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Disseminated Superficial Actinic Porokeratosis Like Drug Eruption : A Case Report

We report a 54-year-old male patient who developed an unusual form of generalized drug eruption. He had pain and breathlessness on the left chest wall. He had history of taking several drugs at private clinics under a diagnosis of herpes zoster. Two weeks later he had a generalized skin eruption. Examination showed multiple variable sized, mild pruritic, erythematous macules and papules on the face and upper extremities. Skin lesions take the form of a clinically consistent with disseminated superficial actinic porokeratosis (DSAP). Methylprednisolone 16 mg, astemisole 10 mg, oxatomide 60 mg was prescribed. Topical corticosteroid cream was applied. Within two months, his eruption had cleared almost completely. The pathogenetic mechanisms of this case are unclear, but drug and UV light have been considered.

Key Words : *Porokeratosis; Drug eruptions*

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INTRODUCTION

Drug eruption is an undesirable clinical manifestation subsequent to, and caused by, administration of a particular drug. It may depend on a toxic reaction, such as overdosage, accumulation, interaction of more than one drug, idiosyncracy, or an anaphylactoid reaction due to histamine liberation (1-2). The most common cutaneous manifestations of drug reaction are a variety of maculopapular, mobiliform, scarlatiniform, or exanthematous eruption. Up to date drug eruptions mimicking special form of skin diseases have been reported. Recently O'Donnell et al. (3) had described the suramin induced disseminated superficial actinic porokeratosis (DSAP). We report an unusual form of drug eruption mimicking DSAP.

CASE REPORT

A 54-year-old farmer was admitted to Department of Dermatology, Wonju Christian Hospital, Wonju College of Medicine, for skin eruptions and itching sensations over his entire body. He had breathlessness and pain on the left chest wall. He had taken several kinds of drugs at private clinics under a diagnosis of herpes zoster. The patient's medications included acyclovir, tramadol, amitriptyline, cimetidine, prednisolone, hydroxyzine and some unidentified ones. Chest pain had subsequently improved, but 2 weeks later he had a generalized skin eruption.

Examination showed multiple variable sized, mild pruritic, erythematous macules and papules on the face and upper extremities. They had, interestingly, narrow, slightly indurated annular ridges and were most prevalent on his face and upper extremities (Fig. 1). His family and past history were

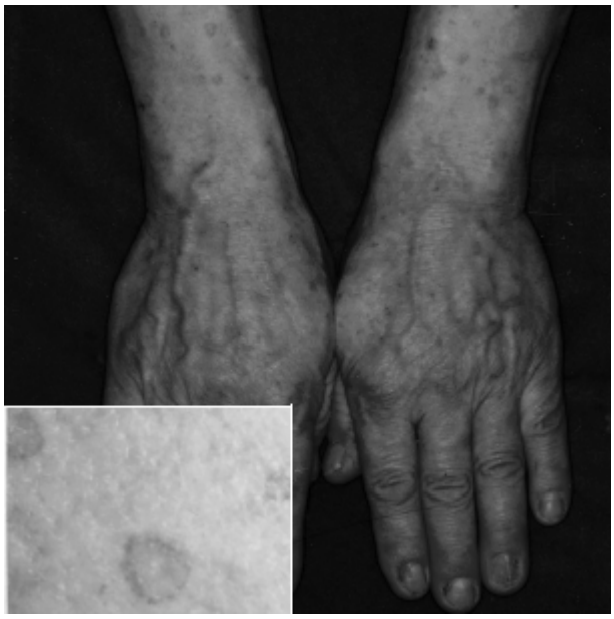

Fig. 1. Multiple variable sized erythematous annular macules and papules on both forearm. Inset: slightly indurated narrow annular ridge with central depression.

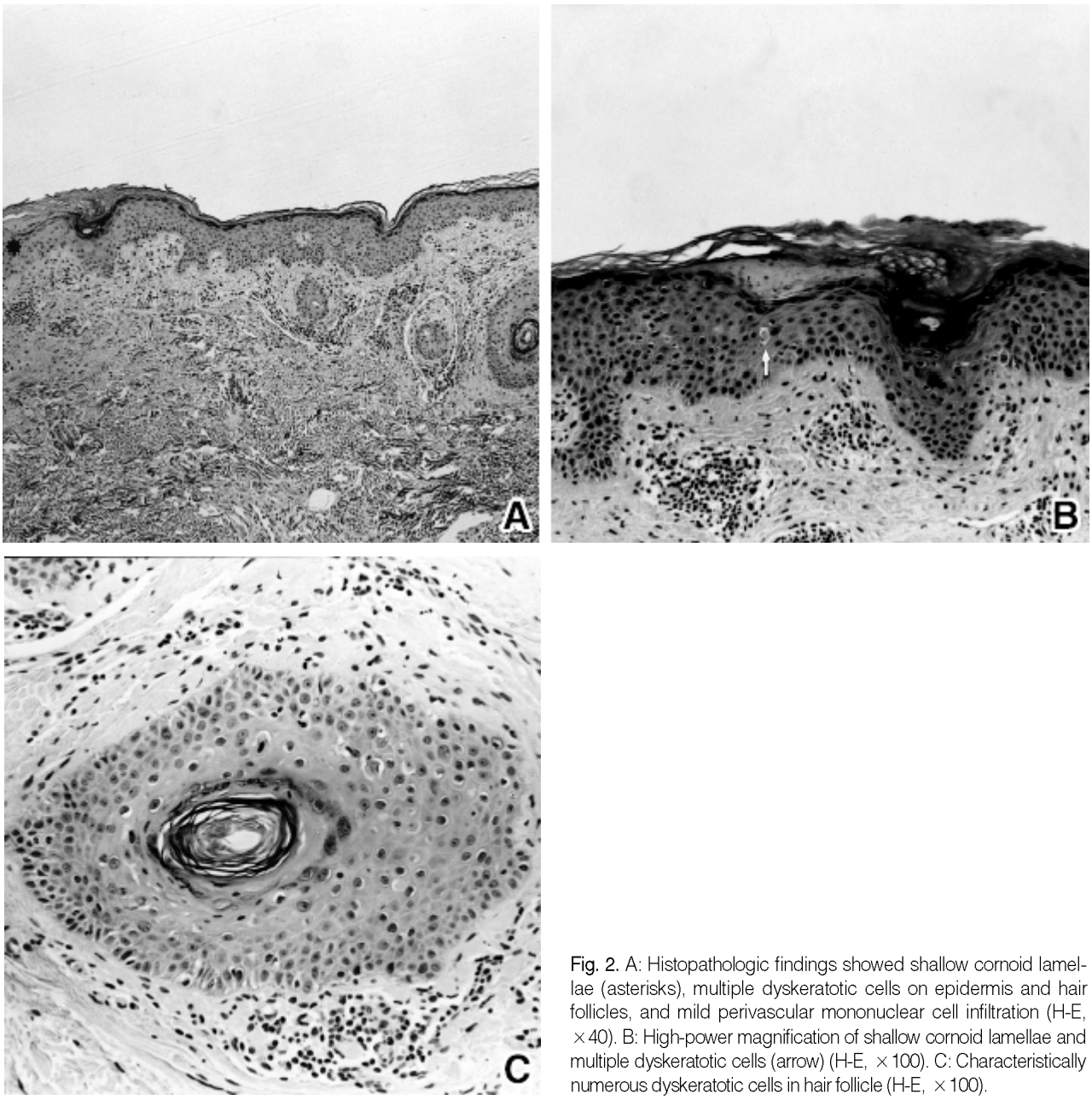

Fig. 2. A: Histopathologic findings showed shallow cornoid lamellae (asterisks), multiple dyskeratotic cells on epidermis and hair follicles, and mild perivascular mononuclear cell infiltration (H-E, ×40). B: High-power magnification of shallow cornoid lamellae and multiple dyskeratotic cells (arrow) (H-E, ×100). C: Characteristically numerous dyskeratotic cells in hair follicle (H-E, ×100).

not contributory. Laboratory findings showed leukocytosis (WBC 16,580; neutrophil 78.4%) and weakly positive antinuclear antibodies (1:20, speckled pattern). Histopathologic findings from DSAP-like lesion showed localized minimal hyperkeratotic horny invagination with parakeratosis (shallow cornoid lamellae), multiple dyskeratotic cells on the epidermis and hair follicles, and mild perivascular mononuclear cell infiltration (Fig. 2A, B). Especially, there were characteristic features of numerous dyskeratotic cells in the hair follicle (Fig. 2C).

The diagnosis was made as drug induced DSAP. Methylprednisolone 16 mg, astemisole 10 mg, oxatomide 60 mg was prescribed. Topical corticosteroid cream was applied. Within two months, his eruption had cleared almost completely.

DISCUSSION

With the rapid development of new therapeutic agents, there are many reports of new causative drugs and clinical patterns of drug induced dermatitis each year. The various

clinical types of drug eruptions include maculopapular, fixed drug eruption, erythema multiforme, toxic epidermal necrolysis, Steven-Johnson syndrome, urticaria, and erythroderma (1-2). Clinical improvement, retrial of medication, and then flare-up of clinical symptomes or positive patch test are diagnostic clues.

In this case, there was a photoactive distribution and clinically and histopathologically consistent with DSAP. DSAP, characterized by one or more annular plaques of hyperkeratosis with central atrophy and histopathologically shallow cornoid lamellae, is an uncommon autosomal dominant localized keratinization disorder. The pathogenesis is unknown. Activation of abnormal clones in the epidermis, induced by drug and/or UV irradiation, and genetic predisposition are pathogenetically linked (4-6). The association of these lesions with UV exposure (7), immunosuppression (8-9), electron beam radiation (10) or drug (6), has been previously reported. Inamoto et al. (6) reported that an 72-year-old man taking benzylhydrochlorothiazide for hypertension, had a flare up of porokeratosis which histopathologically revealed eosinophilic spongiosis and cornoid lamellae.

Unfortunately we could not identify the offending drug, because he had visited many doctors and taken many drugs for relief of his pain and refused further studies. However, withdrawal of all suspected drugs and application of topical and systemic steroid was followed by rapid disappearance of the eruption, suggesting the drug eruption mimicking DSAP. Chernovsky and Anderson (4) and Inamoto et al. (6) suggested that specific factors such as drugs and UV irradiation may be responsible for changing dyskeratotic cells in cornoid lamellae. In our case, there were characteristically abundant dyskeratotic cells in the follicular epithelium, but these were never observed in association with cornoid lamellae and/or porokeratosis.

In conclusion, we present a case of DSAP-like drug eruption which was histopathologically characterized by cornoid lamellae and many dyskeratotic cells in the follicular epithelium. Notwithstanding the fact that we could not elucidate the causative drug, drug and UV exposure have now been associated with the development of this adverse reaction. The physician should be aware of clinical diversity of drug eruptions and consider the drug eruption in patient who show its sudden appearance with medication and subsidence of reaction on withdrawal of drug.

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