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Editorial

Diabetes and Dyslipidemia

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Dyslipidemia is common in patients with type II diabetes mellitus. It is one of the most important cardiovascular risk factors and it is a major reason for mortality in diabetic patients. The principal factors which influence lipid metabolism in diabetes are the type of diabetes, the treatment of the disease, the extent of the glucose control, the extent of insulin resistance, the visceral obesity , and the presence and extent of diabetic nephropathy.

In the pathogenesis of diabetic dyslipidaemia, hepatic insulin resistance plays an important role. Free fatty acids, which flow into the liver from visceral adipose tissue, alter the liver metabolism. The liver becomes insulin-resistant, stores fatty acids as triglycerides and develops a steatosis. Hepatic insulin resistance causes dyslipidemia. As a result, the production of triglyceride-rich VLDL (Very Low Density Lipoproteins) increases, causing food-independent hypertriglyceridemia. High VLDL levels lead to increased concentrations of VLDL remnants and of small dense LDL. Both remnants and small dense LDL (Low Density Lipoproteins) increase risk for cardiovascular disease. At the same time, the HDL (High Density Lipoproteins) levels fall, which in turn promotes atherosclerosis. Remnants and small dense LDL are difficult to detect and are not measured in the routine laboratory. However, a determination is not necessary in everyday clinical practice: the measurement of triglyceride levels is sufficient.

Increased triglycerides are associated with cardiovascular disease in diabetics. The triglycerides do not increase the cardiovascular risk, but high triglycerides have metabolic consequences, i.e. low HDL and the production of small dense LDL. In contrast severe hypertriglyceridemia is associated with genetic factors which impair the clearance of triglyceride-rich lipoproteins, such as mutations in the LPL gene and in the apolipoprotein C-III gene. These are main modulators of hypertriglyceridemia in type II diabetes and may lead to chylomicronemia, which is a major risk factor for acute pancreatitis.

In type 1 diabetes mellitus, these abnormalities can usually be reversed with glycemic control. In contrast, in type 2 diabetes mellitus, although lipid values improve, abnormalities commonly persist even after optimal glycemic control has been achieved.

Screening for dyslipidemia is strongly recommended in subjects with diabetes mellitus. A goal of LDL cholesterol of less than 1.8 mmol/l and of triglycerides lower 2.3 mmol/l should be sought.

Conclusions

The increased risk of coronary artery disease in subjects with diabetes mellitus can be partially explained by the lipoprotein abnormalities associated with diabetes mellitus. Hypertriglyceridemia and low levels of high-density lipoprotein are the most common lipid abnormalities. In type 1 diabetes mellitus, these abnormalities can usually be reversed with glycemic control. In contrast, in type 2 diabetes mellitus, although lipid values improve, abnormalities commonly persist even after optimal glycemic control has been achieved. Screening for dyslipidemia is recommended in subjects with diabetes mellitus. A goal of low-density lipoprotein cholesterol of less than 1.8 mmol/l and triglycerides lower than 2.3 mmol/l should be achieved. Several secondary prevention trials, which included subjects with diabetes, have demonstrated a significant benefit of lowering low-density lipoprotein cholesterol in preventing death from coronary

artery disease. The benefit of lowering triglycerides is less clear. Initial approaches to lowering the levels of lipids in subjects with diabetes mellitus should include glycemic control, diet, weight loss, and exercise. When goals are not met, the most common drugs used are statins.

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