Product Name= RGI PREDNISOLONE
Generic Name= PREDNISOLONE BP 5MG

SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

RGI PREDNISOLONE (Prednisolone BP 5mg)

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each Tablet contains:

Prednisolone B.P 5mg

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

A white small round tablet with inscription of RGI on one side and P/5 on the other side.

4. CLINICAL PARTICULARS

Therapeutic indications

Allergy and anaphylaxis: bronchial asthma, drug hypersensitivity reactions, serum sickness, angioneurotic oedema, anaphylaxis.
Arteritis/collagenosis: giant cell arteritis/polymyalgia rheumatica, mixed connective tissue disease, polyarteritis nodosa, polymyositis.
Blood disorders: haemolytic anaemia (auto-immune), leukaemia (acute and chronic lymphocytic), lymphoma, multiple myeloma, idiopathic thrombocytopenic purpura.
Cardiovascular disorders: post-myocardial infarction syndrome, rheumatic fever with severe carditis.
Endocrine disorders: primary and secondary adrenal insufficiency, congenital adrenal hyperplasia.
Gastro-intestinal disorders: Crohn's disease, ulcerative colitis, persistent coeliac syndrome (coeliac disease unresponsive to gluten withdrawal), auto-immune chronic active hepatitis, multisystem disease affecting liver, biliary peritonitis.
Hypercalcaemia: sarcoidosis, vitamin D excess.
Infections (with appropriate chemotherapy): helminthic infestations, Herxheimer reaction, infectious mononucleosis, miliary tuberculosis, mumps orchitis (adult), tuberculous meningitis, rickettsia disease.
Muscular disorders: polymyositis, dermatomyositis.
Neurological disorders: infantile spasms, Shy-Drager syndrome, sub-acute demyelinating polyneuropathy.
Ocular disease: scleritis, posterior uveitis, retinal vasculitis, pseudo-tumors of the orbit, giant cell arteritis, malignant ophthalmic Grave's disease.
Renal disorders: lupus nephritis, acute interstitial nephritis, minimal change glomerulonephritis.
Respiratory disease: allergic pneumonitis, asthma, occupational asthma, pulmonary aspergillosis, pulmonary fibrosis, pulmonary alveolitis, aspiration of foreign body, aspiration of stomach contents, pulmonary sarcoid, drug induced lung disease, adult respiratory distress syndrome, spasmodic croup.
Rheumatic disorders: rheumatoid arthritis, polymyalgia rheumatica, juvenile chronic arthritis, systemic lupus erythematosus, dermatomyositis, mixed connective tissue disease.

Skin disorders: pemphigus vulgaris, bullous pemphigoid, systemic lupus erythematosus, pyoderma gangrenosum.
Miscellaneous: sarcoidosis, hyperpyrexia, Behçets disease, immunosuppression in organ transplantation.

Posology and method of administration

Posology

Adults including the elderly

The lowest effective dose should be used for the minimum period in order to minimize side effects

Initially:

The initial dosage may vary from 5mg to 60mg daily in divided doses, as a single dose in the morning after breakfast, or as a double dose on alternate days. Dosage depends on the disorder being treated. The dose can often be reduced within a few days but may need to be continued for several weeks or months.

Maintenance:

2.5 to 15mg daily, but higher doses may be needed. Cushingoid side-effects more likely above 7.5mg daily.

Intermittent dosage regimen: A single dose of prednisolone in the morning on alternate days or at longer intervals is acceptable therapy for some patients. When this regimen is practical, the degree of pituitary-adrenal suppression can be minimised.

Specific dosage guidelines: The following recommendations for some corticosteroid-responsive disorders are for guidance only. Acute or severe disease may require initial high dose therapy with reduction to the lowest effective maintenance dose as soon as possible. Dosage reductions should not exceed 5-7.5mg daily during chronic treatment.

Allergic and skin disorders: Initial doses of 5-15mg daily are commonly adequate.

Collagenosis: Initial doses of 20-30mg daily are frequently effective. Those with more severe symptoms may require higher doses.

Rheumatoid arthritis: The usual initial dose is 10-15mg daily. The lowest daily maintenance dose compatible with tolerable symptomatic relief is recommended.

Blood disorders and lymphoma: An initial daily dose of 15-60mg is often necessary with reduction after an adequate clinical or haematological response. Higher doses may be necessary to induce remission in acute leukaemia.

Special populations

Elderly

Treatment of elderly patients, particularly if long-term, should be planned bearing in mind the more serious consequences of the common side-effects of corticosteroids in old age.

Children

Although appropriate fractions of the actual dose may be used, dosage will usually be determined by clinical response as in adults. Prednisolone should be used only when specifically indicated, in a minimal dosage and for the shortest possible time.

Method of administration

Tablets for oral administration. The tablets should be taken with or after food.

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

• Ocular herpes simplex because of possible perforation.

• Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine

Special warnings and precautions for use

- Patients and/or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids. Symptoms typically emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/systemic exposure, 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 tampering/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.
- Tumorigenicity: direct tumour-inducing effects of the glucocorticoids are not known, but the particular risk that malignancies in patients undergoing immunosuppression with these or other drugs will spread more rapidly is a well-recognised problem.
- Calciphylaxis may occur very rarely during treatment with corticosteroids. Although calciphylaxis is most commonly observed in patients who have end stage kidney failure, it has also been reported in patients taking corticosteroids who have minimal or no renal impairment and normal calcium, phosphate and parathyroid hormone levels. Patients/carers should be advised to seek medical advice if symptoms develop.Caution is necessary when oral corticosteroids, including prednisolone, are prescribed in patients with the following conditions, and frequent patient monitoring is necessary.
- Tuberculosis: Those with a previous history of, or X-ray changes characteristic of, tuberculosis. The emergence of active tuberculosis can however, be prevented by the prophylactic use of anti-tuberculosis therapy.

• Inflammatory bowel disease: Symptoms recurred in a patient with Crohn's disease on changing from conventional to enteric-coated tablets of prednisolone. This was not an isolated occurrence in the author's unit, and it was advocated that only non-enteric coated prednisolone tablets should be used in Crohn's disease, and that the enteric coated form should be used with caution in any condition characterized by diarrhea or a rapid transit time.

Adrenocortical insufficiency

Pharmacologic doses of corticosteroids administered for prolonged periods may result in hypothalamic-pituitary-adrenal (HPA) suppression (secondary adrenocortical insufficiency). The degree and duration of adrenocortical insufficiency produced is variable among patients and depends on the dose, frequency, time of administration, and duration of glucocorticoid therapy.

In addition, acute adrenal insufficiency leading to a fatal outcome may occur if glucocorticoids are withdrawn abruptly. Drug-induced secondary adrenocortical insufficiency may therefore be minimized by gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any situation of stress occurring during that period, hormone therapy should be reinstituted. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently. 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 minimize 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 septicemia and tuberculosis may be masked and may reach an advanced stage before being recognized when corticosteroids, including prednisolone, are used. The immunosuppressive effects of glucocorticoids may result in the activation of latent infection or exacerbation of intercurrent infection.

Chickenpox

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 immunization 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 taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs.

Administration of live vaccines

Live vaccines should not be given to individuals on high doses of corticosteroids, due to impaired immune response. Live vaccines should be postponed until at least 3 months after stopping corticosteroid therapy.

Ocular Effects

Prolonged use of corticosteroids may produce posterior subcapsular cataracts and nuclear cataracts (particularly in children), exophthalmos, or increased intraocular pressure, which may result in glaucoma

with possible damage to the optic nerves. Establishment of secondary fungal and viral infections of the eye may also be enhanced in patients receiving glucocorticoids.

Corticosteroids should be used cautiously in patients with ocular herpes simplex because of possible perforation.

Systemic glucocorticoid treatment can cause severe exacerbation of bullous exudative retinal detachment and lasting visual loss in some patients with idiopathic central serous chorioretinopathy.

Cushing's disease

Because glucocorticoids can produce or aggravate Cushing's syndrome, glucocorticoids should be avoided in patients with Cushing's disease

There is an enhanced effect of corticosteroids in patients with hypothyroidism.

Psychic derangements may appear when corticosteroids, including prednisolone, are used, ranging from euphoria, insomnia, mood swings, personality changes, and severe depression, to frank psychotic manifestations.

Raised intracranial pressure

Raised intracranial pressure with papilledema (pseudotumor cerebri) associated with corticosteroid treatment has been reported in both children and adults. The onset usually occurs after treatment withdrawal.

Scleroderma renal crisis

Caution is required in patients with systemic sclerosis because of an increased incidence of (possibly fatal) scleroderma renal crisis with hypertension and decreased urinary output observed with a daily dose of 15 mg or more prednisolone. Blood pressure and renal function (s-creatinine) should therefore be routinely checked. When renal crisis is suspected, blood pressure should be carefully controlled.

Use in the elderly

Treatment of elderly patients, particularly if long term, should be planned bearing in mind the more serious consequences of the common side-effects of corticosteroids in old age, especially osteoporosis, diabetes, hypertension, hypokalemia, susceptibility to infection and thinning of the skin. Close clinical supervision is required to avoid life threatening reactions.

Pediatric population

Corticosteroids cause growth retardation in infancy, childhood and adolescence, which may be irreversible, and therefore long-term administration of pharmacological doses should be avoided. If prolonged therapy is necessary, treatment should be limited to the minimum suppression of the hypothalamus-pituitary adrenal axis and growth retardation. The growth and development of infants and children should be closely monitored. Treatment should be administered where possible as a single dose on alternate days.

Interaction with other medicinal products and other forms of interaction

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.

Antacids	The absorption of prednisolone may be reduced by large doses of some antacids such as magnesium trisilicate or aluminium hydroxide.
Antibacterials	Rifamycins accelerate metabolism of corticosteroids and thus may reduce their

	effect. Erythromycin inhibits metabolism of methylprednisolone and possibly other corticosteroids. Prednisolone can lower plasma levels of isoniazid. Where a reduced response during concurrent use is noted, dosage adjustment of isoniazid may be necessary.
Anticoagulants	Response to anticoagulants may be reduced or, less often, enhanced by corticosteroids. Close monitoring of the INR or prothrombin time is required to avoid spontaneous bleeding.
Antidiabetic agents	Glucocorticoids may increase blood glucose levels. Patients with diabetes mellitus receiving concurrent insulin and/or oral hypoglycemic agents may require dosage adjustments of such therapy.
Antiepileptics	Carbamazepine, phenobarbital, phenytoin, and primidone accelerate metabolism of corticosteroids and may reduce their effect.
Antifungals	Risk of hypokalaemia may be increased with amphotericin, therefore concomitant use with corticosteroids should be avoided unless corticosteroids are required to control reactions; ketoconazole inhibits metabolism of methylprednisolone and possibly other corticosteroids.
Antimuscarinics (Anticholinergics)	Prednisolone has been shown to have antimuscarinic activity. If used in combination with another antimuscarinic drug could cause impairment to memory and attention in the elderly.
Antithyroids	Prednisolone clearance increased by the use of carbimazole and thiamazole.
Antiviral	Plasma concentrations of prednisolone may be increased with antiviral drugs such as ritonavir and indinavir.
Cardiac Glycosides	Increased toxicity if hypokalaemia occurs with corticosteroids.
Ciclosporin	Concomitant administration of prednisolone and ciclosporin may result in decreased plasma clearance of prednisolone (i.e. increased plasma concentration of prednisolone). The need for appropriate dosage adjustment should be considered when these drugs are administered concomitantly.
Cytotoxics	Increased risk of haematological toxicity with methotrexate.
Hepatic microsomal enzyme inducers	Drugs that induce hepatic enzyme cytochrome P-450 (CYP) isoenzyme 3A4 such as phenobarbital, phenytoin, rifampicin, rifabutin, carbamazepine, primidone and aminoglutethimide may reduce the therapeutic efficacy of corticosteroids by increasing the rate of metabolism. Lack of expected response may be observed and dosage of Deltacortril Gastro-resistant Tablets may need to be increased.
Hepatic microsomal enzyme inhibitors	Drugs that inhibit hepatic enzyme cytochrome P-450 (CYP) isoenzyme 3A4 (e.g. ketoconazole, troleandomycin) may decrease glucocorticoid clearance. Dosages of glucocorticoids given in combination with such drugs may need to be decreased to avoid potential adverse effects.
Hormonal contraceptives	Oral contraceptives increased prednisolone concentrations by 131%. May increase AUC and reduce clearance in oral contraceptives containing ethinylestradiol, mestranol, desogestrel, levonorgestrel, norgestrel or norethisterone.
Immunosuppressants	Tumorigenicity: direct tumour-inducing effects of the glucocorticoids are not known, but the particular risk that malignancies in patients undergoing immunosuppression with these or other drugs will spread more rapidly is a well-recognised problem. Mutual inhibition of metabolism may occur between ciclosporin and prednisolone, and may increase the plasma concentration of either drug.
Liquorice	Glycyrrhizin can delay the clearance of prednisolone
Mifepristone	Effect of corticosteroids may be reduced for 3-4 days after mifepristone

Non-steroidal anti-inflammatory drugs	Concomitant administration of ulcerogenic drugs such as indomethacin during corticosteroid therapy may increase the risk of GI ulceration. Aspirin should be used cautiously in conjunction with glucocorticoids in patients with hypoprothrombinaemia. Although concomitant therapy with salicylate and corticosteroids does not appear to increase the incidence or severity of GI ulceration, the possibility of this effect should be considered. Serum salicylate concentrations may decrease when corticosteroids are administered concomitantly. The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication. Salicylates and corticosteroids should be used concurrently with caution. Patients receiving both drugs should be observed closely for adverse effects of either drug.
Oestrogens	Oestrogens may potentiate the effects of glucocorticoids and dosage adjustments may be required if oestrogens are added to or withdrawn from a stable dosage regimen.
Protease inhibitors	Ritonavir possibly increases plasma concentrations of prednisolone and other corticosteroids by reduction in clearance of prednisolone through the inhibition of P450 isoenzyme CYP3A4.
Other	The desired effects of hypoglycaemic agents (including insulin), antihypertensives and diuretics are antagonised by corticosteroids; and the hypokalaemic effect of acetazolamide, loop diuretics, thiazide diuretics, carbenoxolone and theophylline are enhanced.
Somatropin	Growth promoting effect may be inhibited.
Sympathomimetics	Increased risk of hypokalaemia if high doses of corticosteroids given with high doses of bambuterol, fenoteral, formoteral, ritodrine, salbutamol, salmeterol and terbutaline.

Undesirable effects

Tabulated list of adverse reactions
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.
Assessment of undesirable effects is based on the following frequency groupings:
Very common: ≥1/10
Common: ≥1/100 to <1/10
Uncommon: ≥1/1,000 to <1/100
Rare: ≥1/10,000 to <1/1,000
Very rare: <1/10,000
Not known: cannot be estimated from the available data

System Organ Class	**Frequency**	**Undesirable Effect**
Infections and Infestations	Not known	Increases susceptibility to, and severity of infections, opportunistic infections, recurrence of dormant tuberculosis, oesophageal candidiasis.
Blood and lymphatic system disorders	Not known	Leucocytosis
Immune system disorders	Not known	Hypersensitivity including anaphylaxis.
Endocrine disorders	Not known	Suppression of the hypothalamo-pituitary adrenal axis, cushingoid facies, impaired carbohydrate tolerance with

		increased requirement for antidiabetic therapy, manifestation of latent diabetes mellitus.
Metabolism and nutrition disorders	Not known	Sodium and water retention, hypokalaemic alkalosis, potassium loss, negative nitrogen and calcium balance, glucose intolerance and protein catabolism. Increase both high and low density lipoprotein cholesterol concentration in the blood. Increased appetite. Weight gain, obesity, hyperglycaemia, dyslipidaemia.
	Very rare	Calciphylaxis
Psychiatric disorders	Common	Irritability, depressed and labile mood, suicidal thoughts, psychotic reactions, mania, delusions, hallucinations, and aggravation of schizophrenia. behavioural disturbances, irritability, anxiety, sleep disturbances, and cognitive dysfunction including confusion, restlessness, nervousness and amnesia.
	Not known	Euphoria, psychological dependence, depression.
Nervous system disorders	Not known	Depression, insomnia, dizziness, headache, vertigo. Raised intracranial pressure with papilloedema (pseudotumor cerebri). Aggravation of epilepsy, epidural lipomatosis. vertebrobasilar stroke
Eye disorders	Not known	Glaucoma, papilloedema, posterior subcapsular cataracts, nuclear cataracts (particularly in children), exophthalmos, corneal or scleral thinning, exacerbation of ophthalmic viral or fungal disease. Severe exacerbation of bullous exudative retinal detachment; lasting visual loss in some patients with idiopathic central serous chorioretinopathy.
Ear and labyrinth disorders	Not known	Vertigo.
Cardiac disorders	Not known	Congestive heart failure in susceptible patients, hypertension, increased risk of heart failure. Increased risk of cardiovascular disease, including myocardial infarction. Bradycardia.
Vascular disorders	Not known	Thromboembolism
Gastrointestinal disorders	Not known	Dyspepsia, nausea, peptic ulceration with perforation and haemorrhage, abdominal distension, abdominal pain, diarrhoea, oesophageal ulceration, acute pancreatitis.
Skin and subcutaneous tissue disorders	Not known	Hirsutism, skin atrophy, bruising, striae, telangiectasia, acne, increased sweating, pruritus, rash, urticaria
Musculoskeletal and connective tissue disorders	Not known	Proximal myopathy, osteoporosis, vertebral and long bone fractures, avascular osteonecrosis, tendon rupture, tendinopathies (particularly of the Achilles and patellar tendons), myalgia, growth suppression in infancy, childhood and adolescence.
Renal and urinary disorders	Not known	Scleroderma renal crisis
Reproductive system and breast disorders	Not known	Menstrual irregularity, amenorrhoea.
General disorders and administration site conditions	Not known	Fatigue, malaise, impaired healing

Investigations	Not known	Increased intra-ocular pressure, may suppress reactions to skin tests.

Fertility, pregnancy and lactation

Pregnancy

The ability of corticosteroids to cross the placenta varies between individual drugs, however, 88% of prednisolone is inactivated as it crosses the placenta. Administration of corticosteroids to pregnant animals can cause abnormalities of fetal 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. However, when administered for prolonged periods or repeatedly during pregnancy, corticosteroids may increase the risk of intra-uterine growth retardation. The use of corticosteroids, including prednisolone, during pregnancy may also result in stillbirth. Hypoadrenalism may, in theory, occur in the neonate following prenatal exposure to corticosteroids but usually resolves spontaneously following birth and is rarely clinically important. Cataracts have been observed in infants born to mothers treated with long-term prednisolone during pregnancy. 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. Patients with pre-eclampsia or fluid retention require close monitoring

Breast-feeding

Corticosteroids are excreted in small amounts in breast milk. Corticosteroids distributed into breast milk may suppress growth and interfere with endogenous glucocorticoid production in nursing infants. Since adequate reproductive studies have not been performed in humans with glucocorticoids, these drugs should be administered to nursing mothers only if the benefits of therapy are judged to outweigh the potential risks to the infant. The concentration of the steroid in the milk can be between 5 and 25% of those in the serum and the two roughly parallel one another after an oral dose.There are no reports found regarding neonatal toxicity following exposure to corticosteroids during lactation, however if maternal doses >40mg/day of prednisolone is prescribed, the infant should be monitored for adrenal suppression.

5. PHARMACOLOGICAL PROPERTIES

Pharmacodynamic properties

Pharmacotherapeutic group: Corticosteroids for systemic use. Naturally occurring glucocorticoids (hydrocortisone and cortisone), which also have salt – retaining properties, are used as replacement therapy in adrenocortical deficiency states. Their synthetic analogs are primarily used for their potent anti-inflammatory effects in disorders of many organ systems.
Glucortocoids cause profound and varied metabolic effects. In addition, they modify the body's immune responses to diverse stimuli.

Pharmacokinetics Properties

Prednisolone is rapidly and apparently almost completely absorbed after oral administration; it reaches peak plasma concentrations after 1-3 hours. There is however wide inter-subject variation suggesting impaired absorption in some individuals. Plasma half – life is about 3 hours in adults and somewhat less in children, Its initial absorption, but not its overall bioavailability, is affected by food. Prednisolone has a biological half-life lasting several hours, making it suitable for alternate-day administration regimens.

Although peak plasma prednisolone levels are somewhat lower after administration of Prednisolone Tablets and absorption is delayed, total absorption and bioavailability is delayed, total absorption and bioavailability are the same as after plain prednisolone. Prednisolone shows dose dependent pharmacokinetics, with an increase in dose leading to an increase in volume of free, pharmacologically active drug. Reduced doses are necessary in patients with hypoalbuminaemia.
Prednisolone is metabolised primarily in the liver to a biologically inactive compound. Liver disease prolongs the half-life of prednisolone and, if the patient has hypoalbuminaemia, also increases the proportion of unbound drug and may thereby increase adverse effects.
Prednisolone is excreted in the urine as free and conjugated metabolites, together with small amounts of unchanged prednisolone.
Significant differences in the pharmacokinetics of prednisolone amongst menopausal women have been described. The postmenopausal women had reduced unbound clearance (30%), reduced total clearance and increased half-life of prednisolone.

6. PHARMACEUTICAL PARTICULARS

List of excipients

Starch
DCP
Lactose
Sodium Starch Glycollate
Gelatin
Methyl Paraben
Propyl Paraben
Purified Talc
Magnesium Stearate
Colloidal Anhydrous Silica

Incompatibilities

Not applicable.

Shelf life

3 years

Special precautions for storage

Keep this medicine out of the sight and reach of children.
Do not store above 30°C, protect from light.

Nature and contents of container

10 tablets in blisters (PVC /Aluminum foil).

Special precautions for disposal

Do not throw away any medicines via wastewater or household waste. Ask your pharmacist how to throw away medicines you no longer use. These measures will help protect the environment.

7. MARKETING AUTHORISATION HOLDER

Richygold International Limited (Pharmaceutical formulations)
103C Amuwo-Odofin Industrial Scheme Off Oshodi Apapa Express Way, Lagos Nigeria

8. MARKETING AUTHORISATION NUMBER(S)

None

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Not applicable

10. DATE OF REVISION OF THE TEXT

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