

IATROGENIC HYPERCORTICISM IN VULGAR PSORIASIS – CASE REPORT

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Summary

High-potency topical glucocorticoids represent the first-line therapeutic cornerstone in psoriasis vulgaris. However, prolonged and unsupervised use may lead to systemic absorption and suppression of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in iatrogenic Cushing syndrome [1,2].

We present the case of a 40-year-old female patient with a 25-year history of psoriasis vulgaris, admitted for intense diffuse cutaneous erythema with a tendency toward erythroderma, extensive lamellar desquamation, and multiple erythematous-squamous plaques, developed on a background of marked skin atrophy. The patient had chronically used clobetasol propionate, applied topically twice daily over long periods, without medical recommendation or specialist monitoring. Clinical examination revealed cushingoid facies, severe scalp involvement, and significant nail changes. Paraclinical investigations showed an extremely low serum cortisol level (3.97 ng/ml) and osteopenia (DEXA score). Treatment was initiated with oral Prednisone (10 mg/day) to support the HPA axis, associated with systemic Methotrexate (15 mg/week subcutaneously), folic acid, and non-steroidal topical emollient therapy. Clinical and paraclinical evolution at 2 and 5 months was markedly favorable, with a significant reduction in severity indices (PASI from 37.1 to 19.8) and recovery of HPA axis function, with serum cortisol increasing to 105.2 µg/dL at two months.

This case highlights the dangers of self-medication with super-potent dermatocorticosteroids. Interdisciplinary collaboration and periodic medical monitoring are essential to prevent severe iatrogenic complications.

Keywords: psoriasis vulgaris, clobetasol propionate, iatrogenic Cushing syndrome, HPA axis suppression.

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Introduction

Psoriasis is a chronic, relapsing, immune-mediated inflammatory dermatosis with an estimated global prevalence of approximately 2–3% [3]. Clinically, it is characterized by well-demarcated erythematous plaques covered with silvery-white scales, predominantly located on extensor surfaces, the scalp, and nails, often associated with a significant impairment in patients' quality of life [4].

In the management of mild and moderate forms, topical glucocorticoids remain the first-

line therapy due to their potent anti-inflammatory, antimitotic, and immunosuppressive properties [3]. However, prolonged use or uncontrolled application of high-potency molecules (class IV, such as clobetasol propionate) leads to significant local adverse effects (skin atrophy, telangiectasia, striae). Although considered rare in dermatology, systemic transcutaneous absorption may occur when applied over large areas or for prolonged periods, causing profound suppression of the hypothalamic–pituitary–adrenal (HPA) axis and leading to iatrogenic Cushing syndrome [3,4].

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Materials and Methods

This paper presents a retrospective analysis of a clinical case of iatrogenic hypercorticism associated with severe psoriasis vulgaris exacerbated secondary to therapeutic abuse. Clinical evaluation was performed using standardized dermatological indices: Psoriasis Area and Severity Index (PASI), Psoriasis Scalp Severity Index (PSSI), Nail Psoriasis Severity Index (NAPSI), and Dermatology Life Quality Index (DLQI).

Paraclinical assessment included monitoring morning serum cortisol dynamics, serum ionogram, inflammatory markers (fibrinogen), and bone mineral density evaluation by DEXA osteodensitometry at the lumbar spine (L1–L4). Follow-up was conducted through periodic reassessments at 2 and 5 months after initiation of steroid tapering and introduction of systemic immunomodulatory therapy.

Case presentation

A 40-year-old female patient from an urban area was admitted with a severe cutaneous clinical picture characterized by intense diffuse erythema with progression toward erythroderma, accompanied by extensive lamellar desquamation and multiple well-defined erythematous-squamous plaques on a background of generalized skin atrophy.

From her medical history, the patient had been diagnosed with psoriasis vulgaris 25 years prior. In an attempt to self-manage disease flares, she chronically and abusively used clobetasol propionate (applied topically twice daily over large body areas) for prolonged periods, without medical recommendation or specialist monitoring.

Clinical examination revealed pathognomonic signs of hypercorticism: cushingoid ("moon face") facies and severely atrophic, fragile skin with pronounced vascular pattern (disseminated telangiectasia). On the scalp, confluent erythematous plaques covered with

thick, adherent scales causing intense pruritus were observed. Nail changes were also severe, including nail pitting, leukonychia, red lunula spots, onycholysis, and subungual hyperkeratosis. Initial severity scores indicated extremely severe disease: PASI = 37.1; PSSI = 40; NAPSI = 100; DLQI = 9.

Initial laboratory investigations showed elevated fibrinogen (381 mg/dl) and marked vitamin D deficiency (19.6 ng/ml). Serum ionogram values were: potassium = 4.13 mmol/l; sodium = 141 mmol/l; chloride = 105 mmol/l. The crucial finding was a critically low morning serum cortisol level (3.97 ng/ml), confirming HPA axis suppression induced by topical corticosteroids. DEXA osteodensitometry at the lumbar spine (L1–L4) showed a mean bone mineral density (BMD) of 0.907 g/cm², establishing the diagnosis of secondary iatrogenic osteopenia.

Therapeutic Management and Monitoring

Following endocrinology consultation, uncontrolled topical clobetasol use was discontinued and systemic replacement corticotherapy with Prednisone 10 mg/day was initiated to prevent acute Addisonian crisis [5].

Simultaneously, for dermatological control, non-steroidal systemic therapy with Methotrexate (15 mg/week subcutaneously) was introduced, along with folic acid (5 mg/week, administered 24 hours after Methotrexate) and vitamin D supplementation (2000 IU/day). Topical therapy was limited to emollients (keratolytic cleansing gel and moisturizing emulsion twice daily) and a clobetasol shampoo restricted to two applications per week.

At the 2-month follow-up, the patient showed favorable clinical and biological evolution. Ionogram parameters remained stable (K = 4.4 mmol/l, Na = 138 mmol/l, Cl = 109 mmol/l). Serum cortisol increased significantly to 105.2 µg/dL, indicating progressive recovery of endocrine function. Consequently, abusive dermatocorticosteroids were completely discontinued, and Prednisone was gradually tapered (from 10 mg/day to 5 mg/day for two weeks,



Figure 1: Initial clinical appearance of the patient at admission. Marked diffuse cutaneous erythema with a clear tendency toward erythroderma is observed, along with polycyclic erythematous-squamous plaques and pronounced skin atrophy, with prominent visualization of the subcutaneous vascular network on the anterior thorax.



Figure 2: Severe nail involvement at initial presentation, characterized by massive subungual hyperkeratosis, structural leukonychia, red lunula spots, and secondary dystrophic psoriatic onycholysis (initial NAPSI score = 100).



Figure 3: Scalp involvement showing extensive erythematous plaques extending beyond the frontal hairline, covered by thick, hyperkeratotic, silvery-white scales, associated with intense pruritus (initial PSSI score = 40).



Figure 4: Favorable dermatological clinical evolution at 2 months after initiation of Methotrexate therapy and gradual tapering of systemic corticosteroids. A significant reduction in erythema and remission of truncal lamellar scaling are observed.

Table I. Dynamics of clinical severity scores and quality of life index during evaluations

Parameter biological	Initial admission	Re-evaluation at 2 months	Re-evaluation at 5 months
PASI	37.1	24	19.8
PSSI	40	24	12
NAPSI	100	100	97
DLQI	9	7	4

Legend: PASI – Psoriasis Area and Severity Index; PSSI – Psoriasis Scalp Severity Index; NAPSI – Nail Psoriasis Severity Index; DLQI – Dermatology Life Quality Index.

Table II. Dynamic evolution of monitored biological parameters during treatment

Parameter biological	Initial admission	Re-evaluation at 2 months	Re-evaluation at 5 months
Serum cortisol	3.97	105.2	85.42
Sodium (mmol/L)	141	138	141
Potassium (mmol/L)	4.13	4.40	4.2
Chloride (mmol/L)	105	109	109



Figure 5: Final evaluation at 5 months post-treatment (June 2026). Marked remission of psoriatic lesions and restoration of normal thickness and elasticity of the truncal skin barrier following complete resolution of steroid-induced iatrogenic atrophy.

then to 2.5 mg/day until cessation). Clinical scores improved: PASI = 24; PSSI = 24.

At the third evaluation (June 2026, 5 months), therapeutic success was confirmed after complete cessation of both topical and systemic corticosteroids. Clinical examination showed substantial remission of lesions and structural recovery of skin atrophy. Severity scores decreased significantly: PASI = 19.8; PSSI = 12; NAPSII = 97; DLQI = 4, reflecting improved quality of life. Laboratory parameters confirmed normal ionogram and optimal cortisol levels, with no signs of adrenal insufficiency.

Discussions

This case highlights a strong pathophysiological correlation between the severity of cutaneous lesions and secondary endocrine manifestations. Percutaneous absorption of very high-potency glucocorticoids (class IV), such as

clobetasol propionate 0.05%, follows diffusion kinetics where skin barrier integrity is the main limiting factor [4].

In psoriasis, architectural alterations—parakeratotic hyperkeratosis, acanthosis, and increased dermal angiogenesis—lead to a drastic reduction in barrier function. This structural breach, combined with application over large areas (initial PASI score of 37.1) and natural occlusion (folds, scalp), explains the high systemic bioavailability observed [2].

At the endocrine level, the underlying molecular mechanism was characterized by profound suppression of the hypothalamic–pituitary–adrenal (HPA) axis. Systemically absorbed clobetasol exerted a pronounced negative feedback effect on the hypothalamic paraventricular nuclei and anterior pituitary corticotroph cells, suppressing the transcription of the genes encoding corticotropin-releasing hormone (CRH) and proopiomelanocortin

(POMC), the precursor of adrenocorticotrophic hormone (ACTH). Chronic deprivation of the trophic stimulation normally provided by ACTH to the zona fasciculata of the adrenal cortex resulted in progressive cellular atrophy and cessation of endogenous steroidogenesis, a biological state reflected by the critically low morning serum cortisol level of 3.97 ng/mL [6].

Biological effects were evident on three levels:

- **Extracellular matrix damage:** The marked reduction in dermal thickness and the development of telangiectasias are direct consequences of glucocorticoid-mediated inhibition of type I and type III collagen synthesis and fibroblast proliferation [3].
- **Metabolic disturbances and adipose tissue redistribution:** The development of a cushingoid facies is mediated by differential activation of central versus peripheral glucocorticoid receptors, promoting centripetal lipogenesis and characteristic fat redistribution [5].
- **Secondary osteopenia:** As demonstrated by the DEXA findings (BMD 0.907 g/cm²), osteopenia resulted from the direct inhibitory effects of glucocorticoids on osteoblastogenesis and their stimulatory effects on osteoclast activity, further exacerbated in this case by severe vitamin D deficiency (19.6 ng/mL) [6].

The therapeutic management required a highly nuanced multidisciplinary approach. Abrupt discontinuation of the topical corticosteroid would have exposed the patient to a threefold life-threatening risk: precipitating an acute adrenal crisis secondary to severe adrenal insufficiency, increasing the risk of cardiovascular collapse, and triggering a severe dermatologic rebound phenomenon manifesting as erythrodermic or generalized pustular psoriasis [1].

In accordance with the current guidelines of the European Society of Endocrinology and the European Academy of Dermatology and Venereology (EADV), the optimal strategy consisted of implementing a gradual steroid tapering regimen [3]. Replacing clobetasol with a short-acting systemic glucocorticoid (prednisone 10 mg/day) maintained essential metabolic

homeostasis while allowing for controlled dose reduction to facilitate the recovery of endogenous ACTH pulsatility.

Control of the underlying inflammatory skin disease was achieved through the early initiation of methotrexate (15 mg/week administered subcutaneously), a first-line non-steroidal systemic immunomodulatory agent. Methotrexate effectively inhibited the dihydrofolate reductase metabolic pathway, thereby suppressing keratinocyte hyperproliferation and reducing T-cell infiltration within psoriatic plaques and the scalp [4]. This combined therapeutic approach enabled complete corticosteroid withdrawal, improvement in disease severity indices (PASI 19.8; PSSI 12 at 5 months), and full recovery of adrenal function, as confirmed by an increase in morning serum cortisol to 105.2 µg/dL.

Conclusions

1. There is a substantial iatrogenic risk associated with the extensive use of class IV topical corticosteroids. Chronic, unsupervised application of clobetasol propionate over large body surface areas results in transcutaneous absorption rates comparable to substantial systemic glucocorticoid exposure. This phenomenon negates the safety profile typically associated with topical administration, leading to the full clinical manifestation of iatrogenic Cushing's syndrome accompanied by profound suppression of the hypothalamic-pituitary-adrenal (HPA) axis [3].
2. The complex therapeutic management of patients with psoriasis requires familiarity with steroid withdrawal protocols and close interdisciplinary collaboration. In the presence of iatrogenic hypothalamic-pituitary-adrenal (HPA) axis suppression, topical corticosteroid therapy cannot be discontinued abruptly because of the imminent risk of potentially life-threatening acute adrenal insufficiency and severe erythrodermic rebound. A coordinated, stepwise approach involving both dermatologists and endocrinologists is

therefore essential, combining gradual systemic glucocorticoid replacement and tapering (prednisone) with concomitant control of the underlying dermatosis using non-steroidal immunomodulatory therapy (methotrexate) [3,5].

3. Standardized endocrine and skeletal screening should be considered an integral component of the evaluation of patients with severe psoriasis vulgaris and a documented history of prolonged or inappropriate use of super-potent topical corticosteroids. These patients require proactive laboratory and imaging assessment. Measurement of morning serum cortisol levels and evaluation of bone mineral density by dual-energy X-ray absorptiometry (DEXA) are essential

investigations for the early detection of subclinical adrenal insufficiency and secondary osteopenia or osteoporosis [6].

4. The present case underscores the need to strengthen therapeutic education programs for patients with chronic diseases, with particular emphasis on the risks associated with self-medication and the appropriate use of topical corticosteroids, including correct dosing based on the Finger Tip Unit (FTU) rule. Furthermore, stricter pharmaceutical oversight is warranted to prevent the repeated dispensing of high-potency topical corticosteroids without periodic specialist reassessment [2].

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Conflict of interest
NONE DECLARED

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