HYDROCORTISON 10 MG TABLET

Composition

Hydrocortison 10 mg Tablet is a white, round, flat bevelled-edged, scored uncoated tablet approx. 7 mm diameter with code ORN35. Each tablet contains hydrocortisone 10 mg.

Excipients: Lactose, Gelatin, Magnesium Stearate, Starch, Talc, Purified Water.

Properties

Hydrocortisone or cortisol is a natural glucocorticoid with a mild mineralocorticoid action elaborated by the human adrenal cortex, normally 15–40 mg/day. Like other glucocorticoids, it acts by binding to steroid receptors in the cytoplasm. Its effects are to a great extent mediated through lipocortin synthesis.

Glucocorticoids act catabolically especially in the muscle tissue. They reduce the production of lymphokines and eicosanoids, decrease the quantity of lymphatic tissue, and have an immunosuppressive and anti- inflammatory effect irrespective of the cause of inflammation. They also reduce fibroblast activity and scar formation. Glucocorticoids reduce the secretion of corticotrophin and depress the pituitary-adrenal axis.

Hydrocortisone has a slight mineralocorticoid effect. After a single dose of 250 mg of hydrocortisone, the secretion of corticotrophin is inhibited for about 24–36 hours.

Hydrocortisone may be used with mineralocorticoids as specific treatment for adrenocortical insufficiency. If the adrenocortical insufficiency is of pituitary or hypothalamic origin, hydrocortisone alone should be sufficient. It may be used also for unspecific anti- inflammatory and anti-allergic treatment.

Hydrocortisone is absorbed rapidly and completely from the alimentary tract. Because of first-pass metabolism, its bioavailability varies between 25 % and 90 %. In plasma, the peak concentration of hydrocortisone is reached in 1–2 hours after dosing. It is bound to plasma transcortin and albumin. At low concentrations, 10% of hydrocortisone is free; at higher concentrations, the binding capacity of transcortin is saturated and the proportion of free drug can increase to 40–50%. Its volume of distribution is 0.4–0.7 l/kg. The pharmacologic half- life of hydrocortisone averages 1.5 hours, but the half-life of the biological effect is considerably longer, approx. 10 hours. Hydrocortisone penetrates the placenta and is excreted in small amounts in breast milk. The elimination of hydrocortisone may be prolonged in liver diseases and shortened in thyrotoxicosis.

Indications

Adrenocortical insufficiency (Addison's disease), diminution of anterior pituitary function (hypopituitarism), congenital adrenal hyperplasia.
Conditions where systemic glucocorticoid therapy is indicated.

Dosage and Administration

Adults:
For substitution therapy
20–30 mg/day, of which generally approx. 2/3 in the morning and 1/3 early in the evening. If necessary, the morning dose can be taken in two portions. For other indications, 40–200 mg/day, in short-term use for special indications even higher doses.

Children:
Dosage in children is individualised. In adrenocortical insufficiency, 7.5–15 mg/ m^2/day divided into three equal doses (immediately in the morning, in the afternoon, and late in the evening). The morning dose can also be higher than the other doses. In congenital adrenal hyperplasia, generally 10 mg/m^2/day divided into three doses. In hypopituitarism, 2.5 mg three times daily.

For dosage in stress situations, See Special Warning/Precautions.

Contraindications

Tuberculosis and other acute or chronic bacterial and viral infections without antibiotic or chemotherapeutic protection, hypersensitivity to any ingredient of the preparation.

Side Effects

No adverse reactions are anticipated from substitution therapy with physiologic doses.

Short-term hydrocortisone therapy even at high doses is generally harmless.

In long-term high-dose therapy, adverse reactions occur frequently. Hydrocortisone has the same adverse reactions as other glucocorticoids. It also has a mineralocorticoid effect.

Hydrocortisone causes adrenocortical insufficiency in long-term high-dose therapy. Therefore, stress, e.g. surgery or infection, can cause hypotension, hypoglycemia, or even death, unless the steroid dose is increased to adjust to the stress.

Glucocorticoid withdrawal syndrome will follow abrupt withdrawal of long-term steroid therapy.

Symptoms include, e.g. fever, myalgia and arthralgia, weakness, nausea, elevated intracranial pressure, and hypotension. High-dose hydrocortisone therapy causes symptoms of Cushing syndrome: moon face, lipid accumulation in the neck, tendency to bruises, ecchymoses, and striata.

Glucocorticoids increase susceptibility to infection, and they may mask symptoms of infection. They

prolong tissue healing and scar formation.
Osteoporosis and diabetes occur in as many as 50% of patients on glucocorticosteroid therapy. In addition, cataract, elevated intraocular pressure, aseptic bone necrosis, nephroliths, muscle weakness, skin atrophy, tendency to thromboses, hypokalemia, hyperlipidemia, and increased appetite occur. The risk of gastric ulcer increases slightly in corticosteroid therapy.

High-dose glucocorticoid therapy can cause psychiatric adverse reactions, e.g. depression, psychosis, and insomnia. It retards growth in children, impairs semen quality, and can cause amenorrhea.

Glucocorticoids have been reported to have caused allergy, anaphylactic reactions and blurred vision. Hydrocortisone can aggravate cardiac insufficiency and hypertension.

Warnings/Precautions

In patients receiving hydrocortisone substitution therapy, the dosage of hydrocortisone in stress situations, e.g. trauma, infections, or surgery, must be increased 2–4 fold and, if necessary, the patient must be transferred to parenteral therapy.

Long-term systemic glucocorticoid therapy causes adrenocortical insufficiency which may last for months after the treatment has been stopped. Therefore, in stress situations, e.g. infections, the dosage of Hydrocortisone must be increased.

To avoid the withdrawal syndrome of glucocorticoid therapy, long-term corticosteroid therapy must be withdrawn gradually over a period of several weeks. Dosing on alternate days reduces the risk of adrenocortical insufficiency and the withdrawal syndrome associated with stopping the treatment. Caution should be exercised when administering pharmaceutical doses of hydrocortisone to patients with acute psychosis, peptic ulcer, diabetes, osteoporosis, glaucoma or hypertension.

Glucocorticoids may reduce the potency of vaccines and increase the risk of neurologic complications of vaccination. Live virus vaccines can cause an infection in patients receiving corticosteroids.

Visual disturbance may be reported with systemic and topical corticosteroid use. If a patient presents with symptoms such as blurred vision or other visual disturbances, the patient should be considered for referral to an ophthalmologist for evaluation of possible causes which may include cataract, glaucoma or rare diseases such as central serous chorioretinopathy (CSCR) which have been reported after use of systemic and topical corticosteroids.

Pheochromocytoma crisis, which can be fatal, has been reported after administration of systemic corticosteroids. Corticosteroids should only be administered to patients with suspected or identified pheochromocytoma after an appropriate risk/benefit evaluation.

Use in Pregnancy and Lactation:

Hydrocortisone penetrates the placenta. There is however no reason to abstain from clearly indicated therapy. A newborn of a mother who has received pharmacologic doses of hydrocortisone during pregnancy should be monitored for potential hypoadrenalism.

Hydrocortisone is excreted in breast milk. Breast-feeding is possible during pharmacologic low-dose therapy.
When doses exceed 100 mg/day, nursing should be avoided for a few hours after drug administration.

Interactions with other medications

Phenytoin, phenobarbital, carbamazepine, rifampicin, and antithyroid drugs increase the clearance of hydrocortisone and reduce its half-life. Hydrocortisone may weaken the effect of anticoagulants and antidiabetics. In combination with anticholinesterases, corticosteroids can cause muscle weakness in patients with myasthenia gravis. Systemic glucocorticoid therapy increases the risk of hypokalemia in patients receiving diuretics or amphotericin B. Glucocorticoids may increase the risk of anti-inflammatory analgesic induced peptic ulcer.

Overdosage and Treatment

Acute massive overdose of hydrocortisone is not likely. Remarkably high doses are tolerated without serious adverse reactions. Treatment of oral overdose is supportive; if necessary, medicinal carbon may be given and gastric lavage performed.

Presentations

Bottles 30's, 100's. Store at or below 25°C.

Store in the original package. Protect from light.

Keep out of the reach of children. See the package for the expiry date. Do not use the medicine after the stated date.

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