

Treatment of Solar Lentigo with Cryosurgery

Sir,

Melanocytes are the skin cells most vulnerable to cold; they can be destroyed by temperatures of -4 to -7°C (1). Lower tissue temperatures or repeated freeze–thaw applications lead to skin necrosis (2). Selective destruction of melanocytic lesions by skin freezing can therefore be achieved without epithelial loss or skin necrosis if certain technical requirements are taken into account. In a randomized study of 20 patients, small benign pigmented lesions (lentigenes) were shown to respond efficiently to a regimen of 5 s vs. 10 s contact cryosurgery at -42°C skin surface temperature with good to excellent cosmetic results (3). Moreover, Stern et al. have documented significant superiority of cryotherapy over CO_2 and argon laser treatments in a randomized study with 99 lentigenes in 13 patients (4).

We report here on the successful treatment of 6 patients with large solar lentigo lesions using nitrous oxide contact cryosurgery. To our knowledge, the application of cryosurgery has not previously been evaluated for this indication.

MATERIALS AND METHODS

In a prospective open study of case series, cryosurgery was performed in 6 German patients (1 male, 5 females, aged 64.7 ± 19.1 years) with solitary large solar lentigo lesions ($7.7 \pm 5.7 \text{ cm}^2$, range 3–16 cm^2) and skin type I or II. Diagnosis was confirmed histologically in all patients prior to treatment. Lesions were localized on the cheeks in 4 patients and on the temples in 2 and had been present for 3–20 years. Two patients had had treatment with argon laser and azelaic acid, with unsatisfactory results.

Cryosurgical treatment was performed by the contact technique using a single freeze–thaw cycle of 30–40 s with nitrous oxide as refrigerant (skin surface temperature -86°C). The lesions were treated once; in 2 patients with large lesions (14 and 16 cm^2) treatment was fragmented and subsequently performed in 2 sessions with an interval of 3–4 months.

RESULTS

In all patients treated, full remission of the lesions was achieved with excellent cosmetic results (Fig. 1). Complete depigmentation of the lesions without cosmetically significant colour impairment or scar formation was seen. No recurrence was observed within a 10-month follow-up period after the last treatment.

DISCUSSION

Solar lentigo presents clinically as a solitary light- to dark-brown pigmented macular lesion that can develop at any age on the light-exposed areas, especially on the face. Histological examination reveals a benign increase in melanocytes along the dermo-epidermal junction zone and an elevated melanin content in the melanocytes and basal keratinocytes.

Large, facial solar lentigo lesions can cause severe cosmetic impairment. Treatment with ruby laser and topical tretinoin have been used with varying success. Raulin et al. reported on 2 patients in whom ruby laser treatment led to complete healing of the lesions with no appreciable side effects (5). Topical retinoid application improves solar lentigo only after



Fig. 1. Solar lentigo (4.5 cm^2) over the left eyebrow of a 46-year-old woman (a) before and (b) after complete remission with excellent cosmetic result 5 months after cryosurgery (contact technique, single freeze–thaw cycle with nitrous oxide, 30 s).

a long treatment period (occasionally ranging up to 10 months) (6). The therapeutic and cosmetic outcomes reported here confirm the initial hypothesis of melanocyte vulnerability to low temperatures and corroborates contact cryosurgery with nitrous oxide as a successful therapeutic regimen for large solar lentigo on light-coloured skin.

Since re-pigmentation of human skin after cryosurgical treatment through melanocyte immigration from adjacent skin areas is obvious in 3–4 months (1), a follow-up of 8–12 months can exclude recurrences in solar lentigo. In contrast, in lentigo maligna, the success rates of 90–100% reported do not justify use of cryosurgery because recurrences occur in 10–40% of patients (7). Moreover, in 1 case a cryosurgically managed lentigo maligna has been reported to develop into a lentigo maligna melanoma (8).

REFERENCES

- Gage AA, Meenaghan MA, Natiella JR, Green GW Jr. Sensitivity of pigmented mucosa and skin to freezing injury. *Cryobiology* 1979; 16: 348–361.
- Zouboulis ChC. Principles of cutaneous cryosurgery: an update. *Dermatology* 1999; 198: 111–117.
- Almond-Roesler B, Zouboulis ChC. Milde Kryochirurgie zur Behandlung aktinischer Lentigines. *Allergologie* 1999; 21: 420–421.
- Stern RS, Dover JS, Levin JA, Arndt KA. Laser therapy versus cryotherapy of lentigines: a comparative trial. *J Am Acad Dermatol* 1994; 30: 985–987.
- Raulin C, Petzold D, Hellwig S. Lentigo benigna. Entfernung durch den gütegeschalteten Rubinlaser. *Hautarzt* 1996; 47: 44–46.
- Rafal ES, Griffiths CEM, Ditre CM, Finkel LJ, Hamilton TA, Ellis CN, et al. Topical tretinoin (retinoic acid) treatment for liver spots associated with photodamage. *N Engl J Med* 1992; 326: 368–374.
- Collins P, Rogers S, Goggin M, Manning W. Cryotherapy for lentigo maligna. *Clin Exp Dermatol* 1991; 16: 433–435.
- Green T, Ball RY, Pye RJ. Malignant melanoma developing after cryotherapy for lentigo maligna. *Br J Dermatol* 1991; Suppl 125: 18.

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UVA1 for Treatment of Keloids

Sir,

The various therapies for keloids usually have only limited effect. Encouraged by the good results of UVA1 (340–400 nm) irradiation on localized scleroderma (1, 2) we tried high-dose UVA1 phototherapy on keloid scars. UVA1 irradiation has been shown to stimulate collagenase production by human fibroblasts *in vitro* (3).

PATIENTS AND METHODS

A 21-year-old Caucasian woman, a 21-year-old Asian man and a 40-year-old Caucasian man participated in the study. They presented with a several year history of a stable keloid secondary to tuberculin vaccine in the first case and to acne in the other 2 cases. The location of the keloids was right shoulder, left shoulder and chest, respectively. The size of the keloids ranged 2–7 cm in diameter. None of the keloids had been treated during the last 12 months. The patients received 100 J/cm² UVA1 (UVASUN 2000, Mutzhas Aktiengesellschaft, CH-6002 Luzern, Switzerland) 3 times a week for 5–6 weeks. The final cumulative doses for the patients were 1700, 1800 and 1500 J/cm², respectively, given strictly to the lesion alone. The thickness of the keloid was measured before and after treatment with a DermaScan C[®] Ver. 3 (Cortex Technology, Hadsund, Denmark) 20 MHz ultrasound device.

RESULTS

The treatment was tolerated well, and 2 of the patients experienced subjectively softening of the keloid but none had any macroscopic reduction of the scar. The scars were pigmented, but not in a cosmetically disturbing way. No change in the thickness was observed. The keloids measured 6.9, 10.2 and 6.0 mm before treatment and 6.3±0.5 mm, 9.8±0.4 and 6.2±0.2 after treatment, respectively.

DISCUSSION

We found no effect of UVA1 irradiation on stable keloids, contrary to a recent article reporting a successful treatment in 1 case (4). The dose used in that study was 2860 J/cm², i.e. almost twice as high as we used here. This might explain the

lack of response in the present experiment. The UVA dose, thus, needs to be high enough in future studies. The characteristics of the scar tissue, i.e. hypertrophic scar versus keloid and the thickness of the lesion, are also likely to play a role. The keloid in the above publication seemed thinner than the keloids in the present study. The problem with high dose UVA is that the treatment is time-consuming; a single treatment takes about 30 min, and the patient needs to attend up to 3–4 times a week for 2 months. The treatment might be worth trying in combination with other treatment modalities in order to reduce the UVA dose, or for use postoperatively on early lesions.

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REFERENCES

- Kercher M, Volkenandt M, Gruss C, Reuther T, von Kobyletzki G, Freitag M, et al. Low-dose UVA phototherapy for treatment of localized scleroderma. *J Am Acad Dermatol* 1999; 38: 21–26.
- Stege H, Berneburg M, Humke S, Klammer M, Grewe M, Grether-Beck S, et al. High-dose UVA1 radiation therapy for localized scleroderma. *J Am Acad Dermatol* 1997; 36: 938–944.
- Schaffetter K, Wlaschek M, Hogg A, Bolsen K, Schothorst A, Goerz G, et al. UVA irradiation induces collagenase in human dermal fibroblasts *in vitro* and *in vivo*. *Arch Dermatol Res* 1991; 283: 506–511.
- Asawanonda P, Khoo LSW, Fitzpatrick TB, Taylor CR. UV-A1 for keloid. *Arch Dermatol* 1999; 135: 348–349.

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