Steroid-Induced Rosacea: Is It Time to Redefine?

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Steroids are of great value in the treatment of a wide spectrum of dermatological diseases[1]. However, prolonged steroid use may lead to the development of new rashes, characterized by erythema, telangiectasia, papulopustular lesions, itching, and burning[2]. This condition currently lacks a precise definition and has been referred to as steroid-induced rosacea (SIR) in some studies. Although the term includes the word rosacea, a critical review of the literature, particularly of clinical descriptions and photographic evidence, indicates that many reported cases represent steroid-induced rosacea-like dermatitis (SIRD) rather than true rosacea, which is a well-defined term[3].

In clinical practice, we identified a distinct subset of patients whose disease was either triggered or exacerbated by steroids and who fulfilled the established diagnostic criteria for rosacea. This specific group warrants a more appropriate designation of SIR. Therefore, we propose a clear distinction between SIRD and SIR: SIRD is a form of dermatitis rather than rosacea, whereas SIR represents the "hidden" or "undiagnosed" rosacea that is triggered or exacerbated by steroids and meets established diagnostic criteria.

SIRD was first described in 1969 as "rosacea-like dermatitis"[4]. Since then, various terms have been used to describe this entity in the literature, including steroid rosacea, steroid dermatitis resembling rosacea, and others[5]. Based on clinical descriptions and photographic evidence from published reports, the characteristic clinical features of SIRD can be summarized as follows[3,6]: (1) subjective symptoms including burning sensation, pruritus, pain, xerosis, or a feeling of tightness; (2) objective signs including telangiectasia, erythema or flushing, scaling, papules, pustules, hyperpigmentation, or epidermal atrophy. SIRD is fundamentally a form of dermatitis, and its underlying mechanisms remain incompletely understood. Current hypotheses include the following: (1) glucocorticoid (GC)-induced inhibition of collagen synthesis, leading to skin atrophy and, by reducing structural support, passive vasodilation[7]; (2) dysregulation of vascular tone, wherein GCs, which have vasoconstrictive properties, antagonize vasodilatory mediators such as nitric oxide (NO), and upon GC withdrawal, accumulated NO in the endothelium is released, resulting in vasodilation[8]; (3) disruption of the cutaneous microecology, as the immunosuppressive effects of GCs promote microbial overgrowth and impair skin barrier function, thereby mediating inflammatory responses[9]; (4) accumulation of pro-inflammatory cytokines, which are released upon GC withdrawal, triggering an inflammatory cascade[6].

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Under a permissive genetic background, topical GC use may also act as a trigger for rosacea, a condition referred to as SIR. SIR is a genuine rosacea, with neurogenic inflammation as its core mechanism, characterized by dysregulation of neural responses to emotional stress, thermal stimuli, and other triggers. This leads to the production of inflammatory mediators and subsequent clinical manifestations such as erythema, flushing, papules, and pustules[10]. In this context, GCs serve only as a precipitating or exacerbating factor, akin to ultraviolet radiation, heat, trigger foods, and alcohol, rather than as a primary etiologic agent[11]. Consequently, SIRD typically occurs following prolonged topical GC application, with a duration ranging from months to years (mean approximately 3 months)[12], whereas SIR may present with classic rosacea manifestations after short-term use, even with low-potency GC preparations. Detailed history-taking often reveals overlooked prodromal features of rosacea, such as episodic flushing, transient erythema, and sensitive skin. Of note, some patients exhibit a family history of rosacea (e.g., affected parents or siblings), suggesting a subclinical rosacea diathesis.

Although both conditions are induced by GCs and share clinical features such as erythema and burning sensation, their divergence into two distinct disease entities is determined by the following key variables: (1) Genetic susceptibility. Patients with SIR carry rosacea-associated genetic susceptibility variants (e.g., immune-related genes such as *HLA-DRA* [major histocompatibility complex, class II, DR alpha] and *BTNL2* [butyrophilin like 2], as well as genes involved in neurovascular hyperreactivity)[13]. GC exposure unmasks and perpetuates the latent rosacea pathology. GCs upregulate Toll-like receptor 2 (TLR2) expression, leading to aberrant processing of cathelicidin and generation of the pro-inflammatory peptide Leucine-Leucine-37 (LL-37), which activates downstream inflammatory cascades—a critical initiating event in rosacea pathogenesis[14]. In contrast, patients with SIRD typically lack genetic susceptibility to rosacea, and the disease manifestations arise primarily from the effects of GCs on skin structure, function, and microbiome, without necessarily evolving into chronic neurovascular inflammation. (2) Clinical phenotype. In SIR, lesions are predominantly distributed in the central facial region (nose, cheeks, forehead, and chin), consistent with the classic rosacea pattern. Paroxysmal flushing and burning sensation are hallmark symptoms, reflecting the underlying neurogenic inflammation, while pruritus and scaling are less common. In SIRD, lesions are diffusely distributed across the entire area of GC application, with ill-defined borders. Although erythema and burning are also present, symptoms such as pruritus, xerosis, and scaling are prominent. Upon GC discontinuation, patients experience a classic "steroid withdrawal" phase characterized by intensified erythema, exudation, and burning. Over the course of the disease, SIRD does not progress to phymatous changes. (3) Treatment endpoints. SIR, as genuine rosacea, follows the natural history of rosacea even after complete GC withdrawal, necessitating long-term anti-inflammatory, neuromodulatory, and vascular-targeted therapy. In contrast, SIRD is potentially curable; following GC cessation, the classic steroid withdrawal phase ensues, but with barrier repair and dermal remodeling, the skin gradually recovers without persistent neurovascular erythema or burning. The therapeutic goal for SIRD focuses on eliminating GC dependence and restoring skin barrier function (Figure 1 and Table 1).

The histopathology of SIRD is characterized by eczematous epidermal changes, including mild acanthosis, spongiosis, and parakeratosis, accompanied by dilated post-capillary venules, perivascular lymphocytic infiltration, and dermal edema. Common dermal features include perifollicular inflammation, elastic fiber degeneration, possible *Demodex* infestation, and follicular or sebaceous hyperplasia. Noncaseating epithelioid granulomas with foreign bodies or Langerhans-type giant cells were also observed[9]. The histopathology of SIR typically mirrors that of rosacea, featuring vascular and lymphatic dilation, with mixed perivascular inflammatory infiltrates[10].

Due to the ambiguous definitions of SIRD and SIR, some studies referring to SIR should be classified as SIRD. For example, Xu et al.[15] reported the use of the Janus kinase 1 (JAK1) inhibitor abrocitinib for the treatment of SIR, and Lee et al. reported the use of 1% pimecrolimus cream for the treatment of SIR[16]. The reported cases showed involvement of the periorbital and alar groove regions, sites rarely affected by rosacea, along with scaling and pruritus, supporting the diagnosis of dermatitis rather than rosacea. This discrepancy does not necessarily reflect diagnostic error

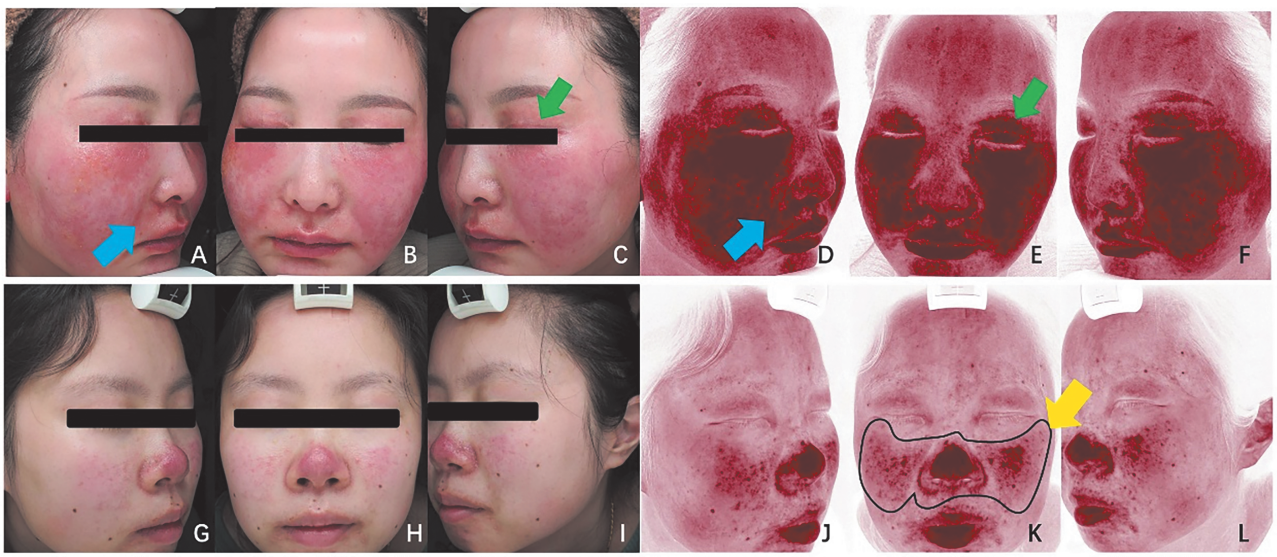

Figure 1: Typical clinical images of SIRD (white light mode [A–C] and red area images [D–F]) and SIR (white light mode [G–I] and red area images [J–L]) captured using the Visia® system.

Patient with SIRD (A–F) exhibit pronounced facial erythema, edema, and serous exudation, with lesions extending into atypical rosacea-affected zones such as the upper eyelids and philtrum area (blue and green arrows). In contrast, patients with SIR demonstrate classical rosacea manifestations confined to characteristic facial regions (yellow arrows). Abbreviation: RBX, red/brown surface; SIRD, steroid-induced rosacea-like dermatitis; SIR, steroid-induced rosacea.

by clinicians, but rather highlights the current lack of clear distinction between SIR and SIRD, two clinically and pathophysiologically distinct entities that are often conflated in the literature.

Treatment strategies for SIR and SIRD both center on glucocorticoid withdrawal, anti-inflammatory therapy, immunomodulation, and anti-angiogenic interventions. However, their specific approaches diverge in the following key aspects: (1) Differences in anti-inflammatory targets and drug selection. SIR is essentially a rosacea, with neurogenic inflammation as its core mechanism. Early anti-inflammatory therapy should focus on the anti-inflammatory effects of tetracyclines (e.g., minocycline and doxycycline) and neurovascular modulators (e.g., carvedilol). Although JAK inhibitors have been explored for the treatment of erythematotelangiectatic or papulopustular rosacea, existing reports are limited by small sample sizes, and most involve non-monotherapy regimens[17]. Mechanistically, while rosacea involves cutaneous inflammation, its core pathogenesis centers on neurogenic inflammation and vascular dysregulation rather than a T helper type 1/T helper type 2 (Th1/Th2)-mediated adaptive immune response; this may limit the clinical efficacy of JAK inhibitors, whose primary mechanism is cytokine pathway blockade. In contrast, JAK inhibitors demonstrate superior efficacy in steroid-induced rosacea-like dermatitis. Studies by Xu et al.[15] have shown that JAK inhibitors significantly improve erythema, papules, and pruritus in SIRD patients, with no notable adverse effects. Misdiagnosing SIR as SIRD and administering long-term potent JAK inhibitors or calcineurin inhibitors such as tacrolimus may suppress inflammation but could paradoxically induce a more severe rosacea-like flare due to chronic immunomodulation. SIRD is fundamentally a dermatitis complicated by steroid withdrawal. Early anti-inflammatory management must address severe epidermal barrier disruption and eczematous changes; during this phase, mild emollients or topical calcineurin inhibitors can be used short-term as steroid-sparing agents to control exudation and xerosis. Anti-inflammatory drugs primarily acting via cytokine pathway blockade, such as JAK inhibitors, may exhibit a more pronounced therapeutic effect in SIRD. (2) Differences in timing of laser and intense pulsed light (IPL) therapy. In SIR, the epidermal barrier is relatively intact, with inflammation predominantly concentrated around the perivascular dermis. Therefore, early and proactive intervention with pulsed dye laser, IPL, or vasoconstrictor agents is feasible once inflammation is controlled, with a favorable safety profile. In contrast, SIRD is often associated with spongiosis,

Table 1: Differences between SIRD and SIR.

	SIRD	SIR
Definition	A rosacea-like inflammatory dermatitis	Rosacea with steroids as the triggering factor
Genetic factors	No susceptible genes have been identified yet	1. Genetic background with the presence of susceptible genes. 2. Patients often experience facial erythema or flushing at an early age
Clinical manifestations	1. Papulopustules, telangiectasia, erythema, with intermittent flushing being subtle; 2. More likely to exhibit exudation, crusting, dryness, scaling, and itching; 3. Steroids withdrawal triad symptoms: intense itching, severe dryness and tightness, frequent scaling; 4. No phymatous lesions	1. Commonly presents with episodic flushing, persistent erythema, papulopustules, and telangiectasia; 2. Pruritus and scaling are relatively uncommon; 3. Phymatous lesions may appear
Commonly affected areas	Skin lesions may appear in atypical sites for rosacea, including the upper eyelids and the skin on both sides of the philtrum	Rosacea predominantly affects the convex areas of the central face, such as the cheeks, forehead, glabella, chin, and nose.
Treatment	**Core goals**: steroid withdrawal, barrier repair, and inflammation control **Pharmacotherapy**: • Topical calcineurin inhibitors (tacrolimus, pimecrolimus) • JAK inhibitors (e.g., abrocitinib) • Oral antibiotics (adjunctive, not core) • Vasculature-targeting agents (beta-blockers, topical alpha-agonists) should be used with caution; timing is critical—use is recommended after barrier restoration to avoid rebound flushing **IPL/Laser therapy**: should be deferred until inflammation subsides and barrier function is restored; laser/IPL should be applied with caution	**Core goals**: long-term control of inflammation and vascular hyperreactivity **Pharmacotherapy**: • Topical metronidazole, azelaic acid, ivermectin • Oral tetracyclines (subantimicrobial doses for long-term maintenance) • Oral isotretinoin • Beta-blockers and topical alpha-agonists can be used as conventional treatment **IPL/Laser therapy**: can be introduced early; laser/IPL serve as important therapeutic modalities
Prognosis	Complete recovery may be achievable with systematic and scientifically guided treatment	Complete resolution may be achievable, but most patients experience recurrent episodes over several years or even decades, necessitating repeated and intermittent treatment

Abbreviation: SIRD, steroid-induced rosacea-like dermatitis; SIR, steroid-induced rosacea; JAK, Janus kinase; IPL, Intense pulsed light.

parakeratosis, and severe barrier dysfunction; thus, the use of energy-based treatment should be approached with caution. Laser treatment should be considered only for residual telangiectasias after complete resolution of inflammation and restoration of barrier integrity. Premature energy-based treatment based on a misdiagnosis of rosacea in SIRD may lead to adverse events such as exacerbated inflammation, burns, and blister formation. (3) Differences in prognosis and management of patient expectations. SIRD is an exogenous, drug-induced dermatitis that achieves clinical cure following successful steroid withdrawal and skin barrier restoration, without the need for long-term maintenance therapy. In contrast, SIR represents genuine rosacea unmasked by corticosteroids in genetically predisposed individuals. These patients have lifelong neurovascular hyperreactivity and require a chronic disease management framework, with treatment goals centered on symptom control rather than cure. Clarifying these differences in therapeutic endpoints is essential for establishing realistic patient expectations (Figure 2).

In clinical practice, SIR and SIRD may indeed coexist in the same patient. To address such complex scenarios, we propose the following sequential management strategy: SIRD should be addressed first (barrier repair, control of pruritus and exudation, and steroid withdrawal), followed by rosacea management.

In summary, while current literature often conflates SIRD with true SIR, these two conditions are likely to be fundamentally distinct disease entities. Our viewpoint

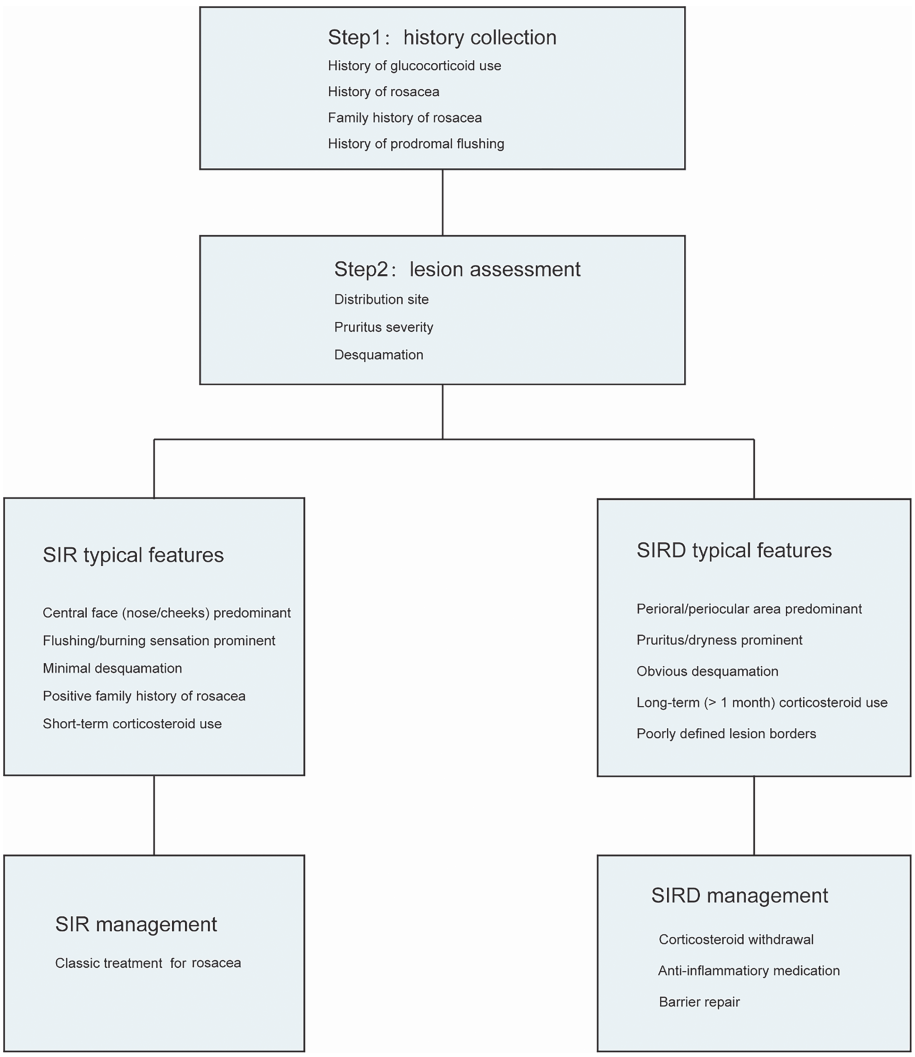

Figure 2: Diagnosis of patients with rosacea-like symptoms.

shares common ground with that recently proposed in the Japanese Rosacea Consensus, which clearly distinguishes between rosacea triggered or exacerbated by glucocorticoids and glucocorticoid-induced dermatitis. In Japan, the latter condition is now referred to as SIRD instead of the previous term SIR, which is consistent with the perspective we have put forward[18]. Overlooking their differences in pathogenic origins and genetic background can lead to errors in treatment timing, drug selection, and long-term prognosis. It should be acknowledged that no direct comparative genetic studies between SIR and SIRD patient populations have been conducted to date. Future large-scale genome-wide association studies or whole-exome sequencing studies comparing the two groups are needed to identify whether they harbor distinct genetic susceptibility loci, thereby providing direct molecular evidence to support their fundamental distinction. Even when both conditions coexist on the same patient's face, they should be clearly differentiated. Therefore, in clinical practice, SIRD should not be mistaken for refractory rosacea and subjected to prolonged laser or immunomodulatory therapy. Conversely, SIR, with its underlying genetic predisposition, should not be managed solely as a barrier disruption or dermatitis, neglecting neurovascular modulation and long-term trigger management. Accurate differentiation between the two is the key to achieving individualized treatment. The above opinions are derived from our own learning and clinical experience, and are put forward for discussion among colleagues.

Author contributions

Xiaoqi Meng drafted the manuscript. Tingwei Liu curated and organized the data. Xin Liu and Yang Xu provided resources, and supervised the work. All authors reviewed, edited, and approved the final version of the manuscript and agree to its publication.

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Ethics statement

Not applicable.

Data availability statement

No datasets were generated or analyzed for this work.

AI statement

The authors declare that no generative artificial intelligence (AI) or AI-assisted technologies were used in the preparation, analysis, or writing of this manuscript.

Conflicts of interest

The authors declare no conflicts of interest.

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