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Review Article

Iatrogenic Cushing Syndrome from Topical Corticosteroid Use in Infants: A Review of Published Cases

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Abstract

Background: Topical corticosteroids are widely used in infants, but thin, highly permeable skin and a high surface-area-to-weight ratio permit systemic absorption sufficient to cause iatrogenic Cushing syndrome.

Objective: To review published cases and case series of iatrogenic Cushing syndrome and hypothalamic-pituitary-adrenal (HPA) axis suppression from topical corticosteroid use in infants.

Methods: Narrative review of case reports, case series, cohort and meta-analytic data, and management literature.

Findings: Most reported infant cases followed unsupervised use of superpotent agents, chiefly clobetasol propionate, applied to the diaper area for 1-7 months; biochemistry uniformly showed secondary adrenal insufficiency (low cortisol with low or inappropriately normal ACTH). A meta-analysis of 522 children found suppression rising from 2.0% with low-potency to 6.6% with high-potency agents. Recovery after physiological hydrocortisone replacement and gradual withdrawal took a median of 2-6 months across series.

Conclusions: Clinicians should recognise cushingoid infants promptly, avoid abrupt cessation of potent topical steroids, and taper under hydrocortisone cover to prevent adrenal crisis.

Keywords: Iatrogenic Cushing syndrome; Topical corticosteroids; Infants; Adrenal suppression; Clobetasol propionate

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Introduction

Topical corticosteroids are a cornerstone of treatment for diaper dermatitis, atopic dermatitis, and other inflammatory skin conditions in infancy, and their apparent familiarity as an over-the-counter remedy in many countries has made unsupervised, prolonged use common [1,2]. Unlike older children and adults, infants combine a markedly higher body-surface-area-to-body-weight ratio with a thinner, less fully keratinised stratum corneum, and the diaper area itself is frequently occluded, all of which magnify

percutaneous absorption of applied steroid [3,4]. When a sufficiently potent agent is applied over a large area for weeks to months, cutaneous absorption can reach systemic concentrations adequate to suppress the hypothalamic-pituitary-adrenal (HPA) axis and to produce the clinical phenotype of Cushing syndrome — a condition that is entirely iatrogenic and, critically, reversible if recognised [5,6]. This review surveys the published individual case reports and case series of iatrogenic Cushing syndrome and HPA-axis suppression attributable to topical corticosteroid use in infants, together with the epidemiological, pathophysiological and management literature that frames those cases.

Epidemiology

Systematic quantification of topical-corticosteroid-induced Cushing syndrome in infants is limited to case reports and small series rather than population-based incidence figures. The best available pooled paediatric estimate comes from a meta-analysis of 12 trials (522 children, drawn from 128 eligible studies) evaluating cutaneous topical corticosteroid use for atopic dermatitis, which found HPA-axis suppression in 3.8% overall (95% CI 2.4-5.8), rising with potency class: 2.0% (3/148) with low-potency agents, 3.1% (7/223, 95% CI 1.5-6.3) with mid-potency agents, and 6.6% (10/151, 95% CI 3.6-11.8) with high-potency agents [7]. Because this pool studied prescribed, monitored dermatitis treatment, it likely under-represents the unsupervised, superpotent exposures — most often clobetasol propionate purchased without prescription for diaper rash or presumed scabies — that produce overt Cushing syndrome in the published infant case literature [8,9,5,10,11,12,13,14,15,16]. A cohort of 14 children with iatrogenic Cushing syndrome from any glucocorticoid route found 85% had adrenal insufficiency at presentation, with 10 of the 14 exposures topical [17]. Case reports of potent topical corticosteroid overuse causing Cushing syndrome are not confined to infants:

similar presentations have been described in older children and adults after unsupervised use of potent agents obtained over the counter, underscoring that availability without prescription, not age alone, drives much of this literature [18,19]. Reviews repeat the finding that most reported paediatric cases arise from a single class-I (superpotent) molecule, clobetasol propionate, often within unlabelled fixed-combination antifungal or antibiotic creams sold for diaper dermatitis or scabies [6,15,14]. In adults, a systematic review of 3753 corticosteroid users across all routes found adrenal insufficiency prevalence rising with cumulative dose and duration of exposure (1.4% for treatment under 28 days versus 27.4% for over 1 year), and this literature is frequently, if imperfectly, extrapolated to justify concern about topical paediatric exposure despite the adult-only denominator [20].

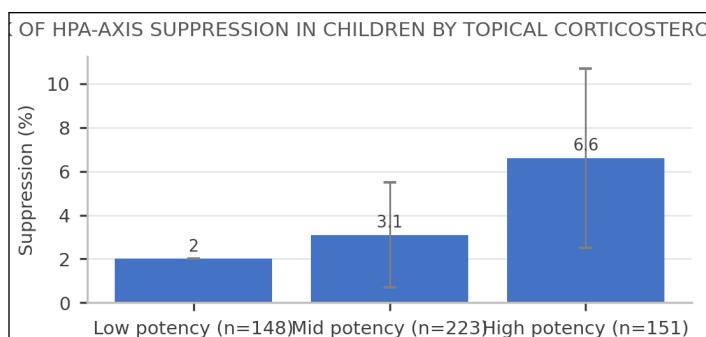


Figure 1: Pooled HPA-axis suppression in 522 children by topical corticosteroid potency class: 2.0% low, 3.1% (95% CI 1.5-6.3) mid, 6.6% (95% CI 3.6-11.8) high potency; overall pooled 3.8% (95% CI 2.4-5.8) [7].

Pathophysiology

Percutaneous absorption of corticosteroid depends on the molecule's lipophilicity and potency, the integrity and thickness of the stratum corneum, the surface area treated relative to body mass, and occlusion. Infant skin at birth and through the first year is thinner than adult skin, and the diaper itself functions as an occlusive dressing that markedly increases penetration compared with exposed skin [3,4]. Classic physiological studies documented measurable suppression of adrenocortical responsiveness

in infants treated with hydrocortisone for severe skin disease, and showed that absorption varies with age and with the severity of the underlying dermatitis, with more inflamed, barrier-disrupted skin absorbing more drug [3,4]. Earlier work using potent topical steroids similarly demonstrated measurable falls in plasma cortisol in infants and children with eczema, establishing decades ago that cutaneous corticosteroid is not pharmacologically inert once applied to compromised or occluded infant skin [21]. Absorbed corticosteroid suppresses hypothalamic corticotrophin-releasing hormone and pituitary ACTH secretion through negative feedback; with sustained suppression the adrenal cortex itself becomes atrophic and unresponsive to ACTH stimulation, producing the biochemical signature seen uniformly across the published infant cases — low basal cortisol together with low or inappropriately normal ACTH, i.e. secondary (central) rather than primary adrenal insufficiency [15,10,12,9]. Children with atopic dermatitis may also have intrinsically altered glucocorticoid sensitivity independent of exogenous exposure, a variable that complicates interpretation of HPA testing in this population [22]. Once the exogenous steroid is stopped, the child is left, at least transiently, without adequate endogenous cortisol production, which is the physiological basis for the risk of adrenal crisis on abrupt withdrawal [23,6].

Clinical Presentation

Reported infant cases share a strikingly consistent phenotype: progressive facial rounding and puffiness ("moon facies"), generalised or truncal adiposity with prominent cheeks, hirsutism or hypertrichosis (often most visible on the forehead and back), cutaneous striae, telangiectasia, and localised skin changes (atrophy, hypopigmentation, or acneiform change) at the site of application [5,10,11,13,14]. Hypertension for age has been documented repeatedly [12,11,24]. A distinctive and diagnostically important pattern is growth failure that can

occur with or without weight gain: one infant showed frank linear growth arrest (height -3.6 SD) despite normal weight after seven months of superpotent clobetasol to the groin, illustrating that failure to thrive is not incompatible with iatrogenic Cushing syndrome [12]. Cardiac involvement has been reported in a young infant with clobetasol-induced Cushing syndrome, who presented with left ventricular hypertrophy and pericardial effusion [8]. Two further infant cases from overuse of topical steroid confined to the diaper area were reported together as a paired case series, reinforcing the diaper region as the single most common site of exposure in this literature [25]. Several clusters arose from misdiagnosis: eight infants developed cushingoid features after a fixed-combination betamethasone/clotrimazole/gentamicin cream was applied for undiagnosed scabies over one to eight months, underscoring that inappropriate use for an unrecognised primary dermatosis is a recurring precipitant [14,11]. No verified paediatric case in the reviewed literature documented hyperglycaemia as a presenting feature, and glucose was explicitly normal where reported; hypertension, not dysglycaemia, is the more consistently documented cardiovascular sign in infants [15,11].

Report	Age at presentation	Preparation / potency	Site / duration	Outcome after withdrawal
Matejek 2022, case 1	6 mo, M	Clobetasol propionate 0.05%, very potent	Whole body, 2 g/day, >=4 wk	Not recovered at 3 wk; lost to follow-up
Tempark 2010	8 mo, F	Clobetasol, very potent	Diaper area, not stated	Hydrocortisone 3 mo, then stopped once normal
Jevalikar 2016	8 mo, F	Clobetasol propionate 0.05% combination, superpotent	Diaper/buttocks, 3 mo	Hydrocortisone; facies and cortisol recovered by 3 mo
Kuikel 2022	15 mo	Clobetasol 0.05%, very potent	Widespread, not stated	Prednisolone taper 45 d; resolved by 3 mo
Razavi 2014	17 mo, M	Clobetasol propionate 0.05%, superpotent	Groin, ~28 g/wk, 7 mo	Hydrocortisone taper 2 mo; catch-up growth by 22 mo
Almansour 2026	18 mo, F	Clobetasol propionate, high potency	Diaper area, prolonged	Monitoring only; partial recovery
Estrada-Chavez 2018, n=8	3 mo-1.5 y	Betamethasone combination, potent	Affected areas, 1-8 mo	Antiscabetic treatment; limited follow-up
Kilci 2025, n=7	3-60 mo	Clobetasol propionate, very potent	Not stated, median 2 mo	Hydrocortisone; median recovery 6 mo

Table 1: Published cases and case series of iatrogenic Cushing syndrome from topical corticosteroid use in infants reviewed here [8,9,5,25,15,6,10,26,11,12,13,19,14,16,24].				
Report	Age at presentation	Preparation / potency	Site / duration	Outcome after withdrawal
Ho 2014	2.5 mo	Clobetasol propionate, very potent	Napkin area, not stated	Steroid stopped; outcome not stated
Alkhuder 2019	3.5 mo, F	Clobetasol propionate, very potent	Napkin area, 5-8x/day, 2 mo	Hydrocortisone taper; axis recovered by week 5
Ahmed 2021	4 mo, F	Betamethasone, potent	Diaper area, 5-8x/day, 3 mo	Steroid stopped, physiological hydrocortisone started
Semiz 2008, n=2	Not stated (infants)	Topical steroid, not stated	Diaper area, not stated	Not stated

Diagnostic Approach

Suspicion should be raised by the combination of cushingoid facies or body-habitus change in an infant with a history, sometimes only elicited on direct questioning, of regular application of a potent topical corticosteroid, frequently obtained without prescription or used within a combination antifungal/antibacterial cream whose steroid component the caregiver did not recognise [6,10,11]. Biochemical confirmation follows the same principles used for endogenous Cushing syndrome, adapted to the fact that the driving process is exogenous and typically causes HPA-axis suppression rather than autonomous cortisol excess [23]. Reported infants have shown low or undetectable basal (morning) serum cortisol

together with low or inappropriately normal ACTH — for example, cortisol 0.22 µg/dL with ACTH 5.9 pg/mL in one napkin-area case, and cortisol <0.3 ng/mL with an ACTH-stimulated peak of only 0.4 ng/mL in another [10,12]. Dynamic ACTH stimulation testing has been used to confirm the degree and reversibility of suppression and to guide the timing of steroid withdrawal [9,15]. Because the population most often affected is preverbal and the exposure history depends entirely on caregiver recall, a deliberately structured medication and product history — including topical antifungal, antibacterial, and "diaper cream" products that may contain unlabelled corticosteroid — is an essential diagnostic step [14,26].

Table 2: Biochemical evidence of adrenal suppression and glucocorticoid replacement given, by case [10,12,9,15,24,17,16].				
Report	Basal cortisol (reference range)	ACTH (reference range)	Replacement given	Document ed recovery
Jevalikar 2016	0.22 ug/dL (4.5-22.6)	5.9 pg/mL	Hydrocortisone 6 mg/m2/day	Cortisol 10.9 ug/dL at 3 mo
Razavi 2014	<0.3 ng/mL (60-280)	Stimulated peak 0.4 ng/mL (>180)	Hydrocortisone 10 mg/m2/24h, 2-mo taper	Cortisol 101.2 ng/mL at 2 mo
Alkhuder 2019	17 ug/dL	0.7 pg/mL basal	Hydrocortisone 12 mg/m2, 6 wk then taper	Stimulated cortisol 28 ug/dL at wk 5
Matejek 2022, case 1	14 nmol/L (155-552)	1.3 pmol/L (2.2-11.0)	Hydrocortisone 9 mg/m2/day	Not recovered at 3 wk
Santos-Oliveira 2025	0.20 ug/dL (6.2-19.4)	3.6 ng/L (7.2-63.3)	Taper over 3 mo, no replacement stated	Cortisol 12.0 ug/dL at 9 mo
Güven 2020, n=14	Median 3.73 ug/dL	Not stated	Hydrocortisone, dose not uniformly stated	Median 60 days (range 30-780)
Kilci 2025, n=7	Not stated per case	Not stated per case	Hydrocortisone, median 10 mg/m2/day (7.5-50)	Median 6 months

Management

The unifying management principle across the published

cases is that the causative topical corticosteroid must be discontinued, but not abruptly, because the child's endogenous HPA axis is typically already suppressed and cannot immediately compensate; abrupt cessation risks acute adrenal insufficiency (adrenal crisis) [6,23]. Most reported infants were therefore covered with physiological-dose hydrocortisone replacement during withdrawal, commonly in the range of 6-12 mg/m /day in individual case reports and a median of 10 mg/m /day (range 7.5-50) across a seven-patient series, with gradual tapering guided by clinical and biochemical recovery [10,9,12,16]. Concurrent treatment of the underlying dermatological indication — for example antiscabetic therapy where scabies had been misdiagnosed and mistreated with steroid — is required alongside steroid withdrawal [11,14]. Broader paediatric dermatology guidance similarly emphasises using the lowest effective potency, limiting duration and surface area, and avoiding occlusion or combination products of unclear composition in infants, particularly in the diaper area [1,27,28]. Where topical corticosteroid is used therapeutically under monitored conditions, such as structured wet-wrap protocols for atopic dermatitis, dosing and duration are deliberately constrained specifically to avoid the exposures seen in the unsupervised case literature [28]. Families and prescribers should be counselled explicitly that visible efficacy does not indicate systemic safety, and that any once-daily, low-potency regimen is preferable to frequent (five-to-eight-times-daily) application of superpotent agents, a pattern present in several of the most severe reported infant cases [9,5].

Outcomes/Prognosis

Where documented, HPA-axis recovery and resolution of cushingoid features after appropriate withdrawal and hydrocortisone cover has generally been favourable. In a cohort of 14 children with iatrogenic Cushing syndrome (10 topical exposures), median time to HPA-axis recovery

was 60 days (range 30-780) [17]. Pooled literature spanning 22 paediatric and 21 adult cases over 35 years reported a mean recovery time of 3.49 ± 2.92 months in children, similar to adults (3.84 ± 2.51 months); a subsequent case-report series drawing on the same broader literature pool of 43 clobetasol-associated cases cited an equivalent 3.49-month mean (range 1-12 months), representing the same underlying dataset rather than an independent replication [6,15]. A more recent series of seven children reported a longer median recovery of six months [16]. Individual reports document full clinical and biochemical normalisation: catch-up linear growth and normalised cortisol by 22 months in the infant with growth arrest [12], resolution of facial and pigmentary changes with cortisol recovery to $10.9 \mu\text{g/dL}$ by three months in a napkin-area case [10], and normalisation of the steroid axis within weeks after dose reduction in a toddler exposed to potent dexamethasone eye drops [15]. No verified case of adrenal crisis following abrupt topical-corticosteroid cessation in an infant was identified in the reviewed literature, though several authors explicitly state that replacement hydrocortisone was given specifically to prevent this complication, reflecting recognised theoretical risk rather than a documented paediatric event in this dataset [6,23].

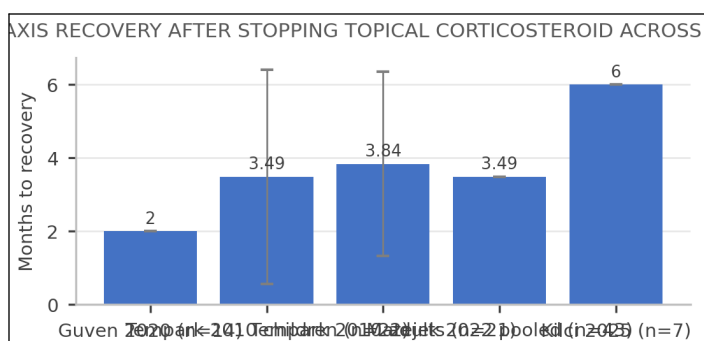


Figure 2: Time to HPA-axis recovery after withdrawal of topical corticosteroid across published series: median 2.0 months (n=14) [17]; mean 3.49 ± 2.92 months in children and 3.84 ± 2.51 months in adults from a pooled 35-year literature series [6]; mean 3.49 months (range 1-12) from a 43-case pooled series [15]; median 6.0 months (n=7) [16].

Conclusion

Iatrogenic Cushing syndrome from topical corticosteroids in infants is a preventable, reversible condition that arises almost exclusively from unsupervised, prolonged application of superpotent agents — most often clobetasol propionate — to the diaper area, frequently within unlabelled combination products. The consistent biochemical picture of secondary adrenal insufficiency means that the central management risk is not the Cushing syndrome itself but abrupt withdrawal of the child's only circulating glucocorticoid; recognition, careful biochemical confirmation, and a physiological hydrocortisone taper over weeks to months are the mainstays of safe recovery. Clinicians should maintain a low threshold to ask about topical steroid and combination-cream use in any infant presenting with unexplained cushingoid features, growth arrest, or hypertension, and should counsel caregivers explicitly on potency, duration, and site limits when prescribing or dispensing topical corticosteroids for infants.

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