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Review Article

### CORTICOSTEROIDS: GENERAL KNOWLEDGE FOR ALL HEALTHCARE PROFESSIONALS-UPDATED REVIEW

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**Background:** Corticosteroids are synthetic analogs of adrenal steroid hormones with potent anti-inflammatory and immunosuppressive properties, widely used across medical specialties. Their therapeutic applications span endocrine disorders, inflammatory conditions, and autoimmune diseases, but their use is complicated by significant adverse effects.

**Aim:** This review synthesizes current knowledge on corticosteroid pharmacology, clinical applications, adverse effects, contraindications, and monitoring strategies to guide healthcare professionals in optimizing therapy while minimizing risks.

**Methods:** A comprehensive literature review was conducted, analyzing peer-reviewed articles, clinical guidelines, and pharmacological data on corticosteroid mechanisms, administration routes, toxicity profiles, and monitoring protocols.

**Results:** Corticosteroids exert effects via genomic and non-genomic pathways, with routes of administration tailored to clinical needs (oral, parenteral, inhaled, topical). Adverse effects—including osteoporosis, HPA axis suppression, diabetes, myopathy, and psychiatric disturbances—are dose- and duration-dependent. Contraindications include systemic fungal infections, uncontrolled diabetes, and live vaccine coadministration. Monitoring requires baseline and periodic assessments of bone density, glucose, adrenal function, and cardiovascular health. Interprofessional collaboration is critical to mitigate risks.

**Conclusion:** While indispensable in modern medicine, corticosteroids demand judicious use, patient education, and vigilant monitoring to balance efficacy and safety. Future research should focus on safer alternatives and personalized dosing strategies.

**Keywords:** Corticosteroids, glucocorticoids, adverse effects, HPA axis suppression, osteoporosis, interprofessional care.

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**INTRODUCTION:**

Corticosteroids have been widely utilized across various medical disciplines since their discovery, administered through multiple routes to address diverse conditions [1]. These synthetic compounds are structural analogs of the natural steroid hormones produced by the adrenal cortex, comprising glucocorticoids and mineralocorticoids. The synthetic derivatives exhibit varying degrees of glucocorticoid and mineralocorticoid activity. Glucocorticoids primarily regulate metabolic functions and exert immunosuppressive, anti-inflammatory, and vasoconstrictive effects, whereas mineralocorticoids maintain electrolyte and fluid homeostasis by modulating ion transport in the renal tubules' epithelial cells [2]. Although corticosteroids encompass both glucocorticoids and mineralocorticoids, the term is frequently used in clinical practice to denote glucocorticoid effects due to their predominant therapeutic applications. Glucocorticoids serve as essential stress hormones, governing a wide range of physiological processes critical for survival [3].

The global corticosteroid market exceeds 10 billion USD annually, reflecting their extensive use in medical treatment [4]. In the United Kingdom, approximately one percent of the adult population is prescribed oral glucocorticoids at any given time [5]. The clinical indications for corticosteroid therapy are vast, encompassing infectious and inflammatory disorders, allergic and autoimmune diseases, shock, hypercalcemia, fluid retention, pathological hypoglycemia, excessive adrenocortical secretion, graft rejection prevention, neurological and hematologic disorders, dermatological conditions, and adrenal replacement therapy [6][7]. These agents are employed in both endocrine and nonendocrine contexts. In endocrine medicine, they are used diagnostically for Cushing syndrome and therapeutically for adrenal insufficiency and congenital adrenal hyperplasia. Their nonendocrine applications leverage their potent anti-inflammatory and immunosuppressive properties to manage a broad spectrum of immunologic and inflammatory conditions. Corticosteroids are administered at physiological doses for adrenal replacement therapy and at supraphysiological doses to achieve anti-inflammatory and immunosuppressive effects [2].

In allergy and pulmonology, corticosteroids are indicated for asthma exacerbations, chronic obstructive pulmonary disease (COPD) exacerbations, anaphylaxis, urticaria, angioedema, rhinitis, pneumonitis, sarcoidosis, and interstitial lung disease [2][5]. Dermatological applications include contact

dermatitis and pemphigus vulgaris, while endocrinological uses involve adrenal insufficiency and congenital adrenal hyperplasia. In gastroenterology, corticosteroids treat inflammatory bowel disease and autoimmune hepatitis, whereas hematological indications include hemolytic anemia, leukemia, lymphoma, and idiopathic thrombocytopenic purpura. Rheumatological conditions such as rheumatoid arthritis, systemic lupus erythematosus, polymyositis, dermatomyositis, and polymyalgia rheumatica also benefit from corticosteroid therapy. Additionally, corticosteroids are used in ophthalmology for uveitis and keratoconjunctivitis, as well as in other specialties for organ transplantation, antenatal lung maturation, nephrotic syndrome, cerebral edema, and multiple sclerosis [2][5].

The extensive use of corticosteroids underscores their therapeutic versatility, though their administration must be carefully monitored due to potential adverse effects. Their ability to modulate immune responses and inflammation has solidified their role as a cornerstone in the treatment of numerous medical conditions. However, the balance between efficacy and safety remains a critical consideration in clinical practice, necessitating judicious prescribing to minimize risks while maximizing therapeutic benefits [2][5].

**Mechanism of Action**

Corticosteroids exert their pharmacological effects through diverse pathways, influencing anti-inflammatory and immunosuppressive responses, protein and carbohydrate metabolism, fluid and electrolyte balance, central nervous system activity, and hematologic function [1][2][7]. Their mechanisms of action can be broadly categorized into genomic and non-genomic pathways. The genomic effects, which account for most anti-inflammatory and immunosuppressive actions, are mediated via intracellular glucocorticoid receptors (GRs) [2][5][8]. Inactive GRs reside in the cytoplasm and, upon corticosteroid binding, rapidly translocate to the nucleus, where they modulate gene transcription. This process suppresses the expression of proinflammatory mediators in leukocytes and structural cells such as epithelial tissues [1][2][7]. Consequently, the synthesis of cytokines, chemokines, cell adhesion molecules, and inflammatory enzymes is significantly reduced, attenuating the overall inflammatory response [2].

In contrast, the non-genomic mechanisms occur more rapidly, mediated either by cytosolic GRs or

membrane-bound receptors [1]. Within seconds to minutes of receptor activation, a signaling cascade is initiated, leading to the inhibition of phospholipase A2—an enzyme critical for generating inflammatory mediators. This suppression disrupts arachidonic acid metabolism and subsequently diminishes the production of prostaglandins and leukotrienes. Additionally, corticosteroids modulate apoptosis in thymocytes and, at higher concentrations, impair B-cell and T-cell proliferation, further contributing to immunosuppression [1][2][7]. These dual genomic and non-genomic actions enable corticosteroids to provide both immediate and sustained therapeutic effects, making them indispensable in managing inflammatory and immune-mediated disorders. However, their broad mechanisms also underlie the potential for systemic side effects, necessitating careful clinical consideration during therapeutic use [7].

#### **Administration of Corticosteroids**

The route of corticosteroid administration is determined by multiple factors, with the primary consideration being the specific disorder being treated. Available routes include parenteral, oral, inhaled, topical, injectable (intramuscular, intra-articular, intralesional, intradermal, etc.), and rectal formulations. When initiating corticosteroid therapy, clinicians must carefully evaluate several key factors, including the most appropriate route of administration, the specific corticosteroid preparation, optimal dosing, frequency, and duration of treatment to maximize therapeutic efficacy while minimizing potential adverse effects [7]. Parenteral administration is typically reserved for emergent clinical situations or for patients who cannot tolerate oral medications. This route ensures rapid systemic absorption, making it particularly useful in acute conditions requiring immediate therapeutic intervention. In contrast, oral administration is more commonly employed for chronic conditions, providing sustained systemic effects. However, due to the risk of systemic side effects associated with oral and parenteral routes, non-systemic therapies—such as inhaled, topical, or locally injected corticosteroids—are preferred whenever clinically feasible. These localized delivery methods target specific tissues while reducing systemic exposure, thereby lowering the risk of adverse effects. The selection of an appropriate administration route ultimately depends on balancing therapeutic goals with patient-specific factors, including disease severity, comorbidities, and potential treatment-related risks [7].

#### **Adverse Effects of Corticosteroids**

Despite their well-documented therapeutic efficacy, corticosteroids are associated with a wide range of adverse effects that significantly limit their clinical utility. These adverse effects are primarily dose- and duration-dependent, with higher doses and prolonged use increasing the likelihood of complications [2][9]. While adverse effects are most commonly observed with chronic high-dose therapy, they can also occur with short-term use [1]. Studies indicate that up to 90% of patients taking corticosteroids for more than sixty days experience some form of adverse effect [1]. The most frequently reported complications include osteoporosis and fractures, hypothalamic-pituitary-adrenal (HPA) axis suppression, Cushingoid features, diabetes and hyperglycemia, myopathy, glaucoma and cataracts, psychiatric disturbances, immunosuppression, cardiovascular disease, gastrointestinal complications, dermatologic effects, and growth suppression in pediatric patients [2]. Synthetic corticosteroids, such as prednisone, methylprednisolone, dexamethasone, and betamethasone, tend to exhibit pronounced glucocorticoid activity with minimal mineralocorticoid, androgenic, or estrogenic effects, making them more likely to induce Cushingoid features and HPA axis suppression [2].

##### **1. Osteoporosis, Fractures, and Osteonecrosis**

Corticosteroids exert detrimental effects on bone metabolism, leading to osteoporosis, increased fracture risk, and, in some cases, osteonecrosis. These drugs disrupt bone remodeling by initially enhancing osteoclast activity (particularly within the first 6 to 12 months of therapy) while simultaneously inhibiting calcium absorption in the gastrointestinal tract [1][2]. Additionally, corticosteroids suppress bone formation by reducing osteoblast activity, shortening their lifespan, and promoting apoptosis in both osteoblasts and osteocytes [2]. Even low-dose corticosteroid therapy ( $\geq 5$  mg/day of prednisone or equivalent) has been associated with significant reductions in bone mineral density (BMD) and an elevated fracture risk within 3 to 6 months of treatment initiation [2][10]. Osteonecrosis, a severe complication characterized by bone tissue death, has been reported in 9–40% of patients receiving long-term corticosteroid therapy, regardless of administration route (systemic or intra-articular) and even in the absence of corticosteroid-induced osteoporosis [2][11]. Other risk factors for osteonecrosis include alcoholism, sickle cell disease, HIV infection, and prior radiation exposure [2].

##### **2. Adrenal Suppression**

The endogenous production of corticosteroids is tightly regulated by the HPA axis, which responds to both inflammatory stimuli and circadian rhythms [1]. Exogenous corticosteroid administration can suppress

this axis, leading to adrenal insufficiency—a condition characterized by inadequate cortisol production. Notably, neither the duration nor the dosage of corticosteroid therapy reliably predicts the onset of HPA axis suppression [2]. Even non-systemic routes of administration, such as inhaled or topical corticosteroids, can contribute to adrenal suppression [1]. Abrupt discontinuation of corticosteroid therapy may precipitate adrenal crisis, necessitating gradual tapering to allow HPA axis recovery [1].

### 3. Cushingoid Features

Corticosteroid use can induce Cushing syndrome, characterized by weight gain and abnormal fat redistribution, including dorsocervical fat accumulation ("buffalo hump"), facial rounding ("moon facies"), and truncal obesity [2]. These changes may manifest within the first two months of therapy, with higher risk observed in patients with elevated baseline body mass index (BMI), younger individuals, and those with excessive caloric intake [12].

### 4. Diabetes and Hyperglycemia

Corticosteroids are the leading cause of drug-induced diabetes mellitus and exacerbate insulin resistance in pre-existing diabetes [2]. Their hyperglycemic effects are dose-dependent and can develop within hours of administration, primarily affecting postprandial glucose levels more than fasting glucose [2][13].

### 5. Myopathy

Corticosteroid-induced myopathy presents as proximal muscle weakness and atrophy, typically developing over weeks to months, though high doses may accelerate onset [2][13]. Unlike inflammatory myopathies, corticosteroid myopathy is not typically associated with muscle pain or tenderness [14]. The underlying mechanism involves protein catabolism and reduced muscle protein synthesis [2]. Symptoms generally improve within 3–4 weeks after discontinuation [2].

### 6. Glaucoma and Cataracts

Corticosteroid use increases the risk of both glaucoma and cataracts in a dose-dependent manner [2]. Glaucoma, the more severe complication, arises from elevated intraocular pressure, which can lead to optic nerve damage and irreversible vision loss if untreated [2]. While intraocular pressure normalizes after corticosteroid cessation, optic nerve damage may persist [2].

### 7. Psychiatric Disturbances

Corticosteroids can induce a spectrum of psychiatric symptoms, including psychosis, agitation, insomnia, irritability, hypomania, anxiety, and mood instability [2]. Short-term use may cause euphoria, whereas prolonged therapy often leads to depressive symptoms [15]. High-dose corticosteroids ( $\geq 20$  mg/day

prednisone or equivalent) are particularly associated with steroid-induced psychosis, sometimes necessitating antipsychotic treatment [2][16].

### 8. Immunosuppression

While immunosuppression is a desired effect in autoimmune and inflammatory conditions, it also increases susceptibility to infections. A meta-analysis of 2000 patients found that infection rates rise significantly with doses  $\geq 10$  mg/day [2]. Risk factors include advanced age, comorbidities, and concomitant immunosuppressive therapies. Notably, corticosteroid-treated patients are at heightened risk for invasive fungal and viral infections [2].

### 9. Cardiovascular Effects

Corticosteroids contribute to hypertension, hyperglycemia, and obesity, with mixed evidence regarding their impact on hyperlipidemia [2]. Their mineralocorticoid activity promotes sodium retention and potassium excretion, exacerbating cardiovascular risk [2]. Studies link corticosteroid uses to increased rates of atrial fibrillation, heart failure, and ischemic heart disease [2][17].

### 10. Gastrointestinal Complications

Corticosteroids are associated with gastritis, peptic ulcer disease, abdominal distension, and dyspepsia [2]. While the ulcerogenic risk of corticosteroids alone is low, concurrent NSAID use nearly quadruples the risk [2][18].

### 11. Dermatologic Effects

Chronic corticosteroid use induces skin atrophy, manifesting as thinning, fragility, striae, and purpura [2]. Striae are typically permanent, whereas other changes may reverse upon discontinuation [2]. Corticosteroids also impair wound healing by suppressing leukocyte migration, collagen synthesis, and keratinocyte growth factor expression [2].

### 12. Growth Suppression in Pediatric Patients

Children and adolescents on long-term corticosteroid therapy are at risk for growth retardation and delayed puberty [19]. Some evidence suggests that final adult height may be reduced in these patients [2]. Growth suppression may also indicate underlying adrenal suppression [2]. Corticosteroids remain indispensable in managing a wide array of conditions, but their adverse effect profile necessitates judicious use. Clinicians must weigh therapeutic benefits against potential risks, employing the lowest effective dose for the shortest duration possible. Prophylactic measures, such as calcium and vitamin D supplementation for bone protection, blood glucose monitoring, and gradual tapering to prevent adrenal crisis, are essential in mitigating complications. Future research should focus on developing corticosteroids with improved safety profiles or alternative therapies that minimize these well-documented adverse effects [19].



### Contraindications to Corticosteroid Therapy

Corticosteroids are contraindicated in patients with a known hypersensitivity to any component of the formulation, as this may precipitate severe allergic reactions. Additionally, their immunosuppressive effects prohibit concurrent administration with live or live-attenuated vaccines when given at immunosuppressive doses, due to the risk of vaccine-induced infection. Systemic fungal infections represent another absolute contraindication, as corticosteroids can exacerbate fungal proliferation and dissemination. Patients with pre-existing osteoporosis, uncontrolled hyperglycemia, or diabetes mellitus should avoid corticosteroids due to their detrimental effects on bone metabolism and glucose homeostasis. Glaucoma is also a contraindication, given that corticosteroids can elevate intraocular pressure, potentially worsening optic nerve damage. Intra-articular corticosteroid injections are contraindicated in cases of joint infection, as they may suppress local immune responses and worsen septic arthritis [9][18].

Other significant contraindications include uncontrolled hypertension, herpes simplex keratitis, and active varicella infection, as corticosteroids can worsen these conditions. Relative contraindications—where risks must be carefully weighed against benefits—include peptic ulcer disease (particularly when combined with NSAIDs), congestive heart failure (due to fluid retention), and uncontrolled bacterial or viral infections, as corticosteroids may impair the immune response necessary for pathogen clearance. Clinicians must exercise caution in these scenarios, considering alternative therapies or implementing stringent monitoring to mitigate risks [2][9][18].

### Monitoring Considerations for Corticosteroid Therapy

#### Pre-Treatment Evaluation

Before initiating corticosteroid therapy, a thorough history and physical examination are essential to identify risk factors or preexisting conditions that may be exacerbated by treatment. Key conditions to assess include diabetes mellitus, hypertension, congestive heart failure, hyperlipidemia, psychiatric disorders, and osteoporosis. Clinicians should aim to use the lowest effective dose for the shortest possible duration to minimize adverse effects. Strategies such as once-daily dosing (preferably in the morning to align with the body's natural cortisol rhythm) and alternate-day dosing may help reduce HPA axis suppression and other systemic complications [2].

Baseline assessments should include:

- **Height and weight measurements** (particularly important in pediatric patients due to growth suppression risks)
- **Blood pressure evaluation** (to detect or monitor hypertension)
- **Bone mineral density (BMD) testing** (for patients at risk of osteoporosis)
- **Ophthalmologic examination** (to screen for glaucoma or cataracts)
- **Fasting blood glucose and lipid panel** (to assess metabolic risk factors)
- **Assessment of pubertal and nutritional status in children and adolescents** (due to potential growth retardation) [2].

#### Bone Health Monitoring

Given the significant risk of osteoporosis and fractures, BMD testing should be performed at baseline and repeated after one year of corticosteroid therapy. If results are stable, subsequent assessments may be spaced to every 2–3 years. For patients with declining BMD, pharmacologic intervention (e.g., bisphosphonates) should be considered, with the Fracture Risk Assessment Tool (FRAX) used to guide treatment decisions [20]. Pediatric patients require serial BMD assessments, and referral to a pediatric bone health specialist is recommended if fragility fractures or significant BMD loss occur [2]. Patients should also be monitored for osteonecrosis, particularly those receiving long-term or high-dose therapy. Symptoms such as joint pain or reduced range of motion should prompt further evaluation. Preventive measures include:

- Smoking cessation
- Reduced alcohol intake
- Weight-bearing exercise
- Calcium and vitamin D supplementation [2].

#### Adrenal Function Assessment

Adrenal suppression is a major concern, particularly in patients receiving supraphysiologic doses for more than two weeks [21]. If adrenal insufficiency is suspected, biochemical testing should include:

- **First-morning cortisol level** (if normal but clinical suspicion remains, proceed with a low-dose ACTH stimulation test)
- **Stress-dose steroid coverage** (if adrenal suppression is confirmed) [2].

#### Gastrointestinal and Metabolic Monitoring

Patients on corticosteroids with concurrent NSAID use or those at high risk for peptic ulcers (e.g., history of GI bleeding, advanced cancer) should receive proton pump inhibitor (PPI) prophylaxis [2]. Additionally, metabolic monitoring is crucial due to corticosteroid-induced hyperglycemia and

dyslipidemia. Patients with diabetes should undergo frequent blood glucose monitoring, with potential adjustments to antihyperglycemic medications. A low-calorie diet may help mitigate weight gain and Cushingoid features, while topical vitamin A, pulsed dye lasers, or radiofrequency treatments may reduce corticosteroid-induced striae [2].

#### **Electrolyte and Cardiovascular Monitoring**

Corticosteroids with mineralocorticoid activity (e.g., hydrocortisone, cortisone) can cause fluid retention, hypertension, and hypokalemia. Regular monitoring of blood pressure, serum electrolytes, and fluid status is essential, particularly in patients with heart failure or renal impairment [2]. Cardiovascular risk assessment should include periodic lipid profiling, as corticosteroids may exacerbate hyperlipidemia and increase the risk of atrial fibrillation, ischemic heart disease, and heart failure [2][17].

#### **Drug Interactions and Immunosuppression Risks**

Corticosteroids interact with several medications, necessitating close monitoring:

- **Anticoagulants (e.g., warfarin):** Increased INR within 3–7 days of corticosteroid initiation may require dose adjustments.
- **Antihyperglycemics:** Reduced efficacy due to corticosteroid-induced insulin resistance.
- **Antiretrovirals:** Some (e.g., efavirenz) decrease corticosteroid effects, while others (e.g., ritonavir) increase exposure, requiring dose modifications.
- **NSAIDs:** Heightened risk of peptic ulcers; concurrent use should be avoided if possible [2].

Due to immunosuppressive effects, live vaccines (e.g., MMR, varicella) should be deferred for at least three months after discontinuing high-dose corticosteroid therapy ( $\geq 40$  mg/day prednisone equivalent for  $> 7$  days) [2].

#### **Psychiatric and Ophthalmologic Surveillance**

Patients should be monitored for mood disturbances, insomnia, or psychosis, particularly at higher doses ( $\geq 20$  mg/day prednisone). Ophthalmologic evaluations are critical for detecting glaucoma or cataracts, especially in long-term users [2]. Effective monitoring of corticosteroid therapy requires a multidisciplinary approach, including regular metabolic, cardiovascular, bone, and adrenal function assessments. Preventive strategies, dose optimization, and early intervention for adverse effects can significantly improve patient outcomes while minimizing risks [2].

#### **Toxicity:**

Corticosteroid toxicity represents one of the most prevalent causes of iatrogenic illness in patients undergoing chronic therapy. Unlike many medications, corticosteroids lack a specific reversal agent, necessitating management through gradual dose reduction and targeted treatment of associated complications such as hyperglycemia, infections, or hypertension. For treatment durations lasting less than one-week, abrupt cessation is generally safe without requiring a taper. However, for therapies spanning one to three weeks, tapering protocols should be individualized based on the patient's clinical condition, as sudden withdrawal may precipitate adrenocorticotrophic hormone (ACTH) suppression and disease flare-ups. Courses exceeding three weeks warrant an initial rapid taper to physiologic doses followed by a slower weaning process, with concurrent assessment of adrenal function to prevent adrenal insufficiency. Notably, long-term, high-dose corticosteroid regimens can suppress the hypothalamic-pituitary-adrenal (HPA) axis for extended periods, with full recovery potentially taking nine to twelve months after discontinuation. During this period, patients remain at risk for adrenal crisis during physiological stress, underscoring the need for careful monitoring and possible stress-dose steroid coverage in appropriate clinical situations [2]. The management of corticosteroid toxicity requires a balanced approach that considers both the necessity of ongoing immunosuppression and the imperative to minimize adverse effects. Clinicians must remain vigilant for signs of HPA axis suppression and be prepared to implement appropriate tapering strategies while addressing specific organ-system toxicities as they arise. This cautious approach helps mitigate the risks associated with corticosteroid withdrawal while ensuring optimal patient outcomes [2].

#### **Enhancing Healthcare Team Outcomes in Corticosteroid Therapy**

Corticosteroids are among the most widely prescribed medications due to their potent anti-inflammatory and immunosuppressive properties. However, their extensive use is accompanied by a significant risk of adverse effects, necessitating a comprehensive understanding among all healthcare team members. The therapeutic effects of corticosteroids can be beneficial in one clinical scenario while potentially harmful in another. For instance, their hyperglycemic effect may be advantageous in managing adrenal insufficiency but detrimental in patients with latent diabetes mellitus [2]. To maximize therapeutic benefits while minimizing risks, careful patient

monitoring and the implementation of preventive strategies are essential.

An interprofessional approach is critical to optimizing corticosteroid therapy. Clinicians, nurse practitioners, pharmacists, and nursing staff must collaborate to educate patients about potential adverse effects and necessary lifestyle or therapeutic modifications. Effective communication among team members ensures consistent messaging and coordinated care. Patients on chronic systemic corticosteroid therapy should be advised to carry a steroid treatment card or wear medical identification to alert healthcare providers in emergency situations. In the era of electronic health records, a digital flag or alert system can help identify patients receiving corticosteroid therapy, reducing the risk of abrupt withdrawal or missed interactions [2].

Preventable morbidity and mortality associated with corticosteroid use can be mitigated through patient education and interprofessional collaboration. Pharmacists play a key role in monitoring for drug interactions, while nurses can assess early signs of complications such as hyperglycemia or infections. Clinicians must regularly evaluate the need for continued therapy and adjust dosages as needed. By fostering a team-based approach with open communication, healthcare providers can enhance patient safety, improve adherence, and ensure that corticosteroids are used both effectively and responsibly. This collaborative model not only optimizes therapeutic outcomes but also reduces the burden of corticosteroid-related complications. [2]

### CONCLUSION:

Corticosteroids remain a cornerstone of therapy for inflammatory, autoimmune, and endocrine disorders due to their potent immunosuppressive and anti-inflammatory effects. However, their extensive use is fraught with significant risks, including osteoporosis, metabolic disturbances, adrenal suppression, and increased infection susceptibility. This review underscores the importance of tailoring corticosteroid therapy to individual patient needs, employing the lowest effective dose for the shortest duration to mitigate adverse effects. The dual genomic and non-genomic mechanisms of corticosteroids explain both their therapeutic benefits and systemic toxicity. Clinicians must carefully select administration routes—prioritizing localized delivery (e.g., inhaled, topical) over systemic forms when possible—to reduce complications. The review highlights critical contraindications, such as uncontrolled infections and metabolic disorders, which necessitate alternative

treatments. Monitoring is paramount, particularly for patients on chronic therapy. Baseline and follow-up assessments of bone mineral density, glucose levels, and adrenal function can preempt complications like fractures and adrenal crises. The Fracture Risk Assessment Tool (FRAX) and routine ophthalmologic exams are recommended for long-term users. Additionally, interprofessional collaboration—engaging physicians, pharmacists, and nurses—ensures comprehensive patient education on medication adherence, warning signs of toxicity, and lifestyle modifications (e.g., calcium supplementation, weight-bearing exercise). Tapering protocols must be individualized to avoid HPA axis suppression, with stress-dose steroids considered for patients undergoing physiological stressors. The review also emphasizes the role of electronic health alerts and steroid treatment cards in preventing treatment interruptions or overlaps. Future directions include developing targeted corticosteroids with reduced off-target effects and exploring non-steroidal alternatives for chronic conditions. Until then, this review equips healthcare teams with evidence-based strategies to optimize corticosteroid therapy, emphasizing vigilance, patient-centered care, and interprofessional communication to achieve the best clinical outcomes while minimizing harm. By integrating these practices, healthcare providers can harness the benefits of corticosteroids while safeguarding patients from their well-documented risks, ensuring both efficacy and safety in clinical application.

### REFERENCES:

1. Ericson-Neilsen W, Kaye AD. Steroids: pharmacology, complications, and practice delivery issues. *Ochsner J*. 2014 Summer;14(2):203-7.
2. Liu D, Ahmet A, Ward L, Krishnamoorthy P, Mandelcorn ED, Leigh R, Brown JP, Cohen A, Kim H. A practical guide to the monitoring and management of the complications of systemic corticosteroid therapy. *Allergy Asthma Clin Immunol*. 2013 Aug 15;9(1):30.
3. Ramamoorthy S, Cidlowski JA. Corticosteroids: Mechanisms of Action in Health and Disease. *Rheum Dis Clin North Am*. 2016 Feb;42(1):15-31, vii.
4. Schäcke H, Döcke WD, Asadullah K. Mechanisms involved in the side effects of glucocorticoids. *Pharmacol Ther*. 2002 Oct;96(1):23-43.
5. Coutinho AE, Chapman KE. The anti-inflammatory and immunosuppressive effects of glucocorticoids, recent developments and

- mechanistic insights. *Mol Cell Endocrinol*. 2011 Mar 15;335(1):2-13.
6. Streeten DH. Corticosteroid therapy. II. Complications and therapeutic indications. *JAMA*. 1975 Jun 09;232(10):1046-9.
  7. Streeten DH. Corticosteroid therapy. I. Pharmacological properties and principles of corticosteroid use. *JAMA*. 1975 Jun 02;232(9):944-7.
  8. Cain DW, Cidlowski JA. Immune regulation by glucocorticoids. *Nat Rev Immunol*. 2017 Apr;17(4):233-247.
  9. Da Silva JA, Jacobs JW, Kirwan JR, Boers M, Saag KG, Inês LB, de Koning EJ, Buttgerit F, Cutolo M, Capell H, Rau R, Bijlsma JW. Safety of low dose glucocorticoid treatment in rheumatoid arthritis: published evidence and prospective trial data. *Ann Rheum Dis*. 2006 Mar;65(3):285-93.
  10. van Staa TP, Leufkens HG, Cooper C. The epidemiology of corticosteroid-induced osteoporosis: a meta-analysis. *Osteoporos Int*. 2002 Oct;13(10):777-87.
  11. Weinstein RS. Glucocorticoid-induced osteonecrosis. *Endocrine*. 2012 Apr;41(2):183-90.
  12. Fardet L, Cabane J, Lebbé C, Morel P, Flahault A. Incidence and risk factors for corticosteroid-induced lipodystrophy: a prospective study. *J Am Acad Dermatol*. 2007 Oct;57(4):604-9.
  13. Schneiter P, Tappy L. Kinetics of dexamethasone-induced alterations of glucose metabolism in healthy humans. *Am J Physiol*. 1998 Nov;275(5):E806-13.
  14. Moghadam-Kia S, Werth VP. Prevention and treatment of systemic glucocorticoid side effects. *Int J Dermatol*. 2010 Mar;49(3):239-48.
  15. Bolanos SH, Khan DA, Hanczyc M, Bauer MS, Dhanani N, Brown ES. Assessment of mood states in patients receiving long-term corticosteroid therapy and in controls with patient-rated and clinician-rated scales. *Ann Allergy Asthma Immunol*. 2004 May;92(5):500-5.
  16. Kershner P, Wang-Cheng R. Psychiatric side effects of steroid therapy. *Psychosomatics*. 1989 Spring;30(2):135-9.
  17. Wei L, MacDonald TM, Walker BR. Taking glucocorticoids by prescription is associated with subsequent cardiovascular disease. *Ann Intern Med*. 2004 Nov 16;141(10):764-70.
  18. Piper JM, Ray WA, Daugherty JR, Griffin MR. Corticosteroid use and peptic ulcer disease: role of nonsteroidal anti-inflammatory drugs. *Ann Intern Med*. 1991 May 01;114(9):735-40.
  19. Allen DB, Mullen M, Mullen B. A meta-analysis of the effect of oral and inhaled corticosteroids on growth. *J Allergy Clin Immunol*. 1994 Jun;93(6):967-76.
  20. Kanis JA, Johansson H, Oden A, McCloskey EV. Guidance for the adjustment of FRAX according to the dose of glucocorticoids. *Osteoporos Int*. 2011 Mar;22(3):809-16.



**الستيرويدات القشرية: معرفة عامة لجميع العاملين في الرعاية الصحية - مراجعة محدثة****الملخص**

**الخلفية:** الستيرويدات القشرية هي نظائر صناعية لهرمونات الغدة الكظرية، تمتلك خصائص قوية مضادة للالتهابات ومثبطة للمناعة، وتستخدم على نطاق واسع في مختلف التخصصات الطبية. تشمل تطبيقاتها العلاجية اضطرابات الغدد الصماء، والحالات الالتهابية، والأمراض المناعية الذاتية، لكن استخدامها معقد بسبب آثارها الجانبية الكبيرة.

**الهدف:** تلخص هذه المراجعة المعرفة الحالية حول فارماكولوجيا الستيرويدات القشرية، وتطبيقاتها السريرية، وآثارها الجانبية، وموانع استخدامها، واستراتيجيات المراقبة، بهدف مساعدة العاملين في الرعاية الصحية على تحسين العلاج وتقليل المخاطر.

**المنهجية:** أجريت مراجعة شاملة للأدبيات، شملت مقالات محكمة، وإرشادات سريرية، وبيانات فارماكولوجية عن آليات عمل الستيرويدات القشرية، وطرق إعطائها، وملفات السمية، وبرتوكولات المراقبة.

**النتائج:** تؤثر الستيرويدات القشرية من خلال مسارات جينية وغير جينية، وتُختار طرق إعطائها حسب الحاجة السريرية (عن طريق الفم، الحقن، الاستنشاق، الموضعية). تشمل الآثار الجانبية هشاشة العظام، كبت محور HPA، السكري، الاعتلال العضلي، والاضطرابات النفسية، وترتبط بالجرعة ومدة الاستخدام. تشمل موانع الاستخدام العدوى الفطرية الجهازية، السكري غير المنضبط، والإعطاء المتزامن للقاحات الحية. تتطلب المراقبة تقييمات أساسية ودورية لكثافة العظام، ومستويات الجلوكوز، ووظيفة الغدة الكظرية، وصحة القلب والأوعية. التعاون بين التخصصات ضروري للحد من المخاطر.

**الاستنتاج:** رغم أن الستيرويدات القشرية أساسية في الطب الحديث، إلا أن استخدامها يتطلب الحذر، وتثقيف المريض، ومراقبة دقيقة لتحقيق التوازن بين الفعالية والسلامة. يجب أن تركز الأبحاث المستقبلية على بدائل أكثر أماناً واستراتيجيات جرعات مخصصة.

**الكلمات المفتاحية:** الستيرويدات القشرية، الجلوكوكورتيكويدات، الآثار الجانبية، كبت محور HPA، هشاشة العظام، الرعاية متعددة التخصصات.