Very low-density lipoprotein (VLDL) is a triglyceride (TG)-rich lipoprotein which is produced by liver. We previously reported that VLDL clearly showed higher values in the order of type 2 diabetic patients with obesity (27.3 ± 22.7 mg/dL), type 2 diabetic patients without obesity (20.1 ± 16.2 mg/dL), subjects with low Framingham risk score (16.6 ± 12.8 mg/dL), and young lean men (4.0 ± 4.6 mg/dL) by using the anion-exchange high performance liquid chromatography data [1]. Further, we reported that the 2-week treatment using a glucagon-like peptide 1 (GLP-1) analog reduced VLDL from 27.3 ± 22.7 to 17.4 ± 7.8 mg/dL in obese patients with type 2 diabetes (BMI, 29.5 ± 7.0 kg/m²; HbA1c, 9.1±2.1%) [2]. In this study, changes in TG were significantly correlated with changes in VLDL (r = 0.99, P < 0.001). These previous studies strongly suggest that VLDL is the leading actor in lipid abnormality in patients with diabetes and/or obesity.

Insulin resistance which is induced by obesity is the main cause of the metabolic syndrome and type 2 diabetes, and increases activity and expression of hormone-sensitive lipase in adipose tissue, which hydrolyses TG, releasing free fatty acids (FFA) (Fig. 1) [3]. In an insulin resistant state, increased FFA entry to liver, reduced degradation of apoB100 and enhanced expression of microsomal TG transfer protein which is a key enzyme involved in VLDL assembly may elevate hepatic production of VLDL [4, 5]. Insulin resistance also decreases the activity of lipoprotein lipase, the rate-limiting enzyme of the catabolism of TG-rich lipoproteins such as VLDL [6].

Hypertriglyceridemia and hypercholesterolemia which are commonly observed in patients with obesity and/or type 2 diabetes may be mainly induced by an increase of VLDL.

**Conflicts of Interest**

The author declares that he has no conflicts of interest concerning this article.

**References**


Figure 1. The abnormal lipid metabolism which may be observed in insulin resistance such as metabolic syndrome and type 2 diabetes.