

A Case of Alopecia Universalis without the Involvement of Scalp Hairs

Kyeong Han Yoon, Seung Hun Lee, Sung Ku Ahn¹ and Won Soo Lee¹

A case of alopecia universalis in a 45-year-old male was reported. The hair loss initiated on the eyebrows and progressed to the whole body, but the scalp hairs were well preserved. Histopathologic features of eyebrows were compatible findings with alopecia areata. This is a unique case of alopecia universalis without any involvement of scalp hairs.

Key Words: Alopecia universalis, scalp hair uninvolved

Alopecia areata is a disease of an insidious patch, or discoid hair loss usually affecting the scalp; however, sites such as the eyebrows, as well as other body sites, may also be involved even though the scalp is not (Bertolino, Freedberg, 1993). The presence of exclamation mark hairs is a pathognomonic manifestation (Nancy, 1987; Bertolino, Freedberg, 1993). Symptoms associated with alopecia areata; nail changes, cataracts, atopic dermatitis, Down syndrome, thyroid diseases, vitiligo, pernicious anemia, Addison's disease, and other endocrine or autoimmune diseases. Alopecia areata progresses to alopecia totalis or alopecia universalis (Nancy, 1987) in one to seven percent of cases. In those cases, the symptoms are even more prominent (Demis, Weiner, 1963; Nancy, 1987).

The histopathology of alopecia areata varies depending on the duration of the disease. In acute cases, lymphocytes predominantly infil-

trate around hair bulbs and are arranged like a "swarm of bees" (Demis, Weiner, 1963; Lever, 1990). In addition, mast cells, plasma cells and occasionally eosinophils are observed (Bergfeld, 1990). Late findings include miniaturization, reduced numbers of hair follicles, and chronic inflammatory infiltrate with increased eosinophils and mast cells (Lever, 1990; Bergfeld, 1992; Pinkus, 1980).

REPORT OF A CASE

A 45-year-old man visited our department because of the loss of the eyebrows for 7 years. He had been diagnosed with occipital alopecia areata which was completely treated about 10 years ago. Physical examinations revealed almost complete hair loss with some broken strands on the whole body except the scalp (Fig. 1). Several pea-sized erythematous papules of indefinite duration were noted near both lateral canthi. Chest radiography, electrocardiogram as well as laboratory tests including a complete blood cell count, serum electrolytes, VDRL, and urinalysis tested within normal limits or negative. However SGOT, SGPT, and γ GT measured 98 IU/L, 170 IU/L, and 211 IU/L respectively. Serum HBs antigen and anti-HBc antibody were negative, but

Received December 5, 1994

Accepted February 22, 1995

Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea

¹Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea

Address reprint requests to Dr. KH Yoon, Department of Dermatology, Yonsei University College of Medicine, C.P.O. Box, Seoul 120-752, Korea

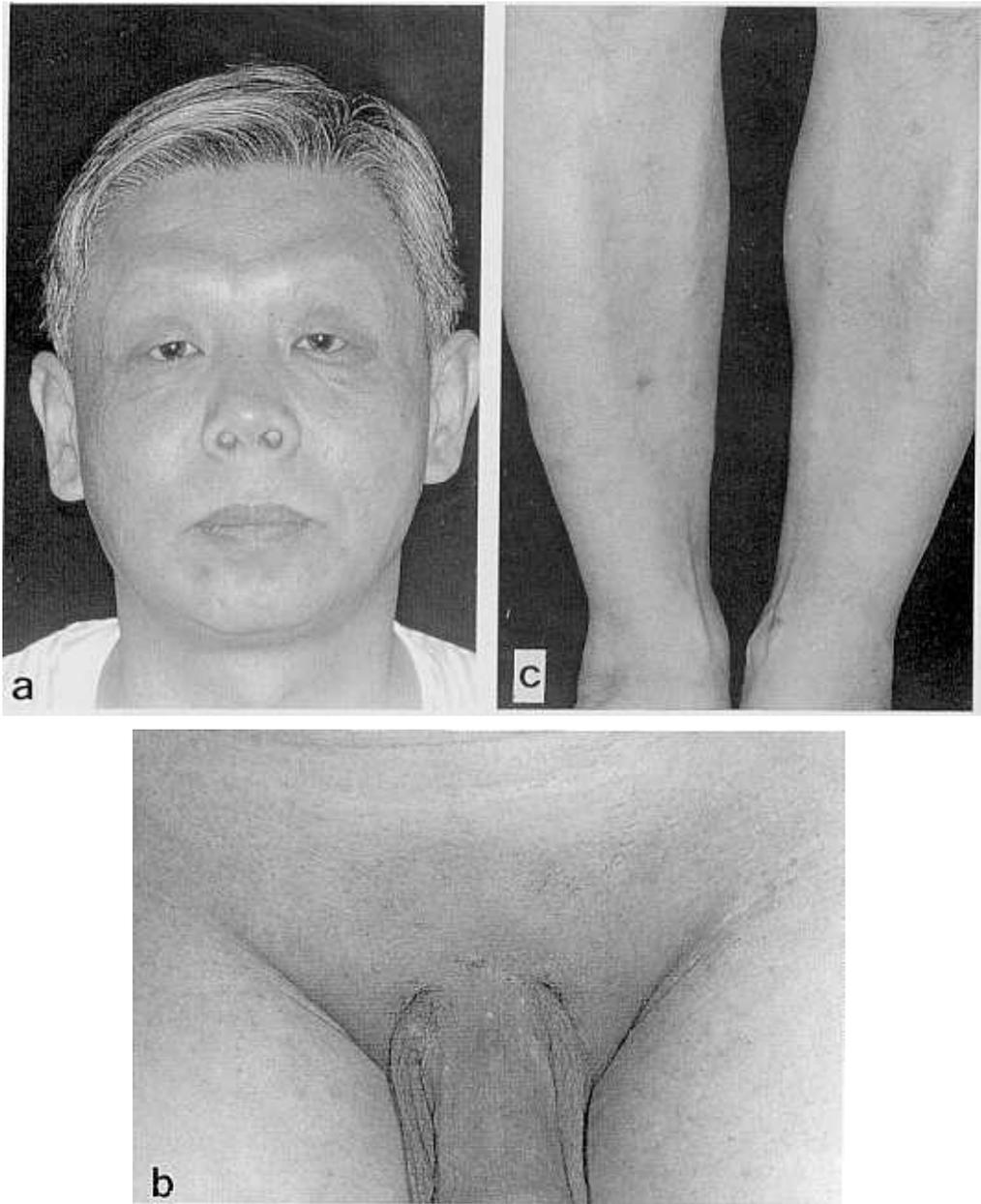


Fig. 1. The hairs on eyebrows and barbae are lost, but scalp hairs are preserved. Erythematous papules are also observed on lateral epicanthus (a). Hairs on whole body are lost including the genitalia (b), and extremities (c).

anti-HBs and anti-HCV antibodies were positive. A liver biopsy demonstrated a histologic pattern resembling chronic active hepatitis. Wade smears performed from both eyebrows

and ear lobes to rule out leprosy, and did not show acid-fast bacilli. Incisional skin biopsies were performed on the lateral margin of the left eyebrow and a papule near the left later-

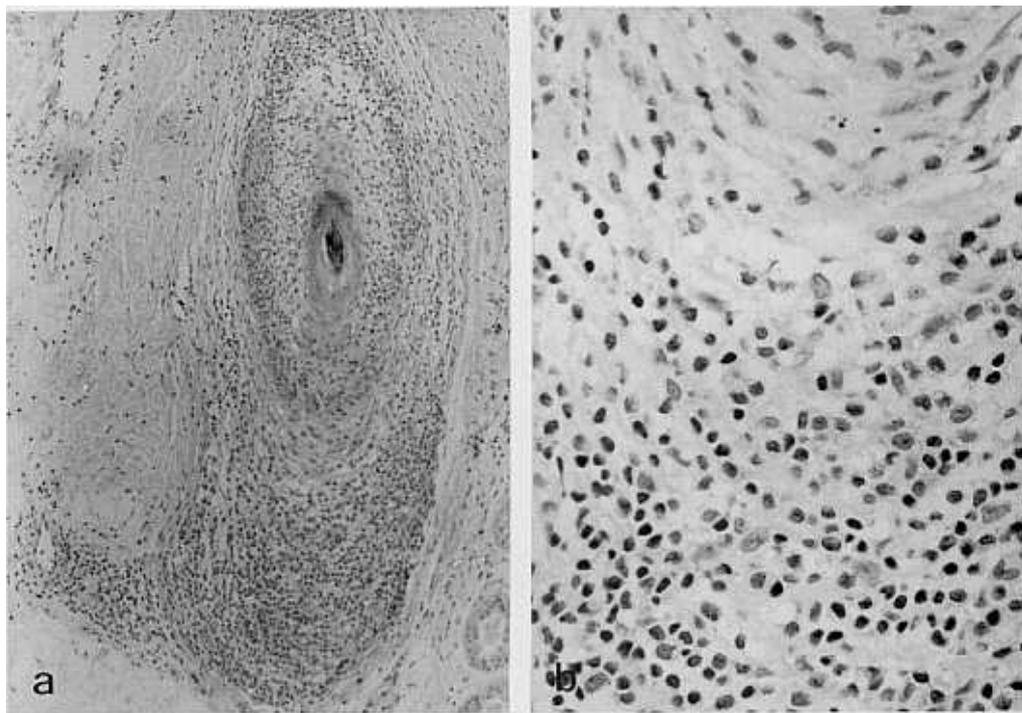


Fig. 2. The dermis around the hair bulbs but not the follicular walls are infiltrated with inflammatory cells (a, H&E, $\times 100$), which are composed largely of lymphocytes and some eosinophils (b, H&E, $\times 400$).

al canthus.

Histopathologic findings of the alopecic patch on the eyebrow showed that the dermis surrounding the hair follicles was infiltrated with lymphocytes (Fig. 2). The infiltrate was more densely aggregated around hair bulbs, yet had not invaded the follicular walls. There were some eosinophils and neutrophils intermixed within the infiltrate.

Histopathologic tests on the lateral epicanthus papule revealed a moderate to dense cellular infiltrate, composed of neutrophils, lymphocytes, and many eosinophils in the dermis. AFB staining on both specimens revealed negative findings. The papules on the face spontaneously disappeared 2 weeks after the first visit. Only the axillary hairs began to regrow with the application of diphencyclone.

DISCUSSION

This case is characterized by a generalized hair loss except for scalp hairs. The initial hair loss began with the eyebrows, and progressed to the whole body. Within a year, some erythematous papules developed on the face. Thus, leprosy was considered as a possible diagnosis (Arnold, 1976; Binford *et al.* 1982). However anesthesia, a major symptom of leprosy, was not present, and Wade smear results were negative. The histopathologic findings of a facial papule were not indicative of leprosy, and the papules spontaneously disappeared without any sequelae within a few weeks.

Alopecia areata is thought to be characterized by a sudden precipitation of anagen stage hair follicles into early catagen (Tobin *et al.* 1990). The anagen phase of human scalp hairs

is quite long, and the approximate speed of growth is much faster than on any other body site (Orentreich, 1969). Thus the scalp hairs are more prone to develop alopecia areata than hairs of other body sites. However even when the scalp is not involved, alopecia areata of any body site occurs in about 5% of all cases. (Gollnick, Orfanos, 1990; Bertolino, Freedberg, 1993).

The advent of hair loss in this case was initiated on the eyebrows. Broken hairs were found on the eyebrows. Histopathologic studies on the alopecic eyebrows were similar with findings of alopecia areata in a later stage. Although there are some reports of alopecia areata which started in areas other than the scalp and progressed to alopecia totalis or alopecia universalis, all of these cases inevitably involve the scalp hairs at some point (Hull *et al.* 1991). Hence, this case seems to be very peculiar in that the scalp hairs were well preserved, while the body hairs were almost completely lost.

Follicles, in particular the cells of the dermal papilla and dermo-epidermal junction of the hair follicle bulb, producing normal terminal hair in uninvolved, normal areas of alopecia areata scalps show a subclinical state of alopecia areata, both immunohistopathologically and electronmicroscopically (Morris, 1955). Thus, it can be proposed that some unexplained local factors act on hair follicles in subclinical states, which attributes to the apparent hair loss. In this case, we did not examine the scalp hairs, and the reason for the scalp hair preservation remains unclear. But this case can provide a good model for studies concerning local factors involved in the pathogenesis of alopecia areata, such as antigenic differences in hair follicles which attribute to, or resist against hair loss.

Aside from occasional nail changes in 10 to 20 percent of patients, there are no other systemic manifestations which regularly accompany alopecia universalis (Farner, 1958), but with which many systemic diseases can be combined (Nancy, 1987). In this case chronic active hepatitis due to hepatitis C virus (HCV) was also present, but it is unclear if there is a concrete relationship between

alopecia areata and hepatitis, since there are no previous reports concerning this issue. The papules on both lateral canthi are thought to be incidentally combined insect bites according to the clinical and histopathologic findings and the clinical course (Barnard, 1966). Despite the fact that infectious agents have never been identified in alopecia areata, some type of infection as a local or systemic trigger mechanism cannot be excluded and could provoke some imbalance of the T cell subpopulations (Gollnick, Orfanos, 1990). Therefore, it would be possible that either or both of the HCV infection and multiple insect bites could act as trigger factors for the hair loss, and thus the loss of total body hair might be more easily induced.

In this paper, we report a unique case of alopecia universalis initiated in the eyebrows and which progressed to the hairs on the whole body without involvement of the scalp hairs.

REFERENCES

- Arnold HL: Leprosy, Differential diagnosis. *Cutis* 18: 53-59, 1976
- Barnard JH: Cutaneous responses to insects. *JAMA* 196: 259-262, 1966
- Bergfeld WF: *Inflammatory reactions of the pilosebaceous unit*. In Farmer ER, Hood AF, ed. *Pathology of the skin*. Connecticut, Appleton & Lange, 1990, 934-939
- Bergfeld WF: *Hair disorders*. In Moschella SL, Hurley HJ, ed. *Dermatology*. 3rd ed, Philadelphia, WB Saunders company, 1992, 1541-1560
- Bertolino AP, Freedberg IM: *Hair*. In Fitzpatrick TB, Eisen AZ, Freedberg IM, Austin KF, ed. *Dermatology in general medicine*, 4th ed, New York, McGraw-Hill, Inc., 1993, 671-695
- Binford CH, Meyers WM, Walsh GP: Leprosy. *JAMA* 247: 2283-2292, 1982
- Demis DJ, Weiner MA: Alopecia universalis, onychodystrophy, and total vitiligo. *Arch Dermatol* 88: 131-137, 1963
- Farner BR: Alopecia universalis. *JAMA* 168: 1837, 1958
- Gollnick H, Orfanos CE: *Alopecia areata, pathogenesis and clinical picture*. In Orfanos CE, Happle R, ed. *Hair and hair diseases*. Berlin, Heidelberg,

Alopecia Universalis with Scalp Hair Uninvolvement

- Spinger-Verlag, 1990, 529-569
- Hull SM, Nutbrown M, Pepal L, *et al*: Immunologic and ultrastructural comparison of the dermal papilla and hair follicle bulb from active and normal areas of alopecia areata. *J Invest Dermatol* 96: 673-681, 1991
- Lever WF, Schaumburg-Lever G: *Histopathology of the skin*. 7th ed. Philadelphia, JB Lipincott company, 1990, 223-224
- Morris L: Progression of alopecia areata through alopecia totalis to alopecia generalizata. *Arch Dermatol* 71: 648-649, 1955
- Nancy BE: Alopecia areata symposium. *Ped Dermatol* 4: 136-158, 1987
- Orentreich N: *Scalp hair replacement in man*. In Montagna W, Dobson RL, ed. *Hair growth*. Oxford, Pergamon, 1969, 99-108
- Pinkus H: Alopecia, clinicopathologic correlations. *Int J Dermatol* 19: 245-253, 1980
- Tobin DJ, Biol GI, David A, *et al*: Ultrastructural observations on the hair bulb melanocytes and melanosomes in acute alopecia areata. *J Invest Dermatol* 94: 803-807, 1990
-