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

Rosuvastatin Tablets IP 5 mg/10 mg
(Brand Name: ROVASTAT® 5 mg / 10 mg Tablets)

2. Qualitative and Quantitative Composition

Each film coated tablet contains:
Rosuvastatin Calcium IP equivalent to Rosuvastatin ……. 5 mg / 10 mg.
Excipients ……. q.s.
Colours: Ferric Oxide USP – NF (Red) and Titanium Dioxide IP.

Each film coated tablet contains:
Rosuvastatin Calcium IP equivalent to Rosuvastatin ……. 10 mg.
Excipients ……. q.s.
Colour: Titanium Dioxide IP.

3. Dosage Form and Strength

Dosage Form: Tablets.
Dosage Strength: Rosuvastatin 5 mg and 10 mg per tablet.

4. Clinical Particulars

4.1 Therapeutic Indication

ROVASTAT Tablets are indicated as an adjunctive therapy to diet in the treatment of lipid (cholesterol and triglycerides) disorders. Rosuvastatin normalizes serum lipid levels i.e., it reduces Total-C, LDL-C, ApoB, non HDL-C, TGs and increases HDL-C.
[Total-C= total cholesterol; LDL-C = low density lipoprotein cholesterol; HDL-C= high density lipoprotein cholesterol; TGs= triglycerides; ApoB= apolipoprotein B].
Lipid disorders are important risk factors for coronary artery diseases (CAD), stroke, and peripheral arterial diseases.
ROVASTAT Tablets are effective in the management of following conditions:

- Hyperlipidemia and mixed dyslipidemia in adults.
- Heterozygous familial hypercholesterolemia (in children between 10 to 17 years of age): In adolescent boys and girls, who are at least one year post-menarche with LDL-C > 160

mg/dl and there is a positive family history of premature cardiovascular disease (CVD) or two or more other CVD risk factors.

- Hypertriglyceridemia in adults.
- Primary dysbetalipoproteinemia (type III hyperlipoproteinemia).
- Homozygous familial hypercholesterolemia.
- Slowing of the progression of atherosclerosis in adults.
- Primary prevention of cardiovascular diseases: To reduce risk of stroke and myocardial infarction (MI) in individuals with an increased risk for first cardiovascular event [risk factors include age, hypertension, elevated high-sensitivity C-reactive protein (hsCRP), low HDL-C, smoking, or a family history of premature coronary heart disease].

4.2Posology and Method of Administration

For oral administration.

Adults

The dose range for rosuvastatin in adults is 5 to 40 mg orally once daily. The usual starting dose is 10 to 20 mg once daily. The usual starting dose in adult patients with homozygous familial hypercholesterolemia is 20 mg once daily.

The maximum dose i.e., 40 mg should be used only for those patients who have not achieved their LDL-C goal utilizing the 20 mg dose.

When initiating rosuvastatin therapy or switching from another HMG-CoA (3-hydroxy-3-methylglutaryl coenzyme A) reductase inhibitor therapy, the appropriate starting dose should be utilized. Then, dose should be titrated according to the patient's response to therapy and individualized goal of therapy.

After initiation or upon titration of rosuvastatin, lipid levels should be analyzed within 2 to 4 weeks and the dosage adjusted accordingly.

Paediatric Dosing

In heterozygous familial hypercholesterolemia, the recommended dose range of rosuvastatin is as follows:

- In children between 6 to 9 years of age: 5 to 10 mg once daily.
- In children between 10 to 17 years of age: 5 to 20 mg once daily.

In homozygous familial hypercholesterolemia, the recommended dose of rosuvastatin is:

- In children between 7 to 17 years of age: 20 mg orally once daily.

ROVASTAT Tablets can be administered as a single dose at any time of day, with or without food. The tablet should be swallowed whole. ROVASTAT Tablets can be combined with other lipid-lowering agents.

Or, as prescribed by the physician.

4.3Contraindications

ROVASTAT Tablets are contraindicated in the following conditions:

- Known hypersensitivity to rosuvastatin or to any component of this product.
- Patients with active liver disease, which may include unexplained persistent elevations of hepatic transaminase levels.
- Pregnancy.
- Lactation.
- Severe renal impairment (creatinine clearance <30 ml/min).
- Patients with myopathy.
- Patients receiving concomitant cyclosporine.

4.4Special Warnings and Precautions for Use

Skeletal Muscle Effects: Cases of myopathy and rhabdomyolysis with acute renal failure secondary to myoglobinuria have been reported with HMG-CoA reductase inhibitors. These risks can occur at any dose, but are increased at the highest dose (40 mg). Rosuvastatin should be prescribed with caution in patients with predisposing factors for myopathy (e.g., age ≥ 65 years, inadequately treated hypothyroidism, renal impairment).

Rosuvastatin therapy should be discontinued if markedly elevated creatine kinase levels occur or myopathy is diagnosed or suspected. Rosuvastatin therapy should also be temporarily withheld in any patient with an acute, serious condition suggestive of myopathy or predisposing to the development of renal failure secondary to rhabdomyolysis (e.g., sepsis, hypotension, dehydration, major surgery, trauma, severe metabolic, endocrine, and electrolyte disorders, or uncontrolled seizures).

There have been rare reports of immune-mediated necrotizing myopathy (IMNM), an autoimmune myopathy, associated with statin use. IMNM is characterized by proximal muscle weakness and elevated serum creatine kinase, which persist despite discontinuation of statin treatment; muscle biopsy showing necrotizing myopathy without significant inflammation; improvement with immunosuppressive agents.

All patients should be advised to promptly report symptoms of myopathy to their physician. These symptoms include unexplained muscle pain, tenderness, or weakness, particularly if accompanied by malaise or fever or if muscle signs and symptoms persist after discontinuing rosuvastatin.

Myopathy with Concomitant Therapy: The risk of myopathy during treatment with rosuvastatin may be increased with concurrent administration of some other lipid-lowering therapies (fibrates or niacin), gemfibrozil, cyclosporine, atazanavir/ritonavir, lopinavir/ritonavir, or simeprevir. Cases of myopathy, including rhabdomyolysis, have been reported with HMG-CoA reductase inhibitors, including rosuvastatin, when co-administered with colchicine, and caution should be exercised when prescribing rosuvastatin with colchicine.

Liver Effects: There have been rare post-marketing reports of fatal and non-fatal hepatic failure in patients taking statins, including rosuvastatin. If serious liver injury with clinical symptoms

and/or hyperbilirubinemia or jaundice occurs during treatment with rosuvastatin, promptly interrupt therapy. If an alternate etiology is not found, do not restart rosuvastatin therapy.

As with other HMG-CoA reductase inhibitors, rosuvastatin should be used with caution in patients who consume excessive quantities of alcohol and/or have a history of liver disease. It is recommended that liver enzyme tests be performed before the initiation of rosuvastatin therapy, and if signs or symptoms of liver injury occur. All patients treated with rosuvastatin should be advised to promptly report any symptoms that may indicate liver injury, including fatigue, anorexia, right upper abdominal discomfort, dark urine or jaundice.

Liver Enzyme Abnormalities: It is recommended that liver enzyme tests be performed before initiation of therapy and at 12 weeks following rosuvastatin therapy and periodically (e.g., semiannually) thereafter. Increases in serum transaminases [AST (SGOT) or ALT (SGPT)] have been reported with HMG-CoA reductase inhibitors, including rosuvastatin. In most cases, the elevations were transient and resolved or improved on continued therapy or after a brief interruption in therapy.

Proteinuria and Hematuria: Proteinuria (dipstick-positive) has been observed in patients treated with higher doses of rosuvastatin, in particular 40 mg, where it was transient or intermittent in most cases. Proteinuria has not been shown to be predictive of acute or progressive renal disease. Also, microscopic hematuria has been observed among rosuvastatin-treated patients. Although the clinical significance of these findings is unknown, a dose reduction and an assessment of renal function should be considered for patients on rosuvastatin therapy with unexplained persistent proteinuria and/or hematuria during routine urinalysis testing.

New Onset Diabetes: Increases in HbA1c and fasting serum glucose levels have been reported with HMG-CoA reductase inhibitors, including rosuvastatin. Based on clinical trial data with rosuvastatin, in some instances these increases may exceed the threshold for the diagnosis of diabetes mellitus.

Endocrine Effects: Although clinical studies have shown that rosuvastatin alone does not reduce basal plasma cortisol concentration or impair adrenal reserve, caution should be exercised if rosuvastatin is administered concomitantly with drugs (such as ketoconazole, spironolactone, and cimetidine) that may decrease the levels or activity of endogenous steroid hormones.

Lactose Intolerance: Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take rosuvastatin.

Interstitial Lung Disease: Exceptional cases of interstitial lung disease have been reported with some statins, especially with long term therapy. Presenting features can include dyspnoea, non-productive cough and deterioration in general health (fatigue, weight loss and fever). If it is suspected a patient has developed interstitial lung disease, statin therapy should be discontinued.

4.5Drug Interactions

Cyclosporine: During concomitant treatment with rosuvastatin and cyclosporine, rosuvastatin AUC values were 7-fold higher than those observed in healthy volunteers. Therefore, in patients

taking cyclosporine, the dose of rosuvastatin should not exceed 5 mg once daily. Concomitant administration did not affect plasma concentrations of cyclosporine.

Gemfibrozil: Gemfibrozil significantly increased rosuvastatin exposure (AUC). Due to an observed increased risk of myopathy/rhabdomyolysis, concurrent administration of rosuvastatin with gemfibrozil should be avoided. If used together, the dose of rosuvastatin should not exceed 10 mg once daily.

Protease Inhibitors: Co-administration of rosuvastatin with certain protease inhibitors has differing effects on rosuvastatin exposure. Simeprevir, which is a hepatitis C virus (HCV) protease inhibitor, or combinations of atazanavir/ritonavir or lopinavir/ritonavir, which are HIV-1 protease inhibitors, increase rosuvastatin exposure (AUC) up to 3-fold. For these protease inhibitors, the dose of rosuvastatin should not exceed 10 mg once daily.

The combinations of fosamprenavir/ritonavir or tipranavir/ritonavir, which are HIV-1 protease inhibitors, produce little or no change in rosuvastatin exposure. Caution should be exercised when rosuvastatin is co-administered with protease inhibitors.

Coumarin Anticoagulants: Rosuvastatin significantly increased International Normalised Ratio (INR) in patients receiving coumarin anticoagulants. Therefore, caution should be exercised when coumarin anticoagulants are given in conjunction with rosuvastatin. In patients taking coumarin anticoagulants and rosuvastatin concomitantly, INR should be determined before starting rosuvastatin and frequently enough during early therapy to ensure that no significant alteration of INR occurs.

Niacin: The risk of skeletal muscle effects may be enhanced when rosuvastatin is used in combination with lipid modifying doses (≥ 1 g/day) of niacin. Caution need to be exercised when co-prescribing rosuvastatin with niacin.

Fenofibrate: When rosuvastatin was co-administered with fenofibrate, no clinically significant increase in the AUC of rosuvastatin or fenofibrate was observed. Because it is known that the risk of myopathy during treatment with HMG-CoA reductase inhibitors is increased with concomitant use of fenofibrates, caution should be exercised when prescribing fenofibrates with rosuvastatin. Rosuvastatin 40 mg dose is contraindicated with concomitant use of a fibrate.

Colchicine: Cases of myopathy, including rhabdomyolysis, have been reported with HMG-CoA reductase inhibitors, including rosuvastatin, when co-administered with colchicine. Thus, caution should be exercised when prescribing rosuvastatin with colchicine.

Antacid: The simultaneous dosing of rosuvastatin with an antacid suspension containing aluminium and magnesium hydroxide resulted in a decrease in rosuvastatin plasma concentration of approximately 50%. This effect was mitigated when the antacid was given 2 hours after rosuvastatin. The clinical relevance of this interaction has not been studied.

Erythromycin: Concomitant use of rosuvastatin and erythromycin resulted in a 20% decrease in AUC and a 30% decrease in C_{max} of rosuvastatin. This interaction may be caused by the increase in gut motility caused by erythromycin.

Oral Contraceptive / Hormone Replacement Therapy (HRT): Concomitant use of rosuvastatin and an oral contraceptive resulted in an increase in ethinyl estradiol and norgestrel AUC of 26%

and 34%, respectively. These increased plasma levels should be considered when selecting oral contraceptive doses. There are no pharmacokinetic data available in subjects taking concomitant rosuvastatin and HRT and therefore a similar effect cannot be excluded.

4.6Use in Special Populations

Pregnant Women

Pregnancy Category X: Teratogenic effects. ROVASTAT Tablets are contraindicated for use in pregnant women. Women of child bearing potential should use appropriate contraceptive measures. Since cholesterol and other products of cholesterol biosynthesis are essential for the development of the fetus, the potential risk from inhibition of HMG-CoA reductase outweighs the advantage of treatment during pregnancy. If a patient becomes pregnant during use of this product, treatment should be discontinued immediately.

Lactating Women

Use of ROVASTAT Tablets is contraindicated during breastfeeding. Limited data indicate that rosuvastatin is present in human milk. There is no available information on the effects of the drug on the breastfed infant or the effects of the drug on milk production. Because of the potential for serious adverse reactions in a breastfed infant, nursing mothers are advised not to breastfeed their infants during treatment with rosuvastatin.

Paediatric Patients

The safety and efficacy of rosuvastatin in children above 6 years of age appears consistent with that observed for adult patients. Rosuvastatin has not been studied in children younger than 6 years of age. Therefore, ROVASTAT Tablets are not recommended for use in children below 6 years. For dosage in children above 6 years, please refer ‘Posology and Method of Administration’ section.

Geriatric Patients

No overall differences in safety or effectiveness have been observed between elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out. Elderly patients are at higher risk of myopathy and thus, rosuvastatin should be prescribed with caution in these patients. The recommended starting dose of rosuvastatin in patients above 70 years of age is 5 mg once daily. No other dose adjustment is necessary in relation to age.

Renal Impairment Patients

No dose adjustment is necessary in patients with mild to moderate renal impairment. The recommended starting dose of rosuvastatin is 5 mg in patients with moderate renal impairment (creatinine clearance of <60 ml/min). The use of rosuvastatin in patients with severe renal impairment is contraindicated.

Hepatic Impairment Patients

Rosuvastatin is contraindicated in patients with active liver disease, which may include unexplained persistent elevations of hepatic transaminase levels. Chronic alcohol liver disease is known to increase rosuvastatin exposure, thus, rosuvastatin should be used with caution in these patients.

4.7Effect on Ability to Drive and Use Machines

Studies to determine the effect of rosuvastatin on the ability to drive and use machines have not been conducted. However, based on its pharmacodynamic properties, rosuvastatin is unlikely to affect this ability. When driving vehicles or operating machines, it should be taken into account that dizziness may occur during treatment.

4.8Undesirable Effects

Rosuvastatin is generally well tolerated. The adverse events are generally mild and transient.

Clinical Trials Experience

The most commonly reported adverse reactions (incidence ≥ 2%) reported in the clinical trial were headache, myalgia, abdominal pain, constipation, asthenia, nausea.

The following serious adverse reactions may occur with use of all statins including rosuvastatin.

- Rhabdomyolysis with myoglobinuria.
- Acute renal failure and myopathy (including myositis).
- Liver enzyme abnormalities (elevated levels of AST and ALT).

Other adverse reactions reported in clinical trials were abdominal pain, dizziness, hypersensitivity (including rash, pruritus, urticaria, and angioedema) and pancreatitis.

Post-Marketing Experience

The following adverse reactions have been identified in post-marketing studies of rosuvastatin. Arthralgia, fatal and non-fatal hepatic failure, hepatitis, jaundice, thrombocytopenia, depression, sleep disorders (including insomnia and nightmares), peripheral neuropathy, hematuria, sexual dysfunction, interstitial lung disease especially with long term therapy, and gynecomastia.

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.

There have been rare post-marketing reports of immune-mediated necrotizing myopathy and cognitive impairment (e.g., memory loss, forgetfulness, amnesia, memory impairment, confusion) associated with statin use. The reports are generally non serious, and reversible upon statin discontinuation.

Laboratory Test Abnormalities

- Dipstick-positive proteinuria and microscopic hematuria.

- Elevated levels of serum creatine phosphokinase, transaminases, glucose, glutamyl transpeptidase, alkaline phosphatase, and bilirubin.
- Thyroid function abnormalities.

4.9Overdose

There is no specific treatment in the event of rosuvastatin overdose. If it occurs, the patient should be treated symptomatically and supportive measures instituted as required. Liver function and creatinine kinase (CK) levels should be monitored. Hemodialysis is unlikely to be of benefit.

5. Pharmacological Properties

5.1 Mechanism of Action

Rosuvastatin is a synthetic lipid lowering agent belongs to a class of medications called as statins. Statins are also called as 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors. Rosuvastatin is a selective and competitive inhibitor of HMG-CoA reductase, the rate limiting enzyme that converts HMG-CoA to mevalonate, a precursor of cholesterol. The primary site of action of rosuvastatin is the liver, the target organ for cholesterol lowering.

In *in vivo* and *in vitro* studies, rosuvastatin produces its lipid-modifying effects in two ways.

- First, it increases the number of hepatic LDL receptors on the cell-surface to enhance uptake and catabolism of LDL.
- Second, rosuvastatin inhibits hepatic synthesis of VLDL (very low density lipoproteins), which reduces the total number of VLDL and LDL particles.

5.2Pharmacodynamic Properties

Rosuvastatin dose dependently reduces elevated LDL-cholesterol and reduces total cholesterol and triglycerides and increases HDL-cholesterol. A therapeutic response to rosuvastatin is evident within 1 week of commencing therapy and 90% of maximum response is usually achieved in 2 weeks. The maximum response is usually achieved by 4 weeks and is maintained after that. Individualization of drug dosage should be based on the therapeutic response.

5.3Pharmacokinetic Properties

Absorption: After oral administration, peak plasma concentration of rosuvastatin is reached in 3 to 5 hours. The absolute bioavailability is approximately 20%. Administration of rosuvastatin with food did not affect the AUC (area under the plasma concentration-time curve) of rosuvastatin. The AUC of rosuvastatin does not differ following evening or morning drug administration.

Distribution: Mean volume of distribution of rosuvastatin is approximately 134 litres. Rosuvastatin is 88% bound to plasma proteins, mostly albumin. This binding is reversible and independent of plasma concentrations.

Metabolism: Rosuvastatin is taken up extensively by the liver which is the primary site of cholesterol synthesis and LDL-C clearance. Rosuvastatin is not extensively metabolized;

approximately 10% of the dose is recovered as metabolite. The major metabolite is N-desmethyl rosuvastatin, which is formed principally by cytochrome P450 2C9, and *in vitro* studies have demonstrated that N-desmethyl rosuvastatin has approximately one-sixth to one-half the HMG-CoA reductase inhibitory activity of the parent compound. Overall, greater than 90% of HMG-CoA reductase inhibitory activity is due to parent compound.

Excretion: Rosuvastatin and its metabolites are primarily excreted in the feces (90%) and the remaining part is excreted via kidney. The elimination half-life (t½) of rosuvastatin is approximately 19 hours.

6. Nonclinical Properties

6.1 Animal Toxicology

Toxicity: In repeated-dose toxicity studies histopathologic liver changes likely due to the pharmacologic action of rosuvastatin were observed in mouse, rat, and to a lesser extent with effects in the gall bladder in dogs, but not in monkeys. In addition, testicular toxicity was observed in monkeys and dogs at higher dosages. Reproductive toxicity was evident in rats, with reduced litter sizes, litter weight and pup survival observed at maternally toxic doses, where systemic exposures were several times above the therapeutic exposure level.

Carcinogenesis: In a 104-week carcinogenicity study in rats at dose levels of 2, 20, 60, or 80 mg/kg/day by oral gavage, the incidence of uterine stromal polyps was significantly increased in females at 80 mg/kg/day at systemic exposure 20 times the human exposure at 40 mg/day based on AUC. Increased incidence of polyps was not seen at lower doses.

In a 107-week carcinogenicity study in mice given 10, 60, or 200 mg/kg/day by oral gavage, an increased incidence of hepatocellular adenoma/carcinoma was observed at 200 mg/kg/day at systemic exposures 20 times the human exposure at 40 mg/day based on AUC. An increased incidence of hepatocellular tumors was not seen at lower doses.

Mutagenesis: Rosuvastatin was not mutagenic or clastogenic with or without metabolic activation in the Ames test with *Salmonella typhimurium* and *Escherichia coli*, the mouse lymphoma assay, and the chromosomal aberration assay in Chinese hamster lung cells. Rosuvastatin was negative in the *in vivo* mouse micronucleus test.

Impairment of Fertility: In rat fertility studies with oral gavage doses of 5, 15, 50 mg/kg/day, males were treated for 9 weeks prior to and throughout mating and females were treated 2 weeks prior to mating and throughout mating until gestation day 7. No adverse effect on fertility was observed at 50 mg/kg/day (systemic exposures up to 10 times the human exposure at 40 mg/day based on AUC). In testicles of dogs treated with rosuvastatin at 30 mg/kg/day for one month, spermatidic giant cells were seen. Spermatidic giant cells were observed in monkeys after 6-month treatment at 30 mg/kg/day in addition to vacuolation of seminiferous tubular epithelium. Exposures in the dog were 20 times and in the monkey 10 times the human exposure at 40 mg/day based on body surface area. Similar findings have been seen with other drugs in this class.

Teratogenicity: Rosuvastatin crosses the placenta in rats and rabbits and is found in fetal tissue and amniotic fluid at 3% and 20%, respectively, of the maternal plasma concentration following a

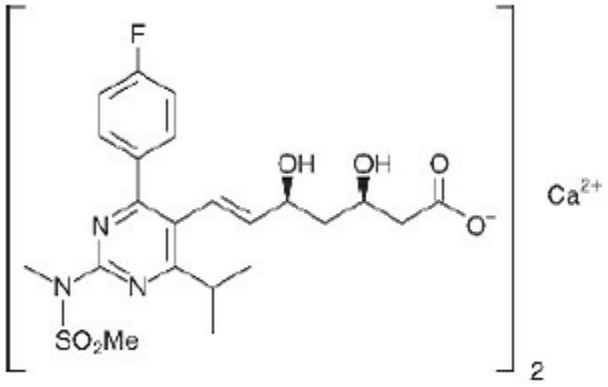
single 25 mg/kg oral gavage dose on gestation day 16 in rats. A higher fetal tissue distribution (25% maternal plasma concentration) was observed in rabbits after a single oral gavage dose of 1 mg/kg on gestation day 18. Rosuvastatin administration did not indicate a teratogenic effect in rats at ≤25 mg/kg/day or in rabbits ≤3 mg/kg/day (doses equivalent to the maximum recommended human dose - MRHD of 40 mg/day based on AUC and body surface area, respectively).

7. Description

ROVASTAT 5 Tablets are reddish coloured, capsule shaped, film coated tablets plain on both the sides.
ROVASTAT 10 Tablets are white coloured, capsule shaped, film coated tablets plain on both the sides.

ROVASTAT 5 Tablets contains 5 mg of rosuvastatin whereas ROVASTAT 10 Tablets contains 10 mg of rosuvastatin for oral administration.

Rosuvastatin calcium is the calcium salt form of rosuvastatin, a statin with antilipidemic activity. Rosuvastatin calcium is a white amorphous powder.
Molecular Weight: 1001.14 g/mol.
Molecular Formula: C22H27FN3O6S) 2Ca.
Chemical Name: bis[(E)-7-[4-(4-fluorophenyl)-6-isopropyl-2- [methyl(methylsulfonyl) amino] pyrimidin-5-yl](3R,5S)-3,5-dihydroxyhept-6-enoic acid] calcium.
Structural Formula:



Inactive ingredients (excipients) of ROVASTAT 5 Tablets contain Tribasic Calcium Phosphate, Cross Povidone XL, Microcrystalline Cellulose, Lactose, Magnesium Stearate, Hydropropyl Methylcellulose, Titanium Dioxide, Talcum, Isopropyl Alcohol, Methylene Chloride, Propylene Glycol, and Colour Red Oxide of Iron.

Inactive ingredients (excipients) of ROVASTAT 10 Tablets contain Tribasic Calcium Phosphate, Cross Povidone XL, Microcrystalline Cellulose, Lactose, Magnesium Stearate, Hydropropyl Methylcellulose, Titanium Dioxide, Talcum, Isopropyl Alcohol, Methylene Chloride, and Propylene Glycol.

8. Pharmaceutical Particulars

8.1 Incompatibilities

None known.

8.2Shelf-life

24 months.

8.3Packaging Information

15 tablets per strip.

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

- Instruct patients to take this medicine exactly as prescribed by your doctor. Do not change the dose or stop therapy without consulting doctor.
- Instruct patients to take ROVASTAT Tablets once a day at any time of the day, with or without food. It may be easier to take your dose if you do it at the same time every day, such as with breakfast or dinner, or at bedtime. Do not take more than one dose at a time.
- If patients miss a dose, they can take it as soon as they remember. Do not take this medicine if it has been more than 12 hours since the last missed dose. Wait and take the next dose at regular scheduled time.
- Pregnant women should strictly avoid use of this medicine. When pregnancy is detected, suspected or planned, discontinue ROVASTAT Tablets as soon as possible.
- This medicine is not recommended for use during lactation. If medicine is necessary, advise nursing mothers not to breastfeed their infants.
- Use of this medicine is not recommended in children less than 6 years of age.
- Patients should wait at least 2 hours after taking ROVASTAT Tablets to take an antacid which contains a combination of aluminum and magnesium hydroxide.
- Patients are advised to consult their doctor immediately if they have unexplained muscle pain, tenderness, weakness, or more tiredness than usual while taking ROVASTAT Tablets.

- Patients are advised not to take ROVASTAT Tablets if they have liver problems.

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 – 249403, Uttarakhand.

11. Details of Permission or License Number with Date

Rovastat 5: Mfg. Lic. No.: 31/UA/2013; Date of FDA Product Permission: 03/06/2020.
Rovastat 10: Mfg. Lic. No.: 31/UA/2013; Date of FDA Product Permission: 30/06/2020.

12. Date of Revision

April 2021.

Marketed by:
BLUE CROSS LABORATORIES PVT LTD.
A-12, M.I.D.C., NASHIK-422 010.
Regd. Off.: Peninsula Chambers, G. K. Marg, Mumbai-400 013.