Psoriatic Scarring Alopecia

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Psoriasis affects approximately 0.123% of the population in China. Scalp is the first site of involvement in nearly 50% of the patients. Hair loss is a possible feature in psoriasis of any type. In 1972, Shuster has first distinguished three types of psoriatic alopecia: circumscribed alopecia, diffuse alopecia, and scarring alopecia. Psoriatic alopecia was found to be circumscribed in 75% of the cases and diffuse in 25%. Scarring alopecia is a rare form, which most often occurs in areas of long-standing lesions. We herein report two cases of psoriatic scarring alopecia and discuss the differential diagnosis of hair loss in psoriasis and the possible pathogenic role of T cells in psoriatic alopecia. (Dermatol Sinica 25: 48-52, 2007)

Key words: Psoriasis, Scarring alopecia

INTRODUCTION

Psoriasis is one of the most common chronic inflammatory skin disorders affecting about 0.123% of the population in China. It is characterized by complex tissue alterations including epidermal hyperproliferation and altered differentiation, angiogenesis and dilatation of dermal blood vessels, and a mixed inflammatory infiltrates of lymphocytes, neutrophils, dendritic cells, and mast cells. Nearly 50% of the psoriatic patients developed their first lesion on the scalp. Acute and chronic hair loss could characterize psoriasis of any type. Shuster first described three types of psoriatic alopecia: circumscribed alopecia, diffuse alopecia, and scarring alopecia. Alopecia was found in...
to be circumscribed in 75% of the cases and diffuse in 25%. Successful treatment of the psoriasis stopped the hair loss and cured most cases of alopecia. Scarring alopecia is a rare complication of scalp psoriasis which in most cases occurs in areas of long-standing lesions. We report two cases of psoriatic scarring alopecia: one in a patient with chronic plaque type psoriasis and the other in a patient with pustular psoriasis. Although the pathogenesis of psoriatic alopecia is still elusive, we will discuss the possible pathogenic role of T cells in psoriatic alopecia.

CASE REPORT

Case 1

A 57-year-old man has suffered from chronic plaque psoriasis for 28 years. He also has psoriatic arthropathy affecting both spinal and peripheral joints with joint deformity. One episode of pustular psoriasis had occurred. Chronic psoriatic plaques appeared on the scalp, the trunk, and the extremities. The scalp has been involved for 28 years. He has been treated with topical corticosteroids, oral retinoid, and non-steroid anti-inflammation drugs. About one month earlier, increased hair loss was noted on one irregular-shaped erythematous plaque on the vertex scalp (Fig. 1A). Skin biopsy from the periphery of the hairless plaque revealed typical features of psoriasis, including parakeratosis, regular acanthosis, and dilated capillaries in the dermal papillae (Fig. 1B). Besides, there were also a decreased number of hair follicles, inflammatory infiltrate of mononuclear cells around the infundibular and isthmic regions of the hair follicles, and atrophy of sebaceous glands (Fig. 1C). The laboratory study, including complete blood counts, antinuclear antibody, serology test for syphilis, and KOH examination didn’t reveal any abnormality. Despite of the continuous treatment with oral retinoid and potent topical steroids, no regrowth of scalp hair within the bald area was observed after a 3-year follow-up.

Case 2

A 27-year-old man presented with a 18-year history of recurrent generalized pustular psoriasis. Psoriatic lesions began on the buttocks, and spread to the trunk, the extremities and the scalp gradually. The scalp has been involved for 15 years. He has been treated with topical corticosteroids, topical calcipotriol and oral retinoid. Beginning approximately half a year earlier, the patient noticed increased hair loss in areas of long-standing psoriatic lesions on the scalp, progressing to several bean-sized bald macules. These bald macules gradually enlarged and coalesced into one irregular-shaped hairless patch on the vertex scalp (Fig. 2A). There was no active psoriasis in the hairless patch, but there was some erythema surrounding the hair follicles bordering the patch. In the area of alopecia, hair follicle markings were conspicuously absent and the skin

Fig. 1

(A) One irregular-shaped erythematous hairless plaque on the vertex scalp. (B) Parakeratosis, regular acanthosis, and dilated capillaries in the dermal papillae. (H & E, x 40) (C) Inflammatory infiltrate of mononuclear cells around the upper permanent portions of the hair follicles and atrophy of sebaceous glands. (H & E, x 100)
Fig. 2
(A) One irregular-shaped shiny hairless patch bordered by some perifollicular erythema on the vertex scalp. There were some tufting hairs in the patch. (B) The interfollicular epidermis was normal and no perifollicular inflammation was seen. There was also some perifollicular fibrosis. (H & E, x 40)

DISCUSSION

The presence of hair loss in psoriasis is now widely recognized and can be attributed to psoriasis per se, antipsoriatic drugs, or other concurrent diseases that could cause hair loss. The diagnosis of psoriatic alopecia is made when hair loss develops after the onset of scalp psoriasis and only in areas affected by psoriasis, and other causes for alopecia are excluded. It is accepted that psoriatic alopecia is a possible feature of any type of scalp psoriasis.

The diagnostic hallmarks of all forms of scarring alopecia are both visible loss of follicular ostia and destruction of the hair follicle on histopathologic examination. The bulge of the hair follicle is thought to be the reservoir of stem cells. Destruction of this critical element required for follicular reconstitution is thought to result in permanent alopecia. The histopathologic features of psoriatic scarring alopecia, besides the typical characteristics of psoriasis, include an accumulation of inflammatory cells around the upper permanent portion of the hair follicle, associated with progressive destruction of the follicular epithelium, atrophy or loss of sebaceous glands, and sometimes keratinized hair shafts lying free in the dermis and provoking a foreign body granulomatous reaction. In the end stage of psoriatic scarring alopecia, the follicular units are greatly decreased and replaced by fibrotic tracts. The epidermis is normal and no inflammatory infiltrates are detectable. The differential diagnosis of psoriatic scarring alopecia includes tinea capitis, syphilitic alopecia, antipsoriatic drug-induced alopecia, alopecia areata, pseudopelade of Brocq, and scarring alopecia due to other specific inflammatory skin disorders, such as discoid lupus erythematosus and lichen planopilaris. A negative special stain for fungus and a negative serology test for syphilis can help exclude tinea capitis and syphilitic alopecia. Retinoids and methotrexate are the commonest drugs responsible for drug-induced alopecia in psoriasis. The pathogenesis of hair loss caused by retinoids or methotrexate is probable premature entry of the hair follicle into telogen, which results in telogen effluvium. Drug-induced hair loss is usually reversible after interruption of treatment. Alopecia areata, discoid lupus erythematosus, and...
and lichen planus have been reported in association with psoriasis. In alopecia areata, the inflammatory infiltrate is predominantly around the lower follicle. In discoid lupus erythematosus, the inflammatory infiltrate is predominantly around the upper follicle and often with plasma cells, and there are also vacuolar interface dermatitis, dermal mucin deposition, and perieccrine inflammation. In lichen planopilaris, the inflammatory infiltrate is predominantly around the upper hair follicle, and there is lichenoid interface dermatitis without dermal mucin deposition and perieccrine inflammation.

Pseudopelade of Brocq is not a distinct disease but the end stage of various forms of scarring alopecia and a diagnosis of exclusion. Pseudopelade of Brocq is clinically manifested as irregular shaped and often widely distributed and grouped bald patches without marked clinical inflammation or folliculitis on the scalp. In most cases of pseudopelade of Brocq, the typical histologic findings are those of end-stage alopecia. If a definite diagnosis of another form of scarring alopecia can be made based on clinical, histologic or immunofluorescent features, then the term pseudopelade of Brocq should not be used. Although the clinical and pathological pictures of case 2 resemble pseudopelade of Brocq, the diagnosis of end-stage psoriatic scarring alopecia is made based on the history that the alopecia occurred in areas of long-standing psoriatic lesions.

It is unclear how psoriasis leads to alopecia. However, based on circumstantial evidence, two lines of argumentation can be delineated supporting a T-cell-based immunopathogenesis of psoriatic alopecia. First, a prominent follicular and perifollicular mononuclear cell infiltrate is seen in some patients with psoriatic alopecia reported in the literature as well as in our patient (case 1). This infiltrate presumably damages the hair follicles. Second, in areas representing the end-stage condition of scarring alopecia without viable hair follicles, psoriatic tissue alterations appear to cease in some patients reported in the literature as well as in our patient (case 2). This observation suggests that T cells in these cases recognize follicular antigens rather than antigens of the interfollicular epidermis, possibly resulting in diminished T-cell attraction to the skin in areas lacking hair follicles. The concept that T cells can attack hair follicles in some cases of psoriasis is supported by a T-cell-based animal model of hyperproliferative inflammatory skin changes resembling several histopathologic and pathophysiologic aspects of psoriasis. Briefly, reconstitution of 8 mice with minor histocompatibility-mismatched naïve CD4+ T lymphocytes resulted in skin alterations that strikingly resembled human psoriasis clinically, histopathologically, and in cytokine expression. Interestingly, in 2 of the 8 recipients (25%), focal alopecia was observed within areas of psoriasiform skin lesions. Immunohistochemical analysis revealed that hair follicles in these lesions, but not in control mice, were surrounded by a dense infiltrate of T cells, suggesting that T cells are the prime pathogenic factor for alopecia. Besides, recent studies consider psoriasis a T-cell-mediated disease that can be triggered by bacterial superantigens. It is possible that scalp bacterial infection in psoriatic patients may lead to stimulated T lymphocytes infiltrating the isthmic regions of hair follicles, thus inducing changes in hair stem cells not compatible with further hair cycles and resulting in scarring alopecia.

A specific hair trichogram has been proposed in psoriasis. Schoorl et al. have noticed the significant increase in the proportion of telogen and catagen hair in psoriatic plaques. Orfanos observed an increased number of dysplastic hairs, a diminished number of anagen hairs, and an insignificantly increased telogen ratio. Runne and Kroneisen-Wiersma found the telogen rate was pathologically elevated in those with hair loss. In our patient (case 2), an increased telogen/anagen ratio was observed by histologic examination. There are two possible explanation for this phenomenon. First, firm attachment of hair shafts to the hyperkeratotic plaque impedes the ability of telogen hairs to shed. Second, the perifollicular inflammatory
infiltrate probably cuts short the growth phase and cause earlier telogen formation.

Successful treatment of psoriasis usually stops the progress of psoriatic alopecia and cures the condition. According to the investigation by Runne and Kroneisen-Wiersma, 3 82.9% of the patients with circumscribed or diffuse psoriatic alopecia receiving local therapy showed a complete regrowth of hair. Recurrences of psoriatic alopecia are possible but seem to be rare. Only 2.4% of the treated patients had new psoriatic hair loss. By contrast, psoriatic scarring alopecia, like other scarring alopecia, is resistant to all medical therapy.

Accumulative studies have shown that psoriatic alopecia is a well-defined entity and probably is not so rare. Due to its good response to antipsoriatic therapy, the dermatologists should always take psoriatic alopecia into consideration when evaluating scalp diseases which coincide with circumscribed or diffuse alopecia either with or without scarring. Early treatment of scalp psoriasis is important to prevent the development of irreversible scarring alopecia.

REFERENCES