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Photobiomodulation as Adjunctive Therapy for Refractory Facial Erosive Pustular Dermatosis Following Ablative CO_2 Laser Resurfacing

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Abstract

Erosive pustular dermatosis (EPD) is a rare, chronic, and often recalcitrant inflammatory dermatosis characterized by sterile pustules, erosions, and crusting. Its cause remains uncertain; cutaneous trauma is a recognized inciting factor, and diagnosis requires exclusion of infectious, autoimmune, neoplastic, and other mimicking disorders. Healing may be prolonged and may leave persistent erythema, textural alteration, dyspigmentation, atrophy, or scarring. We report a 41-year-old woman who developed severe facial erosions and pustules 13 days after fully ablative carbon dioxide (CO_2) laser resurfacing. Bacterial and viral cultures and immunologic testing were negative, and biopsy showed lymphocytic inflammation with nonspecific reparative changes. Multiple topical and systemic therapies produced limited or transient benefit. Photobiomodulation (PBM) was incorporated as an adjunct during the prolonged healing phase. The patient underwent 111 in-office PBM sessions and supplemented treatment at home every 48 hours with 660-nm red light (documented settings: 50 mW/cm², 4 J/cm², 50% duty cycle, 160 seconds). Serial photography from March 2022 through April 2025 documented persistent inflammatory disease followed by progressive re-epithelialization and remodeling. At approximately 3 years, epithelial integrity was restored and skin texture had substantially normalized, with mild residual erythema and minimal scarring or dyspigmentation. The independent contribution of PBM cannot be determined in a single case with concomitant therapies and a prolonged natural history. Nevertheless, the favorable tissue-remodeling outcome supports further investigation of PBM as an adjunct to modulate inflammation and support repair during prolonged healing in refractory EPD.

Introduction

Erosive pustular dermatosis (EPD) is an uncommon inflammatory disorder characterized by sterile pustules, erosions, and crusting, classically affecting atrophic and chronically sun-damaged skin.[1–3] Although the scalp is the most frequently reported site, facial EPD has been described after cosmetic resurfacing procedures.[4] The exact cause is unknown. Trauma appears to act as an inciting event in susceptible skin, but the subsequent inflammatory and reparative response can be idiosyncratic, persistent, and difficult to treat.

EPD is fundamentally a diagnosis of exclusion. Clinical and histopathologic findings may be nonspecific, requiring exclusion of infection, autoimmune disease, neoplasia, and other causes of chronic erosive dermatitis.[1–4] In recalcitrant cases, healing may take months or longer and may result in persistent erythema, altered texture, atrophy, dyspigmentation, or scarring. This prolonged abnormal healing trajectory provides a rationale for adjunctive strategies aimed not only at controlling inflammation but also at supporting orderly tissue repair.

Case report

A 41-year-old woman underwent radiofrequency microneedling for facial acne scars in early 2021, after which she perceived worsening of her scarring. A 4-month course of isotretinoin did not provide

meaningful improvement. In December 2021, she underwent fully ablative CO_2 laser resurfacing abroad (Ultrapulse Encore; 125 mJ; 4–5 passes). Initial healing appeared satisfactory, but extensive facial erosions developed 13 days after the procedure and were aggravated by mask friction during air travel.

The initial differential diagnosis included eczema herpeticum and severe contact dermatitis. Bacterial and viral cultures and immunologic testing were negative. A skin biopsy performed March 16, 2022, demonstrated lymphocytic inflammation and nonspecific reparative changes without evidence of infection or malignancy. In the setting of persistent sterile erosions and pustules following substantial cutaneous trauma and after exclusion of competing diagnoses, facial EPD was favored.

Empirical and subsequent therapies included valacyclovir, ciprofloxacin, prednisone (50 mg/day), petrolatum and saline cleansing, oral cyclosporine, topical tacrolimus, topical corticosteroids, topical clindamycin, doxycycline, trimethoprim-sulfamethoxazole, and low-dose isotretinoin. Responses were limited or transient. Propranolol 40 mg was later used as needed for prominent vascular flushing in the involved facial regions.

Photobiomodulation treatment and clinical course

PBM was incorporated beginning in March 2022 as an adjunctive strategy during the prolonged inflammatory and reparative phase. The patient ultimately received 111 in-office PBM sessions over approximately 3 years and also used a portable home PBM device every 48 hours. The home-device record documented 660-nm red light delivered with a 50% duty cycle (50% on and 50% off), at an irradiance of 50 mW/cm² during the light-on phase, for a total treatment interval of 160 seconds and a radiant exposure of 4 J/cm². The available clinical record does not establish that the in-office system used identical optical parameters.

Serial clinical photographs provide longitudinal documentation of the disease course (Fig. 1). In March 2022, approximately 3 months after resurfacing, confluent erythema with erosive and crusted inflammatory lesions remained prominent. Marked erythema and inflammatory activity persisted in July 2022. In October 2022, erythema remained pronounced with focal erosive and crusted lesions, demonstrating the prolonged and recalcitrant course. By February 2023, substantial re-epithelialization had occurred, although diffuse erythema and textural alteration persisted. By April 2025, epithelial integrity was restored with marked reduction in inflammation, substantial normalization of texture, mild residual erythema, and minimal scarring or dyspigmentation.

Discussion

This case illustrates several defining challenges of EPD: uncertain etiology, an apparent relationship to cutaneous trauma, nonspecific histopathologic findings, an exclusionary diagnosis, treatment resistance, and a potentially prolonged healing period.[1–4] Fully ablative CO_2 resurfacing represented a major epidermal and dermal injury and was temporally associated with onset of the disorder.

PBM was not used as a disease-specific treatment for EPD, but as an adjunct intended to support tissue repair during an unusually prolonged inflammatory wound-healing response. Red and near-infrared PBM have been reported to influence mitochondrial and redox signaling, inflammatory pathways, keratinocyte and fibroblast activity, and other processes relevant to tissue repair.[5–9] These effects provide a biologically plausible rationale for studying PBM in conditions in which persistent inflammation coexists with impaired or dysregulated re-epithelialization.

PBM has also been investigated specifically after ablative fractional resurfacing. In an earlier 14-subject pilot study, 660-nm PBM was evaluated before and/or after 2940-nm Er:YAG fractional resurfacing. Blinded photographic assessment showed less early erythema and edema on PBM-treated sides, and dermaspectrometer measurements demonstrated reduced erythema, with the greatest reduction (15%) at day 1 in the split-face PBM group. The study suggested that PBM could shorten post-resurfacing downtime, particularly by reducing erythema and edema during the first week.10 More recently, in a prospective intraindividual randomized study of 25 volunteers, combined LED PBM after ablative fractional laser treatment showed a numerical tendency toward faster healing in PBM-treated sites, although differences were not statistically significant.11 Together, these observations support the biological and clinical rationale for post-laser PBM while underscoring the need for cautious interpretation of treatment effects.

The longitudinal photographic series is an important feature of the present observation. Rather than a simple before-and-after comparison, it demonstrates persistent active disease over several months followed by gradual epithelial recovery and remodeling over a much longer interval. The final outcome is notable given the severity and duration of the inflammatory injury; however, temporal association should not be interpreted as proof of treatment efficacy.

Causal inference is not possible from this single case. The patient received multiple concomitant and sequential therapies, the optical parameters of the in-office PBM treatments are incompletely documented in the available record, and EPD has a variable natural history. Accordingly, clinical resolution cannot be attributed to PBM alone, and a scar-preventive effect cannot be established. The principal observation is the association of sustained adjunctive PBM with progressive epithelial recovery and a favorable final tissue-remodeling outcome despite severe initial injury and prolonged disease.

The limited residual textural and pigmentary sequelae support the hypothesis that PBM may be useful during the reparative phase of refractory EPD by modulating inflammation and supporting tissue repair. Prospective studies using standardized optical dosimetry and objective measures of erythema, epithelial recovery, texture, pigmentation, and scarring are required to determine whether PBM alters the course or final remodeling outcome of EPD.

Conclusion

Facial EPD after ablative CO_2 resurfacing is rare, diagnostically challenging, and may follow a prolonged and recalcitrant course. In this case, PBM was used as an adjunct throughout a 3-year healing period and was temporally associated with progressive re-epithelialization and a favorable cosmetic outcome. Although efficacy and scar prevention cannot be inferred from a single case, PBM warrants further study as a wound-modulating adjunct in refractory EPD and other prolonged postprocedural inflammatory healing disorders.

Clinical trial number: not applicable.

Declarations

Author Contribution

D.B. and A.B wrote the main manuscript text and D.B. prepared figure 1. All authors reviewed the manuscript.

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Figures

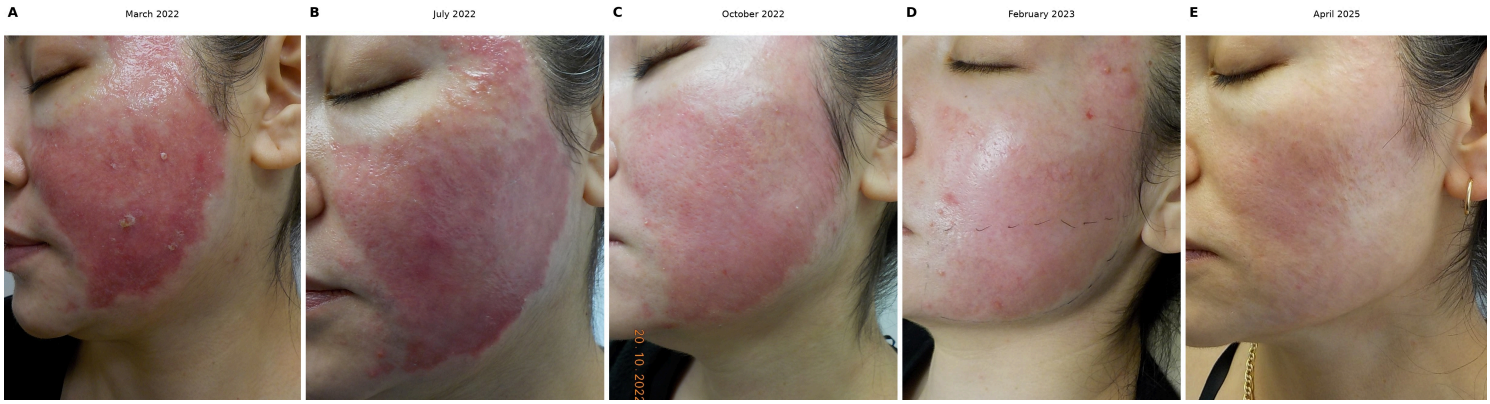


Figure 1

Longitudinal clinical course of facial erosive pustular dermatosis following fully.

Longitudinal clinical course of facial erosive pustular dermatosis following fully ablative CO_2 laser resurfacing and adjunctive photobiomodulation therapy. A, March 2022, approximately 3 months after ablative CO_2 resurfacing, showing persistent confluent facial erythema with erosive and crusted inflammatory lesions at the beginning of specialized management. B, July 2022, persistent marked erythema and inflammatory activity despite multimodal treatment. C, October 2022, ongoing erythema with focal erosive and crusted lesions, illustrating the prolonged and recalcitrant course. D, February 2023, progressive re-epithelialization and substantial reduction in inflammatory activity, with persistent erythema and textural alteration. E, April 2025, approximately 3 years after initiation of adjunctive PBM, showing complete epithelial restoration, marked reduction in erythema, substantial normalization of skin texture, mild residual erythema, and minimal scarring or dyspigmentation. During this period, the patient received 111 in-office PBM sessions supplemented by home treatment every 48 hours. Home-device settings were documented as 660 nm, 50 mW/cm² during the light-on phase, a 50% duty cycle (50% on/50% off), 160 seconds, and 4 J/cm² radiant exposure.