For the use of only a Registered Medical Practitioner or a Hospital or a Laboratory

Not to be sold by retail without the prescription of a Registered Medical Practitioner

Prescribing Information

1. Generic Name

Pantoprazole Gastro-resistant Tablet IP
(Brand Name: P-PPi® Tablets)

2. Qualitative and Quantitative Composition

Each Gastro-resistant Tablet Contains:
Pantoprazole Sodium IP equivalent to Pantoprazole ……. 40 mg
Excipients ……. q.s.
Colours : Yellow Oxide of Iron & Titanium Dioxide IP

3. Dosage Form and Strength

Dosage Form: Tablets.
Dosage Strength: Pantoprazole 40 mg per tablet.

4. Clinical Particulars

4.1 Therapeutic Indication

P-PPi Tablets are indicated for the treatment of following conditions where a reduction of gastric acid secretion is required:

- Duodenal ulcer.
- Gastric ulcer.
- Erosive esophagitis associated with gastro-esophageal reflux disease (GERD).
- *Helicobacter pylori* (*H. pylori*)-associated ulcers (in combination with appropriate antibiotic therapy).
- Pathological hypersecretory conditions including Zollinger-Ellison syndrome.

4.2Posology and Method of Administration

For oral administration.

Adults.

1. **Duodenal Ulcer:** The recommended dose is 40 mg given once daily in the morning. Healing usually occurs within 2 weeks. For patients not healed after this initial course of therapy, an additional course of 2 weeks is recommended.
2. **Gastric Ulcer:** The recommended dose is 40 mg given once daily in the morning. Healing usually occurs within 4 weeks. For patients not healed after this initial course of therapy, an additional course of 4 weeks is recommended.
3. **Erosive Esophagitis Associated with GERD:** The recommended dose is 40 mg once daily for up to 8 weeks. The dose may be doubled (80 mg/day) especially when there

has been no response to other treatment. For adult patients who have not healed after 8 weeks of treatment, an additional course of 8 weeks may be considered.

4. ***Helicobacter Pylori*-Associated Ulcers:** Depending on the resistance pattern, the following combinations can be recommended for the eradication of *H. pylori*. The recommended dose of pantoprazole for *H. pylori* eradication in combination with appropriate antibiotic therapy is as follows:
   A. Pantoprazole 40 mg + Clarithromycin 500 mg + Metronidazole 400 mg / 500 mg or Tinidazole 500 mg: Twice daily. Or,
   B. Pantoprazole 40 mg + Amoxicillin 1000 mg + Clarithromycin 500 mg: Twice daily. Or,
   C. Pantoprazole 40 mg + Amoxicillin 1000 mg + Metronidazole 400 mg / 500 mg or Tinidazole 500 mg: Twice daily.

   In combination therapy for eradication of *H. pylori* infection, the second pantoprazole tablet should be taken 1 hour before the evening meal. The combination therapy is recommended for 7 days in general and can be prolonged for a further 7 days to a total duration of up to 2 weeks.
5. **Pathological Hypersecretory Conditions Including Zollinger-Ellison Syndrome:** The recommended dose is 40 mg twice daily. Dosage regimens should be adjusted to individual patient needs and should continue for as long as clinically indicated. Doses up to 240 mg daily have been administered (temporary increase of the dose). With doses above 80 mg daily, the dose should be divided and given twice daily.

Children (5 years and older).

1. **Short-Term Treatment of Erosive Esophagitis Associated With GERD.**
   - **≥ 15 kg to < 40 kg:** 20 mg once daily for up to 8 weeks.
   - **≥ 40 kg:** 40 mg once daily for up to 8 weeks.

P-PPi Tablets may be administered with or without food. P-PPi Tablets should be swallowed whole with water not to be cut, crush or chew.
Or, as prescribed by the physician.

4.3Contraindications

P-PPi Tablets are contraindicated in the following:

- Patients with known hypersensitivity to pantoprazole or to any substituted benzimidazole or to any component of the formulation. Hypersensitivity reactions may include anaphylaxis, anaphylactic shock, angioedema, bronchospasm, acute interstitial nephritis, and urticaria.
- In patients receiving rilpivirine-containing products.

4.4Special Warnings and Precautions for Use

Presence of Gastric Malignancy: In adults, symptomatic response to therapy with pantoprazole does not preclude the presence of gastric malignancy. Consider additional

follow-up and diagnostic testing in adult patients who have a suboptimal response or an early symptomatic relapse after completing treatment with a pantoprazole.

Acute Interstitial Nephritis: Acute interstitial nephritis has been observed in patients taking pantoprazole. Acute interstitial nephritis may occur at any point during pantoprazole therapy and is generally attributed to an idiopathic hypersensitivity reaction. Discontinue pantoprazole if acute interstitial nephritis develops.

***Clostridium Difficile*-Associated Diarrhea (CDAD):** Published observational studies suggest that pantoprazole therapy may be associated with an increased risk of CDAD, especially in hospitalized patients. This diagnosis should be considered for diarrhea that does not improve.

Risk of Bone Fractures: Proton pump inhibitors (PPIs), especially if used in high doses and over long durations (> 1 year), may modestly increase the risk of hip, wrist and spine fracture, predominantly in the elderly or in presence of other recognized risk factors. Patients should use the lowest dose and shortest duration of PPI therapy appropriate to the condition being treated. Patients at risk of osteoporosis should receive care according to current clinical guidelines and they should have an adequate intake of vitamin D and calcium.

Cutaneous and Systemic Lupus Erythematosus: Cutaneous lupus erythematosus (CLE) and systemic lupus erythematosus (SLE) have been reported in patients taking PPIs, including pantoprazole. These events have occurred as both new onset and an exacerbation of existing autoimmune disease. The most common form of CLE reported in patients treated with PPIs was subacute CLE (SCLE) and occurred within weeks to years after continuous drug therapy in patients ranging from infants to the elderly. The occurrence of CLE with previous PPI treatment may increase the risk of CLE with other PPIs. Systemic lupus erythematosus (SLE) is less commonly reported than CLE in patients receiving PPIs. The majority of patients presented with rash. If lesions occur, especially in sun-exposed areas of the skin, and if accompanied by arthralgia, the patient should seek medical help promptly and pantoprazole therapy should be stopped immediately. Most patients improve with discontinuation of the PPI alone in 4 to 12 weeks.

Cyanocobalamin (Vitamin B_{12}) Deficiency: Generally, daily treatment with any acid-suppressing medication over a long period of time (e.g., longer than 3 years) may lead to malabsorption of vitamin B_{12} caused by hypo- or achlorhydria. Rare reports of cyanocobalamin deficiency occurring with acid-suppressing therapy have been reported in the literature. This diagnosis should be considered if clinical symptoms consistent with cyanocobalamin deficiency are observed.

Hypomagnesemia: Hypomagnesemia, symptomatic and asymptomatic, has been reported rarely in patients treated with PPIs for at least 3 months, in most cases after a year of therapy. Serious adverse events include tetany, arrhythmias, and seizures. In most patients, treatment of hypomagnesemia required magnesium replacement and discontinuation of the PPI. For patients expected to be on prolonged treatment or who take PPIs with medications such as digoxin or drugs that may cause hypomagnesemia (e.g., diuretics), monitoring of magnesium levels prior to initiation of PPI treatment and periodically thereafter should be considered.

4.5Drug Interactions

Antiretroviral Drugs: The effect of PPIs on antiretroviral drugs is variable. The clinical importance and the mechanisms behind these interactions are not always known.

- Decreased exposure of some antiretroviral drugs (e.g., rilpivirine atazanavir, and nelfinavir) when used concomitantly with pantoprazole may reduce antiviral effect and promote the development of drug resistance. Concomitant use of rilpivirine-containing products with pantoprazole is contraindicated. Also, concomitant use of nelfinavir with pantoprazole should be avoided.
- Increased exposure of other antiretroviral drugs (e.g., saquinavir) when used concomitantly with pantoprazole may increase toxicity.
- There are other antiretroviral drugs which do not result in clinically relevant interactions with pantoprazole.

Coumarin Anticoagulants/Warfarin: There have been post-marketing reports of increased international normalized ratio (INR) and prothrombin time in patients receiving PPIs, including pantoprazole, and warfarin concomitantly. Increases in INR and prothrombin time may lead to abnormal bleeding and even death. Monitor INR and prothrombin time and adjust the dose of warfarin, if needed, to maintain the target INR range.

Clopidogrel: Concomitant administration of pantoprazole and clopidogrel in healthy subjects had no clinically important effect on exposure to the active metabolite of clopidogrel or clopidogrel-induced platelet inhibition. No dose adjustment of clopidogrel is necessary when administered with an approved dose of pantoprazole.

Methotrexate: Literature suggests that concomitant use of PPIs with methotrexate (primarily at high dose) may elevate and prolong serum levels of methotrexate and/or its metabolite, possibly leading to methotrexate toxicities. A temporary withdrawal of pantoprazole therapy may be considered in some patients receiving high-dose of methotrexate.

Drugs for Which Gastric pH Can Affect Bioavailability (iron salts, erlotinib, dasatinib, nilotinib, mycophenolate mofetil, and ketoconazole): Pantoprazole causes long-lasting inhibition of gastric acid secretion. Therefore, pantoprazole may reduce absorption of other drugs where gastric pH is an important determinant of their bioavailability.

Mycophenolate Mofetil (MMF): Co-administration of pantoprazole sodium in healthy subjects and in transplant patients receiving MMF has been reported to reduce the exposure to the active metabolite, mycophenolic acid (MPA), possibly due to a decrease in MMF solubility at an increased gastric pH. The clinical relevance of reduced MPA exposure on organ rejection has not been established in transplant patients receiving pantoprazole therapy and MMF. Use pantoprazole with caution in transplant patients receiving MMF.

<u>Drug/Laboratory Tests Interactions</u>

False Positive Urine Tests for THC: There have been reports of false positive urine screening tests for tetrahydrocannabinol (THC) in patients receiving PPIs, including pantoprazole. An alternative confirmatory method should be considered to verify positive results.

Increased Chromogranin A (CgA) Lvel: Increase in CgA may interfere with investigations for neuroendocrine tumours. To avoid this interference, pantoprazole treatment should be

stopped for at least 5 days before CgA measurements. If CgA and gastrin levels have not returned to reference range after initial measurement, measurements should be repeated 14 days after cessation of pantoprazole treatment.

4.6Use in Special Populations

Pregnant Women

Pregnancy Category C. Reproduction studies have been performed in rats at oral doses up to 88- times the recommended human dose and in rabbits at oral doses up to 16-times the recommended human dose and have revealed no evidence of impaired fertility or harm to the fetus due to pantoprazole. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, pantoprazole should be used during pregnancy only if the potential benefit justifies the possible risk to the fetus.

Lactating Women

Animal studies have shown that pantoprazole and its metabolites are excreted in the milk. Excretion of pantoprazole in human milk has also been reported (insufficient information). However, the clinical relevance of this finding is not known. Many drugs which are excreted in human milk have a potential for serious adverse reactions in nursing infants. Similarly, with pantoprazole, a risk to the newborns/infants cannot be excluded. Therefore, a decision should be made whether to discontinue nursing or to discontinue/abstain from pantoprazole therapy, taking into account the benefit of the drug to the mother.

Paediatric Patients

In children 5 years and older, for the short-term treatment of erosive esophagitis associated with GERD, pantoprazole can be administered as recommended. For dosage, please refer 'Posology and Method of Administration' section. Except for use in erosive esophagitis associated with GERD, P-PPi Tablets are not recommended for use in children and adolescents below 18 years of age due to insufficient data on safety and efficacy.

Geriatric Patients

Elderly patients may be given the same dose as recommended for adults. No dose adjustment is necessary in these patients.

Renal Impairment Patients

Dosage modification is not necessary when pantoprazole is administered to patients with impaired renal function (including dialysis patients).

Hepatic Impairment Patients

In patients with severe liver impairment, a daily dose of pantoprazole 20 mg should not be exceeded. P-PPi Tablets contains 40 mg of pantoprazole; thus, P-PPi Tablets are not recommended in patients with severe hepatic impairment. However, P-PPi Tablets can be administered in patients with mild to moderate hepatic impairment. The liver enzymes should

be monitored regularly, particularly on long-term use of pantoprazole. In the case of a rise in liver enzymes, the treatment should be discontinued.

4.7Effect on Ability to Drive and Use Machines

Pantoprazole has no or negligible influence on the ability to drive and use machines. However, adverse reactions such as dizziness and visual disturbances may occur. If affected, patients should not drive or use machines.

4.8Undesirable Effects

Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug may not reflect the rates observed in clinical practice.

Common adverse reactions reported with pantoprazole therapy in clinical trials with frequency > 2% include: Headache, diarrhoea, nausea, abdominal pain, vomiting, flatulence, dizziness, and arthralgia.

Additional adverse reactions reported in clinical trials with a frequency of ≤ 2% include:

Body as a Whole: Allergic reaction, pyrexia, photosensitivity reaction, facial edema.

Gastrointestinal: Constipation, dry mouth, hepatitis.

Hematologic: Leukopenia, thrombocytopenia.

Metabolic/Nutritional: Elevated creatine kinase (CK), generalized edema, elevated triglycerides, elevated liver enzymes.

Musculoskeletal: Myalgia.

Nervous: Depression, vertigo.

Skin and Appendages: Urticaria, rash, pruritus.

Special Senses: Blurred vision.

Post-Marketing Experience

Acute kidney injury as an adverse drug reaction reported with the use of proton pump inhibitors.

The following adverse reactions have been identified during post-approval use of pantoprazole. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.

General Disorders and Administration Conditions: Asthenia, fatigue, malaise.

Hematologic: Pancytopenia, agranulocytosis.

Hepatobiliary Disorders: Hepatocellular damage leading to jaundice and hepatic failure.

Immune System Disorders: Anaphylaxis (including anaphylactic shock), SLE.

Infections and Infestations: *Clostridium difficile*-associated diarrhea.

Investigations: Weight changes.

Metabolism and Nutritional Disorders: Hyponatremia, hypomagnesemia.

Musculoskeletal Disorders: Rhabdomyolysis, bone fracture.

Nervous System: Ageusia, dysgeusia.

Psychiatric Disorders: Hallucination, confusion, insomnia, somnolence.

Renal and Urinary Disorders: Interstitial nephritis.
Skin and Subcutaneous Tissue Disorders: Severe dermatologic reactions, including erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis (TEN), angioedema (Quincke's edema) and CLE.

4.9Overdose

Experience in patients taking very high doses of pantoprazole (> 240 mg) is limited. Pantoprazole is not removed by hemodialysis. In case of overdose, treatment should be symptomatic and supportive.

5. Pharmacological Properties

5.1 Mechanism of Action

Pantoprazole is a proton pump inhibitor (PPI) class of antisecretory agent. Pantoprazole is a lipophilic weak base that crosses the parietal cell membrane and enters the acidic parietal cell canaliculus where it becomes protonated, producing the active metabolite sulfenamide. Sulfenamide forms an irreversible covalent bond with two sites of the H+/K+-ATPase enzyme located on the gastric parietal cell. Thus, pantoprazole suppress the final step in gastric acid (hydrochloric acid – HCl) production by covalently binding to the H+/K+-ATPase enzyme (also called as proton pump) system at the secretory surface of the gastric parietal cell. This effect leads to inhibition of both basal and stimulated gastric acid secretion, irrespective of the stimulus. The binding to the H+/K+-ATPase results in duration of antisecretory effect that persists longer than 24 hours.

5.2Pharmacodynamic Properties

With a single oral dose of 20 to 80 mg of pantoprazole, a dose-dependent decrease in gastric acid secretion occurs. Following the initial oral dose of 40 mg pantoprazole, a 51% mean inhibition was achieved by 2.5 hours. With once-a-day dosing for 7 days, the mean inhibition was increased to 85%. Acid secretion had returned to normal within a week after the last dose of pantoprazole; there was no evidence of rebound hypersecretion.
Pantoprazole reduces acidity in the stomach and thereby increases gastrin in proportion to the reduction in acidity. The increase in gastrin is reversible. Since pantoprazole binds to the enzyme distal to the receptor level, it can inhibit hydrochloric acid secretion independently of stimulation by other substances (acetylcholine, histamine, and gastrin).
The fasting gastrin values increase under pantoprazole. On short-term use, in most cases they do not exceed the upper limit of normal. During long-term treatment, gastrin levels double in most cases.

5.3Pharmacokinetic Properties

Absorption: Like other PPIs, pantoprazole is an acid-labile drug and therefore, administered orally in the form of gastro-resistant tablets. Absorption of pantoprazole, therefore, begins in the intestine only after the tablet leaves the stomach.
After administration of a single or multiple oral doses of pantoprazole 40 mg, the peak plasma concentration of pantoprazole was achieved in approximately 2.5 hours, and C_{max} was

2.5 mcg/ml. Peak serum concentration (C_{max}) and area under the serum concentration time curve (AUC) increases in a dose-dependent manner (with dose range from 10 to 80 mg). Pantoprazole does not accumulate, and its pharmacokinetics is unaltered with multiple daily dosing. Pantoprazole undergoes little first-pass metabolism, resulting in an absolute bioavailability of approximately 77%.

Effect of Antacid / Food: Pantoprazole absorption is not affected by concomitant administration of antacids. Administration of P-PPi Tablets with food may delay its absorption up to 2 hours or longer; however, the C_{max} and the extent of pantoprazole absorption (AUC) are not altered. Thus, pantoprazole may be taken without regard to timing of meals.

Distribution: The apparent volume of distribution of pantoprazole is approximately 11 to 23.6 liters, distributing mainly in extracellular fluid. The plasma protein binding of pantoprazole is about 98%, primarily to albumin.

Metabolism: Pantoprazole is extensively metabolized in the liver through the cytochrome P450 (CYP) system. The main metabolic pathway is demethylation, by CYP2C19, with subsequent sulfation; other metabolic pathways include oxidation by CYP3A4. There is no evidence that any of the pantoprazole metabolites have significant pharmacologic activity.

Excretion: Renal elimination represents the major route of excretion (about 80 %) for the metabolites of pantoprazole, the rest is excreted in the faeces. There is no renal excretion of unchanged pantoprazole. The main metabolite in both the serum and urine is desmethyl-pantoprazole which is conjugated with sulphate. Following oral administration, the serum concentration of pantoprazole declines biexponentially, with a terminal elimination half-life of approximately one hour.

6. Nonclinical Properties

6.1 Animal Toxicology

Carcinogenesis: In a 24-month carcinogenicity study, Sprague-Dawley rats were treated orally with pantoprazole doses of 0.5 to 200 mg/kg/day, about 0.1 to 40 times the exposure on a body surface area basis of a 50 kg person dosed with 40 mg/day. In the gastric fundus, treatment with 0.5 to 200 mg/kg/day produced enterochromaffin-like (ECL) cell hyperplasia and benign and malignant neuroendocrine cell tumors in a dose-related manner. In the forestomach, treatment with 50 and 200 mg/kg/day (about 10 and 40 times the recommended human dose on a body surface area basis) produced benign squamous cell papillomas and malignant squamous cell carcinomas. Rare gastrointestinal tumors associated with pantoprazole treatment included an adenocarcinoma of the duodenum with 50 mg/kg/day and benign polyps and adenocarcinomas of the gastric fundus with 200 mg/kg/day. In the liver, treatment with 0.5 to 200 mg/kg/day produced dose-related increases in the incidences of hepatocellular adenomas and carcinomas. In the thyroid gland, treatment with 200 mg/kg/day produced increased incidences of follicular cell adenomas and carcinomas for both male and female rats.

In a 24-month carcinogenicity study, B6C3F1 mice were treated orally with doses of 5 to 150 mg/kg/day of pantoprazole, 0.5 to 15 times the recommended human dose based on body surface area. In the liver, treatment with 150 mg/kg/day produced increased incidences of

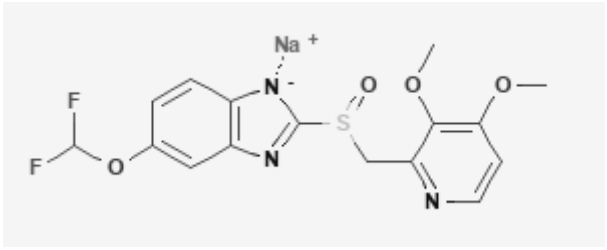
hepatocellular adenomas and carcinomas in female mice. Treatment with 5 to 150 mg/kg/day also produced gastric-fundic ECL cell hyperplasia.
A 26-week p53 +/-transgenic mouse carcinogenicity study was not positive.
Mutagenesis: Pantoprazole was positive in the *in vitro* human lymphocyte chromosomal aberration assays, in one of two mouse micronucleus tests for clastogenic effects, and in the *in vitro* Chinese hamster ovarian cell/HGPRT forward mutation assay for mutagenic effects. Equivocal results were observed in the *in vivo* rat liver DNA covalent binding assay. Pantoprazole was negative in the *in vitro* Ames mutation assay, the *in vitro* unscheduled DNA synthesis (UDS) assay with rat hepatocytes, the *in vitro* AS52/GPT mammalian cell-forward gene mutation assay, the *in vitro* thymidine kinase mutation test with mouse lymphoma L5178Y cells, and the *in vivo* rat bone marrow cell chromosomal aberration assay.
Impairment of Fertility: There were no effects on fertility or reproductive performance when pantoprazole was given at oral doses up to 500 mg/kg/day in male rats (98 times the recommended human dose based on body surface area) and 450 mg/kg/day in female rats (88 times the recommended human dose based on body surface area).
Teratogenicity: Reproduction studies have been performed in rats at oral pantoprazole doses up to 450 mg/kg/day (about 88 times the recommended human dose based on body surface area) and in rabbits at oral doses up to 40 mg/kg/day (about 16 times the recommended human dose based on body surface area) with administration of pantoprazole sodium during organogenesis in pregnant animals. The studies have revealed no evidence of impaired fertility or harm to the fetus due to pantoprazole.

7. Description

P-PPi Tablets are Yellow coloured, round, biconvex, plain on both sides & gastro-resistant tablets.

P-PPi Tablets contains 40 mg of pantoprazole (in a gastro-resistant form) for oral administration.

Pantoprazole sodium is the sodium salt form of a substituted benzimidazole with proton pump inhibitor action which leads to reduction of gastric acid production.
Pantoprazole sodium is a white to off-white crystalline powder. Pantoprazole sodium is freely soluble in water, very slightly soluble in phosphate buffer at pH 7.4, and practically insoluble in n-hexane.
Molecular Weight: 405.4 g/mol.
Molecular Formula: C16H14F2N3NaO4S.
Chemical Name: Sodium; 5-(difluoromethoxy)-2-[(3,4-dimethoxypyridin-2-yl)methylsulfinyl]benzimidazol-1-ide .
Structural Formula:



Inactive ingredients (excipients) of P-PPi Tablets contain Sodium Carbonate, Sodium Starch Glycolate, Sodium Lauryl Sulphate, Colloidal Silicon Dioxide, Starch (Maize), Hydroxy Propyl Methyl Cellulose, Polysorbate 80, Magnesium Stearate, Talcum, Crospovidone, Poly Ethylene Glycol 6000, Procoat ECM Aqua & Colour Iron Oxide Yellow.

8. Pharmaceutical Particulars

8.1 Incompatibilities

None known.

8.2Shelf-life

24 Months

8.3Packaging Information

15 tablets per alu-alu blister.

8.4Storage and Handling Instructions

Store protected from light and moisture, at a temperature not exceeding 30°C.
Keep out of reach of children.

9. Patient Counseling Information

Instructions to Patients

- Take P-PPi Tablets exactly as prescribed by your doctor, at the lowest dose possible and for the shortest time needed. Do not change the dose or stop therapy without consulting to your doctor.
- Swallow P-PPi Tablets whole with water, especially in the morning. Do not split, chew, or crush tablets.
- If you miss a dose of P-PPi Tablets, take it as soon as possible. If it is almost time for your next dose, do not take the missed dose. Take the next dose at your regular time. Do not take 2 doses at the same time to make up for the missed dose.
- This medicine is not recommended for use in children under 5 years of age.
- Instruct patients not to take this medicine during pregnancy and lactation unless advised by their healthcare professionals.

- Instruct patients not to share this medication with other people even though symptoms are similar. It may harm them.
- Tell your doctor about all the medicines you take, including prescription and over-the-counter medicines, vitamins and herbal supplements. P-PPi Tablets and certain other medicines can interact with each other causing serious side effects.

10. Details of Manufacturer

Pure & Cure Healthcare Pvt. Ltd.
(A subsidiary of Akums Drugs & Pharmaceuticals Ltd.)
Plot No. 26A, 27-30, Sector-8A, I.I.E., SIDCUL, Ranipur,
Haridwar – 249 403, Uttarakhand.

11. Details of Permission or License Number with Date

Mfg. Lic. No. : 31/UA/2013, Date of Product Permission: 03/11/2014

12. Date of Revision

February 2023

Marketed by:
EXCEL BLUE CROSS || EXCEL
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