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CASE REPORT

A Case of Intense Pulsed Light Aggravated Rosacea Successfully Treated by Abrocitinib

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Abstract: Rosacea is a chronic inflammatory skin condition characterized by facial erythema, papules, pustules, telangiectasia, and flushing. Currently, various treatment options are available, but no definitive cure has been established. Phototherapy is primarily effective for treating telangiectatic rosacea because it helps alleviate erythema and telangiectasia. However, it can also pose risks; when applied inappropriately, phototherapy may worsen rosacea symptoms, making the condition more difficult to manage. This case report presents a patient with rosacea who experienced acute exacerbation after intense pulsed light therapy, characterized by persistent erythema, edema, pustules, exudation, and a burning sensation with pain. Subsequent treatment with oral abrocitinib for 12 weeks led to a gradual resolution of the patient's facial symptoms. Therefore, we hypothesized that the oral JAK-1 inhibitor abrocitinib not only serves as a promising new treatment option for rosacea but also offers therapeutic benefits in cases of inappropriate phototherapy.

Keywords: rosacea, intense pulsed light, JAK inhibitor, abrocitinib

Introduction

Rosacea is a chronic inflammatory skin disease of the glandula sebacea, primarily affecting the cheeks, nose, and chin. It is characterized by persistent facial symptoms, such as papules, erythema, pustules, and telangiectasia, and may occasionally lead to nodular lesions and ocular involvement.[1] Although the pathophysiology of rosacea remains unclear, factors such as genetic predisposition, immune system dysregulation, vascular and neuronal dysfunction, and microbial involvement are believed to contribute to its development.[1] Current treatment options for rosacea include topical and oral medications, photodynamic therapy, injection therapy, and various surgical interventions. [2] In recent years, phototherapy, particularly laser therapy and intense pulsed light (IPL), has gained attention as a promising and innovative therapeutic approach for managing rosacea. In contrast to oral or topical pharmacological treatments, which are often associated with various adverse effects, phototherapy has demonstrated significant efficacy with minimal invasiveness.[3] Several studies have demonstrated that IPL effectively reduces erythema and telangiectasia in rosacea.[4,5] Although the side effects of phototherapy typically manifest as transient erythema and swelling, it can occasionally result in persistent and uncontrollable worsening of rosacea symptoms.[6] In cases of acute exacerbation of rosacea, clinicians may turn to treatments such as minocycline, cold compresses, physical therapies like red light or yellow light, or even short-term systemic corticosteroid therapy.[7] In this case, we present a patient with acute exacerbation of rosacea induced by IPL, who was resistant to conventional treatments but experienced alleviation of symptoms after treatment with abrocitinib.

Case Report

A 29-year-old female patient presented to the clinic with complaints of "persistent erythematous papules on the cheeks and paroxysmal flushing for several months." She reported experiencing a burning sensation and mild pruritus, which worsened with sun exposure. However, she did not exhibit other symptoms, such as fatigue, arthralgia, or oral ulcers. She also emphasized that the erythema had a considerable adverse effect on her quality of life, social interactions, and work performance. A review of her medical, personal, and family history revealed no significant findings. Clinical examination

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revealed symmetrical facial erythema, primarily affecting the bilateral cheeks, along with scattered papules, mild edema, infiltration, and subtle capillary dilation. Based on the clinical presentation, a diagnosis of rosacea was made. The patient had previously used oral minocycline, topical metronidazole, azelaic acid, and a functional moisturizer for two months, but experienced insufficient improvement in symptoms. The treatments were discontinued for at least 1 week before presentation (Figure 1A). Therefore, we treated the patient with a single session of DPL (7.8 mJ, 12 ms) (HarmonyXL). However, at the 2-week follow-up, the patient exhibited signs of worsening erythema and swelling, indicating acute exacerbation after phototherapy (Figure 1B). Thus, we prescribed prednisone (40 mg, Qd) along with adjunctive treatments, including yellow light therapy and cold compresses. Despite four days of oral prednisone administration at 40 mg per day, the patient's symptoms persisted and worsened, with obvious facial swelling and the development of numerous papulopustules and pustules on the cheeks (Figure 1C). Subsequently, we increased the prednisone dosage to 60 mg, Qd. However, at the 3-day follow-up, there was no significant improvement in symptoms, and the patient reported increased facial pain and tightness (Figure 1D). Given the progression of the disease, ineffectiveness of the conventional treatment regimen, and patient concerns, we decided to adopt a more aggressive approach. After careful discussion, we opted to use the JAK-1 inhibitor abrocitinib. Before treatment, the patient underwent routine blood routine tests, biochemical analyses, coagulation function tests, and chest CT scans, all of which showed no abnormalities. Consequently, we initiated oral treatment with 200 mg of abrocitinib daily. Within 2 weeks, the burning sensation significantly decreased, the erythema gradually improved, and partial pustules formed crusts. This was accompanied by a gradual decrease in hormonal dose (Figure 1E). The dosage was then reduced to 100 mg daily. After 4 weeks of abrocitinib treatment and follow-up, the patient reported feeling better, with improvements in swelling and telangiectasia.

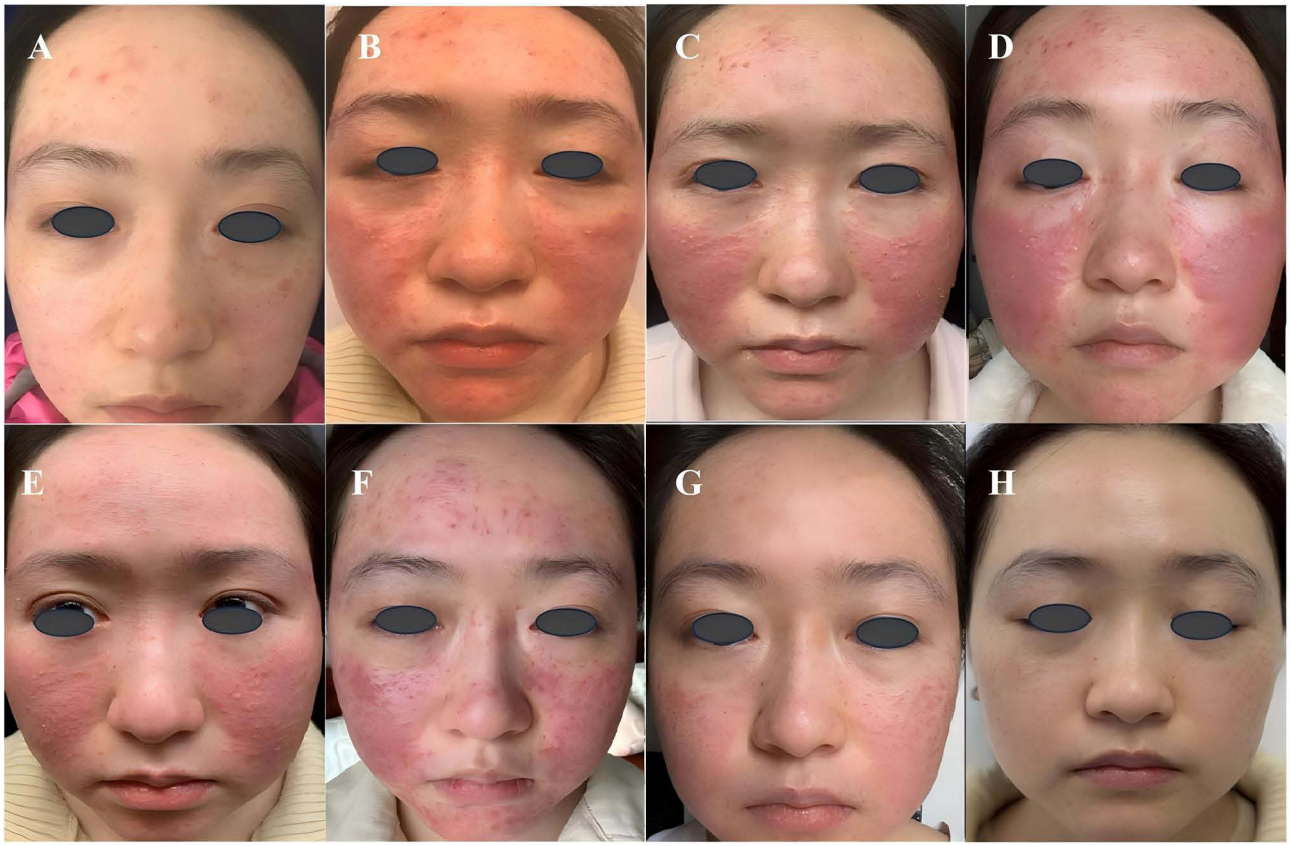

Figure 1 Clinical photographs showing the progression of the patient's condition. (**A**) Facial erythema, flushing, and telangiectasia before DPL (7.8 mJ, 12 ms) treatment; (**B**) Worsen erythema, exudation, and burning facial pain 2 weeks after DPL treatment; (**C**) Severe erythema and swelling 4 days after treatment with prednisolone 40 mg/d and yellow light; (**D**) Increased facial erythema, exudation, and swelling 3 days after treatment with prednisolone 60 mg/d and yellow light; (**E**) Improvement of severe erythema and swelling 2 weeks after treatment with abrocitinib 200 mg daily; (**F**) Extensive post-inflammatory erythema and depressed scars 4 weeks after treatment with abrocitinib 100 mg daily; (**G**) Follow-up at 12 weeks after receiving abrocitinib showing gradual clearance of post-inflammatory erythema and depressed facial scars; (**H**) Follow-up at 5 months after receiving abrocitinib showing complete clearance of erythema and partial resolution of depressed scars.

However, extensive post-inflammatory erythema and depressed scars remained on the patient's face (Figure 1F). After 12 weeks of follow-up (with a total abrocitinib treatment duration of 12 weeks), the patient's symptoms continued to improve, with clearance of post-inflammatory erythema, although depressed facial scars remained (Figure 1G). During the following 5 months of monitoring, no symptom recurrence or significant adverse reactions were observed, except for regional depressed scars (Figure 1H).

Discussion

Laser and light-based therapies, such as pulsed dye, IPL, and pro-yellow lasers, reduce erythema and demodex density in rosacea.[8] For instance, IPL therapy targets superficial blood vessels and dermal inflammation, making it a standard treatment option for erythematotelangiectatic rosacea.[9] However, individual responses vary, and in rare cases—such as with our patient—light-based treatments may trigger acute inflammatory flares. In certain individuals, IPL may compromise the skin barrier, increase vascular reactivity, or stimulate a surge in local inflammatory cytokines, potentially leading to paradoxical symptom exacerbations.[10] These effects may be influenced by individual immune responses or underlying skin sensitivity. Understanding this dual potential is crucial when selecting treatment for patients with rosacea who have reactive or sensitive skin. In some cases, laser-induced rosacea flares may resolve spontaneously once the treatment is discontinued.[11] However, our case demonstrated progressive worsening despite the early discontinuation of phototherapy after 2 weeks and 1 week of corticosteroid treatment. The rapid and sustained improvement observed only after initiating abrocitinib supports a pharmacological benefit beyond natural resolution.

The JAK/STAT signaling pathway plays a crucial role in the regulation of immune and inflammatory responses, acting as a central axis for receptor-mediated signal transduction triggered by extracellular cytokines.[12] This pathway is involved in various biological processes, including cellular proliferation and differentiation, organ development, and the maintenance of immune homeostasis.[13] JAK inhibitors suppress downstream inflammatory cytokine production by inhibiting JAK phosphorylation, preventing T cells from exerting anti-inflammatory and immunomodulatory effects.[14] Ultimately, JAK inhibitors exert their therapeutic effects by suppressing the production and release of various inflammatory cytokines.[14] Currently, JAK inhibitors are widely used as a therapeutic option for managing various inflammation-related disorders, including inflammatory bowel disease, rheumatoid arthritis, and atopic dermatitis.[15,16]

In rosacea, dysregulated innate immunity and the overexpression of inflammatory mediators such as interleukin-6 (IL-6), interleukin-8 (IL-8), and interferon-gamma (IFN-γ) have been implicated.[17,18] Emerging evidence indicates that the activation of the JAK/STAT signaling pathway plays a crucial role in exacerbating rosacea-related inflammation. This pathway interacts with Toll-like receptor 2 (TLR2) signaling mechanisms and the oxidative stress system induced by reactive oxygen species (ROS).[19,20] Activation of TLR2 and the oxidative stress system induced by ROS can trigger inflammatory and vasodilatory responses, which are associated with the erythema observed in rosacea. On the other hand, by inhibiting JAK phosphorylation, JAK inhibitors can suppress the downstream signaling of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interleukin-8 (IL-8), and monocyte chemoattractant protein-1 (MCP-1). Inhibition of JAK1 signaling may indirectly affect TLR activity and reduce cathelicidin (LL-37) expression, both of which play roles in the pathogenesis of rosacea.[18,21,22] In addition, abrocitinib, a selective JAK1 inhibitor, reduces the expression of Th1/Th17 cytokines and chemokines involved in neutrophilic and lymphocytic infiltration, which are believed to contribute to papulopustular rosacea.[23]

Given these interactions, it is reasonable to hypothesize that inhibiting the JAK/STAT signaling pathway could be an effective therapeutic strategy for rosacea. Emerging evidence from numerous studies indicates that oral JAK inhibitors, such as tofacitinib, may be a promising treatment option for patients with rosacea and rosacea-like dermatitis.[24] A single case report highlighted the positive clinical efficacy of tofacitinib in the treatment of steroid-induced erythematotelangiectatic rosacea.[24] In addition, a retrospective analysis was conducted on 21 patients with erythematotelangiectatic rosacea who were treated with oral tofacitinib.[25] This study aimed to assess the therapeutic efficacy of tofacitinib in this patient population. The results indicated that tofacitinib can improve the symptoms of erythematotelangiectatic and papulopustular rosacea. Tofacitinib is a JAK-1/3 inhibitor, and compared with the highly selective JAK-1 inhibitor abrocitinib, it is associated with more adverse effects. Abrocitinib may provide a faster onset of action, improved safety profile, and better tolerability, potentially enhancing patient adherence to treatment regimens.[26] In 2022, the US Food and

Drug Administration (FDA) approved abrocitinib for the treatment of moderate-to-severe atopic dermatitis (AD).[27] Numerous studies have demonstrated that in addition to AD, abrocitinib is most commonly used to treat conditions such as vitiligo, prurigo nodularis, and hand eczema.[28] To date, three case reports have documented the use of abrocitinib for the treatment of rosacea.[29] These reports include two case series—one involving four patients with erythematotelangiectatic rosacea and another with four patients with steroid-induced rosacea—as well as a single case report describing the treatment of granulomatous rosacea in one patient. The aforementioned reports demonstrate that abrocitinib results in significant clinical improvement in treating rosacea, with two patients showing mild improvement and one patient exhibiting no response.[29] In this case report, we report for the first time the beneficial effects of the JAK-1 inhibitor abrocitinib in improving erythema, swelling, exudation, and pustules induced by intense pulsed light for treating rosacea.

Conclusion

This case report is the first to demonstrate that JAK inhibitors can serve as an effective treatment for the exacerbation of rosacea inflammation induced by phototherapy. However, this study has several limitations regarding the use of abrocitinib for rosacea. First, this study's reliance on a single case report restricts the ability to generalize its findings to a larger patient population. Although abrocitinib was effective in this case, its efficacy cannot be assumed to be universally applicable. Second, there is currently no standardized protocol for the mechanism of action or the use of JAK inhibitors for the treatment of rosacea. Therefore, continuous follow-up studies are crucial to assess the long-term efficacy and safety profile of abrocitinib for the treatment of rosacea.

Consent Statement

The patient in this manuscript have given written informed consent to the publication of her clinical details and accompanying images. Institutional approval was not required to publish the case details.

Informed Consent

This case report has obtained consent from the patient and family, and has been approved by the Clinical Trial Ethics Committee of the Affiliated Hospital of Southwest Medical University, with the approval number: KY2024414.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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