improvement was observed in the area without any complication. We gradually expanded the treatment area. During the sessions, mild blisters developed after treatment with 2.7 J/cm² or higher; however, they disappeared without leaving hyper- or hypopigmentation. A total of seven sessions of laser treatment, with energy between 2.0 and 2.7 J/cm², were successfully made from March 2002 to November 2007 (Fig. 2), and the interval between treatments was several months to 2 years. Thereafter, she was lost to follow-up for 4 years because of personal reasons. In March 2012, she visited our clinic for treatment of the remaining lesions. There was neither a recurrence of RAPK nor treatment-related adverse effects on the previously treated area. We treated the residual lesions with same parameters as previously used, and the clinical effect was also good.

Recently, Fahad et al.⁴ introduced the use of 755-nm Q-switched alexandrite laser as an effective modality to treat RAPK. In our case, the successful depigmentation with

532-nm QSND was made without a significant adverse reaction. We also observed the maintenance of clinical improvement for 10 years without any repigmentation on the treated area. Thus, 532-nm QSND may play a central role in the treatment of RAPK. As this is a single case of clinical experience, a follow-up study will be needed.

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A Case of Psoriasis Exacerbated by Radioactive Iodine Therapy

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Dear Editor:

Psoriasis is a chronic inflammatory skin disease that is characterized by erythematous, sharply demarcated papules and plaques covered by scales. Although the exact causes of psoriasis still remains obscure, it is known that genetic,

immunological, and environmental factors contribute to its etiopathogenesis. Several factors such as trauma, stress, infections, and medications may exacerbate psoriasis. The most common medications known to trigger or worsen existing psoriasis include lithium, gold salts, beta blockers,

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Fig. 1. Generalized, scaly erythroderma with papules and pustules was observed on the arm (A) and abdomen (B) 5 days after radioactive iodine therapy.

and antimalarials. Exacerbation of psoriasis due to medications such as adrenergic antagonists, interferon, gemfibrozil, iodine, digoxin, and chlonidine has also been observed¹. Radioactive iodine (RAI) therapy has been used for the remnant ablation and adjuvant therapy of thyroid cancer. The mechanism of RAI treatment is as follows: the thyroid gland needs iodine and absorbs it from the bloodstream, and then radiation destroys both cancerous and normal thyroid cells. Herein, we report the first case of psoriasis exacerbated by RAI therapy.

A 32-year-old Korean woman presented with a 5-day history of erythroderma with papules and pustules on the whole body. On general inspection, generalized, scaly erythroderma with papules and pustules was observed (Fig. 1). She had a history of psoriasis for about 20 years and had undergone systemic narrow band ultraviolet (UV)-B phototherapy.

She received a diagnosis of papillary thyroid cancer and underwent total thyroidectomy 14 months previously. She was scheduled to have RAI therapy for thyroid cancer and was treated with phototherapy for psoriasis for up to 2 days before RAI therapy. Since then, she had applied topical steroid and calcipotriol ointment for the treatment of psoriasis. Five days after RAI therapy, erythroderma with papules and pustules developed over her whole body. Thus, this case was diagnosed as psoriasis exacerbated by RAI therapy. She applied topical desonide lotion and calcipotriol cream for 3 weeks, and clinical improvement was observed. She restarted to undergo systemic narrow band UV-B phototherapy for the treatment of psoriasis.

Shelley² reported two cases of generalized pustular psoriasis induced by potassium iodide. Kaufman³ observed that iodine/iodides specifically activate the enzyme dihydrofolic reductase. Methotrexate and aminopterin, as folic acid analogues, bind this enzyme, which blocks the metabolic steps in the synthesis of deoxyribonucleic acid and also blocks cell division^{4,5}. Thus, activation of dihydrofolic reductase is associated with aggravation of the disease and inactivation associated with remission. This suggests that dihydrofolic reductase plays an important role in the pathogenesis of pustular psoriasis.

We suspected that the mechanism in this case was related to the activation of dihydrofolic reductase through RAI. Further study about the folic acid pathway in psoriasis and the connection between RAI and psoriasis may be necessary.

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