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Clinical Pharmacology and Therapeutic Applications of Topical Corticosteroids in Contemporary Pharmacy Practice-An Updated Review for Pharmacists

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Abstract

Background: Topical corticosteroids (TCS) are cornerstone therapies in dermatology owing to anti-inflammatory, immunosuppressive, antiproliferative, and vasoconstrictive actions. They are indicated across steroid-responsive dermatoses, with potency, vehicle, site, and duration determining benefit–risk.

Aim: To provide pharmacists with an updated, practice-focused review of the clinical pharmacology, therapeutic indications, dosing, safety, and patient counseling considerations for TCS.

Methods: Narrative synthesis of regulatory labeling, dermatology guidelines, and key clinical literature on mechanisms, pharmacokinetics, potency classification, administration principles, dosing by fingertip units (FTUs), special populations, adverse effects, contraindications, monitoring, and interprofessional roles.

Results: TCS effectively manage psoriasis, eczematous disorders, lichen planus/ simplex, discoid lupus, lichen sclerosus, alopecia areata, phimosis, and radiation dermatitis; selected off-label uses (e.g., scalp psoriasis maintenance; limited evidence in urticaria) may be appropriate with oversight. Pharmacokinetics are driven by percutaneous absorption (affected by vehicle, site thickness, inflammation, occlusion). Seven potency classes guide selection; once- or twice-daily application suffices. FTU-based dosing standardizes quantity. Risks include local atrophy, striae, perioral dermatitis, tachyphylaxis, and rare systemic HPA suppression—heightened in children, older adults, thin or damaged skin, high potency, large areas, or occlusion. Absolute contraindications include untreated bacterial infections.

Conclusion: Rational agent/vehicle selection, limited duration, site-appropriate potency, FTU dosing, and vigilant monitoring optimize outcomes and minimize harms. Pharmacists are pivotal in education, adherence support, and safety surveillance.

Keywords: topical corticosteroids; potency classes; fingertip unit; pharmacokinetics; adverse effects; dermatology; pharmacy practice; patient counseling.

Introduction

Topical corticosteroids remain a foundational therapy in dermatologic practice due to their anti-inflammatory, immunosuppressive, antiproliferative, and vasoconstrictive properties. These pharmacodynamic effects make them highly effective in managing inflammatory and pruritic dermatoses. Their therapeutic role extends across a broad spectrum of acute and chronic dermatologic disorders characterized by immune dysregulation, epidermal hyperproliferation, and inflammatory cell infiltration. Regulatory approval by the United States Food and Drug Administration supports their use in steroid-responsive dermatoses, reinforcing their position as first-line or adjunctive therapy in many clinical settings [1][2][3][4][5][6]. Among the most established indications is psoriasis, particularly plaque-type disease. In psoriasis, keratinocyte hyperproliferation and T-cell–mediated inflammation drive lesion formation. Topical corticosteroids suppress proinflammatory cytokine expression, reduce epidermal turnover, and alleviate erythema and scaling. Potent agents are frequently employed for thick plaques located on extensor surfaces, whereas lower-potency preparations are preferred for sensitive areas to minimize adverse effects. Limited areas of vitiligo also respond to topical corticosteroids, especially during early inflammatory stages. By modulating local immune activity directed against melanocytes, these agents may promote repigmentation in selected patients [1][2].

Eczematous disorders represent another principal indication. Atopic dermatitis, contact dermatitis, and other eczema variants involve barrier dysfunction and immune-mediated inflammation. Topical corticosteroids decrease pruritus, reduce erythema, and restore skin integrity. Clinical guidelines from the American Academy of

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Dermatology recommend their use as primary anti-inflammatory therapy in adults with atopic dermatitis. Intermittent application of medium-potency agents twice weekly as maintenance therapy has been shown to reduce relapse frequency and disease flares [11]. This proactive approach reflects an evolution from reactive treatment toward long-term disease control strategies. Other inflammatory dermatoses, including lichen planus, lichen simplex chronicus, discoid lupus erythematosus, and lichen sclerosus, also benefit from topical corticosteroid therapy [3][4][5]. In lichen planus, corticosteroids attenuate T-cell–mediated epidermal damage and relieve associated pruritus. In lichen simplex chronicus, they interrupt the itch-scratch cycle by decreasing inflammation and skin thickening. Discoid lupus erythematosus, a chronic autoimmune condition, often requires high-potency topical corticosteroids to suppress localized immune activity and prevent scarring. Lichen sclerosus, particularly in anogenital involvement, responds favorably to potent formulations, which reduce inflammation and prevent disease progression when used under close supervision.

Alopecia areata represents another immune-mediated indication. Topical corticosteroids suppress peribulbar lymphocytic infiltration, thereby promoting hair regrowth in limited patchy disease [6]. Although intralesional corticosteroids are often preferred for extensive involvement, high-potency topical agents remain a valuable alternative in selected patients who cannot tolerate injections. Topical corticosteroids are also indicated for phimosis, where inflammation contributes to foreskin tightness. Short-term application of moderate-to-high-potency agents can reduce inflammation and facilitate retraction, potentially avoiding surgical intervention. In acute radiation dermatitis, these agents mitigate inflammatory skin reactions associated with radiotherapy, reducing erythema, edema, and discomfort [2]. Specific FDA-approved agents demonstrate tailored indications. Alclometasone, desonide, desoximetasone, diflorasone, fluocinolone, halcinonide, halobetasol, hydrocortisone, and flurandrenolide are indicated for inflammation and pruritus associated with steroid-responsive dermatoses. Clobetasol is specifically indicated for moderate to severe plaque-type psoriasis and other steroid-responsive conditions. Fluticasone and mometasone, while available in topical formulations for dermatoses, also possess respiratory indications in nasal and inhaled forms for allergic rhinitis and asthma, reflecting their versatile anti-inflammatory activity [8][9]. Triamcinolone, in addition to dermatologic use, is indicated for inflammatory oral lesions when formulated for dental application [10].

The pharmacologic effectiveness of topical corticosteroids derives from their ability to inhibit phospholipase A2 activity, suppress arachidonic acid pathways, and reduce production of prostaglandins and leukotrienes. They also decrease expression of adhesion molecules and inflammatory mediators, thereby limiting leukocyte migration to affected tissues [7]. This broad anti-inflammatory action explains their efficacy across disorders characterized by immune activation and cutaneous inflammation. Off-label applications further expand their clinical utility. According to joint guidelines from the American Academy of Dermatology and the National Psoriasis Foundation, class 1 through class 5 topical corticosteroids may be used for up to four weeks in plaque psoriasis not involving intertriginous areas [12]. Scalp psoriasis often requires at least four weeks of therapy due to hair-bearing anatomy and increased barrier thickness. Extended use beyond twelve weeks may be considered under dermatologic supervision to balance efficacy and safety. Emerging evidence also suggests potential benefit in urticaria management. A systematic review and meta-analysis indicate that moderate-to-high-potency topical corticosteroids may reduce urticaria severity and improve pruritus compared with placebo; however, the certainty of evidence remains limited [13]. Reported adverse event rates were comparable between treatment and control groups, underscoring the need for further investigation before widespread adoption in this indication.

From a pharmaceutical care perspective, potency selection, vehicle formulation, anatomical site, patient age, and duration of therapy must be considered when recommending topical corticosteroids. Ointments enhance penetration and hydration, making them suitable for chronic, lichenified lesions. Creams and lotions may be preferred for moist or intertriginous areas. Foam and solution formulations facilitate scalp application. Appropriate matching of potency class to disease severity and body site reduces the risk of cutaneous atrophy, telangiectasia, and hypothalamic-pituitary-adrenal axis suppression. In summary, topical corticosteroids are integral to the management of numerous dermatologic conditions characterized by inflammation, immune dysregulation, and hyperproliferation. Their FDA-approved indications encompass psoriasis, eczema, lichen disorders, autoimmune dermatoses, and other steroid-responsive diseases [1–6][8–10]. Guideline-supported off-label applications further broaden their therapeutic scope [12][13]. Rational selection, appropriate duration, and careful monitoring remain essential to maximize clinical benefit while minimizing risk.

Mechanism of Action

Topical corticosteroids exert their therapeutic effects through a multifaceted mechanism involving anti-inflammatory, anti-mitotic, and immunosuppressive actions, which collectively address the underlying pathophysiology of inflammatory and immune-mediated skin disorders [14]. These pharmacological properties make them highly effective in the management of a wide range of dermatologic conditions, including psoriasis, atopic dermatitis, lichen planus, and other steroid-responsive

dermatoses. The anti-inflammatory action of topical corticosteroids is central to their clinical efficacy. Vasoconstriction of dermal blood vessels reduces local blood flow, limiting the delivery of inflammatory mediators and leukocytes to affected tissues [14]. This effect is immediate and contributes to decreased erythema and edema. At a molecular level, corticosteroids induce the synthesis of lipocortin-1, a glucocorticoid-regulated protein that inhibits phospholipase A2, thereby reducing the production of arachidonic acid derivatives, including prostaglandins and leukotrienes [15]. These lipid mediators play critical roles in the inflammatory cascade, promoting vasodilation, leukocyte recruitment, and pruritus. By decreasing their availability, corticosteroids effectively attenuate local inflammation. Additionally, corticosteroids modulate gene expression by interacting with glucocorticoid receptors in the cytoplasm, which then translocate to the nucleus and bind to glucocorticoid response elements on DNA. This process upregulates anti-inflammatory genes and suppresses transcription factors such as NF-κB, resulting in reduced expression of pro-inflammatory cytokines, chemokines, and adhesion molecules [14].

The anti-mitotic effects of topical corticosteroids are particularly relevant in hyperproliferative disorders like psoriasis. Epidermal hyperplasia in psoriatic plaques is characterized by increased keratinocyte proliferation and abnormal differentiation. Corticosteroids decrease epidermal mitosis, partly through the upregulation of lipocortin, which indirectly inhibits cell cycle progression [16]. In the dermis, corticosteroids also reduce fibroblast proliferation and collagen synthesis, which contributes to decreased thickness and induration of chronic lesions [17]. This dual effect on both epidermal and dermal layers facilitates the rapid resolution of hyperproliferative and lichenified lesions. Immunosuppressive properties further enhance the efficacy of topical corticosteroids. They inhibit humoral immune responses by reducing cytokine secretion and antibody production while simultaneously suppressing the maturation, differentiation, and proliferation of immune cells, including T and B lymphocytes, macrophages, and dendritic cells [16]. This modulation limits both acute inflammatory responses and chronic immune-mediated tissue damage, reducing pruritus, erythema, and edema. Overall, the combined anti-inflammatory, anti-mitotic, and immunosuppressive mechanisms allow topical corticosteroids to target multiple pathological pathways simultaneously. This comprehensive action accounts for their effectiveness across a spectrum of inflammatory, hyperproliferative, and immune-mediated skin conditions, forming the foundation for their widespread use in dermatologic therapy [14][15][16][17].

Pharmacokinetics

The pharmacokinetic profile of topical corticosteroids is determined by the processes of absorption, distribution, metabolism, and elimination, each of which is influenced by both the drug's chemical characteristics and patient-specific factors. Percutaneous absorption is the primary determinant of systemic exposure. Factors such as the formulation, thickness of the stratum corneum, anatomical site of application, and skin integrity significantly affect absorption. Inflammation and disruption of the epidermal barrier increase corticosteroid penetration, while occlusive dressings or bandages enhance absorption by creating a hydrated environment that facilitates drug diffusion. Potency and lipid solubility of the corticosteroid also dictate the degree of percutaneous absorption, with more lipophilic and potent agents achieving higher systemic concentrations [18]. Once absorbed, corticosteroids distribute into the systemic circulation, where they bind variably to plasma proteins, primarily albumin and corticosteroid-binding globulin. Protein binding affects both the free active fraction of the drug and its overall pharmacologic activity. Tissue distribution tends to favor highly perfused organs, although the majority of topically applied corticosteroids exert their effects locally rather than systemically. Metabolism occurs primarily in the liver through oxidative and reductive pathways, producing inactive metabolites that reduce systemic exposure and minimize toxicity. Structural modifications to corticosteroid molecules, such as halogenation in clobetasol, prevent rapid de-esterification and extend local potency, allowing prolonged anti-inflammatory activity at the site of application. Topical corticosteroids may also undergo limited local metabolism in the skin, which can contribute to their clinical efficacy and safety profile [18]. Elimination of corticosteroids and their metabolites occurs predominantly through renal excretion, with a smaller proportion eliminated via the bile. The renal route ensures clearance of systemically absorbed corticosteroids, while biliary excretion facilitates removal of certain conjugated metabolites. Understanding these pharmacokinetic properties is essential for optimizing topical corticosteroid therapy, selecting appropriate potency, and minimizing systemic side effects, particularly in vulnerable populations such as infants, elderly patients, and those with extensive or damaged skin [18].

Administration

Topical corticosteroids are administered directly to the skin, and their clinical efficacy depends on several critical factors, including an accurate diagnosis, selection of the appropriate corticosteroid, choice of vehicle, potency, and frequency of application [1]. The vehicle serves as the carrier for the active drug and influences both patient adherence and percutaneous absorption. Selection of the vehicle is guided by the anatomical site, type of lesion, and desired pharmacokinetic profile. Certain vehicles

provide additional hydration, enhancing absorption and local drug activity, which is particularly important in hyperkeratotic or dry lesions [19]. Ointments are considered the most potent topical corticosteroid vehicles due to their occlusive properties, which allow for prolonged contact with the epidermis and enhanced penetration. They are particularly effective for thick, hyperkeratotic lesions such as chronic plaque psoriasis. However, ointments are not recommended for hair-bearing areas because their occlusiveness can trap bacteria and lead to folliculitis. Creams offer a less occlusive alternative and are cosmetically preferred for acute inflammatory conditions, exudative dermatitis, and intertriginous areas. Their non-greasy nature ensures patient comfort and compliance, although they are less potent than ointments due to reduced occlusion [1]. Lotions provide a lightweight, non-greasy formulation that is particularly useful for application over large areas or hair-bearing regions, as they facilitate uniform drug distribution without matting hair. Gels share similar properties with lotions, being non-occlusive and easy to spread, which makes them suitable for scalp application and other areas where hair interference is a concern. Their alcohol-based formulations may also provide a cooling effect, which can help alleviate pruritus and inflammation. Foams represent a specialized delivery system designed for efficient corticosteroid deposition on the scalp, achieving high local concentrations with minimal residue. Despite their clinical advantages, foams are more expensive and may not be accessible for all patients [20]. Effective administration also requires attention to application frequency and duration. Topical corticosteroids should be applied in a thin layer over affected areas, typically once or twice daily depending on potency, lesion severity, and the patient's age. Higher-potency corticosteroids are generally reserved for short-term use or for chronic lesions requiring intensive therapy, while lower-potency formulations are preferred for sensitive areas such as the face or intertriginous regions. Patient education regarding proper application techniques, avoidance of excessive use, and monitoring for local or systemic adverse effects is essential to optimize outcomes and minimize complications. Proper selection of both drug and vehicle, aligned with lesion type and site, ensures maximum therapeutic benefit while limiting the risk of side effects.

Strength, Classification, and Administration

The potency of topical corticosteroids is defined by the concentration and effectiveness of the drug in producing the desired therapeutic response, primarily through anti-inflammatory, anti-proliferative, and immunosuppressive mechanisms. Determination of potency is standardized using the vasoconstrictor assay, which measures the extent of cutaneous blanching as a proxy for the drug's vasoconstrictive and, consequently, anti-inflammatory effect [21]. This standardized assessment allows clinicians to compare corticosteroids objectively and guides the selection of the appropriate agent based on disease severity, anatomical site, and patient-specific factors. In the United States, topical corticosteroids are classified into seven potency classes, ranging from Class I, the superpotent agents, to Class VII, which includes the least potent preparations [22]. Class I corticosteroids are designated as superpotent and are typically reserved for recalcitrant or thick hyperkeratotic lesions, such as chronic plaque psoriasis. Examples include clobetasol propionate 0.05% in any vehicle, augmented betamethasone dipropionate 0.05% gel or ointment, diflorasone diacetate 0.05% ointment, fluocinonide 0.1% cream, and halobetasol propionate 0.05% cream or ointment [1]. These agents are highly effective but must be applied cautiously to avoid local adverse effects, particularly on thin or sensitive skin areas. Class II agents are high-potency corticosteroids, which provide strong anti-inflammatory effects suitable for moderate to severe dermatoses. This category includes amcinonide 0.1% ointment, augmented betamethasone dipropionate 0.05% cream or lotion, betamethasone dipropionate 0.05% ointment, desoximetasone in cream, gel, or ointment formulations, diflorasone diacetate 0.05% cream, fluocinonide 0.05% cream, gel, or ointment, and halcinonide 0.1% cream, ointment, or solution [1]. Clinicians typically reserve Class II corticosteroids for short-term use on thicker skin or areas resistant to lower-potency formulations.

Potency	Class	Generic (Brand)	Strength (%)
High	1	Betamethasone dipropionate	0.05
		Clobestasol propionate	0.05
		Halobetasol propionate	0.05
	2	Desoximetasone	0.05, 0.25
		Fluocinonide	0.05
Medium	3	Betamethasone valerate	0.1
		Triamcinolone acetate	0.1, 0.5
	4	Flurandrenolide	0.05
	5	Fluticasone propionate	0.05
Low	6	Desonide	0.05
	7	Hydrocortisone	0.5-2.5

Fig. 1: Topical Corticosteroids Classification.

Class III corticosteroids are categorized as medium- to high-potency, including amcinonide 0.1% cream, betamethasone dipropionate 0.05% cream, fluticasone propionate 0.005% ointment, and triamcinolone acetonide 0.5% cream or ointment [1]. These agents offer a balance between efficacy and safety, making them suitable for broader patient populations and less chronic lesions. Classes IV and V represent medium-potency corticosteroids. Examples include betamethasone valerate 0.1% cream, lotion, or foam; desoximetasone 0.05% cream; fluocinolone acetonide 0.025% cream or ointment; fluticasone

propionate 0.05% cream; hydrocortisone butyrate 0.1% ointment; hydrocortisone probutate 0.1% cream; hydrocortisone valerate 0.2% cream or ointment; mometasone furoate 0.1% cream, lotion, or ointment; and triamcinolone acetonide 0.025% or 0.1% cream, lotion, or ointment [1]. These corticosteroids are often used for widespread inflammatory conditions, where long-term management or maintenance therapy is needed. Class VI includes low-potency agents, such as alclometasone dipropionate 0.05% cream or ointment, desonide 0.05% in any vehicle, fluocinolone 0.01% cream, and hydrocortisone butyrate 0.1% cream [1]. These are preferred for sensitive skin areas, including the face, intertriginous zones, and in pediatric populations, as they minimize the risk of atrophy and systemic absorption.

Class VII corticosteroids, representing the least potent group, include hydrocortisone 1% and 2.5% in cream, lotion, or ointment forms [1]. These agents are often used for mild inflammatory conditions, maintenance therapy, or as initial treatment in vulnerable populations. Administration of topical corticosteroids should be individualized, taking into account potency, anatomical site, disease severity, and patient age. Superpotent and high-potency corticosteroids are generally reserved for limited-duration therapy on resistant lesions, whereas lower-potency agents are suitable for long-term use or application to sensitive areas. Proper selection, appropriate dosing, and monitoring for adverse effects are essential to achieve maximal therapeutic benefit while minimizing local or systemic complications. The absorption and permeability of topical corticosteroids are heavily influenced by the thickness of the epidermis at the site of application. Areas with thin epidermis, such as the eyelids, demonstrate significantly higher drug penetration compared to thicker regions, such as the soles of the feet. Studies have demonstrated that this difference in percutaneous absorption can vary by as much as 300-fold, emphasizing the importance of anatomical site selection in clinical practice [23]. Furthermore, diseased or inflamed skin enhances corticosteroid penetration by 2- to 10-fold due to disrupted epidermal barriers, desquamation, and increased vascularity [1][24]. These physiological differences necessitate careful consideration of both steroid potency and site of application to maximize therapeutic efficacy while minimizing local and systemic adverse effects.

High-potency corticosteroids are typically reserved for areas of thick stratum corneum, such as the palms and soles, or for non-facial, non-intertriginous regions affected by severe dermatoses, including psoriasis, severe atopic dermatitis, and contact dermatitis [1]. These potent agents achieve adequate tissue levels despite the barrier posed by the thick epidermis. Conversely, medium- to high-potency corticosteroids are suitable for thin epidermal regions or areas subject to occlusion, such as the eyelids and axillae, where excessive absorption can occur if potency is too high [20]. For large surface areas, low- to medium-potency corticosteroids are recommended to reduce the risk of systemic absorption and potential hypothalamic-pituitary-adrenal axis suppression [1]. The quantity of corticosteroid applied also significantly influences both therapeutic effectiveness and safety. The fingertip unit (FTU) serves as a practical measure, with one FTU equating to approximately 0.5 grams of ointment or cream. The recommended FTU dosage varies depending on the body region being treated, and adherence to proper dosing is critical to avoid local adverse effects such as skin atrophy, striae, and telangiectasia [25]. Frequency of application is another key consideration. Evidence from studies in atopic dermatitis indicates that applying topical corticosteroids more than once daily provides no additional clinical benefit but does increase the risk of adverse effects [26]. Consequently, once- or twice-daily administration remains the standard recommendation for most dermatologic conditions, balancing efficacy with safety. Overall, understanding the interplay between epidermal thickness, disease state, potency, and application parameters is essential for optimizing corticosteroid therapy. Tailoring treatment to these variables ensures adequate therapeutic outcomes while mitigating the potential for systemic absorption and local complications.

Dosage:

The dosage of topical corticosteroids should be carefully tailored to the treated body area to optimize therapeutic effects while minimizing adverse events. According to the American Academy of Family Physicians (AAFP) guidelines, the fingertip unit (FTU) is a practical method for quantifying the amount of ointment or cream required for one application. One FTU corresponds to approximately 0.5 grams of topical medication applied from the distal crease to the tip of an adult index finger. This standardized measurement ensures accurate dosing across various body regions [27]. For a single application, the recommended FTUs vary depending on the anatomical site. The face and neck require approximately 2.5 FTUs, reflecting the sensitivity and smaller surface area of these regions. Larger areas such as the front and back of the trunk each require seven FTUs due to their extensive surface area. Each arm generally requires three FTUs, while one hand, including both the palmar and dorsal surfaces, needs one FTU. These recommendations provide a practical framework to avoid over- or under-application, which could either lead to local side effects or suboptimal therapeutic response [27]. When considering chronic use, particularly twice-daily application over 30 days, the cumulative weight of the corticosteroid must be calculated. For the face and neck, the total required weight is approximately 75 grams. Larger areas, including the front or back of the trunk, require 210

grams for the same period. One arm necessitates 90 grams, and one hand 30 grams. These calculations assist clinicians and patients in planning appropriate supply, ensure adherence to treatment regimens, and help mitigate the risks of systemic absorption or local adverse effects from excessive use. By adhering to FTU-based dosing and calculating cumulative requirements, clinicians can provide safe, effective, and standardized corticosteroid therapy across various dermatologic conditions [27].

Specific Patient Populations

Topical corticosteroid therapy in pregnancy has been extensively studied, and current evidence indicates that maternal use, irrespective of potency, is generally not associated with significant adverse outcomes for the fetus. Systematic reviews suggest that the majority of topical corticosteroids, when used appropriately, do not increase the risk of congenital anomalies or major obstetric complications. Nevertheless, there remains a potential association between cumulative exposure to high-potency corticosteroids and the risk of reduced birth weight. This finding underscores the need for careful monitoring of dosage and duration in pregnant patients, and it highlights the necessity for further research to clarify the impact of prolonged or high-dose topical corticosteroid use during gestation [28].Regarding lactation, short-term application of topical corticosteroids is unlikely to pose a substantial risk to the breastfeeding infant. Extensive or prolonged use of potent formulations may lead to systemic absorption in the mother, potentially affecting the neonate. Clinical recommendations emphasize the use of the least potent corticosteroid on the smallest necessary area of skin, with precautions to avoid direct contact between the treated skin and the infant. When topical corticosteroids are applied to the nipple or areola, water-miscible preparations such as creams or gels are preferred, and the area should be thoroughly cleansed prior to nursing to minimize exposure [29][30]. Pediatric patients represent a particularly sensitive population due to a relatively higher skin surface area-to-body weight ratio, which increases the risk of systemic absorption. Very potent topical corticosteroids should be reserved for cases under specialist dermatological supervision. For conditions such as mild atopic dermatitis, treatment should begin with low-potency corticosteroids, reserving higher-potency agents for acute flares. Maintenance therapy with intermittent application of mild corticosteroids, for example twice weekly, has been shown to reduce the frequency of flares while minimizing systemic risks [31]. Older adults also require careful dosing strategies due to age-related changes in skin structure, including increased fragility and altered barrier function, coupled with a relatively higher surface area-to-weight ratio. These factors elevate the risk of systemic absorption and adverse effects, necessitating cautious selection of potency, frequency, and duration of topical corticosteroid therapy in this population [20].

Adverse Effects

Topical corticosteroids are widely used in dermatologic therapy; however, their prolonged or inappropriate use can lead to a spectrum of adverse effects, which can be categorized as local or systemic. Local adverse effects are primarily influenced by the potency of the corticosteroid, the vehicle used for delivery, the frequency and duration of application, and the anatomical site of administration. These effects often emerge with chronic application and include skin atrophy, striae, steroid-induced rosacea, perioral dermatitis, acneiform eruptions, and purpura [17]. Skin atrophy represents the most common local adverse effect and results from the anti-mitotic and vasoconstrictive actions of corticosteroids [17]. The progression of corticosteroid-induced skin changes occurs in three phases: pre-atrophy, atrophy, and tachyphylaxis. Persistent use on the same site leads to thinning of the epidermis and resorption of the dermal ground substance, causing loss of connective tissue integrity. Clinically, atrophic skin manifests as erythema, telangiectasias, purpura, and a burning sensation. Paradoxically, the vasoconstrictive property of corticosteroids can temporarily alleviate this burning sensation [15]. Intertriginous areas, including axillae and groin, are particularly susceptible due to thinner epidermis and occlusive conditions. Although cessation of therapy can reverse atrophy, complete recovery may take several months, underscoring the need for judicious use [17]. Tachyphylaxis is another phenomenon resulting from repeated corticosteroid exposure, leading to diminished vasoconstrictive response in the capillaries. Capillary responsiveness typically returns after approximately four days, which is why pulse therapy or brief discontinuation is often recommended when efficacy diminishes [25].

Striae formation occurs due to mechanical stress and dermal injury. Chronic inflammation and edema contribute to collagen deposition in affected regions, producing histologically permanent scarring [17]. Similarly, acneiform eruptions are facilitated by follicular epithelium degradation and increased free fatty acid concentration on the skin surface, creating an environment conducive to bacterial proliferation and comedone formation [17]. Steroid-induced rosacea may develop when corticosteroids are applied for erythematous lesions with or without pustules. Low-dose corticosteroid therapy can temporarily control symptoms; however, persistent flares may necessitate escalation to higher-potency agents, perpetuating the condition [17]. Perioral dermatitis is commonly observed with prolonged corticosteroid application on the face. It presents as follicular papules and pustules on an erythematous base surrounding the perioral region, sparing the vermillion border [17]. Less frequent local adverse effects include hypertrichosis, pigmentary alterations, and delayed wound healing. Systemic adverse effects are rare due

to the generally low percutaneous absorption of topical corticosteroids. Nonetheless, high-potency agents applied extensively or on thin-skinned areas can lead to significant systemic exposure. Potential systemic complications include hypothalamic-pituitary-adrenal (HPA) axis suppression, Cushing syndrome, hypertension, hyperglycemia, and, in ocular applications, glaucoma [17]. Drug-drug interactions with topical corticosteroids are uncommon but warrant consideration. Certain agents, including hydrocortisone, fluocinolone, and mometasone, may interact with systemic drugs such as live vaccines, insulin, methotrexate, and diuretics. Beclomethasone and clobetasol may require caution when co-administered with ketoconazole or rifampicin. Despite these potential interactions, most topical corticosteroids remain safe when administered in accordance with labeling instructions and recommended dosing regimens [32]. In conclusion, while topical corticosteroids remain essential in dermatologic therapy due to their anti-inflammatory, anti-mitotic, and immunosuppressive properties, awareness of their adverse effect profile is crucial. Clinicians must balance efficacy with safety by selecting appropriate potency, limiting treatment duration, and monitoring at-risk populations to minimize both local and systemic complications. Understanding the pharmacologic nuances, including regional absorption differences, vehicle effects, and patient-specific factors, is critical to optimizing outcomes and preventing long-term adverse sequelae.

Contraindications

Topical corticosteroids are contraindicated in the presence of bacterial skin infections because their anti-inflammatory and vasoconstrictive properties can mask the underlying infection, delaying diagnosis and appropriate treatment. Conditions such as impetigo, cellulitis, erysipelas, lymphangitis, furuncles, and carbuncles represent absolute contraindications for corticosteroid application. Their use in these infections may worsen outcomes by suppressing the local immune response and allowing bacterial proliferation. Relative contraindications include fungal infections, such as candidiasis and dermatophytosis, where the immunosuppressive effect of corticosteroids can perpetuate infection. In some cases, inappropriate corticosteroid use in fungal infections leads to tinea incognito, characterized by atypical morphology with increased inflammation, pustule formation, and rapid spread of the dermatophyte, posing additional diagnostic and therapeutic challenges [20][33].

Warnings and Precautions

Chronic misuse of topical corticosteroids, particularly for cosmetic purposes, can result in a withdrawal phenomenon. Although poorly characterized and relatively rare, corticosteroid withdrawal manifests with erythema, pruritus, burning sensations, and secondary scaling. Patients may require education on the appropriate duration and application of corticosteroids to prevent this condition [34]. Endocrine adverse effects are an important consideration with high-potency topical corticosteroids. Systemic absorption can suppress the hypothalamic-pituitary-adrenal (HPA) axis, particularly in cases of prolonged use, large application areas, or application over compromised skin barriers. Monitoring may be required for adrenal insufficiency, which can necessitate systemic corticosteroid supplementation. Additionally, systemic absorption may precipitate Cushing syndrome, hyperglycemia, or unmask latent diabetes, with pediatric populations being particularly susceptible due to a higher skin surface area-to-body weight ratio [20]. Local adverse reactions are common and include skin thinning, striae, telangiectasias, folliculitis, acneiform eruptions, hypertrichosis, perioral dermatitis, hypopigmentation, and miliaria. Occlusion, prolonged application, or high-potency formulations increase the likelihood of these effects. Allergic contact dermatitis should be considered when a patient's condition fails to improve. Patch testing can confirm corticosteroid allergy, and discontinuation of the offending agent with initiation of alternative therapy is recommended. Concomitant skin infections require temporary cessation of topical corticosteroids until the infection is adequately managed with appropriate antibacterial or antifungal agents. This precaution ensures that underlying infectious processes are not exacerbated by immunosuppression.

Monitoring

Careful monitoring of patients using topical corticosteroids is essential to minimize the risk of both local and systemic adverse effects. Treatment duration should generally not exceed two to four weeks, regardless of steroid potency, with high-potency formulations limited to two weeks before tapering is considered necessary to prevent complications [1][25]. Adherence to clinical guidelines is critical, including the preferential use of lower-potency steroids, restricting applications to morning hours, employing alternate-day dosing schedules, and minimizing the use of occlusive dressings [35]. These measures collectively reduce the likelihood of cutaneous atrophy, striae, telangiectasias, and systemic complications, particularly in vulnerable populations such as pediatric and elderly patients. Regular assessment of the treated areas, including evaluation for signs of irritation, erythema, or excessive thinning, supports timely intervention.

Toxicity

Systemic absorption of topical corticosteroids can lead to hypothalamic-pituitary-adrenal (HPA) axis suppression, hyperglycemia, glucosuria, and features of Cushing syndrome, such as central obesity, moon facies, and cutaneous changes. Pediatric patients are particularly susceptible due to a higher skin surface area-to-body weight ratio, which increases systemic exposure. The risk is amplified

with prolonged use, application over large surface areas, or when occlusive dressings are employed. Management of overdose involves monitoring for HPA axis suppression through urinary-free cortisol levels and ACTH stimulation testing. Adjustments may include reducing application frequency or transitioning to a lower-potency steroid. Discontinuation of the offending corticosteroid generally allows recovery of adrenal function; however, some patients may experience steroid withdrawal, necessitating temporary systemic corticosteroid supplementation. Local irritation should prompt immediate cessation of the topical agent and appropriate treatment of the affected skin [36].

Enhancing Healthcare Team Outcomes

Topical corticosteroids are widely used in dermatology due to their rapid ability to alleviate inflammation, pruritus, and other distressing symptoms. Despite their effectiveness, improper prescription or insufficient patient education can lead to misuse and adverse effects [37]. Optimal management of these medications requires coordinated efforts from dermatologists, nurses, general practitioners, and pharmacists to ensure correct application and adherence to treatment protocols. However, in practice, verbal instructions from clinicians are often forgotten, and patients frequently rely solely on product labeling, which can be vague or anxiety-inducing, such as instructions to "apply sparingly" or "use thinly." Such wording may lead to underuse, poor adherence, and suboptimal treatment outcomes. To address this, the fingertip unit (FTU) system has been developed to standardize and simplify patient education regarding topical corticosteroid dosing. By demonstrating the appropriate amount of medication for different body regions using FTUs, practitioners can improve patient confidence and compliance. Recommendations suggest including images of FTUs and charts on packaging to clearly illustrate the quantity needed for specific areas, along with instructions to discontinue use under medical supervision if symptoms resolve [37]. Pharmacists occupy a critical role as the final point of contact before patients initiate therapy. They ensure that patients understand dosage, application frequency, and safety measures, reinforcing the guidance provided by the interprofessional team. Effective communication between clinicians, nurses, and pharmacists supports adherence, reduces the risk of adverse effects, and improves clinical outcomes. Implementing an interprofessional approach ensures that topical corticosteroids are used safely, efficiently, and effectively, optimizing therapeutic benefits while minimizing potential harm [38].

Conclusion:

Topical corticosteroids remain indispensable across a spectrum of inflammatory and immune-mediated dermatoses, provided their use is individualized and time-limited. Optimal practice hinges on matching potency and vehicle to lesion type and anatomical site, applying once or twice daily, and using FTU-based quantities to achieve efficacy while curbing toxicity. Awareness of absorption modifiers—thin skin, inflamed or disrupted barrier, occlusion, large treatment areas, and higher potencies—is essential to prevent local atrophy, striae, perioral dermatitis, and the rare but serious risk of HPA-axis suppression. Special consideration is warranted in pediatrics, pregnancy/lactation, and older adults. Absolute avoidance in untreated bacterial infections and caution in fungal disease help avert tinea incognito and diagnostic delay. As accessible medication experts, pharmacists should reinforce clear instructions, dispel "steroid phobia" and misuse, monitor for red flags, and coordinate with prescribers on tapering, maintenance strategies, and alternatives. Through precise selection, dosing, and education, pharmacists can maximize therapeutic benefit and minimize harm.

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