A Case of Juvenile Spring Eruption of the Ears

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Juvenile spring eruption (JSE) of the ears is an unusual type of photodermatosis, which develops on the light exposed areas of the ears of boys and young male adults in the early spring months. JSE has received little attention in the literature, and to our knowledge no cases have been reported in Korea until now.

Herein we report a case of JSE occurring in a 17-year-old man who has suffered from a recurrent pruritic erythematous papulovesicular eruption of both helix, followed by crusting and healing without scarring within one to two months early each spring for six years. (Ann Dermatol 9:(2) 139–142, 1997).

Key Words: Ears, Juvenile spring eruption

Juvenile spring eruption (JSE) of the ears is a rare photodermatosis originally described by Anderson et al.1 in 1954. It affects mainly boys aged 5-12 or young men in the early spring months. It is characterized by grouped papules and vesicles on the light-exposed helix of the ears, followed by crusting and healing within 1 to 2 weeks to leave little scarring. Recurrences during following springs can be seen. Although the disease is self-limiting and not in itself serious, it may cause the patients considerable discomfort and concern.

Herein we report a case of JSE occurring in a 17-year-old man who has suffered from a recurrent pruritic erythematous papulovesicular eruption of both helix, followed by crusting and healing without scarring within 1 to 2 months early each spring for 6 years.

REPORT OF A CASE

A 17-year-old man visited our department complaining of recurrent redness, itching, and blistering over the light exposed areas of both ears. For 6 consecutive years the lesions had appeared in March to April and persisted for 1 to 2 months. There was a close relationship to sunlight exposure. Erythema and pruritus typically developed within a few hours of exposure to sun and, in a severe episode, progressed to papules or vesicles. This was generally followed by crusting and healing within 1 to 2 months to leave little scarring. Over the last 2 years he had simultaneously been affected by a more generalized photosensitive, pruritic, erythematous eruption over the face, backs of the hands, and the V-neck area. A medical history disclosed that no topical photosensitizing agents had been applied on the ears. There was no family history of a similar eruption or other photodermatoses.

A physical examination showed diffuse erythema and crusts over the helix and lobules of both ears (Fig. 1). The ears were protuberant. Minimal scarring was present from previous lesions. Other sun-exposed areas involving the face, backs of the hands, and V-neck revealed only diffuse erythema.

On laboratory examination, a complete blood count, anti-nuclear antibodies and porphyrin levels in the urine were all normal or negative.

Histopathological examination of the crusted lesion of the helix of the left ear showed acanthosis with mild to moderate hyperkeratosis in the epidermis, and chronic perivascular lymphocytic infiltration in
the upper dermis (Fig. 2). Direct immunofluorescent staining revealed no abnormal deposition of immunoglobulin, fibrin or complement.

We used a demalight 2001 (Dr. Honle, Germany) as a polychromatic UVA, whose emission spectrum reaches from 320-400 nm with a maximum between 370-380 nm. For irradiation with polychromatic UVB, PANOSOL II UVB (National Biological Corporation, U.S.A.) was used. The emission spectrum reaches from 230-400 nm with a maximum between 300-310 nm. Minimal erythemal dose (MED) of our patient for UVA and UVB was 30 J/cm² and 40 mJ/cm², respectively. Attempts to reproduce the reaction on the upper part of the ears by two MEDs of UVA and UVB irradiation were unsuccessful.

Symptomatic improvement was obtained from topical steroids, oral antihistamines and sunscreen during each attack. A three-year-follow up has revealed a self-limiting course and no recurrence until now.

**DISCUSSION**

JSE of the ears is a distinctive photodermatosis characterized by the onset of papules and vesicles on the light-exposed helix of the ears, followed by crusting and healing within 1 to 2 weeks to leave little scarring. This condition usually develops in the early spring months. Recurrences during following springs can be seen. It affects mainly boys or young men but it is less common in girls because their ears are normally shielded by long hair. In our patient the typical clinical features on both ears and a past history of recurrent attacks every March to April are well compatible with those of JSE. Over the last 2 years he had been affected simultaneously by a more generalized photosensitive, pruritic, erythematous eruption over the face, backs of the hands, and the V-area of the chest. But a family history, obtained in other sporadic cases of JSE, was not found in our case.

The aetiology of JSE of the ears and the relationship with other photodermatoses has not been fully understood. Anderson et al. originally described an outbreak in a children’s summer camp in Surrey, where 121 of 150 children at the camp in early April 1953 were affected. In all but one case, in which there were lesions on a hand, the eruption involved only the ears. Although the camp was closed due to alarm over a possible infectious aetiology, this was not confirmed and the authors concluded that the eruption may have been due to a combination of sunlight and cold air. The affected children were outdoors for most of the day in sunny but cold weather with a moderate wind. Requena et al. reported an additional outbreak in which 27 soldiers who participated in military manoeuvres were affected. In these two reports there were two common striking clinical features: first, the isolated location to the ears without other sun-exposed skin areas involved and second, its presentation as small outbreaks. Both features were due to an unknown mechanism.

Burckhardt in 1942 described a very similar condition in 18 school children in Zurich. He also studied a school class containing 50 boys and 50 girls during a period of unusually fine spring
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weather; 44 of the boys developed papules and crusting of the ears, whereas only one girl with swept back hair developed the changes. Baran and Civatte reported a series of 10 similar cases. In these reports histological similarity to erythema multiforme has been emphasized, suggesting that JSE may represent a photo-induced variant of this condition.

Recently, Berth-Jones et al. reported 18 sporadic cases with JSE of the ears. Four of these patients had, on other occasions, suffered from typical polymorphous light eruptions (PMLE). Among them, MEDs of one patient were mildly decreased at 340 nm and 360 nm, respectively. The clinical features, histological findings, and results of phototesting suggested that JSE was a localized form of PMLE. Warin reported JSE of the ears developing in four brothers, one of whom also gave a history of PMLE and hydroa vacciniforme.

Histologically the picture evolves according to the evolutionary stage of the lesions from a nodular dermal inflammatory infiltrate in papular lesions to subepidermal blisters in vesicles. Burckhardt performed a biopsy in one case and considered the histology compatible with erythema multiforme. Baran and Civatte reported histological findings in two patients and supported the view that the histological features were those of erythema multiforme. In our case histopathologic examination of the crusted lesion of the helix of the left ear showed acanthosis with mild to moderate hyperkeratosis in the epidermis and chronic perivascular lymphocytic infiltration in the upper dermis. Birth-Jones et al. reported that direct immunofluorescence investigations of skin lesions revealed no abnormal deposition of immunoglobulin, fibrin or complement. Staining for surface markers on the dermal infiltrate showed this to be predominantly lymphocytic and composed of approximately equal numbers of CD4+ and CD8+ cells. In our case, immunofluorescence investigation also yield a negative result.

In recent reports of JSE, attempts to reproduce the reaction on the ears using solar-simulated irradiation were not successful. Minimal erythematous responses of the skin of the upper back to a series of increasing doses of narrow wavebands of ultraviolet and visible light from an irradiation monochromator were normal. Reactions of uninvolved skin to irradiation with broad-band ultraviolet A and ultraviolet B were also normal. In our patient, MEDs for UVB were normal but MEDs for UVA were lowered. Our attempts to reproduce the reaction on the upper parts of the ears by two minimal erythema doses of UVA and UVB irradiation were unsuccessful.

The differential diagnosis includes chilblain and a spectrum of other photodermatoses that may give rise to a skin lesion on the ear such as light-induced erythema multiforme, hydroa vacciniforme, actinic prurigo, lupus erythematosus and porphyria. Erythema multiforme may affect the ear and may also be rarely light-induced. No histological resemblance to erythema multiforme was found in our case. Hydroa vacciniforme and actinic prurigo predominantly affect children. Although they may produce lesions on the ears they do not generally occur in isolation. The cheeks, nose and hands are also usually affected. Further, hydroa vacciniforme produces severe scarring, not a feature of JSE. The porphyria may induce photosensitivity and blistering of the skin but are not confined to the ears. In our patient, porphyrins in urine were not detected. Lupus erythematosus may involve the ears. Pits and scarring, however, occur in this disease and there may be atrophy of the lobe and even perforation of the pinna. Antinuclear antibodies were not detectable and direct immunofluorescent staining revealed no abnormal deposition of immunoglobulin, fibrin or complement in our case. Our patient had no history of chilblain on the ears and other parts of the body after cold exposure during the bitter winter. Clinically, recurrent papulovesicular eruption of both helix developed after sunlight exposure during the day time in spring and the patient did not complain of exposure to cold weather. Therefore, we could exclude chilblain.

Symptomatic improvement can be obtained from topical steroid preparations, oral antihistamines and chloroquine. Some reduction in the frequency and severity of attacks was achieved using sunscreen. In our patient, symptomatic improvement was achieved using sunscreen, topical steroid preparations and oral antihistamines during each attack. The course of JSE has been known to be self limiting and this has been testified by our case over a 3-year-follow up period.
REFERENCES