Alopecia Areata: Clinical Aspects

Vera H. Price
Department of Dermatology, University of California, San Francisco, U.S.A.

Alopecia areata is a common disease of the hair follicle affecting about 2% of new patients attending dermatology clinics in the United States and in Britain. A 25-year epidemiological survey in Rochester, Minnesota, showed an incidence of 17.2 per 100,000 per year, or, stated differently, by the age of 50 years about 1% of us will have had a patch of alopecia areata (AA) at some time in our lives.* In alopecia areata the hair follicle, in response to some unknown signal or injury, is suddenly precipitated into premature telogen, and then cycles in a shortened aborted cycle in which it is repeatedly arrested part way through early anagen. The follicle remains in this arrested hibernation-like state and is fully capable of resuming normal growth after months or years. The nature of the noxious signal or injury and the anatomical target for this assault are unknown, and it has been assumed in the past 20 years or so that an autoimmune process is involved. Because the follicles are not destroyed in AA but only switched off, the mechanism in AA may be affecting a control switch of the hair cycle, or some growth-controlling factor or its receptor.

The clinical hallmark of these events, the sudden onset of a round or oval patch of complete hair loss, is seen worldwide. Any hair-bearing site may be affected and the site bears no prognostic significance. AA affects males and females equally and strikes all ages although it is more common in children and young people. Approximately 60% of patients develop alopecia areata before the age of 20 years. A family history of alopecia areata is present in about 20% of patients, and there are reports of monozygotic twins developing AA simultaneously. Immunogenetic studies have so far shown inconsistent findings but we need further HLA studies in well-defined clinical groups and in families having affected members in several generations.

The hair bulb melanocytes are another target for the pathogenic mechanisms. Pigmented hairs are often shed while the unpigmented or white hairs are spared, and new regrowth is often unpigmented, later darkening to its normal shade.

The course of AA is capricious and unpredictable. It may be an acute event, with the few patches regrowing spontaneously within a year. It may have a chronic course with regrowth in some sites and progression to new sites for years. And it may be fulminant, progressing from patchy to 100% scalp hair loss (alopecia totalis) or total body hair loss (alopecia universalis) in a few weeks. Approximately 1–2% of patients develop the latter two extensive forms. About 20% have persisting, unrelenting disease after 10 to 15 years with no sign of regrowth. Nevertheless, the possibility for hair regrowth remains, and patients with alopecia totalis or alopecia universalis may have complete remission, even after many years.

Two controversial aspects of AA are the role of infection and stress. Microbial agents have so far not been identified in the lesions. Nevertheless a microbial agent could be a trigger factor in some cases, provoking an imbalance of immune responses. Immunohistochemical and electron microscopic studies are needed to look for evidence of viral damage of follicle cells. Severe psychologic stress may alter immune function and could serve as an inciting factor in some patients. However, a large recent survey showed no severe stressful event associated with the onset of AA in the majority of participants, and the association is probably an uncommon event.†

The etiology of AA is still entirely unknown. One basic question is whether AA is caused by one of several triggering factors in a genetically susceptible person, or caused by one etiological factor that unleashes an inflammatory response against the anagen follicle. In either case, the disease activity appears to be directed against a control switch of the hair cycle, or a growth-controlling factor or receptor. One clue to the faulty switching mechanism may be provided by three drugs, minoxidil, diazoxide, and pinacidil, all potassium channel openers that might stimulate hair growth by modulating the hair cycle.

AA is an ideal disease for study: the target organ is visible and accessible. We can witness the onset, remissions, and relapses of the disease. However, its wide clinical variability is a potential pitfall for investigators. When designing studies we must specify whether patients have active hair loss or regrowth, and whether studied lesions are in the center of established patches or at the spreading margins. Finally, patients with patchy AA ought to be evaluated separately from those with 100% scalp hair loss, because the latter have a more resistant form of the disease.

GENERAL REFERENCES

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Reprint requests to: Dr. Vera H. Price, Kaiser Permanente Medical Center, 2350 Geary Boulevard, Room 213, San Francisco, CA 94115.

* Muller SA: Incidence of alopecia areata (unpublished data).

† Koo J (personal communication).

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