

A Case of Halo Dermatitis around Seborrheic Keratosis

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A case of circular eczematous dermatitis around seborrheic keratosis was presented. A 54-year-old man presented with two weeks history of a round pruritic eruption around a preexisting lesion of seborrheic keratosis. Microscopic findings showed central tumor nest composed mainly of basaloid cells with occasional spongiosis and exocytosis. A diffuse, dense, mononuclear cell infiltrate with increased contents of melanophages was seen beneath the tumor. The surrounding lesion of halo dermatitis showed mild dilatation of the capillaries and perivascular infiltration of mononuclear cells in the upper and mid-dermis.

The lesion of halo dermatitis disappeared gradually over a three week period following excision of the central lesion. This peculiar phenomenon seemed to be different from that of Sutton's halo nevus, and might be a manifestation of an eczematous condition which might have a dermatitis provoking factor. (*Ann Dermatol* 5:(2) 83-85, 1993)

Key Words: Halo dermatitis, Seborrheic keratosis

A circular eczematous reaction around pigmented nevi was first described by Meyerson¹, and was called halo dermatitis², halo eczema³⁻⁴, or Meyerson's nevus⁵. After Meyerson's report, many authors reported similar cases in pigmented nevi. It differed from traditional Sutton's halo nevus clinically and histopathologically¹⁻¹⁵. But, this halo dermatitis was reported mainly in the pigmented nevi, and reports of halo dermatitis around tumors other than pigmented nevi were scarce⁶⁻⁷. We present a case of halo dermatitis around seborrheic keratosis.

REPORT OF A CASE

A 54-year-old man presented to the Department of Dermatology, Gyeongsang National University Hospital in January 1992, with a two-week histo-

ry of a round pruritic eruption around previously noted seborrheic keratosis on his right shin. The lesion was a 4×5cm sized round dark reddish patch with a crusted border, and had a 0.7×0.7cm sized black crusted paule in its center (Fig. 1).

Routine examination, including complete blood count, blood chemistry, and chest X-ray was within normal limits. The lesion of seborrheic keratosis and portions of surrounding eczematous lesions were excised.

Microscopically, the central lesion showed a greatly thickened epidermis, composed mostly of basaloid cells with mild hyperkeratosis and papillomatosis. The surrounding lesion of halo dermatitis showed mild dilatation of the capillaries and perivascular infiltration of mononuclear cells in the upper and mid-dermis (Fig. 2). A diffuse, dense mononuclear cell infiltrate with increased content of melanophages was seen beneath the tumor (Fig. 3).

The lesion of halo dermatitis disappeared gradually over a three week period following excision of the central lesion without any other treatment.

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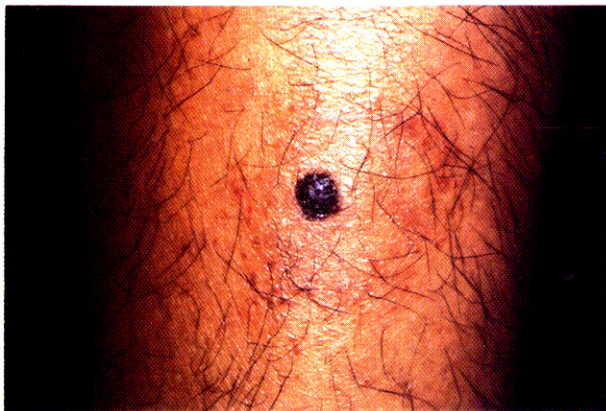


Fig. 1. A round dark reddish eruption around previously noted seborrheic keratosis on the patient's right shin.

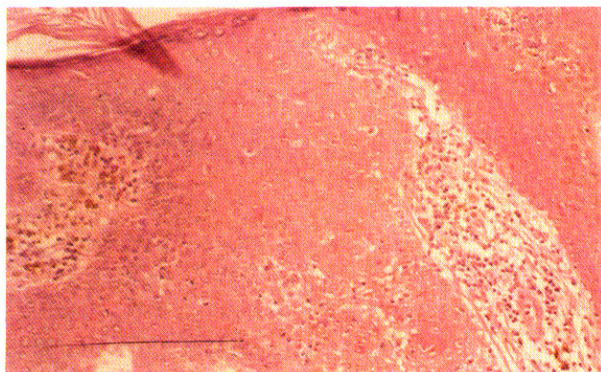


Fig. 3. Exocytosis, and spongiosis were observed in some foci of the lesion. A diffuse dense mononuclear cell infiltrate with increased content of melanophages was seen beneath the tumor (H&E, $\times 100$).

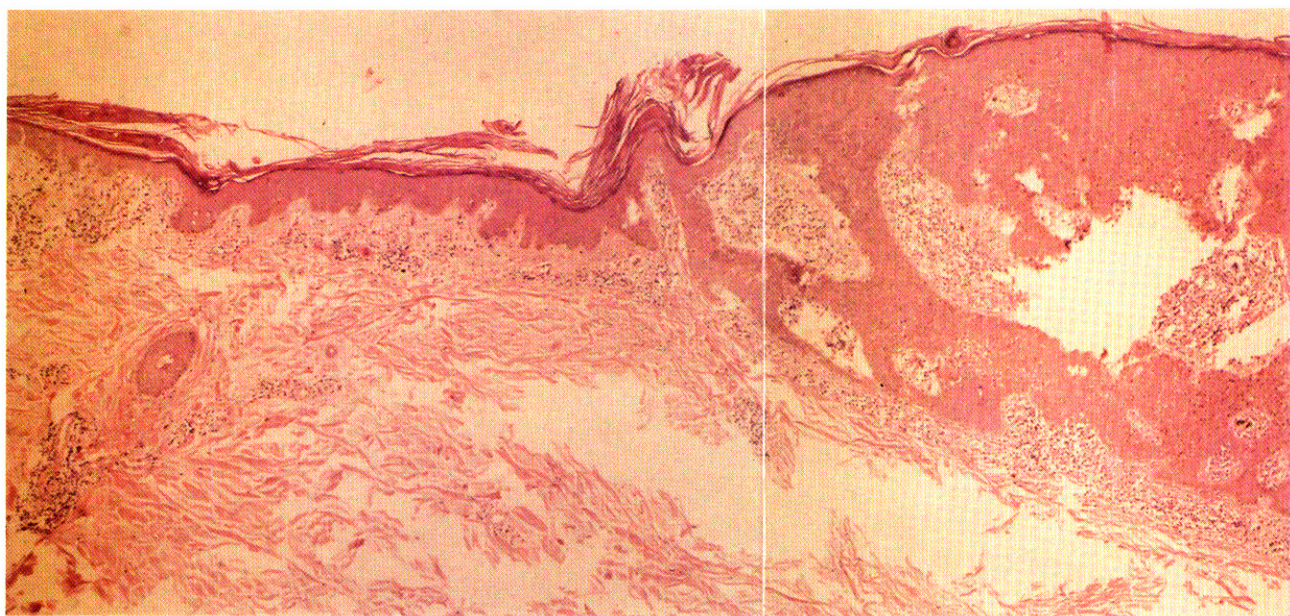


Fig. 2. The right half of the picture, showing a greatly thickened epidermis composed of basaloid cells with mild hyperkeratosis and papillomatosis, is the central black papule. The left half, showing dilated capillaries and perivascular infiltration of mononuclear cells in the upper and mid-dermis, is the surrounding halo dermatitis (H&E, $\times 40$).

DISCUSSION

Halo dermatitis around tumors might be a distinct disease process quite different from that of Sutton's halo nevus. It is pruritic, common in summer, and found more frequently in males, with a male: female ratio of 2.7:1. It also appears to affect a relatively young age group (average age 30 years). It has a rapid onset of 1-6 weeks and sudden change^{2,4}.

Clinically, halo dermatitis is manifested by the appearance of an itchy erythematous focally crust-

ed symmetric zone around the central lesion. In contrast, classical Sutton's halo nevi are surrounded by an area of depigmentation that usually is asymptomatic, and are not associated with clinical evidence of inflammatory changes. The central nevus of halo dermatitis persists despite resolution of the dermatitis either spontaneously or following the use of topical steroids, in contrast to their usual regression in traditional halo nevi^{4,8}.

Histologically, case of halo dermatitis showed focal parakeratosis, punctated crusts, variable

amounts of spongiosis with focal microvesciculation, epidermal hyperplasia often of a psoriasiform type, and a moderately dense cellular infiltrate in the upper part of the dermis. The infiltrate was mostly perivascular and composed mostly of lymphocytes and histiocytes. The inflammatory cells were present between nests of cells of the nevi but were not in actual contact with them, and cytotoxic changes in the central nevus cell nests were absent. Eosinophils were found regularly in the dermal inflammatory reaction. In contrast, classical Sutton's halo nevi showed a predominant lymphocytic inflammatory reaction that might have a lichenoid pattern at the epidermal interface^{4,8}. In addition, T-lymphocytes of the infiltrate in halo dermatitis were mostly T₄ (+) as opposed to previous findings where a relatively large number of cytotoxic/suppressor T cells were reported in halo nevus³. The appearance of eosinophils is an interesting feature and may imply a hypersensitivity phenomenon².

The actual contact of the inflammatory cells with tumor nests in this case would be explained by the fact that the central lesion of seborrheic keratosis showed eczematous pattern of seborrheic keratosis⁹. Because not all cases of halo dermatitis showed a regular presence of eosinophils in the dermal infiltrate⁸, absence of the feature in this case seemed to be feasible. Other clinical and microscopic features were consistent with those of halo dermatitis previously noted.

The exact nature of the halo phenomenon still remains obscure. It has been suggested that it could be related to subacute allergic dermatitis⁸, nummular eczema^{4,7} or pityriasis rosea^{1,5}. In terms of subacute allergic dermatitis, Herrera et al³ observed negative results for expression of interleukin 2 receptor on T lymphocytes, whereas T lymphocytes bearing interleukin 2 receptor have always been found in the acute phase of allergic contact dermatitis. Nummular eczema might be a cause of this phenomenon. But, the lesion of nummular eczema was not found in other sites

at that time or during the follow up period⁶. Our case did not show evidence of nummular eczema either. Koebner's phenomenon in pityriasis rosea was also suggested in some patients having halo dermatitis^{1,5}, but the concomitant association was not found frequently. It would be feasible to suggest that it might be a manifestation of an allergy to unknown causes of dermatitis-provoking factors, such as the central lesion itself or minor trauma to the lesion⁶.

It is of interest that there was no reference to this condition in major dermatology textbooks and only a few reports in the literature. But this phenomenon could be more common than the sparse documentation would suggest, and this phenomenon could occur in diseases other than pigmented nevi. Although the exact nature of halo phenomenon is unclear, it could be a eczematous condition which might have a dermatitis provoking factor.

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