Dexamethasone

Decilone® 500 mcg Tablet
Decilone® Forte 4 mg Tablet
Corticosteroid

FORMULATION

Each Decilone® tablet contains:
Dexamethasone ..... 500 mcg

Each Decilone® Forte tablet contains:
Dexamethasone ..... 4 mg

PRODUCT DESCRIPTIONS

Dexamethasone (Decilone® 500 mcg) is a round, two-layered tablet which is cut across the pink side and plain on the yellow side.

Dexamethasone (Decilone® Forte 4 mg) is a round and white tablet.

CLINICAL PHARMACOLOGY

Pharmacodynamics

Dexamethasone is a highly potent and long-acting glucocorticoid whose main pharmacological actions include: (a) general effects on metabolism, water and electrolyte imbalance; (b) negative feedback effect on the hypothalamus and pituitary glands; (c) anti-inflammatory and immunosuppressive effects.

It has negligible sodium-retaining properties and has powerful anti-inflammatory and immunosuppressive effects.

Pharmacokinetics

Dexamethasone is well absorbed orally. Peak plasma levels are reached between 1 to 2 hours after ingestion. The area under the curve (AUC), volume of distribution, plasma clearance, and maximum plasma concentration are not dose-dependent but vary by a factor of about 0.6-0.7 after oral doses of 0.5-1.5 mg. The systemic bioavailability of dexamethasone is approximately 90% and the mean plasma half-life is 3.6 ± 0.9 hours.

Up to 77% of dexamethasone is bound to plasma proteins. Percentage binding of dexamethasone, unlike that of cortisol, remains practically unchanged with increasing steroid concentrations.

Dexamethasone's metabolism in the liver is slow and limited and 60% of the administered dose is excreted in the urine within 24 hours largely as unconjugated steroids.

Dexamethasone's plasma half-life is shortened in chronic renal failure. Metabolic clearance is reduced and plasma half-life is prolonged in patients with liver disease. Dexamethasone is cleared more rapidly from the circulation of the fetus and neonate than in the mother.

INDICATIONS

Cerebral Edema
Cerebral edema associated with primary or metastatic brain tumor, craniotomy or head injury. Use in cerebral edema is not a substitute for careful neurosurgical evaluation and definitive management such as neurosurgery or other specific therapy.
Endocrine Disorders
• Congenital adrenal hyperplasia • Nonsuppurative thyroiditis • Hypercalcemia associated with cancer • Primary or secondary adrenocortical insufficiency (hydrocortisone or cortisone is the first choice; synthetic analogs like dexamethasone may be used in conjunction with mineralocorticoids where applicable; in infancy, mineralocorticoid supplementation is of particular importance)
Allergic States
Control of severe incapacitating allergic conditions intractable to adequate trials of conventional treatment in adults and children with: • Bronchial asthma • Seasonal or perennial allergic rhinitis • Contact dermatitis • Atopic dermatitis • Serum sickness • Drug hypersensitivity reactions
Rheumatic Disorders
As adjunctive therapy for short-term administration (to tide the patient over an acute episode or exacerbation) in: • Post-traumatic osteoarthritis • Synovitis of osteoarthritis • Rheumatoid arthritis, including juvenile rheumatoid arthritis (selected cases may require low-dose maintenance therapy) • Ankylosing spondylitis • Acute and subacute bursitis • Acute nonspecific tenosynovitis • Acute gouty arthritis • Acute rheumatic carditis • Psoriatic arthritis • Epicondylitis • Dermatomyositis, polymyositis
Collagen Diseases
During an exacerbation or as maintenance therapy in selected cases of: • Systemic lupus erythematosus • Acute rheumatic carditis • Arteritis
Dermatologic Diseases
• Pemphigus • Bullous dermatitis herpetiformis • Severe erythema multiforme (Stevens-Johnson syndrome) • Exfoliative dermatitis • Mycosis fungoides • Severe seborrheic dermatitis • Severe psoriasis
Ophthalmic Diseases
Severe acute and chronic allergic and inflammatory processes involving the eye and its adnexa, such as: • Herpes zoster ophthalmicus • Iritis, iridocyclitis • Chorioretinitis • Diffuse posterior uveitis and choroiditis • Optic neuritis • Sympathetic ophthalmia • Anterior segment inflammation • Allergic conjunctivitis • Keratitis • Allergic corneal marginal ulcers • Retinopathy
Respiratory Diseases
• Symptomatic sarcoidosis • Berylliosis • Fulminating or disseminated pulmonary tuberculosis when used concurrently with appropriate antituberculous chemotherapy • Idiopathic eosinophilic pneumonias • Loeffler's syndrome not manageable by other means • Aspiration pneumonitis • Prevention of neonatal respiratory distress syndrome
Hematologic Diseases
• Idiopathic thrombocytopenic purpura in adults • Pure red cell aplasia • Secondary thrombocytopenia in adults • Acquired (autoimmune) hemolytic anemia • Congenital (erythroid) hypoplastic anemia (Diamond-Blackfan anemia)
Neoplastic Diseases
• For palliative management of leukemias and lymphomas in adults • Acute leukemia in children • Chemotherapy - Induced Nausea and Vomiting
Edematous States
To induce diuresis or remission of proteinuria in nephrotic syndrome, without uremia, of the idiopathic type, or that due to lupus erythematosus
Gastrointestinal Diseases
To tide the patient over a critical period of the disease in: • Ulcerative colitis • Regional enteritis
Other Uses
• Tuberculous meningitis with subarachnoid block or impending block when used concurrently with appropriate antituberculous chemotherapy • Acute exacerbations of multiple sclerosis • Trichinosis with neurologic or myocardial involvement • Diagnostic testing of adrenocortical hyperfunction

DOSAGE AND ADMINISTRATION

INDIVIDUALIZE DEXAMETHASONE DOSE BASED ON THE CONDITION BEING TREATED AND THE RESPONSE OF THE PATIENT.

Initial Oral Adult Dose: Varies from ***0.75 mg to 9 mg a day*** depending on the type and severity of disease being treated

- In less severe cases, doses below 0.5 mg (500 mcg) may suffice
- In severe diseases, doses higher than 10 mg may be required.

Initial Oral Pediatric Dose: Varies from **0.02 to 0.3 mg/kg body weight per day (0.6 to 9 mg/m² body surface area/day)** depending on the type and severity of the disease being treated given in three or four divided doses.

Maintain and/or adjust initial dosage until a satisfactory response is noted. Discontinue dexamethasone and shift the patient to other appropriate therapy if clinical response is unsatisfactory.

Signs that may require dosage adjustment include changes in clinical status resulting from remissions or exacerbations of the disease, individual drug responsiveness, and stress which may be attributed to surgery, infection, or trauma. In times of stress (e.g., surgery, infection, or trauma) temporarily increasing corticosteroid dose in patients with adrenal insufficiency may be necessary.

Upon obtaining a satisfactory response, decrease dose in small increments until the lowest level that maintains an adequate clinical response is reached.

Gradually withdraw dexamethasone when stopping treatment after long-term therapy (more than 3 weeks) since significant adrenal suppression is expected to occur in patients receiving corticosteroids for more than 3 weeks.

Treatment of Acute Exacerbations of Multiple Sclerosis:
Usual Recommended Oral Dexamethasone Dose:
- 30 mg daily for a week, then 4 to 12 mg every other day for 1 month.

Treatment of Acute, Self-Limited Allergic Disorders or Acute Exacerbations of Chronic Disorders:

Day 1:	4-8 mg dexamethasone sodium phosphate injection given IM
Days 2 & 3:	3 mg oral dexamethasone in two divided doses
Day 4:	1.5 mg oral dexamethasone in two divided doses
Days 5 & 6:	0.75 mg (750 mcg) oral dexamethasone once daily

Then the drug is discontinued.

Treatment of Cerebral Edema:
- Dexamethasone sodium phosphate injection is generally administered initially until symptoms of cerebral edema subside.
- When possible, **oral dexamethasone 1-3 mg three times daily** should replace parenteral dexamethasone.

Treatment of Chemotherapy-Induced Nausea and Vomiting:

Treatment of Chemotherapy-Induced Nausea and Vomiting		
Chemotherapy Regimen with High *Emetic Risk (>90%)	**Chemotherapy Regimen with Moderate *Emetic Risk (30 – 90 %)**	**Chemotherapy Regimen with Low *Emetic Risk (10 – 30 %)**
Includes IV antineoplastic agents such as: cisplatin, cyclophosphamide ≥ 1,500 mg/m², carmustine, dacarbazine, etc.	*Includes IV antineoplastic agents such as: oxaliplatin, cytarabine >1 g/m², carboplatin, ifosfamide, cyclophosphamide < 1,500 mg/m², doxorubicin, daunorubicin, epirubicin, idarubicin, irinotecan, etc*	*Includes IV antineoplastic agents such as: paclitaxel, docetaxel, mitoxantrone, topotecan, etoposide, pemetrexed, methotrexate, doxorubicin HCl liposome injection, mitomycin, gemcitabine, 5-fluorouracil, cytarabine ≤ 100 mg/m², bortezomib, cetuximab, trastuzumab, etc.*
Recommended Antiemetic Regimen:	**Recommended Antiemetic Regimen:**	**Recommended Antiemetic Regimen:**
Dexamethasone 12 mg single oral dose before chemotherapy on Day 1, then 8 mg orally once daily on Days 2 to 4. **Note: The regimen also includes a 5-HT_3 receptor antagonist and aprepitant	**Without aprepitant:** Dexamethasone 8 mg single IV dose before chemotherapy on Day 1, then 8 mg orally once daily on Days 2 & 3 or **With aprepitant:** Dexamethasone 12 mg single oral dose before chemotherapy on Day 1 **Note: The regimen also includes a 5-HT_3 receptor antagonist with or without aprepitant	Dexamethasone 8 mg single oral dose (given alone) before chemotherapy on Day 1
Or, as prescribed by a physician		

*Emetic Risk is the incidence of emesis without antiemetics
**See individual product monograph for complete information on specific 5-HT_3 receptor antagonist and aprepitant

Dexamethasone Suppression Tests

A. Test for Cushing's Syndrome:

- 1 mg Dexamethasone orally at 11 PM, then blood is extracted for plasma cortisol determination at 8 AM the following morning.

OR

- 0.5 mg (500 mcg) Dexamethasone orally every 6 hours for 48 hours, then to determine urine free cortisol and 17 – hydroxycorticosteroid excretion. This test provides greater accuracy.

B. Test for distinguishing Cushing's Syndrome due to pituitary Adrenocorticotropic Hormone (ACTH) excess from Cushing's syndrome due to other causes:

- 2 mg Dexamethasone orally every 6 hours for 48 hours, then 24 – hour urine free cortisol and 17 – hydroxycorticosteroid excretion.

CONTRAINDICATIONS

- Systemic fungal infections
- Hypersensitivity to dexamethasone or other ingredients in this product
- Gastrointestinal ulcer
- Acute or chronic infections
- Osteoporosis
- Pregnancy
- Diabetes mellitus
- Renal insufficiency
- Hypertension
- History of psychotic illness
- Immediately before prophylactic immunization

WARNINGS AND PRECAUTIONS

General:

Increased corticosteroid dosage is required in patients with adrenal insufficiency who are subjected to stress (e.g., infection, surgery, trauma). Abrupt withdrawal of corticosteroids after prolonged therapy may result in corticosteroid withdrawal syndrome with symptoms such as fever, myalgia, arthralgia, and malaise occurring even in patients without evidence of adrenal insufficiency.

The lowest effective corticosteroid dose should be used to control the condition being treated. Gradual reduction of dosage is recommended whenever possible.

Corticosteroid effects are exaggerated in patients with hypothyroidism and cirrhosis.

Kaposi's sarcoma has been seen mostly in patients receiving corticosteroids for chronic conditions. Clinical improvement may be observed upon discontinuation of corticosteroids.

Endocrine:

Minimize drug-induced secondary adrenocortical insufficiency caused by rapid withdrawal of corticosteroids by gradual dosage reduction. Since drug-induced adrenocortical insufficiency may persist for months after discontinuation of therapy, reinstitute corticosteroid therapy in any situation of stress occurring during that period. Dosage may have to be increased in patients already receiving corticosteroid therapy. Administer a salt and/or mineralocorticoid concurrently since mineralocorticoid secretion may also be impaired.

Metabolic clearance of corticosteroids may be altered in hypothyroid (decreased clearance) or hyperthyroid (increased clearance) patients. Dosage adjustment based on thyroid status may be necessary for these patients.

Vaccination:

Do not administer live or live attenuated vaccines (including small pox vaccine) in patients receiving corticosteroids. Although killed or inactivated vaccines may be administered in patients on corticosteroids, response to such vaccines cannot be predicted.

Patients who are receiving corticosteroids as replacement therapy, e.g., for Addison's disease, may be immunized.

Infections:

Glucocorticoids suppress the immune system and increase susceptibility to or mask symptoms of infection. Corticosteroids may cause decreased resistance and inability to localize infection and may also produce false negative results with nitroblue-tetrazolium test for bacterial infections. Infections caused by any pathogen including viral, bacterial, fungal, protozoan, or helminthic infections in any location of the body may be associated with corticosteroids alone or in combination with other immunosuppressive agents. These infections may be mild to severe and occurs more frequently with increasing doses of corticosteroids.

- ***Viral Infections:*** Viral infections such as chicken pox or measles can be more serious or even fatal in non-immunized children or adults on corticosteroids. Patients who have not had these diseases, should take particular care to avoid exposure. Prophylaxis with varicella zoster immune globulin (VZIG) may be indicated if exposed to chicken pox, while prophylaxis with immunoglobulin (IG) may be indicated if exposed to measles. Consider treatment with antiviral agents if chicken pox develops.

- ***Fungal Infections:*** Systemic fungal infections may be exacerbated by corticosteroids. Do not use corticosteroids in the presence of such infections unless they are needed to control life-threatening drug reactions. Cardiac enlargement and congestive heart failure have been reported with concomitant use of amphotericin B and hydrocortisone.

- ***Special Pathogens:*** Corticosteroids may cause activation of latent diseases or exacerbation of intercurrent infections due to pathogens including Candida, Mycobacterium, Amoeba, Toxoplasma, Pneumocystis, Cryptococcus, Nocardia, etc.

  As corticosteroids may also activate latent amoebiasis, rule out latent or active amoebiasis before initiating corticosteroid therapy in patients who have spent time in the tropics or with unexplained diarrhea.

  In patients with known or suspected Strongyloides (threadworm) infestation, corticosteroid-induced immunosuppression may lead to Strongyloides hyperinfection and dissemination with widespread larval migration which is often accompanied by severe enterocolitis and potentially fatal gram-negative septicemia. Use corticosteroids with great care in these patients.

  Do not use corticosteroids in cerebral malaria since it is associated with prolongation of coma and higher incidence of pneumonia and gastrointestinal bleeding.

- ***Tuberculosis:*** Use dexamethasone in active tuberculosis only in fulminating or disseminated cases in which corticosteroids are used with appropriate antituberculous regimen.

  Since reactivity to the disease may occur in patients with latent tuberculosis or tuberculin reactivity, observe these patients closely and administer chemoprophylaxis during prolonged corticosteroid therapy.

Ophthalmic:

Prolonged use of corticosteroids may cause posterior subcapsular and nuclear cataracts (particularly in children), exophthalmos, or increased intraocular pressure which may result in glaucoma or may occasionally damage the optic nerve. Monitor intraocular pressure if steroid therapy is continued for more than 6 weeks. Development of secondary fungal and viral infections of the eye may be enhanced in patients receiving corticosteroids.
Do not use corticosteroids in active ocular herpes simplex to avoid corneal perforation.

Cardio-Renal:

Sodium retention resulting in edema, potassium loss, hypokalemic alkalosis, and hypertension may occur in patients receiving glucocorticoids. Congestive heart failure may occur in susceptible patients. These mineralocorticoid effects occur mostly with average and large doses of hydrocortisone or cortisone and occur less frequently with synthetic corticosteroids such as dexamethasone. However, these effects may occur when synthetic corticosteroids are used in high dosage for prolonged periods. Dietary salt restriction and potassium supplementation may be advised when necessary. All corticosteroids increase calcium excretion which may result in hypocalcemia.

Use corticosteroids with caution in patients with hypertension, congestive heart failure, or renal insufficiency.

Use corticosteroids with caution in patients who have experienced a recent myocardial infarction since corticosteroid use is associated with left ventricular free wall rupture in these patients.

Neuro-Psychiatric:

Acute myopathy which occurs most frequently in patients with disorders of neuromuscular transmission such as myasthenia gravis, or in patients receiving therapy with neuromuscular blocking agents such as pancuronium, has been observed with use of high doses of corticosteroids. Acute myopathy, which may result in quadriparesis, is generalized and may involve ocular and respiratory muscles. Elevation of creatine kinase may also be observed.

Clinical improvement or recovery may need weeks to years of stopping corticosteroid therapy.

Corticosteroids may lead to mental disturbances including euphoria, mood swings, depression and anxiety, personality changes, and frank psychoses. Corticosteroids may also aggravate emotional instability or psychotic tendencies.

Gastrointestinal:

Corticosteroids have been implicated in the development, reactivation, perforation, hemorrhage, and delayed healing of peptic ulcers and should therefore not be used in patients with peptic ulcers except in life-threatening situations.

Use corticosteroids with caution in patients with diverticulitis, recent intestinal anastomoses, or nonspecific ulcerative colitis especially if there is risk of impending perforation, abscess or other pyogenic infection.

Manifestations of peritoneal irritation following gastrointestinal perforation may not be seen or is minimal in patients receiving corticosteroids.

Musculoskeletal:

Manifestations of protein catabolism which may occur during prolonged corticosteroid therapy include muscle wasting, muscle pain or weakness, delayed wound healing, and atrophy of the protein matrix of the bone resulting in osteoporosis, vertebral compression fractures, aseptic necrosis of femoral or humeral heads, or pathologic fractures of long bones. These catabolic effects may lead to osteoporosis at any age and inhibition of bone growth in children and adolescents. Since postmenopausal women and geriatric or debilitated patients are especially prone to osteoporosis, special consideration must be given to these patients prior to initiation of corticosteroid therapy.

INTERACTION WITH OTHER MEDICAMENTS

Barbiturates, phenytoin, ephedrine, carbamazepine, rifampicin, and other drugs that stimulate hepatic metabolism: These drugs may enhance dexamethasone metabolism, shorten its plasma half-life and lead to less effect of dexamethasone. Phenytoin may decrease dexamethasone's bioavailability by over 50%. Increased dexamethasone dosage may be required.

CYP 3A4 inhibitors such as ketoconazole and macrolide antibiotics: May increase plasma concentrations of corticosteroids.

Indinavir, erythromycin and other drugs metabolized by CYP 3A4: Dexamethasone is a moderate inducer of CYP 3A4 and may increase clearance of these drugs, resulting in decreased plasma concentrations.

Oral contraceptives and estrogen: Can cause alterations in plasma protein binding and metabolism of corticosteroids which can result in exposure of women to increased levels of the unbound corticosteroid for long periods of time.

Aspirin: Concomitant use of aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids increases the risk of gastrointestinal adverse effects. Use aspirin cautiously in conjunction with corticosteroids in hypoprothrombinemia.

Concomitant administration of dexamethasone and aspirin may also cause changes in salicylate plasma protein binding and its rate of metabolism. These changes cause lowering of plasma salicylate levels.

Warfarin: Coadministration of corticosteroids and warfarin usually results in decreased response to warfarin, although there have been conflicting results. Monitor coagulation indices frequently to maintain the desired anticoagulant effect.

Potassium-depleting agents such as diuretics and amphotericin-B: Observe patients closely for the development of hypokalemia. In addition, there have been cases reported in which concomitant use of amphotericin B and hydrocortisone was followed by cardiac enlargement and congestive heart failure.

Anticholinesterase agents: Concomitant use of anticholinesterase agents and corticosteroids may produce severe weakness in patients with myasthenia gravis. If possible, withdraw anticholinesterase agents at least 24 hours before initiating corticosteroid therapy.

Antacids: Large doses of antacids can cause a decrease in corticosteroid absorption.

Insulin and Oral Antidiabetic Agents: Concominant administration of dexamethasone and insulin generally requires higher doses of insulin. Dosage adjustment of antidiabetic agents may be required because corticosteroids may increase blood glucose concentrations.

Ketoconazole: Decreases the metabolism of certain corticosteroids by up to 60% leading to an increased risk of corticosteroid side effects.

Thalidomide: Toxic epidermal necrolysis has been reported when dexamethasone was used concomitantly with thalidomide.

Indomethacin: May cause false negative results in the dexamethasone suppression test (DST).

Aminoglutethimide: May diminish adrenal suppression of dexamethasone.

Cholestyramine: May increase the clearance of dexamethasone.

Ciclosporin: Increased activity of both ciclosporin and corticosteroids may occur when the two are used concurrently. Convulsions have been reported with concurrent use.

Digitalis Glycosides: Patients may be at increased risk of arrhythmias due to hypokalemia.

Ephedrine: May enhance the metabolic clearance of corticosteroids, resulting in decreased blood levels and lessened physiologic activity, thus requiring an increase in corticosteroid dosage.

Other interactions:
- ***Toxoids and live or inactivated vaccines:*** Patients on prolonged corticosteroid therapy may exhibit diminished response to toxoids and live or inactivated vaccines due to inhibition of antibody response. Corticosteroids may also potentiate the replication of some organisms contained in live attenuated vaccines. If possible, postpone routine administration of vaccines or toxoids until corticosteroid therapy is discontinued.
- ***Skin tests:*** Corticosteroids may suppress reactions to skin tests.

STATEMENT ON USAGE FOR HIGH RISK GROUPS

Pregnancy

Pregnancy Category C; D in 1st trimester

Corticosteroids may cause fetal damage when administered to pregnant women. Women who are pregnant or planning on becoming pregnant while receiving corticosteroids should inform their physician. Dexamethasone and other corticosteroids should only be used during pregnancy if the potential benefit justifies the potential risk to the fetus.

Carefully monitor infants born to women who received corticosteroids during pregnancy for symptoms of adrenal insufficiency. Begin appropriate therapy immediately when such symptoms appear.

Lactation

Corticosteroids may be excreted in breast milk and could suppress growth, interfere with endogenous glucocorticoid production, or cause other adverse effects in breastfeeding infants. Dexamethasone and other corticosteroids should be administered to breastfeeding mothers only if the potential benefits outweigh the potential risk to the infant.

Infants and Children

The efficacy and safety of corticosteroids are considered to be similar in adults and children. Periodically evaluate height, weight, ocular pressure, and blood pressure of children receiving corticosteroids. As in adults, children should undergo clinical evaluation for the presence of infection, psychosocial disturbances, thromboembolism, peptic ulcers, cataracts, and osteoporosis.

As much as possible, avoid long-term administration of pharmacologic doses of corticosteroids in children since these drugs may retard bone growth. Closely monitor growth and development of infants and children if prolonged therapy is necessary. Carefully weigh the potential effects on growth against the clinical benefits and the availability of alternative therapy. Minimize the potential effects of corticosteroids on growth by titrating to the lowest effective dose.

ADVERSE EFFECTS/UNDESIRABLE EFFECTS

The "therapeutically effective dose" of a corticosteroid varies with the indication and the requirement of the individual patients which makes it hardly possible to define an excessive dose. However, continued use of high doses necessary for clinical response in severe illness results in exaggeration of usual corticosteroid problems.

Adverse effects caused by dexamethasone and other corticosteroids include decreased carbohydrate tolerance, development of cushingoid state, glycosuria, hirsutism, hyperglycemia, increased requirements for insulin or oral hypoglycemic agents in diabetes, manifestations of latent diabetes mellitus, menstrual irregularities, secondary adrenocortical and pituitary unresponsiveness particularly in times of stress as in trauma, surgery or illness. These drugs may also cause suppression of growth in children, congestive heart failure in susceptible patients, fluid retention, hypertension, hypokalemic alkalosis, potassium loss, sodium retention, bradycardia, cardiac arrest, cardiac arrhythmias, cardiac enlargement, circulatory collapse, fat embolism, hypertrophic cardiomyopathy in premature infants, myocardial rupture following recent myocardial infarction, edema, pulmonary edema, syncope, tachycardia, thromboembolism, thrombophlebitis, and vasculitis.

Musculoskeletal and neurological adverse effects include aseptic necrosis of femoral and humeral heads, loss of muscle mass, muscle weakness, osteoporosis, pathological fracture of long bones, steroid myopathy, tendon rupture, vertebral compression fractures, convulsions, depression, emotional instability, euphoria, headache, increased intracranial pressure with papilledema (pseudotumor cerebri) usually after discontinuation of treatment, insomnia, mood swings, neuritis, neuropathy, paresthesia, personality changes, psychic disorders, and vertigo.

Dexamethasone and other corticosteroids may also cause abdominal distention, elevation of serum liver enzyme levels which is usually reversible upon discontinuation, hepatomegaly, increased appetite, nausea, pancreatitis, peptic ulcer with possible perforation and hemorrhage, perforation of the small and large bowel particularly in patients with inflammatory bowel disease, ulcerative esophagitis; acne, allergic dermatitis, dry scaly skin, erythema, increased sweating, impaired wound healing, petechiae and ecchymoses, thin fragile skin, rash, striae, thinning scalp hair, urticaria and may suppress reactions to skin tests.

Other possible adverse reactions include negative nitrogen balance due to protein catabolism, exophthalmos, glaucoma, increased intraocular pressure, posterior subcapsular cataracts, abnormal fat deposits, decreased resistance to infection, hiccups, increased or decreased motility and number of spermatozoa, malaise, moon face, weight gain, and allergic reactions including anaphylactoid reactions, anaphylaxis, and angioedema.

OVERDOSAGE & MANAGEMENT

As the clinically effective dose of corticosteroids vary according to the indications and requirements of individual patients, it is hardly possible to define "excessive" dosage of corticosteroids and reports of acute toxicity and/or death resulting from corticosteroid overdosage are rare. However, continued use of large doses of corticosteroids (which are often necessary to elicit a clinical response) without proper dose reduction, leads to exaggeration of usual corticosteroid-related problems such as mental symptoms, moon face, abnormal fat deposits, fluid retention, excessive appetite, weight gain, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, increased blood pressure, tachycardia, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, headache, weakness, menstrual disorders, accentuated menopausal symptoms, neuropathy, fractures, osteoporosis, peptic ulcer, decreased glucose tolerance, hypokalemia, and adrenal insufficiency.

No specific antidote is available and acute overdosage should be treated immediately by gastric lavage or emesis followed by supportive and symptomatic therapy.

CAUTION

Foods, Drugs, Devices, and Cosmetics Act prohibits dispensing without prescription.

STORAGE

STORE AT TEMPERATURES NOT EXCEEDING 30°C.
PROTECT FROM LIGHT.

Date of Revision: December 17, 2015

Manufactured by
ASIAN ANTIBIOTICS, INC.
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Mandaluyong City, Philippines for
UNILAB, Inc.
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Metro Manila, Philippines

Trusted Quality Healthcare

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