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Original Research Article

Changes in Lipid Profile in Diabetic Patients and Normal Patients from West Bengal Region

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Abstract

Glycated haemoglobin (HbA1c) is a routinely used marker for long-term glycaemic control. Apart from functioning as an indicator for the mean blood glucose level, HbA1c also predicts the risk for the development of diabetic complications in diabetes patients. Many studies have proposed HbA1c to be used as a biomarker of both glycaemic control and dyslipidemia in type 2 diabetes mellitus. Hence based on above reported findings the present study was planned for Assessment of Changes.

Total 40 cases were enrolled in the present study. The present study was planned in Department of General medicine, ICARE Institute of Medical Sciences and Research & Dr. Bidhan Chandra Roy Hospital, Haldia, West Bengal, India. The study was conducted from the duration one year.The cases were divided in two study groups as Diabetic patients and normal patients of 20 cases each.

The data generated from the present study concludes that there is significant correlation was observed between HbA1c and various parameters of lipid profile, and there is no standardized protocol to compare the results of various other studies hence more structured and long term studies on larger no of patients are needed to validate HbA1c as a marker of dyslipidemia. Diabetic dyslipidemia or atherogenic dyslipidemia is characterized by low HDL, high TG and high small dense LDL. Early screening of diabetic patients for dyslipidemia and early intervention is required to minimize the risk of future cardiovascular mortality.

Keywords: Lipid Profile in Diabetic Patients, Normal patients, West Bengal, etc.

Introduction

Type 2 diabetes mellitus consists of an array of dysfunctions characterized by hyperglycemia and resulting from the combination of resistance to insulin action, inadequate insulin secretion, and excessive or inappropriate glucagon secretion. Type 2 diabetes mellitus consists of an array of dysfunctions characterized by hyperglycemia and resulting from the combination of resistance to insulin action, inadequate insulin secretion, and excessive or inappropriate glucagon secretion. Poorly controlled type 2 diabetes is associated with an array of microvascular,

macrovascular, and neuropathic complications. Microvascular complications of diabetes include retinal, renal, and possibly neuropathic disease. Macrovascular complications include coronary artery and peripheral vascular disease. Diabetic neuropathy affects autonomic and peripheral nerves.

Unlike patients with type 1 diabetes mellitus, patients with type 2 are not absolutely dependent on insulin for life. This distinction was the basis for the older terms for types 1 and 2, insulin dependent and non–insulin dependent diabetes.

However, many patients with type 2 diabetes are ultimately treated with insulin. Because they retain the ability to secrete some endogenous insulin, they are considered to require insulin but not to depend on insulin. Nevertheless, given the potential for confusion due to classification based on treatment rather than etiology, the older terms have been abandoned. [1] Another older term for type 2 diabetes mellitus was adult-onset diabetes. Currently, because of the epidemic of obesity and inactivity in children, type 2 diabetes mellitus is occurring at younger and younger ages. Although type 2 diabetes mellitus typically affects individuals older than 40 years, it has been diagnosed in children as young as 2 years of age who have a family history of diabetes. In many communities, type 2 diabetes now outnumbers type 1 among children with newly diagnosed diabetes.

Diabetes mellitus is a chronic disease that requires long-term medical attention to limit the development of its devastating complications and to manage them when they do occur. It is a disproportionately expensive disease; in the United States in 2012, the direct and indirect costs of diagnosed diabetes were estimated to be $245 billion; people with diagnosed diabetes had average medical expenditures 2.3 times those of people without diabetes. [2-3]

Type 2 diabetes is characterized by a combination of peripheral insulin resistance and inadequate insulin secretion by pancreatic beta cells. Insulin resistance, which has been attributed to elevated levels of free fatty acids and proinflammatory cytokines in plasma, leads to decreased glucose transport into muscle cells, elevated hepatic glucose production, and increased breakdown of fat.

A role for excess glucagon cannot be underestimated; indeed, type 2 diabetes is an islet paracrinopathy in which the reciprocal relationship between the glucagon-secreting alpha cell and the insulin-secreting beta cell is lost, leading to hyperglucagonemia and hence the consequent hyperglycemia. [4]

For type 2 diabetes mellitus to occur, both insulin resistance and inadequate insulin secretion must exist. For example, all overweight individuals have insulin resistance, but diabetes develops only in those who cannot increase insulin secretion sufficiently to compensate for their insulin resistance. Their insulin concentrations may be high, yet inappropriately low for the level of glycemia. Defects in insulin action and hyperglycemia could lead to changes in plasma lipoproteins in patients with diabetes. Alternatively, especially in the case of type 2 diabetes, the obesity/insulin-resistant metabolic disarray that is at the root of this form of diabetes could, itself, lead to lipid abnormalities exclusive of hyperglycemia.

With prolonged diabetes, atrophy of the pancreas may occur. A study by Philippe et al used computed tomography (CT) scan findings, glucagon stimulation test results, and fecal elastase-1 measurements to confirm reduced pancreatic volume in individuals with a median 15-year history of diabetes mellitus (range, 5-26 years). [5] This may also explain the associated exocrine deficiency seen in prolonged diabetes.

Beta-cell dysfunction is a major factor across the spectrum of prediabetes to diabetes. A study of obese adolescents by Bacha et al confirms what is increasingly being stressed in adults as well: Beta-cell dysfunction develops early in the pathologic process and does not necessarily follow the stage of insulin resistance. [6] Singular focus on insulin resistance as the "be all and end all" is gradually shifting, and hopefully better treatment options that address the beta-cell pathology will emerge for early therapy. In the progression from normal to abnormal glucose tolerance, postprandial blood glucose levels increase first. Eventually, fasting hyperglycemia develops as suppression of hepatic gluconeogenesis fails.

During the induction of insulin resistance (such as occurs with a high-calorie diet, steroid administration, or physical inactivity), increased glucagon levels and increased glucose-dependent insulinotropic polypeptide (GIP) levels accompany glucose intolerance. However, the postprandial glucagon like peptide-1 (GLP-1) response is unaltered. [7]

Genome-wide association studies of single-nucleotide polymorphisms (SNPs) have identified a number of genetic variants that are associated with beta-cell function and insulin resistance. Some of these SNPs appear to increase the risk for type 2 diabetes. Over 40 independent loci demonstrating an association with an increased risk for type 2 diabetes have been shown. [8]

Susceptibility to type 2 diabetes may also be affected by genetic variants involving incretin hormones, which are released from endocrine cells in the gut and stimulate insulin secretion in response to digestion of food. For example, reduced beta-cell function has been associated with a variant in the gene that codes for the receptor of gastric inhibitory polypeptide (GIPR). [9]

The high mobility group A1 (HMGA1) protein is a key regulator of the insulin receptor gene (INSR). [10] Functional variants of the HMGA1 gene are associated with an increased risk of diabetes. Amino acid metabolism may play a key role early in the development of type 2 diabetes. Wang et al reported that the risk of future diabetes was at least 4-fold higher in normoglycemic individuals with high fasting plasma concentrations of 3 amino acids (isoleucine, phenylalanine, and tyrosine). Concentrations of these amino acids were elevated up to 12 years prior to the onset of diabetes. [11] In this study, amino acids, amines, and other polar metabolites were profiled using liquid chromatography tandem mass spectrometry.

Although the pathophysiology of the disease differs between the types of diabetes, most of the complications, including microvascular, macrovascular, and neuropathic, are similar regardless of the type of diabetes. Hyperglycemia appears to be the determinant of microvascular and metabolic complications. Macrovascular disease may be less related to glycemia. Telomere attrition may be a marker associated with presence and the number of diabetic complications. Whether it is a cause or a consequence of diabetes remains to be seen. [12]

Thrombotic abnormalities (ie, elevated type-1 plasminogen activator inhibitor [PAI-1], elevated fibrinogen) and hypertension are also involved. Other conventional atherosclerotic risk factors (eg, family history, smoking, elevated LDL cholesterol) also affect cardiovascular risk.

Insulin resistance is associated with increased lipid accumulation in liver and smooth muscle, but not with increased myocardial lipid accumulation. [13] Persistent lipid abnormalities remain in patients with diabetes despite the use of lipid-modifying drugs, although evidence

supports the benefits of these drugs. Statin dose up-titration and the addition of other lipid-modifying agents are needed. [14]

Increased cardiovascular risk appears to begin prior to the development of frank hyperglycemia, presumably because of the effects of insulin resistance. Stern in 1996 [15] and Haffner and D'Agostino in 1999 [30] developed the "ticking clock" hypothesis of complications, asserting that the clock starts ticking for microvascular risk at the onset of hyperglycemia, while the clock starts ticking for macrovascular risk at some antecedent point, presumably with the onset of insulin resistance.

The question of when diabetes becomes a cardiovascular risk equivalent has not yet been settled. Debate has moved beyond automatically considering diabetes a cardiovascular risk equivalent. Perhaps it would be prudent to assume the equivalency with diabetes that is more than 5-10 years in duration.

The etiology of type 2 diabetes mellitus appears to involve complex interactions between environmental and genetic factors. Presumably, the disease develops when a diabetogenic lifestyle (ie, excessive caloric intake, inadequate caloric expenditure, obesity) is superimposed on a susceptible genotype. The body mass index (BMI) at which excess weight increases risk for diabetes varies with different racial groups. For example, compared with persons of European ancestry, persons of Asian ancestry are at increased risk for diabetes at lower levels of overweight. Hypertension and prehypertension are associated with a greater risk of developing diabetes in whites than in African Americans.

In addition, an in utero environment resulting in low birth weight may predispose some individuals to develop type 2 diabetes mellitus. Infant weight velocity has a small, indirect effect on adult insulin resistance, and this is primarily mediated through its effect on BMI and waist circumference. About 90% of patients who develop type 2 diabetes mellitus are obese. However, a large, population-based, prospective study has shown that an energy-dense diet may be a risk factor for the development of diabetes that is independent of baseline obesity. [16]

Some studies suggest that environmental pollutants may play a role in the development and progression of type 2 diabetes mellitus. A structured and planned platform is needed to fully explore the diabetes-inducing potential of environmental pollutants.

Type 1 diabetes, previously termed insulin-dependent diabetes mellitus, provides a much clearer understanding of the relationship among diabetes, insulin deficiency, and lipid/lipoprotein metabolism. In poorly controlled type 1 diabetes and even ketoacidosis, hypertriglyceridemia and reduced HDL commonly occur. Replacement of insulin in these patients may correct these abnormalities, and well controlled diabetics may have increased HDL and lower than average triglyceride levels.

The lipoprotein abnormalities commonly present in type 2 diabetes, previously termed noninsulin-dependent diabetes mellitus, include hypertriglyceridemia and reduced plasma HDL cholesterol. In addition, low density lipoprotein (LDL) are converted to smaller, perhaps more atherogenic, lipoproteins termed small dense LDL. In contrast to type 1 diabetes, this phenotype is not usually fully corrected with glycemic control. Moreover, this dyslipidemia often is found in prediabetics, patients with insulin resistance but normal indexes of plasma glucose. Therefore, abnormalities in insulin action and not hyperglycemia per se are associated with this lipid abnormality. In support of this hypothesis, some thiazoladinediones improve insulin actions on peripheral tissues and lead to a greater improvement in lipid profiles than

seen with other glucose-reducing agents. [17]

Several factors are likely to be responsible for diabetic dyslipidemia: insulin effects on liver apoprotein production, regulation of lipoprotein lipase (LpL), actions of cholesteryl ester transfer protein (CETP), and peripheral actions of insulin on adipose and muscle.

Glycated haemoglobin (HbA1c) is a routinely used marker for long-term glycaemic control. Apart from functioning as an indicator for the mean blood glucose level, HbA1c also predicts the risk for the development of diabetic complications in diabetes patients. Many studies have proposed HbA1c to be used as a biomarker of both glycaemic control and dyslipidemia in type 2 diabetes mellitus. Hence based on above reported findings the present study was planned for Assessment of Changes in Lipid Profile in Diabetic Patients and Normal Patients from Bihar Region.

Methodology:

Total 40 cases were enrolled in the present study. The present study was planned in Department of General medicine, ICARE Institute of Medical Sciences and Research & Dr. Bidhan Chandra Roy Hospital, Haldia , West Bengal, India.The study was conducted from the duration one year. The cases were divided in two study groups as Diabetic patients and normal patients of 20 cases each.

Serum samples were collected for FBS in tubes containing sodium fluoride and ammonium oxalate and for lipid profile 3ml venous blood was drawn aseptically in plain tubes. Serum glucose was determined by GOD-POD end point. Lipid Profile like Total Cholesterol (TC) was measured by CHOD-POD end point method; Triglycerides (TG) by the GPO-PAP end point method and High Density Lipoprotein (HDL) and Low Density Lipoprotein (LDL) by a Direct Enzymatic method. All the parameters which were under investigation were determined in the serum of the subjects by using commercially available reagent kits.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: Type 2 diabetes mellitus patients in the age range of 30-85 years.

Exclusion Criteria: 1. T2DM patients with concomitant diseases or conditions affecting lipid levels like chronic liver disease and hypothyroidism. 2. Patients on drugs like oral contraceptive pills, steroids and diuretics.

Results & Discussion:

Diabetes is characterized by chronic hyperglycemia and disturbances of carbohydrate, lipid and protein metabolism. [18] Dyslipidemia is one of the major risk factor for cardiovascular disease in Type 2 Diabetes mellitus, characterized by elevated Total cholesterol (TC), Triglycerides (TG), Low density lipoprotein (LDL) and decreased High density lipoprotein (HDL). [19] Because detection and treatment of dyslipidemia is one means of reducing Cardiovascular Disease (CVD) risk, determination of serum lipid levels in people with diabetes is now considered a standard of care. [20]

Diabetes causes about 5% of all deaths globally each year. The World Health Organization (WHO) report in 2010 estimates that the number of diabetic patients in Oman will increase up to 190% over the next 20 years, from 75,5000 in 2000 to 217,000 in 2025. [21]. Several previous studies have attempted to correlate blood glucose levels with serum lipid profile parameters. [22] Research findings show that mainly body fat is responsible for increase in prevalence of

this disease among the body composition components. [23]

The term diabetic dyslipidemia comprises a triad of raised triglycerides, reduced High Density Lipoprotein (HDL) and excess of small, dense Low Density Lipoprotein (LDL) particles. The lipid abnormalities are prevalent in diabetes mellitus because insulin resistance or deficiency affects key enzymes and pathways in lipid metabolism. The causal association between atherosclerosis and dyslipidemia is well established. In diabetes the associated hyperglycemia, obesity and insulin changes highly accelerate the progression to atherosclerosis. [24] Type 2 diabetes is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities, including reduced HDL cholesterol, a predominance of small dense LDL particles, and elevated triglycerides. These abnormalities occur in many patients despite normal LDL cholesterol levels. There is evidence that each of these dyslipidemic features is associated with increased risk of cardiovascular disease, the leading cause of death in patients with type 2 diabetes. [25]

For the interpretation of serum lipid reference values, the guidelines of National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) were followed. According to NCEP-ATPIII guidelines, hypercholesterolemia is defined as TC > 200 mg/dl, high LDL-C when value > 100 mg/dl, hypertriglyceridemia as TAG > 150 mg/dl and low HDL-C when value is < 40 mg/dl. Dyslipidemia was defined by presence of one or more than one abnormal serum lipid concentration. [26] In patients with diabetes, many studies have clearly established that complications are mainly due to chronic hyperglycemia that exerts its injurious to health effects through several mechanisms: dyslipidemia, platelet activation, and altered endothelial metabolism. [27-28] Both lipid profile and diabetes have been shown to be the important predictors for metabolic disturbances including dyslipidemia, hypertension and cardiovascular diseases. [29] Lipids play a vital role in the pathogenesis of diabetes mellitus. Dyslipidemia as a metabolic abnormality is frequently associated with diabetes mellitus. Abnormalities in lipid metabolism have been reported in patients with diabetes mellitus accompanied by the risk of cardiovascular arteriosclerosis. [30]

Table 1: Comparison of General Parameter

Type of Patients	Diabetic patients	Controlled study patients
Age Group	29 – 54 years	32 – 57 year
Males	14	16
Females	6	4

Table 2: Comparison of Bio Chemical Parameter

Bio Chemical Parameter	Diabetic patients	Controlled study patients
Triglycerides (mg%)	197.1 ± 21.4	174.1 ± 25.9
Fating glucose level (mg%)	158.1 ± 7.4	92.7 ± 6.8
Glycated haemoglobin (HbA1c) (%)	8.8 ± 1.3	6.2 ± 1.1
Total cholesterol (mg%)	184.1 ± 13.2	165.4 ± 12.5
High Density Lipid (mg%)	42.7 ± 4.5	52.1 ± 6.4
Low Density Lipid (mg%)	117.3 ± 13.8	95.8 ± 16.3

Jayesh et al [31] conducted a prospective study on western Indian population that comprised of 430 type 2 diabetes mellitus patients and 501 non diabetic control subjects. They found significant correlation of HbA1c with TC and LDL.

Zhe Yan et al [32] conducted a study on 128 type 2 diabetes mellitus patients in Sichuan, China. They found significant correlation of HbA1c with LDL. Eglal et al [33] a study on 50 type 2 diabetes mellitus patients in Khartoum Sudan, they found significant correlation of HbA1c with TG.

Alterations in lipid metabolism are recognized concomitant symptoms of diabetes mellitus. It is believed that even before the development of overt diabetes, insulin resistance and a prediabetic state impair the mechanism that suppresses fatty acid release from adipose tissue after food intake. [34] The resultant excess of free fatty acids leads to increased concentrations of triglyceride (TG)–rich particles (very low-density lipoproteins and chylomicrons) and TG enrichment of high- and low-density lipoprotein (HDL and LDL), affecting virtually every lipid and lipoprotein variable. [35] The end result is a dyslipidaemia that is characterized by elevated TG levels, the generation of small, dense LDL particles, and reduced HDL cholesterol (HDL-C) concentrations.

This combination of features is known by many designations, including atherogenic dyslipidaemia, dyslipidaemia of insulin res istance or the atherogenic lipoprotein phenotype. It contributes to the 2 to 4 times excess risk for cardiovascular disease observed in patients with type 2 diabetes mellitus compared with nondiabetic individuals. [36] It is also increasingly recognized that the presence of diabetes places most patients at the same near-term risk for a coronary event as that of a patient with existing coronary heart disease (CHD). Diabetes incidence is increasing rapidly in the general population, drawing attention to the role of atherogenic dyslipidaemia in the evolution of CHD among these patients. Attempts to correlate CHD incidence among patients with diabetes with the classic coronary risk factors, showed that these risk factors account for only 25% to 30% of the excess risk for CHD. [37]

Results strongly suggest that further investigations should relate the effects of dyslipidaemia and abnormalities of insulin resistance in type 2 diabetics. And ethnic specific patterns of lipid profile in type 2 diabetics regardless of their glucose levels, suggesting that ethnic specific strategies and guidelines on risk assessment and prevention of CVD due to dyslipidemia are required. [38]

Conclusion:

The data generated from the present study concludes that there is significant correlation was observed between HbA1c and various parameters of lipid profile, and there is no standardized protocol to compare the results of various other studies hence more structured and long term studies on larger no of patients are needed to validate HbA1c as a marker of dyslipidemia.

Diabetic dyslipidemia or atherogenic dyslipidemia is characterized by low HDL, high TG and high small dense LDL. Early screening of diabetic patients for dyslipidemia and early intervention is required to minimize the risk of future cardiovascular mortality.

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