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Multiple Bowen's Disease: In a Veteran Who Participated in Vietnam War

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Dear Editor:

Many etiological factors have been suggested for Bowen's Disease (BD). However, the relationship between tetrachlorodibenzo-p-dioxin (TCDD) and BD has not yet been clarified. We report here on a case detailing TCDD as a possible cause of multiple BD.

A 63-year-old-man presented with multiple erythematous and scaly plaques over the whole body, which had first appeared over 10 years prior (Fig. 1). He had a history of contact with defoliants while he participated in the Vietnam War (2 years), 40 years ago and was treated for peripheral neuropathies that were possibly related to Agent Orange. The patient was otherwise in good health. The physical examination revealed six scaly erythematous papules and plaques on his back, arm, leg, and finger. According to the patient the lesions occurred spontaneously. Skin biopsies from the back and finger were performed. Histologic examination showed full thickness epidermal atypia with adnexal involvement (Fig. 2). The clinical appearance of the skin lesion and the microscopic descriptions were consistent with a diagnosis of multiple

BD.

TCDD is a kind of dioxin isomer, and TCDD is contained in Agent Orange, which was used as a defoliant during the Vietnam War from 1961 to 1970. TCDD has a half-life of approximately 8 years in humans and it accumulates primarily in fatty tissues over time, so even small exposures may eventually reach dangerous levels¹. Both the National Toxicology Programs and the International Agency for Research on Cancer have classified TCDD as a human carcinogen.

Over the past several years a number of studies have described the cutaneous effects of TCDD toxicity. The common TCDD-induced skin lesions are chloracne, porphyria cutanea tarda, hyperpigmentation, and malignant fibrous histiocytomas of the skin. In Korea, 2 cases of BCC, 1 case of SCC, and 1 case of merkel cell carcinoma in Vietnam Veterans exposed to TCDD have been reported^{2,3}. Ikuta et al.⁴ have reported that in primary cultured keratinocytes, TCDD increases post-confluent proliferation and late differentiation. TCDD also inhibits culture-induced senescence, which is a form of permanent

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Fig. 1. Multiple scaly (A) erythematous papules and plaques on the back, (B) right 4th finger, (C) left arm, and (D) right leg.



Fig. 2. The skin biopsy specimen of the lower back showed full-thickness involvement by atypical keratinocytes. Large, round and hyperchromatic nuclei with mitoses were contained within the dyskeratotic cells (H&E, $\times 200$).

cell cycle arrest. Wyde et al.⁵ identified the development of cutaneous papillomas and squamous cell carcinomas in mice exposed to dermal (≥ 52 ng/kg) and oral (893 ng/kg) TCDD. Thus, TCDD can modulate the differentiation of keratinocytes and promote tumor formation in the skin. BD is SCC *in situ*, and this has the potential to progress to

invasive SCC. The known etiological factors of BD include: (1) irradiation such as sun exposure, photochemotherapy and, radiotherapy (2) carcinogens such as arsenics (3) immunosuppression such as therapeutic immune suppressions and, acquired immune deficiency syndrome, and (4) oncogenic human papilloma virus (HPV) types, such as HPV 16.

Although BD has a predilection for sun-exposed areas, the BD seen in non-sun exposed areas suggest a strong association of BDs with arsenic or HPV. In the present case, PCR of the skin tissue was negative for HPV. Since our patient denied a history of arsenic exposure, we believe that the multiple BD in our case was likely associated with the TCDD exposure.

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Adult Onset of Nevus Unius Lateris

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Dear Editor:

A 48-year-old Korean female patient complained of brownish papules with verrucous surface, and a linear distribution. She had recognized cutaneous lesions about 15 years previously, and these lesions became more remarkable in coloration and linear distribution for the past year. The skin lesions seemed as a linear pattern following the lines of Blaschko, involving the right leg, which were extended to same side of her sole manifest as linear distributed, verrucous, and hyperkeratotic plaques (Fig. 1). She had no past medical history and family history. Histopathological examination from the right calf showed hyperkeratosis, acanthosis and hyperpigmentation of the basal layer (Fig. 2A). The histopathology from her ipsilateral sole showed verrucous surfaced epidermal alterations which were represented by definite hyperkeratosis, epidermal acanthosis, parakeratosis, and rete ridge elongations (Fig. 2B). She was diagnosed with nevus unius lateris. She had a surgical excision of the plantar lesion subsequently reconstruction with a skin graft. Alternatively, topical treatment with retinoic acid and topical

corticosteroid were applied on her right leg lesions.

Epidermal nevus is a mosaic disorder as a result of somatic mutations that have occurred during fetal life¹. Epidermal nevus has a tendency to follow the Blaschko lines. Eighty



Fig. 1. (A, B, C) The skin lesions appeared in a linear fashion, following the lines of Blaschko, involving primarily the right leg, which were extended to ipsilateral sole. Right sole manifest as yellowish, thick, linear distributed, verrucous, and hyperkeratotic plaque.

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