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Methylprednisolone Administration

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Commentary

Methylprednisolone is a synthetic glucocorticoid steroid that was created to have better anti-inflammatory and mineralocorticoid activity than cortisol, the prototype glucocorticoid steroid (hydrocortisone). As a glucocorticoid or anti-inflammatory medication, methylprednisolone is about four times as effective as hydrocortisone, with around 0.8 times the amount of mineralocorticoid. Methylprednisolone also has a longer action time than hydrocortisone, with a plasma half-life of 2.5 hours compared to 1.5 hours for hydrocortisone. In the current treatment of MS, methylprednisolone is particularly crucial in the acute phase of recurrence. It reduces the inflammatory cycle in a variety of ways, including dampening the inflammatory cytokine cascade, inhibiting T cell activation, decreasing immune cell extravasation into the central nervous system, facilitating apoptosis of activated immune cells, and indirectly decreasing the cytotoxic effects of nitric oxide and TNF-a. As more information about these systems becomes available, it may become possible to develop therapy regimens that are better tailored to the individual as well as the disease state. The only effective neuroprotective drug studied in controlled multicentre clinical trials is high-dose methylprednisolone, as suggested by the National Acute Spinal Cord Injury Studies (NASCIS-2 and NASCIS-3). Methylprednisolone was studied as a lipid peroxidation inhibitor, with the goal of reducing posttraumatic degenerative alterations in the damaged spinal cord. However, several researchers and physicians have questioned the usefulness of methylprednisolone for the treatment of acute traumatic SCI due to inconsistent experimental data and the relatively tiny neurological benefits shown in humans. The American Association of Neurological Surgeons' Section on Disorders of the Spine and Peripheral Nerves and the Congress of Neurological Surgeons produced guidelines for the treatment of SCI in the spring of 2002, with MP being the most contentious. Methylprednisolone is a pregnane steroid hormone that is synthesised from hydrocortisone and prednisolone. It is a type of synthetic glucocorticoid, and more broadly, a type of corticosteroid. It's a glucocorticoid and mineralocorticoid receptor agonist. Methylprednisolone has a stronger affinity for glucocorticoid receptors than mineralocorticoid receptors when compared to other exogenous glucocorticoids. Methylprednisolone is a glucocorticoid (GC) that has pleiotropic effects on a number of physiological processes. They have, however, been widely prescribed for their anti-inflammatory and immune-boosting properties. Synthetic glucocorticoids, such as methylprednisolone, have actions that are dependent on their interaction with intracellular glucocorticoid receptors (GRs) and, to a lesser extent, mineralocorticoid receptors (MRs). In contrast to MRs, which have a limited tissue distribution, GRs are broadly dispersed. The ligand-bound receptor translocates to the nucleus and modulates gene expression by this method. Oral and parenteral administration of methylprednisolone is permitted. Methylprednisolone (Medrol) is available in tablet form in strengths of 2 mg, 4 mg, 8 mg, 16 mg, and 32 mg for oral use.

Intramuscular injections of methylprednisolone acetate (Depo-Medrol) and methylprednisolone succinate (Solu-Medrol) are both approved. In addition to intralesional, intraarticular, and soft tissue injections, Depo Medrol is approved for intraleional, intraarticular and soft tissue injections.

Depomedrol is available as a sterile aqueous solution in concentrations of 20 mg/mL, 40 mg/mL, and 80 mg/mL. Hepatic metabolism and renal excretion of metabolites are the primary routes of elimination for methylprednisolone, with unaltered methylprednisolone excretion accounting for just 1.3-9.2% of total renal excretion. Methylprednisolone and methylprednisone can be interconverted. 11 beta-hydroxysteroid dehydrogenases (11[beta]-HSD) and 20 ketosteroid reductases are involved in hepatic metabolism. Hydrophilic inactive metabolites of methylprednisolone, such as 20-carboxymelthylprednisolone and 6[beta]-hydroxy-20[alpha] hydroxymethylprednisolone, are excreted by the kidneys.

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