

CORTINOX INY

TRIAMCINOLONE ACETONIDE
CORTICOSTEROID

PHARMACEUTICAL FORM:
INJECTABLE SUSPENSION


COMPOSITION:

Each 100 ml contains:
Triamcinolone acetonide 0.6 g
Excipients q.s 100 ml

SPECIES:

•Equine

DOSAGE:

DOSAGE				
SPECIES	REFERENCE DOSE	PRACTICAL DOSE	INDICATIONS	ADMINISTRATION ROUTE
 EQUINES	0,022-0,044 mg/Kg	1,5-3 mL /400 kg	Single Dose	Intramuscular Subcutaneous
	6-18 mg/Kg	1-3 mL	Per joint Repeat after 4-13 days if necessary.	Intra-articular

INDICATIONS AND USE:

CORTINOX INY is a depot glucocorticoid indicated for the treatment of inflammatory conditions in horses.

ROUTE OF ADMINISTRATION:

•Intramuscular, subcutaneous, intra-articular

PHARMACOKINETICS:

Endogenously produce corticosteroids and administered glucocorticoids are largely bound to plasma proteins (up to 75%), mainly to a globulin called transcortin, another 10-15% binds to albumin, and the remainder exists in a free form. The binding is weak and reversible, and the more polar the compound, it will be more weakly bound, as is the case with cortisol.

MECHANISM OF ACTION:

Glucocorticoids have effects on almost all cells and systems. Cardiovascular system: Glucocorticoids can reduce capillary permeability and promote vasoconstriction. The increase in blood pressure may result both from the vasoconstrictive properties of the drugs and from an increase in blood volume that can occur. Cells: Glucocorticoids inhibit the proliferation of fibroblasts, the macrophage response to growth factors, migration, lymphocyte sensitization, and the cellular response to inflammatory mediators. They also stabilize lysosomal membranes.

Nervous system / autonomic nervous system: Glucocorticoids can lower the seizure threshold, alter mood and behavior, reduce the response to pyrogens, stimulate appetite, and help maintain the alpha rhythm. Glucocorticoids are necessary for the normal sensitivity of adrenergic receptors.

Endocrine system: When animals are not under stress, glucocorticoids suppress the release of ACTH from the anterior pituitary,

thereby reducing or preventing the release of endogenous corticosteroids. Stress factors (e.g renal, hepatic disease, diabetes) may sometimes override the suppressive effects of exogenous steroids. When glucocorticoids are administered at pharmacological doses, a reduction in the release of thyroid-stimulating hormone (TSH), follicle-stimulating hormone (FSH), prolactin, and luteinizing hormone (LH) may occur.

The conversion of thyroxine (T4) to triiodothyronine (T3) may be reduced by glucocorticoids.

Hematopoietic system: Glucocorticoids can increase the number of circulating platelets, neutrophils, and erythrocytes, but they inhibit platelet aggregation. A decrease in the number of peripheral lymphocytes, monocytes, and eosinophils is observed due to their sequestration in the lungs and spleen and to a rapid reduction in the release of these cells from the bone marrow.

Removal of senescent erythrocytes is reduced. Glucocorticoids can cause involution of lymphoid tissue.

Gastrointestinal tract and liver: Glucocorticoids increase the secretion gastric acid, pepsin, and trypsin. They alter the structure of mucin and reduce the proliferation of mucosal cells.

Hepatic changes may include increased lipid and glycogen deposition within hepatocytes, as well as increased serum levels of alanine aminotransferase (ALT) and gamma-glutamyl transpeptidase (GGT). Immune system: Glucocorticoids can decrease circulating levels of T lymphocytes, inhibit lymphokines, and inhibit the migration of neutrophils, macrophages, and monocytes.

Metabolic effects: Lipogenesis is increased in certain areas of the body (e.g, the abdomen) and adipose tissue may be redistributed, moving away from the extremities toward the trunk.

Musculoskeletal effects: Glucocorticoids can cause muscle weakness, muscle atrophy, and osteoporosis.

Ophthalmic effects: Prolonged use (both systemic and topical ophthalmic) may cause increased intraocular pressure and glaucoma, as well as cataracts and exophthalmos.

Kidneys and hydroelectrolytic balance: Glucocorticoids may increase the excretion of potassium and calcium. Diuresis may develop following glucocorticoid administration.

Skin: Treatment with corticosteroids may be associated with thinning of the dermal tissue and skin atrophy. Hair follicles may become distended, and alopecia may occur.

PRECAUTION, WARNINGS AND CONTRAINDICATIONS:

It should not be administered to animals with systemic fungal infections.

Do not use in musculoskeletal disorders where immobilization is required.

Do not use in viral or bacterial infectious processes.

Do not use in animals with chronic nephritis or Cushing's syndrome, except for emergency therapy.

Do not administer during the last third of gestation.

Use with caution in patients with diabetes mellitus.

Do not administer in cases of demodectic mange, gastric or duodenal ulcers, and ulcerative colitis.

Glucocorticoids in general should not be administered in cases of:

- Amyloidosis
- Chronic erosive arthritis
- Acute pancreatitis
- Use with caution in cases of chronic bacterial infections

Do not administer intravenously. The glucocorticoid dose should be gradually tapered in animals to allow endogenous ACTH secretion and corticosteroid function to slowly return to normal.

Corticosteroids may precipitate parturition.

Keep out of reach of children and domestic animals.

DRUG INTERACTIONS:

-Use with caution in combination with NSAIDs, as gastrointestinal toxicity may be potentiated.

-High doses in combination with CNS depressants may produce profound sedation.

SIDE EFFECTS:

Side effects from corticosteroids are many and include polyphagia, polydipsia and polyuria, and hypothalamic-pituitary-adrenal axis suppression. Injections of triamcinolone acetonide can have long-lasting effects.

WITHDRAWAL:

None .

ANTIDOTE:

Supportive and symptomatic treatment.

STORAGE:

Store in a cool and dry place, protected from light at a temperature between 15°C to 30°C. **Keep out of the reach of children and pets.**

SALE:

Veterinary prescription only.

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