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Disseminated Superficial Actinic Porokeratosis in a Vitiligo Patient Undergoing Treatment with Long-Term Narrowband Ultraviolet B

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Dear Editor:

Disseminated superficial actinic porokeratosis (DSAP) is distinguishable among the porokeratosis by large numbers of lesions on sun-exposed skin, and sparing of the palms and soles. The tendency to develop DSAP is inherited as an autosomal dominant characteristic. However, a certain amount of accumulated sun exposure and other factors such as immunosuppression, hepatitis, and rarely phototherapy can enhance this tendency[1].

A 52-year-old Korean woman had had vitiligo on her forehead, anterior chest, and abdomen for 5 years. There were no other skin lesions except for vitiligo when the patient visited our clinic, but her son has DSAP on the face. The patient was treated with topical application of corticosteroids, tacrolimus, and narrowband ultraviolet B (NB-UVB, Waldmann UV 5040KL; Waldmann, Villingen-Schwenningen, Germany) phototherapy. Several asymptomatic annular brown colored macules with thread-like borders were identified on her whole body after 7 months of irradiation (Fig. 1A, B). The total cumulative irradiation dose of NB-UVB was 42.9 J/cm^2 at that time, and she had noticed these skin lesions 2 to 3 weeks previously. Interestingly, any skin lesions did not appear in the vitiliginous areas, even though over 100 DSAP lesions had developed on the whole body. A biopsy specimen disclosed the characteristic histopathological findings of DSAP (Fig. 1C).

Although we had warned patient about the risk of malignant change, the patient wanted to continue treatment. Therefore, the normal skin without vitiligo lesions was covered with the cloth during phototherapy. A few new lesions occurred within a few months of the initial detection of DSAP, and the color of lesions was getting darker with time. But the number and size of DSAP lesions were not altered until now.

Nine cases of DSAP associated with photo(chemo)therapy were reviewed (Table 1)[1-3]. In this case, it is thought that DSAP lesions on the trunk was induced by NB-UVB be-

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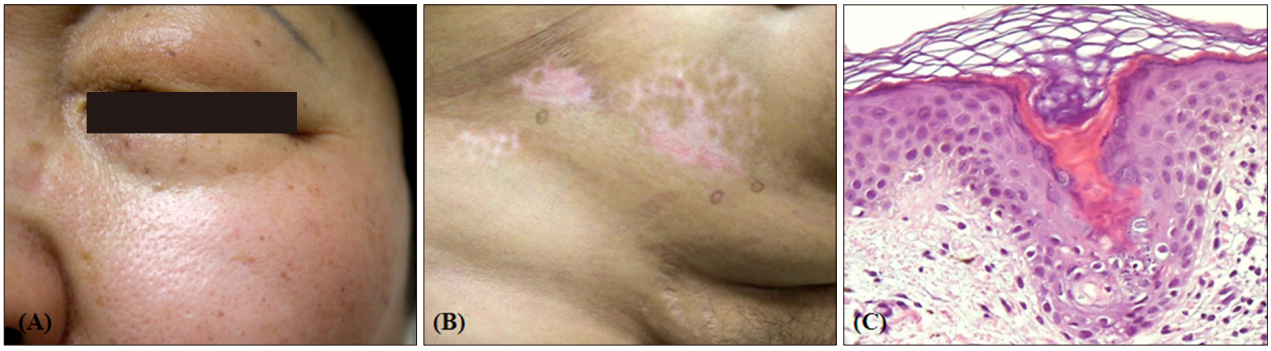

Fig. 1. Clinical and histopathological findings and punch biopsy specimen from disseminated superficial actinic porokeratosis on the abdomen. (A～C) Small round hyperpigmented macules with a keratotic, thread border on her face and abdomen. Histopathological findings show a shallow, keratin-filled invagination. Cornoid lamella with irregularly arranged keratinocytes in the spinous layer (C: H&E, ×400).

Table 1. Summary of photo(chemo)therapy-induced disseminated superficial actinic porokeratosis reported in the literatures

Authors (year)	Sex/age (yr)	Skin disease	Phototherapy	Duration	Total irradiance (J/cm^2)	Site
Reymond et al. (1980)	F/67	Psoriasis	Oral PUVA	24 mo	961	Legs
Farber et al. (1982)	M/66	Psoriasis	Oral PUVA	12 mo	280	Lower limbs, arms, chest, back
	F/45	Psoriasis	Oral PUVA	36 mo	4,173	Limbs
Hazen et al. (1985)	F/30	Psoriasis	Oral PUVA	1.5 mo	19.5	Legs
Cockerell (1991)	M/60	Psoriasis	BBUVB	13 d	0.44	Trunk, legs, arms
Lee et al.[2] (1992)	F/20	Vitiligo	Topical PUVA	Unknown	0.4	Anterior chest
Allen et al. (2000)	F/54	Psoriasis	Topical PUVA	2.5 mo	49.8	Heels, ankles (PUVA-treated area)
Kawara et al.[1] (2011)	M/75	Psoriasis	NB-UVB	33 mo	37.8	Trunk
Present case	F/52	Vitiligo	NB-UVB	7 mo	42.9	Face, anterior chest, abdomen

F: female, M: male, PUVA: psoralen and ultraviolet A therapy, BBUVB: broadband ultraviolet B, NB-UVB: narrowband ultraviolet B.

cause trunk is generally unexposed area to natural sunlight, and DSAP lesions developed after NB-UVB exposure. Kawara et al.[1] reported that DSAP was present after a total dose of 37.8 J/cm^2 for 33 months. In our case, DSAP lesions were found at a 42.9 J/cm^2 in total cumulative dose after 7 months' NB-UVB therapy.

The pathogenesis of DSAP after phototherapy is uncertain. NB-UVB therapy promotes the release of keratinocytic growth factors and is also known to suppress the immune system and reduce inflammation by modulating cytokine patterns, irradiating T cells, and inducing apoptosis[4]. It is possible that chronic immunosuppressive conditions under long-term NB-UVB phototherapy induce clonal proliferation of abnormal keratinocytes.

Though it may be a coincidental finding, the very interesting point in this case is that DSAP lesions did not develop on the areas with vitiligo lesions. So it is thought that the occurrence of DSAP was prevented by vitiligo. Although the underlying mechanism is unknown, it is supposed that this is a similar reason as in vitiligo lesions does not increase the risk of sun induced skin cancer[5]. Therefore, further studies and accumulation of case reports are needed to reveal the relationship between vitiligo and DSAP, as well as the underlying mechanisms for DSAP development.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Medical Comorbidities and the Onset of Androgenetic Alopecia: A Population-Based, Case-Control Study

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Dear Editor:

An association between androgenetic alopecia (AGA) and an increased incidence of metabolic syndrome (MS) has been suggested[1]. AGA might be an indicator of arterial stiffness[2]. A higher prevalence of hypertension has been found in women with early-onset AGA, suggesting that early-onset AGA could be a risk factor for early-onset severe coronary heart disease[3,4]. This study explored differences in medical comorbidities including MS based on the timing of AGA onset.

The medical records of 1,141 subjects who visited the Department of Dermatology and the Occupational Medical Clinic in Wonju Severance Christian Hospital from October 2012 to March 2016 were analyzed retrospectively and classified into early- and late-onset AGA groups. Fifty patients with pattern hair loss who were younger than 35 years old were defined as "early-onset." For comparison, fifty late-onset AGA patients were selected randomly. To evaluate MS, we analyzed the medical history (hypertension, diabetic mellitus and alcohol drinking), blood samples (glucose, lipid profiles, hemoglobin, hematocrit, blood urea nitrogen, creatinine and liver enzymes) and anthropometric indexes (waist, height, weight, body mass index [BMI], and blood pressure) of each group. MS was defined on the basis of the NCEP-ATP III guidelines[5]. This study was approved by the Yonsei University College of Medicine Institutional Review Board (YWMR-15-0-71).

The average age of the early-onset AGA group was 33.7 years and that of the late-onset AGA group was 46.8 years, which did not represent a significant difference ($p=0.244$). The early-onset AGA group was composed of 41 males and 9 females and the late-onset AGA group was composed of 35 males and 15 females. MS was diagnosed in 9 patients in the early-onset AGA group and no patients in the late-onset AGA group. The authors used Fisher's exact test to evaluate medical history, anthropometric and blood abnormalities. The results revealed that the early-onset AGA patients had abnormal BMI, waist circumference and blood parameters relatively, however, there were no statistically significances (Table 1). The comparison of anthropometric and blood parameters using the Mann-Whitney U test revealed no significant differences other than in high density lipoprotein cholesterol level (Fig. 1), which was lower in the early-onset group ($p=0.029$).

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