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Assessment of Lipid Profiles Mainly Triglycerides in Diabetic Patients from Delhi Region

Dr. Umesh Rajak[1], Dr. Gyan Bhushan Raman[2*], Dr. Ravi Bhushan Raman[3], Dilip Kumar[4]

[1] Senior Resident, Department of Medicine, Vardhman Mahavir Medical College & Safdarjang Hospital, New Delhi, India

[2] Senior Resident, Department of Medicine, Vardhman Mahavir Medical College & Safdarjang Hospital, New Delhi, India

[3] Senior Resident, Department of Pathology, IGIMS, Sheikhpura, Patna, Bihar, India

[4] Professor, Head of Department, Vardhman Mahavir Medical College &Safdarjang Hospital, New Delhi, India

[*] Corresponding Author: Dr. Gyan Bhushan Raman

Abstract
Age adjusted incidence of coronary artery diseases is 3 to 5 times higher in both male & female diabetics compare to general population. Individuals with diabetes may have several forms of dyslipidemia leading to additive cardiovascular risk of hyperglycemia. So lipid abnormalities should be aggressively detected & treated as a part of comprehensive diabetic care. Hence based on the above literature findings the current study was planned to assess the triglyceride and serum lipid profiles in diabetic patients. The assessment of triglycerides and other serum lipids in diabetic patients were planned in the Department of Medicine in Vardhman Mahavir Medical College & Safdarjung Hospital. The 20 Diabetic patients and 20 controlled normal patients were enrolled in to the study. The age group of the patients are from 30-70 years. The biochemical parameters like Fasting glucose level, Glycated haemoglobin (HbA1c), Total cholesterol, Triglycerides, High Density Lipid, and Low Density Lipid were estimated. Dyslipidemia is one of the most common complications of diabetes mellitus. It is known to predispose to premature atherosclerosis and macrovascular complications. Since prevention is always better than cure, all persons with impaired glucose tolerance should switch over to a healthy lifestyle and diet, exercise regularly, avoid sedentary habits, increase fibre intake, and get regular check-ups of FBS and lipid profile to reduce the risk of coronary artery disease.

Keywords: triglycerides, lipid profile, diabetic patients. Etc

Introduction

Diabetes is a chronic disease, which occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces. This leads to an increased concentration of glucose in the blood (hyperglycaemia).

Type 1 diabetes (earlier known as insulin-dependent or childhood-onset diabetes) is characterized by a lack of insulin production. Type 2 diabetes (earlier known as non-insulin-dependent or adult-onset diabetes) is caused by the body's ineffective use of insulin. It often results from excess body weight and physical inactivity.

Gestational diabetes is hyperglycemia that is first recognized during pregnancy. It can lead to serious health risks for both the mother and child. Diabetes is a growing challenge in India with estimated 8.7% diabetic population in the age group of 20 and 70 years. The rising prevalence of diabetes and other non-communicable diseases is driven by a combination of factors - rapid urbanization, sedentary lifestyles, unhealthy diets, tobacco use, and increasing life expectancy. Obesity and overweight are the most important risk factors responsible for diabetes. Much of the diabetes burden can be prevented or delayed by behavioural changes favouring a healthy diet and regular physical activity [1].

Globally, an estimated 422 million adults are living with diabetes mellitus, according to the latest 2016 data from the World Health Organization (WHO). Diabetes prevalence is increasing rapidly; previous 2013 estimates from the International Diabetes Federation put the number at 381 million people having diabetes. The number is projected to almost double by 2030. Type 2 diabetes makes up about 85-90% of all cases. Increases in the overall diabetes prevalence rates largely reflect an increase in risk factors for type 2, notably greater longevity and being overweight or obese [2].

Diabetes mellitus occurs throughout the world, but is more common (especially type 2) in the more developed countries. The greatest increase in prevalence is, however, occurring in low- and middle-income countries including in Asia and Africa, where most patients will probably be found by 2030. The increase in incidence in developing countries follows the trend of urbanization and lifestyle changes, including increasingly sedentary lifestyles, less physically demanding work and the global nutrition transition, marked by increased intake of foods that are high energy-dense but nutrient-poor (often high in sugar and saturated fats, sometimes referred to as the Western pattern diet). The risk of getting type 2 diabetes has been widely found to be associated with lower socio-economic position across countries [3].

The WHO estimates that diabetes resulted in 1.5 million deaths in 2012, making it the 8th leading cause of death. However another 2.2 million deaths worldwide were attributable to high blood glucose and the increased risks of associated complications (e.g. heart disease, stroke, kidney failure), which often result in premature death and are often listed as the underlying cause on death certificates rather than diabetes [2].

Until recently, India had more diabetics than any other country in the world, according to the International Diabetes

Federation, although the country has now been surpassed in the top spot by China. Diabetes currently affects more than 62 million Indians, which is more than 7.1% of the adult population. The average age on onset is 42.5 years. Nearly 1 million Indians die due to diabetes every year [4].

According to the Indian Heart Association, India is projected to be home to 109 million individuals with diabetes by 2035. A study by the American Diabetes Association reports that India will see the greatest increase in people diagnosed with diabetes by 2030. The high incidence is attributed to a combination of genetic susceptibility plus adoption of a high-calorie, low-activity lifestyle by India's growing middle class [5].

The cluster of lipid abnormalities associated with type 2 diabetes is defined by a high concentration of TG and small dense LDL and a low concentration of HDL cholesterol. Plasma LDL cholesterol levels are generally normal. Insulin resistance is believed to contribute to this atherogenic dyslipidemia by increasing the hepatic secretion of VLDL and other apolipoprotein (apo) B-containing lipoprotein particles, as a result of increased free fatty acid flux to the liver [6-7]. This may also be the result of a diminished suppressive effect of insulin on apoB secretion, either at the level of the regulation of apoB degradation, or inhibition of microsomal TG transfer protein activity [8]. Through the action of cholesterol ester transfer protein, TGs are transferred from VLDL to HDL, creating TG-rich HDL particles, which are hydrolysed by hepatic lipase and rapidly cleared from plasma [9]. A similar cholesterol ester protein-mediated transfer of TGs from VLDL to LDL contributes to the formation of small dense LDL particles [10]. Other mechanisms, including impaired clearance of lipid and lipoproteins may also be involved.

In uncontrolled diabetes, serum triglycerides, Very Low Density Lipoproteins (VLDL), cholesterol are raised both at fasting & following fixed meal. In post fixed meal Chylomicrons remnants & Low Density Lipoproteins (LDL) remain high for longer period than normal. Total cholesterol & LDL are mild to moderate high in 1/3rd patients. On other end HDL remain significantly low particularly in type-2 diabetes patients with central obesity. Among changes in composition of Lipoproteins high proportion of small, dense triglyceride rich LDL & glycoxidation products of LDL are considered to be most atherogenic. Age adjusted incidence of coronary artery diseases is 3 to 5 times higher in both male & female diabetics compare to general population. Individuals with diabetes may have several forms of dyslipidemia leading to additive cardiovascular risk of hyperglycemia. So lipid abnormalities should be aggressively detected & treated as a part of comprehensive diabetic care. Hence based on the above literature findings the current study was planned to assess the triglyceride and serum lipid profiles in diabetic patients.

Materials & Methodology

The assessment of triglycerides and other serum lipids in diabetic patients were planned in the Department of Medicine in Vardhman Mahavir Medical College & Safdarjung Hospital. The 20 Diabetic patients and 20 controlled normal patients were enrolled in to the study. The age group of the patients are from 30-70 years. All the patients are informed consents. The entire patient's clinical history was collected. The study was conducted from Dec-2015 to July-2016. Approval of the Institutional ethical committee was taken prior to conduct of the study.

The biochemical parameters like Fasting glucose level, Glycated haemoglobin (HbA1c), Total cholesterol, Triglycerides, High Density Lipid, and Low Density Lipid were estimated.

The detail history was taken; relevant clinical examination and all routine investigations were performed. An informed consent was taken from every patient after full explanation of procedure. Every patient was advised for at least 12-14 hours overnight fasting and the 5 ml venous blood sample were collected in a disposable syringe on next morning (before breakfast) for the serum lipid profile.

Results & Discussion

The data from 20 diabetic patients and 20 non-diabetic patients were collected and shown in table 1 as below. The data were discussed with the previous findings.

Table 1: Comparison of BioChemical Parameter in 2 study groups

Group	Group I	Group II
Type of Patients	Diabetic patients	Controlled study patients
No. of Patients	20	20
Age Group	35 – 53 years	38 – 60 year
Males	12	15
Females	8	5
BioChemical Parameter	Observation	
Triglycerides (mg %)	198.4 ±41.3	182.9 ± 45.2
Fasting glucose level (mg %)	158 ± 15.6	98.2 ± 8.2
Glycated haemoglobin (HbA1c) (%)	8.2 ± 1.9	6.9 ± 0.8
Total cholesterol (mg %)	179.3 ± 19.5	171.8 ± 21.7
High Density Lipid (mg%)	42.8 ± 7.2	48.9 ± 6.8
Low Density Lipid (mg %)	95.6 ± 17.1	121.4 ± 24.8

The diabetic patients showed as usual increased in glucose level. The HbA1c level also found increased in the case study group as compared to controlled study group. There is no change in the levels of the total cholesterol, triglycerides, high density and low density lipids.

The concentration of serum sodium and chlorine were slightly lower than the controlled study. The serum potassium ion and bicarbonate level does not show major significant change.

Significant lower level of HDL in diabetic patients compared to nondiabetic subjects. Lower HDL cholesterol level is attributed to triglyceride enrichment by cholesterol ester transfer protein and increased hepatic triglyceride lipase activity [11]. Although liver is produced the HDL particles, a significant part of HDL are formed from remnant particles of TG-rich lipoproteins as metabolized. This metabolism is often defective in diabetes, lowering the production of HDL-C from the liver by protein which is called cholesterol ester transport protein (CETP) transports cholesterol ester away from HDL particles in exchange for TG from the VLDL particles. This transport protein lowers HDL-C in the blood, in addition, it promotes for small, dense LDL particles [12]. Lipid levels affected by glucose levels because metabolism of carbohydrates and lipid is interrelated to each other; because any disorder in metabolism of carbohydrate leads to a disorder in metabolism of lipid, so high concentration of cholesterol, triglycerides and a reduction in HDL cholesterol levels leads to insulin resistance with or without hyperglycemia which is related to qualitative changes in the lipid profile [13].

Diabetic patients have 2 to 3 times increased risk of coronary artery disease as compared to the non-diabetic. The causes for it are multiple, which include dyslipidemia, hypertension, obesity and smoking. Dyslipidemia which is usually present in diabetics in the form of increased triglycerides and decreased HDL cholesterol level. It confers much of accelerated and increased early risk of coronary artery disease (CAD), cerebrovascular disease, peripheral vascular disease and sudden cardiac death. Patients with type 1 diabetes mellitus with very good glycemic control can have normal postprandial lipid levels.

However, as glycemic control worsens because of inadequate insulin, lipoprotein lipase activity decreases, and postprandial hyperlipidemia can result. In patients with type 2 diabetes, the underlying insulin resistance can be associated with mild reductions in lipoprotein lipase, but overproduction of VLDL is a major problem. Increased VLDL competes with chylomicrons for lipoprotein lipase, resulting in postprandial hyperlipidemia in most patients with type 2 diabetes. The fasting triglyceride level is, therefore, a predictor of the severity of postprandial hyperlipidemia.

Disordered metabolism of VLDL and/or chylomicrons may be proatherogenic. Chylomicron remnants and VLDL or its remnants enter the subendothelial space of the vessel wall where the atherogenic process is initiated. The cholesterol content of the triglyceride-rich lipoproteins is important, because persons with more cholesterol enriched particles will be at greater risk for atherosclerotic cardiovascular disease. The plasma concentration of very-low-density lipoprotein, intermediate-density lipoprotein, and low-density lipoprotein cholesterol is linearly related to the flux of these particles into the arterial wall [14].

In Singapore, fasting serum TG levels, but not HDL and LDL concentrations, were found to be higher among persons with type 2 DM than those of non-diabetics [15]. High TG levels cause increased transfer of cholesteryl esters from HDL-C and LDL-C to very VLDL-C via cholesteryl ester transfer protein, thus forming cholesteryl ester depleted, small dense LDL-C particles [16]. These small dense lipoprotein particles are taken up by arterial wall macrophages, resulting in atherogenesis [17].

HDL acts by enhancing the removal of cholesterol from peripheral tissues and so reduces the body's cholesterol pool. Type 2 DM was usually associated with low plasma levels of HDL-C [18]. Low HDL concentrations are often accompanied by high TG levels as seen in this study and this combination has been strongly associated with an increased risk of CHD [19]. The relative insulin deficiency that occurs in type II diabetes mellitus impairs the action of lipoprotein lipase and results in lower HDL cholesterol levels and higher TG levels, which may improve with improved glycaemic control [20].

Conclusion

Hyperlipidemia is one of the most common complications of diabetes mellitus. It is known to predispose to premature atherosclerosis and macrovascular complications. Since prevention is always better than cure, all persons with impaired glucose tolerance should switch over to a healthy lifestyle and diet, exercise regularly, avoid sedentary habits, increase fibre intake, and get regular check-ups of FBS and lipid profile to reduce the risk of coronary artery disease.

References

1. http://www.searo.who.int/india/topics/diabetes_mellitus /en/
2. World Health Organization, Global Report on Diabetes. Geneva, 2016. Accessed 30 August 2016
3. Agardh E, *et al*. Type 2 diabetes incidence and socio-economic position: a systematic review and meta-analysis." International Journal of Epidemiology. 2011; 40(3):804-818.
4. Gale Jason. India's Diabetes Epidemic Cuts Down Millions Who Escape Poverty". Bloomberg. Retrieved, 2010-2012.
5. Kleinfield NR. Modern Ways Open India's Doors to Diabetes. New York Times. Retrieved, 2006, 2012.
6. Krauss RM, Siri PW: Dyslipidemia in type 2 diabetes. Med Clin N Am 2004; 88:897-909 Cross Ref Pub Med Google Scholar
7. Laws A, Hoen HM, Selby JV, Saad MF, Haffner SM, Howard BV. Differences in insulin suppression of free fatty acid levels by gender and glucose tolerance status: relation to plasma triglyceride and apolipoprotein B concentrations: Insulin Resistance Atherosclerosis Study (IRAS) Investigators. Arterioscler Thromb Vasc Biol. 1997; 17:64-71.
8. Malmstrom R, Packard CJ, Caslake M, Bedford D, Stewart P, Yki-Järvinen H, *et al*. Defective regulation of triglyceride metabolism by insulin in the liver in NIDDM. Diabetologia. 1997; 40:454-462.
9. Hopkins GJ, Barter PJ. Role of triglyceride-rich lipoproteins and hepatic lipase in determining the particle size and composition of high density lipoproteins. J Lipid Res. 1986; 27:1265-1277.
10. Berneis KK, Krauss RM. Metabolic origins and clinical significance of LDL heterogeneity. J Lipid Res. 2002; 43:1363-1379
11. DeFronzo RA. The triumvirate: beta cell, muscle, livec a collusion responsible for NIDDM. Diabetes. 1988; 37:667-687.
12. Chatterjee MN, Shinde R. Text book of medical laboratory technology. Metabolism of carbohydrates. (Jaypee Brothers Medical publisher) Sixth edition. Delhi-India, 2005, 266-330.
13. Del Prato S., Bonadonna, RC, Bosom E, *et al*. Characterization cellular defects of insulin action in type 2 (non-insulin dependent) diabetes mellitus. I Clininvest. 1993; 91:4M-494.
14. Golay A. HDL metabolism in NIDDM measurement of HDL turn over using titrated HDL J. Clin Endocrinol Metab. 1987; 65:512-8.
15. Hughes K, Choo M, Kuperan P, Ong CN, Aw TC. Cardiovascular risk factors in non-insulin dependent diabetics compared to non-diabetic controls: a population based survey among Asians in Singapore. Atherosclerosis. 1998; 136:25-31.
16. Sniderman AD, Scantlebury T, Cianfione K. Hypertriglyceridemic hyperpob: the unappreciated atherogenic dyslipidemia in type 2 diabetes mellitus. Ann Intern Med. 2001; 135:447-59.
17. Gowri MS, Vander Westhuyzen DR, Bridges SR, Anderson JW. Decreased protection by HDL from poorly controlled type 2 diabetic subjects against LDL oxidation may be due to the abnormal composition of HDL. Arterioscler Thromb Vasc Biol. 1999; 19:2226-33.
18. Fossati P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen

peroxide. Clin Chem. 1982; 28:2077-80.
19. Assman G, Schulte H. Relation of HDL cholesterol and TG's to incidence of atherosclerotic coronary artery disease. Prospective cardiovascular. Munster study. Am J Cardiol. 1992; 70:733-7.
20. Brunzell JD, Chait A. Diabetic dyslipidemia: pathology and treatment. In: Porte DJ, Sherwin RS, editors. Ellenberg and Rifkin's Diabetes Mellitus. 5th