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Glucocorticoid Impact: Infections and Vaccinations FactFinder

Committed to providing helpful information to our members about key patient safety issues, the International Spine Intervention Society's Patient Safety Committee has developed a FactFinder series. FactFinders will explore and debunk myths surrounding patient safety issues. The intent of this FactFinder is to address issues surrounding use of glucocorticoids and their relationship to infections and the timing of vaccinations.

Myth: Glucocorticoid administration has minimal effect on immune response.

Fact: Systemic glucocorticoids have many effects upon innate and acquired immunity. These effects are dose dependent and increase the risk of infection with common bacterial, viral, and fungal pathogens.

Although there exist no specific data on immune system effects from epidural administration of glucocorticoids, data are available regarding systemic administration. With systemic high-dose glucocorticoid therapy (doses of 40mg or more of prednisone per day) there is an immediate risk of infection due to inhibition of phagocytic cell function, which abates after completion of therapy. In a study of patients with rheumatoid arthritis, the acute affects of 1 gram of intravenous methylprednisolone were evaluated.[1] Patients were given either one or three daily doses of methylprednisolone (1 gram per dose). Leukopenia developed within two hours of the dose, peaked at six hours, and resolved by 24 hours (in both regimens). Patients were followed for 16 weeks, during which PPD testing was unaffected, primary antibody responses to antigens were normal, and serum immunoglobulin levels were unchanged.

Doses of less than 40 mg per day of prednisone in adults can be considered "low to moderate". In this range, T lymphocytes may be slightly reduced in circulation (CD4-positive cells > CD8 positive cells). Delayed-type hypersensitivity responses may be impaired, resulting in cutaneous anergy. With long-term "low-dose" use, the effects on phagocytic cell function are minimal, but some inhibition of immune responses may increase with duration of therapy.

In addition to the direct affects on the immune system, patient-specific factors may have significant influence on the risk of infection. Older patients, hospitalized patients, and those with lowered functional status are at greater risk of infection. Patients with pre-existing conditions such as rheumatoid arthritis (RA) may also have a greater risk of infection related to glucocorticoid use. In one large study of RA patients, current and recent glucocorticoid doses were most strongly associated with such risk, but the data also suggested a cumulative risk effect from doses taken during the preceding 2-3 years.[2]

Myth: Recent administration of systemic glucocorticoid is a contraindication to live-virus vaccination.

Fact: Live-virus vaccination should be deferred for at least 1 month after discontinuation of high-dose systemically absorbed glucocorticoid therapy administered for >14 days.

There are insufficient data to define either dose or duration of therapy of systemically absorbed glucocorticoid needed to suppress the immune system in an otherwise immunocompetent person. "Short term" therapy usually indicates less than 14 days of treatment. "Low to moderate" dosing usually means less than 40mg of prednisone (or equivalent – see table below) per day. Glucocorticoid therapy is generally not considered a contraindication to administering live-virus vaccine when the steroid therapy is short-term or of low to moderate dose. Live-virus vaccination may also be performed when long-term therapy is administered in alternate-day treatment with short acting preparations. Replacement therapy (physiologic doses) is also not generally a contraindication to vaccination. Topical, inhaled, intra-articular, bursal, or tendon injections are also generally not viewed as contraindications to vaccination as long as low to moderate doses of glucocorticoid are administered. Live-virus vaccination should be deferred for at least 1 month after discontinuation of high-dose systemically absorbed glucocorticoid therapy administered for >14 days.

In most patients on glucocorticoid therapy for renal, pulmonary, or rheumatic diseases, pneumococcal vaccine is immunogenic, although antibody titers may be reduced. In one small study of asthmatics, 14 steroid-dependent patients had no difference in strength of response compared to 14 control asthmatics.[3] The doses of glucocorticoids taken by these patients ranged from 10 mg to 35 mg daily or every other day.

Similarly, patients receiving chronic glucocorticoid therapy for rheumatologic or pulmonary disorders generate an adequate antibody response to influenza vaccine, although some have lower antibody titer. [4,5] The significance of lower immunization-induced antibody titers with regard to infection prevention is unclear, and dose thresholds for glucocorticoid use with regard to vaccination success have not been established.

Please see the CDC reference below for further information about common vaccines.[8]

Relative Potency of Glucocorticoids[6]

	Approximate Equivalent Dose (mg)	Duration of Action (hours)	Relative anti-inflammatory activity	Relative mineralocorticoid activity
Cortisol	20	8-12	1	1
Cortisone acetate	25	8-12	0.8	0.8
Hydrocortisone	20	8-12	1	1
Prednisone	5	12-36	4	0.8
Prednisolone	5	12-36	4	0.8
Triamcinolone	4	12-36	5	0
Methyprednisolone	4	12-36	5	0.5
Dexamethasone	0.75	36-72	30	0
Fludrocortisone		12-36	10	125

It is important to re-state that the information above was garnered from the general medical literature, and that there are no data specific to glucocorticoids utilized in spinal procedures. Accordingly, no formal recommendations are provided. This information is provided to raise awareness amongst our membership such that they may have informed discussions with their patients about the risks and benefits or their procedures.

References:

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7. General recommendations on immunization. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR / January 28, 2011 / Vol. 60 / No. 2

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