Original Research Article

A study of glycemic status and lipid profile of obese and non obese type II diabetic patients

S Ramya[1*], V Hariharan[2]

[1]Assistant Professor, Department of Biochemistry, Annapoorana Medical College and Hospital, Salem, Tamil Nadu, INDIA.
[2]Assistant Professor, Department of Biochemistry, Karpagam Faculty of Medical Sciences and Research, Coimbatore, Tamil Nadu, INDIA.
Email: drramyasathish@gmail.com

Abstract

Background: Diabetes mellitus is a group of metabolic disease characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. **Methodology:** The study was approved by the Institutions Human Ethics Committee and informed consents were obtained from all participants. 48 (males =16, females=32) known Type 2 diabetic patients aged 35-55 yrs on oral hypoglycemic drugs were Selected in diabetic out-patient department of tertiary care hospital. Independent 't' test was employed to compare groups and p value less than 0.05 was considered significant. Statistical analysis was performed using SPSS software. **Result:** The type 2 diabetic patients were divided in to two groups. Group 1 with BMI ≥ 23; Group 2 with BMI ≤ 23. There was no significant difference in systolic and diastolic BP between obese and non-obese groups. Serum insulin, HbA_1C, HOMA-IR were significantly increased in group 1. There was no significant difference in fasting plasma glucose and lipid profile parameters between obese and non-obese groups. **Conclusion:** The findings of our study reveal that there is significant increase in fasting plasma glucose, HbA_1C, HOMA-IR but not much significant difference in other biochemical parameters.

Key Words: Glycemic Status, Lipid Profile, Type II Diabetes, HbA1C, HOMA-IR

[*]**Address for Correspondence:**
Dr. S Ramya, Assistant Professor, Department of Biochemistry, Annapoorana Medical College and Hospital, Salem, Tamil Nadu, INDIA.
Email: drramyasathish@gmail.com

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INTRODUCTION

Diabetes mellitus is a group of metabolic disease characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. Diabetes causes about 5% of all deaths globally each year. The chronic hyperglycaemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels.50% of people with diabetes die of cardiovascular disease (primarily heart disease and stroke).[1, 2] Diabetic patients with accompanied (but often unnoticed) dyslipidemia are soft targets of cardiovascular deaths. Patients with type 2 diabetes often exhibit an atherogenic lipid profile, which greatly increases their risk of CVD compared with people without diabetes. An early intervention to normalize circulating lipids has been shown to reduce cardiovascular complications and mortality.[3,4] Glycated hemoglobin (HbA1c) is a routinely used marker for long-term glycemic control. In accordance with its function as an indicator for the mean blood glucose level, HbA1c predicts the risk for the development of diabetic complications in diabetes patients[2].Apart from classical risk factors like dyslipidemia, elevated HbA1c has now been regarded as an independent risk factor for CVD in subjects with or without diabetes. Estimated risk of CVD has shown to be increased by 18% for each 1% increase in absolute HbA1c value in diabetic population[5,6] The UKPDS study has shown that in patients with type 2 diabetes, the risk of diabetic complications were strongly associated with previous hyperglycemia. Glycemic control with decreased level of HbA1c is likely to reduce the risk of complications[7]. Estimated risk of Cardio Vascular Diseases (CVD) has shown to be increased by 18% for each 1% increase in absolute HbA1c value in diabetic[8]. Even in non-diabetic cases with HbA1c levels within normal range, positive relationship between HbA1c and CVD has been demonstrated[9,10]. A few studies have previously tried to find the correlation between HbA1c levels and lipid profile. Some of these have shown that all

the parameters of lipid profile have significant correlation with glycemic control.[11]

MATERIAL AND METHODS

The study was approved by the Institutions Human Ethics Committee and informed consents were obtained from all participants. 48 (males =16, females=32) known Type 2 diabetic patients aged 35-55 yrs on oral hypoglycemic drugs were Selected in diabetic out-patient department of tertiary care hospital. Age- 35 to 55 years ,Duration of diabetes- not more than 5 years were included into study while Smokers , Known Hypertensive patients, H/O coronary/ cerebral vascular disease, Recent major surgeries, Patients on insulin, Diabetes mellitus with complications, Alcoholics, Tobacco chewers were excluded from the study. Clinical parameters: Blood Pressure, Fundus examination, ECG, were done. Anthropometric measurements :Body Mass Index (BMI), Waist: Hip Ratio(WHR). Fasting Blood Glucose (FBG), Lipid Profile(Total Cholesterol, HDL, LDL, Triglycerides) were noted . Patients were Divided into two Groups i.e**.** Obese (Group-1) And Non-Obese (Group-2) Independent 't' test was employed to compare groups and p value less than 0.05 was considered significant. Statistical analysis was performed using SPSS software.

RESULT

Table 1: General Characteristics of Obese (Group-1) and Non-Obese (Group-2) Type 2 Diabetic Patients

Parameters	Group- I (n=28)	Group- II (n=20)	P- value
Age	45.5±6.7	47.1±5.5	NS
Duration	4.3±2.5	4.5±2.3	NS
Family history	1.55±0.5	1.54±0.5	NS
Systolic BP(mm Hg)	127±22	121±17	0.320
Diastolic BP(mm Hg)	82±10	74±10	0.078
Waist Hip Ratio	0.99±0.1	0.85±0.1	0.888
Body Mass Index	26.96±3.6	21.05±1.9	0.091

The type 2 diabetic patients were divided in to two groups. Group 1 with BMI $\geq$ 23; Group 2 with BMI $\leq$ 23.There was no significant difference in systolic and diastolic BP between obese and non-obese groups.

Table 2: Glycemic status and lipid profile of obese and non-obese type 2 diabetic patients

Parameters	Group- I (n=28)	Group- II (n=20)	P value
Fasting plasma glucose (mg/dl)	143.41±29.4	119.21±20.3	0.097
Serum Insulin (U/ml)	26.588±9.2	12.55±6.92	0.047
HbA_1C	9.17±1.11	6.7±0.8	0.021
HOMA-IR	5.56±1.95	2.24±1.65	0.036
Serum cholesterol (mg/dl)	165.90±20.4	163.5±15.36	0.794
Serum Triglycerides (mg/dl)	156.2±24.69	142.6±19.46	0.747
HDL cholesterol (mg/dl)	42.4±3.5	40.5±2.5	0.229
LDL cholesterol (mg/dl)	88.9±26.5	83.5±21.1	0.942

Serum insulin, HbA_1C, HOMA-IR were significantly increased in group 1. There was no significant difference in fasting plasma glucose and lipid profile parameters between obese and non-obese groups.

DISCUSSION

Diabetes mellitus is now emerging as one of the main threats to human health in 21st century. According to Michael Clark, *et al*.,2010, Diabetes mellitus affects more than 120 million people worldwide, and is estimated that it will affect 300 million people by the year 2025. Indians have a genetic phenotype characterized by low body mass index, but with high upper body adiposity, high body fat percentage and high level of insulin resistance. With a high genetic predisposition and the high susceptibility to the environmental insults, the Indian population faces a high risk for diabetes and its associated complications (Ramachandran *et al*., 2009). Type 2 diabetes mellitus seems to be emerging as a major health Problem in the adolescent age group. The increase in the prevalence of type 2 diabetes is closely linked to the upsurge in obesity. About 90% of type 2 diabetes is attributable to obesity. Furthermore, approximately 197 million people worldwide have impaired glucose tolerance, most commonly because of obesity and the associated metabolic syndrome. This number is expected to increase to 420 million by 2025 (Hossain,.2007)[12]. Obesity is frequent in the majority of patients with type 2 diabetes. Obesity is a known contributing factor to most of the Insulin resistance seen in these patients (Bjorntorp., 2008)[13]. The present study was conducted among obese and non-obese type 2 diabetic patients in rural population, to compare their insulin resistance, glycemic status, lipid profile and polymorphism study in these groups. Previous studies have demonstrated that the obesity is highly associated with insulin resistance and obesity is considered the most important risk factor for type 2 diabetes mellitus. In our study, the mean age for group 1 (obese) was 45.5 yrs and group 2 (non-obese) was 47.1 yrs and there was positive family history in both groups. Molisch *et al* 2006, has published that in clinical practice, the BMI and the waist circumference are very good predictors of insulin resistance. In our study, group1 has higher waist circumference and BMI ($\geq$ 23 kg/m^2) than group 2. Pooja *et al* reported that BP was the only significant determinant of atherosclerosis in diabetes patients with HbA_1C of 7% or more and duration of diabetes above 10 years. In our study there is no increase in BP and also no significant difference in both systolic and diastolic blood pressure among these groups and there was a significant increase in HbA_1C value in group 1 obese diabetic patients. In a recent study, endothelial dysfunction and inflammation in the incidence of

complication of diabetes have been explored and found that there was some evidence for a role of endothelial dysfunction but there was no role for low grade inflammation. The study emphasized that importance of glycemic control in amelioration of complications (Spijkerman *et al* 2007) Poor glycemic control was assessed based on high fasting plasma glucose level and high HbA_1C, even though fasting insulin, HOMA-IR were within normal limits. In another study by Kawasumi *et al*, it has been demonstrated that improvement of glycemic control ($HbA_1C < 6.5$) could prevent insulin resistance in type 2 diabetic patients. In our study, fasting plasma glucose level, HbA_1C value and HOMA- IR were significantly increased in group 1 patients. Rashid *et al*., 2006, reported that abdominal obesity and insulin resistance are common grounds for atherogenic dyslipidaemia that includes increased plasma triglyceride levels as well as reduced high density lipoprotein (HDL) cholesterol concentrations. The presence of smaller, denser low density lipoprotein (LDL) particles is also frequently observed in insulin resistant individuals. In our study, it was observed that there is no significant difference in lipid profile parameters in both groups. Viberti *et al* 2006[14], has reported that diabetic nephropathy develops more commonly in patients with poor glycemic control, particularly if the HbA_1C concentration is above 11%. On the other hand patients whose HbA_1C is maintained below 8.5% are at much lower risk of nephropathy. So, based on the above observations of our study, the group 1, obese type 2 diabetic patients having BMI $\geq$ 23 with high fasting plasma glucose and insulin levels are having more insulin resistance than group 2, non-obese type 2 diabetic patients. The fact that obese patients had high levels of insulin denotes that they require higher amount of insulin to metabolise the glucose. However, despite high insulin levels in these patients, the glucose metabolism appears to be deranged as reflected by the high HbA_1C values. This proves that obese patients have higher insulin resistance, since insulin resistance is defined as subnormal physiological action for a given concentration of insulin. The role of visceral adipose tissue (VAT) in influencing insulin resistance needs to be ascertained especially in our population. The renal and liver parameters were within the normal range. Insulin resistance due to genetic predisposition also play an important role.

LIMITATIONS

Our study was done in only 48 type 2 diabetic patients; this small sample size is the limitation of this study.

CONCLUSION

The findings of our study reveal that there is significant increase in fasting plasma glucose, HbA_1C, HOMA-IR but not much significant difference in other biochemical parameters.

REFERENCES

1. Diabtes.http://.who.int/mediacentre/factsheets/fs312/en/index.html (Updated on November 2009)
2. Glycosylaed Haemoglobin, HbA1C.hptt://clinlabnavigator.com/test-interpretations/haemoglobin-a1c.html?letter=h (Updated on 18 June 2010).
3. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med 1998; 339: 229-234.
4. Windler E. What is the consequence of an abnormal lipid profile in patients with type 2 diabetes or the metabolic syndrome? Atheroscler Suppl 2005; 6: 11-14.
5. Marshall SM and Barth JH. Standardization of HbA1c measurements - a consensus statement. Diabetic Medicine 2000; 17:5-176.
6. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, et al. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med 2004; 14: 421-431.
7. Irene M Stratton, Amanda I Alder et al., Association of glycemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35); Brit Med J. 2000 Vol 321 : 405-416
8. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, et al. Meta-analysis: glycosy-lated hemoglobin and cardiovascular disease in diabe-tes mellitus. Ann Intern Med 2004; 14: 421-431.
9. Khaw KT, Wareham N, Bingham S, Luben R, Welch A and Day N. Association of hemoglobin A1c with car-diovascular disease and mortality in adults: the Euro-pean Prospective Investigation into Cancer in Norfolk. Ann Intern Med 2004; 141: 413-420.
10. Deeg R, Ziegenhorn J. Kinetic enzymatic method for automated determination of total cholesterol in serum. ClinChem 1983; 29: 1798-802.
11. Gligor Ramona et al. Relationship between glycosylated hemoglobin and lipid metabolism in patients with type 2 diabetes.StudiaUniversitatis "VasileGoldiş", SeriaŞtiinţeleVieţii 2011;21(2): pp. 313-318
12. Hossain P; Kawar B; and Nahas MEI, Obesity and Diabetes in the Developing World—A Growing Challenge. The New England Journal of Medicine 2007; 356(3):213–5.
13. Bjorntorp P.2008 Abdominal obesity and the development of non-insulin-dependent diabetes mellitus. Diabetes Metab Rev. 4:615–622.
14. Viberti GC, Earle K. Predisposition to essential hypertension and the development of diabetic nephropathy. J Am SocNephrol 2006;3:527-533

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