

ISSN Print: 2617-4693
 ISSN Online: 2617-4707
 IJABR 2021; 5(1): 20-23
www.biochemjournal.com
 Received: 17-11-2020
 Accepted: 23-12-2020

Dr. Pampa Reddy
 Professor and Head,
 Department of Biochemistry,
 Al-Azhar Medical College,
 Thodupuzha, Idukki, Kerala,
 India

Dr. Kiran Kumar Akka
 Associate Professor,
 Department of Biochemistry,
 M R Medical College,
 Kalaburagi, Karnataka, India

Corresponding Author:
Dr. Kiran Kumar Akka
 Associate Professor,
 Department of Biochemistry,
 M R Medical College,
 Kalaburagi, Karnataka, India

Correlations between dyslipidemia and cardiovascular disease in patients of type 2 diabetes mellitus

Dr. Pampa Reddy and Dr. Kiran Kumar Akka

DOI: <https://doi.org/10.33545/26174693.2021.v5.i1a.60>

Abstract

Hyperlipidemia in association with insulin resistance is common in patients with type 2 diabetes mellitus. Insulin resistance and the ensuing hyperinsulinemia are associated with hypertriglyceridemia and low serum high-density lipoprotein (HDL) cholesterol concentrations.

The lipoprotein abnormalities are related to the severity of insulin resistance. The venous blood was collected for both fasting and postprandial for blood sugar estimation. The fasting blood samples were collected for the estimation of serum Total cholesterol, serum Triglycerides and serum HDL cholesterol estimation. In this study, it was found that although the patients were on oral hypoglycaemic drugs and some patients were on Insulin the fasting blood sugar and postprandial blood sugar are significantly higher in study groups when compared to control groups ($p < 0.001$).

Keywords: Correlations, dyslipidemia, cardiovascular disease

Introduction

Diabetes is well recognized as an independent risk factor for cardiovascular disease (CVD) in men and women. CVD is up to four times more common in people with diabetes than in those without, and 50% of diabetic people have evidence of CVD at the time of diagnosis [1]. Post-infarction mortality is significantly higher in people with diabetes than in those without. It is estimated that 75 to 80% of diabetes related deaths are attributable to the macro vascular complications of the disease primarily cardiovascular disease, cerebrovascular disease, and peripheral vascular disease.

Atherosclerotic macrovascular complications occur at an earlier age and with greater severity in people with diabetes, and its pathogenesis is directly influenced by the diabetic state.

Long term exposure to elevated glucose levels can contribute to the endothelial dysfunction observed in diabetes. Increasing evidence suggests that endothelial dysfunction may play a central role in the development of atherosclerosis. Endothelial dysfunction is characterized by inhibited vasodilatation, increased proliferation of vascular smooth muscle, increased thrombogenesis, and proatherogenic cellular processes [2].

Accelerated atherosclerosis, endothelial dysfunction have been linked with increased thrombosis, hypertension, and dyslipidemia, all of which contribute to the pathogenesis of vascular disease in diabetes.

The dyslipidaemia is a condition marked by an abnormal concentration of lipids or lipoproteins in the blood, the typical atherogenic dyslipidemia includes increased plasma triglyceride levels as well as reduced HDL Cholesterol concentration and the presence of small, dense LDL cholesterol particles. The prevalence of dyslipidemia varies with the population being studied. The incidence is highest in patients with premature coronary heart disease (CHD), which can be defined as occurring before 55 to 60 years of age in men and before 65 years in women. In this setting, the prevalence of dyslipidemia is as high as 75 to 85% compared to approximately 40 to 48% in age-matched controls without CHD [3].

Hyperlipidemia in association with insulin resistance is common in patients with type 2 diabetes mellitus. Insulin resistance and the ensuing hyperinsulinemia are associated with hypertriglyceridemia and low serum high-density lipoprotein (HDL) cholesterol concentrations.

The lipoprotein abnormalities are related to the severity of the insulin resistance. A study that measured insulin sensitivity using a euglycemic clamp in patients with and without type 2 diabetes mellitus found that greater insulin resistance was associated with large, very low density lipoprotein (VLDL) particle size, small low density lipoprotein (LDL) particle size, and small HDL particle size. Additionally, the number of VLDL, intermediate density lipoprotein (IDL), and LDL particles increased with increasing insulin resistance.

Hypertriglyceridemia results both from increased substrate availability (glucose and free fatty acids) and from decreased lipolysis of VLDL triglyceride.

According to the centers for disease control and prevention (CDC), 97% of adults with diabetes have one or more lipid abnormalities. The central characteristic of dyslipidemia in type 2 diabetes is an elevated triglyceride level, particularly triglyceride-rich VLDL levels and decreased HDL cholesterol levels. The concentration of LDL cholesterol is usually not significantly different from that seen in non-diabetic individuals [4,5].

Type 2 diabetes typically has a preponderance of small, dense, oxidized LDL particles, which may increase atherogenicity, even if the absolute concentration of LDL cholesterol is not elevated [6].

Methodology

The patients in this study were selected according to the following criteria.

Inclusion criteria

1. Age more than 40 years
2. Type 2 Diabetes Mellitus, previously diagnosed.
3. Cardiovascular disease, freshly and previously diagnosed.
4. Regular follow-up in the diabetic clinic for the last 5 years.

Exclusion criteria

1. Age less than 40 years.
2. Freshly detected patients of Diabetes mellitus.
3. Non Diagnosed patients with cardiovascular diseases.
4. Irregular follow-up.

Specimen collection

The venous blood was collected for both fasting and postprandial for blood sugar estimation. The fasting blood samples were collected for the estimation of serum Total cholesterol, serum Triglycerides and serum HDL cholesterol estimation.

The Random blood samples were collected between 6 to 12 hours in myocardial infarction patients for the estimation of serum CK-MB, serum SGOT and serum Lactate dehydrogenase in the following bulbs,

Fluoride Bulb – Blood sugar.

Plain Bulb – Serum Lipid profile, CK-MB, SGOT, and LDH.

All the above mentioned biochemical investigations were carried out in clinical chemistry laboratories of the Biochemistry department of medical college, Baroda.

The methods used in the study are in accordance with the

facilities available in the clinical Biochemistry Laboratory of Medical College, Baroda. The techniques are recent and used in many Indian laboratories and the results are easy reproducible.

The method used for Estimation of Blood sugar levels Glucose Oxidase and Peroxidase Method

Principle

The substrate β D-Glucose is oxidized by Glucose oxidase to form gluconic acid and hydrogen peroxide. The hydrogen peroxide, so generated, oxidizes the chromogen system consisting of 4-amino antipyrine and phenolic compounds to a red Quinoneimine dye. The intensity of the colour produced is proportional to the glucose concentration and is measured colourimetrically at 505 nm (490-530) or with a green filter.

Results

Table 1: Age and Sex Distribution

Age in Years	No. of Male Patients	No. of Female Patients	Total	Percentage
40 – 49	03	-	03	10%
50 – 59	09	4	13	43.3%
60 – 69	08	4	12	40%
70 – 79	02	0	02	6.6%
	22	8	30	100%

Table shows the age and sex distribution of all 30 patients. It shows an age range of 40 to 75 years. The mean age of presentation was 60.3 years.

Table 2: Risk Factors among 30 patients

S.No.	Risk factor	No. of Patients	Percentage
1	Smoking	14	46.6%
2	Hypertension	10	33.3%
3	Obesity	13	43.3%
4	Family History of Diabetes Mellitus	9	30.0%
5	Alcoholism	4	13.3%
6	Tobacco chewing	5	16.6%

In the present study among 30 patients, there was either a single or a combination of the above mentioned risk factors was found.

The common risk factor encountered in this study was smoking. It was encountered in 14 patients (46.6%) of these 14 patients, 8 patients were heavy smokers, smoking more than 20 cigarettes or bides per day.

The next most frequent risk factor was hypertension. It was present in 10 patients (33.3%) of these 4 patients were hypertensive for more than 5 years and were on regular treatment.

Obesity was present in 13 (43.3%) patients, 10 patients had previously documented evidence of hypercholesteremia. (Blood Cholesterol levels > 250 mg%).

A family history of Diabetes Mellitus was present in 9 patients. (30%)

Other risk factors in the form of alcoholism and tobacco chewing were present in 4 (13.3%) and 5 (16.6%) of patients respectively.

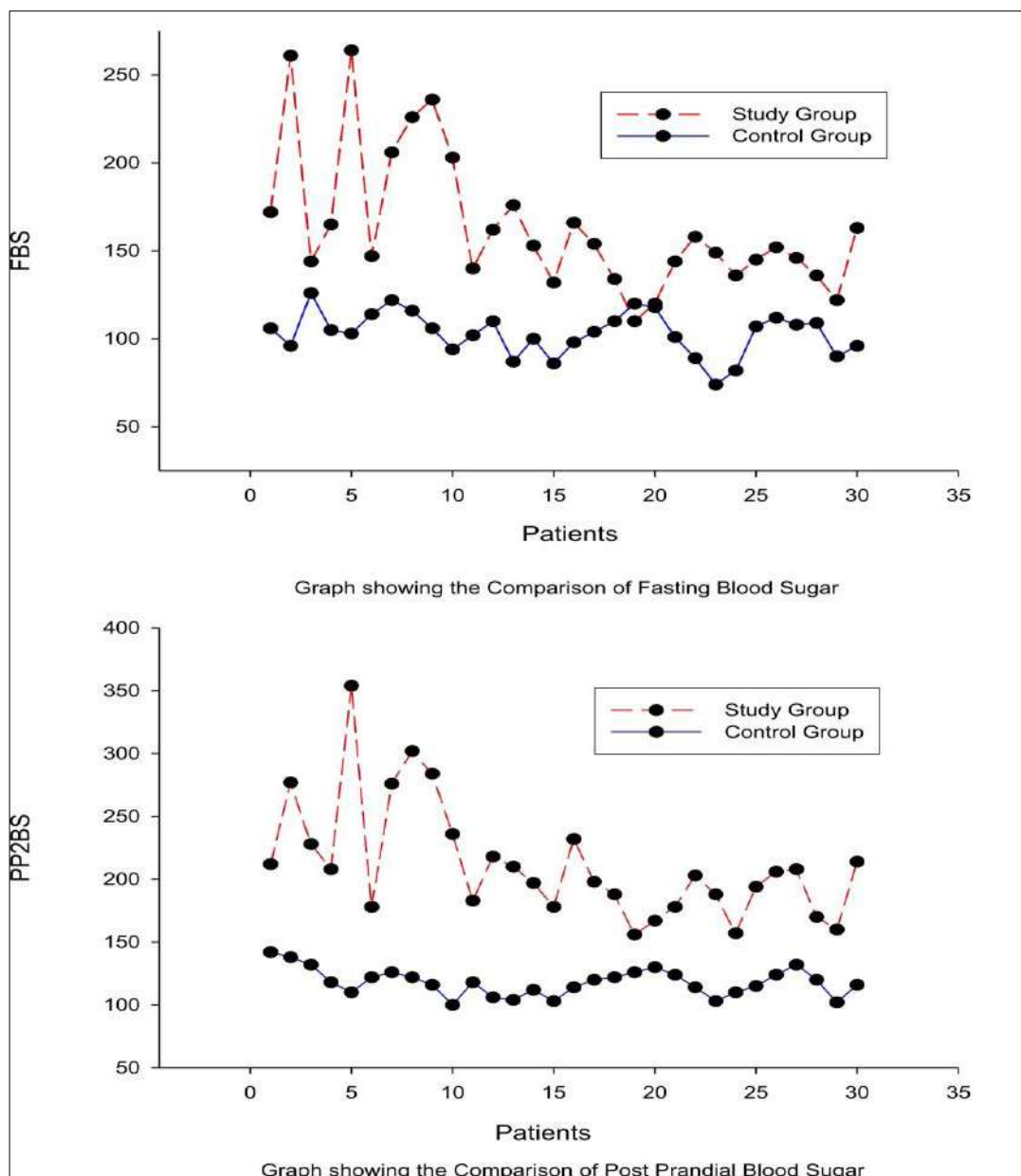


Fig 1: FBS & PPBS

In this study, it was found that although the patients were on oral hypoglycaemic drugs and some patients were on insulin the fasting blood sugar and postprandial blood sugar are significantly higher in study groups when compared to control groups ($P < 0.001$).

This suggests that in diabetic subjects at hyperglycemia, there is both a decrease in responsiveness and a decrease in

sensitivity to insulin. It is well established that in type 2 diabetes exhibits peripheral insulin resistance in target tissues.

In the hyperinsulinemic euglycemic clamp technique, a research method for measuring peripheral insulin resistance shows that the glucose disposal rate is reduced by at least 50% in subjects with type 2 diabetes [7].

Table 3: Comparison between various laboratory parameters in Control [n=30] and Study [n=30] Groups.

Laboratory parameters	Control Group		Study Group		t	P value
	Mean	SD	Mean	SD		
FBS mg/dl	103.0333	12.2938	164.0667	39.4024	8.099	<0.001
PP2BS mg/dl	118.0333	10.7494	212.0000	46.2579	10.83	<0.001
TC mg/dl	185.1000	25.5483	230.7333	43.1437	4.98	<0.001
TRG mg/dl	156.0000	31.9633	228.3667	64.1644	5.52	<0.001
HDL-C mg/dl	38.9333	5.0646	29.4000	6.7650	6.17	<0.001
VLDL-C mg/dl	31.5333	6.4312	46.6333	12.5931	5.84	<0.001
LDL-C mg/dl	114.6333	24.4688	154.7000	38.6747	4.79	<0.001
CK-MB IU/L	19.8667	6.3449	147.5000	46.0163	15.04	<0.001
SGOT IU/L	27.8000	6.0822	118.3333	70.5341	7.004	<0.001
LDH IU/L	360.1667	95.5146	617.1667	135.6845	8.48	<0.001

Discussion

Distribution according to sex showed 73.3% were males and 26.6% were females. There were no female patients below the age of 50 years.

Myocardial infarction can occur at any age and sex, but generally, it is a disease in middle age and older individuals: Minitz-Katz^[8] reported an incidence 66.3% in the age group of 50-59 years. Julien *et al.*^[9] In their prospective study of 100 unselected patients reported a maximum incidence of 62.0% in the age group of 50-59 years.

In the present study, the maximum incidence of acute myocardial infarction was in the age range of 52-60 years (53.3).

Men are frequently the victims of acute myocardial infarction. This has probably to do with the stressful life and the high incidence of smoking. Probably females are protected during their reproductive period by sex hormones. In the present study male to female ratio is (2:1). The ratio further decreased with the advancement of age. Men also developed infarction at an earlier age than females.

In the present study, the average age of presentation was 60.3 years. There were no female patients below the age of 50 years.

The data of the present study regarding age and sex match with the data published by Mintz-Katz^[8] and Julien *et al.*^[9]. This is considered one of the prime risk factors for CHD. The earlier the age of detection, the greater the risk of CHD. Elevated serum cholesterol is associated with the risk of CAD. Specifically, a 10% increase in serum cholesterol is associated with a 20 – 30% increase in the risk of CAD^[10].

In men it has been found that 41% of variants in CAD mortality were related to variation in serum cholesterol, 32% were related to variation in HDL-C and 55% of the variation in the ratio of total cholesterol to HDL cholesterol. It is difficult to define a safe basal level of serum cholesterol. A low risk level, from the point of view of primary prevention should ideally be LDL-C less than 130mg/dl, HDL-C more than 40mg/dl and TRG less than 150mg/dl.

Smoking increases CAD mortality by 50%, it doubles the incidence of CAD, and the risk increases with age and the number of cigarettes smoked. Similar risks have been observed among women.

Smoking is a leading preventable cause of death and CAD worldwide. Those who quit smoking decrease the risk by 50% in 1-2 years and to normal levels by 5-15 years. Smokers have lower HDL-C levels and high VLDL and triglyceride levels.

Hypertension is a well-established risk factor for CAD. Both elevated systolic and diastolic blood pressures are clearly associated with an increased risk of CAD. A 7 mm Hg increase in diastolic blood pressure over any baseline reading was associated with a 27% increase in CAD risk and a 42% increase in stroke risk^[11].

Obesity appears to have an independent risk for CAD, even after controlling the other risk factors. A higher BMI is associated with an increase in all the risk factors of CAD. The distribution of body fat may also play a role in the development of CAD, with abdominal adiposity above the umbilicus posing a substantially greater risk in both men and women. A waist circumference of 35 inches in women and 40 inches in men is an easily measured marker of coronary artery disease risk^[12].

Conclusion

- Serum levels of CK-MB were found significantly elevated in Myocardial Infarction patients with type 2 diabetic individuals when compared to controls. Higher values were obtained in patients who suffered cardiovascular complications and mortality.
- SGOT levels were elevated in the diabetic study group compared to controls.

References

1. Roberts R. The two out of three criteria for the diagnosis of infarction: Is it passé? *Chest*. 1984;86(4):511-513.
2. Multiple Risk Factor interventional trial (MRIFT) circulation. 1987;75(2):6-8.
3. Robert R, MD. Preventing Cardiovascular Complications of Type 2 Diabetes: Focus on Lipid management. *Clinical diabetes*. 2001;19(3):113-20.
4. Durrington P. Dyslipidaemia. University Department of Medicine, Manchester Royal Infirmary, Oxford Road, M13 9WL, Manchester, UK. *Lancet*. 2003;362(9385):717-31.
5. Genest JJ Jr, *et al.* Familial lipoprotein disorders in patients with premature coronary artery disease. *Circulation*. 1992;85(6):2025-33.
6. Roncaglioni MC, *et al.* Role of family history in patients with myocardial infarction. An Italian case-control study. GISSI-EFRIM Investigators. *Circulation*. 1992;85(6):2065-72.
7. Garvey WT; Kwon S, *et al.* Effects of insulin resistance and type 2 diabetes on lipoprotein subclass particle size and concentration determined by nuclear magnetic resonance. *Diabetes*. 2003;52(2):453-62.
8. Mintz-Katz. Recent MI Analysis of 570 cases *Arch. Intern. Med*. 1947;80:205.
9. Sima JS, La-Due JS, Worblewski F. Significance of SGOT activity following acute myocardial infarction circulation. 1955;11:871.
10. Braunwald, Zipes, Libby. Text book of cardiovascular Medicine. 6th Edition. Sander's Publication; c2001, p. 1040-1062.
11. Wilson P, Agostino WD, Levy RB, *et al.* Predictors of coronary heart disease using risk factor categories. *Circulation*. 1998;97:1837-1847.
12. Rimm E. Stampfer A, *et al.* Body size and fat distribution as predictors of CAD. *Am. Jr. Epidemiology*. 1995;141(12):1117-1127.