

**EFFECT OF HYPERGLYCEMIA ON POSTPRANDIAL LIPID PROFILE IN
TYPE 2 DIABETES**

Rajkumari Rathore*, Savita Rathore, Neha Sharma, Anil Bidwai

Department of Biochemistry, Index Medical College Hospital and Research Centre,
Indore-452016, M.P. (India)

Summary

The present study was done to evaluate the effect of hyperglycemia on postprandial serum lipid level in 25 diabetic subjects. The results were compared with 20 healthy subjects. Blood samples were collected for estimating serum lipid level (serum cholesterol, triglyceride, LDL-C, HDL-C) and plasma glucose in fasting as well as post prandial condition. Mean serum cholesterol, LDL and TG were found to be significantly higher ($p < 0.001$) while HDL-C was lower in diabetic cases than the control group in both condition. All the diabetic patients were found to have ($p < 0.001$) increased lipid levels (except HDL-C was decreased) in post prandial condition as compared to the fasting sample. This result indicates that measurement of postprandial lipidemia rather than of fasting plasma TG would be a good atherogenic indicator particularly in hyperglycemic condition.

Keywords: Hyperglycemia, lipid estimation, Low density lipoprotein cholesterol, High density lipoprotein cholesterol, Triglyceride

*Corresponding Author-Rajkumari Rathore, rajkumarirathore@rediffmail.com

Introduction

An estimated 240 million people worldwide have diabetes, which is a leading cause of death in most developed countries. ⁽¹⁾ The worldwide prevalence of diabetes is growing rapidly. The cluster of abnormalities (like increased TG, low HDL-C and increased BP), often referred to as the [dys] metabolic or insulin resistance syndrome, has been shown to increase the risk of cardiovascular disease (CVD) and, more particularly, of diabetes. ⁽²⁾

In people with diabetes or insulin resistance, the incidence of dyslipidemia is high and lipoprotein abnormalities play a key role in the development of atherosclerotic vascular complications. Most studies reporting lipoprotein abnormalities and cardiovascular risk have been performed under fasting conditions. Some data also indicate that postprandial hyperglycemia may have a greater effect on the development of cardiovascular complication compared with elevated fasting plasma glucose not only in patients with type 2 diabetes but also in those with impaired glucose tolerance.^(3, 4) The postprandial metabolic disorders are drawing particular attention as a risk factor for the development of atherosclerotic diseases. It is known that there exist a population of patients whose fasting glucose and triglyceride levels are found to be within a normal range, but who present with hyperglycemia and hypertriglyceridemia postprandially, which has led to both postprandial hyperglycemia and hyperlipidemia becoming recognized as risk factors for cardiovascular disease.⁽⁵⁾ Estimation of lipid pattern in postprandial hyperglycemia condition can often be accounted for by the single concept of “postprandial metabolic disorder”. But it has not established that postprandial lipids might be an independent risk factor of cardiovascular disease.

The present study was aimed at assessing the postprandial metabolic disorder and compared the significance of estimation of postprandial lipid profile over fasting lipid profile of type 2 diabetic cases.

Material & Methods

The study was carried out at the Department of Clinical Biochemistry at Index hospital, Indore. Volunteer patients diagnosed with Type 2 Diabetes Mellitus in the clinics of the department were selected for the study. Blood was obtained from 25 morbid and 20 healthy control groups. The blood collected was allowed to clot and then centrifuged to obtain serum for lipid profile estimation. For glucose test 1ml of blood was collected along with the above sample in sugar bulb and plasma separated was used for estimation of fasting and postprandial blood glucose.

Blood glucose was estimated by GOD-POD method,⁽⁶⁾ serum cholesterol by CHOD-PAP method,⁽⁷⁾ serum TG by GPO/PAP method,^(8, 9) serum HDL-C by direct colorimetric method⁽¹⁰⁾ while LDL-C and VLDL were calculated by the Friedwald formula.

Values have been expressed as Mean±SD. The results were analyzed using Student ‘t’ test. P<0.05 was considered as significant.

Results and Discussion

Mean serum lipid levels were found to be significantly (p<0.001) higher (except HDL-C was decreased) in the diabetic cases than the control group in both fasting as well as postprandial condition.

All the diabetic cases showed significant variation in post prandial lipid levels than the fasting sample of same patients.

Accelerated coronary and peripheral vascular atherosclerosis is one of the common and serious complications of long term diabetes mellitus. Hyperlipidaemia as a metabolic abnormality is frequently associated with diabetes mellitus. Its prevalence is variable, depending on the type and severity of diabetes, glycaemic control, nutritional status, age and other factors. The most characteristic lipid abnormality in diabetics is hypertriglyceridaemia, with or without associated increase in plasma cholesterol.^(11, 12)

Most studies reporting lipoprotein abnormalities and cardiovascular risk have been performed under fasting conditions. Under such circumstances, type 2 diabetes and insulin resistance states are characterized by high levels of plasma TG and VLDL, decreased levels of HDL-C, and predominance of small dense-LDL. However, considering a person's normal eating habits, a great part of the day is in the postprandial state that, according to available evidence, would contribute to the development of atherosclerosis. In people with diabetes, the concomitant increase of postprandial glucose and TG magnifies the phenomenon.⁽¹³⁾

In our study diabetics when compared to the control subjects, showed statistically significant ($p < 0.001$) increase in the levels of serum cholesterol, triglyceride, LDL, VLDL, serum HDL-C levels did not differ significantly ($p < 0.05$) between the two groups in the fasting state. Sharma (1970) and Jain (1980) observed increase in the levels of serum cholesterol and triglyceride in diabetic subjects as compared to normal controls^(14, 15). Same as reported by Zargar et al (1995) in obese diabetics when compared with obese control⁽¹⁶⁾.

Significant ($p < 0.001$) changes were also observed in serum cholesterol, triglyceride and HDL-C levels in postprandial sample of diabetics than control in our study.

And on comparison between fasting and postprandial sample of diabetics, the lipid levels (table-2) significantly ($p < 0.001$) increased (HDL-C was decreased) in postprandial sample than fasting one. Lefebvre (1998) reviewed that Increased postprandial glucose levels and lipid concentrations may damage endothelial cells on blood vessels walls appear to be complex. And high postprandial triglyceride levels and particularly post prandial rich triglyceride remnants contribute an increased risk for cardiovascular disease⁽¹⁷⁾. Heine (2004) reported that the post prandial hyperglycemia is a risk indicator for micro and macro vascular complications, not only in patients with type 2 diabetes but also in those with impaired glucose tolerance. He focused on the relative contributions of postprandial hyperglycemia, the metabolic syndrome and, in particular, raised triglyceride levels in the postprandial state to the development of cardiovascular complications of diabetes⁽¹⁸⁾.

In recent years, there have been reported that postprandial hyperglycemia or hyperlipidemia is an independent risk factor in a cardiovascular event, and that fasting hyperglycemia has a non correlation with the probability of death due to cardiovascular diseases, while persons with the 2 hours blood glucose levels of 200 mg/dl or more have a high correlation with the probability of death due to cardiovascular diseases^(19,20). Mori (2008) reviewed that there exist a population of patients who had hyperglycemia and hypertriglyceridemia postprandially, which has led to both postprandial hyperglycemia and hyperlipidemia becoming recognized as risk factor for cardiovascular disease.⁽⁵⁾

Increased postprandial lipidemia is a characteristic aspect of diabetic dyslipidemia. It has a high prevalence among people with diabetes even when they have normal fasting TG levels. In the postprandial state the metabolism of TG rich lipoproteins is highly altered. There is a significant increase in the concentration and time of presence of chylomicron remnants, VLDL and VLDL remnant (IDL) in plasma, as well as of TG. Therefore the metabolism of the remaining lipoprotein (LDL & HDL) is also affected due to their interrelation, which involves the permanent lipid and apoprotein interchange between them^(21,22). VLDL is the lipoprotein fraction most affected by diabetes in the postprandial period since a greater hepatic synthesis of VLDL is produced to transport endogenous TG to adipose tissue. VLDL plasmatic level depends on two processes: hepatic synthesis and extra hepatic clearance, mainly in adipose tissue. In the diabetes state, both processes appear altered^(21,23).

It can be concluded that the postprandial abnormalities are more evident and occur earlier than those in the fasting state, so the measurement of postprandial lipidemia rather than of fasting plasma triglyceride would be a good atherogenic indicator.

**Table 1: Comparison Between Fasting & Postprandial Condition In Controls
(N= 20)**

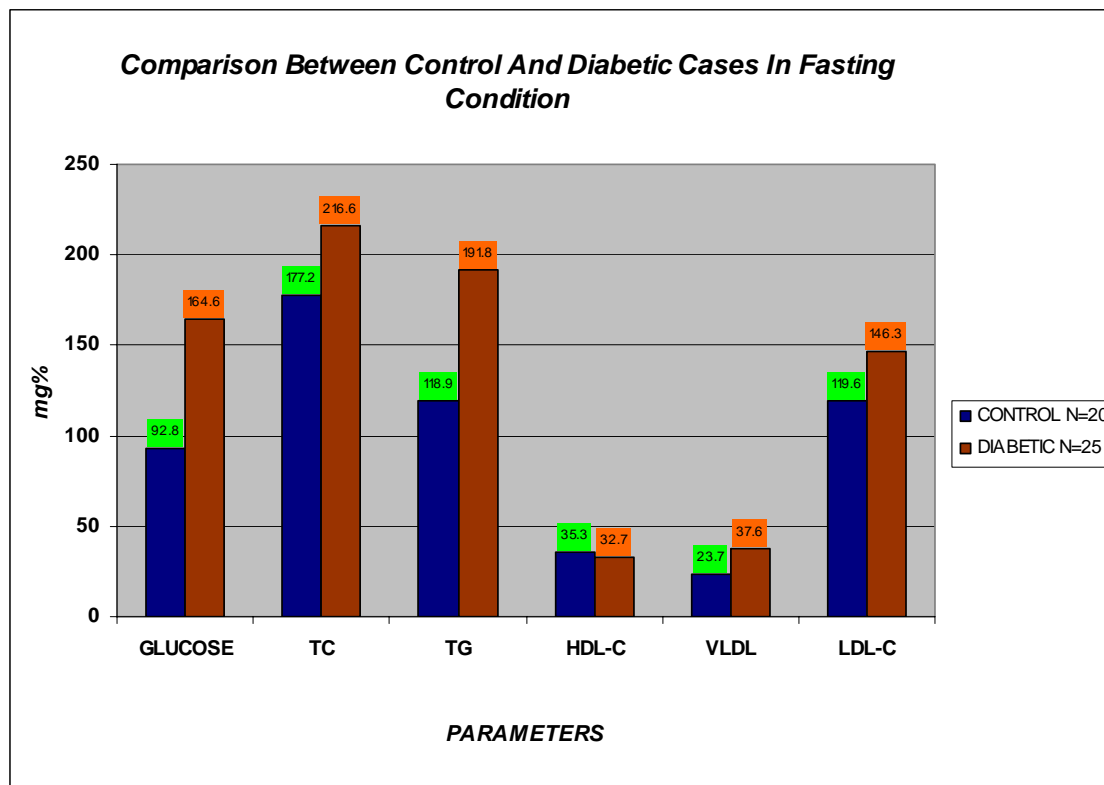
S.No.	Parameters (Mg %)	FBS	PPBS
1	FBS	92.8 ±13.9	125.3 ± 10.2
2	TC	177.2 ± 21.1	192.0 ± 23.9
3	TG	118.9 ± 22.16	170.7 ± 26.8
4	HDL-C	35.3 ± 3.4	31.7 ± 1.4
5	VLDL	23.7 ± 4.4	48.4 ± 5.3
6	LDL-C	119.6 ± 23.8	126.1 ± 24.1

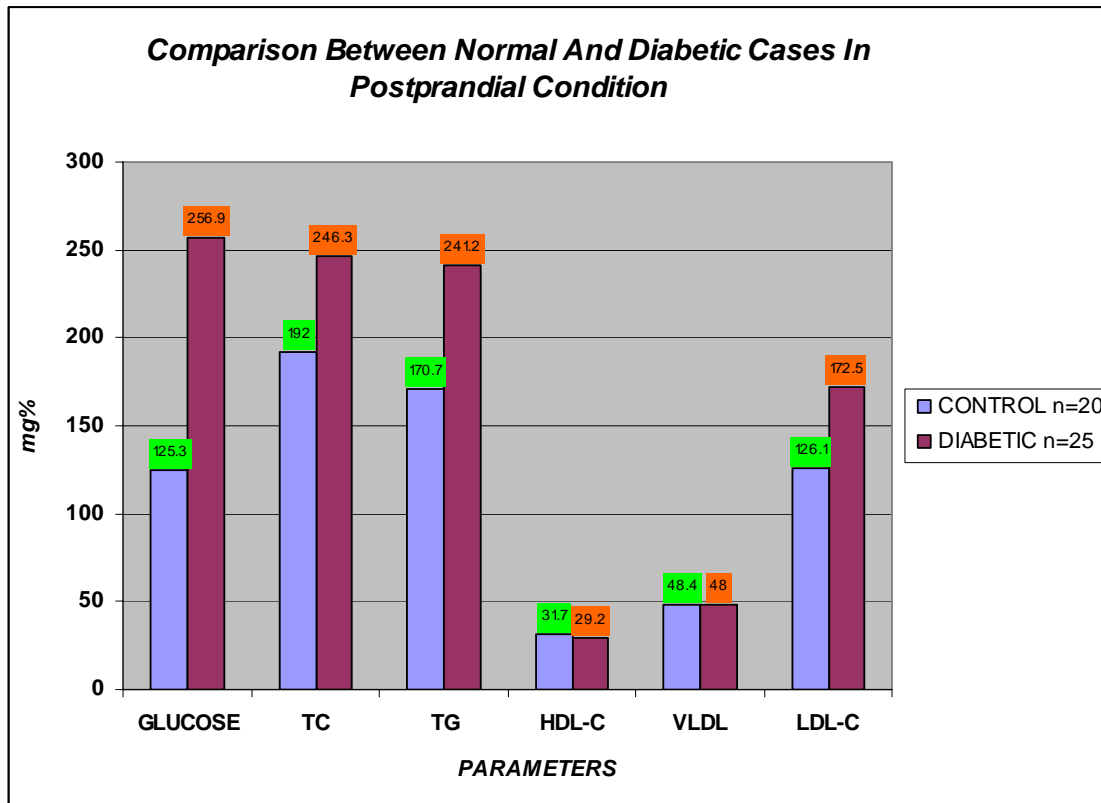
P value < 0.001. Values are expressed as mean ± S.D.

Table 2: Comparison Of Blood Glucose And Lipid Profile Between Fasting And Post-Prandial Condition In Diabetic Patients (N=25)

S.NO.	Parameter (Mg %)	FBS	PPBS
1	GLUCOSE	164.6±62.01	256.9±82.9
2	TC	216.6±32.6	246.3±32.4
3	TG	191.8±46.7	241.2±55.3
4	HDL-C	32.7±2.8	29.2±2.3
5	VLDL	37.6±11.08	48.01±11.5
6	LDL-C	146.3±33.07	172.5±47.3

P value < 0.001. Values are expressed as mean ± S.D.





References

- 1) International Diabetes Federation. Available www.idf.org, 2006.
- 2) Robert J., Heine.-The role of postprandial hyperglycemia and hyperlipidemia in CVD risk. *Current Diabetes Reports*. 2006;6: 1-2.
- 3) Hanefeld M., Kurtchiev T.-Plasma lipids in diabetics. In *New Horizons in Diabetes Mellitus and Cardiovascular Disease*. Ed. CJ Schwartz and GVR Born. London: Current Science. 1995;89-95.
- 4) Grundy S.M.-Hypertriglyceridemia, atherogenic dyslipidemia, and the metabolic syndrome. *Am J Cardiol*, 1998;81: 18B-25B.
- 5) Mori Y.-Postprandial metabolic disorder. *Nippon Rinsho*. 2008;66(6): 1219-29.
- 6) Trinder P.- *Ann Clin Biochem*. 1964;6: 24.
- 7) I.F.C.C.-*Clin Chim Acta*.1978;87/3: 459F.
- 8) Bucolo G., David H.- *Clin Chem*. 1973;19: 476.

- 9) Fossati P., Prencipe L.- Clin Chem. 1982;28: 2077.
- 10) NCCLS Document, "Procedures for the collection of arterial blood specimens", Approved Standard. 3rd Ed, 1999.
- 11) Goldberg R.B.- Lipid disorders in diabetes. Diabetes Care. 1981;4: 561.
- 12) Taskinen M.R.- Hyperlipidemia in diabetes. Clin Endocrinol Metab.1990; 4: 743.
- 13) Ceriello A. The post prandial state and cardiovascular disease: relevance to diabetes mellitus. Diabetes Metab Res Rev, 2000;16: 25-32.
- 14) Sharma D, Bansal BC, Prakash C. serum lipid studies in thin insulin dependent diabetics below the age of 30 years. J Ind Med Ass,1970; 9: 54.
- 15) Jain AP, Gupta DP. Study of blood, lipids diabetes without any manifest vascular complications. J D A I, 1980;20.
- 16) Zargar A H, Wandroo F A, Wadhwa M B, Laway B A. Serum lipid profile in non insulin dependent diabetes mellitus associated with obesity. Int. J. Diab. Dev. Countries, 1995;1510.
- 17) Lefebvre P J, Scheen A J. the postprandial state and risk of cardiovascular disease. Diabet Med, 1998; 15 (4): S63-8.
- 18) Heine R J, Balkau B, Ceriello A, Del Prato S, Horton ES, Taskinen M R. What does postprandial hyperglycaemia mean. Diabet Med,2004; 21(3): 208-13.
- 19) Non Patent Document 1. diabetes Care, 1999;22: 920-924.
- 20) Non Patent Document 2. Arch Intern Med, 2001;161: 397-405.
- 21) Mero N, Syvanne M, Taskinen M R. Post prandial lipid metabolism in diabetes. Atherosclerosis, 1998;141(1): S53-55.
- 22) Verges B L. Dyslipidaemia in diabetes mellitus. Review of the main lipoprotein abnormalities and their consequences on the development of atherogenesis. Diabetes Metab, 1999;25(3): 32-40.
- 23) Tomkin G H, Owens D. Abnormalities in apo B containing lipoproteins in diabetes and atherosclerosis. Diabetes Metab Res Rev,2001; 17: 27-43.