ijlpr 2023; doi 10.22376/ijlpr.2023.13.3.L68-L72

International Journal of Life science and Pharma Research

ISSN 2250-0480

Research Article

Lipid Profile in Type 2 Diabetes Mellitus

Study of Lipid Profile in Type 2 Diabetes Mellitus Patients in South Chennai Population.

V. P. Nivedhini [1] and Dr. A.Jamuna Rani [2]

[1] *Tutor, Department of Biochemistry, Sree Balaji Medical College & Hospital, Chennai.*

[2] *Professor, Department of Biochemistry, Sree Balaji Medical College & Hospital, Chennai.*

Abstract: Diabetes Mellitus is caused by the decreased utilization of glucose by tissues and is attributed either to hypoinsulinemia or hyperinsulinemia induced by the overstimulation of insulin-secreting beta cells. Patients with diabetes mellitus, obesity and dyslipidemia are prone to Metabolic syndrome. The study aims to estimate and compare the lipid profile in type 2 diabetes mellitus patients and healthy individuals. This case-control study was undertaken in type II Diabetic patients, where 48 were male, and 45 were female. The diabetic subjects were compared with 33 male and 55 Healthy female subjects who volunteered as controls. Venous blood samples from healthy and diabetic subjects were collected and used to measure routine parameters such as fasting blood sugar (FBS), post-prandial blood sugar(PPBS), Total cholesterol, HDL, and TGL. The Fasting Plasma Glucose and post-prandial glucose were significantly increased among diabetic subjects than the healthy controls. The triglyceride levels in cases had elevated levels in comparison to controls. HDL showed a marginal decrease in the diabetic group than the control group. Mean LDL levels in people with diabetes increase compared to the healthy controls. Besides, the VLDL levels also significantly increased among people with diabetes. There was a substantial increase in FBS and PPBS levels among the diabetic subjects compared to the healthy controls. The lipid profile constituting Total cholesterol, Triglycerides, HDL, LDL and VLDL levels showed the following changes: There was a significant increase in Total cholesterol, TGL, VLDL, and LDL levels, whereas the HDL levels were decreased among the diabetic subjects. Patients with type 2 diabetes mellitus must keep their blood sugar levels under control through follow-ups because uncontrolled diabetes mellitus leads to lipid dysfunction, which ultimately increases the risk of coronary artery disease.

Keywords: Diabetes mellitus, High-Density Lipoprotein (HDL), Triglyceride (TGL), Low-Density Lipoprotein (LDL).

***Corresponding Author**

Dr.A.Jamuna Rani , Professor, Department of Biochemistry, Sree Balaji Medical College & Hospital, Chennai.

Received On 2 July, 2022

Revised On 24 February, 2023

Accepted On 1 February, 2023

Published On 1 May, 2023

Funding This research did not receive any specific grant from any funding agencies in the public, commercial or not for profit sectors.

Citation V. P. Nivedhini and Dr. A.Jamuna Rani , Study of Lipid Profile in Type 2 Diabetes Mellitus Patients in South Chennai Population..(2023).Int. J. Life Sci. Pharma Res.13(3), L68-L72 http://dx.doi.org/10.22376/ijlpr.2023.13.3.L68-L72

1. INTRODUCTION

Diabetes Mellitus is a metabolic disorder that primarily affects carbohydrate metabolism, causing glucose to be underutilized and resulting in hyperglycemia. [1] Himsworth predicted that Diabetes Mellitus, in which the lesion is caused by a decreased capacity of the tissues to utilize glucose, is attributed to either hypoinsulinemia or hyperinsulinemia. However, both factors may work simultaneously. [2] Environmental and genetic factors cause the fundamental molecular defects that cause hyperinsulinemia and hypoinsulinemia. Environmental factors such as eating habits and physical fitness significantly contribute to insulin-dependent diabetes mellitus. Diabetes is also portrayed as a "geneticist's nightmare" when the mode of inheritance is considered. Obese people are more likely to develop diabetes than people with a family history of diabetes, according to studies. [3] Diabetes is broadly classified as follows: s

Type 1: This category includes about 5-10% of people with diabetes. Patients usually present with sudden onset of symptoms such as polyuria, polydipsia, and weight loss. Patients with insulinopenia have lost pancreatic islet cells and rely on insulin to maintain life and prevent ketosis.

Type 2: diabetes was previously referred to as Non-Insulin Dependent Diabetes Mellitus (NIDDM) or Adult Onset Diabetes. These patients have few symptoms, are not prone to ketosis, and are not insulin dependent. [1] The global epidemic is mainly Type 2 Diabetes. World Health Organization has projected a 122% rise in worldwide diabetic cases from 1995 to 2025. India is expected to have the most significant increase. This twofold rise is attributed to a rising population, ageing, obesity, unhealthy diets, and a sedentary lifestyle.[4] According to the Diabetes Atlas 2012 published by the International Diabetes Federation, the number of people with Diabetes in India is currently around 61.3 million. It is expected to rise to 101.2 million by 2030.[5] A study by Ramachandran *et al.* has shown an increased incidence of diabetes among the Chennai population. This rise is attributed to the change in lifestyle resulting from 100% urbanization and industrialization leading to less exercise, more fast food culture, and oxidative stress.[6] The current study aims to estimate and compare the lipid profile in type 2 diabetes mellitus patients and healthy individuals. In addition, it is done to identify any abnormalities in the lipid profile to prevent complications.

2. MATERIALS & METHODS

A total of 93 (male 48, female 45) cases with Type 2 diabetes in the age group of 30-50 years attending the Diabetic OP of Sri Balaji Medical College & Hospital were enrolled for the study and were compared with eighty-eight (88) age and sex-matched healthy controls (male 33 and female 55).

2.1. Sample Size Calculation[7]

The Calculated sample size was to be 71±10 in each group.

2.2. Inclusion criteria for cases

- Patients diagnosed as type 2 diabetics between 30 and 50 years were chosen for this study.
- Diabetic patients are devoid of related disorders like hypertension, Ischaemic heart disease, oxidative stress and patients under medication for diabetes.

2.3. Inclusion criteria for Healthy controls

- Healthy subjects who were not consuming any antioxidant supplements.

2.4. Exclusion Criteria for cases

- Chronic illness.
- Post-menopausal women.
- Smokers and alcoholics.

2.5. Exclusion criteria for Healthy controls

- Post-menopausal women.
- Smokers and alcoholics.

2.6. Ethical Clearance statement

Ethical clearance for the study was obtained from the Ethical Clearance Committee of SBMCH. The ethical clearance reference number: is CSP/19/SEP/83/356. The study protocol was briefed to the study participants as we gave results of all test parameters except those of special investigation to the participants. Informed written consent was obtained from patients and healthy controls. 4ml venous blood was drawn from the patients in the fasting condition, from which 1ml was taken in a tube with the oxalate-fluoride mixture for estimating the remaining samples of Fasting Plasma Glucose (FPS). Post Prandial Plasma Glucose (PPBS): 1ml venous blood sample, 2 hours after food, was collected into a tube with the oxalate-fluoride mixture for PPPS estimation.

3. METHODS

- Estimating FBS & PPBS was done by Glucose Oxidase Peroxidase (GOD POD) Method (Enzymatic method).

4. ESTIMATION OF LIPID PROFILE

- Total Cholesterol - Liquid Cholesterol oxidase and peroxidase method (CHOD-POD) [Spinreact kit].
- Triglycerides - Glycerol Phosphate Dehydrogenase and Peroxidase method (GPO-POD). [Spinreact kit].
- High-Density Lipoprotein - HDL Precipitant Diatek kit
- Low-Density Lipoprotein (LDL)- The concentration of LDL-Cholesterol can be calculated from the results of a profile including Total cholesterol, HDL-C and triglycerides using the Friedwald equation.

- ***Friedwald equation***

Low Density Lipoprotein C= Total cholesterol - (VLDL + HDL Cholesterol)

- Very Low-Density Lipoprotein: VLDL levels are calculated values. It is obtained by dividing the Triglyceride level by 5 (TGL / 5).

5. STATISTICAL ANALYSIS

Cases and controls had their means, and standard deviations (SD) calculated. The software used to perform the statistical analysis is Statistical Package for the Social Sciences (SPSS) version 19 software was used to perform the statistical analysis. Statistical analysis was done using SPSS 19 software.

The outcome variables, fasting blood sugar, post-prandial blood sugar, total cholesterol, HDL-C, LDL-C, VLDL, and TGL were compared between the cases and controls using the Student *t*-test (two-tailed) to find the significance between the case and control groups. The p-value <0.05 is considered statistically significant.

6. RESULTS

6.1. Study design

A Comparative study of 93 diabetic patients with 88 controls to assess the lipid profile in Type 2 diabetes mellitus.

The age distribution of the study population was
47 diabetic subjects between the age group of 30-40 yrs
46 diabetic subjects between the age group of 41-50 yrs
47 healthy controls between the age group of 30-40 yrs
41 healthy controls between the age group of 41-50 yrs
Samples and controls are age-matched with a p-value of 0.397, showing no significant difference

6.2. Gender distribution

The study population comprised 48 males and 45 females of the diabetic subjects and was compared with 33 males and 55 females in the healthy controls.

Table 1: BMI distribution

BMI (kg/m^2)	Controls			Cases		
	No	%	Mean±SD	No	%	Mean±SD
<18.5	4	4.5	17.3±1.4	1	1.1	15.8±1
18.5-25.0	56	63.6	22.5±1	63	67.7	27.8±2.9
25.1 -30.0	24	27.3	27.1±1.5	26	28.0	27.3±1.7
>30.0	4	4.5	30.5±2.6	3	3.2	22.1±1.3
Total	88	100	24.08±3.5	93	100	23.68±3.36

Samples are BMI matched with P=0.425. p-value <0.05 is considered statistically significant. The mean±SD BMI of the case group (24.08±3.5) is comparatively higher than the control group (23.68±3.36), and there is no statistical significance.

Table 2: Comparison of mean and SD of Age, BMI, FBS, PPBS, total cholesterol, triglyceride, HDL, LDL and VLDL between case and control groups.

Parameter	Healthy Controls (Mean ± SD)	Diabetic patients (Mean ± SD)	p-value
Age			0.397
BMI	24.08±3.5	23.68±3.36	0.425
Fasting glucose (mg/dl)	88.22±13.25	169.63±69.43	t=10.753; P<0.001**
Postprandial glucose (mg/dl)	112.33±24.35	244.46±88.19	t=13.499; P<0.001**
Total cholesterol (mg/dl)	171.36±27.44	185.19±33.12	t=3.0405; P=0.003**
Triglyceride (mg/dl)	108.57±24.56	153.35±51.76	t=7.333; P<0.001**
HDL (mg/dl)	40.37±3.51	36.67±5.75	t=5.168; P<0.001**
LDL (mg/dl)	109.48±27.84	134.18±49.04	t=4.117; P<0.001**
VLDL (mg/dl)	21.89±4.94	30.36±10.12	t=7.053; P<0.001**

The significance of sugar parameters and lipid profile was analyzed by Student t-test (two-tailed, independent). The p-value ** is more significant for the parameters. As per WHO criteria, the normal FBS levels are 70-100 mg/dl. Values ≥126 mg/dl is the diagnostic cut-off for Diabetes Mellitus. In our study, the Fasting Plasma Glucose levels are significantly increased among diabetic subjects compared to the healthy controls. The values are expressed as Mean ± SD. The FBS values of the diabetic subjects are 169.63 ± 69.43 mg/dl, and the FBS of healthy controls are 88.22 ± 13.25 mg/dl. The difference is more significant. The p-value is <0.001. PPBS levels, according to WHO criteria, are 90-140 mg/dl. In our study, the FBS levels of diabetic subjects are elevated. The FPS is expressed as Mean ± SD. The FPS values of people with diabetes are 244.46 ± 88.19mg/dl. The healthy controls had 112.33 ± 24.35mg/dl values with an enormously significant p-value of < 0.001.

6.3. Lipid profile

According to WHO criteria, the average serum cholesterol level ranges from 150-200mg/dl. From Table 2, Our results expressed as mean ± SD in controls is 171.36±27.44 mg/dl, and cases are 185.19±33.12 mg/dl of serum cholesterol level. The p-value is 0.003 showing a strong significance. The normal serum Triglyceride levels range from 50-200mg/dl. From Table 2, In this study, Type 2 diabetic subjects had the serum TGL levels expressed as Mean ± SD to be 108±24.56mg/dl compared to healthy controls who had 185.57±24.56 mg/dl showing a vital significance with p value <0.001. According to WHO, the HDL range in healthy controls is 30-60 mg/dl. From Table 2, In the current study, HDL in diabetic patients expressed as Mean ± S.D.is 40.37±3.51 mg/dl and in the case of healthy controls 36.67± 5.75 mg/dl with more significant p-value of < 0.001. The Normal serum level of LDL is less than 180mg/dl. In type 2 diabetic patients, the mean value expressed as Mean ± was 134.18 ± 49.04 mg/dl. In healthy controls, the values were 109.48 ± 27.84 mg/dl with more significant p-value of < 0.001. From Table 2, In our study, diabetic subjects reported VLDL levels of 30.36 ± 10.12 mg/dl. The VLDL Level in healthy control was 21.89 ± 4.94 mg/dl with more significant p-value of < 0.001.

7. DISCUSSION AND ANALYSIS

Diabetes mellitus is a diverse group of metabolic diseases characterized by abnormally high blood sugar levels, resulting in deficiencies in insulin secretion, insulin action, or both.[8] The current study was undertaken in type II Diabetic

patients. The Glycemic status of the case group was assessed through the analysis of Fasting Plasma Glucose (FBS) and Post Prandial Plasma Glucose (PPBS). The results were expressed as Mean±S.D. In diabetic subjects, the FPS levels were elevated (169.63 ± 69.43 mg/dl) compared to the healthy controls (88.22 ± 13.25 mg/dl), which is more significant. This study found the PPPS values among diabetic subjects and controls are 244.46 ± 88.19 mg/dl) & 112 ± 24.35 mg/dl. Significantly increased FBS, PPBS and HbA_1c indicate poor glycemic control in diabetic patients.

The Lipid Profile

T2DM can also be caused by lipid imbalance. The exact pathogenesis is only partially understood but primarily involves increased triglycerides and decreased HDL. An increase in triglyceride levels causes an increase in free fatty acid levels, which disrupts the cascade connecting insulin receptors to glucose transporters and causes subclinical inflammation. This leads to the dysfunction of insulin receptors and β-cells. This explains why regulating hyperglycemia in patients with elevated triglyceride levels is more complex than in patients with normal TG levels. It also clarifies the reduction in anti-diabetic treatment intensity once hypertriglyceridemia has resolved.[9] The circulatory lipids play an essential role in the progression of diabetes, not only by way of hyperlipidemia and the development of atherosclerosis. [10, 11] Renuka Suvarna *et al*. reported a suggestive significant increase in Total cholesterol in their study. In contrast, we have a more substantial difference in the Total cholesterol level of cases (185.19±33.12 mg) and controls (171.36 ±27.44 mg). [7] In the present study, the triglyceride levels of the diabetic subjects were significantly increased (185 ± 24.56 mg/dl) compared to the controls (108 ± 24.56 mg/dl). The study by Renuka *et al*. correlates with our study reporting a significant difference in triglyceride levels between the controls and cases. This is because of the excessive lipolysis in adipose tissues of diabetics leading to increased free fatty acids in circulation. The free fatty acids undergo esterification in the liver to form triglycerides. Studies report increased triglyceride levels in both types of diabetes than in control. [12] Though there is a significant decrease in HDL among diabetic cases (36.67 ± 5.75mg/dl) compared with the study's healthy controls (40.37 ± 3.51 mg/dl), the levels are in the normal range only. Similar results have been observed by *Renuka Suvarna et al*., with a marginal decrease in HDL levels in the case of diabetics without complications.[13] Inoue *et al*. have reported that the size and composition of HDL are altered in diabetes. This could be the reason for the minimal decrease in HDL. In type 2 diabetes, hypertriglyceridemia is caused by insulin resistance, hyperglycemia, and hyperinsulinemia - accelerated lipolysis after insulin resistance increases the availability of free fatty acids. In contrast, hyperglycemia and hyperinsulinemia stimulate triglyceride synthesis in the liver via the activation of ChREBP and SREBP1c, respectively. As a result, the mass and activity of CETP increase. In these conditions, CETP action enriches HDL particles with triglycerides while depleting them of cholesteryl esters, resulting in lower HDL cholesterol levels. As previously stated, hepatic lipase, which is upregulated by hyperglycemia and has increased activity in states of insulin resistance, rapidly metabolizes the triglyceride-rich HDL, forming small HDL particles that undergo accelerated clearance. Furthermore, hepatic lipase accelerates the depletion of HDL cholesteryl esters. [14,15] This could also be due to a significant increase in Triglycerides rich lipoproteins providing an increase in the substrate for the cholesterol ester transfer protein. This promotes cholesterol flux from HDL particles, leading to decreased HDL levels in Type 2 Diabetes. [16] According to the study reports of Renuka Suvarna *et al*. The LDL cholesterol does not show any difference between the controls and cases, whereas we have seen a significant difference. Similar results have been reported by Samatha et al. [17] This is due to the dyslipidaemia associated with type 2 diabetes. Mean levels of LDL in the case group (diabetic subjects) were 134 ± 49.04 mg/dl, whereas the mean value of the control group was 109.48 ± 27.84 mg/dl. The VLDL levels also showed a significant increase among people with diabetes (30.36 ± 10.12) compared to healthy controls (21.89 ± 4.94). The raised levels of TGL & VLDL in the patients result in increased LDL, a metabolite of VLDL.

8. CONCLUSION

The circulatory lipids play an essential role in the progression of diabetes. In this study, the diabetic subjects had significantly increased triglyceride levels compared to the control. The raised levels of TGL & VLDL in the patients result in increased LDL, a metabolite of VLDL. Uncontrolled diabetes mellitus may lead to increased TGL and VLDL levels which might lead to the development of coronary artery disease. Epidemiological data from human studies revealed that the coexistence of diabetes with other risk factors, particularly dyslipidemia, confers a significantly higher CVD risk. Patients with type 2 diabetes mellitus must keep their blood sugar levels under control through follow-ups because uncontrolled diabetes mellitus leads to lipid dysfunction, which ultimately increases the risk of coronary artery disease. Body weight reduction is associated with improved lipid profile (lower TG, higher HDL), glycemic control, and IR; hence lifestyle modifications like exercise, healthy diet and follow-up with the treatment are required to keep risks at a minimum.

9. ACKNOWLEDGEMENT

We acknowledge the Department of Biochemistry, Departments of General Medicine of Sree Balaji Medical College and Hospital for supporting us in completing this project.

10. ETHICAL STANDARDS

The study involved human participants following the ethical standards of the tertiary healthcare institution where the study was conducted.

11. AUTHOR'S CONTRIBUTION STATEMENT:

Dr A. Jamuna Rani contributed to the study conception, design, and statistical analysis of the findings, and V.P.Nivedhini contributed to data collection and drafting of the manuscript.

12. CONFLICT OF INTEREST

Conflict of interest declared none.

13. REFERENCES

1. Tietz textbook of clinical biochemistry – Carl A. Burtis & Edward R. Ashwood-III Edition pg. 776.
2. Schadewalt H. The history of diabetes mellitus. In: Van Englehardt D, editor. Diabetes, its medical and cultural history. Berlin Springer Verlag; 1983. p. 43-100.
3. Neel JV. Diabetes Mellitus: A geneticist's nightmare. In: Creutzefeldt W, Kobberling JK, Neel JV, editors. The genetics of diabetes. New York: Springer Verlag; 1976. p. 1-11.
4. World Diabetic scenario published by International Diabetes Federation and World Health Organization. Available from: http://www.idf.org/worlddiabetesday/ [cited 26/1/2023].
5. Epidemiology of type 2 diabetes: Indian scenario Mohan V, Sandeep S, et al. Madras Diabetes Research Foundation & Dr Mohan's diabetes specialities centre, Chennai, India. Indian J Med Res. 2007;125(March):217-30.
6. Ramachandran A, Snehalatha C, Vijay V, King H. Impact of poverty on the prevalence of diabetes and its complications in urban southern India. Diabet Med. 2002;19(2):130-5. doi: 10.1046/j.1464-5491.2002.00656.x, PMID 11874429.
7. Suvarna R, Rao SS, Joshi C, Kedage V et al. Paraoxonase activity in type 2 diabetes mellitus patients with and without complications. J Clin Diagn Res. 2011 February;5(1):63-5.
8. Chakraborty T, Gupta S, Saini V, Talukdar A. Biomarkers: an essential tool for diagnosing and treating diabetes mellitus. Int J Life Sci Pharm Res. 2021 March;11(2):123-9. doi: 10.22376/ijpbs/lpr.2021.11.2.P123-129.
9. Parhofer KG. Interaction between glucose and lipid metabolism: more than diabetic dyslipidemia. Diabetes Metab J. 2015;39(5):353-62. doi: 10.4093/dmg.2015.39.5.353, PMID 26566492.
10. Chen X, Scholl TO, Leskiw M, Savaille J, Stein TP. Differences in maternal circulating fatty acid composition and dietary fat intake in women with gestational diabetes mellitus or mild gestational hyperglycemia. Diabetes Care. 2010 September;33(9):2049-54. doi: 10.2337/dc10-0693, PMID 20805277.
11. Han X., Yang J., Yang K., Zhao Z., Abendschein D.R., Gross R.W. Alterations in myocardial cardiolipin content and composition occur at the very earliest stages of diabetes: a shotgun lipidomics study. Biochemistry. 2007 May 29;46(21):6417-28. doi: 10.1021/bi7004015, PMID 17487985.
12. Pi J, Zhang Q, Fu J, Woods C.G., Hou Y., Corkey B.E., et al. ROS signalling, oxidative stress and Nrf2 in pancreatic beta-cell function. Toxicol Appl Pharmacol. 2010 April 1;244(1):77-83. doi: 10.1016/j.taap.2009.05.025, PMID 19501608.
13. Zhang X, Bao Y, Ke L, Yu Y. Elevated circulating free fatty acids levels causing pancreatic islet cell dysfunction through oxidative stress. J Endocrinol Invest. 2010 June;33(6):388-94. doi: 10.1007/BF03346609, PMID 19915385.
14. Farbstein D, Levy AP. HDL dysfunction in diabetes: causes and possible treatments. Expert Rev Cardiovasc Ther. 2012;10(3):353-61. doi: 10.1586/erc.11.182, PMID 22390807.
15. Ikeda Y, Suehiro T, Inoue M, Nakauchi Y., Morita T., Arii K., et al. Serum paraoxonase activity and its relationship to diabetic complications in patients with non-insulin-dependent diabetes mellitus. Metabolism. 1998 May;47(5):598-602. doi: 10.1016/s0026-0495(98)90246-3, PMID 9591753.
16. Gowri M.S., Van der Westhuyzen D.R., Bridges S.R., Anderson J.W. 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(9):2226-33. doi: 10.1161/01.atv.19.9.2226, PMID 10479666.
17. Samantha P, Venkateswarlu M, Prabodh S. Lipid profile levels in type 2 diabetes mellitus from the tribal population of Adilabad in Andhra Pradesh, India. J Clin Diagn Res. 2012;6(4):593-6.