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Editorial

Type 2 Diabetes Mellitus and Dyslipidemia

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Poor lipoprotein synthesis and clearance is one of the metabolic problems associated with diabetes. Genetic and epigenetic factors play a key role in lipid homeostasis by influencing the regulation of cell surface receptors.The metabolic syndrome is a collection of metabolic dysfunctions, it is also known as type 2 diabetes mellitus. Insulin resistance, inflammation, dyslipidemia, obesity, hypertension, atherosclerosis, and endothelial dysfunction are all cardiovascular risk factors.

According to the World Health Organization (WHO), diabetes caused 6% of fatalities in 2012 and accounted for 9% of deaths in 2014. In 2015, 90 percent (312 million) of diabetic cases were identified as type 2 diabetes mellitus, according to the WHO. Diabetes affects 6.3 million people in Pakistan. According to projections, the ratio will rise to by 2030, it is expected that the ratio will have risen to 11.4 million. Although the specific mechanism is unknown, type 2 diabetes mellitus is connected to atherosclerosis. Type 2 diabetes mellitus has a variety of problems. One of them is dyslipidemia. Diabetic dyslipidemia is defined by an increase in triglyceride levels in the blood and a reduction in HDL cholesterol levels in the blood.

Dyslipidemia is a disorder characterised by aberrant lipid metabolism, as evidenced by elevated cholesterol, LDL cholesterol, and triglyceride levels in the blood plasma, as well as lower levels of HDL cholesterol. Dyslipidemia should be treated since it can lead to the development of atherosclerosis, which can raise the threat of stroke, cardiovascular disease, and even loss of life. Deranged lipids have been documented as dyslipidemia. Diabetic dyslipidemia is defined by an increase in triglyceride levels in the blood and a reduction in HDL cholesterol levels in the blood. Dyslipidemia is a disorder characterized by aberrant lipid metabolism, as evidenced by elevated cholesterol, LDL cholesterol, and triglyceride levels in the blood plasma, as well as lower levels of HDL cholesterol. Dyslipidemia should be treated since it can lead to the development of atherosclerosis. It may leads to increase the risk of cardiovascular disease. Insulin resistance has been linked to a lipid profile that is abnormal. Low levels of high density lipoproteins and excessive levels of triglycerides cause dyslipidemia. Diabetic dyslipidimia occurs when the reverse cholesterol transport pathway is disrupted. It is a major main influence for heart disease. Type 2 diabetes mellitus and insulin resistance are linked to abnormalities in plasma a lipoprotein. Hypercholesterolemia is a condition marked by elevated LDL, VLDL, and TG levels. The main problem is a decrease in LDL receptors and an increase in apoB lipoprotein. A rise in plasma LDL-cholesterol (LDL-C) levels in the liver and impairment in LDLC clearance may also be seen.

Insulin resistance and type 2 diabetes are linked to abnormalities in plasma a lipoprotein. Hypercholesterolemia is a condition marked by elevated LDL, VLDL, and TG levels. The main problem is a decrease in LDL receptors and an increase in apoB lipoprotein. An rise in plasma LDL cholesterol (LDLC) levels in the liver and impairment in LDLC clearance may also be seen.

Although cholesterol is mostly obtained through diet yet it may also stay generated endogenously. Bile acid metabolism, synthesis of steroid hormone and vitamin D requires cholesterol [1]. Cholesterol transport is essential because it provides the lipids, which are obligatory for the protection and healing of cell membrane of retinal neurons. Low density lipoprotein (LDL) receptors, which may absorb circulating LDL are found in the retinal pigmented epithe-

lium like, Muller cells. Interplay of neuronal and glial cells is used to regulate cholesterol metabolism. Cholesterol homeostasis is critical for the retina's structural and functional integrity.

Atherosclerosis and other associated illnesses are caused by hyperlipidemia, a modifiable risk factor. Both reduced and raised cellular cholesterol levels are hallmarks of brain diseases, like retinal neurodegenerative disease. Hypercholesterolemia has also been demonstrated to increase the production of nitric oxide (NO) synthase 2 in the retina. It promotes peroxidation of lipids. It results into oxidative injury of retina. Heterozygous familial hypercholesterolemia (FH) is the greatest genetic illness, which is autosomal dominant. [2]. It may affect roughly one out of every 200-300 people [3]. Serum LDL- cholesterol ranks are significantly excessive in people with FH. Mutations in one of the genes involved in LDL receptor-mediated catabolism, namely the LDL receptor and apolipoprotein B, cause FH. LDL receptor mutations affect 85-90% of FH patients, while apolipoprotein B mutations affect 1-12% of patients. The major feature of patients with FH is an elevated risk of developing atherosclerotic cardiovascular disease at a young age. The goal of the physical exam is to find indications of abnormal cholesterol deposits in the skin and eyes (tendon xanthomata, xanthelasmas, arcus cornealis). FH is diagnosed based on family and clinical history, physical examination, genetic testing and LDL-C levels.

Conflict of Interest

The author declares no conflict of interest.

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