International Journal of Biotechnology and Biochemistry
ISSN 0973-2691 Volume 13, Number 2 (2017) pp. 123-131

http://www.ripublication.com

Lipid profile in Diabetes Mellitus

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Abstract

Diabetes mellitus is the most common metabolic disorder affecting the people all over the world. Diabetes mellitus has been known to be associated with lipid disorders and cardiovascular complications. This study is planned to assess the lipaemic changes in diabetes mellitus patients attending the out-patient department of medicine department in Karwar Institute of Medical Sciences and Hospital. Total Cholesterol(TC), Triglycerides(TG), LDL Cholesterol(LDL-C), HDL Cholesterol (HDL-C) levels were studied in serum of diabetes patients. This is a case control study which included 76 patients of diabetes as cases and 50controls of the same age group and sex. All the samples were taken from subjects who fasted for at least 12 hours before the blood collection. The parameters were determined by using fully automated XL-640. The Triglycerides, Total cholesterol, LDL Cholesterol were higher in cases as compared to controls in both IDDM and NIDDM. The HDL-C was lower in NIDDM subjects who were on glibenclamide or metformin and was not significantly altered in IDDM patients on insulin. There was significant correlation between FBS and TC, TG, LDL-C and HDL-C in NIDDM patients. TC, TG, LDL-C showed significant correlation in IDDM subjects.

Key words: Diabetes mellitus, IDDM, NIDDM, lipid profile.

INTRODUCTION

Diabetes mellitus (DM) is a group of metabolic disease characterized by increase blood glucose level resulting from defects in insulin secretion, insulin action, or both[1]. The chronic hyperglycemia of diabetes is associated with longterm damage, dysfunction and disturbance in failure of various organs, especially the eyes, kidneys,

nerves, heart and blood vessels[2]. Patients with type-2 diabetes have increased risk of cardiovascular disease associated with atherogenic abnormalities and dyslipidaemia. Coronary artery disease, especially myocardial infarction is the leading cause of morbidity and mortality worldwide[3]. Hyperglycaemia and atherosclerosis are related in type-2 diabetes[4]. Persistent hyperglycaemia causes glycosylation of all proteins, especially collagen cross linking and matrix proteins of arterial wall. This eventually causes endothelial cell dysfunction, contributing to atherosclerosis. The prevalence of dyslipidemia in diabetes mellitus is 95%[5]. Early detection and treatment of hyperlipidemia in diabetic patients reduces the risk for cardiovascular and cerebrovascular diseases. Lifestyle changes such as diet and exercise are very important in improving diabetic dyslipidemia, but often pharmacological therapy is needed[6]. Lipoprotein metabolism[7]. The rationale of the study was to detect the lipid abnormalities associated with chronic hyperglycemia due to IDDM or NIDDM.

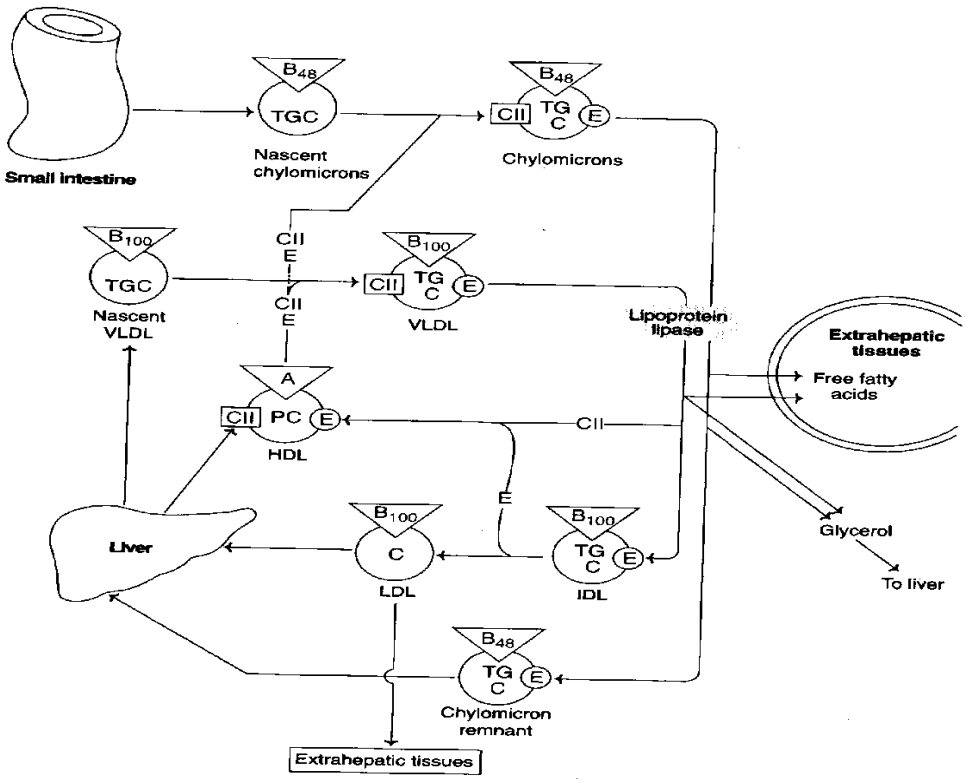
MATERIALS

This study was conducted in Medicine Department, Karwar Institute of Medical Hospital, Karwar. A total number of 50 control who were healthy non smokers non alcoholics and at the time of study all of them were keeping good health and 76 diabetics who were on treatment were studied. The diabetics were on either glibenclamide/metformin or insulin treatment for type l or type Il diabetes. In our study, we excluded diabetic subjects who were smokers, alcoholics and who were hypertensives, familial hyperlipidemia patients and patients with complications.

METHODS

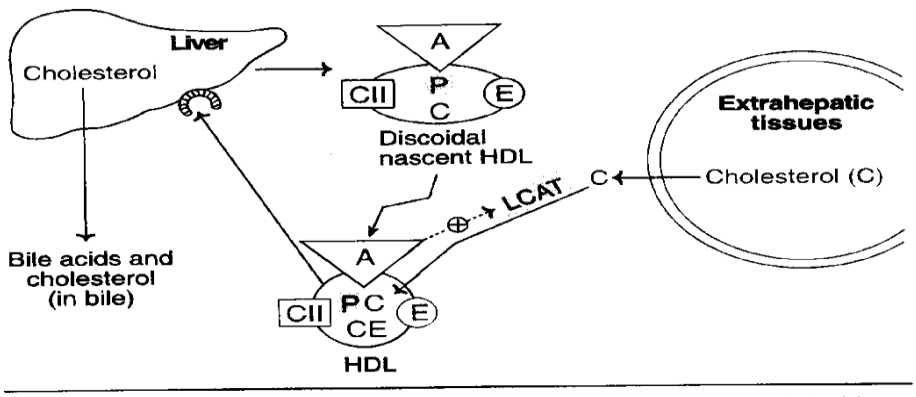
1. Serum glucose estimation by GOD POD (with mutarotase) Method[8].
2. Determination of total cholesterol by CHOD POD method[9].
3. Determination of serum triglycerides by GPO method[10].
4. Determination of HDL-Cholesterol by Modified PEGME method[11].
5. Serum LDL-Cholesterol was calculated by Friedwald's Formula[12].

Figure 01. Summary of metabolism of lipoproteins (Apoproteins–A, B48, B100, CII and E; TG–Triacylglycerol; C–Cholesterol; P–Phospholipid; VLDL–Very low density lipoprotein; IDL–Intermediate density lipoprotein; LDL–Low density lipoprotein; HDL–High density lipoprotein)[7].



Summary of metabolism of lipoproteins (Apoproteins–A, B_{48}, B_{100}, CII and E; TG–Triacylglycerol; C–Cholesterol; P–Phospholipid; VLDL–Very low density lipoprotein; IDL–Intermediate density lipoprotein; LDL–Low density lipoprotein; HDL–High density lipoprotein).

Figure: 02. Metabolism of high density lipoproteins (P–Phospholipid; C–Cholesterol; CE Cholesteryl ester; A, CII, E–Apoproteins; LCAT–Lecithin cholesterol acyltransferase)[7].



Metabolism of high density lipoproteins (P–Phospholipid; C–Cholesterol; CE–Cholesteryl ester; A, CII, E–Apoproteins; LCAT–Lecithin cholesterol acyltransferase).

RESULTS

Table 1: Showing Comparison of FBG. TC.TG.HDL-c. LDL-c Between Insulin Treated Diabetic subjects (IDDM and Control Subjects)

Groups	**FB Glucose Mg/dl**	**TC mg/dl**	**TG mg/dl**	**HDL-c Mg/dl**	**LDL-c Mg/dl**
Control	92.2±11.0	173.0±31.6	122.5±28.7	56.9±17.9	93.3±36.3
IDDM Subjects	229.8±46.2	222.2±23.3	197.9±49.7	58.4±14.6	124.2±25.0
p-value	<.001	<.001	<.001	>.05	>.01
Statistical Significance	H. S	H.S	H.S	N. S	S. S

Table 2: Showing Comparison of FBG. TC.TG.HDL-c. LDL-c Between Diabetic (NIDDM) and Control Subjects.

Groups	**FB Glucose Mg/dl**	**TC mg/dl**	**TG mg/dl**	**HDL-c Mg/dl**	**LDL-c Mg/dl**
Control	92.2±11.0	173.0±31.6	122.5±28.7	56.9±17.9	93.3±36.3
IDDM Subjects	206.2±37.9	229.9±30.5	226.4±59.7	39.5±15.4	145.2±32.5
p-value	<.001	<.001	<.001	<.001	<.001
Statistical Significance	H. S	H.S	H.S	N. S	S. S

Table 3: Showing Comparison of FBG. TC.TG.HDL-c. LDL-c between control and Glibenclamide Treated Diabetic subjects and also with Metformin Treated Diabetics

Groups	**FB Glucose Mg/dl**	**TC mg/dl**	**TG mg/dl**	**HDL-c Mg/dl**	**LDL-c Mg/dl**
Control	92.2±11.0	173.0±31.6	122.5±28.7	56.9±17.9	93.3±36.3
IDDM Subjects	211.7±41.7	226.0±30.3	220.2±64.4	37.9±17.2	146.0±28.2
p-value	<.001	<.001	<.001	<.001	<.001
Statistical Significance	H. S	H. S	H. S	H. S	H. S
Control	92.2±11.0	173.0±31.6	122.5±28.7	56.9±17.9	93.3±36.3
IDDM Subjects	191.7±27.1	223.6±27.1	246.2±50.5	41.2±10.7	143.2±39.2
p-value	<.001	<.001	<.001	<.001	<.001
Statistical Significance	H. S	H. S	H. S	H. S	H. S

1. The values are expressed as their mean ± S.D

2. H.S — highly significant, S. S-Statistically significant, N. S- not significant.

Table No 1: Shows the comparison between the estimated levels of FBG, Tc, Tg HDL-c in healthy controls and in IDDM subjects. It is evident from the table that there are increased levels of FBG, TC, TG, HDL-c and LDL-c in IDDM subjects as compared to controls. The "p" value is highly significant for FBG, TC and TG, statistically significant for LDL-c and not significant for HDL-c.

Table No 2: Shows the comparison between the estimated levels of FBG, Tc, Tg, HDL-c in healthy controls and in NIDDM subjects on oral hypoglycemic. It is evident from the table that there are increased levels of FBG, TC, TG and LDL-c, but decreased levels of HDL-c in NIDDM subjects as compared to controls. The "P" value is highly significant statistically for all the parameters.

Table No 3: Shows the drug wise comparison between the estimated levels of FBG. TC, TG, HDL c in healthy controls and in NIDDM subjects treated with glibenclamide and metformin separately. It is evident from the table that there are increased levels of FBG, TC, TG and LDL-C, but decreased levels of HDL-C in both glibenclamide and metformin treated NIDDM subjects as compared to controls. The "p" value is highly significant statistically for all the parameters.

DISCUSSION

FASTING BLOOD GLUCOSE

The FBG levels in all the diabetics were highly significant ($p<0.001$) as compared to their respective controls[13,14].

TOTAL CHOLESTEROL

Our study in IDDM and NIDDM are in accordance with earlier studies of John D Bagdade2[15] and James M Falko[13]. Diabetic state appears to be associated with increased synthesis of cholesterol. It has been hypothesized that hyperphagia of diabetes induces increased activity of HMG-CoA reductase of the intestine resulting in increased synthesis of cholesterol leading to raised levels in plasma. Dietary cholesterol also adds up to total cholesterol by increased absorption[16].

TRIGLYCERIDES

In our study the TG levels in IDDM as well as NIDDM on insulin as well as sulfonylurea are glibenclamide treated diabetics are raised and statistically highly significant. The hypertriglyceridemia may be due to higher rates of production of triglyceride rich VLDL by the liver[17] and to decreased removal of TG by peripheral tissues-primarily adipose tissue and muscle. Insulin deficiency leads to high TG production and subsequent high packaging in VLDL. Several studies using radioactive substrates to trace the metabolism of plasma VLDL are consistent with their simultaneous overproduction and reduced clearance as the common etiologic mechanism for hypertriglyceridemia in poorly controlled IDDM[18] . Furthermore, the structural composition of the VLDL itself may change with increase in protein components such as apolipoprotein C-III that inhibits the lipase enzyme and the uptake of VLDL remnants by the liver[19].

In NIDDM when TG are elevated above 200 mg/dl higher production rates of triglyceride and VLDL particles have been the most commonly identified metabolic abnormalities[20]. Many hyper-triglyceridemic NIDDM patients also appear to have defect in the clearance of triglyceride with lipoproteins. structural composition of the

VLDL itself may change with increase in protein components such as apolipoprotein C-III that inhibits the lipase enzyme and the uptake of VLDL remnants by the liver[19] . In NIDDM when TG are elevated above 200 mg/dl higher production rates of triglyceride and VLDL particles have been the most commonly identified metabolic abnormalities[20]. Many hypertriglyceridemic NIDDM patients also appear to have defect in the clearance of triglyceride with lipoproteins.

HIGH DENSITY LIPOPROTEIN

The mean levels of HDL-C in our study in IDDM on insulin therapy are not statistically significant as compared to match controls which is in accordance the study of Kennedy and associates but not in variance with that reported by Nikkilaet al[21] . Celvert et al have reported low HDL-C in patients on glibenclamide our study shows low HDL-C in both glibenclamide and metformin treated diabetics[22]. Lower HDL-C in diabetes may be due to reduced Lipoprotein Lipase activity. The activity of cholesterol ester transfer protein is increased in IDDM[18]. Hepatic TG lipase (HTGL) which lines the sinusoids of the liver which break TG added HDL is increased in the diabetic an inversely correlated with HDL LOW DENSITY LIPOPROTEINS Our study confirmed the stud. of Sosenko et al who have reported increase levels of LDL-C in IDDM[23]. The mean LDL-c levels in total NIDDM subjects on glibenclamide and patients on metformin are increased ($p < 0.001$) as compared to the matched controls. LDL production rates are reported to be elevated in IDDM but return to normal after insulin infusion[20]. It may be due to increased synthesis of VLDL or impaired removal of VLDL remnant. Impaired receptor mediated clearance of LDL has also been postulated[21]. In NIDDM there is alteration in the LDL lipid composition, and the LDL is enriched with triglycerides. LDL in patients with hypertriglyceridemia show decreased receptor binging to cultured skin fibroblasts, which may be the mechanism of increase in LDL-C in NIDDM[22].

CONCLUSION

This showed that every patient had at least one type of dyslipidemia. Overall diabetes mellitus is closely associated with dyslipidemia in both IDDM and NIDDM. It is mandatory to treat this dyslipidemia to prevent adverse lipemic status and long term complications to ensure a healthy and happy life inspite of diabetic dyslipidemia.

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