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Case Report

Familial coexistence of vitiligo and alopecia areata: successful treatment with intralesional corticosteroids

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ABSTRACT

Vitiligo and alopecia areata are autoimmune dermatological conditions that may coexist due to shared immunopathogenic mechanisms, although their occurrence within the same anatomical site is uncommon. We report the case of a 33-year-old Indian male with a familial history of autoimmune skin disease who presented with co-localized vitiligo and alopecia areata on the posterior scalp, with an additional vitiligo lesion on the left tibia. Vitiligo preceded the onset of alopecia areata within the same scalp lesion by approximately four months. Dermoscopic examination confirmed features of both conditions, including depigmentation with loss of pigment network and characteristic alopecia areata findings such as yellow dots, black dots, and exclamation mark hairs. The patient was treated with intralesional triamcinolone acetonide injections for the scalp lesion, along with topical corticosteroids, topical tacrolimus, minoxidil, and vitamin D supplementation. Marked hair regrowth and near-complete repigmentation of the scalp lesion were observed at follow-up. Notably, the vitiligo lesion on the tibia demonstrated significant repigmentation following intralesional corticosteroid therapy after limited response to topical treatment alone. This case highlights the rare co-localization of vitiligo and alopecia areata, supports a shared autoimmune pathogenesis, and suggests that intralesional corticosteroids may be an effective therapeutic option for selected localized vitiligo lesions, particularly when associated with alopecia areata.

Keywords: Vitiligo, Alopecia, Autoimmune

INTRODUCTION

Autoimmunity refers to the immune system's abnormal response against the body's own tissues. In dermatology, vitiligo and alopecia areata (AA) are two classic models of autoimmune skin disorders, each targeting distinct structures: melanocytes in vitiligo and hair follicles in AA.[1] Vitiligo is characterized by depigmented macules resulting from the immune-mediated destruction of melanocytes, primarily in the epidermis.

In contrast, AA presents with non-scarring hair loss caused by lymphocytic infiltration around hair bulbs in the deep dermis. Although they affect different cell types and skin layers, both conditions share several immunopathogenic mechanisms. These include CD8+ T-cell infiltration, activation of interferon-γ (IFN-γ) and interferon-α (IFN-α) pathways, and elevated levels of the chemokines CXCL9 and CXCL10. Additional shared pathways involve oxidative stress, autophagy dysfunction, type 2 cytokine signaling, and disruption of the Wnt/β-catenin pathway.[2]

Treatment strategies differ primarily due to the depth of inflammation. Vitiligo is typically treated with topical corticosteroids, calcineurin inhibitors, or narrow-band UVB therapy, which are effective due to the superficial location of melanocyte destruction in the epidermis. In contrast, alopecia areata requires treatments that target deeper dermal inflammation around hair follicles, such as intralesional steroids or contact immunotherapy with agents like DPCP. However, DPCP can induce depigmentation and is therefore unsuitable for vitiligo.[3]

The co-localization of AA and vitiligo within the same anatomical patch is rare but increasingly recognized as more than a coincidental finding.[4–6] This overlap syndrome highlights their shared autoimmune nature and presents unique diagnostic and therapeutic challenges.

In our case, both vitiligo and AA appeared within the same lesion, and intralesional corticosteroid therapy was initiated. Unexpectedly, repigmentation of vitiligo occurred more rapidly than hair regrowth, prompting the decision to extend intralesional steroid treatment specifically to the vitiligo lesion on the left shin of the tibia.

This observation contrasts with typical disease behavior, where AA often responds faster than vitiligo and may reflect differences in stem cell regeneration dynamics and tissue responsiveness.[7,8]

Various treatment combinations such as corticosteroids, JAK inhibitors, vitamin D analogs and phototherapy - have been used to improve therapeutic outcomes in vitiligo and AA.[1,3,9]

CASE REPORT

A 33-year-old Indian male presented with coexisting AA and vitiligo localized to the posterior scalp. The vitiligo lesion appeared first, approximately four months prior to presentation, followed by the development of alopecia areata in the same area. Additionally, a vitiligo patch has been present on the left shin of tibia for approximately one year.

The patient has a significant family history of the same condition; his brother also has combined vitiligo and alopecia areata.

On physical examination, a rounded patch of hair loss with milky white depigmented skin on the occipital scalp, along with an oval depigmented patch on the shin of the tibia. There were no signs of erythema, scaling or scarring.

Dermatoscopic evaluation of the scalp lesion revealed, central depigmented area with a milky-white background and absence of pigment network, consistent with vitiligo. Within this region, there is loss of hair, a few black dots, yellow dots, and numerous fine hairs on the peripheral border of the lesion, which are characteristic features of alopecia areata. The presence of white hairs (leukotrichia) further supports the diagnosis of vitiligo involving both epidermal and follicular melanocytes.

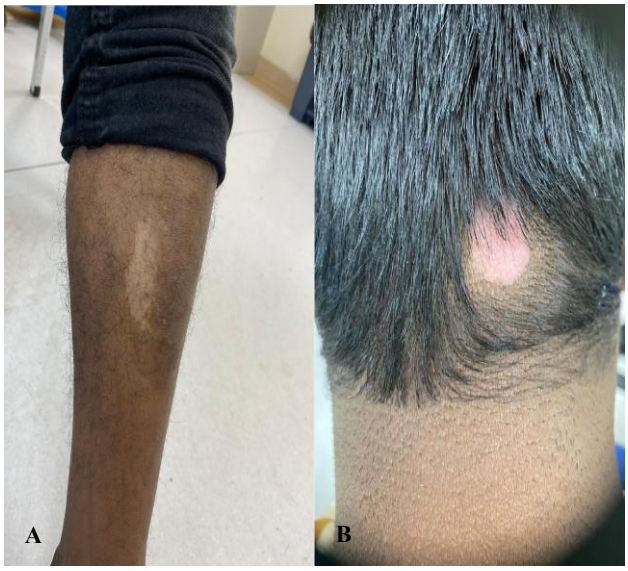

Figure 1 (A and B): Initial scalp lesion showing coexistent alopecia areata and vitiligo before treatment.

Initial laboratory investigations, including CBC, TSH, HbA1c, and vitamin B12, were within normal limits. Only the vitamin D level was borderline at 20 ng/ml. The patient was diagnosed with alopecia areata combined with vitiligo.

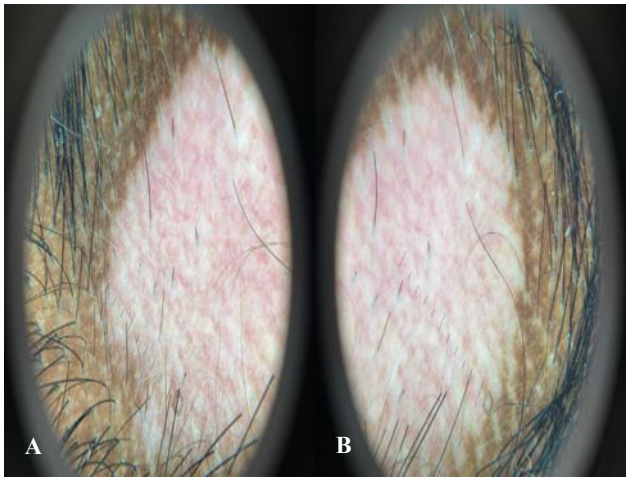

Figure 2 (A and B): Dermascopy of alopecia and vitiligo overlap in scalp patch.

The patient was treated for a scalp lesion in the clinic with an intralesional injection of triamcinolone acetonide (TAC) at a concentration of 5 mg/ml, followed by the topical application of clobetasol cream to the affected areas after one month. For the lesion of left shin of tibia, mometasone furoate 0.1% ointment was prescribed once

daily along with tacrolimus 0.1% ointment to be applied at bedtime for 3 months.

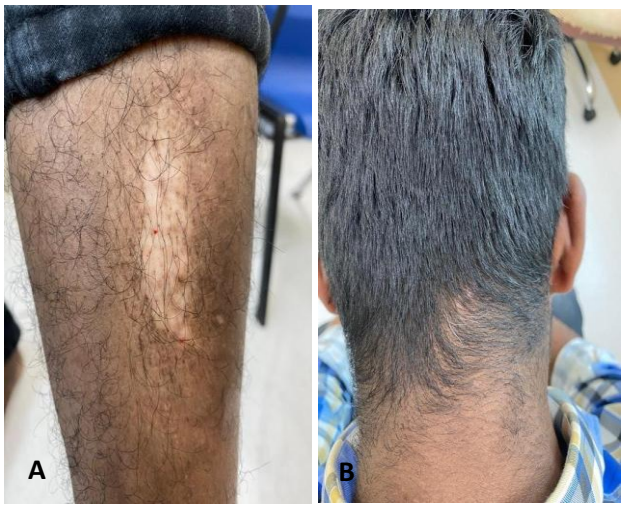

Figure 3 (A and B): Follow-up after intralesional triamcinolone showing hair regrowth and repigmentation.

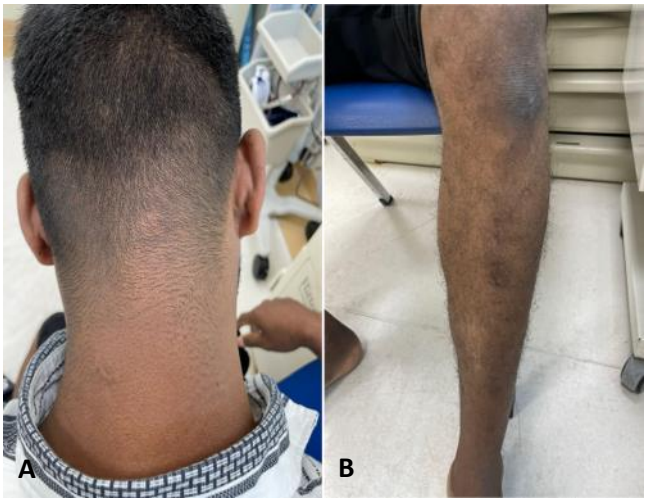

Figure 4 (A and B): Vitiligo lesion on the left tibia before and after intralesional corticosteroid injection.

To promote hair regrowth, minoxidil 5% solution was prescribed for twice-daily application to the scalp. Supplementation included vitamin D3 (cholecalciferol) at a dose of 50,000 IU weekly for two months, folic acid 1 mg daily, and multivitamins.

A follow-up visit was scheduled after 3 months to assess the clinical response. At the first follow-up, the scalp lesion was markedly improved with hair regrowth and nearly complete regimentation of the vitiligo. There was no significant improvement in the lesion on the right leg; therefore, intradermal Triamcinolone Acetonide (the dose: 2.5 mg/ml) was subsequently injected into the vitiliginous patches on the leg, 0.1 ml per cm^2 of the lesion, with further evaluation planned after one month. Laboratory investigations remained within normal limits.

At the second follow-up visit, the patient was re-evaluated in our clinic, and further improvement was observed. Notably, both the scalp and the tibial vitiligo lesions showed visible repigmentation, with the latter demonstrating a marked clinical response following the intralesional triamcinolone injection (2.5 mg/ml) to the left leg lesion.

The patient reported no adverse effects such as pain, atrophy, or dyspigmentation at the injection sites. Clinical photographs were obtained at this visit to document the progression and improvement in both lesions.

DISCUSSION

Vitiligo and AA are two distinct autoimmune skin disorders that may co-occur due to shared immunopathogenic pathways. The co-localization of both conditions in the same anatomical region, as observed in our patient's posterior scalp, highlights the complex interplay of immune mechanisms targeting melanocytes and hair follicles. This overlap has been increasingly recognized in the literature and supports the notion of a shared autoimmune diathesis.

Both conditions are mediated by CD8+ cytotoxic T cells, with prominent roles for interferon-γ (IFN-γ), interferon-α (IFN-α), and chemokines such as CXCL9 and CXCL10 in recruiting immune cells to target sites.2 Additionally, oxidative stress and dysregulated Wnt/β-catenin signaling contribute to tissue-specific destruction. Despite these similarities, the depth of inflammation differs; melanocyte destruction in vitiligo is limited to the epidermis, whereas AA involves deeper dermal infiltration around hair follicles.[7] This anatomical distinction explains the differences in treatment response and strategies. The familial occurrence of combined vitiligo and AA in our patient and his brother suggests a genetic predisposition. Previous studies have identified shared susceptibility loci and immune-related variants particularly within the HLA region as potential contributors to this overlap.[2] Furthermore, alterations in immune regulatory pathways and cytokine signaling further support a common genetic and immunological foundation for both disorders.[1] In most reported cases, alopecia areata precedes the onset of vitiligo.[4–6] However, in our patient, vitiligo developed first, followed by AA in the same region approximately four months later. This reverse sequence suggests that vitiligo may, in certain instances, act as a local initiator of autoimmune activity in the skin, creating a pro-inflammatory microenvironment conducive to subsequent follicular involvement.

In terms of treatment, our patient responded well to a combination of treatment for the scalp lesion, including intralesional corticosteroid injection, high-potency topical corticosteroids, topical minoxidil and vitamin D

supplementation. Notably, in addition to early signs of hair regrowth on the scalp, the vitiligo lesion on the shin showed visible repigmentation within one month, highlighting the efficacy of this combined approach even in extra-facial areas that typically respond more slowly. This suggests that preserved melanocyte reservoirs and a modulated immune microenvironment may enhance treatment responsiveness. Supporting these observations, Alshiyab et al demonstrated a significant difference in repigmentation response among treatment groups (p=0.017) following intralesional triamcinolone acetonide injections. The same study also found that epidermal thickness decreased (indicating atrophy) and collagen fibers became disorganized, particularly at concentrations of 2.5 and 5 mg/ml. These histological changes suggest that local tissue remodeling and immune suppression may play a role in facilitating repigmentation in vitiligo and hair regrowth in AA.[8] Although data on this specific combination therapy in AA remain limited, the shared autoimmune pathogenesis of these two conditions provides a strong rationale for such targeted immunomodulatory treatment.

Further supporting our observations, Jafarzadeh et al conducted a systematic review of 42 clinical studies and confirmed the efficacy of injectable corticosteroids - particularly compound betamethasone formulations - in vitiligo management. Injectable corticosteroids were shown to halt disease progression, promote repigmentation, and yield better outcomes when combined with topical agents. The authors attributed these effects to the immunosuppressive action of corticosteroids, which reduce cytotoxic T-cell activity, suppress antibody production, and promote melanocyte recovery.[10] Similarly, Wang et al reported favorable results with intralesional corticosteroid injections for localized vitiligo, further supporting their role as an effective therapeutic option for refractory lesions. Our follow-up observations confirmed sustained improvement in both alopecia areata and vitiligo lesions after targeted intralesional corticosteroid therapy, even in the lower limb lesion that initially responded poorly to topical therapy. This supports the therapeutic potential of localized intralesional corticosteroids in treating vitiligo patches associated with AA, with no notable adverse effects observed during the follow-up period.

CONCLUSION

This case highlights the rare coexistence of alopecia areata and vitiligo within the same anatomical region, illustrating the shared autoimmune mechanisms underlying both conditions. The familial occurrence suggests a possible genetic predisposition, reinforcing the importance of evaluating personal and family history in autoimmune dermatologic diseases. Early recognition and targeted therapy, including intralesional corticosteroid, may improve outcomes and provide insights into overlapping pathogenic pathways. This emphasizes the importance of more personalized therapeutic approaches.

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