Original Article

"Association Between Glycaemic Control And Lipid Profile In Type 2Diabetes Mellitus"

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Abstract

Introduction

Diabetes mellitus (DM) is the most common metabolic disorder affecting the people worldwide. Even though diabetes has been known since antiquity, onlyin the last few decades new discoveries have provided great hopes to minimize morbidity and mortality. It is estimated that for one diagnosed diabetes there is undetected diabetes. Diabetic ketoacidosis, a major fatal complication of diabetes has virtually come down with advent of insulin. However, the vascular complications have remained the same and they have replaced diabetic ketoacidosis as the frequent cause death in diabetes.

The main underlying cause of DM is the defective production or the action of insulin, the hormone which controls glucose, fat and amino acid metabolism inthe human body. Characteristically, the diabetes is a chronic disease and with variable clinical manifestation. Chronic hyperglycaemia leads to a number of complications like cardiovascular, renal, infections etc. Diabetes mellitus is broadly classified as follows

i) Type 1 or Insulin Dependent Diabetes Mellitus

ii) Type 2 or Insulin Independent Diabetes Mellitus

iii) Malnutrition related diabetes mellitus (MRDM)

iv) Other types (Secondary to pancreatic, drugs, hormonal)

Diabetes is –Iceberg‖ disease. Type 2 diabetes is much more common than type 1 diabetes. It is discovered by chance.[4]

Aim and Objectives

Aim: To study the link between glycacrmic control and serum lipid profile in type-2 diabetic mellitus

Objective

To measure lipid profile and HbA1c level in patients of type 2 diabetes mellitus

Review of Literature

Definition of Diabetes Mellitus

According to the recommendation report of W.H.O. in 2006 following are thecriteria to diagnose diabetes mellitus and intermediate hyperglycemia

Diabetes

Fasting plasma glucose ≥7.0mmol/l (126mg/dl)or
2–h plasma glucose ≥11.1mmol/l (200mg/dl)

Impaired Glucose Tolerance (IGT)

Fasting plasma glucose <7.0mmol/l (126mg/dl)and
2–h plasma glucose ≥7.8 and <11.1mmol/l (140mg/dl and 200mg/dl)

Impaired Fasting Glucose (IFG)

Fasting plasma glucose: 6.1 to 6.9mmol/l (110mg/dl to 125mg/dl)2–h plasma glucose (if measured): <7.8mmol/l (140mg/dl) [8].

HISTORY:

The first ever documented evidence of DM came from a discovery made by German archaeologist named George Ebers which was papyrus in Egypt in 19th century and ever since then its known as the Ebers Papyrus in hishonour.

DM in ancient India

In ancient India DM was known as prameha [pra –excess, meha –urine], a term use to refer to the disease even today in certain Indian languages.

DM is not a new disease, the charaka samhita, dating back to 1500 bc, describes prameha in great detail. It recognizes 20 types of prameha which if not treated can lead to madhumeha [madhu- honey, meha- urine, literally sweet urine, an unambiguous description of DM.

It is accepted generally that the term DM literally means ‟siphon‘, in reference to the polyuria characteristic of uncontrolled disease. The word ‟siphon‘ means sucking water out of the body through the urine. [15]

History timeline [46]

APPROXIMATELY 1550BC

An Egyptian papyrus mentions a rare disease that causes the patient to lose weight rapidly and urinate frequently. This is thought to be the first reference to DM.

- **250 BC**

The creation of the term ―DM‖ is credited to Apollonius of Memphis, which refers to a disease which drains patients of more fluid than they can consume.

- **131-201 CE**

A Greek physician, Galen of Pergamum, theorises that DM is an affliction of the kidneys. After this period, DM is rarely mentioned.

- **1025**

A Persian polymath called Avicenna (980-1037) publishes ―The Canon of Medicine‖ in 1025, providing a detailed account on DM. The sweet urine of people with DM is described, as is abnormal appetite, diabetic gangrene and sexual dysfunction.

- **UP TO 11th CENTURY**

Around this period, ‗uroscopy' becomes a way of identifying disease, which involves examining the colour, sediment and odour of urine to try to establish what is wrong with the patient. Some physicians even taste the urine, and this is apparently how DM is given its second name, mellitus, meaning ‗honey' in Latin.

- **1770-1800**

Matthew Dobson identifies that the sweet taste in the urine of people with DM is due to excess sugar in the urine and the blood. He also observes that DM isfatal for some, leading to death within five weeks, while others live much longer. This is the first indication of two different types of DM: type 1 and type 2.

- **1800S**

Claude Bernard coins the term ―glycogen‖ after discovering a substance formed by the liver that he reports is the same sugar found in the urine of those with DM. This is the first link between DM, glycogen and metabolism.

- **1919**

Dr Frederick Allen publishes a book, ―Total Dietary Restriction in the Treatment of Diabetes‖, which reveals case records of diabetes patients treated with the ‗starvation diet'. The treatment helps extend the lives of diabetes patients, but many of his patients die as a result of starvation.

- **1920S**

It is not until 1920 that an American called Moses Barron links the Langerhanscells with the basis of DM. Picking up on the research of Barron, a doctor called Frederick Banting conducts critical experiments linking the pancreas and diabetes. Banting and Charles Best demonstrate that administering the islets of Langerhans from healthy dogs to dogs which have had their pancreases removed can treat high sugar levels.

James Collip purifies this insulin and it is treated for the first time on a human, 14-year-old Leonard Thompson. The work is considered a great success. The average life expectancy for a child with type 1 diabetes at the beginning of the 20th Century is roughly a year; Leonard lives until the age of 27, when he eventually dies of pneumonia.

- **1955**

The first oral drug is developed, carbutamide, to help lower blood glucose levels.

- **1960S**

Urine strips are made available for home testing helping people with diabetes get faster readings.

Blood testing technology also makes advances. Miles Laboratories releases Dextrostix testing strips which require a drop of blood for a minute. The blood is then washed off and an indication of blood sugar levels is revealed on a colour chart.

Doctors at the University of Minnesota attempt the first pancreas transplantation in an attempt to cure type 1 diabetes.

- **1970S**

The first insulin pump is invented by Dean Kamen. Another insulin breakthrough is made in the form of U-100 insulin.

The HbA1c test is introduced into clinical laboratories for the first time in 1977.

The first synthetic human insulin is produced using recombinant DNA techniques. Prior to this development insulin manufacturers have had to stockpile pancreatic tissue from animals.

- **1996**

The first recombinant DNA human analogue insulin is marketed by Eli Lilly. Humalog (lispro) is genetically engineered insulin which contains an amino acid sequence and alters how insulin is absorbed.

- **1997**

Dr Richard Bernstein publishes ‗Diabetes Solution' which addresses lowering carbohydrate intake as a means of achieving good blood glucose control and avoiding diabetes-related complications.

- **2000**

There is growing interest in islet cell transplantation as Shapiro et al publish findings from seven patients with type 1 diabetes who undergo the procedure as a means of helping them achieve insulin independence.

- **2008**

Suzanna M. de la Monte proposes the term ―type 3 diabetes‖ to describeinsulin resistance in the brain.

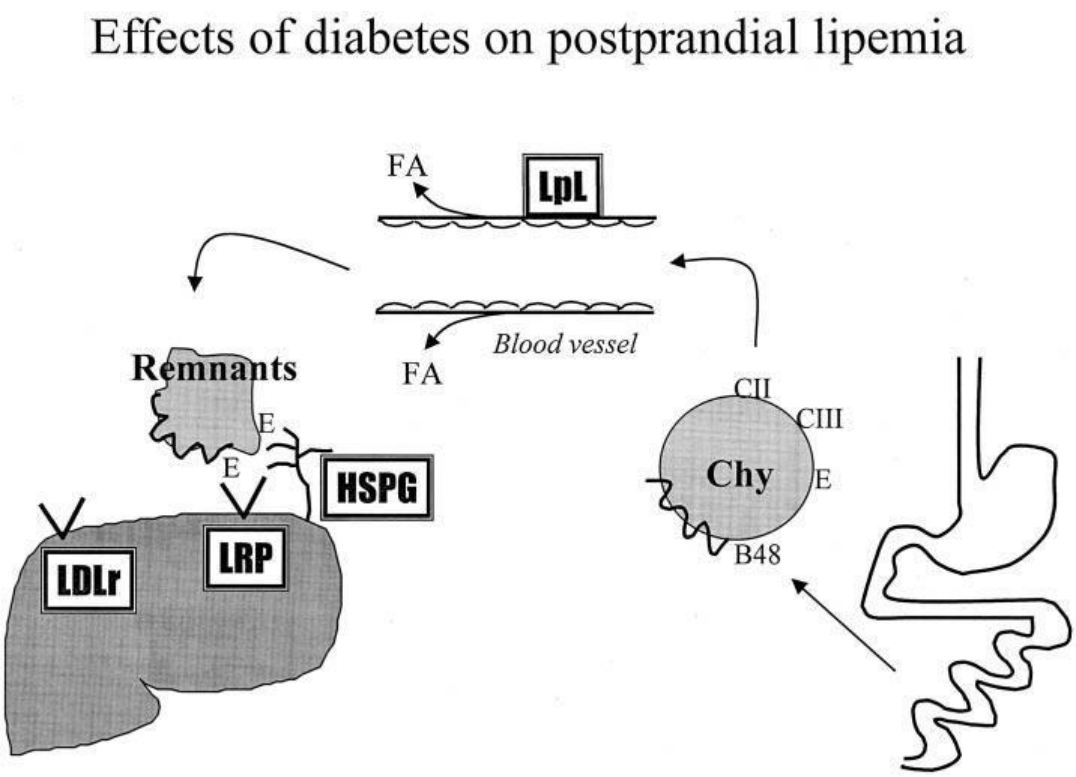
- **2015**

Dr Edward Damiano introduces the iLet, a bionic pancreas that delivers bothinsulin and glucagon every five minutes. Damiano describes the device as a
―bridge to a cure‖.

Effect of Diabetes on Post Prandial Lipemia

Effects of diabetes on postprandial lipemia. A defect in removal of lipids from the bloodstream after a meal is common in patients with diabetes. Chylomicron metabolism requires that these lipoproteins

obtain apo-CII after they enter the bloodstream from the thoracic duct. Triglyceride within the particles can then be hydrolyzed by LpL, which is found on the wall of capillaries. LpL activity is regulated by insulin, and its actions are decreased indiabetes. Triglyceride-depleted remnant lipoproteins are primarily degraded in the liver. This requires them to be trapped by liver heparan sulphate proteoglycans (HSPG) and then internalized by lipoprotein receptors, LDL receptor and LRP. Because remnants contain a truncated form of apoB, apoB48, that does not interact with these receptors, this uptake is mediated by apoE.[13]



Although it is clear, that diabetic patients have an elevated risk for atherosclerosis and although dyslipidemia seems to be an important link it is unclear which of the aforementioned abnormalities actually causes vascular damage. An increased number of apoB containing lipoproteins (either asVLDL or as remnants or as LDL, particularly small dense LDL) seem to be crucial. There is an enhanced interaction of small dense LDL with the scavenger receptor, which promotes foam cell formation and atherogenesis.[10]

Pathogenesis of Type 2 DM:

Genetic factors:

There are genetic factors in the development of insulin resistance, impaired insulin secretion, and type 2 diabetes has been known for many years.[47] There are supporting evidence which includes familial clustering of thesetraits, the higher concordance rate of type 2 diabetes in monozygotic versus dizygotic twins, and the high prevalence of type2 diabetes in certain ethnic groups. 25% and 70% of the occurrence of type 2 Diabetes is attributed to genetic factors.[9]

Beta cell dysfunction:

The distinctive β-cell defect in type 2 diabetes is the loss of the first phase of glucose-induced insulin secretion. The second phase is also impaired but to a lesser degree. Further investigation showed that this defect was fully established by the time the fasting glucose level reached 115mg/dL, but the finding was absent in persons with truly normal glucose levels in whom type 2 diabetes developed later. Thus, it first appears in the pre-diabetes state, termed impaired glucose tolerance [IGT], which is clinically manifested as excess postprandial excursions of glycaemia.[9]

Insulin resistance:

The term insulin resistance usually refers to resistance which occurs to the metabolic effects of insulin, this includes the suppressive effects of insulin on endogenous glucose production, the stimulatory effects of insulin on peripheral (predominantly skeletal muscle) glucose uptake and glycogen synthesis, and the inhibitory effects of insulin on adipose tissue causing lipolysis. Insulin resistance plays a major role in the development of type 2 diabetes as there is an estimated 170 million people worldwide having Type 2 Diabetes, and there is a association seen. There could be acquired factors which play a role like:

1. Beta cell cytotoxic chemical / virus.
2. Autoimmunity.
3. Ageing,
4. Obesity.
5. TNF alpha, leptin.
6. Physical inactivity,
7. High glycemic diet.

Complications of DM:

- Microvascular
- Macrovascular complications.

There could be acute complications too, like Diabetic ketoacidosis (DKA) and hyperglycaemic hyperosmolar state (HHS). DKA and HHS exist along a continuum of hyperglycaemia, with or without ketosis.[10]

Chronic complications can be further divided into

- Vascular
- Non vascular complications.

The vascular complications of DM are further subdivided into

- microvascular which includes
    - Retinopathy

- Neuropathy
- Nephropathy

- Macrovascular which includes complications like
  - Coronary Artery Disease(CAD),
  - Peripheral Arterial Disease (PAD),
  - Cerebrovascular Disease.

Nonvascular complications include problems such as gastroparesis, infections, and skin changes and dyslipidemia.[10]

A cross sectional study conducted by Aclan Ozder to assess the lipid profile abnormality in type 2 DM patients at primary healthcare in Turkey showed thatof the total number of patients, 61.4% (81) were females, and 38.6% (51) of the patients were males. The age range and mean age of female patients was 37–75 years and 55.5 ± 9.2 while the age range and mean age for male patients was 39–84 years and 54.8 ± 12.6 years, respectively. The mean BMI of the participants was 28.59 ± 3.25 kg/m2 (range: 26.10-44.98). Eleven outof 132 patients were obese in this study with BMI ranging from 31.02 to 44.98.Results of the blood glucose profile showed that all individuals were hyperglycaemic. The mean fasting blood sugar in female was 196.7 ± 61.2 and in male it was 217.1 ± 57.5. While, HbA1c in female was 8.46 ± 1.49 and in male was found to be 8.84 ± 1.53 indicating poor glycemic control. Mean value of the TG/HDL ratio which is considered a surrogate marker of insulin resistance was 7.98 ± 3.87 among entire group. In correlation studies, TG/HDL ratio showed significant positive correlation with HbA1c ($p < 0.01$). Among all patients, hypercholesterolemia was found in 93 (70.5%) individuals.

Similarly, increased LDL cholesterol was found in 117 (88.6%) individuals. Of the participants only 39 (29.5%) were found to have normal values for TG. Grade I and II hepatosteatosis were found in nearly one third of diabetics and Grade III hepatosteatosis in 63 (47.7%) patients while Grade IV hepatosteatosis was found in 27 (20.5%) of the study group by hepatic ultrasound. In correlation studies, FBG showed significant positive correlation with cholesterol ($p < 0.05$) and TG ($p < 0.05$). The correlation of cholesterol with TG ($p < 0.001$) and LDL cholesterol ($p < 0.001$) were positive. However, HDL cholesterol showed a negative correlation with LDL cholesterol ($p < 0.01$).[16]

The study ―Correlation of Dyslipidemia and Type 2 Diabetes Mellitus Amongstthe People of Vidarbha Region of India.‖ By Ajay Meshram and et al was case control study comprising of 50 non-diabetic control and 50 type 2 Diabetic cases. The mean age of T2DM patients study group and the corresponding control group was 53.8 ± 7.11 (years) and 52.8 ± 7.83 (years) respectively; whereas, the percentage of gender in studied population showed that control group consisted of 60% male and 40% female while, among T2DM patients 52% are male and 48% are female. Hence, the results suggest that the study comprised of equal distribution of age group and gender. The FBS and serum LDL cholesterol levels were significantly elevated in the study group 97.99 ± 12.05 and 121.79 ± 44.33 respectively with respect to the control group 94.3 ± 19.5 and 120.4 ± 30.57 respectively, which implies a strong significance at $p<0.002$ and $p<0.001$ respectively. Whereas, HDL cholesterol levels are found to be lower in patients 35.5 ± 9.27 than the control group 45.0 ± 4.10, which also shows a highly significance at $p<0.001$.[17]

A review by Chitra R. Hinge et al on body mass index, blood pressure and lipid profile in type 2 diabetes showed that CVD is the most prevalent complication of DM. The age adjusted cardiovascular mortality

is at least 2- fold higher in diabetic men than in non-diabetic patients in the presence of many numbers of major risk factors. The classical dyslipidemia in type 2 DMis so called atherogenic dyslipidemia. Majority of Indian type 2 DM are dyslipidemic at baseline. The most common pattern of dyslipidemia is high LDL-C and low HDL-C among both males and females contributing to 22.7% and 33% patients of diabetic dyslipidemia, respectively. The most prevalent problem among males is high LDL–C while among females low HDL –C emerged as a bigger threat.[20]

The study conducted by Dharmesh N. Gamit and Avanish Mishra showed that Out of total 140 diabetic patients examined, the mean age of patients was
48.93 ± 12.1 years. In present study we found the mean Fasting Blood Sugar
(FBS) was 188.76 ± 54.63 mg/dl. The prevalence rates in our study for high Total Cholesterol (TC) and Triglycerides (TG) were 13.6% and 41.4% respectively. The prevalence rates for high LDL-C, very high LDL-C and low HDL-C in the diabetic patients were 8.6%, 5.0% and 72.9% respectively.[21] While study conducted by Emina Panjeta et al showed that Median glucose concentration in the serum of patients with good glycemic control was 6.99 ±
1.65 mmol/L, and for the group of patients with poor glycemic control, it was

13.63 ± 4.48 mmol/L. Median serum triglyceride concentrations in the group ofpatients with good glycemic control were 1.48 (1.15–2.22) mmol/L and inpatients with poor glycemic control 2.15 (1.4–3.32) mmol/L. Patients with good glycemic control had significantly lower median serum triglyceride concentrations when compared to a group of patients with poor glycemic control (p = 0.03). There was a significant positive correlation between the concentration of glycated hemoglobin with serum glucose concentrations (r = 0.560) in patients with type 2 diabetes mellitus (Figure 1). Furthermore, thereis a significant positive correlation between the concentration of glycated hemoglobin and serum triglyceride concentrations (r = 0.375).[22]

The study conducted by Hetal Pandya et al showed that the mean duration from the first diagnosis of diabetes for the study patients was 5.1 ± 4.64 years.Serum cholesterol level was 188.9 ± 43.70, mean serum triglyceride was
174.6 ± 69.44, mean serum HDL was 46.2 ± 17.08, mean serum LDL was

105.9 ± 34.06 and mean serum VLDL level was 33.4 ± 11.08. Out of 171 DM patients, 36.3% (n = 62) patients were having high serum cholesterol level, while almost similar no. of patients, 35.7% (n = 61) had low serum HDL levels.About 56.1% (n = 96) had high serum triglyceride level, while almost similar number of patients, 57.3% (n = 98) also had serum LDL levels above normal range. About 49.7% (n = 85) also showed high serum VLDL levels. Only 19.3% of study patients were well-controlled.[25] A cross sectional study conducted by JK Baranwal et.al. involving 168 type 2 diabetic patients showed that the correlation of long term glycemic control represented byHbA1c has been found out with parameters of lipid profile viz. TC, HDL-C, LDL-C and TG. Out of 168 patients, there were 91 females and 77 males. The means ± SD of age, TC, HDL-C, LDL-C, HDL/LDL ratio and HbA1c of patientswere 52.2 ± 11.9 years, 182.9 ± 41.9 mg/dl, 41.6 ± 8 mg/dl, and 94.9 ± 20 mg/dl, 0.47 ± 0.18 and 6.5 ± 1.5 % respectively.[26]

Kay-Tee Khaw et al. conducted study in Norflok showed that those with known diabetes had higher mean (±SD) hemoglobin A1c concentrations (8.0% ± 1.9%) than the rest of the study sample (5.3% ± 0.7%). They were older and had a higher body mass index, waist-to-hip ratio, and systolic blood pressure; they were also more likely to report having had a previous heart attack or stroke. The study also concludes that persons with known or undiagnosed diabetes had a greater risk for all-cause mortality and cardiovascular or coronary heart disease than those without diabetes. Risk for coronary heart or cardiovascular disease and total mortality increased throughout the whole range of hemoglobin A1c concentrations; those with hemoglobin A1c concentrations less than 5% had the lowest rates. In men, known diabetes predicted coronary heart and cardiovascular disease events and total mortality with approximate 2-fold relative risks. While, in women, known diabetes status predicted an approximate 5-fold increase in risk for coronary heart and 3-fold increase in risk for cardiovascular disease events. Also, in men and women, hemoglobin A1c concentrations predicted and increased risk for coronary heart and

cardiovascular disease events and total mortality.[7]

A case control study was conducted by Mehnaz Khattak et.al. to assess the correlation between plasma glucose level and lipid profile in Type 2 DMpatients. About 60 individuals were placed in each group. Group A comprised of Type 2 diabetics and group B were healthy controls with no major illness. FPG levels (mean ± SD) in group A was 11.23±3.65 as compared to group B

4.35±0.68, HbA1c was (6.84±0.482 group A vs 5.31±0.487 group B) and inthe lipid profile, serum total cholesterol was (4.68±0.96 group A vs 3.99±1.01 group B p< 0.001), triglycerides (TG) were (2.42±1.22 group A vs 1.56±0.87 group B p< 0.001) and LDL cholesterol was (2.46±0.77 group A vs 2.17±0.72 group B p< 0.05). Whereas HDL cholesterol was (1.04±0.224 group A vs 1.21±0.222 group B p< 0.001). The BMI was raised significantly in group A than group B (28.57±1.97 vs 24.46±2.32 p< 0.001). It showed significant increase in levels of BMI,

FBG, HbA1c, total cholesterol and TG in the diabetic females as compared to controls while HDL cholesterol was significantly lower in the diabetic group. In females, LDL cholesterol showed no significance may be due the small sample size. While, there was significant increase in levels of BMI, FBG, HbA1c, Total cholesterol, TG and LDL cholesterol in diabetic males as compared to controls while HDL cholesterol was lower in the diabetic male.[29]

R. Yasmin A. Majeed A. Rashid, et al conducted study to find the risk of dyslipidaemia in diabetic patients of different age groups. In this study we came to know that of the 40 patients, 17(42.5%) were men and 23(57.5%) women. The blood glucose control of younger age group (30-40 years) was 234±90.37mg/dl compared to older age group (60-70 years) with a mean of 173.4±54.2 mg/dl. Serum triglyceride levels of the youngest age group (30-40 years) were the highest with a mean value of 216±137 mg/dl. There was a significant association between age and triglyceride levels (p=0.03), while the serum high-density lipoprotein level of this group was lowest with a mean of 29.6±13.4 mg/dl.[33] Also, study conducted by Satish Biradar showed that all the biochemical parameters were significantly raised in IGT and T2DM patients as compared to controls. In T2DM, FBG showed significant positive correlation with TC (p=0.048) and significant negative correlation with HDL (p=0.000). PPBG and HbA1c showed significant positive correlation with TGL,TC VLDL and LDL and significant negative correlation with HDL, p value was

for all parameters. The correlation in IGT, FBG showed significantpositive correlation with TC (p=0.000) and LDL (p=0.004), significant negative correlation with HDL (p=0.000). PPBG showed significant positive correlation with TGL, TC and VLDL and significant negative correlation with HDL (p=0.000).[34]

Thapa Subarna Dhoj et al. in the study of Dyslipidemia in type 2 diabetes mellitus showed that out of 199 patients with diabetes mellitus 30.7% had totalcholesterol >200 mg/dl, 64.4% had elevated low-density lipoprotein, 53.77% patient had elevated triglyceride and 64% patients had low high-density lipoprotein level. Cholesterol showed significant correlation with triglyceride (P < 0.001), low density lipoprotein (p < 0.001). Triglyceride showed a significant negative correlation with high density lipoprotein (p < 0.01), while a highly significant positive correlation was observed with cholesterol and high density lipoprotein (p < 0.001).[38] The study by Ullasini Kolhar and Priyanka P. stated that the prevalence of dyslipidaemia in DM patients in our study was 90% and there was no statistically significant difference in the prevalence among males and females. Poorly controlled diabetics had high prevalence of dyslipidaemia as compared to well controlled diabetics. The prevalence of Diabetic nephropathy in our study was 41%. There was significant association of DM with high Total Cholesterol, high Low-density lipoprotein (LDL-C) and high Triglycerides. There was no significant association of DN with High density lipoprotein (HDL-C).[39]

Vasant Devkar et. al studied correlation between HbA1cand dyslipidemia in patients with type 2 diabetes mellitus and found that a total of 100 patients with Type 2 diabetes mellitus were followed (52 males and 48 females). The mean age was 62.91 years with age range of 30-85 years. Poor glycemic control (HbA1c>8) was seen in 62% of total patients. Poor glycemic control was associated with dyslipidemia

in 41.5% of total patients, whereas 20.5% accounted for poor glycemic control without dyslipidemia, the maximum frequency of abnormal lipid profile status in all patients was low HDL cholesterol (HDL-C) and the age group with maximum patients with bothdyslipidemia and higher HbA1c levels was 51-60 years.[40]

Yaru Li et al. in their study found that the prevalence of dyslipidemia was 39.9%, 46.8%, and 59.3% in participants with normal glucose, prediabetes, and type 2 diabetes mellitus (T2DM). Women had lower dyslipidemia prevalence than men (38.7% vs. 43.3%). Dyslipidemia prevalence was positively associated with the education level and inversely correlated with the physical activity level, and negatively related to age only among prediabetes and T2DM groups (P for trend < 0.05). Obesity, abdominal obesity, and hypertension were associated with dyslipidemia.[41]

Diabetes mellitus (DM) is a non-communicable disease which has worldwide distribution and it varies in different populations. DM is increasing rapidly which is already an epidemic in India. The first edition of the IDF Diabetes Atlas, which was released in 2000, estimated global DM prevalence to be 151 million. Since then, there is a constant rise up to 285 million, representing 6.4% of the world's adult population, with a prediction that by 2030 the number of people with DM will reach 438 million. [49 -50]

DM is a disease of higher socioeconomic group and is very much a disease associated with poverty, which is a burden to the low and middle income countries and disproportionately affecting the lower socio-economic groups.[51] Estimated 40 million people had DM in India in 2007 and the predicted number by 2025 is around 70 million people. [52]

DM is of 2 types Type1 and Type 2. Type 2 DM is most common form of DM and characterized by a combination of peripheral insulin resistance and inadequate insulin secretion by pancreatic beta cells. The risk factors for developing Type 2 DM are increasing age, obesity, and physical activity. There is also a strong familial aggregation, so that persons with a parent or sibling with the disease are at increased risk, as are individuals with obesity, hypertension, or dyslipidemia and women with a history of gestational DM. Even though the disease is commonly seen in adults, the age of onset can be earlier too. The disease can occur at any age and is now seen in children andadolescents. [53]

The International Federation of DM, reported that 415 million adults around the world are suffering from DM, the numbers will reach around 642 million by 2040.[57] The World Health Organization (WHO) global report on DM shows that the number of adults living with DM has quadrupled since 1980 to 422 million adults.[58] India leads the World and stands at the second position after China, with 69 million persons affected by DM. This is a challenge to the sustainable development of the nation as every tenth adult (9.3%) in India is estimated to be affected by DM. [IDF] The WHO estimated death of every 26 per 100,000 persons due to DM in India.[59] The international DM federation in its reported 425 million people who have DM in world and among these 82 million are present in South East Asian region. India being one of the countries of SEA region contributes to 72,946 cases of DM. The prevalence ofinsulin-dependent DM (IDDM) or type 1 DM is 10-15% of the diabetic population. [57]

The cause of this epidemic is the rapid epidemiological transition associated with dietary patterns which have changed and decreased physical activity as evident from the increased DM cases in the urban population.[60] A study by **Liu S et al**, has showed positive association of low-fibre and high glycemic index diet with a higher risk of type 2 DM (DM).[61]

There are studies which suggest two-third of all Type 2 and almost all Type 1 diabetics are expected to develop dyslipidemia and diabetic retinopathy (DR) over a period of time. Up to 21% of patients with type 2 DM have retinopathyat the first diagnosis of DM and most develop many complications along with dyslipidemia and some degree of retinopathy over time. DM is one of the major causes of preventable blindness and cardiac complications due to dyslipidemia in both the developing and the developed countries.[60]

MATERIALS AND METHODS

Study area and setting:

The study was conducted at Krishna Hospital and Medical Research Centre,a tertiary care hospital and teaching institute in Maharashtra.

Source of Data: The study was carried on patients with diagnosis of type 2 Diabetes mellitus who were admitted in wards and attending Out PatientDepartment over the period of October 2017 to March 2019 in Krishna Institute of Medical Sciences, Karad.

Study design:

A cross sectional study of type 2 diabetic patients.

Study period:

The present study was conducted for 18 months, from October 2017 to March 2019.

Sample size:

According to a study conducted by Nyasatu G. Chamba et al. the prevalenceof dyslipidemia in type 2 diabetes patients was 83 %. So

P = 83 %

Q = 1-p = 1-0.83 = 0.17

D = absolute error = 10% = 0.1

$$N = \frac{4 \times 0.83 \times 0.17}{0.1 \times 0.1}$$

= 56 64. N ~ 60

For statistical reasons and better yield, sample size taken as 100. N =100

SELECTION OF PARTICIPANTS

Type 2 diabetic patients attending the Out Patient Department, Indoor patients, patients admitted in Intensive Care Unit at KIMSDU, Karad were selected for the study.

Inclusion Criteria:

All the patients who were willing to participate in the study, provided they were

- Patients above 18 years of age

- Patients diagnosed as Type 2 Diabetes Mellitus

Exclusion Criteria:

- Newly diagnosed diabetic patients who are not on treatment.
- Non diabetics.
- Patients diagnosed as Type 1 diabetes mellitus.
- Patients with hepatic, renal, endocrine disorders.
- Patients who are on lipid lowering agents.
- Patients who are not willing to participate in the study.

Informed consent:

Patients presenting with type 2 diabetes mellitus were screened for the eligibility. The patients fulfilling the selection criteria were explained about the nature as well as purpose of the study in English and / or local language they understand. A written informed consent was obtained from those who were willing to participate in the study.

Ethical clearance:

The study also obtained the permission of the institutional ethics committee (IEC). Permission was also taken from head of departments.

Data Collection:

The study used a pre-designed and pre-tested proforma. Patients were interviewed to obtain their demographic characteristics, personal details, age, gender, presenting chief complains, diabetic history, history of other co- morbidities, addictions. General and systemic examination of the patients was done and the findings were noted.

Statistical analysis:

The data collected was entered in Microsoft Excel sheet.

The results were expressed as Mean ± SD (Standard Deviation) and which compares population. Appropriate statistical tests were applied and SPSS statistical package trial version 20 was used for data analysis. Chi Square testwas used for association and student‘s t test was used for correlation. Statistically significant level was put at P <0.05 unless otherwise stated.

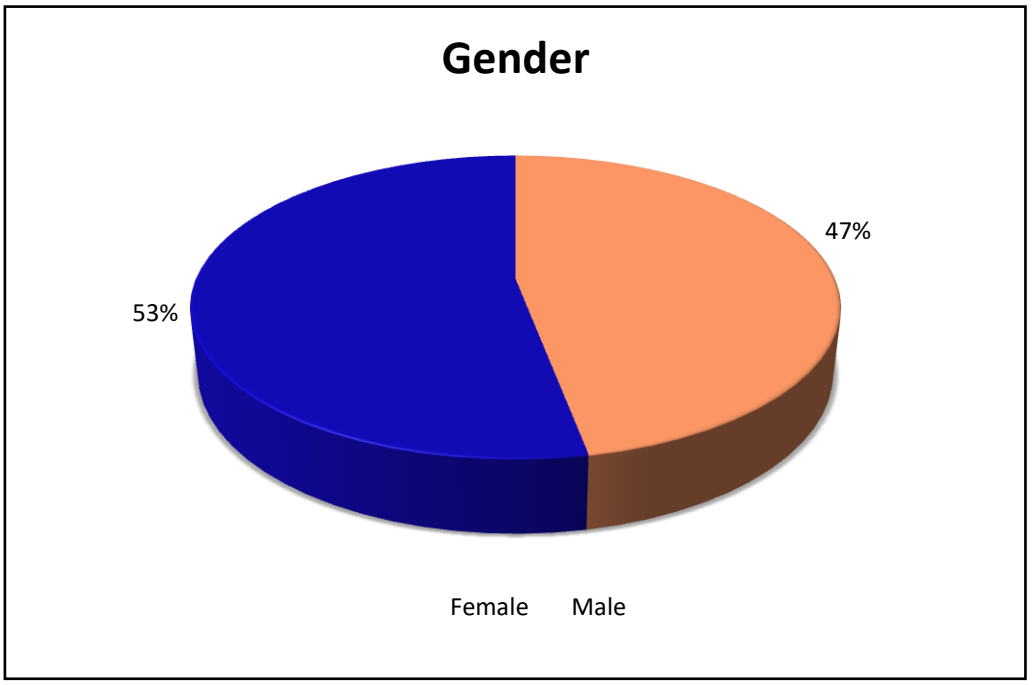
OBSERVATION AND RESULTS

There were 53 males (53%) and 47 females (47%) in our study. (**Table 1,**

Figure 1)

Table 1: Gender-wise distribution of patients		
	Total number (n = 100)	**Percent (%)**
Male	**53**	**53**
Female	**47**	**47**
Total	**100**	**100**

Figure 1: Gender-wise distribution of patients

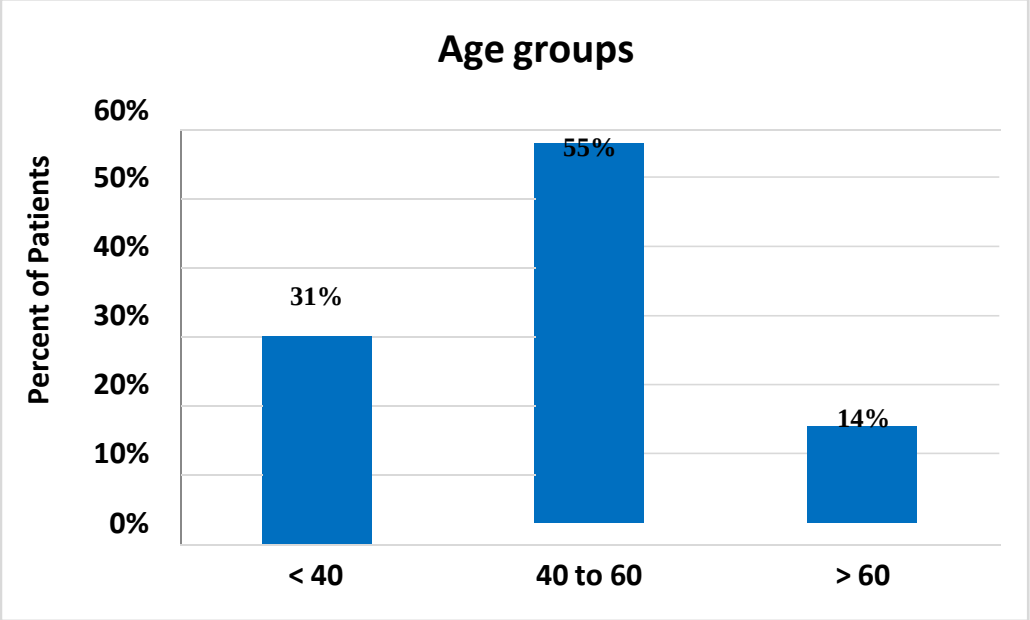


In current study, most common age group was 40-60 years with 55 patients (55%). This was followed by 31 patients (31%) under the age group of <40 years.

There were 14 patients (14%) from the age group more than 60 years. The mean age of the patients was found to be 55.66 ± 12.21 years. (**Table 2, Figure 2)**

Table 2: Age groups		
Age in years	**Frequency (n)**	**Percent (%)**
< 40	**31**	**31**
40 to 60	**55**	**55**
> 60	**14**	**14**
Total	**100**	**100**
Mean:55.66 ± 12.21		

Figure 2: Age groups



Our study found that the most common duration of diabetes was 6 to 10 years seen in 31 patients (31%), followed by more than 15 years seen in 27 patients (27%), less than 6 years seen in 22 patients (22%) and 11 to 15 years in 20 patients (20%). The mean duration of diabetes was 10.36 ± 6.10 years.

Table 5: Sugar Control		
	Frequency (n)	**Percent (%)**
Good Control	**15**	**15**
Poor Control	**85**	**85**
Total	**100**	**100**

Figure 5: Sugar Control

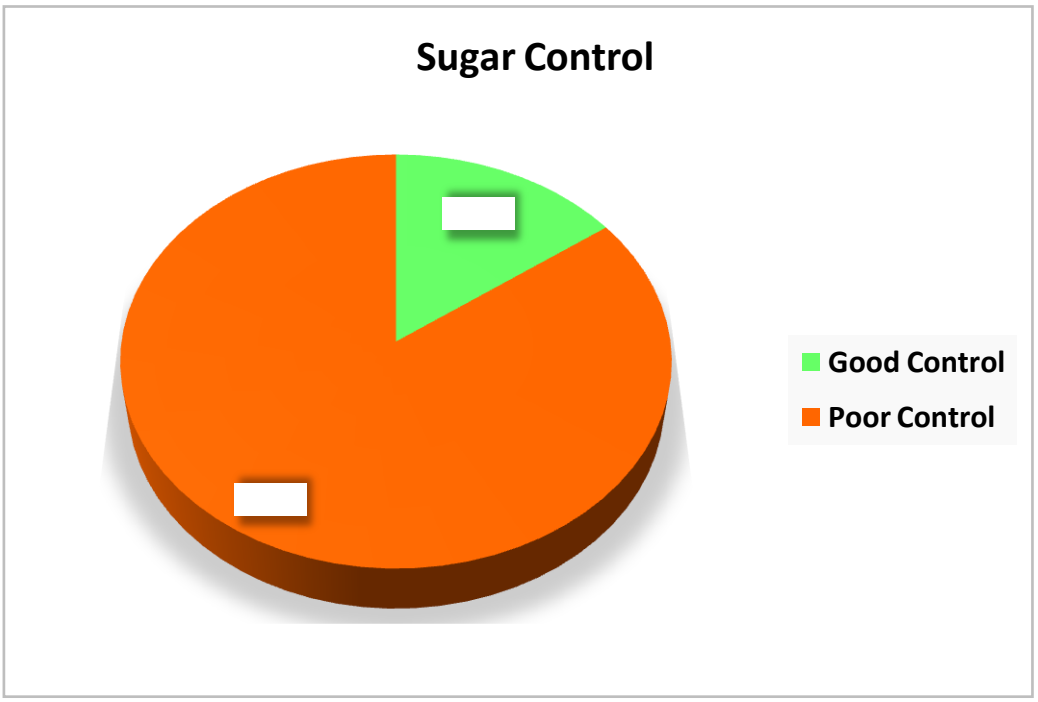


Table 6: Descriptive Statistics of quantitative variables

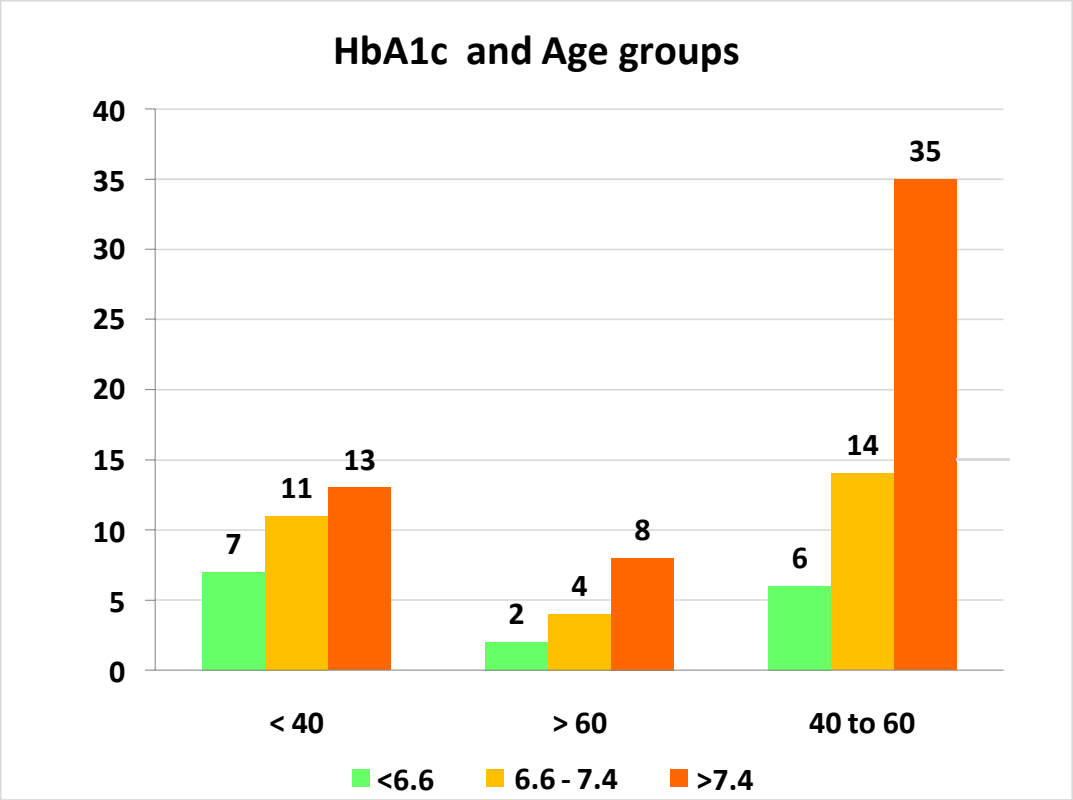
	Minimum	Maximum	Mean	S.D.
Age (years)	**25**	**85**	**55.66**	**12.21**
Duration of DM (years)	**1**	**26**	**10.36**	**6.10**
FBS (mg/dl)	**67**	**388**	**159.79**	**60.57**
PPBS (mg/dl)	**67**	**463**	**259.89**	**79.12**
TC (mg/dl)	**100**	**297**	**175.99**	**48.69**
TRIG (mg/dl)	**56**	**375**	**187.53**	**76.78**
HDL (mg/dl)	**23**	**74**	**44.07**	**11.13**
VLDL (mg/dl)	**21**	**83**	**44.38**	**16.06**
LDL (mg/dl)	**27**	**242**	**100.49**	**44.69**
HbA1c (%)	**5.1**	**12.1**	**7.85**	**1.34**

Different descriptive study variables are as shown in the above table.

In our study, the mean age of study participants was 55.66 ± 12.21 years. Themean duration of diabetes was 10.36 ± 6.10 years.

Table 8: HbA1c levels and Age Groups				
HbA1c levels	**Age Groups**			**Total**
	< 40	**40 to 60**	**> 60**	
<6.6	**7**	**6**	**2**	**15**
6.6 - 7.4	**11**	**14**	**4**	**29**
>7.4	**13**	**35**	**8**	**56**
Total	**31**	**55**	**14**	**100**
X^2= 4.12 p = 0.38 (statistically insignificant)				

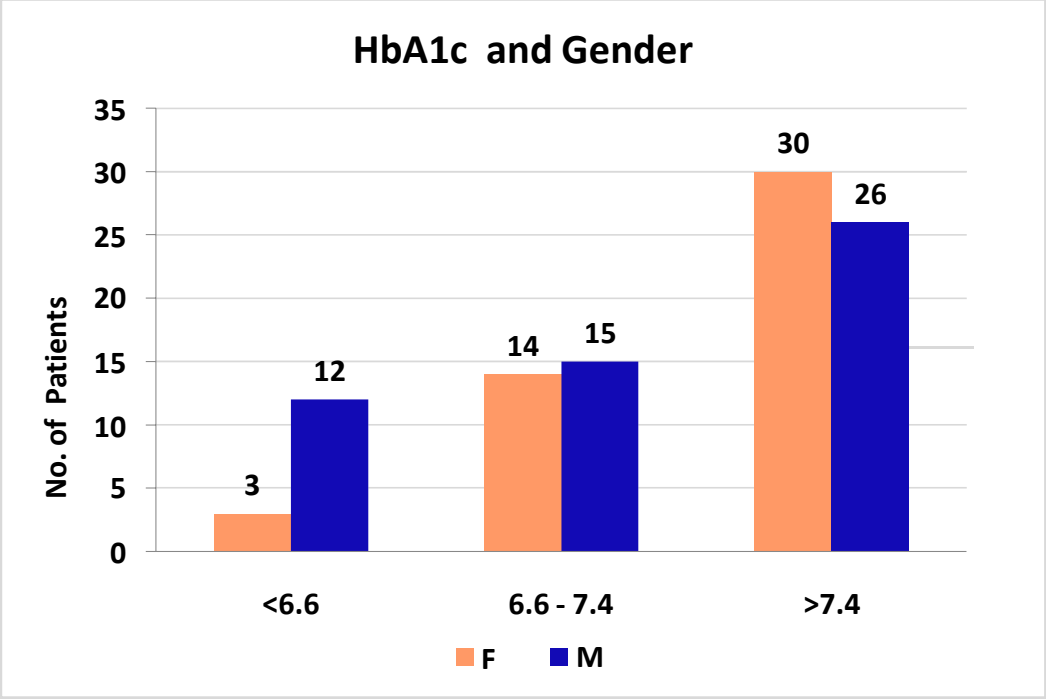
Figure 6: HbA1c levels and Age Groups



No any significant association was seen between the HbA1c levels andgender. (p = 0.07)

Table 9: HbA1c levels and Gender			
HbA1c levels	**Gender**		**Total**
	F	**M**	
<6.6	**3**	**12**	**15**
6.6 - 7.4	**14**	**15**	**29**
>7.4	**30**	**26**	**56**
Total	**47**	**53**	**100**
X^2= 5.38 p = 0.07 (statistically insignificant)			

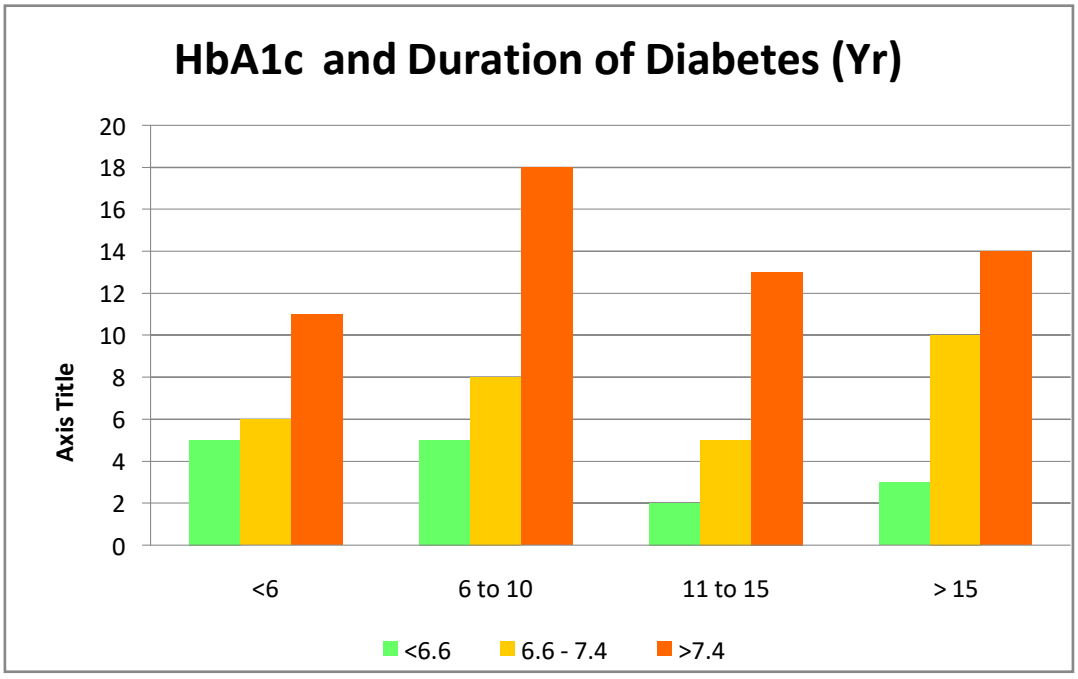
Figure 7: HbA1c levels and Gender



No any significant association was seen between the HbA1c levels andduration of diabetes. (p = 0.82)

Table 10: HbA1c levels and Duration of Diabetes					
HbA1c levels	**Duration of Diabetes**				**Total**
	<6	**6 to 10**	**11 to 15**	**> 15**	
<6.6	**5**	**5**	**2**	**3**	**15**
6.6 - 7.4	**6**	**8**	**5**	**10**	**29**
>7.4	**11**	**18**	**13**	**14**	**56**
Total	**22**	**31**	**20**	**27**	**100**
X^2= 2.88 p = 0.82 (statistically insignificant)					

Figure 8: HbA1c levels and Duration of Diabetes

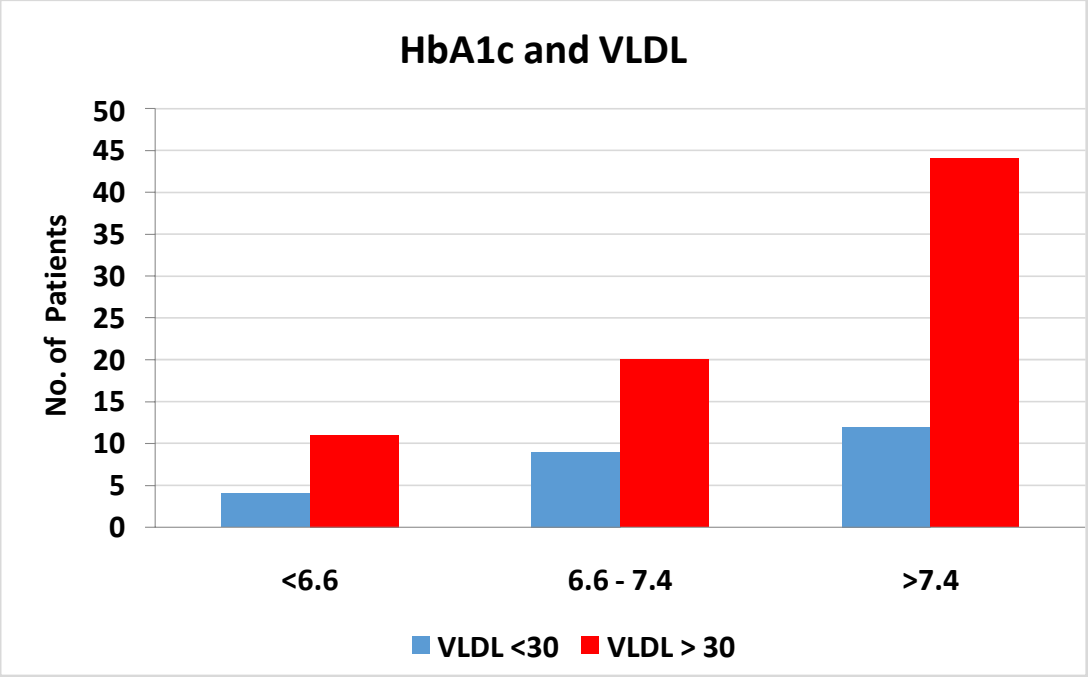


No significant association was seen between the HbA1c Groups andVLDL > 30 (p = 0.62).

Table 14: HbA1c levels and VLDL > 30

HbA1c levels	VLDL > 30		Total
	No	Yes	
<6.6	4	11	15
6.6 - 7.4	9	20	29
>7.4	12	44	56
Total	25	75	100
X^2= 0.96 p = 0.62 (statistically insignificant)			

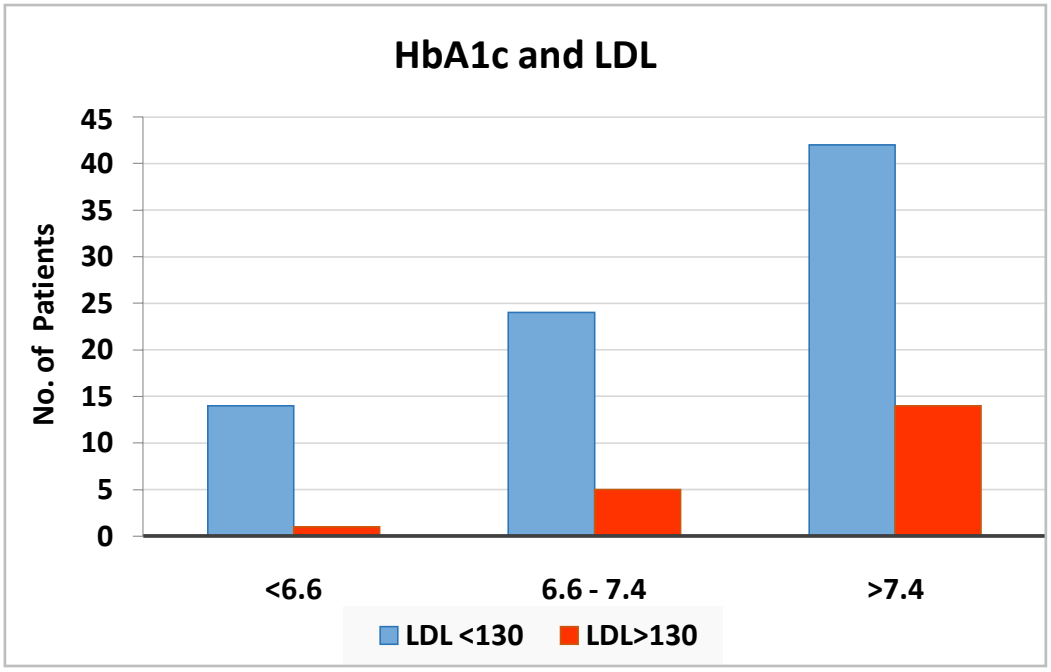
Figure 12: HbA1c levels and VLDL



No any significant association was seen between the HbA1c Groups and LDL > 130 ($p = 0.26$).

Table 15: HbA1c levels and LDL > 130			
HbA1c levels	**LDL > 130**		**Total**
	No	**Yes**	
<6.6	**14**	**1**	**15**
6.6 - 7.4	**24**	**5**	**29**
>7.4	**42**	**14**	**56**
Total	**80**	**20**	**100**
X^2= 2.68 p = 0.26 (statistically insignificant)			

Figure 13: HbA1c levels and LDL



DISCUSSION

The patients having dyslipidemia in our study were 89% of the total population. Those having hypercholesterolemia were 35%.

There were 53 males (53%) and 47 females (47%) in our study.

- The study by **Samatha P et al**, had 52% males, similar to our study,majority were males. [66]
- The study by **Devkar, et al**. had 52% males who were majority similarto the current study. [67]
- The study by **Abdulazeez Sulaiman Safo** had majority females whichis in contradiction to the current study. [68]

Most common age group was 40-60 years with 55 patients (55%) followed by

<40 years having 31 patients (31%) and more than 60 years with 14 patients

(14%).

Mean age of study participants was 55.66 ± 12.21 years

- The study by **Yadav S** had majority from age group of 50-69 years, similar to the current study. [65]
- The study by **Devkar et al**. had mean age of 62.91 which is higher than our study which had mean of 55.66 ± 12.21 years. [67]
- In the study by **Samatha P et al**, mean age was 58.56 and 52.48 among males and females respectively, this is comparable to thecurrent study where mean was 55.66 ± 12.21 years. [66]
- The study by **Abdulazeez Sulaiman Safo** had mean age of 55.56 ± 10.48 years similar to the current study. [68]

Our study found that the most common duration of diabetes was 6 to 10 years seen in 31 patients (31%), followed by more than 15 years seen in 27 patients (27%), less than 6 years seen in 22 patients (22%) and 11 to 15 years in 20 patients (2%). Mean duration of diabetes was 10.36 ± 6.10 years.

Table: Comparison with prevalence of dyslipidemia in T2DM patients from previous studies	**Year**	**Prevalence**
R M Parikh et al[74]	2010	85.5%
A G Crawford et al[75]	2011	66.7%
KM Uttra et al[76]	2011	72%
Tseng LN et al [77]	2012	72%
AK Dixit et al [78]	2013	85%
SR Joshi et al[79]	2014	79%
G Y Chen et al [80]	2015	31.9%
Li Yan et al[81]	2016	67.1%
S Palazhy et al [82]	2017	75.28%
R Anari et al [83]	2017	92%
S Sharma et al[84]	2018	86%
Current Study	**2019**	**89%**

SUMMARY AND CONCLUSION

HbA1c is a predictor of glycaemic control and is widely used worldwide to assess the sugar control status of diabetic patients. Out of all the complications of diabetes mellitus, cardiovascular complications are most dangerous. HbA1c level can be used as a good parameter for predicting the lipid profile of diabetic patients.

Lipid profile is one of the best predictors of the cardiac health. Our study found no any difference between glycaemic status of the males and females as measured by HbA1c. HbA1c had showed positive correlations with lipid profile parameters such as TC, LDL, TG, and VLDL and negative correlations between HbA1c and the HDL levels.

Deranged lipid parameters are indicative of cardiovascular risk, and these deranged lipid parameters are shown by HbA1c levels in diabetic patients. Our findings suggest that HbA1c level can be most useful tool for assessment of dyslipidaemia in diabetic patients.

So, HbA1c should be utilized for screening the diabetic patients for risk of cardiovascular events and also for timely intervention with lipid lowering drugs.

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