BEST: International Journal of Humanities, Arts, Medicine and Sciences (BEST: IJHAMS)
Vol. 1, Issue 1, Oct 2013, 27-36
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STUDY OF ATHEROSCLEROSIS IN NORMAL AND DIABETIC PATIENTS

A. S. M RUHUL QUDDUS[1], S. M. HOSSAIN[2] & ZAKARIA AHMED[3]

[1]Department of Pathology and Endocrinology, BIRDEM, Dhaka, Bangladesh

[2]University Dental College and Hospital, Dhaka, Bangladesh

[3]Department of Microbiology, Priemasia University, 9 Banani, Dhaka, Bangladesh

ABSTRACT

Atherosclerosis disease and its complication in Bangladesh population are similar to those else where in the world. Low density lipoprotein cholesterol is considered as an independent rich factor may positively be associated with atherosclerotic diseases of future coronary artery disease, cerebrovascular disease or peripheral arterial disease. The risk for CAD (Chronic Arterial Disease) is higher in diabetic subject than non-diabetics. Glycation of tissue proteins and other macromolecules and excess production of polyl compounds from glucose are among the mechanisms thought to produce tissue damage from chronic hyperglycaemia. Type 2 Diabetes Mellitus is associated with a marked increase in atherosclerotic micro and macro- vascular diseases involving cardiac, cerebral and peripheral large vessels.

KEYWORDS: IMT (Intima Media Thickness) Atherosclerosis, Glycosylated Haemoglobin: Type 2 Diabetes, Low Density Lipoprotein Cholesterol

INTRODUCTION

Atherosclerotic risk of diabetes and the patients with the recently recognized metabolic syndrome is increasing. Enlargement of intima media thickness (IMT) is an early predictor of atheroma formation. Vasorelaxation especially endothelium dependent is impaired in type 2 diabetes and other insulin resistant states. Diabetes mellitus is a group of metabolic disorder characterized by a state of hyperglycaemia resulting from defects in insulin secretion, insulin action or both (1-3). The chronic hyperglycaemia is associated with long term damage dysfunction and failure of various organs, especially the eyes, kidneys, liver, heart and blood vessels. Diseases of arteries are responsible for more morbidity and mortality than any other type of human disease. Atherosclerosis is characterized by intimal lesions called atheromas or atheromatous fibrofatty (lipid) plaques which protrude into and obstruct vascular lumens and weaken than underlying media. Dyslipidemia is defined as an abnormal level of one or more blood lipids which must typically are total cholesterol low density lipoprotein (LDL), High density lipoprotein (HDL) and/or triglyceride (TG) there is strong evidence that elevated LDL and decreased HDL levels directly contribute to the formation of atherosclerotic plaques, in turn, increase the patient's risk of cardiovascular disease. Hyperglycaemia may cause increased glycation leading to accumulation of basement membrane collagen and membrane leakiness.

Stimulation of intracellular polyol pathway leading to basement membrane and capillary endothelial cell damage. Overall effects of hyperglycaemia include capillary basement membrane thickening, protein leakage, micro thrombus formation and tissue ischemia.Diabetes mellitus induces hypercholesterolemia and a markedly increased predisposition to atherosclerosis. The incidence of myocardial infection is twice as high in diabetes as in non-diabetes (4-6). There is also an increased risk of strokes and even more striking, perhaps a 100-fold increased risk of atherosclerosis induced gangrene of the lower extremities. Most arterial occlusive disease to produced by atherosclerosis. Atherosclerosis is a generalized response of the artery wall to injury. Atherosclerotic plaque are characterized by smooth muscle migration into the intima

and subsequent proliferation and extracellular lipid deposition. Complex lesions are composed of a fibrous cap containing smooth muscle and inflammatory cells overlying a central core of lipid-rich necrotic debris (7).

Circulating lipoproteins are just as dependent on insulin as in the plasma glucose. In type-1 diabetes, moderately deficient control of hyperglycemia is associated with only a slight elevation of LDL cholesterol & serum triglycerides and little if any change in HDL cholesterol. Once the hyperglycemia is corrected, lipoprotein levels are generally normal. However, in obese patients with type 2 diabetes a distinct diabetic "dyslipidaemia" is characteristic of the insulin resistance syndrome. Its features are a high serum triglyceride level a low HDL cholesterol and a qualitative change in LDL particles producing a smaller dense particle whose membrane carries supernormal amount of free cholesterol.

These smaller dense LDL particles are more susceptible to oxidation which renders them more atherogenic. VLDL production in increased by enhanced delivery of NEFA (Non-esterifies fatty acid) and increased hepatic VLDL apo B synthesis. High density lipoprotein (HDL) levels are reduced because of increased exchange of cholesterol esters for triglyceride medicated by cholesterol ester transfer protein (CETP). Small dense low density lipoprotein (LDL) particle also predominate in insulin resistance state and are highly atherogenic. Lipoprotein lipase (LPL) in adipose tissue hydrolyses triglyceride in circulating chylomicrons and VLDL, thus providing adiposities with NEFA (non esterified fatty acid). Insulin acutely increases the activity increase the activity of adipose- tissue LPL, an effect which appears to be blunted in insulin resistant conditions such as obesity. Insulin normally decreases LPL (lipoprotein lipase) activity in skeletal muscle (8-10).

This action combined with insulin's stimulation of adipose tissue LPL tends to "Keep fat where it belongs" i.e. adipose tissue. In insulin resistant obese subject, insulin may increase rather than decrease LPL activity in skeletal muscle. Many but not all patient with type 2 diabetes show subnormal activity of adipose tissue LPL, and this shows the clearance of VLDL particles and chylomicrons. Procoagulant changes such as impaired fibrinolysis & increased levels of plasminogen activator inhabitor-1 (PAI-1) as well as defects in platelet function are frequently associated with insulin resistance, especially in obese patients with type 2 diabetes. These abnormalities predispose to atherothrombotic vascular disease (9, 11-13).

Arterial structure and compliance- The intima media thickness (IMT) of the arterial wall and the area occupied by atherosclerotic plaques can be measured in vivo using "high-resolution B mode (Two-dimensional) ultrasound scanning). IMT measurements employ multiple recordings with a 10-MHz ultrasound probe and frame grabbing software in the common carotid or femoral artery. In experience hands, measurements are highly reproduced with within subject co-efficient of variation around 2.4% and increased IMT is a sensitive indicator of early atherosclerosis. In post mortem carotid artery and aorta the ultrasound images closely match and histological findings of arterial wall thickening and atherosclerotic plaques and in vivo carotid IMT has been shown to correlate with the extent of coronary atherosclerosis on angiography (14). Arterial wall compliance can be assessed by direct visualization of vessel wall motion with high resolution, edge detection two dimensional ultrasound, mechanically transducing the pulse waveform or detecting arterial pulse waves using Doppler ultrasound.

These techniques have demonstrated that arterial wall compliance is reduced (i.e the wall is stiffer) in type 2 diabetes and in subjects with hypertension and other cardiovascular risk factors that define the metabolic syndrome, recently mechanical transduction of the peripheral arterial waveform using explanation goniometry has emerged as a versatile and reproducible method for measuring arterial compliance and endothelial function (15). It has also been shown that insulin reduces article wall stiffness (Compliance) and that this action is attenuated to some condition of metabolic to

some conditions arterial contractility (Vasomotion) is also impaired in diabetes, hypertension dyslipidaemia and obesity. Arterial vasodilatation depends crucially on the release of metric oxide (no) try the endothelium, which acts to relax the vascular smooth muscle of the arterial media. This endothelium dependent vasodilatation is triggered by numerous stimuli, including acetylcholine, insulin and increased shear stress on the endothelium during hyperaemia. Hyperchromocystinaemia is common among diabetic patient and it may contribute to the accelerated risk of atherosclerosis and cardiovascular disease (16-19). In the general population higher intake of folate & vit B6 has been associated with lower risk of CHD, probably through reducing plasma homcysteine levels. It is now clear that reduced HDL- Cholesterol and increased triglyceride concentrations many be more important in diabetic patients. Several prospective studies have reported highly significant association between LDL- Cholesterol concentrations and the risk of CHD. It is also clear that specific subtypes of LDL- Cholesterol that are prevalent in type 2 diabetes (notably the small dunce LDL fraction) are particularly atherogenic.

There is now considerable evidence that that a low HDL cholesterol concentration is associated with a high risk of CHD: indeed this is the commonest lipoproteins abnormality in patients with CHD. More ever most subjects with low HDL cholesterol who develop CHD also have moderately elevated triglyceride concentration. This high triglyceride low HDL Cholesterol dyslipidaemia is a common feature of patients with type 2 diabetes and predicts a substantially increased risk of CHD. My work has indicated that this specific atherogenic dyslipidaemia frequently result from abdominal obesity which is also accompanied by insulin resistance and the other features of the “insulin resistance” or metabolic syndrome. Individual elements of the metabolic syndrome are atherogenic and in combination, carry a substantially increased risk of CHD. Risk remains high for overweight obese patients with features of the metabolic syndrome and high triglyceride low HDL cholesterol dyslipidaemia even in the absence of hyperglycaemia or type2 diabetes and even if LDL- cholesterol concentration are apparently within the normal range.

Thus although insulin resistance is a major risk factor for glucose intolerance CHD may develop before hyperglycemia or type 2 diabetes supervene (20). This emphasizes the importance of implementing appropriable preventive measures in overweight individuals at high risk of both type diabetes and CHD. Indeed an “atherogenic metabolic triad” can be identified comprising fisting hyperinsulinaemia elevated alp B concentrations (the main component of atherogenic lipo proteins, including VLDLs, IDLs and LDLs) and an increased proportion of small dense LDL particles asymptomatic middle aged men with these three key features show a 20 fold increase in the risk of developing ischemic heart disease. Both type 1 and 2 diabetes increase cardiovascular risk 2-4 fold compound develops curlier and faster in diabetes, leading to widespread lesion throughout the arterial tree including the smaller arteries. Thickening of the intima is an early change “Hyaline degeneration and thickening” of the muscular media may contribute to hypertension and often undergoes calcification (Medial Sclerosis).

An important functional abnormality is impaired arterial relaxation, due to failure of the endothelium to produce nitric oxide (N.O) a potent vasodilator. Procoagulant changes on the endothelial surface promote adhesion of macrophages (the precursors of foam cells of the atheromatous plaque) and platelets, favoring thrombosis (21). Platelet rich thrombus in the coronary arteries is unstable and likely to rupture, causing acute coronary occlusion. Presence of diabetes amplifies the effect of other coexistent risk factors. Disorders of coagulation and fibrinolysis are also associated with metallic syndrome and probably contribute athero-thrombotic disease. Hyperglycaemia leads to age advanced glycation end product (AGE) formation in the arterial wall, damaging structural proteins and generating toxic reactive oxygen species, sequelae increased endothelial permeability; impaired NO (Nitric Oxide) mediated vasorelaxation, up regulation of procoagulant and attraction of macrophages that form foam cells. AGEs inter act with specific receptors (RAGEs) on endothelial and

other cells to causes specific effects; genetic polymorphism of the RNGE gene way modulate production of inflammatory mediators in arteries, and thus cardiovascular risk. Insulin resistance is strongly associated with cardiovascular risk. Insulin stimulates vascular smooth muscle cell proliferation & production of the fibrinolysis inhibitor, plasminogen activator inhibitor 1 (PAI-1) high insulin levels in insulin resistant states may therefore by atherogenic. Other commonly associated risk factors are hypertension (30-40% of diabetic people), dyslipidaemia and obesity, an independent risk factor (22).

Hypercoagulability may contribute to vascular risk in diabetes, insulin resistance is associated with high levels of procoagulant proteins (e.g fibrinogen, factor VII, Von Willebrand factor) with suppressed fibrinolysis due to increased concentrations of PAI-1, Underlying mechanisms may include raised insulin, triglyceride and inflammatory cytokine levels. Atheromatous plaques in patient with diabetes are not qualitatively different from those affecting the non-diabetic population. However, atheroma in diabetes is more extensive more difficult and involves distal vessels in both the coronary and peripheral circulations. Coronary lesions are more prone to plague ulceration and instability which predisposes to thrombus formation and arterial occlusion and thus to unstable angina or myocardial infarction.

The excess of recognized risk factors (notably hypertension and dyslipidaemia) in diabetic patients probably accounts for a considerable proportion of the increased burden of atheroma. In diabetic patient the arterial intima in thickened and this can be measured clinically using high resolution Doppler scanning in the carotids and other large arteries. Intimal thickening tends to increase with duration of diabetes and severity of hyperglycemia. Media layers of arterioles throughout the circulation show “hyaline” change an amorphous ground glass appearance resulting from breakdown of structural proteins (largely thought to be collagen) and the uptake into the vessel wall of glycated plasma proteins. Similarly changes are seen in older non-diabetic subjects as part of the ageing process and in hypertension.

The hyaline material has a high glycoprotein content and characteristically stains positive with periodic acid Schiff (PAS) reagent. Small & medium arteries and arterioles in diabetic subjects are further damaged by reduced blood supply resulting from microvascular disease affecting the vasa vasorum.Lipid metabolism is commonly deranged by the diabetic milieu often with additional contributions from coexistent renal or hepatic disorders this lipid alterations plays an important role in the development of atherosclerosis and contribute to the development of atheromatous plaques. Gross hypercholesterolaemia is not a feature of diabetic dyslipidaemia but at any given level of cholesterol a diabetic subject has two to three times and cardiovascular risk of a non diabetic.

Lipid abnormalities associated with type 1 diabetes are largely related to the level of glycaemic control. Hyperglycaemia is associated with raised low density lipoprotein (LDL) cholesterol and triglyceride concentrations and low HDL cholesterol, abnormalities which are received by normalizing glycaemia (8, 11, 23). The patter of dyslipidaemia in type 2 diabetes is that characteristically seen in the insulin resistant states and the metabolic syndrome. Low HDL and raised triglyceride concentrations are accompanied by normal LDL cholesterol levels, although this is likely to be dominated by highly atherogenic small dense LDL particles non-diabetic and insulin resistance first degree relatives of type 2 diabetic subjects share this atherogenic profile, suggesting that it preceds the development of clinical diabetics so the reduction or control of blood glucose level may lower the lipid risk factor for cardiovascular diseases. The purpose of the present study was to reduce the chronic complications of diabetes mellitus i,e micro and macro vascular complication which are occur due to atherosclerosis. Atherosclerosis is several fold more frequent in persons with diabetes. In diabetic person atheromatous lesions are more severe and wide spread. It was aimed to compare lipid profiles with special attention to low density lipoprotein cholesterol between uncontrolled (Hb A1c>9%) moderately controlled (≥7.0%-≤9.0% Hb A1c) and controlled (HBA1c <7.0%) diabetic subjects to explore the association of glycaemic status with CHD risk factors.

MATERIALS AND METHODS

It is a cross sectional study. Patient from Azimpur, New Market, Elephant road, hatirpul area of Dhaka city. Study was carried out in the Department of Pathology and Endocrinology BIRDEM Dhaka, Bangladesh in 600 patients, normal person, diabetic patient well controlled, moderately controlled and uncontrolled patients during June 2009 to August 2010. Positive Sample were taken for study and all patients were given an explanation of the study and informed consent was taken before entry into the study. Clinical assessment was done at baseline. Then confirmed by laboratory investigations.Clinical data were collected by structured questionnaire. Questionnaires are- (a) Name of the Patient, (b) Age, (c) Sex, (d) Type of Symtoms, (e) Site of Lesion, (f) Site of first involvement, (g) Duration of illness (in year), (h) Family history, (i) Medication History, (j) Weight of the patient, (k) Waist circumference in centimeter, (l) Blood pressure measurement, (m) History of lifestyle and (n) Laboratory test report.

All data were collected and edited after collection. Then the data were entered into computer and analyzed with the help of SPss (Statistical package of social science) with 12 software programmed. Six hundred specimens obtained from confirmed diabetic subjects during June 2009 to August 2010 were analyzed. Total study subjects were grouped into there categories i.e controlled, moderately controlled and uncontrolled diabetes depending on HbA1c values. Total study subjects were also grouped into male and female categories. According to age, subjects were grouped into three age groups (upto 40 years, from 41 to 60 years and above 61 years).

Fasting and post prandial plasma glucose levels were measured by GOD-PAP method in Dimension RxL max auto-analysis (Simens Healthcare Diagnostics Ltd) Glycosylated Haemogloin level (HbA1c) was measured by HPLC based method (D-10 TM, Hemoglobin testing system, Bio-Rad Laboratories, inc, Hercules, CA 94547, USA) as a marker of glycaemic status and serum total cholesterol, serum triglycerides and serum high density lipoprotein cholesterol concentration were measured by Dimension RxL max auto analyzer (Siemens Health Care Diagnostics Ltd) sir William Siemens of frimly, camberly, UK GU 16 8QD) serum low density lipoprotein cholesterol was calculated by friedewald's formula i.e [LDL Cholesterol]= [Total cholesterol]- [HDL Cholesterol]- [Plasma TG]/2.175. Results are expressed as mean ±SD and compared by unpaired *t* test. Statistical analysis was performed by STATISTICA 6 and graph pad prism 5. Lipid profiles were compared by unpaired *t* test in different diabetic groups. Lipid profiles were also compared between male and female groups and also compared is different age groups.

RESULTS AND DISCUSSIONS

The manse age of the total study subjects was 49.92< 11.7 years. The mean SD of HbA1c Level was 9.37+2.64 serum total cholesterol was 186.46+ 42.06 mg/dl, Serum high density lipoprotein cholesterol was 36.85+8.02 mg/dl, serum triglycerol was 191.31+ 123.57 mg/dl and calculated serum low density lipoprotein cholesterol was 112.82+35.78 mg/dl in the total study subjects 21% of the study population were within the controlled diabetic group (HbA1c<7.0%), 30% were moderately controlled diabetic group (HbA1c-7.1<9.0%) and 49% were uncontrolled diabetic group (HbA1c>9.0%) of the total study subject6s 301 were male and 299 were female 25% of the study subject were within the age group of 41-60 years and 17% were above 61 years age group. comparison of plasma glucose and lipid parameters in different HbA1c groups shown in table1, it is evedent from table 1 that fasting plasma glucose and post prandial plasma glucose levels differ significantly among different HbA1c groups. serum total cholesterol did not differ significanlty in the moderately controlled diabetic groups compared to controlled diabetic group but the difference between moderately controlled diabetic group and uncontrolled diabetic group and also between uncontrolled and controlled diabetic groups were statistically significant (Table1), serum triglycerol level differs significantly between uncontrolled and controlled diabetic gropes but

the difference was not significant between controlled and moderately controlled diabetic groups and not between moderately controlled diabetic groups and not between moderately controlled and uncontrolled diabetic groups (Table 1). There is significant difference of serum high density lipoprotein cholesterol level among three diabetic groups.

The difference of the serum low density lipoprotein cholesterol between moderately controlled and controlled diabetic groups is not significant but LDLC level in the uncontrolled diabetic group is significantly different from controlled and moderately controlled diabetic groups, HbA1c, fasting plasma glucose post-prandial pleasure glucose, serum TG and LDL level did not differ significantly between male and female diabetic groups but serum total cholesterol and serum HDLC level was significantly higher in female than male diabetic groups (Table 2). HbA1c Fasting plasma glucose, post-prandial plasma glucose. Serum TG level did not differ significantly among three different age groups but serum TG & HDL was significantly lower in the age group of >61 years than other two groups and serum LDLC level was significantly higher in the age group of 41-60 years than other two age groups (Table 3).

Distribution of serum LDLC into 04 (Four) different LDL-C ranges slowed that the controlled diabetic group has the maximum subjects with optimal level of LDL-C ranges slowed that the controlled diabetic group has the maximum subjects with optimal level of LDL-C (Up to 100 mg/dl) and least number of subjects has LDL-C level above 130 mg/dl (17%) than moderately and uncontrolled diabetic subjects (Table 4). In moderately and uncontrolled percentages of diabetic patients having LDL-C above 130 mg/dl are higher than controlled diabetic subjects (26% and 38% respectively) (Table 4)

In diabetics advanced glycosylation end products induce endothelial activation critical to the pathogenesis of adhere sclerosis. High blood glucose level damages the endothelial cells living the plod vessels making them thick, hard and less elastic. This makes it difficult for the blood to flow through. People with diabetes have higher level of fat in the blood (24-27). Again high blood glucose contributes to this. The fat or lipids in the blood vessels may clog the vessels and restrict that elevated LDL and decreased HDL levels directly contribute to the formation of atherosclerotic plaques, in turn, increase the patient's risk of cardiovascular disease. Though the consecration of total & LDL cholesterol in type2 diabetic patients in usually not significant different from non-diabetic individuals, the diabetics may have elevated levels of non-HDL cholesterol (LDL & VLDL).

However type 2 diabetic patients typically have a preponderance of small and dense LDL particles, which possibly increase atherogenicity even if the absolute concentration of cholesterol is not significantly increased (28). This study was first of its kind conducted in a urban population of Bangladesh and it could investigate all cluster components needed for the three diagnostic criteria proposed by WHO, NCEP-ATP III (National cholesterol education program adult treatment panel III and IDF (International Diabetic Federation). The study revealed that except TC (Total Cholesterol) and HDLC, there was no significant difference of mean TG and LDLC level between male and female diabetic groups. Male diabetic subjects have lower level of TC and HDLC than their female counterparts.

As the differences of mean HDLC was highly significantly than that of total cholesterol, females are more cardiovascular disease protection than male. This finding is consistent with results of other research work carried out in our country as well as in different parts of the world. The influence of age on lipid parameters was not significant except TG and LDLC, but comparison of lipid parameters showed that TG level was significantly lower in the age above 61 years than other age groups and LDLC level was significantly higher in the age group of 41-60 years than other age groups. So the middle age group may be less cardioprotective.

The result is consistent with the finding of other work. High LDLC level was also obtained in moderately controlled and uncontrolled diabetic subjects. The distribution of LDLC in different LDLC range also showed the higher risk of uncontrolled and moderately controlled diabetic subjects than controlled diabetic subjects significant positive conflation was observed between HbA1c with TC (r= 0.205, P= 0.0000004) and LDLC (r=0.193, P=0.0000004). In our study put with triglyceride was weak (r=0.0825, P- 0.043). LDL hyperlipoproteinemeia (LDLC>100 mg/dl) was 46% and HDL Dyslipidemia (HDLC<35mg/dl) was 60% of the total study subjects which is some what higher than obtained in a study carried out in India so the overall risk of CAD is alarming in our population. It is evident from the above findings that uncontrolled diabetic subjects have higher risk of CAD than controlled and moderately controlled diabetic subjects.

Diabetic mellitus as a disease has earned its importance by its complications. Duration as well as degree of hyperglycemia is associated with both macrovascular e.g cardiovascular, cerebrovascular, peripheral vascular disease and microvasular e.g retinopathy, nephropathy, neuropathy, neuropathy angiopathies in diabetic individual. Both lessens may result in organ and tissue dysfunctions which are designated as chronic complications of diabetics individual (29).

Now it has become clear that diabetes mellitus is not just an acute metabolic threat to life, but also causes chronic complications. Some of which may lead to premature death or considerable morbidity result from many studies namely DCCT (Diabetic Complication and control Treatment) and UKPDS (United kingdom prospective diabetic syndrome) have shown that the morbidity as well as mortality risk associated with diabetes can be reduced by strick dslipedemia and intensive glycaemic control (30). Our perspective studies with diabetic persons have documented that reduction of blood lipids particularly TG and LDL cholesterol reduces their cardiovascular events.

On the basis of that evidences now it is advice to lower the triglyceride and LDL cholesterol and to rise HDL cholesterol. Since the study conducted in one selected hospital only, the study result may however differ from other hospitals and may not reflect the actual picture of Bangladeshi population. Epidemiological evidences suggest that the incidence of diabetic is increasing worldwide.

The managements of diabetes mellitus and the management & prevention of the complications are important challenges of the presents time. There are ample evidences from applied clinical research that morbidity and mortality risks associated with diabetes are preventable (21, 17, 23). So management of hyperglcaemia by the lifestyle modification become more important as becomes that needs to be synchronized with drug/insulin administration. The important issues of lifestyle of a diabetic induce their dietary habit and involvement in monitoring blood glucose such as self monitoring of blood glucose.

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APPENDICES

Table 1: Comparison of Plasma Glucose and Lipid Parameters in Different HbA_{1c} Groups

Group I	Group II	Group III
FPG (mmol/L)		
5.597±1.249	7.554±1.943***	11.48±4.287*"
PPG (mmol/L)		
8.639±2.643	11.32±3.143*"	16.53±5.467***
HbA1c (%)		
6.251±0.579	7.969±0.566"*	11.61±1.88***
TC (mg/dL)		
174.3±33.91	182.2±42.15^{NS}	194.4±43.65","*
TG (mg/dL)		
169.3±90.94	190.7±127.8^{NS}	201.3±132.0^{NS}"*
HDLC(mg/dL)		
37.12±8.33	36.85±7.65^{NS}	34.4±7.11
LDLC (mg/dL)		
104.7±30.79	108.8±35.11^{NS}	118.9±37.24","*

Group I, controlled; Group II, uncontrolled and Group III, uncontrolled diabetic subjects; NS, not significant; *=P<0.05; **=PO.01;*"= P<0.001.

Table 2: Comparison of HbA_{1c}, Plasma Glucose and Lipid Parameters Between Male and Female

Parameter	Male	Female
FPG (mmol/L)	8.887±3.851	9.357±4.125^{NS}
PPG (mmol/L)	13.48±5.612	13.08±5.299^{NS}
HbA_{1c}(%)	9.351 ±2.698	9.387±2.583^{NS}
TC (mg/dL)	182.5±42.16	190.4±41.65*
TG (mg/dL)	199.1±141.7	183.5±101.7^{NS}
HDLC (mg/dL)	34.4±7.11	39.31±8.14***
LDLC (mg/dL)	110.5±36.44	115.2±35.00^{NS}

NS, not significant; *, P<0.05; ***, P<0.001.4).

Table 3: Comparison of Plasma Glucose and Lipid Parameters in Different Age Groups

Up to 40 Age(yrs)	41-60 Age(yrs)	>60 Age(yrs)
FPG(mmol/L)		
8.837±3.455	9.159±3.951	9.402±4.801
PPG(mmol/L)		
12.78±4.976	13.43±5.539	13.50±5.839
HbA1c (%)		
9.342±2.705	9.332±2.498	9.534±3.011
TC (mg/dL)		
183.3±38.85	188.8±42.85	183.1±43.66

TG(mg/dL)		
206.9±152.7	193.5±121.6^{NS}	161.3±65.37*,**
HDLC (mg/dL)		
36.60±7.623	36.71 ±8.045 ^{NS}	(37.68±8.517*S.NJ9
LDLC (mg/dL)		
108±33.56	114.8±35.26* 1	13±40.08NS,NS

NS, not significant;*, PO.05;**, P<0.01

Table 4: Distributions of LDLC in Controlled, Moderately Controlled and Uncontrolled Diabetic Subjects in Different LDLC Level

LDLC Range	upto lOO mg/dL	101-130 mg/dL	131-150 mg/dL	>150 mg/dL
Group I	49%	34%	9%	8%
Group II	43%	31%	14%	12%
Group III	34%	27%	17%	21%

Group I=controlled diabetic subjects; Group II=moderately controlled diabetic subjects and Group III=uncontrolled diabetic subjects.