



Corticosteroids: An Updated Review for Healthcare Professionals

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Abstract:

Background: Corticosteroids, synthetic analogs of adrenal cortex hormones, are widely used across various medical domains due to their anti-inflammatory and immunosuppressive properties. These drugs, comprising glucocorticoids and mineralocorticoids, play critical roles in regulating metabolism, immune responses, and electrolyte balance.

Aim: This review provides an updated overview of corticosteroids, including their mechanisms of action, therapeutic applications, adverse effects, and contraindications, offering essential insights for healthcare professionals.

Methods: A comprehensive literature review was conducted, focusing on the pharmacological properties, clinical uses, and adverse effects of corticosteroids. Key mechanisms, routes of administration, and considerations for therapy initiation were analyzed.

Results: Corticosteroids demonstrate multifaceted mechanisms through genomic and non-genomic pathways, enabling suppression of inflammatory mediators and modulation of immune responses. They are used for conditions such as asthma, rheumatoid arthritis, inflammatory bowel disease, and adrenal insufficiency. However, their usage is constrained by adverse effects, including osteoporosis, adrenal suppression, hyperglycemia, myopathy, and psychiatric disturbances. The choice of administration route impacts efficacy and side-effect profiles, with systemic exposure often linked to increased risks.

Conclusion: Corticosteroids remain indispensable in clinical practice, but their potential adverse effects necessitate cautious use, tailored dosing, and regular monitoring. Understanding their mechanisms, therapeutic potential, and risks is vital for optimizing patient outcomes.

Keywords: Corticosteroids, glucocorticoids, mineralocorticoids, anti-inflammatory, immunosuppressive, adverse effects, therapeutic applications

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Introduction:

Since their initial discovery, corticosteroids have become integral to nearly every domain of medicine and are administered through a wide variety of routes [1]. These compounds are synthetic analogs of endogenous steroid hormones produced by the adrenal cortex, comprising glucocorticoids and mineralocorticoids. The synthetic derivatives exhibit diverse glucocorticoid and mineralocorticoid properties. Glucocorticoids predominantly regulate metabolic processes and exhibit immunosuppressive, anti-inflammatory, and vasoconstrictive actions, while mineralocorticoids primarily modulate electrolyte and water homeostasis through ion transport within the epithelial cells of renal tubules [2]. In clinical practice, the term corticosteroids is generally synonymous with the glucocorticoid effect. Glucocorticoids, as primary stress hormones, regulate numerous physiological processes vital to life [3]. Globally, corticosteroids are among the most frequently prescribed drug classes, with an estimated market value exceeding 10 billion USD annually [4]. In the United Kingdom, approximately 1% of the adult population receives oral glucocorticoid therapy at any given time [5]. The indications for corticosteroid therapy encompass a vast spectrum of conditions, broadly categorized into infectious and inflammatory diseases, allergic and autoimmune disorders, shock, hypercalcemia reduction, promotion of water excretion, management of pathologic hypoglycemia, suppression of excessive adrenocortical secretion, prevention of graft rejection, and treatment of neurological, hematologic, dermatological disorders, as well as corticosteroid replacement therapy [6][7].

Corticosteroids serve both endocrine and non-endocrine purposes. Endocrine applications include diagnosing Cushing syndrome, managing adrenal insufficiency, and addressing congenital adrenal hyperplasia. Non-endocrine applications leverage their robust anti-inflammatory and immunosuppressive properties to treat a wide array of immunological and inflammatory disorders. Corticosteroids are employed at physiological doses for adrenal insufficiency replacement therapy, whereas supraphysiological doses are utilized for their anti-inflammatory and immunosuppressive effects [2]. Common indications for corticosteroids span numerous specialties, including but not limited to: asthma exacerbation, chronic obstructive pulmonary disease (COPD) exacerbation, anaphylaxis, urticaria, angioedema, rhinitis, pneumonitis, sarcoidosis, and interstitial lung disease in pulmonology; contact dermatitis and pemphigus vulgaris in dermatology; adrenal insufficiency and congenital adrenal hyperplasia in endocrinology; inflammatory bowel disease and autoimmune hepatitis in gastroenterology; hemolytic anemia, leukemia, lymphoma, and idiopathic thrombocytopenic purpura in hematology; rheumatoid arthritis, systemic lupus erythematosus, polymyositis, dermatomyositis, and polymyalgia rheumatica in rheumatology; uveitis and keratoconjunctivitis in ophthalmology; and other conditions such as organ transplantation, antenatal lung maturation, nephrotic syndrome, cerebral edema, and multiple sclerosis [2][5].

The mechanism of action of corticosteroids is multifaceted, encompassing anti-inflammatory and immunosuppressive effects, as well as influences on protein and carbohydrate metabolism, water and electrolyte balance, the central nervous system, and hematologic profiles [1][2][7]. These effects are mediated via both genomic and non-genomic pathways. The genomic mechanism involves activation of intracellular glucocorticoid receptors, subsequent nuclear translocation, and modulation of gene transcription, leading to the suppression of inflammatory mediators such as cytokines, chemokines, cell adhesion molecules, and enzymes involved in inflammatory pathways [1][2][7]. The non-genomic mechanisms operate more rapidly, within seconds to minutes, through interactions with intracellular or membrane-bound glucocorticoid receptors. These interactions initiate cascades that inhibit phospholipase A2, impair arachidonic acid release, and regulate apoptosis in thymocytes, effectively curtailing the inflammatory response [1][7]. At elevated concentrations, corticosteroids also inhibit the production of B cells and T cells [1][2]. The route of corticosteroid administration is determined by the specific condition being treated and may include parenteral, oral, inhaled, topical, intramuscular, intra-articular, intralesional,

intradermal, or rectal routes. Parenteral administration is generally reserved for emergent situations or when oral intake is contraindicated, while oral administration is common for chronic conditions. Whenever feasible, clinicians are advised to opt for non-systemic routes to reduce systemic exposure and minimize potential adverse effects. Several factors must be considered before initiating corticosteroid therapy, including the route, preparation, dosing, frequency, and duration of treatment.

Adverse Effects of Corticosteroids

Despite their profound therapeutic efficacy, the utilization of corticosteroids is considerably constrained due to their numerous adverse effects. These undesirable outcomes are influenced by both the average dosage and the cumulative duration of corticosteroid therapy [2][9]. Adverse effects are notably more prevalent at higher dosages and with prolonged usage; however, they are not exclusively confined to these conditions [1]. Studies indicate that up to 90% of patients who have undergone corticosteroid treatment for over 60 days experience adverse effects [1]. The most commonly reported adverse effects include osteoporosis and fractures, suppression of the hypothalamic-pituitary-adrenal (HPA) axis, Cushingoid features, diabetes and hyperglycemia, myopathy, glaucoma and cataracts, psychiatric disturbances, immunosuppression, cardiovascular complications, as well as gastrointestinal and dermatologic effects [2]. Synthetic corticosteroids, such as prednisone, methylprednisone, dexamethasone, and betamethasone, are particularly associated with pronounced Cushingoid features, suppression of HPA axis function, and minimal mineralocorticoid, androgenic, or estrogenic activity.

Osteoporosis, Fractures, and Osteonecrosis

Corticosteroids significantly impair bone mineralization by favoring osteoclast activity within the initial 6 to 12 months of therapy and concurrently inhibiting calcium absorption in the gastrointestinal tract [1][2]. These agents also reduce bone formation by diminishing osteoblast activity and lifespan while inducing apoptosis in osteoblasts and osteocytes [2]. Research has demonstrated that prednisone doses as low as 5 mg/day can contribute to bone loss, with reductions in bone mineral density (BMD) and elevated fracture risks observed within three to six months of initiating therapy at doses of 5 mg or more per day [2][10]. Furthermore, osteonecrosis has been reported in 9% to 40% of patients undergoing long-term corticosteroid therapy, whether systemic or intra-articular, even in the absence of corticosteroid-induced osteoporosis [2][11]. Factors such as alcoholism, sickle cell disease, HIV infection, and radiation exposure further exacerbate the risk of osteonecrosis.

Adrenal Suppression

The endogenous production of corticosteroids is tightly regulated by the hypothalamic-pituitary-adrenal (HPA) axis, which responds rapidly to inflammation and other stressors while adhering to circadian rhythms [1]. Adrenal suppression arises when exogenous corticosteroid exposure disrupts the HPA axis, resulting in insufficient cortisol production. Importantly, the likelihood of adrenal suppression cannot be reliably predicted based solely on the dosage or duration of corticosteroid therapy [2]. Even inhaled or topical corticosteroids have been implicated in causing adrenal suppression [1]. Abrupt cessation of corticosteroid therapy is a major precipitant of adrenal insufficiency, necessitating a gradual tapering protocol to mitigate this risk [1].

Cushingoid Features

Cushingoid features, indicative of excess cortisol, manifest as adipose tissue redistribution leading to truncal obesity, moon facies, and dorsocervical fat deposition, commonly referred to as "buffalo hump" [2]. These changes can occur within the initial two months of corticosteroid therapy and are more prevalent in individuals with higher baseline body mass indices, younger age, and increased caloric intake [12].

Diabetes and Hyperglycemia

Corticosteroids are a primary cause of drug-induced diabetes mellitus and exacerbate insulin resistance in diabetic patients [2]. A dose-dependent hyperglycemic effect is evident within hours of corticosteroid

administration, disproportionately affecting postprandial glucose levels compared to fasting glucose levels [2][13].

Myopathy

Corticosteroid-induced myopathy is characterized by proximal muscle weakness and atrophy, typically developing over weeks to months and exacerbated by higher doses [2][13]. Unlike other myopathies, these effects are not associated with muscle tenderness or myalgias [14]. The underlying mechanism involves the catabolic effects of corticosteroids, including reduced protein synthesis and increased protein breakdown. Symptoms generally resolve within three to four weeks after discontinuation [2].

Glaucoma and Cataracts

The use of corticosteroids poses a dose-dependent risk for both glaucoma and cataracts [2]. Glaucoma, the more severe of the two, arises from increased intraocular pressure, potentially resulting in optic nerve atrophy and irreversible vision loss. Although intraocular pressure typically normalizes after corticosteroid cessation, the damage to the optic nerve is permanent [2].

Psychiatric Disturbances

Corticosteroids can induce a spectrum of psychiatric effects, ranging from mild euphoria during short-term use to severe psychosis, agitation, insomnia, and mood instability during prolonged therapy [2][15]. Psychosis, generally associated with high doses exceeding 20 mg/day of prednisone, may necessitate antipsychotic intervention if symptoms persist [2][16].

Immunosuppression

While corticosteroids are valued for their immunosuppressive and anti-inflammatory properties, they also heighten susceptibility to infections. A meta-analysis revealed significantly increased infection rates among patients on systemic corticosteroids, especially at doses exceeding 10 mg/day [2]. Susceptibility to invasive fungal and viral infections is notably higher, influenced by factors such as patient age, comorbidities, and concurrent immunosuppressive therapies [2].

Cardiovascular Adverse Effects

Corticosteroid use has been linked to hypertension, hyperglycemia, obesity, and conflicting evidence regarding hyperlipidemia [2]. These effects, along with corticosteroid-induced water and sodium retention and potassium excretion, elevate the risk of cardiovascular events, including atrial fibrillation, heart failure, and ischemic heart disease [2][17].

Gastrointestinal Adverse Effects

Corticosteroids are associated with various gastrointestinal complications, such as gastritis, peptic ulcers, dyspepsia, and abdominal distension [2]. Although the risk of peptic ulcers from corticosteroid use alone is relatively low, it increases nearly fourfold when combined with nonsteroidal anti-inflammatory drugs (NSAIDs) [2][18].

Dermatologic Adverse Effects

Corticosteroids induce skin thinning, fragility, striae, and purpura, with striae being typically irreversible [2]. Additionally, corticosteroid therapy impairs cutaneous wound healing by inhibiting leukocyte and macrophage migration, reducing collagen synthesis, and suppressing keratinocyte growth factor expression [2].

Growth Suppression

In pediatric populations, corticosteroid therapy is linked to growth suppression, delayed puberty, and, in some cases, reduced final height [19]. This effect may serve as an indicator of adrenal suppression and underscores the importance of careful monitoring in younger patients [2].

Contraindications

The administration of corticosteroids is contraindicated in cases of hypersensitivity to any ingredient in the formulation and during concurrent use of live or live-attenuated vaccines, particularly when administered in immunosuppressive doses. Additionally, systemic fungal infections, uncontrolled hyperglycemia, diabetes mellitus, osteoporosis, joint infections, glaucoma, uncontrolled hypertension, herpes simplex keratitis, and varicella infections are notable contraindications. The use of corticosteroids in such conditions can exacerbate underlying disease processes or increase the risk of complications due to their immunosuppressive and metabolic effects. Relative contraindications warranting cautious use include peptic ulcer disease, congestive heart failure, and active viral or bacterial infections that are not adequately managed by antimicrobial therapy. Corticosteroids may worsen these conditions by impairing the body's natural defense mechanisms, altering tissue repair, or exacerbating fluid retention and cardiovascular strain. In the case of infections, their immune-modulating effects can mask clinical signs, delaying appropriate treatment and potentially worsening outcomes. Clinicians must carefully assess the risk-benefit profile when prescribing corticosteroids, especially in patients with these contraindications. Strategies such as dose adjustments, close monitoring, and the concurrent use of prophylactic measures may help mitigate risks in certain circumstances. However, in instances where the contraindications are absolute, alternative therapies should be prioritized to avoid adverse effects associated with corticosteroid use.

Monitoring

Prior to initiating corticosteroid therapy, it is essential to conduct a comprehensive history and physical examination to identify any preexisting conditions or risk factors that could be exacerbated by the treatment. These evaluations should focus on identifying conditions such as diabetes mellitus, hypertension, congestive heart failure, hyperlipidemia, psychiatric disorders, and osteoporosis. To mitigate potential adverse effects, healthcare providers should aim to use the lowest effective dose for the shortest feasible duration, and dosing regimens should prioritize once-daily, morning, or alternate-day schedules to reduce systemic impact [2]. Baseline assessments are fundamental and should encompass the patient's height, weight, blood pressure, bone mineral density (BMD), ophthalmologic evaluation, blood glucose levels, and lipid profile. Special attention must be given to children and adolescents due to their heightened susceptibility to growth suppression. For this population, evaluations of pubertal development and nutritional status are imperative prior to commencing therapy [2]. BMD testing is a critical component of monitoring, with initial assessment recommended at baseline and again after one year of therapy. Stable results may allow subsequent evaluations to occur every two to three years. Pharmacological intervention should be considered for patients with reduced BMD, using the World Health Organization's Fracture Risk Assessment Tool (FRAX) to identify those who might benefit from treatment [20]. Pediatric patients with declining BMD scores should undergo serial assessments and may require referral to a pediatric bone health specialist. Regular evaluations for joint pain and restricted motion are also warranted to monitor for osteonecrosis. Preventive measures in adults, such as smoking cessation, reduced alcohol intake, weight-bearing exercises, and supplementation with calcium and vitamin D, are crucial to minimizing side effects [2].

Adrenal suppression is a significant concern for patients receiving supraphysiologic corticosteroid doses for over two weeks. If suspected, biochemical assessment of the hypothalamic-pituitary-adrenal (HPA) axis is indicated, beginning with a first-morning cortisol measurement. If results are inconclusive, a low-dose adrenocorticotrophic hormone stimulation test may be performed. Confirmed cases of adrenal suppression necessitate treatment with daily physiologic corticosteroid doses and additional stress dosing when required [21]. Patients on concurrent corticosteroid and nonsteroidal anti-inflammatory drug (NSAID) therapy, or those at elevated risk of gastrointestinal (GI) complications (e.g., history of ulcers or advanced comorbidities), should receive prophylactic proton pump inhibitor (PPI) therapy to prevent ulcers and GI bleeding. To reduce the risk of striae, patients are advised to maintain a low-calorie diet, and some benefit has been reported with treatments such as topical vitamin A creams, pulsed dye lasers, and non-ablative radiofrequency devices [2]. Corticosteroids with significant mineralocorticoid activity can result in fluid

and electrolyte imbalances, including water and salt retention and potassium excretion. As a result, regular monitoring of fluid and electrolyte levels is essential, especially in patients receiving corticosteroids with heightened mineralocorticoid activity [2].

Drug interactions are a critical aspect of corticosteroid management, as these agents can significantly alter the effects of concurrent medications. For instance, corticosteroids may potentiate the effects of anticoagulants like warfarin, necessitating closer monitoring and potential dosage adjustments, particularly between three to seven days after initiating corticosteroid therapy. Glucocorticoids may reduce the efficacy of antihyperglycemic agents, necessitating increased blood glucose monitoring and potential dose modifications. Interactions with antiviral agents can also occur; certain antivirals, such as efavirenz and nevirapine, may reduce corticosteroid effects, while others, including ritonavir and atazanavir, may enhance their effects, increasing the risk of toxicity. Combining corticosteroids with NSAIDs raises the risk of peptic ulcer disease, warranting the use of antacids or PPIs and, if possible, avoiding concomitant use [2]. Live or live-attenuated vaccines pose an infection risk when administered during immunosuppressive corticosteroid therapy (e.g., doses of ≥ 40 mg/day of prednisolone for over seven days). Consequently, it is recommended to defer vaccination for three months following the cessation of immunosuppressive corticosteroid treatment to minimize complications [2].

Toxicity

Corticosteroid toxicity is a significant concern in chronic therapy and is recognized as one of the most frequent causes of iatrogenic illness. Unfortunately, there is no specific antidote or reversal agent for corticosteroid toxicity. Management revolves around addressing individual complications—such as hyperglycemia, infections, or hypertension—and gradually tapering the corticosteroid dose. The approach to tapering depends on the duration and intensity of corticosteroid therapy. For patients undergoing therapy for less than a week, abrupt discontinuation is generally safe and does not necessitate tapering. However, for those receiving corticosteroids for one to three weeks, the decision to taper must be informed by the patient's clinical condition and the reason for initiating corticosteroid therapy. Rapid discontinuation in these cases can precipitate adrenocorticotrophic hormone (ACTH) suppression and result in a rebound exacerbation of the underlying disease. For prolonged corticosteroid courses exceeding three weeks, a carefully orchestrated tapering plan is crucial. This involves a rapid reduction to physiological doses followed by a more gradual weaning process, during which adrenal function should be periodically assessed. Long-term, high-dose corticosteroid therapy can suppress the hypothalamic-pituitary-adrenal (HPA) axis, a condition that may persist for nine to twelve months after cessation of treatment. This prolonged suppression necessitates a cautious approach to withdrawal to avoid adrenal insufficiency, which can have life-threatening consequences. Monitoring for signs of HPA axis recovery, such as normal cortisol levels, is essential during this phase to guide the tapering process effectively [2].

In managing corticosteroid toxicity, early recognition and treatment of specific complications are imperative. Hyperglycemia, one of the most common adverse effects, requires vigilant glucose monitoring and potentially the use of antihyperglycemic agents. Similarly, corticosteroid-induced hypertension necessitates blood pressure management through lifestyle modifications or pharmacologic interventions. The immunosuppressive properties of corticosteroids heighten infection risks, underscoring the importance of preventive measures, including vaccination and infection control protocols. Bone health is another critical aspect of managing toxicity, as prolonged corticosteroid use can lead to osteoporosis. Patients should be assessed for bone mineral density, and prophylactic measures such as calcium and vitamin D supplementation or bisphosphonate therapy should be implemented as needed. By addressing these complications proactively, healthcare providers can minimize the harmful effects of corticosteroids and optimize therapeutic outcomes. Despite their toxicity, corticosteroids remain indispensable in managing numerous medical conditions. However, their potential for causing significant harm highlights the necessity for clinicians to exercise judicious prescribing practices. Adopting a risk-benefit approach when initiating corticosteroid therapy ensures that these medications are reserved for cases where their benefits clearly outweigh their risks. Close collaboration among healthcare team members and regular patient monitoring are essential to identify early signs of toxicity and adapt treatment strategies

accordingly. Through a combination of careful dosing, tailored tapering protocols, and vigilant complication management, healthcare providers can mitigate the risks associated with corticosteroid toxicity while maximizing their therapeutic potential.

Enhancing Healthcare Team Outcomes

Corticosteroids, though immensely beneficial in treating various medical conditions, are accompanied by a broad spectrum of adverse effects. These effects, which can be therapeutic for one patient and detrimental for another, underscore the importance of comprehensive understanding and careful management by the entire interprofessional healthcare team. For instance, the glucose-elevating properties of corticosteroids are invaluable in patients with adrenal insufficiency but can prove detrimental in individuals with latent or overt diabetes mellitus. Consequently, the effective use of corticosteroids requires a collaborative approach to ensure optimal outcomes while minimizing risks [2]. Healthcare providers, including physicians, mid-level practitioners, nurses, and pharmacists, must engage in consistent monitoring of patients receiving corticosteroid therapy. Preventive strategies should be prioritized to mitigate adverse effects, such as recommending lifestyle modifications, prescribing prophylactic treatments, and monitoring for complications like hyperglycemia, hypertension, or osteoporosis. Educating patients about the potential side effects of corticosteroids and the necessary preventive measures is a critical responsibility shared by all members of the healthcare team. This includes discussing dietary adjustments, the importance of adhering to prescribed doses, and the need for regular follow-ups. To ensure seamless communication within the healthcare team, electronic medical records (EMRs) can serve as a valuable tool. By tagging patients on chronic corticosteroid therapy within the EMR system, healthcare professionals are promptly alerted to this critical aspect of their medical history. This facilitates coordinated care, reducing the likelihood of errors or oversights in medication management. Additionally, patients should be encouraged to carry a treatment card or another form of identification indicating their corticosteroid use. Such measures are particularly important in emergency situations where healthcare providers may lack immediate access to the patient's medical history.

Patient education remains a cornerstone of minimizing corticosteroid-related morbidity and mortality. Patients must be informed about recognizing early signs of complications, such as infections, severe fatigue, or unexplained pain, and the importance of seeking timely medical attention. For patients on long-term therapy, education should also include discussions about the risks of adrenal insufficiency during abrupt discontinuation or periods of high stress. By empowering patients with knowledge, healthcare providers can enhance adherence to treatment plans and encourage proactive management of potential side effects. Interprofessional collaboration is pivotal to optimizing corticosteroid therapy outcomes. Pharmacists, for example, can play a crucial role in identifying potential drug interactions, such as those between corticosteroids and anticoagulants or antihyperglycemic agents, and recommending appropriate adjustments. Nurses, meanwhile, are often the first to identify signs of adverse effects during routine care and can provide critical feedback to the prescribing physician. Regular team meetings and open communication channels ensure that all members of the healthcare team remain aligned in their efforts to deliver safe and effective care. Ultimately, the success of corticosteroid therapy hinges on a comprehensive, team-based approach that prioritizes patient safety, education, and meticulous monitoring. By leveraging the strengths of an interprofessional team, healthcare providers can maximize the therapeutic benefits of corticosteroids while minimizing their associated risks.

Conclusion:

Corticosteroids are among the most versatile pharmacological agents in modern medicine, demonstrating extensive utility across numerous medical specialties. Their ability to regulate immune and inflammatory responses has established their role in treating conditions such as asthma, rheumatoid arthritis, and autoimmune diseases. Additionally, their endocrine applications, including adrenal insufficiency management and congenital adrenal hyperplasia treatment, underscore their significance in hormonal regulation. The dual mechanisms of corticosteroids—genomic and non-genomic—provide a robust framework for their efficacy. Genomic pathways enable transcriptional modulation of pro-inflammatory

mediators, while non-genomic mechanisms rapidly attenuate inflammatory responses. Despite their therapeutic potential, the extensive adverse effect profile of corticosteroids poses significant challenges. Long-term use is associated with osteoporosis, adrenal suppression, Cushingoid features, hyperglycemia, cardiovascular complications, and psychiatric disturbances. These effects necessitate a risk-benefit assessment before initiating therapy, alongside strategies to mitigate side effects, such as opting for non-systemic administration routes when feasible. Healthcare professionals must be vigilant in monitoring patients on corticosteroids, especially those on prolonged or high-dose regimens. The importance of gradual tapering to prevent adrenal insufficiency and the potential contraindications, including interactions with live vaccines or systemic fungal infections, cannot be overstated. Emerging research into corticosteroid-sparing agents and adjunct therapies holds promise for minimizing dependence on these agents while preserving therapeutic efficacy. In conclusion, corticosteroids, while indispensable in managing a spectrum of diseases, require judicious use and comprehensive patient management to balance their benefits against the risks. Enhanced understanding and continuous research are essential to optimize their clinical applications and improve patient outcomes.

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الكورتيكوستيرويدات: مراجعة محدثة لمتخصصي الرعاية الصحية

الملخص:

الخلفية: الكورتيكوستيرويدات هي نظائر اصطناعية لهرمونات قشرة الغدة الكظرية، وتستخدم على نطاق واسع في العديد من المجالات الطبية بسبب خصائصها المضادة للالتهابات والمثبطة للمناعة. تشمل هذه الأدوية الجلوكوكورتيكويدات والمينيرالوكورتيكويدات، وتلعب أدوارًا حيوية في تنظيم التمثيل الغذائي، والاستجابات المناعية، وتوازن الإلكتروليتات.

الهدف: تقدم هذه المراجعة نظرة عامة محدثة حول الكورتيكوستيرويدات، بما في ذلك آليات عملها، وتطبيقاتها العلاجية، وأثارها الجانبية، وموانع استخدامها، لتوفير رؤى أساسية لمتخصصي الرعاية الصحية.

المنهجية: تم إجراء مراجعة شاملة للأدبيات العلمية، مع التركيز على الخصائص الدوائية، والاستخدامات السريرية، والآثار الجانبية للكورتيكوستيرويدات. تم تحليل الآليات الرئيسية، وطرق الإعطاء، والاعتبارات الخاصة ببدء العلاج.

النتائج: تظهر الكورتيكوستيرويدات آليات متعددة الجوانب من خلال المسارات الجينية وغير الجينية، مما يمكنها من تثبيط الوسائط الالتهابية وتعديل الاستجابات المناعية. تُستخدم في علاج حالات مثل الربو، والتهاب المفاصل الروماتويدي، وأمراض الأمعاء الالتهابية، وقصور الغدة الكظرية. ومع ذلك، يحد من استخدامها الآثار الجانبية مثل هشاشة العظام، وتثبيط الغدة الكظرية، وارتفاع سكر الدم، والاعتلال العضلي، والاضطرابات النفسية. يؤثر اختيار طريقة الإعطاء على الكفاءة ومظاهر الآثار الجانبية، حيث يرتبط التعرض الجهازى غالبًا بزيادة المخاطر.

الخلاصة: تظل الكورتيكوستيرويدات لا غنى عنها في الممارسة السريرية، ولكن أثارها الجانبية المحتملة تستدعي استخدامها بحذر مع تحديد الجرعات بعناية والمراقبة المنتظمة. فهم آلياتها وإمكاناتها العلاجية ومخاطرها أمر ضروري لتحسين نتائج المرضى.

الكلمات المفتاحية: الكورتيكوستيرويدات، الجلوكوكورتيكويدات، المينيرالوكورتيكويدات، مضاد للالتهابات، مثبط للمناعة، الآثار الجانبية، التطبيقات العلاجية