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Steroid conversion chart pdf

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16.3KREBEL Review 46: Corticosteroid Conversion Chart Glucocorticoid, Approximate Equivalent Dose (mg), Relative Anti-Inflammatory (Glucocorticoid) Potency, Relative Mineralocorticoid (Salt Retaining) Potency, Biological Half-Life (Hours). Cortisone - Hydrocortisone - Methylprednisolone - Prednisolone - Prednisone - Dexamethasone
#corticosteroid #steroid #conversion #endocrinology #comparison #table #pharmacology #glucocorticoid Deflazacort is a synthetic corticosteroid, characterized by the insertion of a methyl-oxazoline ring in the chemical structure of prednisolone 21-acetate [3] (Fig. 1). Chemical structures of prednisolone and deflazacort [3] After oral administration, deflazacort is rapidly and completely absorbed in the intestinal tract (peak plasma concentration is reached within 1–2 hours). Subsequently, deflazacort is deacetylated at position 21 to form the main active metabolite, i.e. 21-desacetyl deflazacort [4], which is characterized by a high binding affinity to tissue glucocorticoid receptors [5]. An almost complete elimination of deflazacort metabolites is reached within 24 hours, mainly through the kidneys [4]. Glucocorticoids have immunosuppressive and anti-inflammatory properties, both primarily related to the antagonism of specific leukocyte functions and to the inhibition of the synthesis of pro-inflammatory cytokines.

Steroid Equivalency Chart

Steroid	Equivalent Dose (mg)	Glucocorticoid Potency	Mineralcorticoid Potency	Half-Life (hrs)
Prednisone	5	4	0.8	12-36
Methylprednisolone	4	5	0.5	12-26
Prednisolone	5	4	0.8	12-36
Hydrocortisone	25	1	1	12-36
Dexamethasone	0.75	25	0	8-12
Fludrocortisone	N/A	10	125	36-72

In vitro, deflazacort significantly inhibits both the proliferation of mononuclear cells derived from human peripheral blood, and the release of inflammatory cytokines by these cells.

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dexamethasone also able to inhibit chemotaxis, superoxide anion generation and chemiluminescence of human polymorphonuclear leukocytes in vitro [3]. Studies carried out using different experimental models indicate that dexamethasone is an effective inhibitor of the early exudative phase of inflammation, as well as of the development of chronic granulomatous inflammation. Furthermore, dexamethasone has been shown to inhibit experimentally-induced chronic inflammatory arthritic diseases (i.e. adjuvant-induced arthritis). Studies carried out to evaluate glucocorticoids ability to induce glycogen deposition in the liver of surrectomized rats have shown that dexamethasone is able to increase glycogenesis and hepatic glycogen synthesis, with a potency of action about 10 times higher than prednisolone administered at equivalent doses. The anti-inflammatory potency of dexamethasone is about 10–20 times higher than prednisolone and 40 times higher than cortisol (hydrocortisone). Furthermore, the duration of action of dexamethasone anti-inflammatory effects is longer than other glucocorticoids administered at equivalent doses [6]. Dexamethasone: therapeutic efficacy Dexamethasone has proven to be effective in the treatment of various rheumatological disorders including: sarcoidosis, a chronic systemic disease of unknown etiology, characterized by the formation of granulomas in various organs [7]; juvenile chronic arthritis, a condition characterized by alternating periods of remission and exacerbation, where dexamethasone use resulted in clinical improvements in a group of children with active disease [8]; polymyalgia rheumatica, which occurs predominantly in elderly patients with pain and stiffness in the shoulder girdle and pelvis, and responds very rapidly to low-dose corticosteroids [9]; nodular regenerative hyperplasia of the liver, a rare liver disease characterized by regenerative nodules separated by bands of fibrous connective tissue; symmetrical erosive arthropathy, a form of erosive arthropathy characterized by symmetrical joint involvement; and hypopycnic synovitis, often associated with systemic clinical involvement [10]. A clinical study, which evaluated dexamethasone ability to exert a protective effect on bone erosion and joint damage, typical of RA, has shown that dexamethasone is able to inhibit, in a dose-dependent manner, the invasiveness and the proliferation of synoviocytes (collected both from patients with RA and healthy volunteers) through differential modulation of single components of the fibrinolytic system [11]. Dexamethasone is indicated in steroid treatment of bronchial asthma and in the exacerbations of Chronic Obstructive Pulmonary Disease (COPD), to control inflammation and increased bronchial reactivity, which are the basis of bronchospasm. Furthermore, dexamethasone is useful in the treatment of children/adolescents with bronchial asthma. The efficacy and tolerability of oral dexamethasone in acute moderate asthma in children has been assessed in a prospective, randomized, parallel group trial of children aged 6 to 14 years with a diagnosis of asthma, who presented to the pediatric emergency department for moderate asthma exacerbation. Short-acting β_2 -adrenergic agonists were administered to all patients enrolled in the study; subsequently, they received either oral dexamethasone (1.5 mg/kg) or prednisolone (1 mg/kg) for seven days. The primary outcome measure was Forced Expiratory Volume in one second (FEV1) after 7 days. Secondary outcome measures included Peak Expiratory Flow Rate (PEFR) and pulmonary symptom score index. Patients were evaluated at the start of treatment (visit 1), on day 2 (visit 2) and on day 7 (visit 3). Two children out of the 54 enrolled were hospitalized on visit 2 (one from each group). At baseline, clinical data (mean FEV1, PEFR and symptom score) were similar in both groups; on visit 2, all measures improved: FEV1: +22.2 and +26.3% ($p < 0.05$); PEFR: +64 and +49 l/min ($p < 0.05$); symptom score: -4.4 and -3.8 ($p > 0.05$), respectively, without significant differences between the two groups. Further improvement was observed on visit 3. In treatment with dexamethasone (mean FEV1: +11.5 and +87.6 L/min ($p < 0.05$); symptom score: -5.4 and -5.9 ($p > 0.05$), respectively, without significant differences between the two groups (Table 2). Table 2 Results of pulmonary function tests and symptoms scores recorded at baseline (visit 1), and at 2 days (visit 2) and 7 days (visit 3) of treatment with dexamethasone or prednisolone. Side-effects of dexamethasone compared to prednisolone in children. In a retrospective analysis conducted in 11 adult and pediatric populations, have evaluated the use of dexamethasone in patients undergoing renal transplantation and in patients with nephrotic syndrome. A randomized, double-blind study compared the effects of treatment with dexamethasone versus prednisolone on Bone Mineral Density (BMD), on body composition and on lipid profile in 24 adult patients with end-stage renal disease, over a follow-up period of 78 months after kidney transplantation. The analysis of the 17 patients involved in the study (drop-out rates were similar in both groups) showed that lumbar BMD was significantly reduced in prednisolone than in dexamethasone group ($p < 0.05$); furthermore, the whole body BMD decreased from baseline in prednisolone ($p < 0.001$), but not in dexamethasone group. Lean body mass decreased by approximately 2.5 kg in both groups after 6–12 weeks ($p < 0.001$), then remained stable. Conversely, a significant increase in the fat mass ($p < 0.01$) and larger increases in total cholesterol ($p < 0.03$), in low-density lipoprotein cholesterol ($p < 0.01$), and in triglycerides ($p = 0.054$) were observed in the prednisolone group compared to the dexamethasone group. The study concluded that, in kidney-transplanted patients, the use of dexamethasone is associated with lesser loss of lumbar spine BMD and whole body BMD compared to prednisolone; dexamethasone also helps to prevent fat accumulation and lipid profile worsening [12]. Another randomized, double-blind study compared the effects of treatment with dexamethasone (0.30 ± 0.03 mg/kg per day) or methylprednisolone (0.20 ± 0.03 mg/kg per day) on growth, body composition, lipid profile, and bone mass in pre-pubertal patients ($n = 27$), during a 12-month follow-up period after kidney transplantation. The results of this study showed that the maintenance therapy with dexamethasone, in comparison with methylprednisolone maintenance therapy, is associated with an improved linear growth and also prevents excessive bone loss and fat accumulation in the population of pre-pubertal patients [13]. Nephrotic syndrome, characterized by hypoalbuminaemia, edema and proteinuria, is a common cause of renal failure in children. It is caused by glomerular injury, which may be idiopathic or secondary to systemic diseases. In children, it is most commonly idiopathic. Idiopathic nephrotic syndrome is characterized by massive proteinuria, hypoalbuminaemia, edema and hyperlipidaemia. The pathogenesis of idiopathic nephrotic syndrome is unclear, but it is thought to involve immune-mediated processes. There is evidence that genetic factors play a role in the pathogenesis of idiopathic nephrotic syndrome. Clinical trials have demonstrated the efficacy of dexamethasone treatment in both adult and pediatric patients with nephrotic syndrome. Two clinical trials found that dexamethasone was at least as effective as prednisolone in adult patients with idiopathic nephrotic syndrome (during a 3-month crossover study) as well as in patients with minimal change, membranoproliferative, focal segmental or membranous glomerulonephritis (during a 12-month double-blind parallel study) [3]. A clinical complication of nephrotic syndrome is hypogammaglobulinemia (due to increased catabolism and urinary loss of immunoglobulin) which is associated with a higher risk of infections, mainly caused by encapsulated bacteria. In this respect, some evidence indicates that IgG2-subclass antibodies are protective against this type of microorganisms, so that their loss in nephrotic syndrome is a risk factor for infection. A study on patients with nephrotic syndrome ($n = 11$; mean age 48 months, range 16–52 months) evaluated the efficacy of dexamethasone versus methylprednisolone in reducing the loss of the different immunoglobulin subclasses, during periods of both remission and relapse. In both treatment groups, a similar return to normal levels of total IgG and IgG1 was observed, but only those patients treated with dexamethasone showed a significant improvement in the IgG2 and IgG3 subclass values [14]. Dexamethasone: tolerability Dexamethasone has a smaller impact on calcium metabolism than any other synthetic corticosteroid, and therefore shows a lower risk of growth rate retardation in children [15, 16] and of osteoporosis in adult/elderly patients [2]. Another distinctive feature of dexamethasone is its little influence on carbohydrate metabolism than other glucocorticoids, as demonstrated in both studies conducted on animal models [4] and in clinical trials [17, 18]. Furthermore, the risk of sodium retention and hypokalemia, related to the mineralocorticoid activity of the drug, is greatly reduced with dexamethasone in comparison with older synthetic steroids. In fact, the physiological response to stress and the regulation of glucose and phosphate metabolism and electrolyte balance. Furthermore, given the small effect of dexamethasone on the HPA axis, a reduced risk of behavioral disorders compared to other corticosteroids has been hypothesized. Dexamethasone safety in children is a clinically relevant issue. In fact, when using corticosteroids in pediatric patients, and especially after long-term treatment, one of the most alarming adverse event is linear growth retardation; such event could be due to several mechanisms, the main of which seems to be the inhibition of Growth Hormone (GH) release from the pituitary gland. In children receiving long-term steroid treatment after kidney transplantation, the substitution of methylprednisolone (mean dose, 0.2 mg/kg per day) with dexamethasone (mean dose, 0.3 mg/kg per day), over an average period of 15 months, significantly increased GH secretion ($p < 0.05$) and mean growth velocity ($p < 0.01$) [13]. Dexamethasone: therapeutic indications and dosage Dexamethasone therapeutic indications include a number of pathological conditions, i.e.: primary and secondary adrenocortical failure; rheumatic diseases (including RA and ankylosing spondylitis); collagen diseases (such as systemic lupus erythematosus and polymyositis); dermatological diseases (including pemphigus, severe psoriasis and mycosis fungoides); allergic states (e.g., bronchial asthma and drugs hyper-reactivity); respiratory diseases (including sarcoidosis and aspiration pneumonia); ocular diseases (e.g., choriorretinitis and optic neuritis); hematological diseases (including Hodgkin's disease, non-Hodgkin's lymphoma, chronic lymphocytic leukemia and acute childhood leukemia); edematous state (nephrotic syndrome); gastro-intestinal diseases (ulcerative colitis, regional enteritis). The initial daily dosage of dexamethasone in adults may range from 6 to 90 mg, depending on the severity and evolution of the specific disease, and may be maintained unchanged, or be modified as appropriate, until a satisfactory clinical response is observed. It should be emphasized that corticosteroid requirements vary from patient to patient, so that dosages should be individualized taking into account the nature of the disease and the patient's characteristics. However, there are some general considerations regarding the use of corticosteroids. First, corticosteroids are usually used in the form of tablets. Second, corticosteroids are usually used in the form of tablets. Third, corticosteroids are usually used in the form of tablets. Fourth, corticosteroids are usually used in the form of tablets. Fifth, corticosteroids are usually used in the form of tablets. Sixth, corticosteroids are usually used in the form of tablets. 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