Fenofibric acid delayed-release capsules are a prodrug of fenofibrate, a PPAR α agonist indicated:

- In combination with a statin to reduce LDL-C and increase HDL-C in patients with hypercholesterolemia.
- As monotherapy to reduce LDL-C in patients with severe hypercholesterolemia.
- As monotherapy to reduce elevated total cholesterol, LDL-C, TAG, and Apo B in patients with primary hypercholesterolemia, mixed dyslipidemia, or hypertriglyceridemia.

**DOSAGE AND ADMINISTRATION**

- **Mixed dyslipidemia**: 135 mg once daily (3.2).
- **Hypertriglyceridemia**: 45 to 135 mg once daily (3.2).
- **Heterozygous familial hypercholesterolemia**: 135 mg once daily (3.2).
- **Maximum dose**: 135 mg once daily (2.1).
- **Use in liver disease**: Metabolic steady-state dosing (3.3).

**Coadministration with other lipid-lowering therapies**: Coadministration with bile acid sequestrants is not recommended. Coadministration with fibrates is not recommended. If astatin and a fibrate are used concomitantly, patients should be monitored for increased muscle adverse events, including myalgias and rhabdomyolysis.

**SIDE EFFECTS**

- **Musculoskeletal pain**: Probably dose-related.
- **Myalgia**: Usually mild to moderate and usually did not result in discontinuation of therapy.
- **Rhabdomyolysis**: Occurred with combination therapy with a statin.
- **Increased serum creatinine levels**: Occurred with combination therapy with a statin.
- **Increased serum creatine kinase levels**: Occurred with combination therapy with a statin.

**WARNINGS AND PRECAUTIONS**

- **Paradoxical Decreased in HDL Cholesterol Levels (PPAR α antagonist effect)**: HDL-C and other cholesterol components are affected by the conformation of the HDL particle. HDL particles can take two different conformations (small, dense, or large, buoyant). The conformational shift to the large, buoyant form is associated with an increased anti-inflammatory and anti-thrombotic activity. Decreased HDL-C levels in patients treated with fibrates are largely due to a decrease in the large, buoyant form of HDL.

**ADVERSE REACTIONS**

- **Gastrointestinal Disorders**
- **Drug Interactions**
- **Use in Specific Populations**
- **Pregnancy**
- **Contraindications**
- **Pediatric Use**
- **Reproductive Toxicology and Nonclinical Toxicology**
- **Carcinogenicity and Mutagenicity**
- **Nonclinical Toxicology**
- **Clinical Pharmacology**
- **Pharmacokinetics**
- **Pharmacodynamics**

**Full Prescribing Information: Contents**

1. **INDICATIONS AND USAGE**
   - 1.1 Treatment of Severe Hypertriglyceridemia
   - 1.2 Treatment of Mixed Hyperlipidemias
   - 1.3 Treatment of Primary Hypercholesterolemia or Mixed Dyslipidemia
   - 1.4 Important Limitations of Use
   - 1.5 General Considerations for Treatment

2. **DRUG INTERACTIONS**
   - 2.1 Coadministered with other lipid-lowering agents
   - 2.2 Coadministration with other lipid-lowering therapies
   - 2.3 Severe Hypertriglyceridemia
   - 2.4 Primary Hypercholesterolemia or Mixed Dyslipidemia

3. **DOSE FORM STRENGTHS**

4. **CONTRAINDICATIONS**

5. **PRECAUTIONS**
   - 5.1 Mortality and Coronary Heart Disease Morbidity
   - 5.2 Safety in Patients with Renal Impairment
   - 5.3 Safety in Patients with Hepatic Impairment
   - 5.4 Safety in Elderly Patients
   - 5.5 Safety in Patients with Renal Function
   - 5.6 Safety in Patients with Severe Hypertriglyceridemia
   - 5.7 Safety in Patients with Pregnancy

6. **ADVERSE REACTIONS**
   - 6.1 Gastrointestinal Disorders
   - 6.2 Pulmonary Edema
   - 6.3 Dermatological

7. **DRUG INTERACTIONS**

8. **USE IN SPECIFIC POPULATIONS**
   - 8.1 Use in Renal Impairment
   - 8.2 Use in Liver Impairment
   - 8.3 Use in Elderly Patients
   - 8.4 Use in Pregnancy
   - 8.5 Use in Pregnancy
   - 8.6 Use in Pregnancy

9. **DRUG USE DURING PREGNANCY**
   - 9.1 Pregnancy Category B
   - 9.2 Excretion into Breast Milk

10. **DRUG USE IN LACTATING MOTHERS**

**DRUG USE IN NURSING MOTHERS**

**FULL PRESCRIBING INFORMATION: CONTENTS**

**1. INDICATIONS AND USAGE**

- **1.1 Treatment of Severe Hypertriglyceridemia**
- **1.2 Treatment of Mixed Hyperlipidemias**
- **1.3 Treatment of Primary Hypercholesterolemia or Mixed Dyslipidemia**
- **1.4 Important Limitations of Use**

**2. DRUG INTERACTIONS**

- **2.1 Coadministered with other lipid-lowering agents**
- **2.2 Coadministration with other lipid-lowering therapies**
- **2.3 Severe Hypertriglyceridemia**

**3. DOSE FORM STRENGTHS**

**4. CONTRAINDICATIONS**

**5. PRECAUTIONS**

- **5.1 Mortality and Coronary Heart Disease Morbidity**
- **5.2 Safety in Patients with Renal Impairment**
- **5.3 Safety in Patients with Hepatic Impairment**
- **5.4 Safety in Elderly Patients**
- **5.5 Safety in Patients with Renal Function**

**6. ADVERSE REACTIONS**

- **6.1 Gastrointestinal Disorders**
- **6.2 Pulmonary Edema**
- **6.3 Dermatological**

**7. DRUG INTERACTIONS**

**8. USE IN SPECIFIC POPULATIONS**

- **8.1 Use in Renal Impairment**
- **8.2 Use in Liver Impairment**
- **8.3 Use in Elderly Patients**
- **8.4 Use in Pregnancy**
- **8.5 Use in Pregnancy**

**9. DRUG USE DURING PREGNANCY**

- **9.1 Pregnancy Category B**
- **9.2 Excretion into Breast Milk**

**10. DRUG USE IN LACTATING MOTHERS**

**11. DRUG USE IN NURSING MOTHERS**
A plasma level of fenofibrate acid occurs in 0 to 3 hours after a single dose administration of fenofibrate acid delayed-release capsules while taking fenofibrate acid capsules. The drug is stable in the gastrointestinal tract and is absorbed 1 to 2 hours after oral administration. Absorption is not significantly different when a single 135 mg dose of fenofibrate acid delayed-release capsules is administered under fasting or non-fasting conditions.

The absolute bioavailability of fenofibrate acid is 62%.

The pharmacokinetics of fenofibric acid was examined in patients with mild, moderate, and severe renal impairment. In patients with severe renal impairment, the renal clearance of fenofibric acid was significantly decreased, and the half-life was prolonged. In patients with moderate and mild renal impairment, the pharmacokinetics of fenofibric acid were similar to those in patients with normal renal function.

The use of fenofibric acid in patients with severe renal impairment should be cautious. In patients with moderate or mild renal impairment, no dosage adjustment is necessary.

Efficacy and safety of fenofibric acid delayed-release capsules coadministered with statins were assessed in three 12-month studies. The studies were randomized, double-blind, placebo-controlled, and involved patients with hypertriglyceridemia and elevated LDL-C. The studies were conducted at 193 study centers in 24 countries.

The effects of fenofibrate on serum triglycerides were studied in two randomized, double-blind, placebo-controlled clinical trials. Patients were randomized to fenofibrate 135 mg once daily or placebo. The duration of the studies was 3 to 6 months. Mean baseline lipid values were: Total-C 306.9 mg/dL; LDL-C 213.8 mg/dL; HDL-C 52.3 mg/dL; and triglycerides 281.0 mg/dL.

A total of 1,060 patients were included in the studies. The studies were conducted in 20 countries. Mean baseline lipid values were: Total-C 310.4 mg/dL; LDL-C 214.5 mg/dL; HDL-C 49.7 mg/dL; and triglycerides 291.0 mg/dL.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.

The mean percentage change from baseline in the primary endpoint was -10.2% for total cholesterol, -21.9% for LDL-C, and 11.8% for triglycerides. The mean percentage change from baseline in HDL-C was 11.8%. The mean percentage change from baseline in apolipoprotein B was -3.1%.