

Pump it up: IV Glucocorticoid Use in the ED

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Board Bombs

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Introduction

This guide serves a quick hit synopsis of IV glucocorticoid usage in the ED. Practitioners frequently abuse giving both PO and IV glucocorticoid as a nonspecific management option for many conditions. Glucocorticoids should be respected – and feared – for their potential to greatly disrupt human physiology. In general, practitioners overuse IV glucocorticoids even when oral dosing is shown to be equivocal. Here, we dispel a few myths and go through the top conditions that have evidence supporting clear benefit from IV glucocorticoid therapy in the ED.

Side effects of glucocorticoids

In the past, folks in the ED “ignored” the side effects of short-term glucocorticoid usage. We would say “I’m only giving one dose, or 5 days, or 10 days or therapy. Its less than one month, what could happen?” Possibly a lot. More and more data are coming out concerning glucocorticoid usage and their associated side effects. One population-based cohort study of >1.5 million Americans showed that within 30 days of a short-term prescription of oral glucocorticoid use, there were increase rates of sepsis, venous thromboembolism, and fracture within that 30-day period. Yes, there are plenty of confounding variables in this study, but it should raise awareness that glucocorticoids are not “angelic” therapies. Estimation regarding the frequency and severity of side effects and the highest risk dose associated with them are limited by small numbers of trials.

Glucocorticoids are associated a variety of cardiovascular effects, including fluid retention, hypertension, worsening atherosclerotic disease, certain arrhythmias, and even hyperlipidemia.

Glucocorticoids, not just NSAIDs, increase the risk of gastritis, ulcers, and upper-GI bleeding. NSAIDs and glucocorticoids together greatly increase the risk, a nearly 2-fold increased risk.

Glucocorticoids cause dose-dependent hyperglycemia, making glycemic control difficult for those with pre-existing diabetes mellitus.

Frequent usage of glucocorticoids

Infections

Let’s break the ice with the most controversial topic: glucocorticoids **in septic shock**

As of this writing (late 2020), glucocorticoid therapy should be a *case-by-case decision* in select patients with refractory shock (SBP <90 mmHg for more than one hour following both adequate fluid resuscitation *and* vasopressor administration). This does NOT include patients who are glucocorticoid dependent or when adrenal insufficiency is suspected.

-Many RCTs and meta-analyses have shown glucocorticoids lead to faster resolution of shock; however, *there appears to be minimal or no effect on mortality.*

-If using steroids, only use hydrocortisone. Hydrocortisone 1 mg/kg delivered as a bolus or as an infusion was more likely than placebo and methylprednisolone to result in shock reversal.

Meningitis: controversial. Dexamethasone has been studied as an adjuvant therapy to reduce the neurological complications in adults and children. It is controversial and there’s a lot of chit-chat on this subject, but we’ll spare you and tell you what you need to know.

- In general, in developed countries, the guidelines recommend giving 0.15 mg/kg dexamethasone before or at the same time as antimicrobials if there is strong suspicion for bacterial meningitis, but the organism is unknown. It should only be continued if the organism is found to be *S. pneumoniae*. There is no proven benefit of continuing glucocorticoids if the organism is not *S. pneumoniae*.

- Dexamethasone should *not* be given after antibiotics as its benefit is very limited and there might even be an *increase* in mortality.

-In developing countries, dexamethasone has a less clear role and no proven benefit.

- In children, steroids are *only* indicated in those with *H. influenzae B* meningitis. It can reduce the risk of hearing loss. Glucocorticoids should be given prior to, or with the first dose of antibiotics and continued for 4 days. Since *H. influenzae* is exceedingly rare, we only give steroids in unvaccinated children with suspected meningitis.

- There has been a long-held concern that glucocorticoids may reduce antibiotic penetration into CSF (especially Vancomycin). This is controversial and will NOT be on boards.

Rheumatologic/autoimmune diseases

Multiple sclerosis exacerbation: well-established. IV methylprednisone reduces disability at 28 days. Oral prednisone has been studied and, in some cases, accepted, but we know from experience IV is preferred.

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Temporal arteritis: well-established in preventing dreaded, irreversible vision loss. If visual symptoms or rapidly progressive disease symptoms are occurring, IV is absolutely indicated. If no visual symptoms are present, oral prednisone is perfectly acceptable.

Severe ITP: Glucocorticoid therapy is indicated when there is active bleeding and platelet counts of 10-20,000, or in those with no bleeding but platelet counts <10,000. Dexamethasone and prednisone raise the platelet count in approximately two-thirds of patients, with most patients responding within two to five days, thus decreasing the need for IVIG.

Thyrotoxicosis: IV glucocorticoids reduce T4-to-T3 conversion and possibly reduce the autoimmune process in Graves' disease. They should *not* be given in hyperthyroidism absent of storm. They also should be given *last*. Here is the order you should give therapies:

Propranolol → PTU/MMZ → Potassium-Iodine → Hydrocortisone

Inflammatory bowel disease (IBD): if an exacerbation is suspected, consultation with a gastroenterologist is preferred prior to dosing. There are lots of reasons why a patient with IBD presents with a "flare". Some causes are as simple as medication noncompliance and exacerbation of immunologic symptoms. Others are life-threatening such as toxic megacolon, abscesses, large fistulas, or small bowel obstruction. Obviously, steroids are not the solution in these cases. The clinician must exert utmost caution in having a large differential when these complicated patients present to the ED.

There is no data detailing the effectiveness of IV versus oral steroids in IBD. Methylprednisone is preferred and the patient can be transitioned to oral dosing once inpatient. Glucocorticoids are seen as short-term induction therapies or for exacerbations and should never be given long term.

Severe SLE flare: a "severe" flare is any patient with new renal insufficiency and significant proteinuria. This is suspected to be lupus nephritis which is the *most common cause of death in SLE*. Labs will show low C3, C4, and elevated acute phase reactants. IV methylprednisone is preferred. We suggest calling rheumatology first, as IV glucocorticoids may be very harmful if the diagnosis is not SLE, but scleroderma.

Respiratory illness

COPD/asthma exacerbations: IV glucocorticoids have been found to be equally efficacious as oral glucocorticoids. *However*, when a patient is in any sort of respiratory distress, we recommend IV as every effort should be made to optimize patient breathing. Don't forget to give inhaled corticosteroids as well!

Glucocorticoids in COPD/asthma have a bevy of evidence behind them: decreased hospital stay, improved lung function and symptoms, reduced treatment failure risk by 50%, and even reduced relapse risk at 1 month.

***Pneumocystis jiroveci* pneumonia:** glucocorticoids have been shown to decrease the mortality and risk of respiratory failure in those with severe *Pneumocystis* pneumonia. They should be given if:

- partial pressure of O₂ <70 mmHg on room air **and/or**
- A-a gradient >35 on arterial blood gas

Even easier, if there is clear evidence of persistent hypoxemia (<92%) on pulse oximetry, don't wait for the blood gas. Glucocorticoids can be given. Oral route is fine and works well, but given their respiratory distress, IV is preferable.

References: for a complete list of references please visit our website under this title's heading.