Package insert for use in India only

℞

Selexipag Tablets 200 mcg/ 400 mcg/ 600 mcg/ 800 mcg/ 1000 mcg/ 1200 mcg/ 1400 mcg/ 1600 mcg

Selepeg 200/400/600/800/1000/1200/1400/1600

सेलापेग २००/४००/६००/८००/१०००/१२००/१४००/१६००

To be sold by retail on the prescription of a Cardiologist/Pulmonologist only.

PRESCRIBING INFORMATION

1. GENERIC NAME

Selexipag Tablets 200 mcg
Selexipag Tablets 400 mcg
Selexipag Tablets 600 mcg
Selexipag Tablets 800 mcg
Selexipag Tablets 1000 mcg
Selexipag Tablets 1200 mcg
Selexipag Tablets 1400 mcg
Selexipag Tablets 1600 mcg

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Selexipag Tablets 200 mcg
Each film coated tablet contains
Selexipag.....200 mcg
Selexipag Tablets 400 mcg
Each film coated tablet contains
Selexipag.....400 mcg
Selexipag Tablets 600 mcg
Each film coated tablet contains
Selexipag.....600 mcg
Selexipag Tablets 800 mcg
Each film coated tablet contains
Selexipag.....800 mcg
Selexipag Tablets 1000 mcg
Each film coated tablet contains
Selexipag.....1000 mcg
Selexipag Tablets 1200 mcg
Each film coated tablet contains
Selexipag.....1200 mcg
Selexipag Tablets 1400 mcg
Each film coated tablet contains
Selexipag.....1400 mcg
Selexipag Tablets 1600 mcg
Each film coated tablet contains
Selexipag.....1600 mcg

3. DOSAGE FORM AND STRENGTH

Selexipag is available as 200 mcg, 400 mcg, 600 mcg, 800 mcg, 1000 mcg, 1200 mcg, 1400 mcg and 1600 mcg tablets.

CLINICAL PARTICULARS

4.1. Therapeutic Indications

Selexipag is indicated for the treatment of pulmonary arterial hypertension (PAH, WHO Group I) to delay disease progression and reduce the risk of hospitalization for PAH.

Effectiveness was established in a long-term study in PAH patients with WHO Functional Class II-III symptoms.

Patients had idiopathic and heritable PAH (58%), PAH associated with connective tissue disease (29%), PAH associated with congenital heart disease with repaired shunts (10%).

4.2. Posology and Method of Administration

Treatment should only be initiated and monitored by a physician experienced in the treatment of PAH.

Posology

Individualised dose titration

Each patient should be up-titrated to the highest individually tolerated dose, which can range from 200 micrograms given twice daily to 1,600 micrograms given twice daily (individualised maintenance dose).

The recommended starting dose is 200 micrograms given twice daily, approximately 12 hours apart. The dose is increased in increments of 200 micrograms given twice daily, usually at weekly intervals. At the beginning of treatment and at each up-titration step it is recommended to take the first dose in the evening. During dose titration some adverse reactions, reflecting the mode of action of Selexipag (such as headache, diarrhoea, nausea and vomiting, jaw pain, myalgia, pain in extremity, arthralgia, and flushing), may occur. They are usually transient or manageable with symptomatic treatment. However, if a patient reaches a dose that cannot be tolerated, the dose should be reduced to the previous dose level.

In patients in whom up-titration was limited by reasons other than adverse reactions reflecting the mode of action of Selexipag, a second attempt to continue up-titration to the highest individually tolerated dose up to a maximum dose of 1,600 micrograms twice daily may be considered.

Individualised maintenance dose

The highest tolerated dose reached during dose titration should be maintained. If the therapy over time is less tolerated at a given dose, symptomatic treatment and/or a dose reduction to the next lower dose should be considered.

Interruptions and discontinuations

If a dose is missed, it should be taken as soon as possible. The missed dose should not be taken if the next scheduled dose is within approximately 6 hours.

If treatment is missed for 3 days or more, Selexipag should be restarted at a lower dose and then up-titrated.

There is limited experience with abrupt discontinuation of Selexipag in patients with PAH. No evidence for acute rebound has been observed.

However, if the decision to withdraw Selexipag is taken, it should be done gradually while an alternative therapy is introduced.

Dosage Adjustment with Co-administration of Moderate CYP2C8 Inhibitors

When co-administered with moderate CYP2C8 inhibitors (e.g., clopidogrel, deferasirox and teriflunomide), reduce the dosing of Selexipag to once daily. If the therapy is not tolerated at a given dose, symptomatic treatment and/or a dose reduction to the next lower dose should be considered. Revert to twice daily dosing frequency of Selexipag when co-administration of moderate CYP2C8 inhibitor is stopped.

Method of administration

Oral use.
The film-coated tablets are to be taken orally in the morning and in the evening. To improve tolerability, it is recommended to take Selexipag with food and, at the beginning of each up-titration phase, to take the first increased dose in the evening.
The tablets should not be split, crushed or chewed, and are to be swallowed with water.
Patients who have poor vision or are blind must be instructed to get assistance from another person when taking Selexipag during the titration period.

4.3. Contraindications

- Hypersensitivity to the active substance or to any of the excipients used in the formulation.
- Severe coronary heart disease or unstable angina.
- Myocardial infarction within the last 6 months.
- Decompensated cardiac failure if not under close medical supervision.
- Severe arrhythmias.
- Cerebrovascular events (e.g., transient ischaemic attack, stroke) within the last 3 months.
- Congenital or acquired valvular defects with clinically relevant myocardial function disorders not related to pulmonary hypertension.
- Concomitant use of strong inhibitors of CYP2C8 (e.g., gemfibrozil).

4.4. Special Warnings and Precautions for Use

Hypotension

Selexipag has vasodilatory properties that may result in lowering of blood pressure. Before prescribing Selexipag, physicians should carefully consider whether patients with certain underlying conditions could be adversely affected by vasodilatory effects (e.g., patients on antihypertensive therapy or with resting hypotension, hypovolaemia, severe left ventricular outflow obstruction or autonomic dysfunction).

Hyperthyroidism

Hyperthyroidism has been observed with Selexipag. Thyroid function tests are recommended as clinically indicated in the presence of signs or symptoms of hyperthyroidism.

Pulmonary veno-occlusive disease

Cases of pulmonary oedema have been reported with vasodilators (mainly prostacyclins) when used in patients with pulmonary veno-occlusive disease. Consequently, if signs of pulmonary oedema occur when Selexipag is administered in patients with PAH, the possibility of pulmonary veno-occlusive disease should be considered. If confirmed, treatment with Selexipag should be discontinued.

Elderly (≥ 65 years)

There is limited clinical experience with selexipag in patients over the age of 75 years, therefore Selexipag should be used with caution in this population (see section 4.2).

Hepatic impairment

There is no clinical experience with selexipag in patients with severe liver impairment (Child-Pugh class C), therefore Selexipag should not be administered in these patients. The exposure to selexipag and its active metabolite is increased in subjects with moderate hepatic impairment (Child-Pugh class B; see section 5.2). In patients with moderate hepatic impairment, Selexipag should be dosed once daily (see section 4.2).

Renal impairment

In patients with severe renal impairment (eGFR < 30 mL/min/1.73 m^2), caution should be exercised during dose titration. There is no experience with Selexipag in patients undergoing dialysis (see section 5.2), therefore Selexipag should not be used in these patients.

Women of childbearing potential

Women of childbearing potential should practise effective contraception while taking selexipag.

4.5. Drug Interactions

Effect of other medicinal products on Selexipag

Selexipag is hydrolysed to its active metabolite by carboxylesterases. Selexipag and its active metabolite both undergo oxidative metabolism mainly by CYP2C8 and to a smaller extent by CYP3A4. The glucuronidation of the active metabolite is catalysed by UGT1A3 and UGT2B7. Selexipag and its active metabolite are substrates of OATP1B1 and OATP1B3. Selexipag is a weak substrate of the P-gp efflux pump. The active metabolite is a weak substrate of the breast cancer resistance protein (BCRP).

The pharmacokinetics of selexipag and its active metabolite are not affected by warfarin.

Inhibitors of CYP2C8

In the presence of 600 mg gemfibrozil, twice a day, a strong inhibitor of CYP2C8, exposure to selexipag increased approximately 2-fold, whereas exposure to the active metabolite, the major contributor to efficacy, increased approximately 11-fold. Concomitant administration of Selexipag with strong inhibitors of CYP2C8 (e.g., gemfibrozil) is contraindicated.

Concomitant administration of Selexipag with clopidogrel (loading dose of 300 mg or maintenance dose of 75 mg once a day), a moderate inhibitor of CYP2C8, had no relevant effect on the exposure to Selexipag but increased the exposure to the active metabolite approximately 2.2 and 2.7-fold following loading dose and maintenance dose, respectively. Dosing frequency of Selexipag should be reduced to once daily when co-administered with moderate CYP2C8 inhibitors (e.g., clopidogrel, deferasirox, teriflunomide). Dosing frequency of Selexipag should be reverted to twice daily when co-administration of moderate CYP2C8 inhibitor is stopped.

Inducers of CYP2C8

In the presence of 600 mg rifampicin, once a day, an inducer of CYP2C8 (and UGT enzymes), the exposure to selexipag did not change, whereas exposure to the active metabolite was reduced by half. Dose adjustment of selexipag may be required with concomitant administration of inducers of CYP2C8 (e.g., rifampicin, carbamazepine, phenytoin).

Inhibitors of UGT1A3 and UGT2B7

The effect of strong inhibitors of UGT1A3 and UGT2B7 (valproic acid, probenecid, and fluconazole) on the exposure to Selexipag and its active metabolite has not been studied. Caution is required when administering these medicinal products concomitantly with Selexipag. A potential pharmacokinetic interaction with strong inhibitors of UGT1A3 and UGT2B7 cannot be excluded.

Inhibitors and inducers of CYP3A4

In the presence of 400/100 mg lopinavir/ritonavir twice daily, a strong CYP3A4 inhibitor, exposure to Selexipag increased approximately 2-fold, whereas the exposure to the active metabolite of selexipag did not change. Given the 37-fold higher potency of the active metabolite, this effect is not clinically relevant. Since a strong inhibitor of CYP3A4 did not affect the pharmacokinetics of the active metabolite, indicating that the CYP3A4 pathway is not important in the elimination of the active metabolite, no effect of inducers of CYP3A4 on the pharmacokinetics of the active metabolite is expected.

PAH-specific therapies

In the Phase 3 placebo-controlled trial in patients with PAH, the use of selexipag in combination with both an ERA and a PDE-5 inhibitor resulted in a 30% lower exposure to the active metabolite.

Transporter inhibitors (lopinavir/ritonavir)

In the presence of 400/100 mg lopinavir/ritonavir twice daily, a strong OATP (OATP1B1 and OATP1B3) and P-gp inhibitor, exposure to selexipag increased approximately 2-fold, whereas the exposure to the active metabolite of selexipag did not change. Given that the majority of the pharmacological effect is driven by the active metabolite, this effect is not clinically relevant.

Effect of Selexipag on other medicinal products

Selexipag and its active metabolite do not inhibit or induce cytochrome P450 enzymes and transport proteins at clinically relevant concentrations.

Anticoagulants or inhibitors of platelet aggregation

Selexipag is an inhibitor of platelet aggregation *in vitro*. Selexipag did not influence the pharmacodynamic effect of warfarin on the international normalised ratio.

Midazolam

At steady state after up-titration to 1,600 µg selexipag twice a day, no clinically relevant change in exposure to midazolam, a sensitive intestinal and hepatic CYP3A4 substrate, or its metabolite, 1-hydroxymidazolam, was observed. Concomitant administration of selexipag with CYP3A4 substrates does not require dose adjustment.

Hormonal contraceptives

Specific drug-drug interaction studies with hormonal contraceptives have not been conducted. Since selexipag did not affect the exposure to the CYP3A4 substrates midazolam and R-warfarin or to the CYP2C9 substrate S-warfarin, reduced efficacy of hormonal contraceptives is not expected.

4.6. Use in Special Populations (such as pregnant women, lactating women, paediatric patients, geriatric patients etc.)

Women of childbearing potential

Women of childbearing potential should practise effective contraception while taking selexipag.

Pregnancy

There are no data from the use of selexipag in pregnant women. Animal studies do not indicate direct or indirect harmful effects with respect to reproductive toxicity. Selexipag and its main metabolite showed 20- to 80-times lower prostacyclin (IP) receptor potency in vitro for animal species used in reproductive toxicity testing compared to humans. Therefore, safety margins for potential IP receptor-mediated effects on reproduction are accordingly lower than for non-IP-related effects.

Uptravi is not recommended during pregnancy and in women of childbearing potential not using contraception.

Breast-feeding

It is unknown whether selexipag or its metabolites are excreted in human milk. In rats, Selexipag or its metabolites are excreted in milk. A risk to the suckling child cannot be excluded. Uptravi should not be used during breast-feeding.

Fertility

There are no clinical data available. In rat studies, selexipag at high doses caused transient disturbances in oestrus cycles that did not affect fertility (see section 5.3). The relevance for humans is not known.

Elderly (≥ 65 years)

No adjustment to the dose regimen is needed in elderly people. There is limited clinical experience in patients over the age of 75 years, therefore Selexipag should be used with caution in this population.

Hepatic impairment

Selexipag should not be administered in patients with severe liver impairment (Child-Pugh class C). For patients with moderate hepatic impairment (Child-Pugh class B), the starting dose of Selexipag should be 200 micrograms once daily, and increased at weekly intervals by increments of 200 micrograms given once daily until adverse reactions, reflecting the mode of action of selexipag, that cannot be tolerated or medically managed are experienced. No adjustment to the dose regimen is needed in patients with mild hepatic impairment (Child-Pugh class A).

Renal impairment

No adjustment to the dose regimen is needed in patients with mild or moderate renal impairment. No change in starting dose is required in patients with severe renal impairment (estimated glomerular filtration rate [eGFR] < 30 mL/min/1.73 m^2); dose titration should be done with caution in these patients.

Paediatric population (< 18 years)

The safety and efficacy of Selexipag in children aged 0 to less than 18 years have not yet been established. No data are available. Administration of Selexipag in the paediatric population is not recommended. Animal studies indicated an increased risk of intussusception, but the clinical relevance of these findings is unknown.

4.7. Effects on ability to drive and use machines

Selexipag has minor influence on the ability to drive and use machines. The clinical status of the patient and the adverse reaction profile of Selexipag (such as headache or hypotension) should be kept in mind when considering the patient's ability to drive and use machines.

4.8. Undesirable Effects

The most commonly reported adverse reactions are headache, diarrhoea, nausea and vomiting, jaw pain, myalgia, pain in extremity, arthralgia, and flushing. These reactions are more frequent during the up-titration phase. The majority of these reactions are of mild to moderate intensity.

System organ class	Very common (≥ 1/10)	Common (≥ 1/100 to < 1/10)	Uncommon (≥ 1/1,000 to < 1/100)
Blood and lymphatic disorders		Anaemia Haemoglobin decreased	
Endocrine disorders		Hyperthyroidism Thyroid-stimulating Hormone decreased	
Metabolism and nutrition disorders		Decreased appetite Weight decrease	
Nervous system disorders	Headache		
Cardiac disorders			Sinus tachycardia
Vascular disorders	Flushing	Hypotension	
Respiratory, thoracic and mediastinal disorders	Nasopharyngitis (of non-infectious origin)	Nasal congestion	
Gastro-intestinal disorders	Diarrhoea Vomiting Nausea	Abdominal pain	
Skin and subcutaneous tissue disorders		Rash Urticaria Erythema	
Musculoskeletal and connective tissue disorders	Jaw pain Myalgia Arthralgia Pain in extremity		
General disorders and administration site conditions		Pain	

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. To report Suspected Adverse Reactions, contact MSN Laboratories Private Limited at pharmacovigilance@msnlabs.com or through company website www.msnlabs.com->Contact us->Medical Enquiry/ to report a side effect.

4.9. Overdose

Isolated cases of overdose up to 3200 mcg were reported. Mild, transient nausea was the only reported consequence. In the event of overdose, supportive measures must be taken as required. Dialysis is unlikely to be effective because selexipag and its active metabolite are highly protein-bound.

5. PHARMACOLOGICAL PROPERTIES

5.1 Mechanism of action

Selexipag is an oral prostacyclin receptor (IP receptor) agonist that is structurally distinct from prostacyclin. Selexipag is hydrolyzed by carboxylesterase 1 to yield its active metabolite, which is approximately 37-fold as potent as selexipag. Selexipag and the active metabolite are selective for the IP receptor versus other prostanoid receptors (EP1-4, DP, FP, and TP).

5.2 Pharmacodynamic Properties

Pharmacotherapeutic group: Platelet aggregation inhibitors excl. heparin
ATC code: B01AC27

Cardiac electrophysiology:

At the maximum tolerated dose of 1600 mcg twice daily, selexipag does not prolong the QT interval to any clinically relevant extent.

Platelet aggregation:

Both selexipag and its active metabolite caused concentration-dependent inhibition of platelet aggregation in vitro with an IC50 of 5.5 µM and 0.21 µM, respectively. However, at clinically relevant concentrations, there was no effect on platelet aggregation test parameters as seen following multiple-dose administrations of selexipag in healthy subjects from 400 mcg up to 1800 mcg twice daily.

Drug interaction:

Selexipag (400 mcg twice a day) did not influence the pharmacodynamic effect of warfarin on the international normalized ratio.

Pulmonary hemodynamics:

A Phase 2 clinical study assessed hemodynamic variables after 17 weeks of treatment in patients with PAH WHO Functional Class II–III and concomitantly receiving endothelin receptor antagonists (ERAs) and/or phosphodiesterase type 5 (PDE-5) inhibitors. Patients titrating selexipag to an individually tolerated dose (200 mcg twice daily increments up to 800 mcg twice daily) (N=33) achieved a statistically-significant mean reduction in pulmonary vascular resistance of 30.3% (95% confidence interval [CI] −44.7%, −12.2%) and an increase in cardiac index (median treatment effect) of 0.41 L/min/m^2 (95% CI 0.10, 0.71) compared to placebo (N=10) [**Reference: Uptravi EU SmPC, dated: 12-Aug-2019**]

5.3 Pharmacokinetic Properties

The pharmacokinetics of selexipag and its active metabolite have been studied primarily in healthy subjects. The pharmacokinetics of selexipag and the active metabolite, both after single- and multiple-dose administration, were dose-proportional up to a single dose of 800 micrograms and multiple doses of up to 1,800 micrograms twice daily. After multiple-dose administration, steady state conditions of selexipag and the active metabolite were reached within 3 days. No accumulation in plasma, either of parent compound or active metabolite, occurred after multiple-dose administration.

In healthy subjects, inter-subject variability in exposure (area under the curve over a dosing interval) at steady state was 43% and 39% for selexipag and the active metabolite, respectively. Intra-subject variability in exposure was 24% and 19% for selexipag and the active metabolite, respectively.

Exposure to selexipag and the active metabolite at steady state in PAH patients and healthy subjects was similar. The pharmacokinetics of selexipag and the active metabolite in PAH patients were not influenced by the severity of the disease and did not change with time. [**Reference: Uptravi EU SmPC, dated: 12-Aug-2019**]

Absorption

Selexipag is rapidly absorbed and is hydrolysed by carboxylesterases to its active metabolite.
The absolute bioavailability of selexipag is approximately 49%. Upon oral administration, maximum observed plasma concentrations of selexipag and its active metabolite are reached within about 1–3 hours and 3–4 hours, respectively.
In the presence of food, the absorption of selexipag was prolonged resulting in a delayed time to peak concentration (Tmax) and ~30% lower peak plasma concentration (C_{max}). The exposure to selexipag and the active metabolite (AUC) did not significantly change in the presence of food.

Distribution

The volume of distribution of selexipag at steady state is 11.7 L.
Selexipag and its active metabolite are highly bound to plasma proteins (approximately 99% in total and to the same extent to albumin and alpha1-acid glycoprotein).

Metabolism

Selexipag is hydrolyzed to its active metabolite, (free carboxylic acid) in the liver and intestine by carboxylesterases. Oxidative metabolism, catalyzed mainly by CYP2C8 and to a smaller extent by CYP3A4, leads to the formation of hydroxylated and dealkylated products. UGT1A3 and UGT2B7 are involved in the glucuronidation of the active metabolite. Except for the active metabolite, none of the circulating metabolites in human plasma exceeds 3% of the total drug-related material.

Elimination

Elimination of selexipag is predominately via metabolism with a mean terminal half-life of 0.8-2.5 hours. The terminal half-life of the active metabolite is 6.2-13.5 hours. There is minimal accumulation of the active metabolite upon twice daily repeat administration suggesting that the effective half-life is in the range of 3-4 hours. The total body clearance of selexipag is 17.9 L/hour.

Excretion

In a study in healthy subjects with radiolabeled selexipag, approximately 93% of radioactive drug material was eliminated in feces and only 12% in urine. Neither selexipag nor its active metabolites were found in urine.

Specific Populations:

No clinically relevant effects of sex, race, age or body weight on the pharmacokinetics of selexipag and its active metabolite have been observed in healthy subjects or PAH patients.

Age:

The pharmacokinetic variables (Cmax and AUC) were similar in adult and elderly subjects up to 75 years of age. There was no effect of age on the pharmacokinetics of selexipag and the active metabolite in PAH patients.

Hepatic Impairment:

In subjects with mild (Child-Pugh class A) or moderate (Child-Pugh class B) hepatic impairment, exposure to selexipag was 2-and 4-fold that seen in healthy subjects. Exposure to the active metabolite of selexipag remained almost unchanged in subjects with mild hepatic impairment and was doubled in subjects with moderate hepatic impairment.

Renal Impairment:

A 40-70% increase in exposure (maximum plasma concentration and area under the plasma concentration-time curve) to selexipag and its active metabolite was observed in subjects with severe renal impairment (estimated glomerular filtration rate ≥ 15 mL/min/1.73 m^2 and < 30 mL/min/1.73 m^2).

Drug Interaction Studies:

In vitro studies

Selexipag is hydrolyzed to its active metabolite by carboxylesterases. Selexipag and its active metabolite both undergo oxidative metabolism mainly by CYP2C8 and to a smaller extent by CYP3A4. The glucuronidation of the active metabolite is catalyzed by UGT1A3 and UGT2B7. Selexipag and its active metabolite are substrates of OATP1B1 and OATP1B3. Selexipag is a substrate of P-gp, and the active metabolite is a substrate of the transporter of breast cancer resistance protein (BCRP).

Selexipag and its active metabolite do not inhibit or induce cytochrome P450 enzymes and transport proteins at clinically relevant concentrations.

6. NONCLINICAL PROPERTIES

6.1 Animal Toxicology or Pharmacology

In the repeated-dose toxicity studies in rodents, strong blood pressure decrease as a result of exaggerated pharmacology induced transient clinical signs and reduced food consumption and body-weight gain. In adult and juvenile dogs, intestine and bone / bone marrow were identified as the main target organs after treatment with selexipag. A delay in the closure of the femoral and/or tibial epiphyseal growth plate was observed in juvenile dogs. A no-observed-adverse-effect level was not established. In juvenile dogs, intussusception due to prostacyclin-related effects on intestinal motility was observed sporadically. Safety margins adapted for IP receptor potency for the active metabolite were 2-fold (based on total exposure) in relation to human therapeutic exposure. The finding did not occur in mouse or rat toxicity studies. Because of the species-specific sensitivity of dogs to develop intussusception, this finding is considered not relevant for adult humans.

Increased bone ossification and related changes in the bone marrow in dog studies are considered to be due to the activation of EP4 receptors in dogs. As human EP4 receptors are not activated by selexipag or its active metabolite, this effect is species-specific and, therefore, not relevant to humans. Selexipag and the active metabolite are not genotoxic.

Selexipag was not teratogenic in rats and rabbits (exposure margins above therapeutic exposure of 13-fold for selexipag and 43-fold for the active metabolite, based on total exposure). Safety margins for potential IP receptor-related effects on reproduction were 20 for fertility and 5 and 1 (based on free exposure) for embryo-foetal development in rats and rabbits, respectively, when adapted for differences in receptor potency. In the rat pre-/post-natal development study, selexipag induced no effects on maternal and pup reproductive function.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenesis: Selexipag revealed no evidence of carcinogenic potential.
Mutagenesis: Selexipag and the active metabolite are not genotoxic on the basis of the overall evidence of conducted genotoxicity studies.
Fertility: The no effect dose for effects on fertility.

7. PHARMACEUTICAL PARTICULARS

8.1 Incompatibilities

None

8.2 Packing Information

Blister of 10's count.

8.3 Shelf-life

Please see Mfg. Date/ Expiry Date printed on pack.

Do not use the product after the expiry date which is stated on the packaging. The expiry date refers to the last day of that month.

8.4 Storage and Handling Instructions

Store at controlled room temperature between 20˚C to 25˚C

8. PATIENT COUNSELLING INFORMATION

Advise the patient to read the package insert.
Inform patients:

- what to do if they miss a dose
- not to split, crush, or chew tablets.

9. DETAILS OF MANUFACTURER

MSN Laboratories Private Limited,
Formulation Division,
Unit II, Survey No. 1277 & 1319 to 1324,
Nandigama (Village and Mandal),
Rangareddy District, Pin Code: 509216,
Telangana, India.

10. DETAILS OF PERMISSION OR LICENCE NUMBER WITH DATE

5/MN/TS/2014/F/G, 26/08/2019

11. DATE OF REVISION

Jan 2022

D031471-00