

Systemic corticosteroids

A family of synthetic glucocorticoids - prednisone, prednisolone, methylprednisolone, dexamethasone and hydrocortisone - that all work through the same intracellular receptor but differ enough in potency and duration of action that mixing them up changes both the dose and the tapering plan a patient actually needs.

Online guide: <https://www.medguideglobal.com/pharmacology/endocrine-and-metabolic/systemic-corticosteroids>

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Quick revision

One Liner: All systemic corticosteroids bind the same intracellular glucocorticoid receptor and suppress inflammatory gene transcription, but they are not interchangeable dose-for-dose or duration-for-duration - potency and half-life vary five- to twenty-five-fold across the class, and any of them given at supraphysiologic doses for more than a few weeks can suppress the HPA axis badly enough that stopping abruptly, rather than tapering, is what actually causes harm.

1. Corticosteroids bind the intracellular glucocorticoid receptor, and the activated receptor translocates to the nucleus to suppress transcription of pro-inflammatory genes - the genomic mechanism responsible for most of the drug class's anti-inflammatory effect. [3]
2. Hydrocortisone is the potency reference point (short-acting, roughly equivalent to physiologic cortisol); prednisone/prednisolone/methylprednisolone are intermediate-acting and several times more potent; dexamethasone is long-acting and roughly 25 times more potent than hydrocortisone on a milligram basis. [2]
3. Exogenous corticosteroids suppress ACTH via negative feedback on the hypothalamic-pituitary-adrenal (HPA) axis, and neither the dose nor the duration of therapy reliably predicts which patient will develop clinically significant suppression. [3]
4. After roughly three weeks or more of supraphysiologic dosing, labels and guidance recommend a taper to physiologic doses followed by slow weaning, rather than abrupt discontinuation, because HPA suppression can persist for months after the drug is stopped. [1][3]
5. The major adverse effect profile spans hyperglycemia, osteoporosis, adrenal suppression, increased infection risk from immunosuppression, psychiatric effects ranging from insomnia to frank psychosis, cushingoid features, and growth suppression in children. [4]
6. "Physiologic" dosing replaces the roughly 15-25 mg/day of hydrocortisone-equivalent cortisol the body normally makes; "stress dosing" temporarily raises that during acute illness or surgery in a patient with HPA suppression; "pharmacologic" dosing is the much higher, supraphysiologic dosing used for its anti-inflammatory or immunosuppressive effect rather than to replace a deficiency. [3]

Overview

1. "Corticosteroid" covers two different hormone families - glucocorticoids and mineralocorticoids - but the drugs grouped on this page are the glucocorticoids used systemically for their anti-inflammatory and immunosuppressive effect: prednisone, prednisolone, methylprednisolone, dexamethasone and hydrocortisone. All five are synthetic or semisynthetic analogs of cortisol, the body's own principal glucocorticoid, and all five act through the identical intracellular glucocorticoid receptor. What differs across the five is not the mechanism but the pharmacology built on top of it - relative potency, mineralocorticoid activity, and duration of action - and those differences are exactly what a clinician is choosing between when selecting one drug over another for a given indication. [3]
2. The receptor-level mechanism has two components. The genomic pathway, responsible for most of the clinically relevant effect, has the corticosteroid-bound receptor translocate to the nucleus and alter gene transcription - chiefly suppressing genes for pro-inflammatory cytokines and other inflammatory mediators, though the receptor can also

increase transcription of some anti-inflammatory proteins. A faster, non-genomic pathway, acting within seconds to minutes rather than requiring new protein synthesis, includes effects such as inhibition of phospholipase A2, which reduces the availability of arachidonic acid for prostaglandin and leukotriene synthesis. The genomic pathway explains why corticosteroid benefit in an inflammatory flare typically takes hours, not minutes, to become apparent. [3]

3. Because the drugs are glucocorticoid-receptor agonists, they inevitably reproduce cortisol's other physiologic roles as well as its anti-inflammatory one, and the HPA axis is the clearest example. Cortisol normally suppresses its own further production through negative feedback on the hypothalamus and anterior pituitary; an exogenous corticosteroid does exactly the same thing to the patient's own axis, suppressing ACTH and, over time, the adrenal cortex's own capacity to produce cortisol. Critically, StatPearls notes that neither the dose nor the duration of corticosteroid therapy reliably predicts which patient develops clinically significant suppression - even inhaled or topical corticosteroids can suppress the axis in a susceptible patient - which is why the tapering decision is made case by case rather than by a fixed rule. [3]

4. That HPA-suppression risk is the reason abrupt discontinuation after any meaningfully prolonged course of supraphysiologic corticosteroid therapy is discouraged. The prednisone label states plainly that if the drug is to be stopped after long-term therapy, it should be withdrawn gradually rather than abruptly, because drug-induced secondary adrenocortical insufficiency is minimised by a gradual dose reduction. Suppression can persist for months after the corticosteroid itself is stopped, so the practical consequence for a patient during that window is that any new physiologic stress - an infection, surgery, trauma - may need exogenous "stress dose" corticosteroid coverage because their own adrenal glands cannot yet respond adequately on their own. [1][2]

Classification and drug examples

Intro: The five drugs on this page are grouped by duration of action, which tracks reasonably well with relative anti-inflammatory potency: shorter-acting agents tend to be weaker per milligram, longer-acting agents stronger, with the intermediate group doing most of the day-to-day work in general medicine.

1. Name: Short-acting glucocorticoid

1. Note: The potency and duration reference point for the whole class; also the closest analog to the body's own cortisol and the standard choice for physiologic replacement and stress dosing.

Name: Hydrocortisone

1. Cortisol

2. Solu-Cortef (IV form)

Route: Oral or IV

Note: The reference agent for relative-potency tables (potency = 1); has meaningful mineralocorticoid activity, unlike the more potent synthetic agents. [2] [2]

2. Name: Intermediate-acting glucocorticoid

2. Note: The workhorse group for most systemic anti-inflammatory and immunosuppressive indications; roughly four to five times more potent than hydrocortisone per milligram, with substantially reduced mineralocorticoid activity relative to hydrocortisone.

1. Name: Prednisone

1. Route: Oral

1. Note: A prodrug converted hepatically to prednisolone, its active form; the most commonly prescribed oral corticosteroid in general practice. [1]

2. Name: Prednisolone

2. Route: Oral

2. Note: The active metabolite of prednisone; used directly when hepatic conversion is a concern, such as significant liver disease. [2]

3. Name: Methylprednisolone

1. Medrol

2. Solu-Medrol (IV form)

3. Route: Oral or IV

3. Note: Similar potency class to prednisolone with slightly less mineralocorticoid activity; commonly used IV for acute exacerbations. [2] [2]

3. Name: Long-acting glucocorticoid

3. Note: Substantially more potent per milligram and essentially free of mineralocorticoid activity, but with a longer duration of HPA suppression per dose than the intermediate-acting agents.

Name: Dexamethasone

Decadron

Route: Oral or IV

Note: Roughly 25-30 times more potent than hydrocortisone per milligram with negligible mineralocorticoid activity; used where a longer, more potent effect is wanted, including cerebral edema and antiemetic protocols. [2] [2]

Mechanism of action

Summary: Corticosteroids diffuse across the cell membrane and bind the cytoplasmic glucocorticoid receptor. The hormone-receptor complex translocates to the nucleus, where it binds glucocorticoid response elements in DNA and, predominantly, suppresses transcription of genes encoding pro-inflammatory cytokines, chemokines, adhesion molecules and enzymes such as cyclooxygenase-2 and inducible nitric oxide synthase, while modestly increasing transcription of some anti-inflammatory proteins. A faster non-genomic component, including inhibition of phospholipase A2 (which reduces arachidonic acid release and downstream prostaglandin/leukotriene synthesis), contributes an additional, more rapid layer of anti-inflammatory action.

Target: The intracellular glucocorticoid receptor (NR3C1)

Cidal Static: Anti-inflammatory and immunosuppressive via transcriptional regulation rather than cytotoxic - suppresses the inflammatory cascade rather than killing a cell population, though it does induce apoptosis in some lymphocyte populations at pharmacologic doses

Kill Kinetics: Non-genomic effects begin within minutes; the dominant genomic, transcription-mediated effect takes hours to become clinically apparent and requires ongoing receptor occupancy to sustain

1. Step: 1

1. Title: The corticosteroid crosses the cell membrane and binds the cytoplasmic glucocorticoid receptor

1. Detail: Because the drug is lipophilic, it diffuses freely into the cell rather than requiring a surface receptor or transporter. [3]

2. Step: 2

2. Title: The hormone-receptor complex translocates to the nucleus

2. Detail: Ligand binding causes a conformational change that releases the receptor from cytoplasmic chaperone proteins and allows nuclear entry. [3]

3. Step: 3

3. Title: The receptor complex binds glucocorticoid response elements and alters gene transcription

3. Detail: The dominant effect is transrepression of pro-inflammatory genes (cytokines, chemokines, adhesion molecules, COX-2), with some transactivation of anti-inflammatory genes. [3]

4. Step: 4

4. Title: Reduced inflammatory mediator synthesis dampens the inflammatory and immune response

4. Detail: This is the basis of the class's anti-inflammatory and, at higher doses, immunosuppressive effect across the many conditions it is used for. [3]

5. Step: 5

5. Title: The same receptor mediates negative feedback on the patient's own HPA axis

5. Detail: Exogenous glucocorticoid binding in the hypothalamus and anterior pituitary suppresses corticotropin-releasing hormone and ACTH release, which over time reduces the adrenal cortex's own cortisol output -

the mechanistic basis of HPA-axis suppression and the reason for tapering rather than abrupt withdrawal. [3]

Major clinical uses

1. Drug: Prednisone / prednisolone

1. Organism: Not applicable

1. Indication: A very broad range of inflammatory and autoimmune conditions, including asthma and COPD exacerbations, rheumatoid arthritis flares, inflammatory bowel disease, many dermatologic and allergic conditions, and as part of some chemotherapy and transplant-immunosuppression regimens

1. Line: the default oral corticosteroid for most outpatient indications

1. Note: Prednisone requires hepatic conversion to prednisolone, its active form, which matters in significant liver disease. [1]

2. Drug: Methylprednisolone

2. Organism: Not applicable

2. Indication: Similar indication range to prednisone, plus common use for IV pulse dosing in acute severe flares (for example, severe asthma exacerbation, multiple sclerosis relapse, acute transplant rejection)

2. Line: preferred when an IV route or a slightly different potency/mineralocorticoid profile is wanted

2. Note: Not a prodrug in the way prednisone is, so it does not depend on the same hepatic activation step. [2]

3. Drug: Dexamethasone

3. Organism: Not applicable

3. Indication: Cerebral edema, antenatal fetal lung maturation, chemotherapy-associated nausea, croup, and diagnostic use in the dexamethasone suppression test for Cushing syndrome

3. Line: chosen specifically for its long duration, high potency and negligible mineralocorticoid activity

3. Note: Its long duration of action also means a longer period of HPA suppression per dose than the intermediate-acting agents. [2]

4. Drug: Hydrocortisone

4. Organism: Not applicable

4. Indication: Physiologic glucocorticoid replacement in adrenal insufficiency, and stress-dose coverage during acute illness or surgery in a patient with known or suspected HPA suppression

4. Line: the standard choice specifically because its potency and mineralocorticoid activity most closely mirror the body's own cortisol

4. Note: Rarely used at the higher end of its dose range for pure anti-inflammatory effect, since the volume/mineralocorticoid load becomes impractical. [2]

Pharmacokinetics

1. Drug: Prednisone

1. Route: Oral

1. Absorption: Well absorbed orally

1. Csf Penetration: See prescribing reference

1. Metabolism: Hepatic conversion (prodrug) to prednisolone, its active form

1. Elimination: Hepatic and renal

1. Dose Adjust: Hepatic impairment can reduce conversion to the active metabolite, a reason to consider prednisolone directly instead [1]

2. Drug: Methylprednisolone

2. Route: Oral or IV

2. Absorption: Well absorbed orally; immediate with IV administration

2. Csf Penetration: See prescribing reference

2. Metabolism: Hepatic

- 2. Elimination: Hepatic and renal
- 2. Dose Adjust: Hepatic impairment [2]
- 3. Drug: Dexamethasone
- 3. Route: Oral or IV
- 3. Absorption: Well absorbed orally
- 3. Csf Penetration: See prescribing reference
- 3. Metabolism: Hepatic, partly via CYP3A4
- 3. Elimination: Hepatic and renal
- 3. Dose Adjust: Concomitant strong CYP3A4 inducers/inhibitors; hepatic impairment [2]
- 4. Drug: Hydrocortisone
- 4. Route: Oral or IV
- 4. Absorption: Well absorbed orally
- 4. Csf Penetration: See prescribing reference
- 4. Metabolism: Hepatic
- 4. Elimination: Hepatic and renal
- 4. Dose Adjust: Hepatic impairment [2]

- 1. This page gives no dose regimens by design. Physiologic replacement, stress dosing and pharmacologic anti-inflammatory dosing are entirely different orders of magnitude for the same drug, and the correct one depends on the indication, the individual patient's HPA status, and current guideline or prescribing-reference dosing tables.
- 2. Relative potency and duration of action, not absorption or elimination route, are the pharmacologic properties that most differentiate members of this class from one another - which is why a comparison table of milligram-equivalence is more clinically useful here than a conventional pharmacokinetic table. [2]
- 3. Prednisone's dependence on hepatic conversion to prednisolone is the one metabolism detail in this class with a direct prescribing consequence: in a patient with significant liver disease, prednisolone is sometimes used directly to avoid relying on impaired hepatic activation. [1]

Adverse effects

- 1. Effect: Hyperglycemia
 - 1. Detail: A dose-dependent rise in fasting and postprandial glucose is described with systemic corticosteroid use, relevant to both diabetic and non-diabetic patients on higher or prolonged doses. [4]
- 2. Effect: Cushingoid features
 - 2. Detail: Truncal obesity, a buffalo hump and moon facies develop with sustained supraphysiologic dosing, reflecting the same fat-redistribution effect seen in endogenous Cushing syndrome. [4]
- 3. Effect: Mood and sleep effects
 - 3. Detail: Insomnia, mood fluctuation and euphoria are common even at moderate doses; more severe psychiatric effects are described separately below. [4]
- 4. Effect: Fluid retention and hypertension
 - 4. Detail: Agents with meaningful mineralocorticoid activity, particularly hydrocortisone, cause sodium retention, fluid retention, hypertension and potassium loss more than the low-mineralocorticoid-activity agents such as dexamethasone. [1]
- 1. Effect: HPA axis suppression and adrenal insufficiency
 - 1. Detail: Prolonged ACTH suppression leads to adrenal cortical atrophy; the label states that neither dose nor duration reliably predicts which patient is affected, and suppression can persist for months after stopping the drug.
 - 1. Action: Gradual dose reduction (tapering) rather than abrupt discontinuation after any meaningfully prolonged course of supraphysiologic dosing; stress-dose coverage during acute illness or surgery in a patient with known suppression. [1][3]
- 2. Effect: Osteoporosis

2. Detail: Up to roughly 40% of patients on long-term glucocorticoids develop bone loss significant enough to raise fracture risk, which is the clinical link between this class and the glucocorticoid-induced osteoporosis guidance covered on the bisphosphonates and bone agents page.
2. Action: Fracture-risk assessment and, where indicated, bone-protective therapy are recommended for patients expected to need more than about three months of glucocorticoid treatment at meaningful doses. [4]
3. Effect: Immunosuppression and infection risk
3. Detail: Risk of serious and opportunistic infection rises roughly linearly with both dose and duration of corticosteroid therapy.
3. Action: Screening for latent infections such as tuberculosis is considered before prolonged high-dose therapy, and new infectious symptoms during treatment are evaluated promptly. [4]
4. Effect: Psychiatric effects
4. Detail: A wide range is reported, from aggression, insomnia and mood fluctuation at lower or shorter exposure to depression, manic behaviour and frank psychosis at higher or more prolonged exposure.
4. Action: New or worsening psychiatric symptoms during corticosteroid therapy are reported to the treating clinician rather than managed by the patient alone. [4]
5. Effect: Growth suppression in children
5. Detail: Growth deceleration is documented with prolonged systemic corticosteroid use in the pediatric population.
5. Action: Growth is monitored during prolonged pediatric courses, and the lowest effective dose and shortest effective duration are used where possible. [1][4]
1. Drug: Hydrocortisone
1. Effect: Retains meaningful mineralocorticoid activity relative to the synthetic agents, making sodium retention, edema and hypokalemia more prominent at equivalent anti-inflammatory potency. [1]
2. Drug: Dexamethasone
2. Effect: Longest duration of action and highest anti-inflammatory potency per milligram of the drugs on this page, with negligible mineralocorticoid activity - a longer single dose translates to a longer single episode of HPA suppression. [2]
3. Drug: Prednisone
3. Effect: Requires hepatic conversion to its active form, prednisolone, a step that is not needed with the other agents on this page. [1]

Contraindications, precautions and interactions

1. Known hypersensitivity to the specific corticosteroid or its formulation excipients. [1][2]
2. Systemic fungal infection is a contraindication to systemic corticosteroid use in the prednisone label, given the immunosuppressive effect. [1]
1. Active or latent infection, including tuberculosis, is a major caution given the immunosuppressive effect of this class. [4]
2. Diabetes mellitus or a strong predisposition to it is a caution because of the hyperglycemic effect of systemic corticosteroids. [4]
3. Osteoporosis or elevated fracture risk is a caution for any course expected to exceed roughly three months, per the reasoning behind the ACR glucocorticoid-induced osteoporosis guideline. [5]
4. Peptic ulcer disease and psychiatric illness are both cautions given the class's gastrointestinal and psychiatric adverse-effect profile. [1]
5. Any patient recently on a prolonged course of supraphysiologic corticosteroid dosing should be assumed to have some degree of HPA suppression when facing acute illness, trauma or surgery, even after the drug itself has been stopped for weeks to months. [2]
1. With: NSAIDs and aspirin
1. Effect: Additive gastrointestinal irritation and ulcer risk when combined with systemic corticosteroids. [1]
2. With: Strong CYP3A4 inducers or inhibitors

2. Effect: Alter dexamethasone and, to a lesser extent, other corticosteroid exposure; dose adjustment may be needed. [2]

3. With: Live or live-attenuated vaccines

3. Effect: Generally avoided during immunosuppressive-dose corticosteroid therapy because of blunted immune response and theoretical risk of vaccine-strain infection. [1]

Comparison tables

Title: Relative potency and duration of action

Caption: Approximate equivalent doses and relative anti-inflammatory potency, referenced to hydrocortisone; mineralocorticoid activity falls as synthetic potency rises. Figures are for comparison and education only, not for dose conversion in a specific patient.

1. Key: drug

1. Label: Drug

1. Is Row Header: true

2. Key: duration_class

2. Label: Duration class

3. Key: equivalent_dose

3. Label: Approx. equivalent dose

4. Key: mineralocorticoid

4. Label: Mineralocorticoid activity

1. Drug: Hydrocortisone

1. Duration Class: Short-acting (biologic half-life ~8-12h)

1. Equivalent Dose: 20 mg

1. Mineralocorticoid: High, relative to the synthetic agents [2]

2. Drug: Prednisone / prednisolone

2. Duration Class: Intermediate-acting (~18-36h)

2. Equivalent Dose: 5 mg

2. Mineralocorticoid: Low [2]

3. Drug: Methylprednisolone

3. Duration Class: Intermediate-acting (~18-36h)

3. Equivalent Dose: 4 mg

3. Mineralocorticoid: Minimal [2]

4. Drug: Dexamethasone

4. Duration Class: Long-acting (~36-54h)

4. Equivalent Dose: 0.75 mg

4. Mineralocorticoid: Negligible [2]

High-yield exam pearls

1. Pearl: The genomic mechanism (transcriptional suppression of inflammatory genes) is slow, over hours; the non-genomic mechanism (e.g. phospholipase A2 inhibition) is fast, over minutes.

1. Why: It explains why a patient in acute anaphylaxis is given epinephrine, not just a corticosteroid, even though a corticosteroid is part of the treatment - its main benefit takes hours to appear. [3]

2. Pearl: Neither dose nor duration of corticosteroid therapy reliably predicts which patient develops clinically significant HPA suppression - even topical or inhaled steroids can do it in a susceptible patient.

2. Why: It is the reason clinicians err toward tapering and toward assuming possible adrenal suppression in any patient with a meaningful corticosteroid exposure history, rather than relying on a fixed dose or duration cutoff. [3]

3. Pearl: Dexamethasone's near-zero mineralocorticoid activity is precisely why it, not hydrocortisone, is preferred in

situations needing high glucocorticoid potency without fluid retention, such as cerebral edema.

3. Why: The dissociation between glucocorticoid and mineralocorticoid potency across the class is a recurring exam theme distinguishing dexamethasone from hydrocortisone. [2]

4. Pearl: "Physiologic," "stress," and "pharmacologic" dosing are three different orders of magnitude of the same drug, not three different drugs.

4. Why: Confusing these is a common source of dosing errors and of exam-question traps that ask what dose a patient with adrenal insufficiency needs during surgery versus during routine daily replacement. [3]

5. Pearl: Prednisone itself is inactive until hepatically converted to prednisolone.

5. Why: It is the one drug on this page where significant liver disease is a specific reason to choose a different, already-active agent instead. [1]

Common exam traps

1. Trap: Assuming all systemic corticosteroids are dosed the same way milligram-for-milligram.

1. Correction: Relative potency varies roughly 25-fold across this class (hydrocortisone 20 mg being roughly equivalent to dexamethasone 0.75 mg), so any dose comparison must use an equivalence table, not a 1:1 substitution. [2]

2. Trap: Believing a short course of corticosteroids can always be stopped abruptly without consequence.

2. Correction: Even courses as short as one to three weeks may need a tapering decision depending on dose and clinical context, because dose and duration do not reliably predict which patient has developed HPA suppression. [3]

3. Trap: Treating hydrocortisone as simply a weaker version of prednisone or dexamethasone.

3. Correction: Hydrocortisone's lower glucocorticoid potency comes with proportionally higher mineralocorticoid activity, which is exactly why it - not a more potent synthetic agent - is the standard choice for physiologic adrenal-insufficiency replacement, where that mineralocorticoid activity is wanted rather than avoided. [2]

4. Trap: Assuming corticosteroid adverse effects require months of therapy to appear.

4. Correction: Psychiatric effects, hyperglycemia and fluid retention can appear within days at higher doses; osteoporosis, cushingoid features and HPA suppression are the ones that specifically accumulate with duration. [4]

Self-test questions

1. Question: What is the dominant molecular mechanism by which systemic corticosteroids suppress inflammation?

1. Direct inhibition of cyclooxygenase enzyme activity

2. Binding the intracellular glucocorticoid receptor and altering transcription of inflammatory genes

3. Blocking histamine H1 receptors

4. Inhibiting bacterial cell wall synthesis

1. Explanation: Corticosteroids bind the cytoplasmic glucocorticoid receptor; the hormone-receptor complex translocates to the nucleus and predominantly suppresses transcription of pro-inflammatory genes, which is the genomic mechanism responsible for most of the class's anti-inflammatory effect. [3]

2. Question: What does the prednisone label say about stopping the drug after long-term therapy?

1. It should be stopped abruptly to avoid dependency

2. It should be withdrawn gradually rather than abruptly, to minimise drug-induced adrenocortical insufficiency

3. No tapering is ever necessary regardless of dose or duration

4. It can only be stopped by switching directly to dexamethasone

2. Explanation: The label states that after long-term therapy, prednisone should be withdrawn gradually rather than abruptly, because gradual dose reduction minimises drug-induced secondary adrenocortical insufficiency. [1]

3. Question: Which of the following statements about predicting HPA axis suppression is correct?

1. High doses for less than one week never cause suppression

2. Neither dose nor duration of corticosteroid therapy reliably predicts which patient develops clinically significant suppression

3. Only IV corticosteroids can suppress the HPA axis

4. Suppression always resolves within 48 hours of stopping the drug

3. Explanation: StatPearls specifically notes that duration and dosage of corticosteroid therapy are not reliable predictors of HPA suppression, and that suppression can persist for months after the drug is stopped. [3]

4. Question: Why is dexamethasone often preferred over hydrocortisone when a high-potency glucocorticoid effect without fluid retention is needed, such as in cerebral edema?

1. Dexamethasone has higher mineralocorticoid activity

2. Dexamethasone has substantially higher glucocorticoid potency and negligible mineralocorticoid activity compared with hydrocortisone

3. Dexamethasone cannot cross into the central nervous system

4. Hydrocortisone is not absorbed orally

4. Explanation: Dexamethasone is roughly 25-30 times more potent than hydrocortisone on a glucocorticoid basis while having negligible mineralocorticoid activity, avoiding the sodium retention and edema that a mineralocorticoid-active agent like hydrocortisone would cause at an equivalent anti-inflammatory dose. [2]

5. Question: Which adverse effect is specifically tied to the duration of corticosteroid therapy rather than appearing rapidly?

1. Insomnia

2. Osteoporosis

3. Acute hyperglycemia

4. Injection-site reaction

5. Explanation: Osteoporosis develops with sustained glucocorticoid exposure - up to roughly 40% of patients on long-term therapy develop clinically significant bone loss - in contrast to effects such as insomnia or hyperglycemia, which can appear within days. [4]

6. Question: Why is prednisolone sometimes used directly instead of prednisone in a patient with significant liver disease?

1. Prednisolone has no adverse effects

2. Prednisone is a prodrug requiring hepatic conversion to prednisolone, its active form, so impaired liver function can reduce its effect

3. Prednisolone is not metabolised at all

4. Prednisone cannot be given orally

6. Explanation: Prednisone must be converted hepatically to prednisolone to become active; in a patient with significant hepatic impairment that conversion may be reduced, so prednisolone is sometimes given directly instead. [1]

Frequently asked questions

1. Question: Are all systemic corticosteroids equally strong?

1. Answer: No. They share the same receptor mechanism but differ substantially in potency and duration of action. Hydrocortisone is the weakest and shortest-acting of the group discussed here; prednisone, prednisolone and methylprednisolone are several times more potent and intermediate-acting; dexamethasone is the most potent per milligram and the longest-acting, with negligible mineralocorticoid activity. Equivalent-dose tables exist specifically because a 1:1 substitution between them is not appropriate. [2]

2. Question: Why can't a patient just stop taking a corticosteroid once they feel better?

2. Answer: Exogenous corticosteroids suppress the body's own hypothalamic-pituitary-adrenal axis, and neither the dose nor the length of the course reliably predicts how much suppression has occurred. Stopping abruptly after a meaningfully prolonged or high-dose course risks adrenal insufficiency, because the patient's own adrenal glands may not yet be able to produce enough cortisol on their own - this is why tapering, rather than stopping outright, is the standard approach after longer courses. [1][3]

3. Question: What is the difference between physiologic, stress, and pharmacologic corticosteroid dosing?

3. Answer: Physiologic dosing replaces the amount of cortisol the body would normally make on its own, roughly 15-25

mg/day of hydrocortisone-equivalent. Stress dosing temporarily increases that amount during acute illness, injury or surgery in a patient whose own adrenal response is impaired. Pharmacologic dosing is much higher again, used specifically for an anti-inflammatory or immunosuppressive effect rather than to replace a deficiency, and it is pharmacologic dosing that carries most of this class's characteristic adverse-effect profile. [3]

4. Question: What are the main long-term risks of taking a systemic corticosteroid?

4. Answer: The major risks described in the literature are hyperglycemia, osteoporosis and increased fracture risk, adrenal suppression, increased susceptibility to infection from the drug's immunosuppressive effect, psychiatric effects ranging from insomnia to, less commonly, frank psychosis, cushingoid features such as truncal weight redistribution, and growth suppression in children on prolonged courses. Risk generally rises with both dose and duration of therapy. [4]

References

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