ROSLAREN-F-10

Rosuvastatin 10 mg and Fenofibrate 160 mg Tablets

Background:

- Dyslipidemia, and especially atherogenic dyslipidemia, a combination of small low-density lipoproteins cholesterol (LDL-C), decreased high-density lipoprotein cholesterol (HDL-C) and increased triglyceride (TG) concentrations, represents a major cardiovascular (CV) risk factor.
- Nuclear receptor peroxisome proliferator-activated receptors (PPARs) are involved in the regulation of lipid metabolism; PPAR ligands are used to treat dyslipidemias. Fibrates have a major impact on TG metabolism as well as on modulating LDL size and subclasses.

Description:

- ROSLAREN-F-10 contains Rosuvastatin and Fenofibrate. Both are antilipemic agent which reduces both cholesterol and triglycerides in the blood.
- Rosuvastatin competitively inhibits hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase to reduce cholesterol synthesis in liver.
- Fenofibrate belongs to the class of organic compounds known as benzophenones.

Composition:

Each Film coated tablet of ROSLAREN-F-10 contains Rosuvastatin 10 mg and Fenofibrate 160 mg.

Indication:

For use as adjunctive therapy to diet to reduce elevated LDL-C, Total-C and Triglycerides and to increase HDL-C in adult patients with primary hypercholesterolemia or mixed dyslipidemia.

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.
- Fenofibrate exerts its therapeutic effects through activation of peroxisome proliferator activated receptor alpha (PPARα). This increases lipolysis and elimination of triglyceride-rich particles from plasma by activating lipoprotein lipase and reducing production of Apoprotein C-III.
- The resulting fall in triglycerides produces an alteration in the size and composition of LDL from small, dense particles, to large buoyant particles. These larger particles have a greater affinity for cholesterol receptors and are catabolized rapidly.

Dosage:

The recommended daily dosage of ROSLAREN-F-10 is one Tablet per day.

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La Renon®

The Magic of Dual Power

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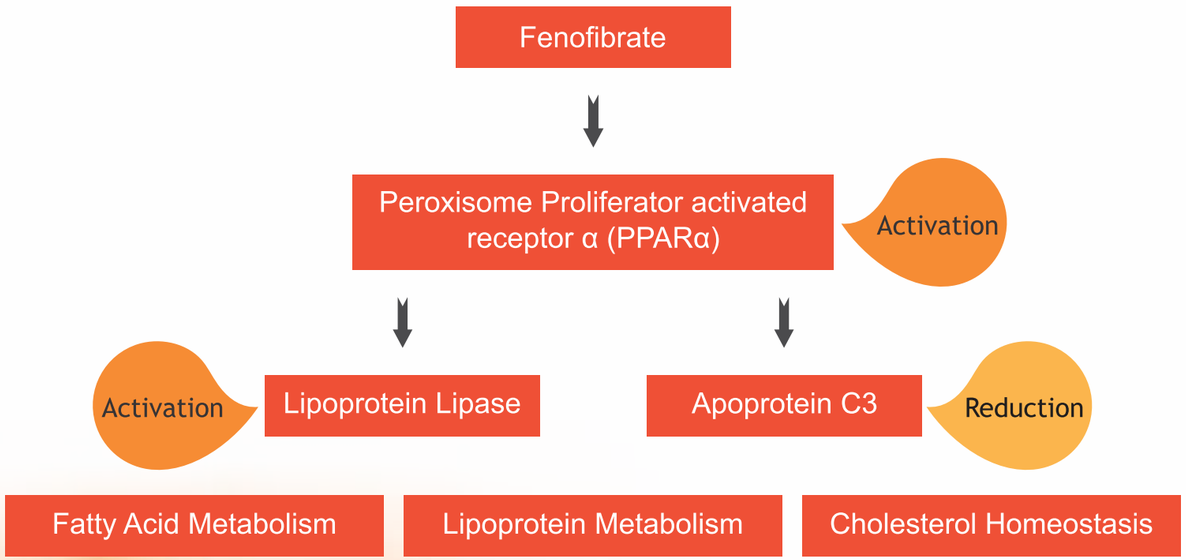
Dyslipidemia:

- Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein level that contributes to the development of atherosclerosis.
- Diagnosis is by measuring plasma levels of total cholesterol, TGs, and individual lipoproteins. Treatment involves dietary changes, exercise, and lipid-lowering drugs.

Fenofibrate Mode of Action:

- The peroxisome proliferator-activated receptors (PPARs) are a group of nuclear receptor proteins that function as transcription factors regulating the expression of genes.
- PPARs play essential roles in the regulation of cellular differentiation, development, and metabolism (carbohydrate, lipid, protein) and tumorigenesis of higher organisms.
- Fenofibrate acts through activation of peroxisome proliferator activated receptor α (PPARα).
- Peroxisome proliferator activated receptor α (PPARα) principally regulates the fatty acid and lipoprotein metabolism.
- Fenofibrate lowers the blood cholesterol and other lipid levels by activating PPARα, which in turn activates lipoprotein lipase and reduces Apoprotein C3.
- Active lipoprotein lipase and reduced Apoprotein C3 cause dramatic degradation (lipolysis) and elimination of triglyceride-rich particles from plasma.
- Activation of PPARα also reduces very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) containing Apoprotein B.
- Apart from this PPARα also enhances the synthesis of Apoprotein AI, AII, and high-density lipoprotein (HDL).



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CLINICAL STUDY

Effect of Rosuvastatin monotherapy or in combination with fenofibrate or ω -3 fatty acids on lipoprotein subfraction profile in patients with mixed dyslipidemia and metabolic syndrome.

Background:

Raised triglycerides (TG), decreased high-density lipoprotein cholesterol (HDL-C) levels and a predominance of small dense low density lipoproteins (sdLDL) are characteristics of the metabolic syndrome (MetS).

Objective:

To compare the effect of high-dose Rosuvastatin monotherapy with moderate dosing combined with fenofibrate or ω-3 fatty acids on the lipoprotein subfraction profile in patients with mixed dyslipidemia and MetS.

Methods:

- We previously randomised patients with low-density lipoprotein cholesterol (LDL-C) > 160 and TG > 200 mg /dl to Rosuvastatin monotherapy 40 mg / day (R group, n = 30) or Rosuvastatin 10 mg / day combined with fenofibrate 200 mg / day (RF group, n = 30) or ω -3 fatty acids 2g/day (Rω group, n = 30).
- In the present study, only patients with MetS were included (24, 23 and 24 in the R, RF and Rω groups respectively). At baseline and after 12 weeks of treatment, the lipoprotein subfraction profile was determined by polyacrylamide 3% gel electrophoresis.

Results:

- The mean LDL size was significantly increased in all groups. This change was more prominent with RF than with other treatments in parallel with its greater hypotriglyceridemic capacity ($p < 0.05$ compared with R and Rω).
- A decrease in insulin resistance by RF was also noted. Only RF significantly raised HDL-C levels (by 7.7%, $p < 0.05$) by increasing the cholesterol of small HDL particles.
- The cholesterol of larger HDL subclasses was significantly increased by R and Rω.

Conclusion:

All regimens increased mean LDL size; RF was the most effective. A differential effect of treatments was noted on the HDL subfraction profile.

Reference:
Int J Clin Pract, September 2012, 66, 9, 843-853