Complete Regression of a Melanocytic Nevus under Intense Pulsed Light Therapy for Axillary Hair Removal in a Cosmetic Center

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Key Words

Intense pulsed light • Laser • Pseudomelanoma • Regression • Melanocytic lesions

Abstract

Intense pulsed light (IPL) therapy using non-coherent broad-spectrum light has been reported to be effective for hair removal, and also for treating superficial pigmented lesions like ephelides and solar lentigines. We report complete regression of a pigmented melanocytic nevus, histologically confirmed, after hair removal treatment with IPL. The use of lasers and IPL is a common procedure used by dermatologists and even other professions for the treatment of cosmetically troubling skin conditions. The main advantage of such treatment is a reduction of surgical scars, thus producing a favorable cosmetic outcome, but a major limitation is that histopathologic diagnosis is not usually obtained prior to treatment. Such devices should be carefully used in patients with potentially dangerous melanocytic lesions. We also review the recent literature regarding inadequate treatment of melanocytic lesions with lasers.

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Introduction

The use of lasers and intense pulsed light (IPL) technology has become established practice in dermatology and aesthetic medicine. The number of treatments performed with lasers and IPL has experienced a notable increase in the last two decades [1]. Unfortunately, the increase in popularity and widespread use of such equipment, sometimes used by cosmetologists and aestheticians, has been accompanied by a sharp increase in the number of professional errors [1, 2]. The consequences can be severe if cutaneous lesions are improperly treated by staff with limitations in dermatology skills. In this context, treatment of dysplastic nevi during photorepilation must be mentioned.

Case Report

A 42-year-old woman with multiple acquired melanocytic nevi was regularly evaluated in our Department of Dermatology using digital dermoscopy. In one of her routine revisions, dermoscopy revealed that one of her nevi, located on the left axilla, had significantly changed since her last visit. This melanocytic nevus,

which initially measured 6 mm and was dark-brownish in color (fig. 1a), showing a homogeneous reticular pattern on dermoscopy (fig. 1b), had virtually lost all the aforementioned features. Dermoscopy showed several blotches of brownish pigment without any reminiscent pattern of a melanocytic lesion, together with centrally located slight whitish areas mimicking regression or scarring. A very subtle reticule could only be seen at the periphery (fig. 1c). Anamnesis revealed that the patient had undergone eight IPL sessions to remove the hair from her axillae in a cosmetic center. The sessions were performed every five weeks, and she realized that the nevus changed over the last two sessions.

We initially decided on observation and careful follow-up of the patient. Three months later, the lesion showed similar characteristics. The brownish spots of pigment were smaller, and new brownish dots, in a diffuse distribution, could also be seen together with whitish areas (fig. 1d). Therefore, we decided to remove the lesion.

Histologically, the epidermis showed a slight acanthosis and papillomatosis, and prominent basket-wave hyperkeratosis with small superficial microcrusts. Delicate fibrosis, few perivascular lympho-

cytes, and aggregates of melanophages were present in the papillary dermis (fig. 2, 3). No junctional or dermal melanocytic nests were found. Melan-A immunostaining disclosed a normal periodical distribution of epidermal melanocytes. Melanophages were found in the dermis where no Melan-A-labeled melanocytes were present.

Discussion

Melanin is synthesized in tyrosinase-containing melanosomes within melanocytes. Human melanocytes rapidly transfer the produced melanin to keratinocytes and do not accumulate it. These melanosome accumulations (melanosome clusters or packaged melanosomes) form cap-like structures over the nuclei of keratinocytes to protect the nuclei from the damaging effects of ultraviolet radiation. The highest density of melanin is present in the epidermal basal layer as supranuclear melanin caps [3].

Melanin absorbs a wide range of light, making different light sources useful for melanocytic lesion removal, including argon, ruby, alexandrite, Nd:YAG, and more recently, noncoherent intense pulsed flashlamps. IPL is a broadband visible light emitted from a noncoherent, filtered flashlamp. IPL sources emit light in the 500–1,200 nm range and allow hair removal, treatment of vascular lesions, and removal of either superficial or deep melanocytic lesions [4].

The target of IPL treatment of melanocytic lesions is the melanosome. Selective thermal damage has been observed with a wide range of wavelengths, from 351 to 1,064 nm. Long wavelengths are able to penetrate more than 3 mm and are suitable for dermal melanocytic lesion removal. On the contrary, short wavelengths reach 200 nm beneath the epidermis and are safe for treatment of superficial melanocytic lesions due to preservation of the dermis. The use of long wavelength would explain why epidermal melanocytes persisted in this case. The IPL pulse duration is on the order of milliseconds. In considering the thermal relaxation time of a melanosome, selective photothermolysis requires a pulse duration of nanoseconds (70–250 ns). Long pulses, greater than the melanosome thermal relaxation time, may provoke thermal diffusion from melanosomes to their surroundings, with potential deleterious side effects, and may stim-

Fig. 1. Clinical (a) and dermoscopic (b) morphology of the nevus prior to treatment with IPL. c Dermoscopic morphology after 8 sessions of IPL treatment. d Dermoscopic picture 3 months after the end of IPL treatment.

Fig. 2. Low-power magnification showing slight epidermal hyperplasia with hyperkeratosis. H&E, $\times 40$.

ulate a rapid differentiation of epidermal cells by heating effects [4].

Regarding the dermoscopic-pathologic correlation in this case, the brownish blotches of crusted pigmentation shown with dermoscopy could be probably related to the microcrusts shown by reflectance mode confocal microscopy after treatment of solar lentigines and ephelides with IPL [5].

Electron microscopy studies have analyzed the microscopic structure of these microcrusts, showing that they were composed of numerous melanosomes together with cellular debris. The microcrusts usually appeared on day 5 after IPL irradiation. The mechanism of IPL photothermolysis of pigmented lesions consists of denaturation of melanin caps-containing cells and promotion of a rapid differentiation of keratinocytes, accompanied by an upward transfer of melanocytes along with necrotic keratinocytes, which results in the elimination of melanosomes as the microcrusts are removed from the skin [5].

Microcrusts could not be seen on the adjacent non-pigmented skin, and their formation seemed to depend on the amount and density of pigments contained in the lesion. They usually disappear within two weeks after irradiation [6]. We do not have any plausible explanation for the persistent accumulation of the microcrusts in this patient because she denied new pulsed light sessions. Otherwise, these microcrusts are not a feature associated to spontaneous regression of melanocytic nevi.

The slight whitish areas appreciated dermoscopically would correspond histologically to a particular type of fibrosis composed of bundles of extremely fine and ordered collagen fibers present in the papillary dermis. This subtle fibrosis, very unusually seen in the histopathological evaluation of regression in melanocytic nevi, may be explained by the exposition of IPL and may be related to the rapid evolution of the regression process.

The increased use of lasers and light sources for the removal of pigmented skin lesions may lead to a growing risk of erroneously treated melanocytic tumors. In recent years, several errors (or complications) related to laser treatment of melanocytic lesions have been reported [1, 7–21]. Although lentigines and café au lait spots are frequently managed with IPL [4], there are few cases of melanocytic nevi treated with this technique in the literature, main-

Fig. 3. A microcrust was present in the epidermis above a papillary dermis with delicate fibrosis and a mild superficial inflammatory component with melanophages. H&E, $\times 100$.

ly because the use of laser therapy and IPL in the treatment of pigmented melanocytic lesions is a controversial issue. A complete regression of melanocytic nevi after laser treatment, as occurred in our case, is not usually described.

IPL and melanin-targeting lasers can eliminate superficial melanocytes. However, reported studies about laser treatment of melanocytic lesions show that, in most cases, the complete removal of melanocytic nevi cannot be obtained either clinically or histologically, so repigmentation is frequently seen [2, 8, 13, 14]. Ablative systems such as CO₂ and argon lasers are not pigment-specific and carry a special risk of relapse [2].

Some publications about misdiagnosis induced by laser therapy reported so-called pseudomelanoma [7–10]. The term pseudomelanoma has been used to describe a lesion that histologically resembles superficial spreading melanomas and is clinically seen as areas of repigmentation. It typically occurs following incomplete treatment of a melanocytic nevus either by surgery, dermabrasion or laser therapy [2, 13, 14].

Moreover, several cases wherein melanoma was diagnosed after laser treatment of presumed benign nevi have been recently described [11–21]. It is still unclear whether the lesions were primary malignant melanomas treated with laser or if a malignant melanoma was induced by the laser treatment. Presumably, most cases represent an incorrect treatment based on a clinical misdiagnosis. However, a modification of the melanocytic lesion induced by the invasive procedure could not be ruled out in some cases, since the interval between laser therapy and progression was conspicuously short [11, 13]. Table 1 summarizes a collection series of patients who developed melanoma or pseudomelanoma subsequent to treatment of a melanocytic lesion with laser, obtained from MEDLINE-cited journals.

Conclusion

In this case, fortunately, the dermoscopic characteristics of the lesion prior to IPL treatment and the follow-up supported the diagnosis of a benign melanocytic nevus. The presence of dermoscopic mi-

Table 1. Collection series of patients who developed melanoma or pseudomelanoma subsequent to treatment of a melanocytic lesion with laser

First author	Age/ sex	Location	Initial clinical evaluation	Laser type	Histologic diagnosis	Interval from laser treatment to diagnosis	Metastasis at follow-up
Arndt, 1986 [16]	64/M	none	LM	argon	LM	4 years	–
Trau, 1986 [8]	45/F	back	congenital nevus	CO ₂	pseudomelanoma	4 months	–
Dummer, 1998 [10]	44/F	upper leg	pigmented nevus	CO ₂	pseudomelanoma	6 weeks	–
Lee, 1998 [21]	71/M	cheek	pigmented actinic keratosis	QSRL	LM	1 year	–
Lee, 1998 [21]	59/M	cheek	lentigo	QSRL	LMM	30 months	–
Grob, 1999 [15]	47/M	–	red papular lesion	CO ₂	amelanotic MM	1 year	–
Hwang, 2002 [9]	18/M	chin	congenital nevus	CO ₂ /QSRL/ alexandrite	pseudomelanoma	43 months	–
Böer, 2003 [17]	37/F	shoulder	flesh-colored papule	ablative laser	nodular MM	several months	subcutaneous cellular tissue metastasis
Böer, 2003 [17]	54/F	cleavage	melanocytic nevus	ablative laser	MM	several months	–
Dummer, 2003 [12]	64/F	forehead	brownish macule	alexandrite	LMM	6 months	lymph node metastasis
Dummer, 2003 [12]	?/F	right arm	brownish papular lesion	CO ₂	MM	12 months	lymph node metastasis
Woodrow, 2003 [14]	27/F	upper back	giant congenital nevus	argon	SMM	11 years	–
Gottschaller, 2006 [13]	64/F	cheek	lentigo	CO ₂	N/A	3 years	parotid gland metastasis
Lee, 2006 [7]	16/F	upper extremity	congenital nevus	CO ₂ /erbium/ alexandrite	pseudomelanoma	–	–
Giacomet, 2008 [11]	42/F	back	small black nodule	N/A	invasive melanoma with satellite metastasis	N/A	hepatic, pulmonary and intracranial metastasis
Zipser, 2010 [2]	52/F	cheek	seborrheic keratosis	CO ₂ /erbium	MM not classifiable	80 months	–
Zipser, 2010 [2]	72/M	forehead	melanocytic nevus	N/A	LMM	20 months	–
Zipser, 2010 [2]	66/M	lentigo	lentigo	CO ₂	LM	7 years	–
Zipser, 2010 [2]	75/F	preauricular	N/A	CO ₂	LM	N/A	–
Zipser, 2010 [2]	63/F	mandibular	melanocytic nevus	N/A	SSM	6 months	–
Zipser, 2010 [2]	46/F	eyebrow	melanocytic nevus	N/A	N/A	9 months	–
Zipser, 2010 [2]	47/M	preauricular	verruca	CO ₂	nodular MM	1 year	–
Zipser, 2010 [2]	21/F	upper arm	melanocytic nevus	N/A	ALM	1 year	–
Zipser, 2010 [2]	45/F	forehead	verrucous nevus	N/A	NMM	5 months	–
Zipser, 2010 [2]	59/F	cheek	lentigo	erbium:YAG	LMM	5 months	–
Zipser, 2010 [2]	75/F	upper arm	basal cell carcinoma	CO ₂	NMM	18 months	–
Zipser, 2010 [2]	40/F	under lower lip	lentigo	CO ₂	nodular MM and ALM	7 years	–

ALM = Acral-lentiginous melanoma; LM = lentigo maligna; LMM = lentigo maligna melanoma; MM = melanoma; N/A = not available; QSRL = Q-switched ruby laser; SSM = superficial spreading melanoma.

crocrusts should help us in the diagnosis of pulsed light- or laser-induced regression of melanocytic nevi. When a laser-treated skin lesion is excised and examined histologically, it is absolutely essential that the dermatopathologist have as much clinical information as possible to establish a correct diagnosis and to avoid unnecessary surgical procedures, and other forms of

adjuvant treatment and follow-up programs [7, 9, 10].

Melanocytic lesions should not be considered a routine indication for laser therapy for cosmetic reasons [11, 13]. Moreover, clinically ambiguous skin tumors should never be treated with laser prior to a diagnostic biopsy. The first-choice treatment of equivocal skin lesions, particular-

ly with a history of changes, remains excision with histopathologic examination [11].

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