The Ivory Colored Hypopigmentation
After Autologous Suction Blister Grafts in Vitiligo
Lesion May be Caused by the Pretreated Deep Freeze

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Background: Ivory colored hypopigmentation has been frequently observed in morphea and lichen sclerosus et atrophicus, and also seen after phenol peels, dermabrasion, cryosurgery and post-laser resurfacing.

Objective: This study was undertaken to investigate the cause of hypopigmentation following autologous suction blister graft (ASBG) in vitiligo patients.

Methods: The ivory lesion and contralateral normal skin were collected by punch biopsies. And the tissues were stained with hematoxylin-eosin, Fontana-Masson, Masson’s trichrome, Verhoeff-van Gieson, and S-100 protein to compare the differences between two specimens.

Results: H-E and Masson’s Trichrome stains showed that compacted hyalinized sclerotic collagens and collapsed, small sized capillaries in the upper dermis were definite in the hypopigmented lesion, whereas normal control sites were unremarkable. Elastic fibers were markedly decreased or fragmented in upper dermis of the hypopigmented lesion. Fontana-Masson stain identified that the lesional epidermis was more hyperpigmented rather than hypopigmented. S-100 stain showed no differences between hypopigmented and control sites.

Conclusion: The results suggest that the ivory colored hypopigmentation is not caused by the failure of procedure, but by scar formation due to deep freeze; therefore, this kind of complication may be prevented by carefully performing the cryotherapy.


Key Words: Vitiligo, Autologous suction blister graft, Ivory hypopigmentation, Cryotherapy

Vitiligo is a relatively common depigmentary disorder, and many therapeutic options are available, such as, medical and surgical methods. Of the surgical methods, until now autologous suction blister graft (ASBG) is widely used to treat a localized or segmental vitiligo, and is found to be very effective. When ASBG was performed, the skin of the recipient site was pretreated with liquid nitrogen one or 2 days before surgery, although PUVA or Laser were used occasionally. The absence of scarring, rapid repigmentation, and the reutilization of the donor site for further repigmentation procedures after time elapses are considered to be the most important advantage of this method. However, several complications have been reported.

Although many dermatologists performing ASBG experience some degree of hyperpigmentation which is inevitably anticipated, hypopigmentation is rarely seen on the grafted site and is supposed to be an unreported risk. Moreover, clinically similar hypopigmentation is frequently observed in morphea and lichen sclerosus et atrophicus, and also
described after phenol peels, dermabrasion, cryosurgery and post-laser resurfacing. The authors experienced three cases showing round ivory colored hypopigmented macules on the grafted sites after ASBG which were more accentuated in hot environments or exercises. Therefore, we examined the ivory hypopigmented lesions to see whether the cause resided in epidermis or dermis.

MATERIALS AND METHODS

Patients
Three Korean patients were segmental vitiligo, and Fitzpatrick skin prototype IV. Two months after ASBG, the ivory colored hypopigmentation was evident, and persisted for 8 to 25 months before this study(Fig.1 a, b). The ASBG was performed as previously described. Briefly, one day before grafting, blisters at the recipient site were made by the application of liquid nitrogen with cryogen, Brumwell(3)(Vernon, CT, USA). Another blister was made by suction from the buttock as donor site. After approximately one to one and a half hours of suction at 450 mmHg, a large bulla was formed. After removal of the roof of the blister at the recipient sites, the roof of the suction blister was carefully dissected, and the epidermal sheet from the donor site was grafted to the recipient site. Two weeks after grafting, topical PUVA, or topical corticosteroid application were recommended. The patients are summarized in Table 1.

Methods
Specimens were collected by punch biopsies 2.5 mm in diameter at the ivory hypopigmented lesion of the ASBG site and contralateral normal skin. And the tissues were stained with hematoxylin-eosin, Fontana-Masson, Masson’s trichrome, Verhoeff-van Gieson, and S-100 stain for the purpose of identifying the general morphologic changes, melanin pigments, collagen fibers, elastic fibers and melanocytes, respectively. Finally, we compared the ivory hypopigmented lesion with the contralateral normal skin.

RESULTS

Hematoxylin-eosin stain showed that compacted hyalinized sclerotic collagens and collapsed, small sized capillaries in the upper dermis were definite in the hypopigmented lesion, whereas normal control sites were unremarkable(Fig. 2a). The epidermis of the hypopigmented lesion showed flattened changes with some rete ridge effacement. Elastic fibers with Verhoeff-van Gieson stain were markedly decreased or fragmented in upper dermis of the hypopigmented lesion as compared with control site(Fig. 2b). Masson’s Trichrome stain revealed the upper dermis of hypopigmented lesions was more sclerotic than that of control. Fontana-Masson stain identified that the lesion epidermis was more hyperpigmented rather than hypopigmented(Fig. 2c). Finally S-100 stain for melanocytes showed that there were no definite differences between hypopigmented lesions and controls (Fig. 2d).

DISCUSSION

Generally normal human skin color is produced by several skin pigments: in the epidermis by melanin and carotenoids; in the dermis by hemoglobin in the capillaries and venules. Of these, melanin is the major determinant of differences in skin color, and vasoconstriction decreases capillary blood flow resulting in a pale skin color. Clinically morphea and lichen sclerosus et atrophicus which are characterized by dermal fibrosis, occasionally represent hypopigmentation.

Also, hypopigmentation is to be expected very

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age(yr)/Sex</th>
<th>Type of vitiligo</th>
<th>Duration of vitiligo</th>
<th>Site</th>
<th>Interval between the ASBG* and biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37 females</td>
<td>Segmental</td>
<td>5 years</td>
<td>Forehead</td>
<td>25 months</td>
</tr>
<tr>
<td>2</td>
<td>28 females</td>
<td>Segmental</td>
<td>14 years</td>
<td>Cheek</td>
<td>16 months</td>
</tr>
<tr>
<td>3</td>
<td>27 females</td>
<td>Segmental</td>
<td>4 years</td>
<td>Neck</td>
<td>8 months</td>
</tr>
</tbody>
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*ASBG: Autologous suction blister graft
Fig 1. Patient (case 2). Before (a), 16 months after (b) autologous suction blister graft. Comparing with the original remnant vitiligo lesions (arrow), multiple round ivory colored hypopigmented patches (arrow heads) were evident and more accentuated in hot environments or exercises.

Figure 2. Patient (case 2). Hematoxylin-eosin (a) stains showed that compacted hyalinized sclerotic collagen changes (*) and collapsed, small capillary lumina in the upper dermis were more definite in the hypopigmented lesion than those of control sites. Elastic fibers (b) were markedly decreased or fragmented in the lesion (*) as compared with control site. Fontanna-Masson stain (c) identified that the lesional epidermis was more hyperpigmented rather than hypopigmented. (d) S-100 stain for melanocytes showed no differences between hypopigmented lesions and controls. (original magnification x 100; L, hypopigmented lesion; C, normal control site)
frequently after Baker-Gordon phenol peels and dermabrasion. From the histologic follow-up study of phenol face peels, a new band of connective tissue 2 to 3 mm in width was laid down in the subepidermal region, and melanocytes were not eliminated, but melanin synthesis was impaired, accounting for the hypopigmentation. Some complications of post-dermabrasion is thought to be consequential to cold injury. Also hypopigmentations after cryosurgery are usually permanent, even though nonfunctioning melanocytes may be recognized in the white area.

Recently, there are several cases of post-laser resurfacing hypopigmentation representing as a permanent effect. Laws et al reported a similar clinical hypopigmentation after CO₂ laser resurfacing, in which case showed a normal number of melanocytes with a decrease in epidermal melanin, and the presence of a zone of dermal fibrosis. The histologic effect of high-energy CO₂ laser on human skin was a subepidermal dermal repair zone consisting of compact new collagen fibers which was similar to those seen with medium-depth chemical peels.

The authors think that all of these conditions including our cases have common dermal sclerotic changes which result in collapsing of vessels leading to hypopigmentation. The hypopigmentations of our cases were developed after epidermal grafting, while the hypopigmentations by other methods, such as dermabrasion, chemical peelings, cryosurgery and laser resurfacing, were produced from destruction of the upper dermis followed by wound healing process. Therefore, our cases need more dermal fibrotic changes for hypopigmented patch to be seen, regardless of epidermal hyperpigmentation. We believe the ivory hypopigmentation is not caused by the failure of procedure, but by a scar formation due to deep freeze for several reasons. First, epidermal melanin pigments in the hypopigmented lesion were more abundant than normal control sites, although ivory colored hypopigmentation was seen clinically in the lesion. Second, thickened sclerotic collagens in upper dermis result in a decreased, narrowed vascularity in the lesion. Third, elastic fibers in the hypopigmented site were more fragmented and lost, which may reveal the depth of cryodamage although collagen changes also could define the damage. Finally immunohistochemical staining with S-100 demonstrated no change in the number of melanocytes compared with the contralateral normal skin. Therefore, when performing cryotherapy with liquid nitrogen as a pretreatment of ASBG, although cryotherapy is usually believed as a scarless method, deep freezing should be avoided if possible.

REFERENCES


