IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 1 Ver.VII (Jan. 2015), PP 13-22
www.iosrjournals.org

Study of Lipid Profile in Diabetes Mellitus Patients Who Were On Glibenclamide and Glimeperide

Dr. Indirakumari N[1], Dr.Vinutha. S[2], Dr. Chandrakala Kambar[3]
[1] *Associate Professor, Andhra Medical College, Visakhapatnam.*
[2] *Civil Assistant Surgeon, King George Hospital, Visakhapatnam*
[3] *Associate Professor, Siddhartha Medical College, Vijayawada.*

Abstract: *Hyperlipidaemia is the most common associated disorder of Diabetes mellitus which is a predisposing factor for cardiovascular complications leading to death. Hypoglycemic drugs are having variable influence on lipid profiles. Hence a study of each hypoglycemic drug is required and is to be compared with another cardio protective anti diabetic agent in relation to risk factors like lipid profile gives the effective information for selectivity of better drug selection basing on lipid profile features as a preliminary cardio protective measure. This study was taken up to show the effect of hypoglycemic drugs on lipid profile, taking two groups A and B with 30 patients in each, given Glibenclamide and Glimepiride drugs for 6 weeks respectively. After 6 weeks of therapy with these agents, Lipid profile and blood sugars were estimated and compared with the pre treatment levels. It was found that, Group"A" with Glibenclamide, there is a significant fall of FBS, TC, TG & HDL. FBS: The mean fall of BS is 19.9↓, TC: The mean fall is 4↓, TG: The mean fall is 5↓, HDL: The mean fall is 2↓, LDL: The mean rise is 2↑ & VLDL: The mean fall is 1↓ and in Group"B" with Glimepiride, there is a significant fall of FBS, TC, TG, LDL, VLDL and rise in HDL FBS: The mean fall of BS is 21.9↓, TC: The mean fall is 24↓, TG:The mean fall is 13.34↓, HDL: The mean raise is 0.5↑, LDL: The mean is 14↓ fall VLDL: The mean fall is 3↓. Patients who were treated with Glimepiride (Group B), there is a significant fall in the levels of total cholesterol, Triglycerides & LDL levels comparatively. And there is slight raise, in the levels of HDL, but not significantly.*

Keywords: *FBS, Glibenclamide, Glimeperide, HDL, LDL, TC, TG, VLDL.*

I. Introduction

Diabetes Mellitus is a metabolic disorder characterized by hyperglycemia, glycosuria, hyperlipidaemia, negative nitrogen balance some times ketonaemia[1]

Diabetes mellitus at the outset is not a single entity, but a combination of many related physiological functions and biochemical features involving different endogenous features. It is an established fact that etiology of diabetes mellitus is mostly hereditary origin and genomic specifications. In this modern age, many advances are coming in usage of therapeutic agents, which are closely and almost directed by the changes in the target organs and these physiological, biochemical and pharmacodynamic changes.

More scientific research is available in relation to antidiabetic therapy with insulin and other non-insulin agent. In this regard different oral hypoglycemic agents are available and evaluation of efficacy of these drugs has been always exposed for further betterment and more margin of safety, keeping in view the development of endogenous complications, which may in turn the causative factors for involvement of the other vital organs like kidney, heart, vulnerable vascular endothelium with altered lipid profiles and finally loss of immunity.

Hypercholesterolemia and onset of high levels of triglycerides, LDL, HDL, VLDL are associated with Diabetes Mellitus. Study of these risk factors is essential because these are intervening with the management of Diabetes Mellitus. Glycated LDL can be more atherogenic than native LDL. The study of these risk factors in DM became essential in following the usage of different therapeutic hypoglycemic agents. Even though much research has been going on in Diabetes Mellitus management, still the overall mortality rate among the population is to be reduced. Hence the study of individual hypoglycemic agents and new agents is always helpful to understand comparatively the influence of these agents on risk factors. These risk factors predominantly got a major role on total mortality rate of diabetes, besides with other controls like diet therapy, exercise etc.

Death may result from acute metabolic decompensation, while longstanding metabolic derangement of is frequently associated with permanent and irreversible functional & structural changes in the cellular level of the body, thus resulting hazardous atherosclerotic changes, being the basic root cause for the target organs pathology. In this connection, exclusive studies on lipid profile are required keeping in view that atherosclerosis

and other risk factors among Diabetes Mellitus patients. As these hypoglycemic drugs are having variable influence on lipid profiles. Hence a study of each hypoglycemic drug is required and is to be compared with another cardio protective anti diabetic agent in relation to risk factors like lipid profile gives the effective information for selectivity of better drug selection basing on lipid profile features as a preliminary cardio protective measure. Exclusively most of the literature of the DM comprises of anti diabetic therapy as main aim to bring down blood sugar levels to the normal range, rather some more literature relating to anti diabetic therapy involving the associated pathogenic complications. In this connection there is no exemption for the risk factors like lipid profiles leading to CVS disorders from a series of studies or research, so as to search for measures to prevent the cardiac risk factors. Hence the lipid profiles along with hypoglycemic features among DM individuals are utmost considerable factors for study.

II. Objectives

In this study the objective is a preliminary study among Diabetes Mellitus patients to secure the information towards cardio protective endogenous inference following anti diabetic therapy in which the feature of lipid profile is enmarked as one aspect of cardioprotection.

III. Methodology

The present study was a prospective and comparative study and was conducted on patients suffering from Diabetes Mellitus attending the local hospital, King George Hospital, Visakhapatnam. This study was approved by the Institutional Ethical Committee, King George Hospital. Informed consent was taken from the patients in local language.

The investigations were carried out in clinical laboratory of King George Hospital. Sixty patients with Diabetes Mellitus were taken into two groups. Each group consisted of 30 patients.

All patients were examined clinically and laboratory investigations done including FBS, TC, TG, HDL, LDL, VLDL.The patients were kept on Tab. Glibenclamide, Glimepiride.

Selection of patients:

60 known Diabetes Mellitus patients attending King George Hospital, Visakhapatnam were selected for present study. These sixty patients were divided into 2 groups, group A and group B of 30 each. They were confirmed as a diabetic by the relevant blood sugar levels.

Inclusion criteria:

1. Both Male and Female patients
2. Patients age between 40-70 years of age.

Exclusion criteria:

1. Patients with cardiac, renal or pancreatic dysfunction.
2. Patients on other alternative medicine.
3. Patients on multiple drug therapy
4. Patients who had irregular dietary habits.
5. Patients with recent infections or major surgery.

Investigations:

1. Lipid profile estimation:

Lipid profile was done in all the patients at the time of entry & after 6 weeks of antidiabetic therapy. FBS, Total Cholesterol, TG, HDL, LDL, VLDL were estimated by the enzymatic method using Auto pack kits. These reagents were manufactured by the standard company Bayer Diagnostics India Limited, The patients were asked to come on 12 hours (overnight) fasting, were made comfortable and reassured before collecting 10ml of whole blood. The data was analyzed by using student "t" test for paired values. Probability was read from available tables.

Serum Total Cholesterol estimation(Sr TC): (Allain CC. Et al 1974) [2]

Principle: This is an enzymatic method.

Cholesterol ester + H2O cholesterol esterase cholesterol +Fatty acids

Cholesterol + 02 cholesterol oxidase Cholestenone + H2O2

2H2O2/Phenol/4 - Aminoantipyrine Peroxidase Redquinine + 4H2O

The concentration of cholesterol in the sample is directly proportional to intensity of the red complex (Red Quinine) which is measured at 500nm. The color of the reaction is stable for 2 hours if not exposed to direct light.

The sample Collection: Serum is preferred or heparinised plasma can also be used. The sample should be used on same day. Samples are stable for a week when stored at 2° to 8°C. Reagents:
Reagent/ (Enzymes/Chromogen) Reagent 1A (Buffer) Standard (cholesterol 200mg/dl) Reagent Reconstitution:
Reagent 1 & Reagent 1A are mixed gently. The reconstituted reagent is stable for 3 months when stored at 2° to 8°C.

Procedure:
The samples and the reconstituted reagent should be brought to room temperature prior to use.

	Blank	Standard	Test
Reconstituted	1ml	1ml	1ml
Standard	-	10µl	-
Sample	-	-	10µl

Incubated for 5 minutes at 37°C. Mix and read the colour obtained. And compare the colour with the standard solution.

Serum Triglyceride Estimation(Sr TG): (Buccolo G. David M. 1973) [3]:
This is an enzymatic calorimetric method. The sample is preferably serum (or) heparinized plasma can also be taken. Sample should be used on the same day. Samples are stable for 2-3 days when stored tightly capped at 2° to 8°C fasting blood sample should be used. Principle:

Triglycerides + H2O Lipoprotein lipase Glycerol + Fatty acid Glycerol + ATP Glycerol Kinase Glycerol – 3-Phosphate + ADP Glycerol - 3- Phosphate + O2 GPO Dehydroxyacetone phosphate + H2O2

2H2O2 + 4 - aminoantipyrine + ADPS Peroxidase Redquinine + 4H2O GPO = Glycerol - 3 - Phosphate oxidase ADPS = N - Ethyl - N - Sulphoprophyl - n - anisidine.

The intensity of purple colored complex formed during the reaction is directly proportional to the triglyceride concentration in the sample and it measured at 546nm. Reagents:

Reagent 1 (Enzymes/ Chromogen) Reagent 1A (Buffer) Standard (Triglycerides 200mg./dl) The reagent should not be exposed to direct light. Reagent reconstitution:

Allow the reagent to attain room temperature and mix reagent l & 1A bottles gently. The reconstituted reagent is stable for 6 weeks when stored at 2°C to 8°C.

Procedure:
The samples and the reconstituted reagent should be brought to room temperature prior to use.

	Blank	Standard	Test
Reconstituted	1ml	1ml	1ml
Standard	-	10 µl	-
Sample	-	-	10 µl

Incubated for 5 minutes at 37°C. Mix and read the color compared with the standard solution. The final color is stable at least for 30 minutes.

High Density lipoproteins Estimation: (HDL)

(Allain C A et al 1974) [1];(Castelli W P etal 1986) [4]
This is a phosphotungstate method used to estimate HDL-C in serum or heparnized plasma.

Principle:
Chylomicrons, VLDL, and LDL fractions in serum or plasma are separated from HDL by precipitation with phosphotungstic acid and magnesium chloride. After centrifugation, the cholesterol in the HDL fraction, which remains in the supernatant is assayed with enzymatic cholesterol method, using cholesterol esterase, cholesterol oxidase, peroxidase and the chromogen 4 -Aminoantipyrine / Phenol.

Sample Collection:
Serum is preferred, EDTA or heparnised plasma can also be used. Use the sample on the same day. Samples are stable for a week when stored at 2°C to 8°C.

Reagents:

Reagent 1 (Enzymes/Chromogen) Reagent 1A (Buffer) Reagent 2 (Precipitating reagent) Standard (HDL - C 50mg/dl). The reagents are protected from direct light. Reagent reconstitution:
Reagent 1 End 1A are mixed gently. The reconstituted reagent is stable for 3 months when stored at 2°C to 8°C.

Procedure:

The samples, the precipitating reagent 2 and the reconstituted reagent should be brought to room temperature prior to use.
Sample and the precipitating reagent 2 are centrifuged at 1500g or 3500-4000 rpm for l0min. Separate the clear supernatant immediately and determine the cholesterol content.

	Blank	Standard	Test
Reconstituted	1ml	1ml	1ml
Standard	-	20 µl	-
Sample	-	-	20 µl

Incubate for 5 minutes at 37°C. Mix and read the colour with the standard solution.

Very Low Density Lipoprotein (VLDL) Estimation: (Fried Wald WT et al 1972)
To estimate the VLDL we should have the Triglyceride values.

Principle:
VLDL = Triglyceride/5

Low density lipoprotein (LDL) estimation:
The value of LDL Cholesterol can be calculated as follows. If the value of Triglycerides, HDL, Total cholesterol are known LDL cholesterol can be calculated based on Fried walds Equation.
LDL cholesterol mg/dl = Total cholesterol - Triglycerides/5
- HDL cholesterol
(Or)
LDL cholesterol mg/dl = Total cholesterol (HDL + VLDL)

IV. Blood Sugar Estimation

Method: Glucose oxidase peroxidase method (GODPOD)
Principle: Glucose + O2 + H2O GOD Gluconic acid + H2O2 2H2O2 + 4 Amino antipyrine + Phenol POD Quinoneimine + 4 H2O Quinoneimine is estimated colorimetrically at 540nm or green filter.
Reagents:
Reagent I - Phosphate buffer (PH 7.0) - 100mmol/l
Phenol - 5mmol/l
4 Amino antipyrine- 0.5mmol/l
Glucose Oxidase - 15 KV/1
Peroxidase- 1 KV/1
Glucose - 100mg/dl

Procedure:

	Test	Standard	Blank
Sample	10µl	-	-
Standard	-	10µl	-
Reagent	1000 µl	1000µl	1000 µl

Mixed well & Incubate for 15 minutes at room temperatue. Read absorbance of sample, Absorbant of the Standard against Blank.

Calculation:
Glucose Mgs/dl = Absorbant Sample/Absorbant Standard x Concentration of Standard (lOOmg/dl)
Nelson - Somogyi Method:
Somogyi (1945 Hoaroldvarley, Modified 1952)

Reagents:
Zinc sulphate, 5% solution of ZnSo4, 7H2O

Barium Hydroxide, 0.3N, add 15gms of Barium Hydroxide, or 28gms of Barioum Hydroxide, 8 M2O to 500 ml of hot water and boil for a few minutes. Stop X allow to cool and filter.

Alkaline Copper tatrate reagent:
Arsenomolybdate reagent (Nelson, 1944)
Standard Glucose Solution (0.1%)

Procedure **:** Place 0.5 ml of blood is placed in 1.0ml of distilled water and add 4.25ml of 0.3N barium hydroxide and 4.25ml of 5% Zinc sulfate. Shake well and filter or centrifuge. Pipette 1ml of the filtrate (0.050ml blood) for test, 1ml of water for the blank, 1ml of standard, into three 6 & ¾ inch test tubes, add 1ml of water and 2 ml of the alkaline copper solution to each and heat in a boiling water bath for 20 minutes cooled quickly for 3 minutes, add 1ml of the arsenomolybolate reagent and make up to 25 ml with water.
Calculations: mg. glucose/100ml of blood.

$$\frac{\text{Reading of unknown}}{\text{Reading of standard}} \times \frac{100}{0.05} \times 0.05$$

$$\frac{\text{Reading of unknown}}{\text{Reading of standard}} \times 100$$

To prepare a standard curve dilute 2.5, 5.0, 7.5 and 10 ml of the strong standard to 100ml with benzoic acid and put 1ml. Portions through as per the standard above.
For higher values use less supernatant or dilute the final color further.

V. Results

In this study 60 patients were included - All are Diabetes Mellitus patients ranging from 40 years to 70 years age. Total patients are grouped into 2 groups. Group A & Group B - Thirty patients in each group.
Group A: were given Glibenclamide 5 mg to l0mg range
Group B: were given Gimepiride 1 to 4mg range

Table 1: Demographic data

Sex	Group-A	Group-B
Male	26	19
Female	4	11
Total	30	30

Age Distribution	Group A	Group B
40-45	4	8
46-50	6	6
51-55	7	7
56-60	6	4
61-65	4	3
66-70	3	2

Estimation of Blood Sugar and lipid profiles (Total cholesterol,Triglycerides, HDL, LDL, VLDL)
Was done before the administration of drugs (0 Week) and after 6 weeks of treatment.
The mean values are noted.

Group A : Results Before Administation of Drug
Fasting Blood Sugar :
Mean Value of Fasting Blood Sugar- 120.7mg/dl
Standard Deviation of Blood Sugar - 28.32
Standard Error of Blood sugar - 5.17

Serum Total cholesterol :
Mean Value of Serum Cholesterol - 209mg/dl
Standard Deviation of Serum Cholesterol - 44.98
Standard Error of Serum Cholesterol- 8.224

Triglycerides :
Mean Value of Triglycerides- 210mg/dl

Standard Deviation of Triglycerides - 86.93
Standard Error of Triglycerides - 15.89

HDL :
Mean Value of HDL - 43.0 mg/dl
Standard Deviation of HDL - 9.45
Standard Error of HDL - 1.727

LDL :
Mean Value of LDL - 136 mg / dl
Standard Deviation of LDL - 41.34
Standard Error of LDL - 7.557

VLDL :
Mean Value of VLDL - 42.0 mg/dl
Standard Deviation f VLDL - 19.95
Standard Error of VLDL - 3.648

Group A- Six weeks after administration of drug.
Fasting blood sugar:
Mean Value of Fasting Blood Sugar - 100.8mg/dl
Standard Deviation of Blood Sugar - 23.08
Standard Error of Blood Sugar - 4.21

Serum Total cholesterol:
Mean Value of Serum Cholesterol - 205 mg/dl
Standard Deviation of Serum Cholesterol - 48.37
Standard Error of Serum Cholesterol - 8.842

Triglycerides:
Mean Value of Triglycerides - 205mg / dl
Standard Deviation of Triglycerides - 82.5
Standard Error of Triglycerides - 15.0012

HDL:
Mean Value of HDL - 41.0mg / dl
Standard Deviation of HDL - 7.57
Standard Error of HDL - 1.384

LDL:
Mean Value of LDL - 138mg /dl
Standard Deviation of LDL - 41.01
Standard Error of LDL - 7.314

VLDL:
Mean Value of VLDL - 41.0 mg / dl
Standard Deviation of VLDL - 17.83
Standard Error of VLDL - 3.261

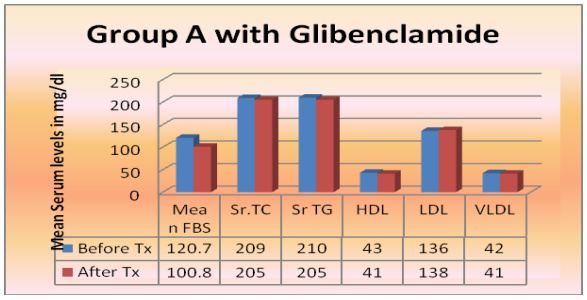

Fig no.1: Group A showing Mean Serum levels before and after treatment

Group B: Results before administation of drug

Fasting blood sugar:

Mean Value of Fasting Blood Sugar	-	132.76 mg /dl
Standard Deviation of Blood Sugar	-	46.27
Standard Error of Blood sugar	-	8.44

Serum Total cholesterol:

Mean Value of Serum Cholesterol	-	224 mg/dl
Standard Deviation of Serum Cholesterol	-	35.90
Standard Error of Serum Cholesterol	-	6.56

Triglycerides:

Mean Value of Triglycerides	-	205 mg/dl
Standard Deviation of Triglycerides	-	73.711
Standard Error of Triglycerides	-	13.47

HDL:

Mean Value of HDL	-	36.66 mg/dl
Standard Deviation of HDL	-	2.60
Standard Error of HDL	-	0.476

LDL:

Mean Value of LDL	-	160 mg / dl
Standard Deviation of LDL	-	31.70
Standard Error of LDL	-	5.79

VLDL:

Mean Value of VLDL	-	41.0 mg/dl
Standard Deviation f VLDL	-	14.54
Standard Error of VLDL	-	2.659

Group B: Results six weeks after administration of drug.

Fasting blood sugar:

Mean Value of Fasting Blood Sugar	-	100.8 mg/dl
Standard Deviation of Blood Sugar	-	17.24
Standard Error of Blood Sugar	-	3.14

Serum Total cholesterol:

Mean Value of Serum Cholesterol	-	200 mg/dl
Standard Deviation of Serum Cholesterol	-	34.7
Standard Error of Serum Cholesterol	-	6.34

Triglycerides:

Mean Value of Triglycerides	-	191.66 mg / dl
Standard Deviation of Triglycerides	-	67.49
Standard Error of Triglycerides	-	12.33

HDL:

Mean Value of HDL	-	37.16 mg / dl
Standard Deviation of HDL	-	2.867
Standard Error of HDL	-	30.52

LDL:

Mean Value of LDL	-	146 mg /dl
Standard Deviation of LDL	-	26.24
Standard Error of LDL	-	4.79

VLDL:

Mean Value of VLDL	-	38 mg / dl

Standard Deviation of VLDL - 13.94
Standard Error of VLDL - 2.548

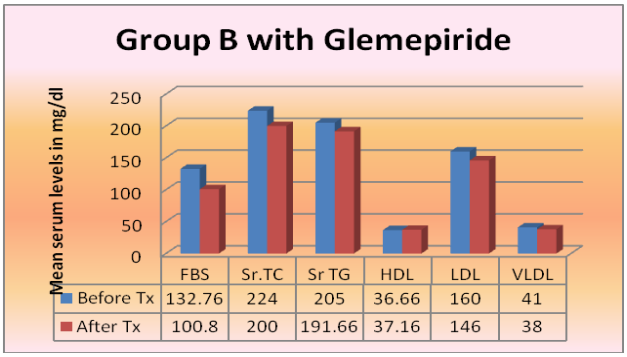

Fig no.2: Group B showing Mean serum levels before and after treatment

Table no.2: Shows the differences of Mean Values in Group A & Group B.

Group	Total Cholesterol	Triglycerides	HDL	LDL	VLDL
Group A	4mg/dl	5mg/dl	2mg/dl	2mg/dl	1.00 mg/dl
Group B	24 mg/dl	13.34 mg/dl	0.5 mg/dl	14mg/dl	3.00 mg/dl

According to the results obtained for us in Group A & Group B before & after the treatments the "t" value & "p" value were calculated and the significance of the values are observed.

Table no. 3: Showing the "t" Value & "p" Values of Group A.

Type	S.D.		S.E.		"t" Value	"p" Value
	0 Wk	6 Wk	0 Wk	6 Wk		
TC	44.98	48.37	8.224	6.842	3.999	0.00
TG	86.93	82.05	15.89	15.0012	1.026	0.313
HDL	9.45	7.57	1.727	1.384	4.055	0.000
LDL	41.34	41.01	7.557	7.314	0.336	0.740
VLDL	19.95	17.83	3.648	3.265	0.883	0.384

Table no. 4: Showing the "t" Value & "p" Values of Group B.

Type	S.D.		S.E.		"t" Value	"p" Value
	0 Wk	6 Wk	0 Wk	6 Wk		
TC	35.90	34.7	6.56	6.34	6.6541	0.00
TG	73.711	67.49	13.47	12.33	5.632	0.00
2.60	2.867	0.476	0.52	-1.897	0.068	0.000
LDL	31.70	26.24	5.79	4.79	9.928	0.00
VLDL	14.54	13.94	2.659	2.548	-2.005	0.054

VI. Discussion

The increased risk of vascular disease in diabetics is in part due to the lipid abnormalities, which are twice as common in type 2 diabetes compared to non-diabetics and are more complex than in type 1 diabetics. The most common lipid abnormality seen is hypertriglyceridemia and reduced HDL cholesterol level. An atherogenic constellation of lipid abnormalities including elevated small dense LDL, IDL and apo-B level and reduced Apo - A levels also occur commonly in type 2 diabetics. Glycated LDL can be more atherogenic than native LDL. Hence, the study of these risk factors of Diabetes Mellitus became essential following the usage of different therapeutic hypoglycemic agents.

The Framingham study has shown that a reduction in risk of coronary heart disease in the patients with diabetes depends more on improvement in ratio between LDL/HDL than on the control of hyperglycemia.

Disturbances in the structure and function of the endothelium are associated with vasoconstriction and intravascular thrombosis (Vanhoutte PM 1985) [5]. These alterations appear early in the courses of hyperlipidemia, hypertension and Diabetes Mellitus and may contribute to the pathogenesis of atherosclerosis and to its manifestations, such as Coronary Artery Disease (Bassenge et al 1990) [6].

In this study, the levels of lipid profiles are decreased after therapy.

In this study Group "A" with Glibenclamide. There is a significant fall of FBS, TC, TG &

HDL.

FBS: The mean fall is 19.9↓,

TC: The mean fall is 4↓,
TG: The mean fall is 5↓,
HDL: The mean fall is 2↓,
LDL: The mean rise is 2↑ &
VLDL: The mean fall is 1↓
In this study Group "B" with Glimepiride. There is a significant fall of FBS, TC, TG, LDL, VLDL and rise in HDL
FBS: The mean fall of BS is 21.9↓,
TC: The mean fall is 24↓,
TG: The mean fall is 13.34↓,
HDL: The mean rise is 0.5↑,
LDL: The mean fall is 14↓
VLDL: The mean fall is 3↓

Besides obviously there is hypoglycemic result is seen with both the drugs. Further, more interesting inference is associated on comparison of the mean fall of each parameter of each individual drug is highly significant.

(Tsunekawa et al 2003) [7] in their study showed that BMI and plasma lipid profile, TC, TG, HDL improved by Glimepiride treatment probably, the mechanism for improvement may be related to decrease in plasma TNF α levels & increase in plasma adiponectin which is a specific plasma Glycoprotein which has anti atherogenic, anti-inflammatory and apoptotic effects.

(Nghyen C et al) [8]in their studies shows Glimepiride possibly has anti atherogenic activity by inhibiting platelet aggregation via suppression of arachidonic acid metabolism.

Most of the antidiabetic drugs besides their hypoglycemic effect, they are having to some extent the antiatherogenic influence by bringing fluctuations in the lipid profile features, but this effect is variable. In case of Glimepiride comparatively influence on the lipid profile is asignificant feature associated with hypoglycemic effect, where as Glibenclamide is not so significant.Both hyper cholesterolemia, hypertriglyceridemia are high risk factors for atherosclerosis. A longold established theory suggested that the higher the circulating levels of lipoprotein the more likely they enter into arterial wall. Chemically modified or oxidized lipoprotein produced in hyperlipidemic disorders like Diabetes Mellitus, enter into the scavenger arterial wall macrophages leading to formation of foam cells. Oxidized LDL promote the following changes. Chemotactic activity of monocytes, facilitate the recruitment of circulating monocytes, inhibition of migration of macrophages within artery back to plasma compartment, increased uptake of LDL by macrophages through acetyl receptor leading to generation of foam cells and Atheroma formation.

A lowering effect of TC, TG, LDL, VLDL is favouring cardioprotective measure against atherogenesis. Besides this, more important is slight rise of HDL also accounts for cardioprotection. A low level of HDL is unfavourable feature for cardioprotection. These parameter's mean values are represented in the respective tabular forms and evaluated.

In case of Glimepiride therapy the mean values and the fall of lipid profile levels, on comparison, supporting the significant inference towards antiathero sclerotic change, which is a cardioprotective measure.After considering the above facts to arrive a conclusion, the inferences of the Glibenclamide and Glimepiride are compared and evaluated for significance.

Finally Glimepiride is found to have a significant cardioprotective inference in relation to lipid profile features comparatively with Glibenclamide. A reasonable rational use of the drugs for antilipidemic purpose is always necessary, to prevent cardiovascular risk factor. It appears that the glimepiride is not a competent substitute for antilipidemic agents, but in Diabetes Mellitus patients from the beginning of the glycemic control, the glimepiride itself precludes the onset of the hyperlipidemia measures comparatively as a cardioprotection.

Particularly selection of Glimepiride drug comparatively is a measure to preclude the hyperlipidemic complications, exclusively in Diabetes Mellitus from the onset of Diabetes Mellitus therapy. Both these two drugs are equivocally used for hypoglycemic effect as usual. But furthermore, in this study there is a profound inference for the selectivity of Glimepiride comparatively for an extension of Cardioprotective measure.

VII. Conclusion

Sixty patients of Diabetes Mellitus were observed for lipid profile fluctuations in relation to Glibenclamide and Glimepiride comparatively.

The changes in the lipoprotein levels noted before and after 6 weeks of drugs administration. After the study of therapy 8& investigations, it is understood that the patients who were treated with Glimepiride (Group B), there is a significant fall in the levels of total cholesterol, Triglycerides & LDL levels comparatively. And

there is slight raise, in the levels of HDL, but not significantly.

Where as in patients who were treated with glibenclamide there is only slight fall in levels of Triglycerides, Total cholesterol and LDL levels comparatively.

This study gives the scope and inference to select a cardioprotective antidiabetic agent to avoid the increased mortality rate among Diabetes Mellitus patients.

References

[1]. K.D.Tripathi ,Accesentials of Medical Pharmacology,7th Edition 2013
[2]. Allian C.A. et al, Clinical Chemistry - 20;490(1974)
[3]. Buccolo G. David M. - Clinical Chemistry - 19(1973,476) Castelli WP et al, Lancet 1, 1965(1977
[4]. Castelli WP, Garrison RJ, Wilson PWF, et al. - Incidence of Coronary Heart Disease and lipoprotein cholesterol levels. The Framingham Study. - JAMA, 256:2835-8, 1986.
[5]. Van houtte, DM, Houstan D.S. Platelets, endothelium, and vasospasm. Circular 1985:72:728-34
[6]. Basseng E, Heusch G. Endothelial and neuro humoral control of Coronary Blood flow in health and disease Rev.PhysioL Biochem. Pharmacol.
[7]. Tsunekawa et al 2003. - Diabetes Care 26:285-289 No.2, Vol.26, Feb,2Q03 - Plasma Adiponectin plays an important role in improving Insulin resistance with Glimepiride in elderly Type Diabetes Mellitus Subjects.
[8]. Nguyan C: Pan J, Charles MA, - Drugs today (Bare), 1998 May: 34(5):391-400.