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## **Fast response in facial vitiligo monitored by non-invasive imaging during topical ruxolitinib treatment**

Francesca Feresin, Francesca Paola Sasso, Alberto Di Caro, Steven Paul Nisticò, Giovanni Pellacani, Annunziata Dattola, Maria Elisabetta Greco

Unit of Dermatology, Department of Clinical Internal, Anesthesiological and Cardiovascular Sciences, Sapienza University of Rome, Italy

*Correspondence: Francesca Feresin, Unit of Dermatology, Department of Clinical Internal, Anesthesiological and Cardiovascular Sciences, Sapienza University of Rome, Viale del Policlinico, 155, 00161 Rome, Italy.*

E-mail: [feresinfrancesca@gmail.com](mailto:feresinfrancesca@gmail.com)

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Dear Editor,

Vitiligo is a chronic autoimmune disorder affecting 0.5-1% of the population, characterized by progressive melanocyte loss and depigmented macules. Non-segmental vitiligo (NSV) is the most common subtype, typically involving the face and acral areas, with relevant psychosocial impact. Conventional therapies frequently provide delayed or incomplete responses. Recently, topical Janus kinase (JAK) inhibitors, such as ruxolitinib 1.5% cream, have shown promise in promoting repigmentation.<sup>1</sup>

The aim of this letter is to report a case of early clinical efficacy of topical ruxolitinib and to highlight the utility of high-resolution non-invasive imaging in monitoring treatment response in vitiligo.

We report the case of a 59-year-old male (Fitzpatrick phototype II) with stable NSV involving the face, hands, axillae, and feet. His past medical history includes allergic diathesis to grasses and pernicious anemia, currently treated with vitamin B12 and folic acid supplementation. No medication use, no smoking, and occasional alcohol consumption were reported. There was no family history of vitiligo or other autoimmune disease.

The patient started ruxolitinib 1.5% cream, which was applied twice daily to facial lesions. Five sessions of narrowband (NB)-UVB phototherapy were performed during the first month of treatment, administered concurrently with the topical application of ruxolitinib. This combined approach of early JAK inhibition supported by targeted photostimulation may have contributed to accelerating the speed of cutaneous repigmentation, as previously hypothesized,<sup>2</sup> but rarely demonstrated using high-resolution dermatologic imaging.

Importantly, in two randomized, double-blind, vehicle-controlled phase 3 studies of adults and adolescents aged  $\geq 12$  years (TRuE-V1/TRuE-V2), ruxolitinib cream was not evaluated in combination with phototherapy, and repigmentation was typically observed only after several months.<sup>3</sup>

In our case, the topical ruxolitinib-NB-UVB combination markedly shortened the time to visible repigmentation, suggesting a potential synergistic effect.

Non-invasive imaging was performed at three standardized time points: baseline before treatment initiation (T0), after 1 month of therapy (T1), and after 3 months of therapy (T2), focusing on the right cheek and forehead. Clinical imaging was obtained using the VISIA<sup>®</sup> system in visible and ultraviolet light, while cellular and structural changes were assessed by reflectance confocal microscopy (RCM) and line-field confocal optical coherence tomography (LC-OCT).

Clinical response was quantified using the Facial Vitiligo Area Scoring Index (F-VASI), retrospectively estimated from standardized clinical and ultraviolet VISIA<sup>®</sup> images. At baseline (T0), approximately 30% of the facial area was involved, with predominantly severe depigmentation,

corresponding to an F-VASI of about 22.5. After 1 month of treatment (T1), facial involvement decreased to approximately 18-20%, with partial repigmentation of most lesions, resulting in an F-VASI of approximately 9-10. After 3 months (T2), only small residual hypopigmented areas persisted (approximately 5-7% of the face, with mild depigmentation), corresponding to an F-VASI of approximately 1.5. Overall, this represented an estimated >90% improvement in facial vitiligo.

At T0, VISIA® visible light imaging (Figure 1A) documented sharply demarcated depigmented facial areas. At T2, VISIA® visible images (Figure 1B) showed near-complete clinical repigmentation, with markedly reduced contrast between lesional and non-lesional skin. Ultraviolet VISIA® images confirmed these findings, showing high-contrast depigmented areas at T0 (Figure 1C) and a marked reduction of hypopigmented areas at T2 (Figure 1D).

RCM at T0 (Figure 1E) showed the absence of bright dermal papillary rings and dendritic melanocytes at the dermo-epidermal junction (DEJ), consistent with active vitiligo.<sup>4</sup> At T2, RCM (Figure 1F) demonstrated the reappearance of bright dermal papillary rings and numerous dendritic melanocytes, confirming widespread melanocyte repopulation.

LC-OCT at T0 (Figure 1G) revealed a hyporeflective basal layer and poorly defined DEJ, suggestive of melanocyte depletion.<sup>5</sup> At T1, LC-OCT (Figure 1H) demonstrated perifollicular hyper-reflective dendritic structures, indicative of early melanocyte reactivation. At T2, LC-OCT (Figure 1I) showed increased basal reflectivity and restoration of the DEJ architecture.

Although LC-OCT was initially developed for structural imaging, its application in pigmentary disorders is emerging as a promising tool and its combination of vertical (B-scan) and *en face* (E-scan) acquisition enables comprehensive visualization of melanocyte architecture and inflammatory features. When integrated with RCM, a technique widely used in inflammatory and pigmentary dermatoses, this multimodal approach proved effective in detecting perifollicular reactivation and tracking pigment recovery in real time.

This case demonstrates how the combination treatment with ruxolitinib and phototherapy can facilitate rapid melanocyte reactivation and repigmentation in vitiligo. Larger longitudinal studies are warranted to validate these findings and to define the predictive role of non-invasive imaging biomarkers in personalized vitiligo management.

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**Figure 1.** Sequential non-invasive imaging of facial vitiligo treated with ruxolitinib 1.5% cream.

A) VISIA® at T0 (visible light): sharply demarcated depigmented facial areas; B) VISIA® at T2 (visible light): near-complete repigmentation with markedly reduced contrast between lesional and non-lesional skin; C) VISIA® at T0 (UV light): high contrast between lesional and non-lesional skin. Small white asterisks highlight depigmented areas; D) VISIA® at T2 (UV light): restored pigmentation and minimized contrast. Small black asterisks indicate repigmented areas; E) RCM at T0: absence of bright dermal papillary rings and dendritic melanocytes at the DEJ. White arrows indicate missing papillary rings; F) RCM at T2: widespread repopulation of melanocytes and bright dermal papillary rings. White arrows point to reappearing bright dermal papillary rings; arrowheads highlight dendritic melanocytes at the DEJ; G) LC-OCT at T0: hyporeflexive basal layer and disrupted DEJ structure (blue arrows indicate DEJ architecture and low basal reflectivity); H) LC-OCT at T1: perifollicular hyper-reflective dendritic structures indicating early melanocyte activity (blue arrows point to focal basal changes and DEJ outline); I) LC-OCT at T2: increased basal reflectivity and restored DEJ (blue arrows highlight normalized DEJ architecture and high reflectivity).

