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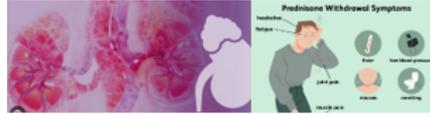
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2024年08月22日

ステロイド剤使用のガイドライン・2024年版

ステロイド剤使用のガイドライン・2024年版

European Society of Endocrinology and Endocrine Society
Joint Clinical Guideline: Diagnosis and therapy
of glucocorticoid-induced adrenal insufficiency



ステロイド剤は最強の抗炎症薬の一つです。症状が安定したら、いかに漸減していくかが問題です。ステロイド剤には、そのものの副作用とステロイド剤誘因の副作用不全も注意しなくてはなりません。

今回、ヨーロッパからガイドラインが出ていましたのでブログします。長い論文で実地医家にとってはやや饒舌な感じのため、journal Watchからも引用し、端折って纏めます。

- ステロイド剤の用量に関係なく、投与の期間が3~4週間以内なら漸減の方法をとらなくても良い。
- 投与の期間が3~4週間を超えた場合は、漸減方式が必要となる。それによってステロイド離脱症状を予防し、下垂体からの副腎刺激を回復できる。
- 漸減は疾患がコントロールされていることを確認する必要があります。
 - プレドニン換算で40mg/日を超えている場合は、5~10mg/週の割合で漸減していく。
 - 40mg/日以下の場合は、10~20mg/日になるまで2.5mg/1~4週間毎に漸減し、10mg/日になったら、1mg/1~4週間毎に漸減していく。
- 長時間作用型ステロイド（例えばデキサメタゾン）を処方している場合は、短期作用型ステロイド（プレドニンやハイドロコルチゾン）に変更してから漸減する。
- 漸減して生理的量（プレドニン換算で4~6mg）に達したら、早朝のコルチゾール値を測定し、10µg/dl以上なら視床下部-下垂体-副腎（HPA）の反応が回復しているのでステロイドを中止しても良い。10µg/dl以下の場合は漸増を継続し、数週間後に再検する。
- 考察
ステロイドを漸減していく際に、ステロイド離脱症状か副腎不全か、それとも本来の疾患の再発かを区別する事は簡単ではない事に留意する。

Glucocorticoid	Approximate equivalent dose ^a	Glucocorticoid potency relative to hydrocortisone ^b	Plasma half-life (mg) ^c	Biological half-life (hours) ^d	Therapeutic indications
Short-acting glucocorticoids with lower potency					
Hydrocortisone	20 mg	1.0	90-120	8-12	Adrenal insufficiency replacement
Corticone acetate	25 mg	0.8	80-120	8-12	Adrenal insufficiency replacement
Dexamethasone	7.5 mg	2.8	70-120	Not defined	Duchenne muscular dystrophy
Intermediate-acting glucocorticoids with moderate potency					
Prednisolone	5 mg	4.0	40	12-36	Anti-inflammatory, immunosuppression, Adrenal insufficiency replacement
Prednisolone	5 mg	4.0	115-200	12-36	Anti-inflammatory, immunosuppression, Adrenal insufficiency replacement
Triamcinolone	4 mg	5.0	30	12-36	Anti-inflammatory, immunosuppression
Methylprednisolone	4 mg	5.0	180	12-36	Anti-inflammatory, immunosuppression
Long-acting glucocorticoids with highest potency					
Dexamethasone	0.5 mg	30-40	200	36-72	Anti-inflammatory, immunosuppression, Usually reserved for short-term use in severe, acute conditions.
Betamethasone	0.5 mg	25-40	300	36-72	Anti-inflammatory, immunosuppression, Usually reserved for short-term use in severe, acute conditions.

^aThese are estimates based on historically accepted conversion factors and should be used as a guide only. There can be considerable variation depending on the route of administration, the individual patient's metabolism and excretion.

^bGlucocorticoid potency equivalence apply to oral use for systemic administration. Intracortical effects are not considered.

^cPlasma half-life does not reflect the biological half-life.

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以降はカテゴリで検索してください。

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Table 4. Suggested tapering regimen depending on glucocorticoid dose

Patient's current daily prednisone equivalent dose	Suggested prednisone decrements	Time interval
>40 mg	5-10 mg decrease	Every week
20-40 mg	5 mg decrease	Every week
10-20 mg	2.5 mg decrease	Every 1-4 weeks
5-10 mg	1 mg decrease	Every 1-4 weeks
5 mg	In absence of clinical symptoms or negative testing for adrenal insufficiency continue 1 mg decrease (if low dosage prednisolone preparations are not available, alternative: 20 mg hydrocortisone with 5 mg decrease)	Every 4 weeks

Table 5. Clinical features of adrenal insufficiency, glucocorticoid withdrawal syndrome and common underlying conditions

	Glucocorticoid withdrawal syndrome	Adrenal insufficiency	Underlying condition for which glucocorticoids were initially prescribed
Symptoms	General malaise, fatigue, nausea, muscle and joint pain, sleep disturbances, mood change	General malaise, fatigue, nausea, muscle and joint pain	Depending on condition (e.g. joint pain in rheumatoid arthritis). Common overlapping signs (general malaise, fatigue)
Signs	Cushingoid features common, especially earlier in the glucocorticoid taper	Weight loss (¹), hypotension, orthostasis	Disease specific signs (e.g. rash)
Timing of symptoms and signs	At any point during glucocorticoid taper, usually when prednisone is decreased <15 mg/day	Only when not treated with optimal glucocorticoid therapy	At any point during glucocorticoid taper if the underlying condition is suboptimally controlled with a non-glucocorticoid agent
Severity	Higher risk with long-term supraphysiologic glucocorticoid therapy	Increased glucocorticoid requirements due to adrenal insufficiency	
Biochemistry	Normal electrolytes	Normal electrolytes	Markers of disease activity (autoantibodies, disease-specific biomarkers)
HPA axis	Tinting is not recommended if normal, ACTH and cortisol are usually undetectable	Initially, low ACTH and cortisol. Later in recovery: normal elevated ACTH, low cortisol	Not applicable
Risk of adrenal crisis	Unlikely, if glucocorticoids are administered to patients with glucocorticoid withdrawal syndrome (do they have adrenal insufficiency?)	Yes, if not optimally treated with glucocorticoid therapy	Not applicable

¹ Weight loss due to reduced caloric intake. Cushing syndrome should be considered. **General remarks:** Patients with glucocorticoid-induced adrenal insufficiency may be asymptomatic at baseline conditions but can develop symptoms – from mild to life-threatening adrenal crisis – when tapered or stopped (Table 4). When present, symptoms of adrenal insufficiency are often non-specific and can overlap with those of the disease for which glucocorticoids are prescribed. Recovery of underlying autoimmune diseases can occur during tapering of exogenous glucocorticoids. Signs and symptoms of adrenal insufficiency can overlap with those of glucocorticoid withdrawal syndrome, which arises from the discontinuation of rapid tapering of glucocorticoid therapy in patients who developed a rebound to supraphysiologic glucocorticoid levels. In patients on glucocorticoids due to the physiological range, adrenal insufficiency and glucocorticoid withdrawal syndrome cannot be distinguished with complete accuracy.

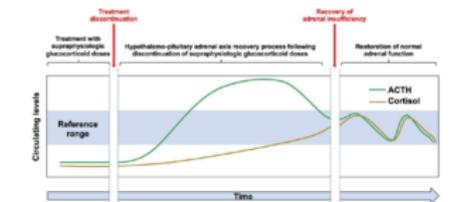
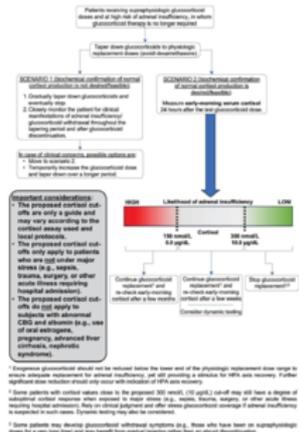


Figure 8. Schematic representation of HPA axis recovery following discontinuation of supraphysiologic glucocorticoid therapy (adapted from: Pheas and Barnes 2021¹⁷).



Proposed approach to systemic glucocorticoid discontinuation.

	Prevalence of glucocorticoid-induced adrenal insufficiency*	Factors increasing the risk of glucocorticoid-induced adrenal insufficiency	Strategies to mitigate the risk of glucocorticoid-induced adrenal insufficiency*
Inhaled glucocorticoids	<ul style="list-style-type: none"> Overall: 7.8% (CI 4.2-13.9) Short-term use (<1 month): 1.4% (CI 0.3-7.4) Medium-term use (1-12 months): 15.7% (CI 1.4-23.1) Long-term use (>12 months): 27.4% (CI 17.3-39.0) Low-dose use (2.4% (0.4-8.3)) Intermediate-dose use (8.1% (4.2-14.8)) High-dose use: 21.1% (12.0-35.1) 	<ul style="list-style-type: none"> Treatment with high-dose[†] for prolonged periods Use of fluticasone propionate Concomitant use of other glucocorticoid formulations (e.g., and glucocorticoids in chronic obstructive pulmonary disease or nasal glucocorticoids for rhinitis/allergies) Lower body mass index Higher compliance with treatment Concomitant treatment with strong cytochrome P450 3A4 inhibitor[‡] (e.g., medications containing ritonavir, atazanavir, darunavir, or efavirenz) 	<ul style="list-style-type: none"> Use the lowest effective glucocorticoid dose for the shortest period Use spacers and mouth rinsing Consider alternative glucocorticoids to fluticasone propionate Avoid co-administration with strong cytochrome P450 3A4 inhibitor[‡]
Intra-articular glucocorticoids	52.2% (40.3-63.6)	<ul style="list-style-type: none"> Repeated injections over a short period (<1 month) Simultaneous injections of multiple joints Use of high glucocorticoid doses Inflammatory arthropathies Concomitant use of other glucocorticoid formulations Concomitant treatment with strong cytochrome P450 3A4 inhibitor[‡] 	<ul style="list-style-type: none"> Reduce the number of injections, if possible Space out injections by at least 3-4 months, if possible Triamcinolone hexacetonide may carry a lower risk of systemic absorption than triamcinolone acetonide Avoid co-administration with strong cytochrome P450 3A4 inhibitor[‡]
Parenteral (parental) glucocorticoids	4.7% (CI 1.3-18.3)	<ul style="list-style-type: none"> Long-term use of high-potency glucocorticoids on large surface areas or areas of increased absorption (e.g., muscle) Prolonged use on inflamed skin with impaired barrier function Oxidative dressings Use on mucous membranes, orifices, and scrotum Concomitant use of other glucocorticoid formulations Concomitant treatment with strong cytochrome P450 3A4 inhibitor[‡] Long-term use Concomitant use of other glucocorticoid formulations Concomitant treatment with strong cytochrome P450 3A4 inhibitor[‡] 	<ul style="list-style-type: none"> Use the smallest effective quantity for the shortest period Use lower potency glucocorticoids, if possible Avoid co-administration with strong cytochrome P450 3A4 inhibitor[‡]
Intra-nasal glucocorticoids	4.2% (CI 0.3-28.9)	<ul style="list-style-type: none"> Long-term use Concomitant use of other glucocorticoid formulations Concomitant treatment with strong cytochrome P450 3A4 inhibitor[‡] 	<ul style="list-style-type: none"> Use the lowest effective glucocorticoid dose for the shortest period Avoid co-administration with strong cytochrome P450 3A4 inhibitor[‡]

Table 7: Signs and symptoms of glucocorticoid-induced (exogenous) Cushing syndrome

Symptoms	Signs	Other manifestations
<ul style="list-style-type: none"> Muscle weakness Sleep disturbances (insomnia) Increased appetite Mood and cognitive disturbances (irritability, impaired memory, depression) 	<ul style="list-style-type: none"> Proximal muscle weakness and wasting Excess weight gain and central obesity Supraclavicular and dorsocervical fat accumulation Facial and upper neck plethora with facial rounding Skin atrophy with easy bruising, red stretchmarks, and poor wound healing Acne Menstrual irregularities in women. 	<ul style="list-style-type: none"> Cardiometabolic risk factors (hypertension, dysglycemia, dyslipidemia, hypercoagulability) Osteoporosis and fragility fractures Hypogonadism, reduced libido, and reduced fertility

specialist. Patients with adrenal insufficiency for more than one year should be treated with standard replacement doses of hydrocortisone or prednisone (Table 1). Furthermore, it is necessary to provide education to these patients regarding the adjustment of glucocorticoid substitution therapy doses during stressful situations to prevent adrenal crises or to manage them³³ (see Section 3).

General considerations	<ul style="list-style-type: none"> Patients present with a shock out of proportion to the severity of the trigger, if a trigger is identified (see below). The shock is typically resistant to isotropes and fluid resuscitation if the adrenal crisis is not recognized and promptly treated with parenteral glucocorticoids. Risk factors for adrenal crisis include a history of previous adrenal crisis, older age (>65 years), adolescence and transition from pediatric to adult care, and a higher comorbidity burden. Glucocorticoid tapering down and discontinuation are critical times, as glucocorticoid-induced adrenal insufficiency can become clinically apparent.
Diagnosis	<p>Hypotension or hypotensive shock, plus at least one of the following:</p> <ul style="list-style-type: none"> Nausea or vomiting Severe fatigue Frustrated Impaired consciousness (incl. lethargy, confusion, somnolence, collapse, delirium, coma, and seizures)
Possible laboratory abnormalities (not required for the diagnosis)	<ul style="list-style-type: none"> Hypotension (especially with renal urinary sodium) Hypokalemia Signs of relative depletion (e.g., raised urea and creatinine) Hypoglycemia Leucocytosis Eosinophilia
Factors that can trigger an adrenal crisis or other symptoms of adrenal insufficiency	<p>Common to all patients with adrenal insufficiency</p> <ul style="list-style-type: none"> Infections (including gastrointestinal, genitourinary, respiratory, and sepsis) Acute illness (including fever) Physical trauma Surgery or other procedures requiring general, regional, or local anesthesia Recent procedures requiring heparin/contrast Labour and delivery Dental procedures Severe stress and pain (including severe anxiety and harassment) Severe exercise <p>Specific to patients with glucocorticoid-induced adrenal insufficiency</p> <ul style="list-style-type: none"> Abrupt glucocorticoid withdrawal in relation to long-term treatment Glucocorticoid tapering below physiologic replacement doses Switch between different types, formulations, and doses of inhaled glucocorticoids, which can lead to considerable variability of glucocorticoid systemic absorption Initiation of strong cytochrome P450 3A4 inducers, which leads to increased first metabolism of several glucocorticoids. Strong inducers include rifampicin, carbamazepine, phenytoin, topiramate, lamotrigine, bosentan, fenofibrate, rifaximin, phenobarbital, phenylethynolone, and ritonavir.

私見)

従来よりステロイド剤を2週間処方している場合は急に中止しても良いとされていましたが、本院では1週間としています。
小児の場合は4日間が無難でしょうか。
今後もステロイド離脱症候群、急性副腎不全（副腎クライシス）に注意して参りましょう。
下記に関連文献を掲載します。

[本論文.pdf](#)

[Adrenal Crisis.pdf](#)

[急性副腎不全（副腎クライシス）.pdf](#)

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いいね!

ポスト

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