

Abrocitinib Treatment of Riehl's Melanosis with Elevated Serum IgE: A Case Report

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Case Report

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Additional Declarations: No competing interests reported.

Abstract

Background Riehl melanosis is a rare acquired pigmentary disorder with an unclear pathogenesis and limited therapeutic options. Effective systemic treatments have rarely been reported. We present a case of Riehl melanosis associated with markedly elevated serum immunoglobulin E that responded favorably to treatment with abrocitinib, highlighting a potential new therapeutic approach.

Case presentation A 61-year-old Chinese woman developed diffuse gray-brown pigmentation involving the face, neck, trunk, extremities, dorsal hands, and dorsal feet after an initial episode of facial flushing, burning, and pruritus. Laboratory examination revealed markedly elevated serum immunoglobulin E, whereas autoimmune-related tests were unremarkable. Histopathological examination demonstrated increased melanin in the basal and lower spinous layers of the epidermis, superficial dermal lymphocytic infiltration, and abundant dermal melanophages, supporting the diagnosis of Riehl melanosis. The patient was treated with oral abrocitinib at a dose of 100 mg once daily. After 3 months of treatment, the pigmentation became lighter and less extensive, no new lesions developed, and the pruritus resolved completely.

Conclusion This case describes a favorable clinical response to abrocitinib in a patient with Riehl melanosis, pruritus, and elevated serum immunoglobulin E. The observation provides a basis for further investigation of the efficacy, safety, and potential mechanisms of abrocitinib in this rare disorder.

Background

Riehl melanosis is an acquired hyperpigmentation disorder involving not only increased basal pigmentation but also interface change, inflammation, and dermal pigmentary incontinence[1]. In RM lesions, dermal stem cell factor (SCF) and c-KIT are increased, epidermal and dermal endothelin-1 (ET-1) is upregulated, and keratinocyte guanine deaminase (GDA) is elevated; GDA, induced by IFN- γ , TNF- α , and IL-1 α , promotes melanogenesis by regulating SCF and ET-1[2]. Thus, RM may be associated with inflammation-related pigmentation.

Case presentation

A 61-year-old woman presented in April 2024 with a 10-month history of diffuse facial flushing with burning and pruritus. Tests for antinuclear antibody (ANA) and an autoantibody panel—including SS-A, SS-B, Ro-52, SM, Scl-70, Jo-1, CENP-B, PCNA, dsDNA, AMA-M2, and PM-Scl—were all negative. Total IgE was not tested initially. She was diagnosed with rosacea and treated with oral minocycline, 100 mg every other day, plus desloratadine citrate disodium and ebastine. After 30 days, the diffuse facial flushing darkened into dark-brown patches and spread to the neck, chest, nape, and upper back. Minocycline was stopped after 60 days, but the lesions became brown-gray and extended to the neck, abdomen, buttocks, extensor upper limbs, and dorsal feet. No trigger was identified. She had no history of allergic rhinitis or asthma and no relevant family history.

Examination showed diffuse gray-brown pigmentation with clear borders on the face, neck, abdomen, buttocks, upper and lower extremities, dorsal hands, and dorsal feet (Fig. 1). The palms, soles, lips, oral mucosa, and vulvar mucosa were spared. Generalized xerosis was present. Serum IgE was 1128.61 U/L; blood counts, biochemistry, and autoantibody-related tests were otherwise unremarkable. Histopathology of the left buttock showed mild parakeratosis, increased basal/lower-spinous melanin, superficial dermal lymphocytes, and abundant free melanin granules and melanophages in the papillary and reticular dermis (Fig. 2). Based on the clinical, histopathologic, and laboratory findings, the diagnosis was revised to RM. Oral abrocitinib, 100 mg/day, was then given. After 3 months, the lesions improved markedly, with lighter, less extensive pigmentation, no new lesions (Fig. 1), and complete relief of pruritus.

Discussion and conclusions

Abrocitinib, a highly selective oral Janus kinase 1 (JAK1) inhibitor, could potentially influence inflammatory and pruritic signaling; however, the mechanism underlying the improvement observed in this patient remains uncertain. In this case, elevated IgE and persistent pruritus in the patient, both closely linked to type 2 inflammation[3], may indicate a more active inflammatory state. Thus, abrocitinib may relieve pruritus and affect the inflammatory hyperpigmentation process of RM. On the other hand, RM and lichen planus pigmentosus (LPP) belong to the category of acquired dermal macular hyperpigmentation (ADMH) and show some histopathologic overlap[4]. JAK inhibitors have been reported for lichen planus pigmentosus, and topical ruxolitinib markedly improved lesions, indirectly supporting the use of abrocitinib for RM[5].

Prolonged oral administration of minocycline in this patient may have been a trigger for RM. Minocycline can present as different clinical types due to deposition of the drug and its metabolites in the skin on the one hand, and may aggravate pigmentation by promoting melanogenesis on the other hand, which may be further enhanced under ultraviolet exposure. Post-inflammatory hyperpigmentation is usually confined to previous definite inflammatory lesions, and was unlikely because this patient had diffuse facial pigmentation with gradual extension, which is consistent with typical RM. Second, atopic dermatitis was also unsupported: despite elevated total serum IgE and pruritus, she had no clinical manifestations of eczema, no history of allergic rhinitis or asthma, and no family history of atopic disease. Combined with the clinical manifestations and pathologic findings such as increased melanin granules in the basal layer and infiltration of melanophages in the superficial dermis, the diagnosis was RM.

Because specific histochemical staining, such as Perls staining, was lacking, and this was a case report, the causation between minocycline and this case could not be determined. However, there are currently few case reports and a lack of more scientific clinical studies, so the efficacy and safety of abrocitinib need to be further verified by larger sample studies.

This case report has several limitations. First, it describes a single patient without a control group, so the observed improvement cannot establish the efficacy of abrocitinib or a causal relationship between treatment and outcome. Second, the follow-up period was limited to 3 months, and longer-term durability of the response and safety could not be assessed. Third, no specific mechanistic biomarkers were evaluated, and therefore the relationship between elevated serum IgE, type 2 inflammation, and the clinical response remains speculative. Finally, although minocycline exposure preceded the development and progression of pigmentation, a causal relationship could not be determined because specific histochemical staining, such as Perls staining, was not performed.

Abbreviations

ADMH

Acquired dermal macular hyperpigmentation

ANA

Antinuclear antibody

ET-1

Endothelin-1

GDA

Guanine deaminase

IFN- γ

Interferon gamma

IgE

Immunoglobulin E

IL-1 α

Interleukin-1 alpha

JAK1

Janus kinase 1

LPP

Lichen planus pigmentosus

PM-Scl

Polymyositis–scleroderma antibody

RM

Riehl melanosis

SCF

Stem cell factor

TNF- α

Tumor necrosis factor alpha

Declarations

Ethics approval and consent to participate

Ethical approval for this case report was obtained from the Ethics Committee of the First Affiliated Hospital of Hebei University of Chinese Medicine. Written informed consent to participate was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

XYB collected the clinical data, reviewed the literature, and drafted the manuscript. YWL, BF, WDX, and LMJ participated in patient management, data interpretation, and critical revision of the manuscript. LMJ supervised the study and approved the final version of the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article. Additional information is available from the corresponding author upon reasonable request.

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Figures



Figure 1

Clinical manifestations before and after abrocitinib treatment. A–F, Diffuse gray-brown pigmentation before treatment. G–L, Marked improvement after 3 months of oral abrocitinib.

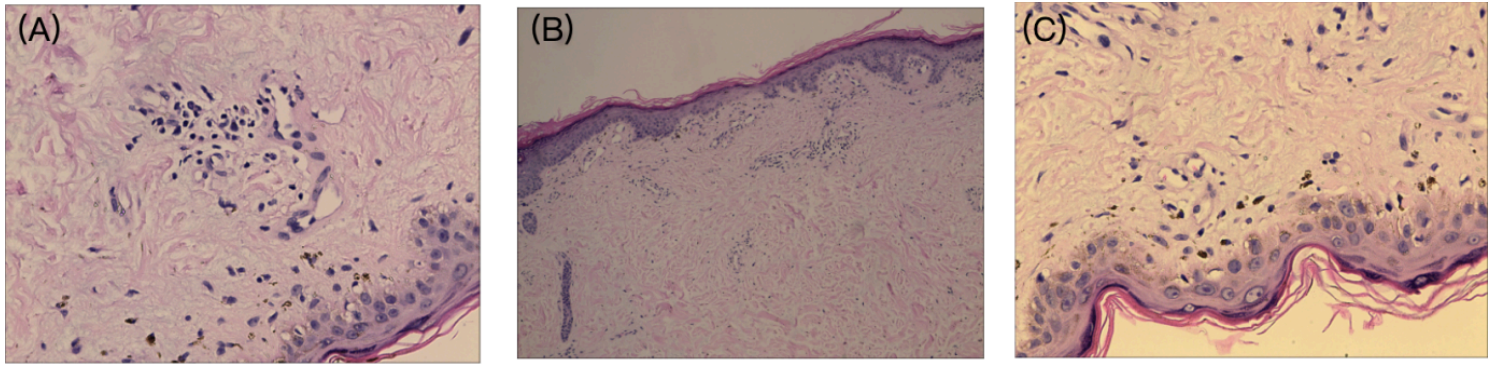


Figure 2

Histopathologic findings. A–C, Mild parakeratosis, increased basal and lower-spinous melanin, superficial dermal lymphocytic infiltration, and abundant free melanin granules and melanophages in the dermis.

Supplementary Files

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