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Fast clinical response with topical ruxolitinib in the management of non-segmental vitiligo detected by VISIA® analysis system

Alberto Di Caro,[1] Maria Elisabetta Greco,[1] Francesca Paola Sasso,[1] Francesca Feresin,[1] Ilaria Proietti,[2] Giovanni Pellacani,[1] Steven Paul Nisticò,[1] Annunziata Dattola[1]

[1]Dermatology Clinic, Department of Clinical Internal, Anesthesiological and Cardiovascular Sciences, Sapienza University of Rome; [2]Dermatology Unit "Daniele Innocenzi", ASL Latina, Department of Medico-Surgical Sciences and Biotechnologies, Sapienza University of Rome, Latina, Italy

Correspondence: Alberto Di Caro, Dermatology Clinic, Department of Clinical Internal, Anesthesiological and Cardiovascular Sciences, Sapienza University of Rome, 00185 Rome, Italy. E-mail: alberto.dicaro@uniroma1.it

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Dear Editor,

Vitiligo is an acquired, chronic depigmenting disorder characterized by the progressive loss of epidermal melanocytes. Leukotrichia, present in approximately 10% to 60% of cases, results from the loss of melanocytes within the hair follicles. Clinically, it manifests as well-demarcated, non-scaling depigmented macules that may coalesce and expand over time.[1] While the condition is not life-threatening, its psychological and social impact, particularly in cases with visible involvement, such as the face or hands, can be substantial and often disproportionate to the extent of skin involvement.[2] Although the pathogenesis of vitiligo is multifactorial, increasing evidence supports a central role of autoimmune mechanisms, including the activation of cytotoxic $CD8^+$ T cells and the interferon-γ–mediated Janus kinases/signal transducers and activators of transcription (JAK/STAT) signaling pathway.[3] With a global prevalence of up to 2%, vitiligo poses a therapeutic challenge, especially in patients with extensive involvement or poor response to conventional treatments.[4]

We present the case of a 64-year-old female patient, phototype III according to the Fitzpatrick classification, with extensive non-segmental vitiligo affecting her face, neck, trunk, and upper extremities. The first manifestation appeared at the age of 40 and gradually progressed despite multiple treatment attempts. Her past medical history was notable for type 2 diabetes mellitus and essential hypertension, both well controlled with oral medications (metformin). The facial involvement was especially noticeable, with near-complete depigmentation of the forehead and central facial region, and confluent lesions affecting the periorbital and perioral areas. A distinctive hyperpigmented border was observed along the margins of the lesions on both cheeks. Notably, there was no leukotrichia in the affected areas, a finding that is associated with a better response to treatment due to the preserved follicular melanocyte reservoir. This distribution pattern significantly contributed to the patient's psychological distress and social discomfort. Over the years, she has undergone various conventional treatments, including the application of topical corticosteroids and tacrolimus, as well as narrowband UVB phototherapy. Despite good adherence, these approaches failed to produce meaningful or sustained repigmentation, particularly in the facial areas. This had a significant impact on her self-esteem and social interactions. At the time of evaluation in our outpatient clinic, the Facial Vitiligo Area Scoring Index (F-VASI) was 2, and her Dermatology Life Quality Index (DLQI) was 25, reflecting a major impairment in quality of life. Clinical documentation included standardized photographs, Wood's lamp examination, and imaging with the VISIA® skin analysis system (Canfield Scientific Inc., Parsippany, NJ, USA). These assessments were repeated at each follow-up to objectively track changes in pigmentation. Given her lack of response to previous therapies and the high psychosocial burden, topical ruxolitinib 1.5% was initiated, applied twice daily

to affected areas. The treatment was well tolerated, and the patient adhered to the regimen without difficulties.

By week 14, the patient achieved a 50% reduction in F-VASI, improving further to 75% by week 28. The response was particularly notable in the facial regions, which had been unresponsive to all prior treatments. Clinical photographs and imaging confirmed a predominantly perifollicular repigmentation pattern, with additional pigment reappearance at the periphery of the lesions – both features suggestive of active stimulation of the melanocyte reservoir. The remaining depigmented areas were primarily located on the frontal, periocular, and periorbital regions. However, these areas showed clear signs of active repigmentation. Importantly, no local or systemic adverse effects were reported during treatment, and no signs of irritation or hypopigmented rebound were observed. The patient reported a significant improvement in her quality of life, appearance-related distress, and social engagement.

This case provides real-world support for the use of topical ruxolitinib in patients with refractory non-segmental vitiligo. Unlike traditional therapies, which primarily aim to reduce inflammation or stimulate melanocyte migration, ruxolitinib directly inhibits key immune pathways – most notably the JAK-STAT axis – believed to sustain melanocyte destruction in vitiligo. Its topical formulation allows for localized effect while minimizing systemic exposure, which is especially important in patients with comorbidities. In conclusion, topical ruxolitinib represents a valuable therapeutic option for patients with vitiligo unresponsive to standard treatments. In our patient, it led to substantial clinical improvement, both objectively (F-VASI reduction, photographic evidence) and subjectively (DLQI improvement, quality-of-life restoration).

This case adds to the growing evidence supporting JAK inhibitors as a mechanism-based treatment for vitiligo. Further real-world data and long-term follow-up are needed to better define optimal protocols and the durability of response.

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Figure 1. Patient before and after 28 weeks of ruxolitinib cream applied twice daily. **A**) Clinical picture before starting treatment; **B**) high-quality photography with VISIA® system at week 0; **C**) UV light photography with VISIA® system at week 0; **D**) clinical picture after 14 weeks of treatment; **E**) high-quality photography with VISIA® system at week 14; **F**) UV light photography with VISIA® system shows a significant response after 14 weeks of therapy; **G**) clinical picture after 28 weeks of treatment; **H**) high-quality photography with VISIA® system at week 28; **I**) UV light photography with VISIA® system, demonstrating a 75% reduction of F-VASI after 28 weeks of treatment.