by 4 mg every 4 hours on days 5 to 8 of treatment.
The reafter, the dose is gradually reduced by 4 mg daily. A maintenance dose of 2 mg given by intravenous or intramuscular injection 2 or 3 times daily has been used in patients with recurrent or inoperable neoplasms.
Dexamethasone is given intravenously for the prevention of nausea and vomiting induced by cancer chemotherapy.
The sodium phosphate ester is given by intra-articular,intralesional, or soft-tissue injection. For intra-articular injection doses equivalent to 0.8 to 4 mg of dexamethasone phosphate are used depending upon the size of The joint. For soft-tissue injection doses of 2 to 6 mg are used. Injections are repeated every 3 to 5 days to every 2 to 3 weeks.
A biodegradable intravitreal implant containing dexamethasone is used for the treatment of macular oedema after branch or central retinal vein occlusion.
Other esters of dexamethasone that have occasionally been used include the hemisuccinate, linoleate, palmitate,pivalate, propionate, sodium succinate, tebutate, andvalerate.
The phenpropionate and troxundate esters have been used in veterinary medicine.
Dexamethasone-releasing stents may be used to reduce restenosis after coronary artery stent placement.

PRECAUTIONS:

Increased susceptibility to and severity of infection; activation or exacerbation of tuberculosis, amoebiasis, strong yloidiasis; risk of severe chickenpox in non-immune patient(varicella-zoster immunoglobulin required if exposed to chickenpox); avoid exposure to measles (normal immunoglobulin possiblyrequired if exposed); diabetes mellitus;peptic ulcer; hypertension; precautions relating to long-term use of corticosteroids;glaucoma, epilepsy; drug should not be abruptly withdrawn; interactions

INTERACTIONS:

The interactions of corticosteroids in general are described on. Various drugs may interfere with the dexamethasone suppression test.Antiepileptics. As mentioned on, dexamethasone may decrease or increase plasma concentrations of phenytoin.Like other enzyme-inducing drugs, phenytoin also
has the potential to increase the metabolism of dexamethasone.
There have been reports of false positive dexamethasone suppression tests (see Diagnosis and Testing, in patients taking carbamazepine.

ADVERSE EFFECTS:

Nausea, dyspepsia, malaise, hiccups;hypersensitivity reactions including anaphylaxis; perineal irritation after intravenous administration; adverse effects associated with long-term corticosteroid treatment; hyperglycaemia, abdominal distension, angioedema, bradycardia,acne, erythema, Cushing's syndrome, oropharangeal candidiasis, hypothalamicpituitary adrenal axis suppression.

Dexamethasone has little or no effect on sodium and water retention.When applied topically, particularly to large areas, when the skin is broken, or under occlusive dressings, or when given intranasally, corticosteroids may be absorbed insufficient amounts to cause systemic effects. Prolonged use of ophthalmic preparations containing corticosteroids has caused raised intra-ocular pressure and reduced visual function.

EFFECTS ON THE NEONATE:

Adverse effects noted in premature neonates with bronchopulmonary dysplasia receiving dexamethasone treatment to enable weaning from assistedventilation have included hypertension often accompanied by bradycardia, gastroduodenal perforation,ulceration and thinning of the gastric wall, development of a catabolic state, renal calcification, and transientmyocardial hypertrophy. There is some evidence of a suppressive effect on motor activity and spontaneous movement. It has been postulated that neonatal dexamethasone may both increase and decrease retinopathy of prematurity; its true effect is uncertain. There is also a concern that longer term development of the child may be adversely affected Although data are scanty, a meta-analysis has concluded that postnatal use ofcorticosteroids to treat or prevent bronchopulmonarydysplasia is associated with dramatic increases in the incidence of cerebral palsy and neurodevelopment alimpairment, and suggested that such use should be abandoned.Pulsed dosage may reduce the adverse effects but may also reduce efficacy.

OVERDOSE:

Reports of acute toxicity and/or death following overdosage of glucocorticoids are rare. In the event of overdosage, no specific antidote is available; treatment is supportive and symptomatic.
The oral LD50 of dexamethasone in female mice was 6.5 g/kg. The intravenous LD50 of dexamethasone sodium phosphate in female mice was 794 mg/kg

USES IN PREGNANCY:

Since adequate human reproduction studies have not been done with corticosteroids, Use of these drugs in pregnancy or in women of child bearing potential requires that the anticipated benefits be weighed against the possible hazards to the mother and embryo or fetus. Infants born of mothers who have received substantial doses of corticosteroids during pregnancy should be carefully observed for signs of hypoadrenalism.
Corticosteroids appear in breast milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other unwanted effects. Mothers taking pharmacologic doses of corticosteroids should be advised not to use.

Storage: - Store protected from light at a temperature not exceeding 30°C.
Improper Storage may Deteriorate the product
Keep the medicine out of reach of children.
Presentation: -Each pack contains 2 ml Vial of Dexamethasone Sodium Phosphate Injection IP

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For the use only of a registered medical practitioner or a hospital or a laboratory

Dexamethasone Sodium Phosphate Injection IP

AMERICAN
Dexacan
Injection

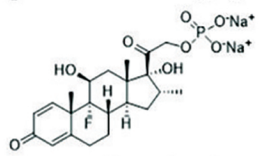
FOR IM/IV USE ONLY

Composition: -
Each ml Contains: -

Dexamethasone Sodium Phosphate	IP	
Eq. to Dexamethasone Phosphate		4 mg.
Water for Injections	IP	q.s.

DESCRIPTION:

Dexamethasone sodium phosphate injection IP is a sterile solution of dexamethasone sodium phosphate, and is supplied in 4 mg/ ml, this is a sterile solution for intravenous, intramuscular, intra-articular, intralesional and soft tissue administration.
Dexamethasone sodium phosphate, a synthetic adrenocortical steroid, is a white or slightly yellow, crystalline powder. It is freely soluble in water and is exceedingly hygroscopic. The molecular weight is 516.41. It is designated chemically as 9-fluoro-11β,17-dihydroxy-16α-methyl-21-(phosphonooxy) pregna-1, 4-diene-3,20-dione disodium salt. The empirical formula is $C_{22}H_{28}FNa_2O_8P$ and the structural formula is:



Dexamethasone sodium phosphate

PHARMACOLOGY:

CLINICAL PHARMACOLOGY:

Dexamethasone sodium phosphate injection has a rapid onset but short duration of action when compared with less soluble preparations. Because of this, It is suitable for the treatment of acute disorders responsive to adrenocortical steroid therapy.
Naturally occurring glucocorticoids (hydrocortisone and cortisone), which also have salt-retaining properties, are used as replacement therapy in adrenocortical deficiency states. Their synthetic analogs, including dexamethasone, are primarily used for their potent anti-inflammatory effects in disorders of many organ systems.
Glucocorticoids cause profound and varied metabolic effects. In addition, they modify the body's immune responses to diverse stimuli.
At equipotent anti-inflammatory doses, dexamethasone almost completely lacks the sodium-retaining property of hydrocortisone and closely related derivatives of hydrocortisone.

PHARMACOKINETICS:

Dexamethasone is readily absorbed from the gastrointestinal tract. Its biological half-life in plasma is about 190 minutes. Binding of dexamethasone to plasma proteins is about 77%, which is less than for most other corticosteroids.Up to 65 % of a dose is excreted in urine within 24 hours.
Clearance in premature neonates is reported to be proportional to gestational age, with a reduced eliminationrate in the most premature. It readily crosses the placenta with minimal inactivation.

USES AND ADMINISTRATION:

Dexamethasone is a corticosteroid with mainly glucocorticoid activity; 750 micrograms of dexamethasone is equivalent in anti-inflammatory activity to about 5 mg of prednisolone.
It has been used, either in the form of the free alcohol or in one of the esterified forms, in the treatment of conditions for which corticosteroid therapy is indicated, except adrenocortical insufficiency for which hydrocortisone with supplementary fludrocortisone is preferred. Its lack of mineral ocorticoid properties makes dexamethasone particularly suitable for treating conditions where water retention would be a disadvantage.
However, esterification generally alters potency and compounds with equivalent dexamethasone content may not have equivalent clinical effect. Dexamethasone sodium phosphate 1.1 mg is equivalent to about 1 mg of dexamethasone phosphate.
For parenteral administration in intensive therapy or in emergencies, the sodium phosphate ester may be given **intravenously by injection or infusion or intramuscularly by injection**; doses are sometimes expressed in terms of the free alcohol, the phosphate, or the sodium phosphate and
confusion has sometimes arisen in the literature because of these variations. Initial doses used, expressed in terms of dexamethasone phosphate, range from about 0.5 to 24 mg daily (about 0.4 to 20 mg of dexamethasone).

Intravenous

doses equivalent to 2 to 6 mg/kg of dexamethasone phosphate given slowly over a minimum period of several minutes have been suggested for the treatment of severe shock. These high doses may be repeated within 2 to 6 hours and this treatment should be continued only until the patient's condition is stable and usually for no longer than 48 to 72 hours. Alternatively, the initial intravenous injection may be followed by. a continuous intravenous infusion of 3 mg/kg per 24 hours.
Dexamethasone sodium phosphate is also used in the treatment of cerebral oedema caused by malignancy. Aninitial intravenous dose equivalent to 10 mg of dexamethasone phosphate is followed by 4 mg intramuscularly every 6 hours until the symptoms of oedema subside. Aresponse is usually obtained after l2 to 24 hours and dosage may be reduced after 2 to 4 days, then gradually stopped over 5 to 7 days. A much higher dosage schedule has also been suggested for use in acute life-threatening cerebraloedema. An initial intravenous dose equivalent to 50 mg of dexamethasone phosphate is followed by 8 mg intravenously every 2 hours for the first 3 days of treatment. The dose is then reduced to 4 mg every 2 hours on day 4,followed