

# Dexamethasone 0.5 mg Tablets

## 1. Name of the medicinal product

Dexamethasone Tablets BP 0.5 MG.

## 2. Qualitative and quantitative composition

Each tablet contains 0.5mg dexamethasone BP.

## 3. Pharmaceutical form

Tablet

White, Circular bi convex uncoated round and a diameter of 5.5 mm. and caplets with "DEXA" embossed on one side and plain on other side.

## 4. Clinical particulars

### 4.1 Therapeutic indications

Indicated in a wide variety of disorders amenable to glucocorticoid therapy, as well as an adjunct in the control of cerebral oedema.

### 4.2 Posology and method of administration

#### Posology

In general, glucocorticoid dosage depends on the severity of the condition and response of the patient. Under certain circumstances, for instance in stress and changed clinical picture, extra dosage adjustments may be necessary. If no favourable response is noted within a couple of days, glucocorticoid therapy should be discontinued.

#### *Adults*

Usually, daily oral dosages of 0.5 - 10 mg are sufficient. In some patients higher dosages may be temporarily required to control the disease. Once the disease is under control the dosage should be reduced or tapered off to the lowest suitable level under continuous monitoring and observation of the patient. **(See Section 4.4)**

For a short dexamethasone suppression test, 1mg dexamethasone is given at 11 p.m. and plasma cortisol measured the next morning. Patients who do not show a decrease in cortisol can be exposed to a longer test: 500 micrograms dexamethasone is given at 6 hourly intervals for 48 hours followed by 2mg every 6 hours for a further 48 hours. 24 hour-urine collections are made before, during and at the end of the test for determination of 17-hydroxycorticosteroids.

#### *Paediatric population*

0.01-0.1mg/kg of body weight daily.

Dosage of glucocorticoids should be adjusted on the basis of the individual patient's response.

### 4.3 Contraindications

Systemic infection unless specific anti-infective therapy is employed.

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.

**Avoid live vaccines in patients receiving immunosuppressive doses (serum antibody response diminished).**

In general no contraindications apply in conditions where the use of glucocorticoids may be lifesaving.

### 4.4 Special warnings and precautions for use

**A patient information leaflet should be supplied with this product.**

In post-marketing experience tumour lysis syndrome (TLS) has been reported in patients with haematological malignancies following the use of dexamethasone alone or in combination with other chemotherapeutic agents. Patients at high risk of TLS such as patients with high proliferative rate, high tumour burden, and high sensitivity to cytotoxic agents, should be monitored closely and appropriate precaution taken.

Patients and/or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids (see section 4.8). Symptoms typically emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/systemic exposure (see also section 4.5 for pharmacokinetic interactions that can increase the risk of side effects), although dose levels do not allow prediction of the onset, type, severity or duration of reactions. Most reactions recover after either dose reduction or withdrawal, although specific treatment may be necessary. Patients/carers should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected. Patients/carers should also be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids, although such reactions have been reported infrequently.

Particular care is required when considering the use of systemic corticosteroids in patients with existing or previous history of severe affective disorders in themselves or in their first degree relatives. These would include depressive or manic-depressive illness and previous steroid psychosis.

The results of a randomised, placebo-controlled study suggest an increase in mortality if methylprednisolone therapy starts more than two weeks after the onset of Acute Respiratory Distress Syndrome (ARDS). Therefore, treatment of ARDS with corticosteroids should be initiated within the first two weeks of onset of ARDS (see also section 4.2.).

#### ***Preterm neonates:***

Available evidence suggests long-term neurodevelopmental adverse events after early treatment (<96 hours) of premature infants with chronic lung disease at starting doses of 0.25 mg/kg twice daily.

Undesirable effects may be minimised by using the lowest effective dose for the minimum period, and by administering the daily requirement as a single morning dose or whenever possible as a single morning dose on alternative days. Frequent patient review is required to appropriately titrate the dose against disease activity.

#### ***Dexamethasone withdrawal***

Adrenal cortical atrophy develops during prolonged therapy and may persist for years after stopping treatment. Withdrawal of corticosteroids after prolonged therapy must therefore always be gradual to avoid acute adrenal insufficiency, being tapered off over weeks or months according to the dose and duration of treatment.

In patients who have received more than physiological doses of systemic corticosteroids (approximately 1mg dexamethasone) for greater than 3 weeks, withdrawal should not be abrupt. How dose reduction should be carried out depends largely on whether the disease is likely to relapse as the dose of systemic corticosteroids is reduced. Clinical assessment of disease activity may be needed during withdrawal. If the disease is unlikely to relapse on withdrawal of systemic corticosteroids but there is uncertainty about HPA suppression, the dose of systemic corticosteroid may be reduced rapidly to physiological doses. Once a daily dose of 1mg dexamethasone is reached, dose reduction should be slower to allow the HPA-axis to recover.

Abrupt withdrawal of systemic corticosteroid treatment, which has continued up to 3 weeks is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses of up to 6mg daily of dexamethasone for 3 weeks is unlikely to lead to clinically relevant HPA-axis suppression in the majority of patients. In the following patient groups, gradual withdrawal of systemic corticosteroid therapy should be *considered* even after courses lasting 3 weeks or less:

- Patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than 3 weeks.
- When a short course has been prescribed within one year of cessation of long-term therapy (months or years).
- Patients who may have reasons for adrenocortical insufficiency other than exogenous corticosteroid therapy.
- Patients receiving doses of systemic corticosteroid greater than 6mg daily of dexamethasone.
- Patients repeatedly taking doses in the evening.

During prolonged therapy any intercurrent illness, trauma or surgical procedure will require a temporary increase in dosage; if corticosteroids have been stopped following prolonged therapy they may need to be temporarily re-introduced.

Patients should carry 'Steroid treatment' cards which give clear guidance on the precautions to be taken to minimise risk and which provide details of prescriber, drug, dosage and the duration of treatment.

#### *Anti-inflammatory/Immunosuppressive effects and Infection*

Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. The clinical presentation may often be atypical, and serious infections such as septicaemia and tuberculosis may be masked and may reach an advanced stage before being recognised.

Appropriate anti-microbial therapy should accompany glucocorticoid therapy when necessary e.g. in tuberculosis and viral and fungal infections of the eye.

*Chickenpox is of particular concern since this normally minor illness may be fatal in immunosuppressed patients.* Patients (or parents of children) without a definite history of chickenpox should be advised to avoid close personal contact with chickenpox or herpes zoster and if exposed they should seek urgent medical attention. Passive immunisation with varicella zoster immunoglobulin (VZIG) is needed by exposed non-immune patients who are receiving systemic corticosteroids or who have used them within the previous 3 months; this should be given within 10 days of exposure to chickenpox. If a diagnosis of chickenpox is confirmed, the illness warrants specialist care and urgent treatment. Corticosteroids should not be stopped and the dose may need to be increased.

**Measles. Patients should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs; prophylaxis with intramuscular normal immunoglobulin may be needed.**

#### *Visual disturbance*

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.

Particular care is required when considering the use of systemic corticosteroids in patients with the following conditions and frequent patient monitoring is necessary

- a. Osteoporosis (post-menopausal females are particularly at risk)
- b. Hypertension or congestive heart failure
- c. Existing or previous history of severe affective disorders (especially previous steroid psychosis)
- d. Diabetes mellitus (or a family history of diabetes)
- e. History of tuberculosis
- f. Glaucoma (or a family history of glaucoma)
- g. Previous corticosteroid-induced myopathy
- h. Liver failure
- i. Renal insufficiency
- j. Hypothyroidism
- k. Epilepsy
- l. Peptic ulceration
- m. Migraine
- n. Certain parasitic infestations in particular amoebiasis
- o. Incomplete natural growth since glucocorticoids on prolonged administration may accelerate epiphyseal closure

**Caution should be exercised when using corticosteroids in patients who have recently suffered myocardial infarction as myocardial rupture has been reported.**

After administration of glucocorticoids serious anaphylactoid reactions such as glottis oedema, urticaria and bronchospasm have occasionally occurred particularly in patients with a history of allergy.

If such an anaphylactoid reaction occurs, the following measures are recommended: immediate slow intravenous injection of 0.1-0.5ml of adrenaline (solution of 1:1000: 0.1-0.5mg adrenaline dependent on body weight), intravenous administration of aminophylline and artificial respiration if necessary.

**Dexamethasone Tablets contain lactose.** Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

#### Paediatric population

Corticosteroids cause dose-related growth retardation in infancy, childhood and adolescence, which may be irreversible.

#### *Use in the Elderly*

The common adverse effects of systemic corticosteroids may be associated with more serious consequences in old age, especially osteoporosis, hypertension, hypokalaemia, diabetes, susceptibility to infection and thinning of the skin. Close clinical supervision is required to avoid life-threatening reactions.

#### **4.5 Interaction with other medicinal products and other forms of interaction**

Rifampicin, rifabutin, carbamazepine, phenobarbital, phenytoin, primidone, and aminoglutethimide enhance the metabolism of corticosteroids and its therapeutic effects may be reduced.

Dexamethasone is a moderate inducer of CYP 3A4. Co-administration of dexamethasone with other drugs that are metabolized by CYP 3A4 (e.g., indinavir, erythromycin) may increase their clearance, resulting in decreased plasma concentrations.

Co-treatment with CYP3A inhibitors, including cobicistat-containing products, is expected to increase the risk of systemic side-effects. The combination should be avoided unless the benefit outweighs the increased risk of systemic corticosteroid side-effects, in which case patients should be monitored for systemic corticosteroid side-effects.

#### **Ephedrine also accelerates the metabolism of dexamethasone.**

The effects of anticholinesterases are antagonised by corticosteroids in myasthenia gravis.

The desired effects of hypoglycaemic agents (including insulin), anti-hypertensives and diuretics are antagonised by corticosteroids, and the hypokalaemic effects of acetazolamide, loop diuretics, thiazide diuretics and carbenoxolone are enhanced.

The efficacy of coumarin anticoagulants may be enhanced by concurrent corticosteroid therapy and close monitoring of the INR or prothrombin time is required to avoid spontaneous bleeding.

Oral contraceptives (oestrogens and progestogens) increase plasma concentration of corticosteroids. The antiviral drug ritonavir also increases the plasma concentration of dexamethasone.

#### **Dexamethasone reduces the plasma concentration of the antiviral drugs indinavir and saquinavir.**

The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication.

Patients taking NSAIDs should be monitored since the incidence and/or severity of gastro-intestinal ulceration may increase.

Patients taking methotrexate and dexamethasone have an increased risk of haematological toxicity.

Antacids, especially those containing magnesium trisilicate have been reported to impair the gastrointestinal absorption of glucocorticoid steroids. Therefore, doses of one agent should be spaced as far as possible from the other.

#### **4.6 Fertility, pregnancy and lactation**

##### Pregnancy

The ability of corticosteroids to cross the placenta varies between individual drugs, however, dexamethasone readily crosses the placenta.

Administration of corticosteroids to pregnant animals can cause abnormalities of foetal development including cleft palate, intra-uterine growth retardation and effects on brain growth and development. There is no evidence that corticosteroids result in an increased incidence of congenital abnormalities, such as cleft palate/lip in man (see also section 5.3). However, when administered for prolonged periods or repeatedly during pregnancy, corticosteroids may increase the risk of intra-uterine growth retardation. Hypoadrenalism may, in theory, occur in the neonate following prenatal exposure to corticosteroids but

usually resolves spontaneously following birth and is rarely clinically important. As with all drugs, corticosteroids should only be prescribed when the benefits to the mother and child outweigh the risks. When corticosteroids are essential however, patients with normal pregnancies may be treated as though they were in the non-gravid state.

#### Breast-feeding

Corticosteroids may pass into breast milk, although no data are available for dexamethasone. Infants of mothers taking high doses of systemic corticosteroids for prolonged periods may have a degree of adrenal suppression.

#### **4.7 Effects on ability to drive and use machines**

None known.

#### **4.8 Undesirable effects**

The incidence of predictable undesirable effects, including hypothalamic-pituitary-adrenal suppression correlates with the relative potency of the drug, dosage, timing of administration and the duration of treatment (see section 4.4).

##### *Endocrine/metabolic*

Suppression of the hypothalamic-pituitary-adrenal axis, growth suppression in infancy, childhood and adolescence, menstrual irregularity and amenorrhoea, Cushingoid faces, hirsutism, weight gain, premature epiphyseal closure, impaired carbohydrate tolerance with increased requirement for anti-diabetic therapy, negative protein and calcium balance, increased appetite

##### *Anti-inflammatory and Immunosuppressive effects*

Increased susceptibility and severity of infections with suppression of clinical symptoms and signs, opportunistic infections, recurrence of dormant tuberculosis (see section 4.4), decreased responsiveness to vaccination and skin tests

##### *Musculoskeletal*

Osteoporosis, vertebral and long bone fractures, avascular osteonecrosis, tendon rupture, proximal myopathy

##### *Fluid and electrolyte disturbance*

Sodium and water retention, hypertension, potassium loss, hypokalaemic alkalosis

##### *Neuropsychiatric*

A wide range of psychiatric reactions including affective disorders (such as irritable, euphoric, depressed and labile mood and suicidal thoughts), psychotic reactions (including mania, delusions, hallucinations and aggravation of schizophrenia), behavioural disturbances, irritability, anxiety, sleep disturbances and cognitive dysfunction including confusion and amnesia have been reported. Reactions are common and may occur in both adults and children. In adults, the frequency of severe reactions has been estimated to be 5-6%. Psychological effects have been reported on withdrawal of corticosteroids; the frequency is unknown.

Increased intra-cranial pressure with papilloedema in children (pseudotumour cerebri), usually after treatment withdrawal. Aggravation of epilepsy. Psychological dependence.

##### *Ophthalmic*

Increased intra-ocular pressure, glaucoma, papilloedema, posterior subcapsular cataracts, corneal or scleral thinning, exacerbation of ophthalmic viral or fungal diseases, chorioretinopathy

##### *Eye disorders*

Vision, blurred (see also section 4.4)

##### *Gastrointestinal*

Dyspepsia, peptic ulceration with perforation and haemorrhage, acute pancreatitis, oesophageal ulceration and candidiasis, abdominal distension and vomiting

##### *Dermatological*

Impaired healing, skin atrophy, bruising, telangiectasia, striae, acne

## General

Hypersensitivity, including anaphylaxis and angioedema, have been reported. Leucocytosis, thromboembolism, myocardial rupture following recent myocardial infarction, nausea, malaise, hiccups

### *Withdrawal symptoms and signs*

Too rapid a reduction of corticosteroid dosage following prolonged treatment can lead to acute adrenal insufficiency, hypotension and death (see section 4.4).

A 'withdrawal syndrome' may also occur including, fever, myalgia, arthralgia, rhinitis, conjunctivitis, painful itchy skin nodules and loss of weight.

### Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via the Yellow Card Scheme at [www.mhra.gov.uk/yellowcard](http://www.mhra.gov.uk/yellowcard).

## 4.9 Overdose

It is difficult to define an excessive dose of a corticosteroid as the therapeutic dose will vary according to indication and patient requirements. Exaggeration of corticosteroid related adverse effects may occur. Treatment should be asymptomatic and supportive as necessary.

## 5. Pharmacological properties

### 5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Corticosteroids for systemic use, Glucocorticoids, ATC code: H02AB02

Dexamethasone is a synthetic glucocorticoid whose anti-inflammatory potency is 7 times greater than prednisolone. Like other glucocorticoids, dexamethasone also has anti-allergic, antipyretic and immunosuppressive properties.

Dexamethasone has practically no water and salt-retaining properties, and is therefore particularly suitable for use in patients with cardiac failure or hypertension. Because of its long biological half-life (36-54 hours), dexamethasone is especially suitable in conditions where continuous glucocorticoid action is desired.

### 5.2 Pharmacokinetic properties

#### Absorption

Corticosteroids, are, in general, readily absorbed from the gastro-intestinal tract. They are also well absorbed from sites of local application. Water-soluble forms of corticosteroids are given by intravenous injection for a rapid response; more prolonged effects are achieved using lipid-soluble forms of corticosteroids by intramuscular injection.

#### Distribution

Corticosteroids are rapidly distributed to all body tissues. They cross the placenta and may be excreted in small amounts in breast milk.

Most corticosteroids in the circulation are extensively bound to plasma proteins, mainly to globulin and less so to albumin. The corticosteroid-binding globulin has high affinity but low binding capacity, while the albumin has low affinity but large binding capacity. The synthetic corticosteroids are less extensively protein bound than hydrocortisone (cortisol). They also tend to have longer half-lives.

#### Biotransformation and Elimination

Corticosteroids are metabolised mainly in the liver but also in the kidney, and are excreted in the urine. The slower metabolism of the synthetic corticosteroids with their lower protein-binding affinity may account for their increased potency compared with the natural corticosteroids.

### 5.3 Preclinical safety data

In animal studies, cleft palate was observed in rats, mice, hamsters, rabbits, dogs and primates: not in horses and sheep. In some cases these divergences were combined with defects of the central nervous system and of the heart. In primates, effects in the brain were seen after exposure. Moreover, intra-uterine growth can be delayed. All these effects were seen at high dosages.

## 6. Pharmaceutical particulars

**6.1 List of excipients**

Maize Starch

Di basic calcium Phosphate

Bronopol

Talcum

Magnesium Stearate

**6.2 Incompatibilities**

None known.

**6.3 Shelf life**

3 years

**6.4 Special precautions for storage**

Store below 25°C protected from light.

**6.5 Nature and contents of container**

100 Tablets Packed in a amber coloured Tubeler glass vials printed with a Pvc Cap.

1000 caplets packed in poly bag each poly bag packed in a HDPE jar and each jar packed in printed box

**6.6 Special precautions for disposal and other handling**

No special requirements.

**SOLE AGENT : LIFELINE PHARMACEUTICALS**

**NAFDAC REG. NO. : A4-5794**

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<p><b>COMPOSITION:</b> Each uncoated tablet contains: Dexamethasone B.P. .... 0.5 mg. Excipients .... q.s. <b>DOSAGE:</b> As directed by the physician . Store in a cool, dry &amp; dark place below 30°C. Protect from sunlight. Keep all medicines out of reach of children.</p> <p>Manufactured By : <b>Mancare</b> PHARMACEUTICALS PVT. LTD. Plot No. 59, 60, 85, 86, V.M.I.E, Dowali Village, Vasai (W), Dist. Thane, Maharashtra, India. E-mail: mancerp@yahoo.co.in</p>	<p>1000 Tablets</p> <p><b>Lifeline</b> <b>Dexamethasone</b></p> <p><b>0.5 mg</b></p> <p><b>Dexamethasone</b> <b>Tablets B.P. 0.5 mg</b></p> <p>NAFDAC Reg. No.: A4-5794</p>	<p>Mfg. Lic. No. : KD-354</p> <p>Batch No. : <input type="text"/></p> <p>Mfg Date : <input type="text"/></p> <p>Exp Date : <input type="text"/></p>  <p>18906062950958</p> <p>Sole Agent: <b>Lifeline Pharmaceuticals</b> 30 Umuoji Street, Nkpor, Anambra State, Nigeria.</p>
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184(L) x 55(H) mm Prepared On : 20July2017

<p>For the use of Registered medical Practitioner or a Hospital or a Laboratory only.</p> <p><b>Lifeline</b> <b>Dexamethasone</b></p> <p><b>(Dexamethasone Tablets B.P)</b></p>	
<p><b>COMPOSITION</b> Each uncoated tablet contains:- Dexamethasone B.P. 0.5 mg</p> <p><b>PHARMACOLOGICAL CLASSIFICATION:-CORTICOSTEROID</b></p> <p><b>PHARMACOLOGY</b> Dexamethasone is a long acting fluorinated glucocorticoid with 25-30 times more potency than an equal weight of hydrocortisone. It is faster acting with longer duration than the intermediate oral agents prednisone and prednisolone. In dogs, the drug has been shown to have a half-life of 2 to 5 hours with biologic activity of up to 48 hours. Dexamethasone stimulates synthesis of enzymes required to decrease the inflammatory response. Its primary efficacy is that of a strong anti-inflammatory, as well as an antiallergic agent. Besides being an anti-inflammatory, the drug will also suppress the immune response, stimulate bone marrow, and influence fat, protein, and carbohydrate metabolism. Although it is able to cause diuresis, there are more effective drugs for reducing edema. The drug is readily absorbed into tissue, and rapidly distributed to kidneys, liver, muscle, and intestines. It is metabolized in the liver and primarily excreted by the kidneys. The drug can be given orally, used topically, or by injection.</p> <p><b>INDICATIONS</b> Used as a quick agent to reduce inflammation in ear infections causing wry neck/head tilt. Can be used as support therapy in respiratory infections unresponsive to antimicrobials alone, or in serious to advanced lung infections (e.g., pneumonia) to reduce allergic response (especially where pathogen suspected to be mycoplasma). Also used for swelling caused by injury, cerebral edema, neoplasia, and septic shock.</p> <p><b>DRUG INTERACTIONS OR CONTRAINDICATIONS</b> Do not give in active untreated infections or viral, and fungal diseases of the eye. Use with caution in CHF and colitis.</p>	<p>Incompatible mixed in with doxorubicin HCL, daunorubicin HCL, and vancomycin. (*note updated correction: may be used concurrently with doxycycline) Has only minor mineralocorticoid effect and should not be used alone to treat adrenal insufficiency. When used with NSAIDs will increase GI distress or cause bleeding, use together cautiously.</p> <p><b>ADVERSE REACTIONS</b> EENT: cataracts, glaucoma CV: CHF, edema GI: Known to increase appetite but may have opposite effect in rats with resulting weight loss. Skin: delayed wound healing, thinning of dermal tissue, skin atrophy Local: atrophy at injection site</p> <p><b>OTHER: MAY MASK OR EXACERBATE INFECTIONS ALREADY PRESENT</b></p> <p><b>PRESENTATION</b> Pack of 100 tablets in a glass bottle &amp; 1000 tablets in a plastic jar.</p> <p><b>STORAGE INSTRUCTIONS</b> Store in a cool, dark &amp; dry place below 30°C. Keep all medicines out of reach of children.</p> <p>NAFDAC Reg. No: A4-5794</p> <p>Mfg. Lic. No.: KD-354</p> <p>Manufactured By : <b>Mancare</b> PHARMACEUTICALS PVT. LTD. Plot No. 59, 60, 85, 86, V.M.I.E, Dowali Village, Vasai (W), Dist. Thane, Maharashtra, India. E-mail: mancerp@yahoo.co.in</p> <p>Sole Agent: <b>Lifeline Pharmaceuticals</b> 30 Umuoji Street, Nkpor, Anambra State, Nigeria.</p>

Lifeline Dexa \_\_ Size: 110 x 100 mm Dated 20 July 2017




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1000 Tablets

**Lifeline**  
**Dexamethasone**



**Dexamethasone**  
**Tablets B.P. 0.5 mg**

 **NAFDAC Reg. No.: A4-5794**





