



RENBÖK PHENOMENON IN A PATIENT WITH ALOPECIA AREATA ALONG WITH PSORIASIS

Dermatology

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ABSTRACT

The Renbök phenomenon/reverse Koebner phenomenon designates the withdrawal of a lesion when a different one appears. The mechanism underlying the Renbök phenomenon is not entirely clear, although cross reaction between cytokines is hypothesized to play a prominent role in the competition between the two overlapping immune responses. We report a case of Renbök phenomenon with rapid development of two dermatoses; Alopecia Areata and Psoriasis.

KEYWORDS

Renbök phenomenon, alopecia areata, psoriasis, immune system.

INTRODUCTION:

Phenomenon literally means a fact or situation that is observed to exist or happen, especially one whose cause or explanation is in question. In clinical practice any sign or objective symptom or any observable occurrence or fact is defined as phenomenon. In context of dermatology, the term has been applied to several peculiar clinical occurrences or laboratory observations.¹ The Renbök phenomenon (Inverse Koebner phenomenon) was first reported by Happle et al. in 1991.² Renbök phenomenon was first described in a patient suffering from two autoimmune diseases concomitantly, i.e. psoriasis and alopecia universalis.¹ The phenomenon relates to sparing among two concomitant active diseases often with autoimmune etiology.² The term was later extended to include patients with mosaic phenomena, one with alopecia areata (AA) without a nevus flammeus, and another without a congenital nevus. Whereas the Koebner phenomenon involves psoriasis of traumatic or inflammatory origin, the Renbök phenomenon (RP) occurs when some inflammatory process is inhibited by psoriasis.³ Psoriasis can often coexist with AA. Reports on psoriasis-induced Renbök phenomenon in AA have been exceedingly sparse and do not demonstrate the interchanging nature of these two disorders.⁴

CASE REPORT:

This 28-year-male, student by occupation developed small single asymptomatic patch of alopecia areata over the scalp that enlarged to the present extent over a period of 2 months. One month later patient experienced scaling over the entire scalp except at the site of patch of alopecia areata. Cutaneous examination showed the diffuse involvement of the scalp with erythematous indurated scaly plaques with single well defined patch of non-scarring hair loss and dermoscopic examination revealed perifollicular pigmentation, exclamation hairs and yellow dots corresponding to AA. Interestingly, there was no hair loss within the plaques of psoriasis, showing that the psoriatic plaque engulfed the AA area, but sharply stopped at its border. The patient was treated with topical medications only, for both alopecia areata and scalp psoriasis and showed good response to treatment.



(a) A 2.5-cm diameter patch of hair loss on the scalp.



(b) Thick psoriatic plaques shown adjacent to the area of hair loss, but sharply stops at its border.

DISCUSSION

Co-localization of diseases has always remained elusive and a puzzle, difficult to unscramble especially in dermatology. Co-localization of two rare and disparate dermatoses has been reported on several occasions in the past.⁵ AA is well known as an autoimmune T-cell mediated disease, however its exact pathogenesis is still obscure. Although it has been reported to occur in association with other autoimmune disorders, such as vitiligo, co-localization of these two disorders remains a rare clinical entity, co-localization with psoriasis, another autoimmune disease, is also rare, and usually there is remission of AA in psoriatic plaques, the so called Renbök phenomenon.⁴ In addition to development of Renbök phenomenon in two patients with genetic mosaicism, there are other case reports that seem to fit in the Renbök phenomenon including diltiazem-induced morbilliform drug eruption that spared seborrheic keratoses, widespread maculopapular drug reaction to phenobarbital that spared nevus depigmentosus, guttate psoriasis sparing Becker's melanosis and widespread drug reaction that spared psoriatic plaques.² The phenomenon represents a natural experiment in which distinct but overlapping immune responses favored psoriasis or contact dermatitis, halting the development and progress of AA.¹ Although recent advances in the pathogenesis of AA and psoriasis points towards the possibility that changes in the local cytokine milieu based on the presence of different T-cell populations result in interchange between these two disorders.⁴ Psoriasis is well known to be a T-cell-mediated chronic inflammatory disorder, while alopecia areata as a B-cell-mediated autoimmune condition.¹ The T helper 17 psoriatic inflammation antagonizes the T-helper 1 AA inflammatory response, presumably through cytokines, as each inflammatory pathway increases its own response and inhibits opposing pathways, therefore preventing the co-existence of two different diseases in the same location.² In this patient, it was observed that psoriatic plaques on the scalp retained hair follicles, whereas single patch of hair loss was evident elsewhere showing that clinically active psoriasis suppressing alopecia areata, thereby limiting alopecia areata to a single patch in our patient as he had diffuse involvement of the scalp with scalp psoriasis.

Our patient is unique in that it represents a case of rapid development of two dermatoses one after the other. In a short time frame of 3 months, the patient developed two disorders with distinct pathogenesis and different cytokine milieu on scalp sparing each other's territory.

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CONFLICT OF INTEREST: None declared

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