinical Immunology* **11**, 22–28.
6. Ohshiro, T. (1991). *Low Reactive-Level Laser Therapy: Practical Application*. John Wiley & Sons Ltd., p4.
7. Asada, K., Yutani, Y. and Shimazu, A. (1989). Diode laser therapy for rheumatoid arthritis: A clinical evaluation of 102 joints treated with low reactive-level laser therapy (LLLT). *Laser Therapy* **1**(3), 147–151.
8. Obara, J., Yanase, M., Motomura, A., *et al.* (1987). The pain relief of low energy laser irradiaiton on rheumatoid arthritis. *Pain Clinic* **8**(1), 18–22.
9. Sampson, H.A., (1988). The role of food allergy and mediator release in atopic dermatitis. *Journal of Allergy and Clinical Immunology* **81**, 635–645.
10. Mitchell, E.B., Crow, J., Williams, G., *et al.* (1986). Increase in skin mast cells following chronic house dust mite exposure *British Journal of Dermatology* **114**, 65–73.
11. Atherton, D.J., Carabott, F., Glover, M.T., *et al.* (1988). The role of psoralen chemotherapy (PUVA) in the treatment of severe atopic eczema in adolescence. *British Journal of Dermatology* **118**, 791–795.