Drug/ Drug Class:

Rosuvastatin
Fadztat®

20 mg Film-Coated Tablet
Anti-hypercholesterolemia/
Anti-dyslipidemia

Pharmacodynamics

Rosuvastatin is administered orally in the active form with peak plasma levels occurring 5 hours after dosing. Exposure increases linearly over the dose range. The haff life is 19 hours and does not increase with increasing dose. Absolute bioavailability is 20%.
Them is minimal accumulation on repeated once daily dosing.
Rosuvastatin undergoes first pass extraction in the liver, which is the primary site of cholesterol synthesis and LDL-C clearance. Rosuvastatin is approximately 90% bound to plasma proteins, mostly albumin. The parent compound, accounts for greater than 90% of the circulating active HMG CoA reductase inhibitor activity. Rosuvastatin undergoes limited metabolism (approximately 10%), mainly to the N-desmethyl form, and 90% is eliminated as unchanged drug in the feces with the remainder being excreted in the urine.

Special Populations: Age and sex: There was no clinically relevant effect of age or sex on the pharmacokinetics of Rosuvastatin in adults. The exposure in children and adolescents with heterozygous familial hypercholesterolemia appears to be similar to or lower than that in adult patients with dyslipidemia.

Race: Pharmacokinetic studies show an approximate 2-fold elevation in median AUC in Asian subjects compared with Caucasians. A population pharmacokinetic analysis revealed no clinically relevant differences in pharmacokinetics among Caucasian, Hispanic and Black or Afro-Caribbean groups.
Renal insufficiency: In a study in subjects with varying degrees of renal impairment, mild to moderate renal disease had little influence on plasma concentrations of Rosuvastatin. However, subjects with severe impairment (CrCI <30 mi./min) had a 3-fold increase in plasma concentration compared to healthy volunteers.
Hepatic insufficiency: In a study in subjects with varying degrees of hepatic impairment there was no evidence of increased exposure to Rosuvastatin other than in the 2 subjects with the most severe liver disease (Child-Pugh scores of 8 and 9). In these subject's systemic exposure was increased by at least 2-fold compared to subjects with lower Child-Pugh scams.
Genetic polymorphisms: Disposition of HMG-CoA reductase inhibitors, including Rosuvastatin, involves OATP1B1 and BCRP transporter proteins. In patients with SLCO1 B1 (0ATP1B1) and/or ABCG2 (BCRP) genetic polymorphisms there is a risk of increased Rosuvastatin exposure. Individual polymorphisms of SLCO1 B1 c.521CC and ABCG2 c.421 AA am associated with an approximate 1.6-fold higher Rosuvastatin exposure (AUC) or 2.4-fold higher exposure, respectively, compared to the SLCO1B1 c.521TT or ABCG2 c.421 CC genotypes.
Toxicology: Preclinical safely data: Preclinical data reveal no special hazards for humans based on conventional studies of safety pharmacology, repeat-dose toxicity, genotoxicity, carcinogenic potential, and reproductive toxicity.

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Formulation

Each film-coated tablet contains:

Rosuvastatin Calcium
Equivalent to Rosuvastatin.....20 mg

Availability

- Alu/Alu Blister Pack x 10's
- Box of 30's

Mechanism of Action

Rosuvastatin is a selective, potent and competitive inhibitor of HMG-CoA reductase, the rate-limiting enzyme that converts 3-hydroxy-3-methylglutaryl coenzyme A to mevalonate, a precursor of cholesterol. Triglycerides (TG) and cholesterol in the liver are incorporated, with apolipoprotein B (Apse), into very low-density lipoprotein (VLDL) and released into the plasma for delivery to peripheral tissues. VLDL particles are TG-rich. Cholesterol-rich low-density lipoprotein (LDL) is formed from VLDL and is cleared primarily through the high affinity LDL receptor in the liver.
Rosuvastatin produces its lipid-modifying effects in two ways; it increases the number of hepatic LDL receptors on the cell-surface, enhancing uptake and catabolism of LDL and it inhibits the hepatic synthesis of VLDL, thereby reducing the total number of VLDL and LDL particles.
High density lipoprotein (HDL), which contains Apo A-I is involved, amongst other things, in transport of cholesterol from tissues back to the liver (reverse cholesterol transport). The involvement of LDL-C in atherogenesis has been well documented. Epidemiological studies have established that high LDL-C, TG, low HDL-C and Apo A-I have been linked to a higher risk of cardiovascular disease. Intervention studies have shown the benefits on mortality and CV event rates of lowering LDL-C and TG or raising HDL-C. More recent data has linked the beneficial effects of HMG-CoA reductase inhibitors to lowering of non-HDL (i.e., all circulating cholesterol not in HDL) and ApoB or reducing the ApoB/ApoA-I ratio.

Dosage & Administration

The usual dose is 10-40 mg orally once a day.
The dosage of Rosuvastatin should be individualized according to the goal of therapy and patient response. The majority of patients are controlled at the start dose. However, if necessary, dose adjustment can be made at 2-to-4-week intervals. Rosuvastatin may be given at any time of day, with or without food.
Adults:
Primary Hypercholesterolemia (including Heterozygous Familial Hypercholesterolemia), Mixed Dyslipidemia, Dysbetalipoproteinemia, Isolated Hypertriglyceridemia and Treatment of Atherosclerosis and Prevention of Cardiovascular Events
The usual start dose is 10 mg once a day. A 5 mg start dose is available for special patient populations if needed. For patients with severe hypercholesterolemia (including heterozygous familial hypercholesterolemia) or those with aggressive lipid targets, a start dose of 20 mg may be considered.
Homozygous Familial Hypercholesterolaemia
For patients with homozygous familial hypercholesterolemia a start dose of 20 mg once a day is recommended.
Children and Adolescents 6 to 17Years of Age:
In children 6 to 9 years of age with heterozygous familial hypercholesterolemia, the usual dose range is 510 mg orally once daily. Safety and efficacy of doses greater than 10 mg have not been studied in this population. In children 10 to 17 years of age with heterozygous familial hypercholesterolemia, the usual dose range is 5-20 mg orally once daily. Safety and efficacy of doses greater than 20 mg have not been studied in this population.
The dose should be appropriately titrated to achieve treatment goal.
In children and adolescents with homozygous familial hypercholesterolemia experience is limited to a small number of patients (aged 8 years and above).

Indication

Rosuvastatin should be used as an adjunct to diet when the response to diet and exercise is inadequate.

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Contraindication

-in patients with hypersensitivity to any component of this product.
- in patients with active liver disease.
- during pregnancy, while breast-feeding and in women of child-bearing potential not using appropriate contraceptive measures.

Over Dose Treatment

There is no specific treatment in the event of overdose. In the event of overdose, the patient should be treated symptomatically and supportive measures instituted as required. Hemodialysis is unlikely to be of benefit

Storage Condition

- Store at temperatures not exceeding 30°C.

Manufactured by: Stallion Laboratories PA. Ltd.
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