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Two Cases of Linear Lichen Planus Pigmentosus of the Chin in Korean Women Treated by Fractional Lasers and Polydeoxyribonucleotide Injection

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Linear lichen planus pigmentosus is a rare subtype of lichen planus pigmentosus that follows Blaschko's lines, leaving long-standing residual atrophy and pigmentation, especially in dark-skinned populations. Conventional treatments have several limitations regarding the alleviation of pigmentation and atrophy. We report two cases of Korean women with linear lichen planus pigmentosus on their faces who were successfully treated with fractional lasers and intralesional injection of polydeoxyribonucleotide.

Keywords: Atrophy, Fractional laser, Linear lichen planus pigmentosus, Pigmentation, Polydeoxyribonucleotides

INTRODUCTION

Lichen planus pigmentosus, characterized by the insidious onset of mottled or reticulated dark-brown to gray macules or papules on sun-exposed areas and flexural folds, is an uncommon variant of lichen planus[1]. The involvement pattern is mostly diffuse; however, it rarely presents with reticular, blotchy, perifollicular, and linear patterns. Linear lichen planus pigmentosus, likely a combination of lichen planus pigmentosus and linear lichen planus, is a rare subtype of lichen planus pigmentosus associated with Blaschko's lines[2].

Little has been reported about linear lichen planus pigmentosus treatments, especially for facial lesions. Several potential treatments have been tested to try to stabilize the disease and prevent its progression, but they have failed to manage disease-induced atrophy and pigmentation[3].

Herein, we report two cases of linear lichen planus pigmentosus on the face in Korean women who were treated with fractional lasers and polydeoxyribonucleotide injections.

CASE REPORT

Case 1

A 59-year-old healthy Korean woman (Fitzpatrick skin type IV) presented with a 6-month history of asymptomatic dark-brown pigmented atrophic linear macules on the chin following a Blaschko's line (Fig. 1A). She reported no history of trauma on the lesion site. One grayish macule initially appeared on the mental protuberance and gradually spread to near the inferior vermilion border. No other cutaneous lesions or involvement of the oral mucosa and nails were found. She had not taken any medications during the previous 6 months. Routine laboratory parameters, including complete blood cell counts and liver function, were normal. Skin biopsy revealed hypergranulosis, liquefactive degeneration of the basal layer, and dense perivascular lymphohistiocytic infiltration with melanin incontinence (Fig. 2); early, active, linear lichen planus pigmentosus was diagnosed.

The lesion was treated with 0.1% tacrolimus cream, a CO_2

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fractional laser (Fraxis duo; ilooda; 10.2 mj pulse energy, 1.81% coverage, 0.34 msec pulse duration), and a 1 ml polydeoxyribonucleotide (Recovery injection; Kyoungbo Pharmaceutical) injection every 6 weeks. After 10 treatments, the atrophy and hyperpigmentation improved markedly (Fig. 1B), without aggravation, for over 6 months.

Case 2

A 46-year-old Korean woman (Fitzpatrick skin type III) presented with a 2-year history of asymptomatic, dark-brown, atrophic macules linearly distributed along the Blaschko's line on her chin (Fig. 3A). Initially, several asymptomatic skin-colored papules without scales had appeared, leaving the dark-brown atrophic linear macules. She reported no history of trauma on the lesion site. There were no other cutaneous lesions or involvement of the mucosa or nails. She had been taking anti-hypertensive drugs for a long time without interruption before lesion onset. Laboratory parameters, including complete blood cell count, liver function, antinuclear antibodies, and total serum immunoglobulin E, were normal. Skin

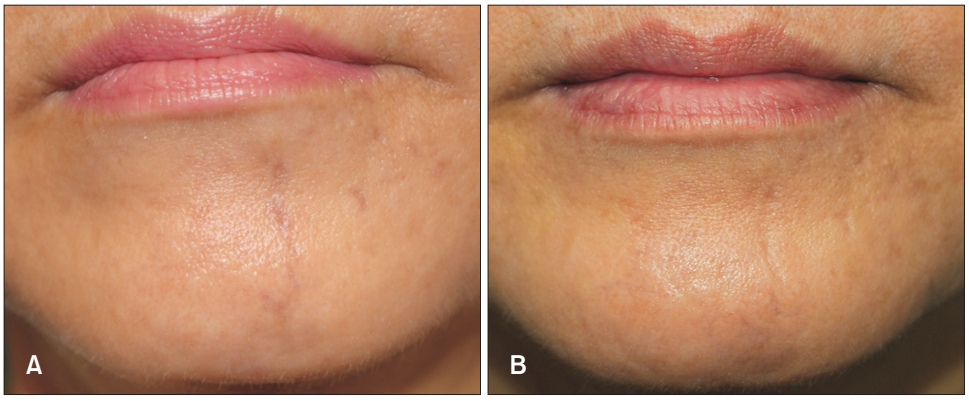


Fig. 1. Photographs showing (A) brown to grayish linear atrophic macules on the chin before treatment. (B) Considerable improvement is observed after treatment with a CO_2 fractional laser and polydeoxyribonucleotide injection.

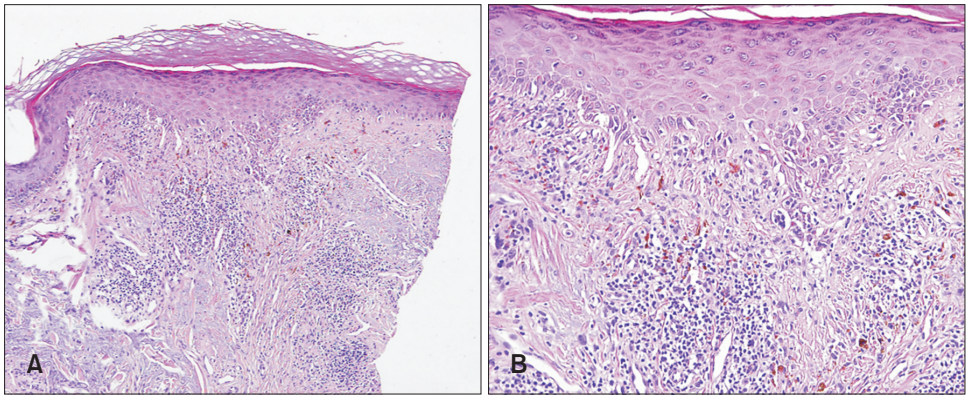


Fig. 2. Histopathology shows hypergranulosis, liquefactive degeneration of the basal layer, dense perivascular lymphohistiocytic infiltrates, and prominent melanin incontinence (H&E, original magnifications: ×100 [A], ×200 [B]).

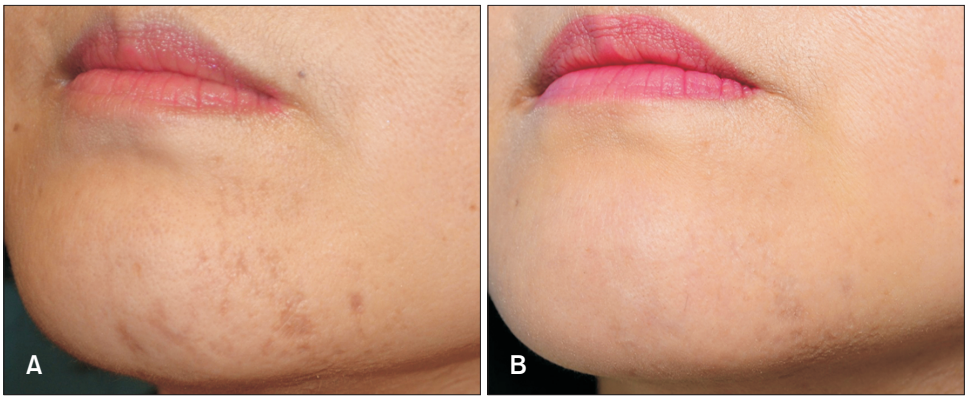


Fig. 3. Photographs showing (A) brown to grayish mottled atrophic macules linearly distributed on the chin before treatment. (B) The lesion improved with mild residual atrophy after treatment with a 1,927-nm nonablative fractional laser and polydeoxyribonucleotide injection.

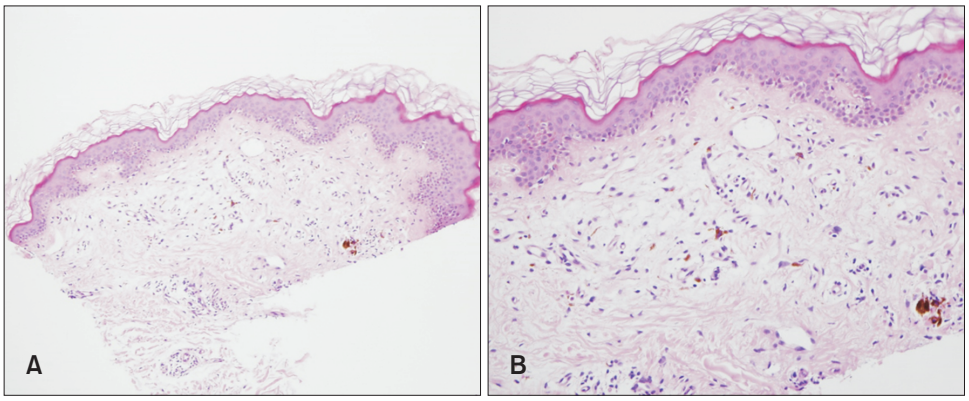


Fig. 4. Histopathology reveals atrophic epidermis, mild basal layer vacuolar degeneration, sparse lymphohistiocytic infiltrates, and pigmentary incontinence (H&E, original magnifications: ×100 [A], ×200 [B]).

biopsy showed atrophic epidermis, focal basal vacuolar degeneration with sparse perivascular lymphohistiocytic infiltrates, and melanin incontinence (Fig. 4), suggestive of a long-standing and evolving linear lichen planus pigmentosus lesion.

Linear lichen planus pigmentosus was diagnosed based on clinical and histopathological findings. To achieve less downtime after the laser treatment, the lesion was treated with a 1,927 nm non-ablative fractional laser (Fraxel SR laser; Reliant Technologies Inc.; 60 mj pulse energy, total density: 304 microthermal zones/cm^2) rather than an ablative fractional laser, and a 1 ml polydeoxyribonucleotide injection every 8 weeks. A 0.1% tacrolimus cream was applied to the lesion as well. After seven treatments, the lesion improved with mild residual atrophy (Fig. 3B).

DISCUSSION

Lichen planus pigmentosus is more likely to occur in darker-skinned populations[2]. Compared with classical lichen planus, lichen planus pigmentosus has a longer clinical course and rarely shows pruritus or scalp, nail, or mucosal involvement[3]. The histopathological findings of lichen planus pigmentosus include atrophic epidermis, hypergranulosis, basal layer vacuolar degeneration, lymphohistiocytic or lichenoid infiltrates in the papillary dermis, and prominent dermal melanophages and pigmentary incontinence. Band-like lichenoid infiltrates may also be found in early lesions, as the intensity on inflammation decreases with disease duration[2].

Although the etiology of lichen planus pigmentosus is unknown, hepatitis C virus infection, hair dye (including henna), and mustard and amla oil may act as triggers in susceptible patients[3]. Inflammatory T-cell infiltrates composed of variable proportions of CD4+ and CD8+ T-cells may play a pivotal role in lichen planus pigmentosus pathogenesis[2]. Autoreactive cytotoxic CD8+ T-cells mediate keratinocyte apoptosis, causing nuclear damage and DNA fragmentation[1]. Furthermore, the expression of nuclear factor kappa B, a transcription factor related to the encoding of several pro-inflammatory cytokines, is elevated in lichen planus lesions, leading to long-lasting inflammation[4].

In both patients, linear lichen planus pigmentosus presented with a unilateral distribution following Blaschko's lines, suggesting that a predisposition to the disease might be determined during embryogenesis. Blaschko's lines are associated with T-cell migration and clonal expansion during skin embryogenesis. Therefore, linear lichen planus pigmentosus may be caused by the genetic mosaicism responsible for cutaneous antigenic mosaicism triggered by various conditions, such as viral infection and medications, resulting in a mosaic T-cell response[1].

Various therapies, including systemic and topical steroids, isotretinoin, dapsone, topical calcineurin inhibitors, chemical peels, and laser therapy (e.g., 1,064 nm Q-switched Nd:YAG), have been used to treat lichen planus pigmentosus and prevent its progression. However, the limitations of conventional treatments for resolving disease-induced pigmentation and atrophy necessitate additional interventions to prevent and improve long-lasting sequelae[3]. Additionally, few studies have dealt with the treatment of linear lichen planus pigmentosus on the face. Further studies are necessary to investigate the possible role of genetic mosaicism in the predilection of dark-skinned populations for linear lichen planus pigmentosus, as well as treatments to stabilize the disease and manage remnant pigmentation and atrophy.

Our patients were first treated with topical tacrolimus, which suppresses T-cell activation. As lichen planus pigmentosus shows a chronic course and treatment resistance compared with classic lichen planus, topical tacrolimus was reported to be only partially effective (approximately 50% improvement)[5]. While topical tacrolimus used in these cases prevented disease progression and aggravation, it was ineffective in controlling remnant pigmentation or atrophy.

We used ablative and non-ablative fractional lasers to effectively treat remnant pigmentation and atrophy. Fractional photothermolysis, which creates vertical columns of thermal injury by fractional emission of light, can improve atrophic scarring by stimulating collagen and elastic fiber synthesis. Pigmentary lesions in patients with dark skin should be treated carefully, as the treatment can exacerbate the pigmentation. Ablative CO_2 fractional lasers can effectively treat recalcitrant pigmentary lesions by transepidermal elimination of the dermal content, such as the dermal melanin pigments in linear lichen planus pigmentosus[6]. Bae et al.[7] suggested that a 1,927 nm non-ablative fractional laser was safe and effective for treating post-inflammatory hyperpigmentation in patients with dark skin. However, using only fractional lasers could not sufficiently improve the atrophic skin lesions.

We injected polydeoxyribonucleotide directly into the lesions to improve pigmentation and atrophy. Polydeoxyribonucleotide, purified from salmon trout sperm and used in a preparation with a high percentage of DNA, stimulates adenosine A2A receptors expressed on various cells, including neutrophils, endothelial cells, and fibroblasts. Activation of these receptors by polydeoxyribonucleotide enhances angiogenesis, extracellular matrix synthesis, and fibroblast proliferation, which are helpful for alleviating atrophic scarring[8]. Additionally, polydeoxyribonucleotide exerts anti-inflammatory properties by activating adenosine A2A receptors, which inhibit nuclear factor kappa B translocation to suppress tumor necrosis factor-α expression[9]. It also exhibits anti-melanogenesis properties by decreasing tyrosinase activity and microphthalmia-associated transcription factor expression[10]. Therefore, polydeoxyribonucleotide may ameliorate long-lasting inflammation, atrophy, and pigmentation in linear lichen planus pigmentosus patients.

We reported two cases of linear lichen planus pigmentosus of the chin in Korean adults. Linear lichen planus pigmentosus following Blaschko's lines involves pigmentation and atrophy that is resistant to conventional treatments. Fractional lasers combined with intralesional injection of polydeoxyribonucleotide may be effective and safe for the treatment of linear lichen planus pigmentosus-induced recalcitrant pigmentary and atrophic lesions.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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