clinic, computer problems, and/or health reasons). An additional six patients had one to two missing observations during the follow-up period. Multilevel models were used to describe the dietary effects and permitted the use of all collected data in the statistical analyses, including incomplete cases. At 42 weeks, although trends in risk factors slightly favored the HiPro-HiMono diet, changes were not significantly different between the AHA and HiPro-HiMono groups for weight (−5.9 vs. −9.1 kg; $P = 0.768$), triglyceride (−0.8 vs. −1.1 mmol/l; $P = 0.920$), fasting glucose (−2.2 vs. −3.2 mmol/l; $P = 0.153$), and LDL cholesterol (0.23 vs. 0.18 mmol/l; $P = 0.217$). The preponderance of patients improved their glycemic control. At 42 weeks, glycemic control was normalized in all 10 patients with impaired fasting glucose; it was also normalized in 2 and reduced to impaired fasting glucose in 3 of 7 patients with diabetes. Food record analyses to evaluate compliance showed that changes from the baseline diets to assigned levels of carbohydrate and total, saturated, and monounsaturated fats were significantly different between the groups, in keeping with different dietary goals. In a similar study (5), with slightly different diets, subjects at the end of 18 months were consuming diets of similar composition.

Our long-term study, enabled by our Internet Management System, was limited by small sample size. The power to detect a 10% difference between groups at $\alpha = 0.05$ with the observed SDs was <18% for LDL cholesterol, triglyceride, and fasting glucose. Weight loss was a potential confounding factor in the analyses. Nevertheless, the study's trends support the hypothesis that a diet high in protein and MUFAs may be advantageous in correcting glucose and lipid metabolism abnormalities. Large, randomized, multicenter trials are needed.

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Fenofibrate Monotherapy Induced Rhabdomyolysis

Fenofibrate (TriCor; Abbott Pharmaceuticals, Abbott Park, IL), a fibric acid derivative, was introduced in the U.S. in 1998. It is frequently used to treat diabetic dyslipidemia and hypertriglyceridemia alone or in combination with statins. Despite reports of rhabdomyolysis with the use of statins or statin-fibrate combinations, there have been no cases of rhabdomyolysis reported in the U.S. when fenofibrate was used alone to treat a patient with normal baseline creatinine. Here, we present the first case of a patient with a normal creatinine who developed life-threatening rhabdomyolysis while under the treatment of fenofibrate monotherapy.

A 56-year-old woman with a medical history significant for type 2 diabetes, peripheral vascular disease, hypertension, hyperlipidemia, and polyneuropathy presented with a complaint of the new onset of diffuse myalgia. She had no recent viral illness or other complaints. Ten days before presentation she was started on 200 mg fenofibrate daily. Before fenofibrate therapy, her serum creatinine was 1.3 mg/dl, thyroid-stimulating hormone was normal, and liver function was normal. Her medications included NPH insulin, metformin 500 mg q.d., amitriptyline 25 mg q.h.s., pioglitazone 30 mg q.d., quinapril 10 mg q.d., fenofibrate 200 mg q.d., and daily aspirin. Physical examination was unremarkable except for diffuse generalized muscle tenderness.

Because of the patient's presenting symptoms and recent initiation of fenofibrate therapy, her creatine phosphokinase (CPK) level was checked and found to be 5,632 units/l. Fenofibrate was discontinued. She was admitted to the hospital with a presumptive diagnosis of rhabdomyolysis. Her admission labs were also remarkable for elevated transaminases and a creatinine of 2.0 mg/dl. Because of the creatinine elevation, metformin was also discontinued.

Following hydration and bicarbonate therapy, the patient's myalgia resolved. She was discharged with fluid intake encouraged. Although her CPK peaked to >23,000 units/l, it returned to baseline (86 units/l) within weeks of discharge. Her renal function also improved gradually within months of hospitalization.

The fibric acid agents have long been shown to be of benefit in the treatment of hyperlipidemia. Early fibrates such as clofibrate and even gemfibrozil have rarely been associated with rhabdomyolysis. Fenofibrate has been one of the newer fibrates to show great promise since its release, with few if any reports of rhabdomyolysis outside the U.S. Taken once daily with a meal, fenofibrate is more effective than gemfibrozil in lowering serum LDL cholesterol and triglyceride concentrations (1,2). It has also been shown to be of benefit in raising the serum concentration of HDL cholesterol and in lowering dense LDL cholesterol. Fenofibrate in combination with low-dose 3-hydroxy-3 methylglutaryl conenzyme A (HMG-CoA) reducatse inhibitors are being used more frequently in the treatment of combined hyperlipidemia and to lower non-HDL cholesterol to