

Rosuvastatin Tablets 5 mg, 10 mg, 20 mg and 40 mg

ROSLAREN

5 | 10 | 20 | 40

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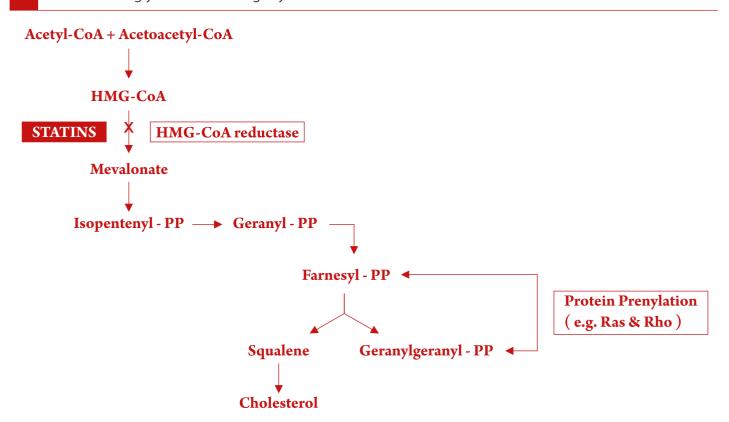
- Hyperlipidemia is too much cholesterol in the blood. Cholesterol is a waxy, fat protein manufactured by the liver and is essential for healthy cell membranes, hormone production, and vitamin storage.
- Even the brain depends on cholesterol for proper functioning. Cholesterol becomes a problem when too much of the bad kind is produced or ingested through regular eating of unhealthy foods.

Causes

- The causes of hyperlipidemia are either genetic (familial or primary hyperlipidemia) or from a poor diet and other specific factors (secondary hyperlipidemia).
- When the body cannot utilize or remove the excess fat, it accumulates in the blood. Over time, the buildup damages the arteries & internal organs. This process contributes to the development of heart disease.

- Rosuvastatin is a competitive inhibitor of HMG - CoA reductase.

- Unlike most others, however, it is a completely synthetic compound. HMG CoA reductase catalyzes the reduction of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) to mevalonate, which is the rate limiting step in hepatic cholesterol biosynthesis.
- Inhibition of the enzyme decreases de novo cholesterol synthesis. Rosuvastatin also reduces blood levels of trigly cerides and slightly increases levels of HDL - cholesterol.



CLINICAL STUDY

Effect of Rosuvastatin on systemic blood pressure in patients with hypercholesterolemia

Objective

To investigate whether Rosuvastatin reduces blood pressure in patients with hypercholesterolemia.

Aethods

The present study investigated the effect of Rosuvastatin on lipids and clinical parameters in 25 patients with a mean (\pm SD) age of 58.4 ± 10.6 years over a three - month period.

- Rosuvastatin (2.5 mg/day to 5.0 mg/day) reduced systolic BP from 136.3 \pm 13.1 mm Hg to 130.8 \pm 10.7 mm Hg, along with a significant reduction in serum low-density lipoprotein cholesterol level.

- The patients were divided into two groups: 13 responders whose BP decreased by >5 mm Hg with Rosuvastatin treatment and 12 nonresponders who showed a BP reduction of \leq 5 mm Hg.
- Baseline systolic BP was significantly higher in responders than nonresponders (143.6 ± 9.6 mm Hg versus 128.4 ± 11.9 mm Hg, respectively).
- Responders also had a lower serum concentration of high-sensitivity C-reactive protein compared with nonresponders (0.11 ± 0.07 mg/dL versus 0.40 ± 0.28 mg/dL).
- The extent of BP reduction was positively correlated with baseline systolic BP but not with the reduction of low density lipoprotein cholesterol level.
- Among the patients with baseline systolic BP >130 mm Hg, all 11 responders (138.3 mm Hg) were nonsmokers, while five of six nonresponders (145.7 mm Hg) were smokers.

Conclusion

- Rosuvastatin had an additive antihypertensive effect in patients with poorly controlled hypertension that was independent of its lipid-lowering effect, which may be related to an inflammatory mechanism.
- The study demonstrated that Rosuvastatin treatment achieved more effective blood pressure reduction in patients with a higher baseline SBP.
- Our results indicate that Rosuvastatin may have a useful antihypertensive effect independent of its lipid-lowering effect in patients with poorly controlled hypertension.
- The response to Rosuvastatin treatment may be affected by smoking-induced impairment of endothelial function or by inflammation that increases serum hs CRP levels.

Reference: Exp Clin Cardiol.2012 Winter; 17 (4):221-5

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Background

- Hyperlipidemia refers to increased levels of lipids (fats) in the blood, including cholesterol & triglycerides.
- Hyperlipidemia can significantly increase your risk of developing cardiovascular disease, including disease of blood vessels supplying the heart (coronary artery disease), brain (cerebrovascular disease), and limbs (peripheral vascular disease).
- These conditions can in turn lead to chest pain, heart attacks, strokes, and other problems. Because of these risks, treatment is often recommended for people with hyperlipidemia.

Description

- Roslaren (Rosuvastatin) is an antilipemic agent that competitively inhibits hydroxymethylglutaryl coenzyme A (HMG-CoA) reductase.
- HMG-CoA reductase catalyzes the conversion of HMG CoA to mevalonic acid, the rate limiting step in cholesterol biosynthesis.
- Rosuvastatin belongs to a class of medications called statins & is used to reduce plasma cholesterol levels and prevent cardiovascular disease.

ndication

- Used as an adjunct to dietary therapy to treat primary hyperlipidemia, mixed dyslipidemia and hypertrigly-ceridemia.
- Furthermore, it is used to slow the progression of atherosclerosis and for primary prevention of cardiovascular disease.

Mechanism of Action

- Rosuvastatin is a competitive inhibitor of HMG CoA reductase.
- HMG CoA reductase catalyzes the conversion of HMG CoA to mevalonate, an early rate limiting step in cholesterol biosynthesis.
- Rosuvastatin acts primarily in the liver. Decreased hepatic cholesterol concentrations stimulate the upregulation of hepatic low density lipoprotein (LDL) receptors which increases hepatic uptake of LDL.
- Rosuvastatin also inhibits hepatic synthesis of very low density lipoprotein (VLDL). The overall effect is a decrease in plasma LDL and VLDL.

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- The dose range for Roslaren in adults is 5 to 40 mg orally once daily.
- The usual starting dose in adult patients with hypercholesterolemia is 20 mg once daily.
- The maximum Roslaren dose of 40 mg should be used only for those patients who have not achieved their LDL-C goal utilizing the 20 mg dose.

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