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Single Case

Upadacitinib Monotherapy in Vitiligo Associated with Atopic Dermatitis: Killing Two Birds with One Stone

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Atopic dermatitis · Upadacitinib · Vitiligo

Abstract
Introduction: An increased risk of developing vitiligo has recently been described in patients with atopic dermatitis (AD). Vitiligo and AD can be associated because of shared pathogenetic pathways, including alterations in the Janus kinases/signal transducer and activator of transcription (JAK/STAT) signaling, suggesting JAK inhibitors as a promising new therapeutic approach in vitiligo. ***Case Presentation:*** We describe a 25-year-old woman diagnosed with AD since childhood and subsequent onset of slowly progressive vitiligo at the age of 16. Systemic therapy with JAK1 inhibitor upadacitinib 15 mg daily was started, after a medical and laboratory evaluation to exclude pregnancy and other contraindications. Progressive improvement of AD was observed after the first weeks of treatment with clinical remission at week 16. At the same time, clear improvement of vitiligo was observed with an almost complete remission achieved at week 28 of treatment. ***Conclusion:*** The remission of both AD and vitiligo achieved with upadacitinib monotherapy supports the therapeutic utility of inhibition of JAK 1 signaling in these patients.

Introduction

An increased risk of developing vitiligo has recently been described in patients with atopic dermatitis (AD) [1]. Vitiligo is a T helper 1 (Th1) immune-mediated disease characterized by patches of skin depigmentation, resulting from destruction of melanocytes. Recently, it has been

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hypothesized that Th2 and Th17 immune responses also play a role in inducing melanocyte dysfunction and skin inflammation. AD is a chronic, itchy inflammatory skin disease characterized by skin barrier dysfunction and activation of the Th2 axis. However, Th1, Th17, and Th22 cytokines are also variably involved, mainly in the chronic phase of the disease.

Vitiligo and AD can be associated because of shared pathogenetic pathways, including alterations in the Janus kinases/signal transducer and activator of transcription (JAK/STAT) signaling. The pro-inflammatory environment of AD would favor melanocytes destruction and the appearance of vitiligo, just as the chronic scratching present in AD could trigger the appearance of patches of vitiligo through the Koebner phenomenon [2]. The overlap of Th1 and Th2 immune responses in vitiligo could amplify melanocyte destruction and skin inflammation through JAK signaling, suggesting JAK inhibitors as a promising new therapeutic approach in vitiligo [3]. Upadacitinib, an oral selective JAK1 inhibitor, currently approved for the treatment of moderate/severe AD, has recently been proposed as an effective treatment in recalcitrant vitiligo [4].

Case Presentation

We describe a 25-year-old woman diagnosed with AD since childhood and subsequent onset of slowly progressive vitiligo at the age of 16. At baseline examination, the patient presented diffuse cutaneous xerosis, excoriated papules and plaques on the back, and lichenified erythematous lesions on the upper limbs and dorsum of the hands with an Eczema Area and Severity Index (EASI) of 18. The patient reported intense itching (Number Rating Scale [NRS] = 10). Confluent achromic macules suggestive of vitiligo were also present on the elbows (Fig. 1a) and on the face, predominantly on the eyelid region (Fig. 2a), bilaterally. The patient also showed multiple irregularly shaped achromic macules on the neck, trunk and dorsum of the hands, and initial hypopigmented manifestations on the left axillary region and left knee with a Vitiligo Area Scoring Index (VASI) of 0.45. Both skin conditions had an impact on her quality of life, resulting in a Dermatology Life Quality Index (DLQI) of 17.

Previous treatments for AD included topical and systemic corticosteroids, topical calcineurin inhibitors, antihistamines, and a short course of cyclosporine, which was discontinued due to intolerance. Previous treatment for vitiligo included short-term treatment with topical and systemic corticosteroids at disease onset and, for the first 2 years, without results. Subsequently, tacrolimus, a topical calcineurin inhibitor, was used for several years following a proactive pulsed maintenance regimen after an initial induction period, yielding minimal therapeutic benefits. Antioxidants and vitamin-based products were also used as adjuvant therapies. Due to logistical reasons, the patient refused phototherapy.

Given the coexistence of AD and vitiligo, and considering the potential additional benefits, systemic therapy with upadacitinib 15 mg daily was initiated after medical and laboratory evaluations to exclude pregnancy and other contraindications. Based on the approved indication of upadacitinib for AD, we opted to start treatment with a view to achieving the lowest effective dose.

Progressive improvement of AD was observed after the first weeks of treatment with clinical remission at week 16 (EASI 0; NRS 0) associated with improved quality of life (DLQI 0). At the same time, clear improvement of vitiligo was observed with complete repigmentation of the achromic areas on the neck, trunk, back of the hands, and elbows (Fig. 1b), and partial but significant repigmentation of the achromic patches of the eyelid region (Fig. 2b) (VASI = 0.25). An almost complete remission of vitiligo was achieved at week 28 of treatment and was maintained up to 1 year of follow-up, with no side effects except for a slight worsening of acne, easily controlled with topical therapies.

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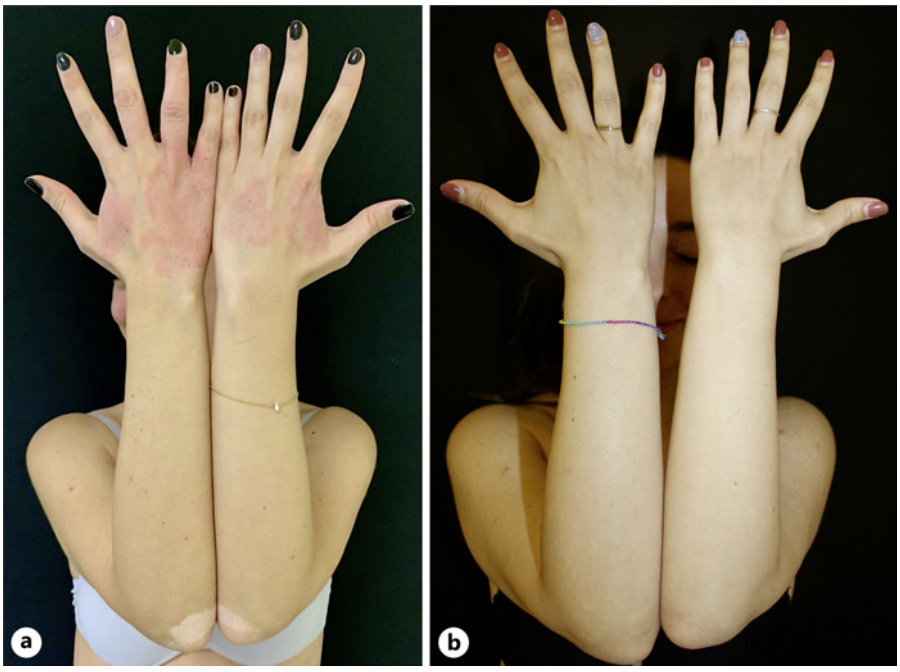

Fig. 1. Clinical image of the patient at baseline (**a**) and after 28 weeks of treatment with upadacitinib (**b**). Repigmentation of the achromic areas on the back of the hands and elbows.

Fig. 2. Clinical image of the patient at baseline (**a**) and after 28 weeks of treatment with upadacitinib (**b**). Repigmentation of the achromic patches of the eyelid region.

The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000544703).

Discussion

Upadacitinib has proven to be an effective and safe drug in the real-life setting for adult and adolescent AD [5], as well as for vitiligo (Phase II trials and real-life data) [6–8]. To date, only 1 case demonstrating an unexpected beneficial effect on vitiligo during treatment with upadacitinib for AD has been reported in the literature [9]; however, upadacitinib was administered in combination with topical crisaborole, a drug also used as monotherapy for vitiligo, introducing a confounding element in the evaluation of the efficacy of upadacitinib therapy. In our patient, treatment with upadacitinib monotherapy induced remission of both AD and vitiligo. Our case report also demonstrated that upadacitinib monotherapy was effective on vitiligo at a lower dose (15 mg) than that recently reported by Magdaleno-Tapial et al. [10], who described adult patients affected by vitiligo treated with upadacitinib 30 mg.

Inhibition of JAK1 signaling, shared in the pathogenesis of both diseases, may have a dual effect of relieving itching in AD and promoting skin repigmentation in vitiligo, potentially representing a valid therapeutic option in these patients. Further studies on larger series will, however, be necessary to confirm this preliminary evidence.

Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. Ethical approval is not required for this study in accordance with local or national guidelines.

Conflict of Interest Statement

L.M.M. and A.D.B. have no conflicts of interest to declare. M.E. has served as a speaker/board member for AbbVie, Almirall, Biogen, Celgene, Eli Lilly, Janssen, Leo Pharma, Novartis, Sanofi, and UCB. M.C.F. has served as consultant/advisor, received speaker honoraria and/or grants, and/or is investigator for AMGEN, Almirall, AbbVie, Boehringer-Ingelheim, BMS, Galderma, Kyowa Kyrin, Incyte, LEO Pharma, Pierre Fabre, UCB, Lilly, Pfizer, Janssen, MSD, Novartis, Sanofi, Regeneron, Sun Pharma, and Takeda.

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Author Contributions

L.M.M.: clinical support, literature review, and writing of the manuscript. A.D.B.: clinical support, clinical documentation, data management, and reporting. M.E. and M.C.F.: conception, clinical supervision, and literature review.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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