A Study on Lipid Profile Pattern of patients with *Diabetes Mellitus* *and relationship with Blood Pressure, Serum Creatinine level and HbA1c*

This Thesis Paper Submitted in Partial Fulfillment of the Requirement for the Degree of Masters of Pharmacy, East West University

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation, entitled "**A Study on Lipid Profile Pattern of patients with *Diabetes Mellitus and relationship with Blood Pressure, Serum Creatinine level and HbA1c***" is an authentic and genuine research work carried out by me under the guidance of Dr. Shamsun Nahar Khan, Associate Professor, Department of Pharmacy, East West University, Dhaka.

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This is to certify that the dissertation entitle "**A Study on Lipid Profile Pattern of patients with *Diabetes Mellitus and relationship with Blood Pressure, Serum Creatinine level and HbA1c***" Research work done by **SHAHRIA SULTANA**, **ID: 2014-1-79-016** in partial fulfillment of the requirement for the Degree of Masters of Pharmacy.

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This is to certify that the dissertation entitled "**A Study on Lipid Profile Pattern of patients with *Diabetes Mellitus and relationship with Blood Pressure, Serum Creatinine level and HbA1c***" is a genuine research work carried out by **Shahria Sultana,ID:2014-1-79-016** under the supervision of Shamsun Nahar Khan (PhD., Postdoc, Harvard University, Associate Professor, Department of Pharmacy, East West University, Dhaka). I further certify that no part of the thesis has been submitted for any other degree and all the resources of the information in thus connection are duly acknowledged.

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December, 2017

Shahria sultana

DEDICATION

DEDICATED TO

MY BELOVED PARENTS

WHO INSPIRED AND

SUPPORTED ME

THROUGHOUT MY WORK

A STUDY ON LIPID PROFILE PATTERN of PATIENTS WITH *DIABETES MELLITUS AND RELATIONSHIP WITH BLOOD PRESSURE, SERUM CREATININE LEVEL AND HbA1c*

Contents

List of Tables

List of Figures

Abstract

This study reviews the relationship between dyslipidemia, chronic kidney disease, and cardiovascular diseases in patients with diabetes. Diabetes mellitus is associated with complications in the cardiovascular and renal system, and is increasing in prevalence worldwide. Modification of the multifactorial risk factors, in particular dyslipidemia, has been suggested to reduce the rates of diabetes-related complications. Dyslipidemia in diabetes is a condition that includes hypertriglyceridemia, low high-density lipoprotein levels, and increased small and dense low-density lipoprotein particles. This condition is associated with higher cardiovascular risk and mortality in diabetic patients. Current treatment guidelines focus on lowering the low-density lipoprotein cholesterol level; multiple trials have confirmed the cardiovascular benefits of treatment with statins. Chronic kidney disease also contributes to dyslipidemia, and dyslipidemia in turn is related to the occurrence and progression of diabetic nephropathy.

1. Introduction

According to the report of International Diabetes Federation on the estimated projections regarding diabetes, South East Asian countries have the highest prevalence of diabetes in the world. Due to the high degree of genetic predisposition and high susceptibility to environmental insulin, characterised by a low BMI, high upper body adiposity, a high body fat percentage and a high level of insulin resistance, Indian population faces higher risk for diabetes and its complications. A low level of HDL-C is a key feature of the type 2 diabetes (Rosenson, 2005 and Martinez et al., 2002) diabetes being one of the strongest risk factors with associated age adjusted risk ratios about 2.2 for men and about 3.7 for women. Diabetes often coexists with obesity, hypertension, dyslipidaemia. Certain racial and ethnic groups (Africans, Americans, and Asians) have a greater risk of developing diabetes. The risk for diabetes increases if waist circumference measurements in men are more than 90 cm and in women they are more than 80.

There are several factors responsible for this tendency including sedentary lifestyle and improper eating habits. An increase in body fat leads to less action of insulin as well increases several other toxic substances in the body. Changes occurring in diabetic dyslipidemia include quantitative and qualitative changes. Quantitative changes include increase in VLDL as compared to normal due to increase availability of glucose for VLDL synthesis and decrease in lipoprotein lipase activity leading to decrease of VLDL from peripheral circulation, increase in LDL-C levels and decrease in HDL-C levels due to increase in hepatic lipase activity decrease in VLDL clearance. Qualitative changes include increase amount of triglycerides, LDL-C and HDL-C, non-enzymatic glycation of LDL and non-enzymatic glycation of HDL, thus increasing risk of heart diseases. Nowadays frequency of diabetes mellitus is increasing many folds. Research findings show that it is the body composition components, mainly body fat and lipid profiles that are responsible for increase prevalence of this disease. Hence the purpose of the present study is to review the relationship between dyslipidemia, chronic kidney disease, and cardiovascular diseases in patients with diabetes.

1.1. Diabetes Mellitus

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels.

In 2012 there were 1.5 million deaths worldwide directly caused by diabetes. It was the eighth leading cause of death among both sexes and the fifth leading cause of death in women in 2012. Blood glucose levels that are higherthan-optimal, even if below the diagnostic threshold for diabetes, are a major source of mortality and morbidity. The diagnostic criterion for diabetes is fasting plasma glucose ≥ 7.0 mmol/L – a diagnostic point selected on the basis of micro-vascular complications such as diabetic retinopathy. The risk of macro-vascular disease, such as heart attack or stroke, however, starts increasing well before this diagnostic point. To better understand the full impact of blood glucose levels on mortality therefore requires a look at mortality related to blood glucose as a risk factor.

The total burden of deaths from high blood glucose1 in 2012 has been estimated to amount to 3.7 million. This number includes 1.5 million diabetes deaths, and an additional 2.2 million deaths from cardiovascular diseases, chronic kidney disease, and tuberculosis related to higher-than-optimal blood glucose. Its magnitude highlights that high blood glucose causes a large burden of mortality beyond those deaths directly caused by diabetes. The largest number of deaths resulting from high blood glucose occur in upper-middle income countries (1.5 million) and the lowest number in low-income countries (0.3 million).

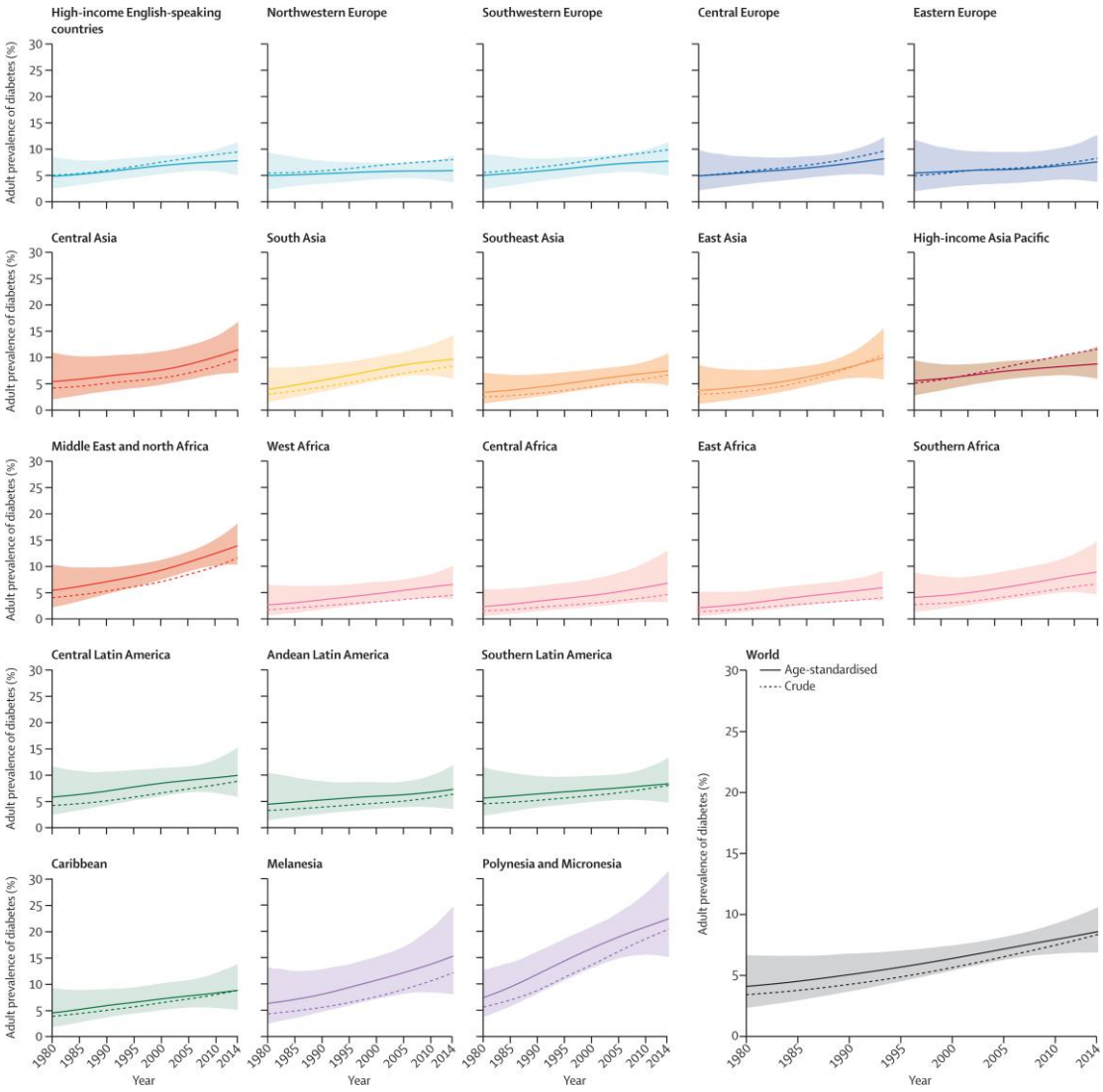
After the age of 50, middle income countries have the highest proportion of deaths attributed to high blood glucose, for both men and women. Except in high-income countries, the proportion of deaths attributable to high blood glucose for both men and women are highest in the age group 60–69 years.

Forty-three per cent of all deaths attributable to high blood glucose occur prematurely, before the age of 70 years – an estimated 1.6 million deaths worldwide. Globally, high blood glucose causes about 7% of deaths among men aged 20–69 and 8% among women aged 20–69. Figure 2 shows that the percentage of premature deaths attributable to high blood glucose is higher in low- and middle-income countries than in high-income countries, and higher among men than women.

WHO estimates that, globally, 422 million adults aged over 18 years were living with diabetes in 2014. The largest numbers of people with diabetes were estimated for the WHO South-East Asia and Western Pacific Regions, accounting for approximately half the diabetes cases in the world.

From 1980 to 2014, worldwide age-standardised adult diabetes prevalence increased from 4·3% (95% CrI 2·4–7·0) to 9·0% (7·2–11·1) in men and from 5·0% (2·9–7·9) to 7·9% (6·4–9·7) in women; the posterior probabilities that these were true increases were 0·994 and 0·954, respectively. Over these years, crude adult prevalence increased from 3·6% (2·0–5·9) to 8·8% (7·0–10·8) in men, and from 4·7% (2·7–7·4) to 8·2% (6·6–9·9) in women.

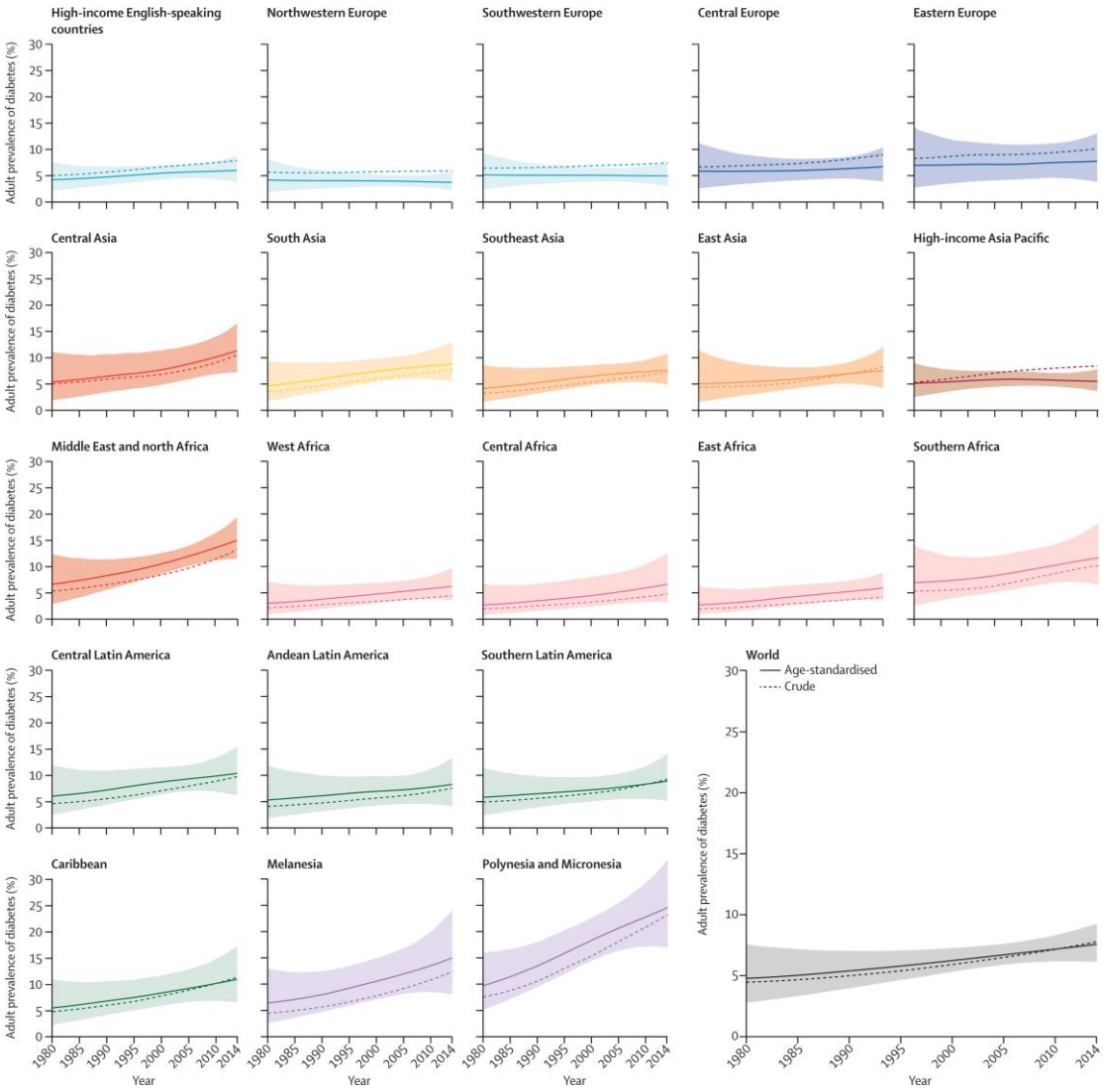
Figure 1: Trends in age-standardised and crude prevalence of diabetes for men by region



Note: The lines (solid for age-standardised and dashed for crude) show the posterior mean estimates; the shaded area shows the 95% credible intervals for age-standardised prevalence.

Source: NCD Risk Factor Collaboration, 2016

Figure 2: Trends in age-standardised and crude prevalence of diabetes for women by region



Note: The lines (solid for age-standardised and dashed for crude) show the posterior mean estimates; the shaded area shows the 95% credible intervals for age-standardised prevalence.

Source: NCD Risk Factor Collaboration, 2016

Age-standardised diabetes prevalence in women in 2014 was lowest in northwestern and southwestern Europe, which each had a prevalence of less than 5% (figure 2). The lowest prevalence in adult men was also in northwestern Europe, at 5·8% (95% CrI 3·6–8·7). Crude adult prevalence in northwestern Europe was 5·9% (3·8–8·6) for women and 7·9% (5·1–11·5) for men in 2014. At the other extreme, age-standardised diabetes prevalence was higher than 20% in adult men and women in Polynesia and Micronesia, and around 15% in Melanesia and in the Middle East and north Africa.

Over the 35 years of analysis, there was almost no change in age-standardised diabetes prevalence in women in northwestern and southwestern Europe, and only a small non-significant increase in central and eastern Europe (figure 2). Adult men in northwestern Europe also had a smaller rise in prevalence than did other regions (figure 1). By contrast, age-standardised prevalence in Polynesia and Micronesia rose by 15·0 (95% CrI 5·5–25·9) percentage points in adult men (posterior probability >0·999) and by 14·9 (4·5–26·2) percentage points in adult women (posterior probability 0·998). Crude adult prevalence increased more than age-standardised prevalence in regions that had substantial ageing—eg, in high-income regions.

Regular physical activity reduces the risk of diabetes and raised blood glucose, and is an important contributor to overall energy balance, weight control and obesity prevention – all risk exposures linked to future diabetes prevalence. The global target of a 10% relative reduction in physical inactivity is therefore strongly associated with the global target of halting the risk in diabetes.

However, the prevalence of physical inactivity globally is of increasing concern. In 2010, the latest year for which data are available, just under a quarter of all adults aged over 18 years did not meet the minimum recommendation for physical activity per week and were classified as insufficiently physically active. In all WHO regions and across all country income groups women were less active than men, with 27% of women and 20% of men classified as insufficiently physically active. Physical inactivity is alarmingly common among adolescents, with 84% of girls and 78% of boys not meeting minimum requirements for physical activity for this age.

The prevalence of physical inactivity is highest in high-income countries where it is almost double that of low-income countries. Among WHO regions, the Eastern Mediterranean Region showed the highest prevalence of inactivity in both adults and adolescents.

Being overweight or obese is strongly linked to diabetes. Despite the global voluntary target to halt the rise in obesity by 2025, being overweight or obese has increased in almost all countries. In 2014, the latest year for which global estimates are available, more than one in three adults aged over 18 years were overweight and more than one in 10 were obese. Women were more overweight or obese than men. The prevalence of obesity was highest in the WHO Region of the Americas and lowest in the WHO South-East Asian Region. The proportion of people who are overweight or obese increases with country income level. High- and middle-income countries have more than double the overweight and obesity prevalence of low-income countries

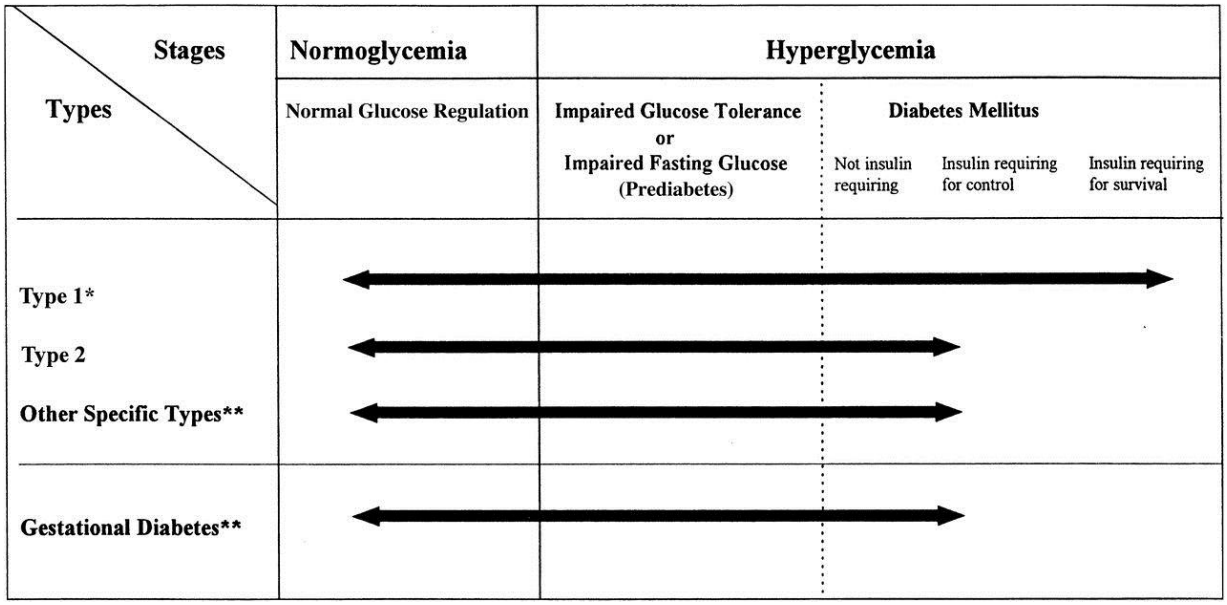
The number of people with diabetes (defined in surveys as those having a fasting plasma glucose value of greater than or equal to 7.0 mmol/L or on medication for diabetes/raised blood glucose) has steadily risen over the past few decades, due to population growth, the increase in the average age of the population, and the rise in prevalence of diabetes at each age. Worldwide, the number of people with diabetes has substantially increased between 1980 and 2014, rising from 108 million to current numbers that are around four times higher. Forty per cent of this increase is estimated to result from population growth and ageing, 28% from a rise in age-specific prevalences, and 32% from the interaction of the two (WHO, 2016).

The vast majority of cases of diabetes fall into two broad etiopathogenetic categories (discussed in greater detail below). In one category, type 1 diabetes, the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers. In the other, much more prevalent category, type 2 diabetes, the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. In the latter category, a degree of hyperglycemia sufficient to cause

pathologic and functional changes in various target tissues, but without clinical symptoms, may be present for a long period of time before diabetes is detected. During this asymptomatic period, it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load or by A1C.

The degree of hyperglycemia (if any) may change over time, depending on the extent of the underlying disease process (figure 1). A disease process may be present but may not have progressed far enough to cause hyperglycemia. The same disease process can cause impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) without fulfilling the criteria for the diagnosis of diabetes. In some individuals with diabetes, adequate glycemic control can be achieved with weight reduction, exercise, and/or oral glucose-lowering agents. These individuals therefore do not require insulin. Other individuals who have some residual insulin secretion but require exogenous insulin for adequate glycemic control can survive without it. Individuals with extensive β-cell destruction and therefore no residual insulin secretion require insulin for survival. The severity of the metabolic abnormality can progress, regress, or stay the same. Thus, the degree of hyperglycemia reflects the severity of the underlying metabolic process and its treatment more than the nature of the process itself.

Figure 3: Disorders of glycemia: etiologic types and stages.



Notes: *Even after presenting in ketoacidosis, these patients can briefly return to normoglycemia without requiring continuous therapy (i.e., "honeymoon" remission); **in rare instances, patients in these categories (e.g., Vacor toxicity, type 1 diabetes presenting in pregnancy) may require insulin for survival.

Source: American Diabetes Association, 2014

1.2 Signs and symptoms

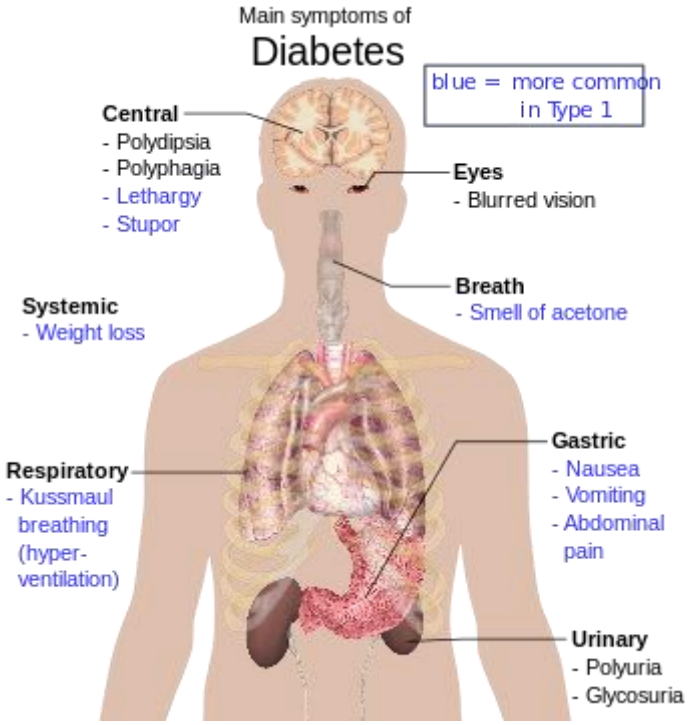


Figure-4: Overview of the most significant symptoms of diabetes (Plotnic, 2008)

The classic symptoms of untreated diabetes are weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger).Symptoms may develop rapidly (weeks or months) in type 1 DM, while they usually develop much more slowly and may be subtle or absent in type 2 DM.Several other signs and symptoms can mark the onset of diabetes, although they are not specific to the disease. In addition to the known ones above, they include blurry vision, headache, fatigue, slow healing of cuts, and itchy skin. Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes. (*WHO. 2013)*

1.3 Diabetic emergencies

Low blood sugar is common in persons with type 1 and type 2 DM. Most cases are mild and are not considered medical emergencies. Effects can range from feelings of unease, sweating, trembling, and increased appetite in mild cases to more serious issues such as confusion, changes in behavior, seizures, unconsciousness, and (rarely) permanent brain damage or death in severe cases.Mild cases are self-treated by eating or drinking something high in sugar. Severe cases can lead to unconsciousness and must be treated with intravenous glucose or injections with glucagon.People (usually with type 1 DM) may also experience episodes of diabetic ketoacidosis, a metabolic disturbance characterized by nausea, vomiting and abdominal pain, the smell of acetone on the breath, deep breathing known as Kussmaul breathing, and in severe cases a decreased level of consciousness. A rare but equally severe possibility is hyperosmolar nonketotic state, which is more common in type 2 DM and is mainly the result of dehydration. (*Holman et al; 2000)*

1.4. Complications

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10–20), but may be the first symptom in those who have otherwise not received a diagnosis before that time. The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease and about 75% of deaths in diabetics are due to coronary artery disease. Other "macro vascular" diseases are stroke, and peripheral vascular disease. The primary complications of diabetes due to damage in small blood vessels include damage to the eyes, kidneys, and nerves. Damage to the eyes, known as diabetic retinopathy, is caused by damage to the blood vessels in the retina of the eye, and can result in gradual vision loss and blindness. Damage to the kidneys, known as diabetic nephropathy, can lead to tissue scarring, urine protein loss, and eventually chronic kidney disease, sometimes requiring dialysis or kidney transplant. Damage to the nerves of the body, known as diabetic neuropathy, is the most common complication of diabetes. The symptoms can include numbness, tingling, pain, and altered pain sensation, which can lead to damage to the skin. Diabetes-related foot

problems (such as diabetic foot ulcers) may occur, and can be difficult to treat, occasionally requiring amputation. Additionally, proximal diabetic neuropathy causes painful muscle wasting and weakness. There is a link between cognitive deficit and diabetes. Compared to those without diabetes, those with the disease have a 1.2 to 1.5-fold greater rate of decline in cognitive functio*n.* (*JN et al; 2009)*

1.5 Classification of Diabetes Mellitus and Other Categories of Glucose Regulation

1.5.1 Type 1 Diabetes (β-Cell Destruction, Usually Leading to Absolute Insulin Deficiency)

Immune-Mediated Diabetes

This form of diabetes, which accounts for only 5–10% of those with diabetes, previously encompassed by the terms insulin-dependent diabetes or juvenile-onset diabetes, results from a cellular-mediated autoimmune destruction of the β-cells of the pancreas. Markers of the immune destruction of the β-cell include islet cell autoantibodies, autoantibodies to insulin, autoantibodies to GAD (GAD65), and autoantibodies to the tyrosine phosphatases IA-2 and IA-2β. One and usually more of these autoantibodies are present in 85–90% of individuals when fasting hyperglycemia is initially detected. Also, the disease has strong HLA associations, with linkage to the DQA and DQB genes, and it is influenced by the DRB genes. These HLA-DR/DQ alleles can be either predisposing or protective.

Idiopathic Diabetes

Some forms of type 1 diabetes have no known etiologies. Some of these patients have permanent insulinopenia and are prone to ketoacidosis, but have no evidence of autoimmunity. Although only a minority of patients with type 1 diabetes fall into this category, of those who do, most are of African or Asian ancestry. Individuals with this form of diabetes suffer from episodic ketoacidosis and exhibit varying degrees of insulin deficiency between episodes. This form of diabetes is strongly inherited, lacks immunological evidence for β-cell autoimmunity, and is not HLA associated. An

absolute requirement for insulin replacement therapy in affected patients may come and go.

1.5.2 Type 2 Diabetes (Ranging From Predominantly Insulin Resistance With Relative Insulin Deficiency to Predominantly an Insulin Secretory Defect With Insulin Resistance)

This form of diabetes, which accounts for ~90–95% of those with diabetes, previously referred to as non–insulin-dependent diabetes, type 2 diabetes, or adult-onset diabetes, encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency. At least initially, and often throughout their lifetime, these individuals do not need insulin treatment to survive. There are probably many different causes of this form of diabetes. Although the specific etiologies are not known, autoimmune destruction of β-cells does not occur, and patients do not have any of the other causes of diabetes listed above or below.

Most patients with this form of diabetes are obese, and obesity itself causes some degree of insulin resistance. Patients who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal region. Ketoacidosis seldom occurs spontaneously in this type of diabetes; when seen, it usually arises in association with the stress of another illness such as infection. This form of diabetes frequently goes undiagnosed for many years because the hyperglycemia develops gradually and at earlier stages is often not severe enough for the patient to notice any of the classic symptoms of diabetes.

Figure 5: Comparison of Type 1 and Type 2 Diabetes

Feature	Type 1 diabetes	Type 2 diabetes
Onset	Sudden	Gradual
Age at onset	Mostly in children	Mostly in adults
Body size	Thin or normal	Often obsess
Ketoacidosis	Common	Rare
Auto antibodies	Usually present	Absent
Endogenous insulin	Low or absent	Normal, decreased or increased
Concordance in identical twins	50%	90%
Prevalence	~10%	~90%

(Dolores et al ; 2011)

1.6 Gestational diabetes

Gestational diabetes mellitus (GDM) resembles type 2 DM in several respects, involving a combination of relatively inadequate insulin secretion and responsiveness. It occurs in about 2–10% of all pregnancies and may improve or disappear after delivery.However, after pregnancy approximately 5–10% of women with gestational diabetes are found to have diabetes mellitus, most commonly type 2. Gestational diabetes is fully treatable, but requires careful medical supervision throughout the pregnancy. Management may include dietary changes, blood glucose monitoring, and in some cases insulin may be required.Though it may be transient, untreated gestational diabetes can damage the health of the fetus or mother. Risks to the baby include macrosomia (high birth weight), congenital heart and central nervous system abnormalities, and skeletal muscle malformations. Increased levels of insulin in a fetus' blood may inhibit fetal surfactant production and cause respiratory distress

syndrome. A high blood bilirubin level may result from red blood cell destruction. In severe cases, perinatal death may occur, most commonly as a result of poor placental perfusion due to vascular impairment. Labor induction may be indicated with decreased placental function. A Caesarean section may be performed if there is marked fetal distress or an increased risk of injury associated with macrosomia, such as shoulder dystocia. (*Katzmarzyk et al; 2012)*

1.7 Other types

Prediabetes indicates a condition that occurs when a person's blood glucose levels are higher than normal but not high enough for a diagnosis of type 2 DM. Many people destined to develop type 2 DM spend many years in a state of prediabetes.Latent autoimmune diabetes of adults (LADA) is a condition in which type 1 DM develops in adults. Adults with LADA are frequently initially misdiagnosed as having type 2 DM, based on age rather than etiology.Some cases of diabetes are caused by the body's tissue receptors not responding to insulin (even when insulin levels are normal, which is what separates it from type 2 diabetes); this form is very uncommon. Genetic mutations (autosomal or mitochondrial) can lead to defects in beta cell function. Abnormal insulin action may also have been genetically determined in some cases. Any disease that causes extensive damage to the pancreas may lead to diabetes (for example, chronic pancreatitis and cystic fibrosis). Diseases associated with excessive secretion of insulin-antagonistic hormones can cause diabetes (which is typically resolved once the hormone excess is removed). Many drugs impair insulin secretion and some toxins damage pancreatic beta cells. The ICD-10 (1992) diagnostic entity, *malnutrition-related diabetes mellitus* (MRDM or MMDM, ICD-10 code E12), was deprecated by the World Health Organization when the current taxonomy was introduced in 1999.Other forms of diabetes mellitus include congenital diabetes, which is due to genetic defects of insulin secretion, cystic fibrosis-related diabetes, steroid diabetes induced by high doses of glucocorticoids, and several forms of monogenic diabetes.

1.8 The following is a comprehensive list of other causes of diabetes:

Table -2

• Genetic defects of β-cell function • Maturity onset diabetes of the young • Mitochondrial DNA mutations • Genetic defects in insulin processing or insulin action • Defects in proinsulin conversion • Insulin gene mutations • Insulin receptor mutations • Exocrine pancreatic defects • Chronic pancreatitis • Pancreatectomy • Pancreatic neoplasia • Cystic fibrosis • Hemochromatosis • Fibrocalculous pancreatopathy	• Endocrinopathies • Growth hormone excess (acromegaly) • Cushing syndrome • Hyperthyroidism • Pheochromocytoma • Glucagonoma • Infections • Cytomegalovirus infection • Coxsackievirus B • Drugs • Glucocorticoids • Thyroid hormone • β-adrenergic agonists • Statins

(Aboyans et al;2012)

1.9 Diagnostic Criteria for Diabetes Mellitus

For decades, the diagnosis of diabetes has been based on glucose criteria, either the FPG or the 75-g OGTT. In 1997, the first Expert Committee on the Diagnosis and Classification of Diabetes Mellitus revised the diagnostic criteria, using the observed association between FPG levels and presence of retinopathy as the key factor with which to identify threshold glucose level. The Committee examined data from three cross-sectional epidemiologic studies that assessed retinopathy with fundus photography or direct ophthalmoscopy and measured glycemia as FPG, 2-h PG, and A1C. These studies demonstrated glycemic levels below which there was little prevalent retinopathy and above which the prevalence of retinopathy increased in an apparently linear fashion. The deciles of the three measures at which retinopathy

began to increase were the same for each measure within each population. Moreover, the glycemic values above which retinopathy increased were similar among the populations. These analyses helped to inform a new diagnostic cut point of ≥126 mg/dl (7.0 mmol/l) for FPG and confirmed the long-standing diagnostic 2-h PG value of ≥200 mg/dl (11.1 mmol/l).

A1C is a widely used marker of chronic glycemia, reflecting average blood glucose levels over a 2- to 3-month period of time. The test plays a critical role in the management of the patient with diabetes, since it correlates well with both microvascular and, to a lesser extent, macrovascular complications and is widely used as the standard biomarker for the adequacy of glycemic management. Prior Expert Committees have not recommended use of the A1C for diagnosis of diabetes, in part due to lack of standardization of the assay. However, A1C assays are now highly standardized so that their results can be uniformly applied both temporally and across populations. In their recent report (3), an International Expert Committee, after an extensive review of both established and emerging epidemiological evidence, recommended the use of the A1C test to diagnose diabetes, with a threshold of ≥6.5%, and ADA affirms this decision. The diagnostic A1C cut point of 6.5% is associated with an inflection point for retinopathy prevalence, as are the diagnostic thresholds for FPG and 2-h PG (3). The diagnostic test should be performed using a method that is certified by the National Glycohemoglobin Standardization Program (NGSP) and standardized or traceable to the Diabetes Control and Complications Trial reference assay. Point-of-care A1C assays are not sufficiently accurate at this time to use for diagnostic purposes.

The established glucose criteria for the diagnosis of diabetes remain valid. These include the FPG and 2-h PG. Additionally, patients with severe hyperglycemia such as those who present with severe classic hyperglycemic symptoms or hyperglycemic crisis can continue to be diagnosed when a random (or casual) plasma glucose of ≥200 mg/dl (11.1 mmol/l) is found. It is likely that in such cases the health care professional would also measure an A1C test as part of the initial assessment of the severity of the diabetes and that it would (in most cases) be above the diagnostic cut point for diabetes. However, in rapidly evolving diabetes, such as the development of

type 1 diabetes in some children, A1C may not be significantly elevated despite frank diabetes.

1.10. Management

Diabetes mellitus is a chronic disease, for which there is no known cure except in very specific situations.Management concentrates on keeping blood sugar levels as close to normal, without causing low blood sugar. This can usually be accomplished with a healthy diet, exercise, weight loss, and use of appropriate medications (insulin in the case of type 1 diabetes; oral medications, as well as possibly insulin, in type 2 diabetes).Learning about the disease and actively participating in the treatment is important, since complications are far less common and less severe in people who have well-managed blood sugar levels.The goal of treatment is an HbA_{1C} level of 6.5%, but should not be lower than that, and may be set higher. Attention is also paid to other health problems that may accelerate the negative effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise.Specialized footwear is widely used to reduce the risk of ulceration, or re-ulceration, in at-risk diabetic feet. Evidence for the efficacy of this remains equivocal, however. (*Urban et al;2009)*

1.11 .Lifestyle

People with diabetes can benefit from education about the disease and treatment, good nutrition to achieve a normal body weight, and exercise, with the goal of keeping both short-term and long-term blood glucose levels within acceptable bounds. In addition, given the associated higher risks of cardiovascular disease, lifestyle modifications are recommended to control blood pressure. (*Chiarelli et al; 2012)*

1.12. Medications

Medications used to treat diabetes do so by lowering blood sugar levels. There are a number of different classes of anti-diabetic medications. Some are available by mouth, such as metformin, while others are only available by injection such as GLP-1

agonists. Type 1 diabetes can only be treated with insulin, typically with a combination of regular and NPH insulin, or synthetic insulin analogs.Metformin is generally recommended as a first line treatment for type 2 diabetes, as there is good evidence that it decreases mortality.It works by decreasing the liver's production of glucose.Several other groups of drugs, mostly given by mouth, may also decrease blood sugar in type II DM. These include agents that increase insulin release, agents that decrease absorption of sugar from the intestines, and agents that make the body more sensitive to insulin.When insulin is used in type 2 diabetes, a long-acting formulation is usually added initially, while continuing oral medications.Doses of insulin are then increased to effect.Since cardiovascular disease is a serious complication associated with diabetes, some recommend blood pressure levels below 120/80 mmHg; however, evidence only supports less than or equal to somewhere between 140/90 mmHg to 160/100 mmHg. Amongst medications that lower blood pressure, angiotensin converting enzyme inhibitors (ACEIs) improve outcomes in those with DM while the similar medications angiotensin receptor blockers (ARBs) do not. Aspirin is also recommended for patient with cardiovascular problems, however routine use of aspirin has not been found to improve outcomes in uncomplicated diabetes. (*Kirkman et al;2010)*

Anti-diabetic drugs:

Drugs used in diabetes treat diabetes mellitus by lowering glucose levels in the blood. With the exceptions of insulin, exenatide, liraglutide and pramlintide, all are administered orally and are thus also called oral hypoglycemic agents or oral antihyperglycemic agents. There are different classes of anti-diabetic drugs, and their selection depends on the nature of the diabetes, age and situation of the person, as well as other factors.Diabetes mellitus type 1 is a disease caused by the lack of insulin. Insulin must be used in Type I, which must be injected.Diabetes mellitus type 2 is a disease of insulin resistance by cells. Type 2 diabetes mellitus is the most common type of diabetes. Treatments include (1) agents that increase the amount of insulin secreted by the pancreas, (2) agents that increase the sensitivity of target organs to insulin, and (3) agents that decrease the rate at which glucose is absorbed from the gastrointestinal tract.Several groups of drugs, mostly given by mouth, are

effective in Type II, often in combination. The therapeutic combination in Type II may include insulin, not necessarily because oral agents have failed completely, but in search of a desired combination of effects. The great advantage of injected insulin in Type II is that a well-educated patient can adjust the dose, or even take additional doses, when blood glucose levels measured by the patient, usually with a simple meter, as needed by the measured amount of sugar in the blood. (Alvin 18th edition)

CATEGORIS OF ANTIDIABETIC DRUGS

1) Insulin
2) Alpha-glucosidase inhibitors (starch inhibitors)
3) Sulfonylureas
4) Biguanides
5) Thizoladinediones
6) Insulin secretagogues

2. Dyslipidemia

Dyslipidemia is a medical term for abnormally high levels of fats (lipids) in the blood. The two major types of lipids found in the blood are triglycerides and cholesterol. Triglycerides are made when your body stores the extra calories it doesn't need for energy. They also come directly from your diet in foods such as red meat and whole-fat dairy. A diet high in refined sugar, fructose, and alcohol raises triglycerides. Cholesterol is produced naturally in your liver because every cell in your body uses it. Similar to triglycerides, cholesterol is also found in fatty foods like

eggs, red meat, and cheese. Dyslipidemia is more commonly known as high cholesterol. Although high cholesterol can be inherited, it's more often the result of unhealthy lifestyle choices.

2.1. Causes of Dyslipidemia

- Hereditary factors are the most common cause.
- A diet high in saturated fat and cholesterol increases blood cholesterol and triglyceride levels.
- Other disorders, such as diabetes mellitus, kidney disease, and hypothyroidism, may promote hypertriglyceridemia.
- Certain drugs, such as estrogen, corticosteroids, retinoids, protease inhibitors, thiazide diuretics, and beta-blockers, may cause hypertriglyceridemia.
- Obesity increases the risk of Dyslipidemia.
- Chronic, excessive alcohol use increases the risk of hypertriglyceridemia.
- Smoking and not exercising may lead to Dyslipidemia.
- Steroid uses, alcoholism, hypothyroidism, oral contraceptives, chronic renal failure, hypopituitarism and nephritic syndrome are other contributors to Dyslipidemia.

2.2. Symptoms of Dyslipidemia

- If Dyslipidemia results in CHD or atherosclerosis at other sites, symptoms may include chest pain (angina), heart attack, or stroke.
- Dyslipidemia itself does not produce symptoms. When levels are exceedingly high, cholesterol may be deposited (xanthomas) in tendons or just beneath the skin under the eyes. Very high triglyceride levels may result in the formation of nodules on the elbows or knees, or the appearance of multiple, pimple-sized, yellowish skin eruptions.

- The skin deposits fats or xanthomas
- Swelling of organs such as the liver, spleen, or pancreas (pancreatitis)
- Blockage of blood vessels in brain and heart

2.3. Prevention of Dyslipidemia

- Eating a diet low in saturated fats and cholesterol to prevent lipid abnormalities.
- Eating foods high in soluble fiber such as oats, beans, peas and certain fruits.
- Exercising regularly.
- Maintaining a healthy weight—or lose weight, if necessary.
- Moderate alcohol consumption increases levels of HDL cholesterol, which decreases the risk of CHD. However, chronic, heavy alcohol use raises triglyceride levels, and is associated with many other harmful effects. Therefore, it is recommended that, on average, women consume no more than one alcoholic beverage per day; men should consume no more than two alcoholic drinks daily. (A drink is considered one 12-ounce beer, 4 ounces of wine, or 1.5 ounces of 80-proof spirits.)

2.4. Dyslipidemia Diagnosis

It is recommended that all adults 20 years and older obtain a fasting lipid profile—a blood test that measures total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides—at least every 5 years. Those with known risk factors, such as diabetes or a family history of CHD, may need to be screened more frequently.

2.5. How to Treat Dyslipidemia

- Underlying causes of the disorder, such as diabetes and hypothyroidism, should be treated.

- Lifestyle changes, including eating a diet low in saturated fats and cholesterol, can have a great impact on lipids. Other dietary measures include the use of foods that contain plant stanols and sterols, which are added to certain margarines and salad dressings, and consuming foods rich in soluble fiber, such as cereal grains, legumes, and many fruits and vegetables.
- Weight loss is the most effective way to lower triglyceride levels.
- Quitting smoking and exercising may raise levels of "good" HDL cholesterol.
- Medications specifically designed to reduce blood cholesterol levels, such as bile acid sequestrants (cholestyramine, colestipol, and coesevelam), statins (lovastatin, pravastatin, simvastatin, fluvastatin, and atorvastatin), or niacin, may be prescribed when dietary modifications prove inadequate.
- Statins, niacin, or fibrates (gemfibrozil, fenofibrate) can be used to lower triglyceride levels when lifestyle measures fail to produce adequate results. Hypertriglyceridemia often requires treatment with a combination of a fibrate and a statin.
- In rare patients with extremely high cholesterol levels (familial hypercholesterolemia), repeated removal of blood plasma (plasmapheresis) may be recommended to lower blood cholesterol levels.
- Most people require long-term, even lifelong, treatment of Dyslipidemia with both lifestyle measures and medications

3. Literature Review

Over the past decade or so, there has been increased international focus on the health burden of diabetes, it increasing incidence and prevalence, and the associated costs to society. In Australia in 1996 it has been estimated that diabetes was responsible for 6.5% of all deaths, 5.2% of all years of life lost, and 4.6% of the years of life lost owing to disability.

Also in 1996, Australian Health Ministers agreed that diabetes be identified as the fifth National Health Priority Area. Following this, the National Diabetes Strategy and Implementation Plan (1998) identified population sub-groups requiring special attention in diabetes prevention and care services as follows: Indigenous Australians, people from culturally and linguistically diverse backgrounds, people living in rural and remote areas, children and adolescents, and older Australians.

Much of the burden of diabetes can be reduced through better diagnosis and management. Improvements in diabetes management have been identified in a range of settings, such as general practice, hospital emergency departments, outpatient diabetes clinics and antenatal care clinics. Considerable work is needed in order to better understand the ways in which people with diabetes self-manage their condition and how living with diabetes impacts upon their lives (Pollard et al. 2000).

Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein–Cholesterol (HDL-C) level that contributes to the development of atherosclerosis, which may be primary (genetic) or secondary and diagnosed by measuring plasma levels of total cholesterol (TC), TGs, and individual lipoproteins. It is traditionally classified by patterns of elevation in lipids and lipoproteins. Dyslipidemia is a well-recognized and modifiable risk factor that should be identified early to institute aggressive cardiovascular preventive management. Patients with type 2 DM are at greater risk of developing vascular diseases because of lipid changes. Lipid abnormalities and insulin use is critically discussed in diabetics. The most typical lipoprotein pattern reported in diabetes, also known as diabetic

dyslipidemia or atherogenic dyslipidemia consists of moderate elevation in TG levels, low HDL-C (Agrawal et al. 2014).

CKD is associated with dyslipidemia associating hypertriglyceridemia, elevated LDL cholesterol, an accumulation of ApoB containing lipoproteins, increased concentrations of lipoprotein (a) particles and low HDL levels (Barter, P et al. 2007). Many recent reviews analyzed this dyslipidemia in detail. Dyslipidemia in CKD is unique for many reasons. First, cardiovascular (CV) diseases are the leading cause of mortality in CKD patients. Number of cardiovascular events has been strongly correlated with GFR decline and despite constant improvement of renal suppletion therapies, such as hemodialysis, this cardiovascular mortality remains at the forefront. Traditional strategies for cardiovascular prevention, including the prescription of statins, failed in some CKD populations. Even if post hoc analysis of large prospective studies sketched a potential benefit in early stages of CKD, this positive effect is diminished in advanced stages (4 and 5), either on intima/media thickness or cardiovascular mortality and related events, as shown by 4D and AURORA studies. Recent meta-analysis from the Cochrane Collaboration confirmed this observation in dialysis patients but also suggested its interest for CKD patients who did not require hemodialysis or transplant recipients. However, beyond its effects on CV mortality, statins exhibited beneficial effects for impeding renal failure progression. Indeed, statins can modulate intracellular pathways of inflammatory and fibrogenic responses and inhibit the proliferation of mesangial and renal tubular epithelial cells. Moreover, recent data corroborate their importance in lipid control to prevent the progression of CKD. The increase of one standard deviation of TG level and TG/HDL-cholesterol ratio was correlated with an increased risk of developing CKD. Additionally, increases of HDL-cholesterol level, LDL-cholesterol/ApoB and HDL-cholesterol/ApoAI ratios seemed to be protective (Florens et al. 2016).

The most important complication in evolution of chronic kidney disease is renal failure; renal failure from diabetes is the most common single cause of entry to renal replacement programs worldwide. Even this disease can be treated by dialysis and transplantation the costs are at high levels all over the world. So it is more important to prevent the apparition of this important complication. The time necessarily for

instauration of renal failure from the apparition of microalbuminuria is 9 years. This time will be double with an adequate treatment of blood pressure, and a optimal control of blood glucose level. [8] Screening for diabetic nephropathy should be performed annually, by measuring urine albumin/creatinine ratio and estimated GFR (eGFR). If urine albumin excretion is raised, an inhibitor of the renin angiotensin system should be commenced and titrated up to the maximum tolerated dose. Maintaining BP<125/75 mmHg will reduce the rate of decline of GFR from 10-12 to 3-5 mL/min/1.73 m2. Reducing dietary protein intake to 0.7-1.0 g/kg body weight per day may slow the deterioration in renal function. Aggressive management of other cardiovascular risk factors and prescription of aspirin reduces the incidence of cardiovascular events and of progression to nephropathy by around 60%.

In US chronic kidney disease is present in 40% of patients with type 2 diabetes (study included 1462 patients who participated in the trial Fourth National Health and Nutrition Examination Survey - NHANES IV in 1999-2004). [11] There is a strong correlation between cardiac and renal pathophysiology in type 2 diabetes, this is expressed by the so-called cardio-renal risk factors: Type 2 diabetes, Smoking, Obesity, Hypertension, Dyslipidemia, Genetic factors, etc.

Regarding the association of diabetes with dyslipidemia, approximately 50% of people with diabetes present simultaneously both hypertension and dyslipidemia (according to a recent study in which the total number of subjects was 137745: 56% presenting diabetes + hypertension + hyperlipidemia, and diabetes + dyslipidemia or diabetes + hypertension or diabetes were only at the rate of about 10-15%).

A detailed overview of risk assessment deciding in whom to use statin therapy is provided in the Vascular Protection chapter (p. S100). Principles of risk assessment also are discussed in the 2012 Canadian Cardiovascular Society (CCS) Guidelines for the Management of Dyslipidemia, and efforts were made to ensure consistency between the guidelines.

The burden of dyslipidemia is high in people with diabetes. A national cross-sectional chart audit study of 2473 Canadians with type 2 diabetes revealed that 55% of

individuals with a diabetes diagnosis of 2 years' duration also had dyslipidemia. This propor- tion rose to 66% in those with diabetes for !15 years (10). Therefore, a fasting lipid profile (total cholesterol [TC], HDL-C, TG and calcu- lated LDL-C) should be conducted at the time of diagnosis of dia- betes, and, if the results are initially normal, the assessment should be repeated annually or as clinically indicated. If treatment for dyslipidemia is initiated, more frequent testing is warranted. A fast of >8 hours may be inappropriate for individuals with diabetes, especially if long-acting basal insulin is part of their treatment regimen. Under these circumstances, non-HDL cholesterol (TC minus HDL-C) or apolipoprotein B (apo B) measurements (see below), which are valid, even in the nonfasting state, may be used. For screening in children and adolescents, please refer to the chapters dedicated to diabetes in children and adolescents (Manda et al., 2016)

In the widely acclaimed popular Scandinavian Simvastatin Survival Study (4S) trial, simvastatin significantly reduced CHD incidence and total mortality in diabetic subjects with high LDL cholesterol or with previous clinical CHD (The Scandinavian Simvastatin Survival Study Group, 1994; Pedersen et al., 2000). In the Cholesterol and Recu rrent Events (CARE) study, pravastatin reduced CHD incidence significantly in diabetic subjects with average LDL levels and with previous clinical CHD (Goldberg et al., 1998). In the Helsinki Heart Study, gemfibrozil was associated with a reduction in CHD in diabetic subjects without prior CHD (Frick et al., 1993). The recently completed Heart Protection Study (HPS) has been the largest study to date, enrolling and randomizing 5,963 patients over the age of 40 years with diabetes and total serum cholesterol more than 135 mg/dl (Collins et al., 2003). In this trial, patients with diabetes assigned to simvastatin had a 22% reduction in the event rate for major cardiovascular disease (Collins et al., 2003). In the Veterans Affairs High - Density Lipoprotein Chole sterol Intervention Trial (VA - HIT), gemfibrozil was associated with a 24% decrease in cardiovascular events in diabetic subjects with prior cardiovascular disease, low HDL (<40 mg/dl) and modestly elevated triglycerides (Robins et al., 2001). Two recent tr ials (Anglo - Scandinavian Cardiac Outcomes Trial - Lipid Lowering Arm [ASCOT - LLA] with atrovastatin 10 mg and Antihypertensive and Lipid – Lowering Treatment to Prevent Heart Attack Trial

[ALLHAT] with pravastatin 10 mg) indicated that further reduction of the LDL - C threshold resulted in additional ben efits for patients in the moderately high - risk category (Sever et al., 2003). A meta - analysis of four large trials revealed that the high - dose statin therapy significantly improves cardiovascular outcomes as compared to the standard low - dose one (The Scand inavian Simvastatin Survival Study Group, 1994) . The ASCOT - LLA and Collaborative Atorvastatin Diabetes Study (CARDS) suggests people with type 2 diabetes could benefit from statin therapy to reduce CVD risk, ev en when they do not have high choles terol (Pe dersen et al., 2000; Goldberg et al., 1998).

4. Methodology

This was a survey based study where among the patients randomly 135 patients were taken as volunteers who were diagnosed with Diabetes during the period from 1st of June 2016 to 31 of November 2016 at Khidmah Hospital (Pvt) & Health Care Development Project (HCDP), Dhaka. A self-administered questionnaire was made to observe the patient's diagnostic reports, prescriptions etc. Patient of known or newly diagnosed cases of diabetes mellitus (type-2) who came through outdoor patients department (OPD) or indoor patients were evaluated and those patients who meet the inclusion criteria were enrolled in the study. The detail history was taken; relevant clinical examination and all routine investigations were performed. An informed consent was taken from every patient after full explanation of procedure. Every patient was advise for at least 12-14 hours overnight fasting and the 5ml venous blood sample were collected in a disposable syringe on next morning (before breakfast) for the serum lipid profile and fasting blood sugar (for the assessment of blood glucose level).The known cases of type 2 *diabetes mellitus* will also be evaluated for their blood sugar (control or un-control) by advising the HbA1C level. The data was collected on predesign proforma and then entered, saved and analyzed in Microsoft Excel 2010.The frequency of dyslipidemia was evaluated while the pattern were determine by serum level for cholesterol, high density lipoprotein HDL-C, low density lipoprotein LDL-C and triglyceride. Data was analyzed using Microsoft Excel 2010 and statistical package.

4.2.Data Variables

Blood pressure: Blood pressure (BP) is the pressure of circulating blood on the walls of blood vessels. Used without further specification, "blood pressure" usually refers to the pressure in large arteries of the systemic circulation. Blood pressure is usually expressed in terms of the systolic pressure (maximum during one heart beat) over diastolic pressure (minimum in between two heart beats) and is measured in millimetres of mercury (mmHg), above the surrounding atmospheric pressure (considered to be zero for convenience).

It is one of the vital signs, along with respiratory rate, heart rate, oxygen saturation, and body temperature. Normal resting blood pressure in an adult is approximately 120 millimetres of mercury (16 kPa) systolic, and 80 millimetres of mercury (11 kPa) diastolic, abbreviated "120/80 mmHg" (Ogedegbe, Gbenga; Pickering, Thomas 2010).

Traditionally, blood pressure was measured non-invasively using a mercury manometer and this is still generally considered the gold standard. More recently other semi-automated methods have become common, largely due to concerns about potential mercury toxicity, although cost and ease of use have also influenced this trend. Early alternatives to mercury sphygmomanometers were often inaccurate, but more modern validated devices have similar accuracy to mercury devices (O'brien, 2001).

Blood pressure is influenced by cardiac output, total peripheral resistance and arterial stiffness and varies depending on situation, emotional state, activity, and relative health/disease states. In the short term it is regulated by baroreceptors which act via the brain to influence nervous and endocrine systems.

Blood pressure that is low due to a disease state is called hypotension, and pressure that is consistently high is hypertension. Both have many causes and may be of sudden onset or of long duration. Long term hypertension is a risk factor for many diseases, including heart disease, stroke and kidney failure. Long term hypertension is more common than long term hypotension. Long term hypertension often goes undetected because of infrequent monitoring and the absence of symptoms.

Glucose Test: A glucose test is a type of blood test used to determine the amount of glucose intheblood.Itismainlyused in screening for prediabetes or diabetes. Patients are instructed not to consume anything but water during the fasting period. Caffeine will also distort the results. If the person eats during the period in which he or she is supposed to have been fasting then they may show blood sugar levels that may cause his or her doctor to think the person has or is at increased risk of having diabetes. In people already having diabetes, blood glucose monitoring is used with frequent intervals in the management of the condition (American Diabetes Association, 2005).

There are several different kinds of glucose tests:

- Fasting blood sugar (FBS), fasting plasma glucose (FPG): 8 or 12 or 14 hours after eating
- Glucose tolerance test: continuous testing
- Postprandial glucose test (PC): 2 hours after eating
- Random glucose test

Fasting blood sugar: A range of 4 to 5.5 mmol/l (70 to 99 mg/dl) before a meal is normal. Continual fasting levels of 5.5 to 7 mmol/l (101–125 mg/dl) causes concern of possible prediabetes and may be worth monitoring. 7 mmol/l (126 mg/dl) and above means a risk of diabetes. After a 12‑hour fast, a range of 3.9 to under 5.5 mmol/l (70.2 to 100 mg/dl) is normal; a level of 5.6 to under 7 mmol/l (100 to 126 mg/dl) is considered a sign of prediabetes.

Postprandial glucose: A level of < 7.8 mmol/l (140 mg/dl) 90 minutes after a meal is normal.

Creatinine Blood Test: A creatinine blood test measures the level of creatinine in the blood. Creatinine is a waste product that forms when creatine breaks down. Creatine is found in your muscle. Creatinine levels in the blood can provide your doctor with information about how well your kidneys are working.

The kidneys are a pair of fist-sized organs located at the bottom of the rib cage. One kidney is on each side of the spine. Each kidney has millions of small blood-filtering units called nephrons. The nephrons constantly filter blood through a very tiny cluster of blood vessels known as glomeruli. These structures filter waste products, excess water, and other impurities out of the blood. The toxins are stored in the bladder and then removed during urination.

Creatinine is one of the substances that your kidneys normally eliminate from the body. Doctors measure the level of creatinine in the blood to check kidney function.

High levels of creatinine may indicate that your kidney is damaged and not working properly.

Creatinine blood tests are usually performed along with several other laboratory tests, including a blood urea nitrogen (BUN) test and a basic metabolic panel (BMP) or comprehensive metabolic panel (CMP). These tests are done during routine physical exams to help diagnose certain diseases and to check for any problems with your kidney function (Samra, M. and Abcar, A.C., 2012).

Creatinine is measured in milligrams per deciliter of blood (mg/dL). People who are more muscular tend to have higher creatinine levels. Results may also vary depending on age and gender. In general, however, normal creatinine levels range from 0.7 to 1.3 mg/dL in men and 0.6 to 1.1 mg/dL in women. High serum creatinine levels in the blood indicate that the kidneys aren't functioning properly.

HbA1c: The term *HbA1c* refers to glycated haemoglobin. It develops when haemoglobin, a protein within red blood cells that carries oxygen throughout your body, joins with glucose in the blood, becoming 'glycated'. By measuring glycated haemoglobin (HbA1c), clinicians are able to get an overall picture of what our average blood sugar levels have been over a period of weeks/months. For people with diabetes this is important as the higher the HbA1c, the greater the risk of developing diabetes-related complications. HbA1c is also referred to as **haemoglobin A1c** *or* simply **A1c**.

When the body processes sugar, glucose in the bloodstream naturally attaches to haemoglobin. The amount of glucose that combines with this protein is directly proportional to the total amount of sugar that is in your system at that time.

In diabetes mellitus, higher amounts of glycated hemoglobin, indicating poorer control of blood glucose levels, have been associated with cardiovascular disease, nephropathy, neuropathy, and retinopathy. A trial on a group of patients with Type 1 diabetes found that monitoring by caregivers of HbA_{1c} led to changes in diabetes treatment and improvement of metabolic control compared to monitoring only of blood or urine glucose. However, a trial designed specifically to determine

whether reducing HbA_{1c} below the normal 6%, using primarily insulin and sulfonylureas (both known to easily drive blood sugar too low), would reduce the rate of cardiovascular events in type 2 diabetes found *higher* mortality—the trial was terminated early. The negative outcomes may well have been a result of the treatment approach, primarily insulin and sulfonylureas, utilized in the "intensive" treatment group instead of LCHF, GlP-1 analogues & SGLT-2 inhibitors, none of which have these problems & lower cardiovascular mortality (Larsen, M.L., Hørder, M. and Mogensen, E.F., 1990).

Because red blood cells in the human body survive for 8-12 weeks before renewal, measuring glycated haemoglobin (or HbA1c) can be used to reflect average blood glucose levels over that duration, providing a useful longer-term gauge of blood glucose control. If your blood sugar levels have been high in recent weeks, your HbA1c will also be greater.

The HbA1c target for people with diabetes to aim for is **48 mmol/mol (6.5%).**

Note that this is a general target and people with diabetes should be given an individual target to aim towards by their health team. An individual HbA1c should take into account your ability to achieve the target based on your day to day life and whether you are at risk of having regular or severe hypos (Gerstein HC, Miller ME, Byington RP, et al. 2008).

HbA1c can indicate people with prediabetes or diabetes as follows:

HbA1c	mmol/mol	%
Normal	**Below 42 mmol/mol**	**Below 6.0%**
Prediabetes	**42 to 47 mmol/mol**	**6.0% to 6.4%**
Diabetes	**48 mmol/mol or over**	**6.5% or over**

Total Cholesterol: Cholesterol is measured enzymatically in serum or plasma in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol. One of the reaction byproducts, H2O2 is measured quantitatively in a peroxidase-catalyzed reaction that produces a color. Absorbance is measured at 500 nm. The color intensity is proportional to cholesterol concentration.

Elevated levels of cholesterol increase the risk for coronary heart disease (CHD). Cholesterol is measured to help assess the patient's risk status and to follow the progress of patient's treatment to lower serum cholesterol concentrations. Desirable cholesterol levels are considered to be those below 200 mg/dL in adults and below 170 mg/dL in children (Artiss, J.D. and Zak, B., 2000).

Triglycerides: Triglycerides are measured enzymatically in serum or plasma using a series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol. Glycerol is then oxidized using glycerol oxidase, and H2O2, one of the reaction products, is measured as described above for cholesterol. Absorbance is measured at 500 nm.

High levels of serum triglycerides help mark conditions that are associated with increased risk for CHD and peripheral atherosclerosis. High triglycerides are associated with increased risk for CAD in patients with other risk factors, such as low HDL-cholesterol, some patient groups with elevated apolipoprotein B concentrations, and patients with forms of LDL that may be particularly atherogenic. Desirable

fasting triglyceride levels are considered to be those below 200 mg/dL, and are further categorized as Borderline, 200-400 mg/dL; High, 400-1,000 mg/dL; and Very High (> 1000 mg/dL). Very high triglycerides can result in pancreatitis and should be promptly evaluated and treated. Triglycerides are also measured because the value is used to calculate low-density lipoprotein (LDL)-cholesterol concentrations.

High-density lipoprotein (HDL) cholesterol: Low serum concentrations of HDL-cholesterol are associated with increased risk for CHD. Coronary risk increases markedly as the HDL concentration decreases from 40- to 30 mg/dL. A low HDL-cholesterol concentration is considered to be a value below 35 mg/dL, and high HDL, >60 mg/dL. HDL-cholesterol values are also used in the calculation of LDL-cholesterol.

Direct HDL method - HDL is measured directly in serum. The basic principle of the method is as follows. The apoB containing lipoproteins in the specimen are reacted with a blocking reagent that renders them non-reactive with the enzymatic cholesterol reagent under conditions of the assay. The apoB containing lipoproteins are thus effectively excluded from the assay and only HDL-chol is detected under the assay conditions.

The reagents are purchased from Roche/Boehringer-Mannheim Diagnostics. The method uses sulfated alpha-cyclodextrin in the presence of Mg+2, which forms complexes with apoB containing lipoproteins, and polyethylene glycol-coupled cholesteryl esterase and cholesterol oxidase for the HDL-cholesterol measurement (Belcher et al. 1991).

LDL-cholesterol: Most of the circulating cholesterol is found in three major lipoprotein fractions: very low-density lipoproteins (VLDL), LDL and HDL.

LDL-cholesterol is calculated from measured values of total cholesterol, triglycerides and HDL cholesterol according to the relationship:

[LDL-chol] = [total chol] - [HDL-chol] - [TG]/5

where [TG]/5 is an estimate of VLDL-cholesterol and all values are expressed in mg/dL.

LDL carries most of the circulating cholesterol in man and when elevated contributes to the development of coronary atherosclerosis. LDL-cholesterol is measured to assess risk for CHD and to follow the progress of patients being treated to lower LDL-cholesterol concentrations. Desirable levels of LDL-chol are those below 130 mg/dL in adults and 110 mg/dL in children. In NHANES 2001-2002, LDL-chol will be reported only for fasting participants >5 years of age (Sugiuchi et al. 1995).

Age and sex: Patients' age and sex were registered.

5. Result

During six month study period, total 135 patients with *diabetes mellitus* were evaluated for lipid profile, Creatinine level, blood pressure and HbA1C. Out of 135, diabetic patients 35 (26%) were males and 100 (74%) were female.according to study

All patients had type 2 *diabetes*. In concern with hyperlipidemia study result female patients (80%) are more prone to hyperlipidemia compared with male patients (68%). The mean age of type 2 diabetic male patients is 55.77±1.29 yrs, with range of 38-73 years and female patients was 47.80±1.30 yrs, ranged 25-68 years As per study result shows male patient ages ranging from (51-60) year are more susceptible to hyperlipidemia whereas female patients ages ranging from (41-50) year are more susceptible to hyperlipidemia. The fasting blood glucose of hyperlipidemic male patients (34%) are within the normal range (4.2-6.2).whereas hyperlipidemic female patients(27%) are in ranging from (8.3-10.2) and the post prandial blood glucose of hyperlipidemic male patients (50%) are within the high range (14.3-25).whereas hyperlipidemic female patients(52%) are in ranging from (14.3-25).The HbA1c of type-2 diabetic Male and Female patients in concern with Hyperlipidemia. In most cases male and female patients HbA1C (6-8).

According to the study of lipid profile, in male patients with type-2 diabetes 4(17%) had raised serum cholesterol (>200mg/dl) and 3(13%) patients have raised triglycerides in serum (>150mg/dl) [10]. Whereas, in female patients serum cholesterol level >200mg/dl was found in 11(14%) patients with type-2 diabetes and serum TG was raised in 16(20%) diabetics. In our study, serum TG levels were found to be much raised among diabetic females as compared to males. According to table-6 Serum TC,TG, LDL-C were significantly higher in diabetic females as compared to diabetic males, while Serum TC,TG, HDL-C was significantly higher in male. Blood pressure of male patients gradually more increases rather than female patients relate

with hyperlipidemia. Creatinine level of both male and female diabetic patients with hyperlipidemia are ranging from(0.6-1) which is in normal limit

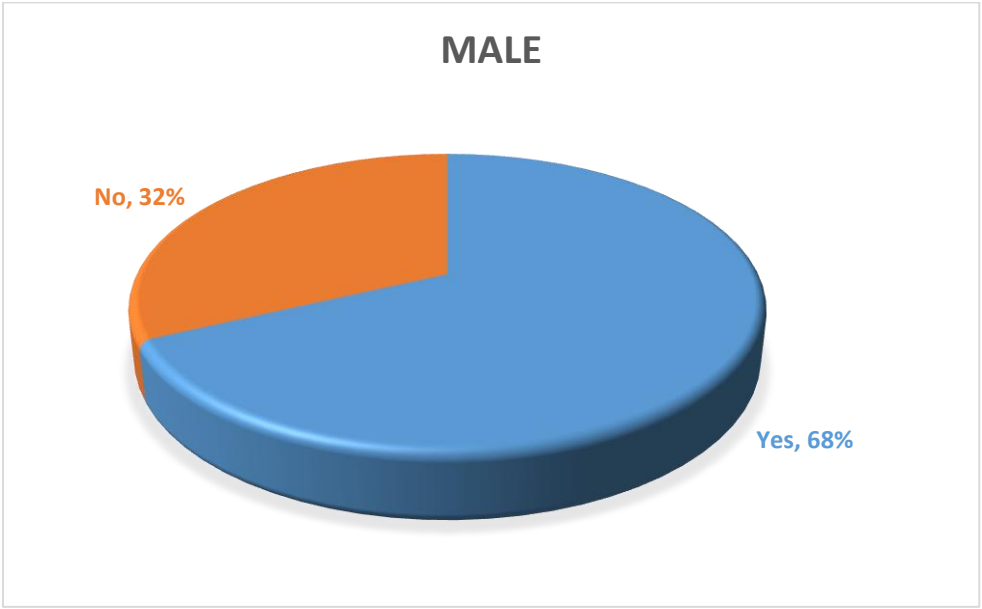
Table 1 Genaral Information of the study sample (N = 135)

Variable	**Frequency**	**Percent (%)**
Gender		
Male	35	26
Female	100	74
Age		
≤40 years		
Male	3	9
Female	27	27
>40 years		
Male	32	91
Female	73	73
Family History		
Male (Yes)	14	40
Male(No)	21	60
Female (Yes)	52	52
Female (No)	48	48
Physical Activity		
Male (Yes)	23	66
Male(No)	12	34
Female (Yes)	38	38
Female (No)	62	62
Daily Exercise		
Male (Yes)	20	58

Male(No)	15	42
Female (Yes)	27	27
Female (No)	73	73

Table 2 -Frequency of presence of hyperlipidemia in type-2 Diabetes mellitus as far as gender is concerned

	Yes	No	Total
Male	25(72%)	10(28%)	35(100%)
Female	80(80%)	20(20%)	100(100%)



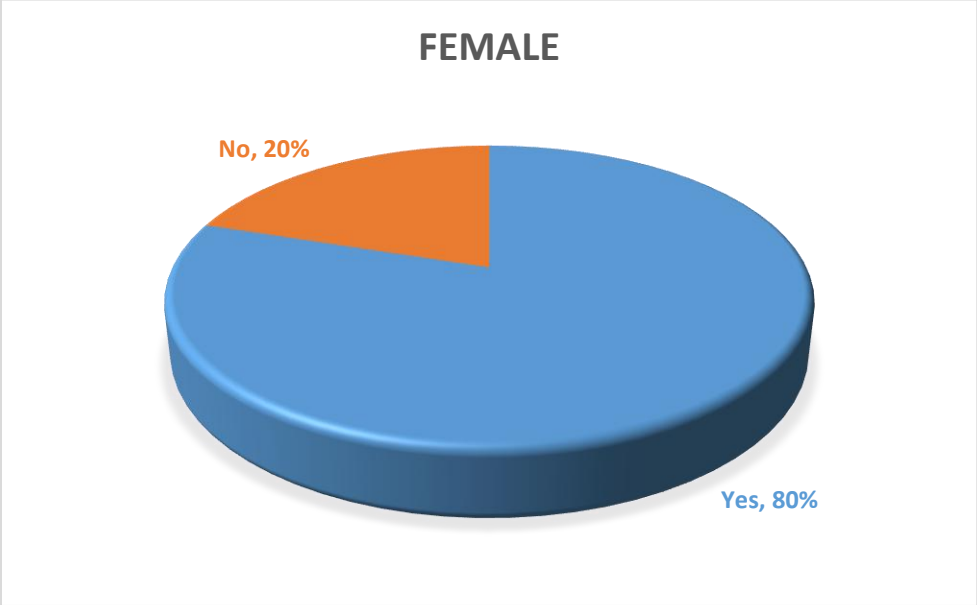


Figure-6 Frequency of presence of hyperlipidemia in type-2 Diabetes mellitus as far as gender is concerned

As per study result female patients (80%) are more prone to hyperlipidemia compared with male patients (68%).

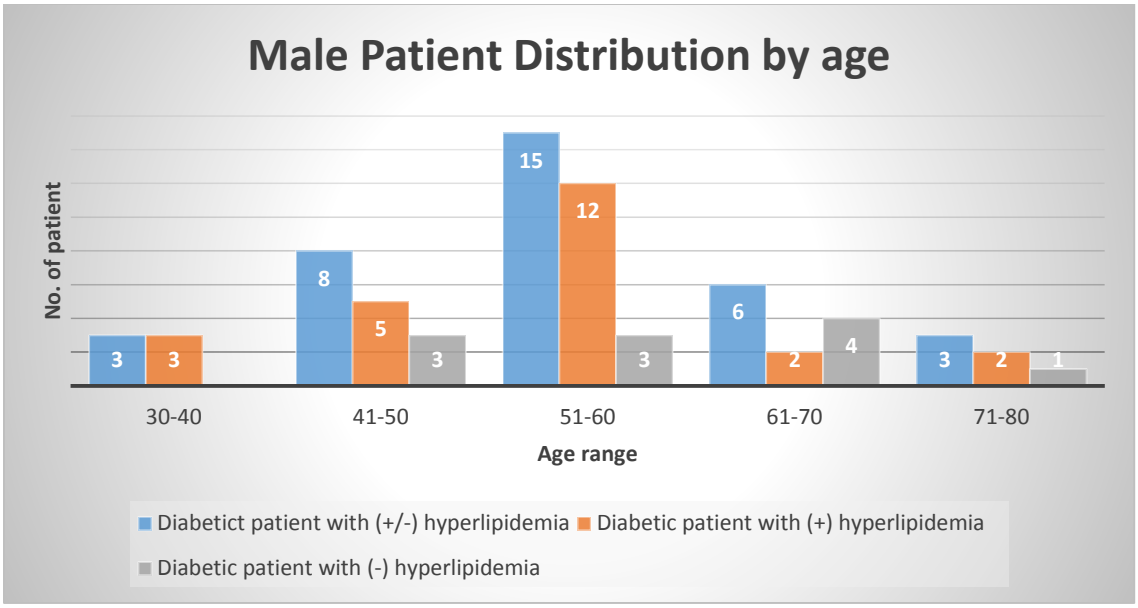
Table 3(a): Distribution of male patients according to age

Age	Diabetic patient with (+/-) hyperlipidemia	Diabetic patient with (+) hyperlipidemia	Diabetic patient with (-) hyperlipidemia
30-40	3	3	1
41-50	8	5	3
51-60	15	12	3
61-70	6	2	4

71-80	3	2	1
Grand Total	**35**	**24**	**11**

Table 3(b): Distribution of Female patients according to age

Age	Diabetic patient with (+/-) hyperlipidemia	Diabetic patient with (+) hyperlipidemia	Diabetic patient with (-) hyperlipidemia
30-40	27	20	7
41-50	34	30	4
51-60	29	28	1
61-70	10	7	3
Grand Total	**100**	**80**	**20**



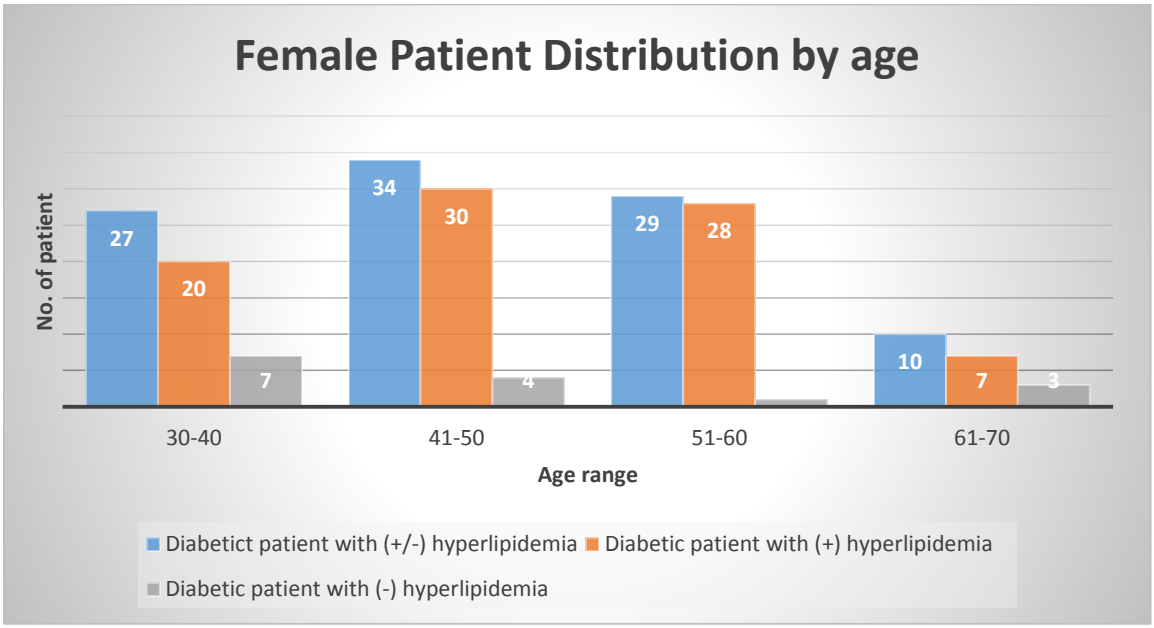


Figure 7: Distribution by age

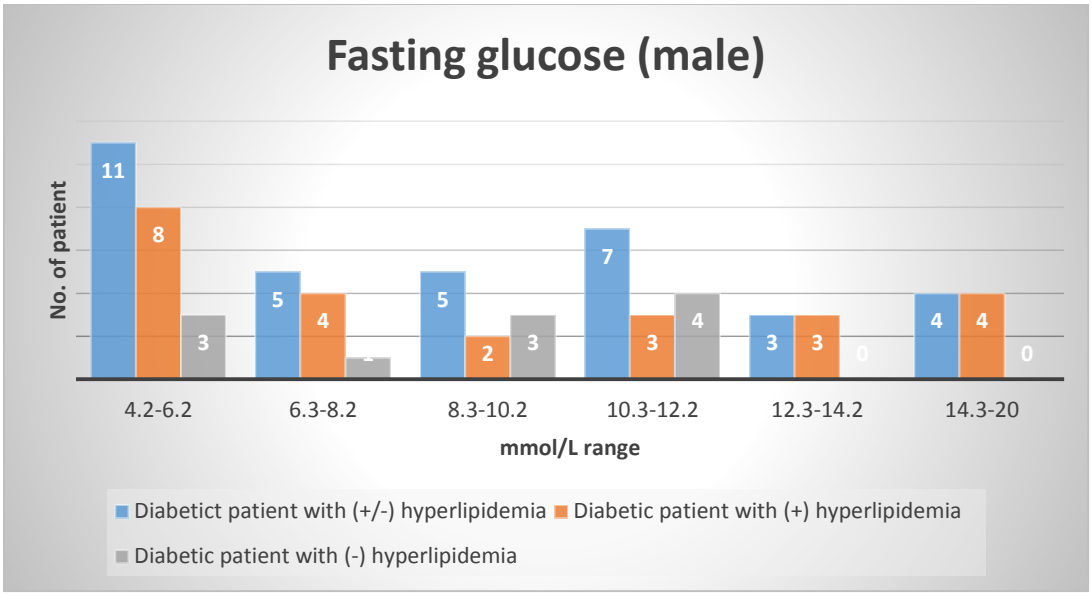
One hundred Thirty five type 2 diabetic subjects were enrolled in the study. The mean age of type 2 diabetic male patients is 55.82±1.29 yrs, with range of 38-73 years and female patients was 55.46±1.30 yrs, ranged 25-68 years. Of the diabetic patients 35 (26%) were males and 100 (74%) were females. Table 3(a)(b) shows age distribution of type 2 DM for male and female patient in concern with hyperlipidemia. As per study result shows male patient ages ranging from (51-60) year are more susceptible to hyperlipidemia whereas female patients ages ranging

from (41-50) year are more susceptible to hyperlipidemia. As per concern male patients are highly susceptible to hyperlipidemia by increasing with their age.

Table 4(a): Fasting blood glucose level of type-2 diabetic Male and Female patients in concern with hyperlipidemia

Male patient(fasting blood glucose)	4.2-6.2	6.3-8.2	8.3-10.2	10.3-12.2	12.3-14.2	14.3-20
Diabetic with (+/-) hyperlipidemia	**11**	**5**	**5**	**7**	**3**	**4**
Diabetic with (+) hyperlipidemia	**8**	**4**	**2**	**3**	**3**	**4**
Diabetic with (-) hyperlipidemia	3	1	3	4	0	0

Female patient(fasting blood glucose)	4.2-6.2	6.3-8.2	8.3-10.2	10.3-12.2	12.3-14.2	14.3-20
Diabetic with (+/-) hyperlipidemia	**10**	**17**	**28**	**15**	**15**	**15**
Diabetic with (+) hyperlipidemia	**7**	**15**	**21**	**12**	**14**	**11**
Diabetic with (-) hyperlipidemia	**3**	**2**	**7**	**3**	**1**	**4**



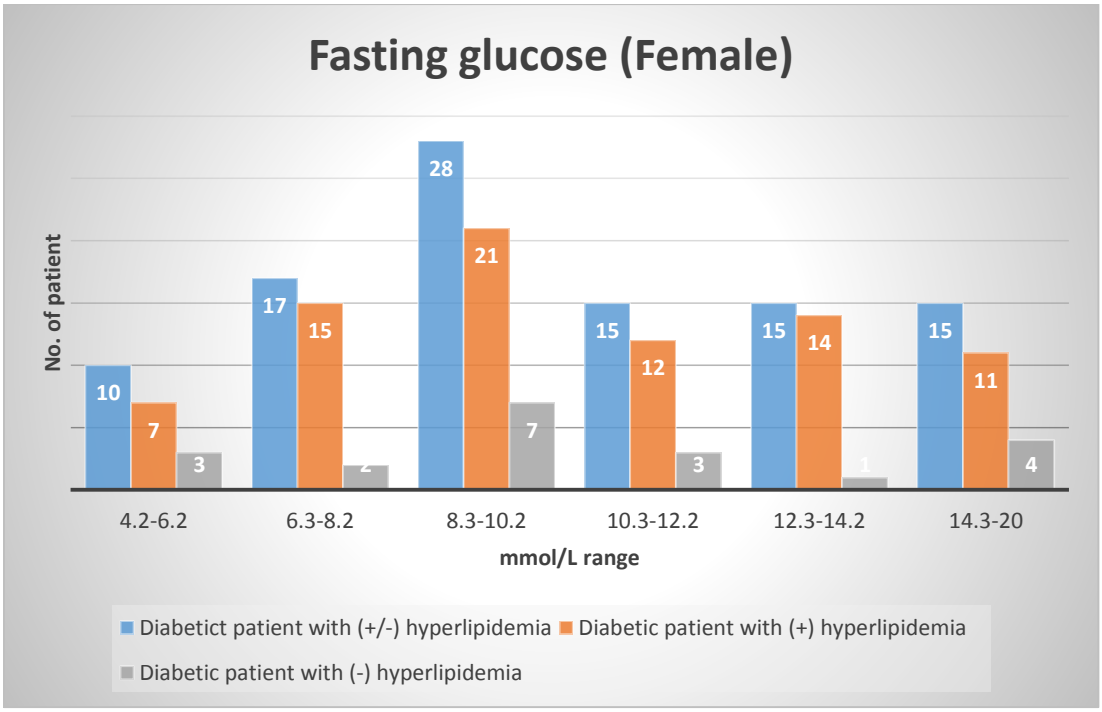
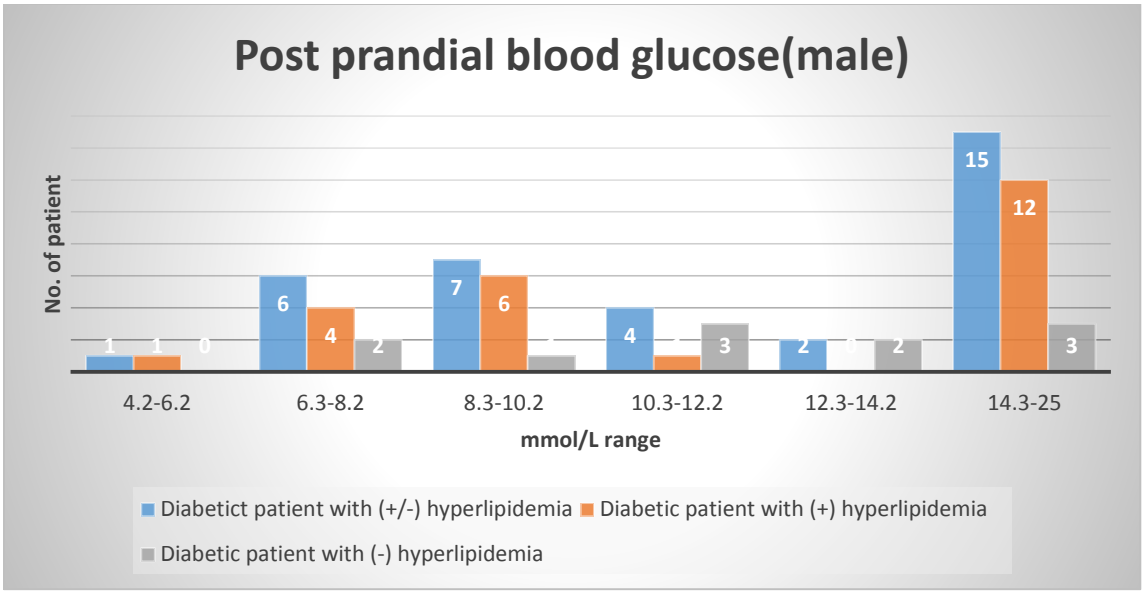


Figure 8: Fasting blood glucose level of type-2 diabetic Male and Female patients in concern with hyperlipidemia

Table 4(b): Post prandial blood glucose level of type-2 diabetic Male and Female patients in concern with hyperlipidemia.

Male patient(post prandial blood glucose)	4.2-6.2	6.3-8.2	8.3-10.2	10.3-12.2	12.3-14.2	14.3-25
Diabetict patient with (+/-) hyperlipidemia	**1**	**6**	**7**	**4**	**2**	**15**
Diabetic patient with (+) hyperlipidemia	**1**	**4**	**6**	**1**	**0**	**12**
Diabetic patient with (-) hyperlipidemia	**0**	**2**	**1**	**3**	**2**	**3**

Female patient(post prandial blood glucose)	4.2-6.2	6.3-8.2	8.3-10.2	10.3-12.2	12.3-14.2	14.3-25
Diabetict patient with (+/-) hyperlipidemia	**1**	**8**	**14**	**16**	**14**	**47**
Diabetic patient with (+) hyperlipidemia	**1**	**6**	**7**	**14**	**11**	**41**
Diabetic patient with (-) hyperlipidemia	**0**	**2**	**7**	**2**	**3**	**6**



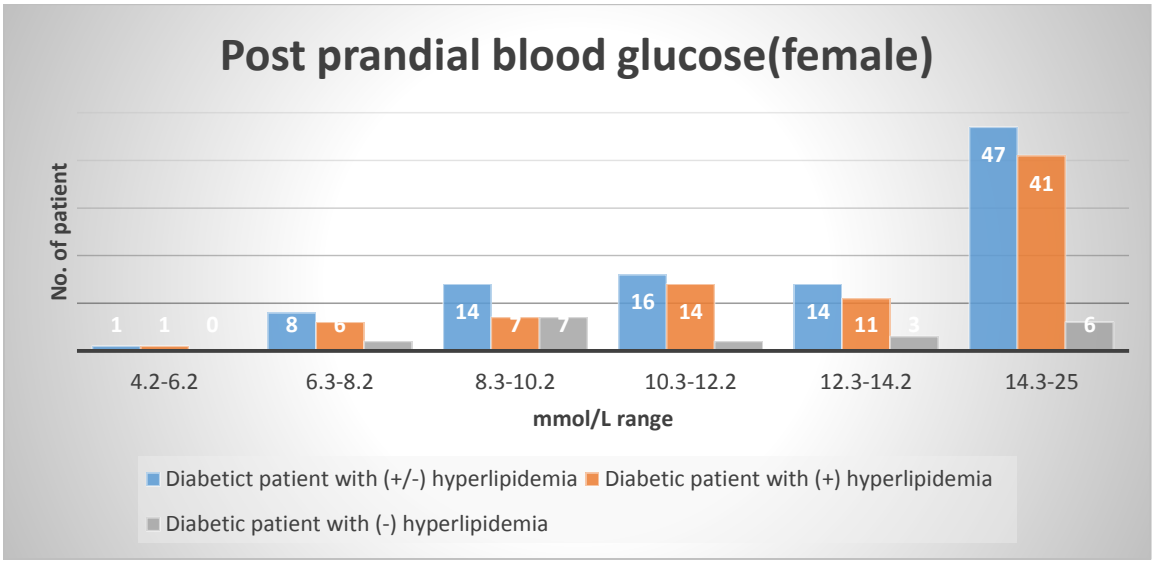


Figure 9: Fasting blood glucose level of type-2 diabetic Male and Female patients in concern with hyperlipidemia

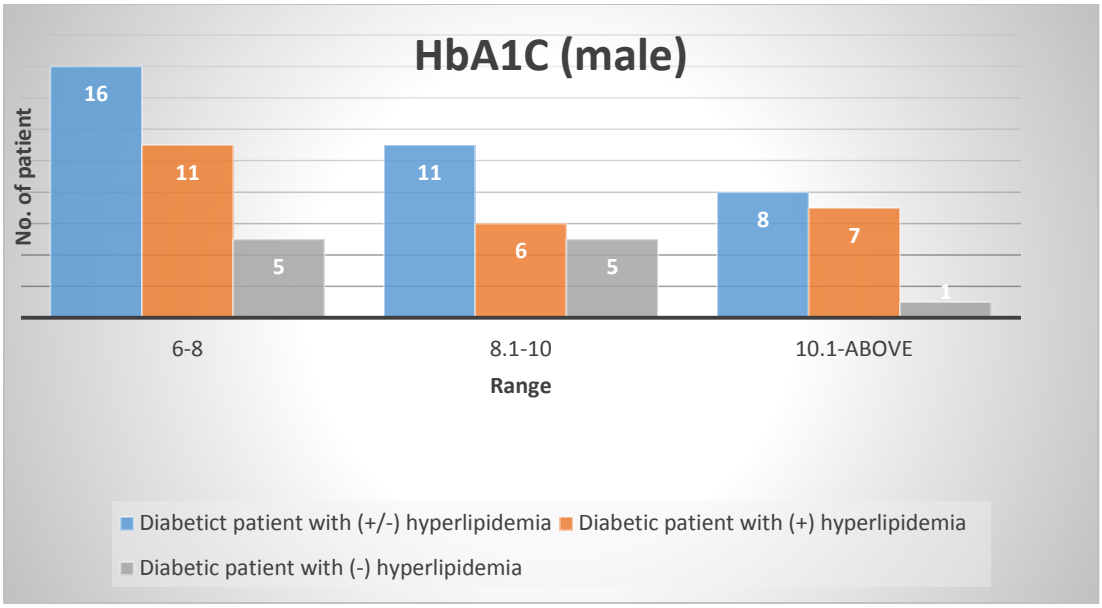
In most cases the fasting blood glucose of hyperlipidemic male patients (34%) are within the normal range (4.2-6.2).whereas hyperlipidemic female patients(27%) are in ranging from (8.3-10.2).As per results show hyperlipidemic female diabetic patients have higher fasting blood glucose in comparison to hyperlipidemic male diabetic patients

According to the study of post prandial blood glucose of hyperlipidemic male and female patients, the post prandial blood glucose of hyperlipidemic male patients (50%) are within the high range (14.3-25).whereas hyperlipidemic female patients(52%) are in ranging from (14.3-25).As per results show hyperlipidemic male and female diabetic patients both have higher post prandial blood glucose.Table-5 shows the HbA1c of type-2 diabetic Male and Female patients in concern with Hyperlipidemia. In most cases male and female patients HbA1C (6-8).

Table-5: HbA1c of type-2 diabetic Male and Female patients in concern with Hyperlipidemia

Male patients(HbA1C)	6-8	8.1-10	10.1-Above
Diabetic patient with (+/-) hyperlipidemia	**16**	**11**	**8**
Diabetic patient with (+) hyperlipidemia	**11**	**6**	**7**
Diabetic patient with (-) hyperlipidemia	**5**	**5**	**1**

Female patients(HbA1C)	6-8	8.1-10	10.1-Above
Diabetic patient with (+/-) hyperlipidemia	**45**	**39**	**16**
Diabetic patient with (+) hyperlipidemia	**35**	**32**	**13**
Diabetic patient with (-) hyperlipidemia	**10**	**7**	**3**



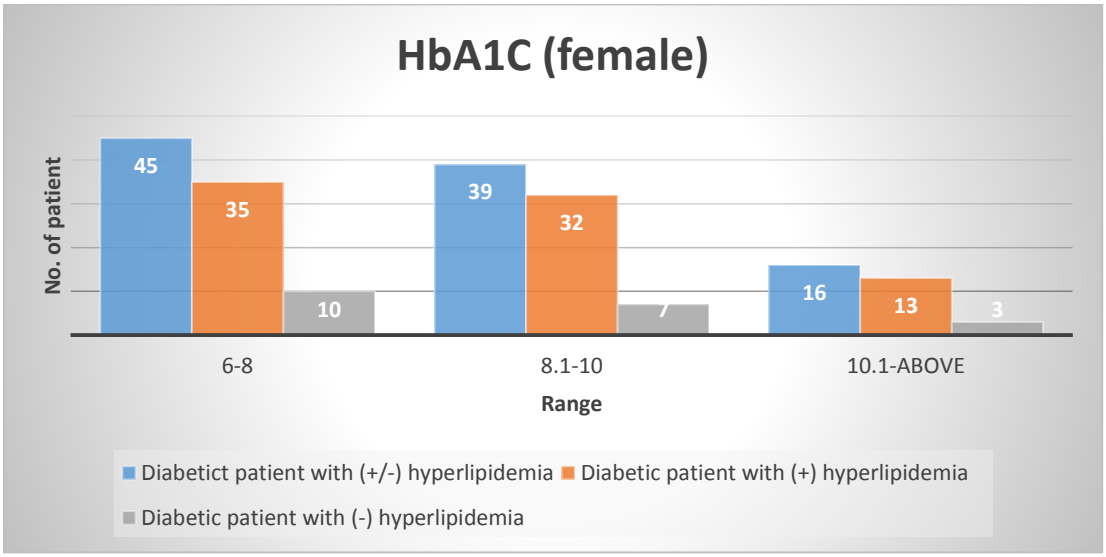


Figure 10: HbA1c of type-2 diabetic Male and Female patients in concern with Hyperlipidemia

Table-6: Lipid profile pattern of type-2 diabetic Male patients.

	Male	
LIPID PROFILE	N= 24	%
HIGH TOTAL CHOLESTEROL (mg/dl)	4	17
HIGH TRIGLYCERIDE (mg/dl)	3	13
HIGH TC,TG,LDL(low density lipoprotein) (mg/dl)	4	17
HIGH TC,TG,low HDL(high density lipoprotein) (mg/dl)	6	25
HIGH TC TG	2	9
HIGH TC,LDL	1	5
HIGH TG,HDL	1	5
TC,TG,LDL,HDL	3	9

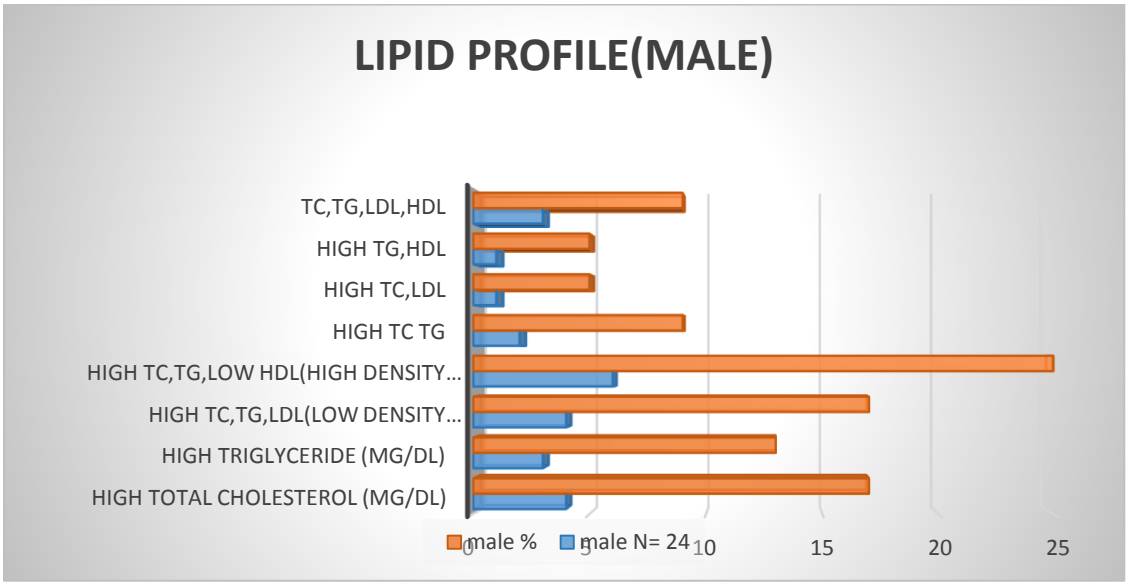


Table-7: Lipid profile pattern of type-2 diabetic Female patients.

	female	%
LIPID PROFILE(mg/dl)	N= 80	%
HIGH TOTAL CHOLESTEROL	11	14
HIGH TRIGLYCERIDE	16	20
HIGH TC,TG,LDL	20	25
HIGH TC,TG, low HDL	5	7
HIGH TC TG	10	13
HIGH TC,LDL	4	5
HIGH TG,HDL	3	4
TC,TG,LDL,HDL	11	14

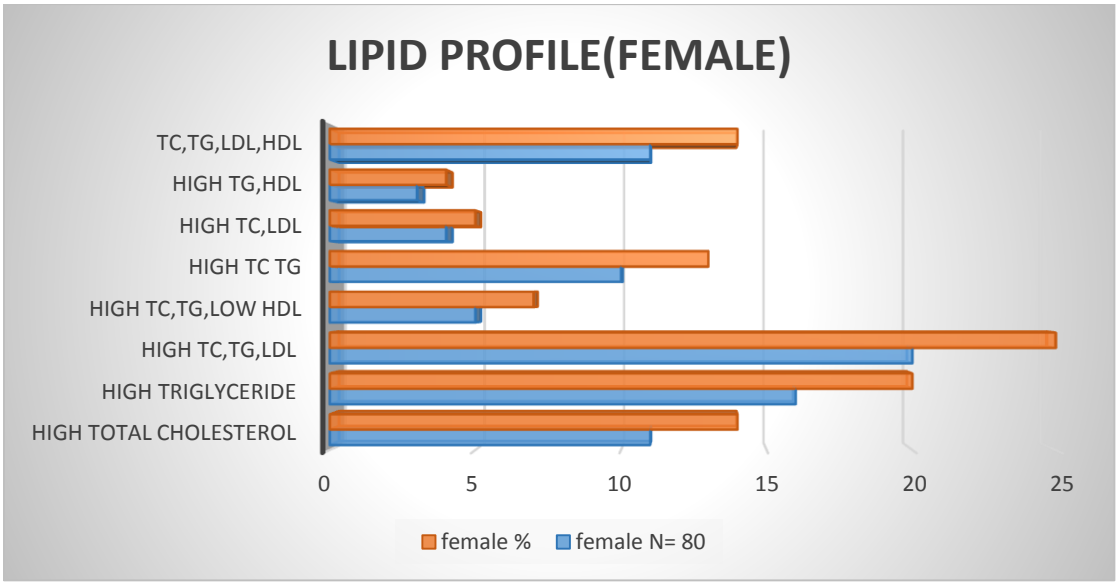


Figure 11: Lipid profile pattern of type-2 diabetic patient

According to the study,in male patients with type-2 diabetes 4(17%) had raised serum cholesterol (>200mg/dl) and 3(13%) patients have raised triglycerides in serum (>150mg/dl) [10]. Whereas, in female patients serum cholesterol level >200mg/dl was found in 11(14%) patients with type-2 diabetes and serum TG was raised in 16(20%) diabetics.In our study, serum TG levels were found to be much raised among diabetic females as compared to males.According to table-6 Serum TC,TG, LDL-C were significantly higher in diabetic females as compared to diabetic males, while Serum TC,TG, HDL-C was significantly higher in male.

Table-8: **Lipid profile(mean)**

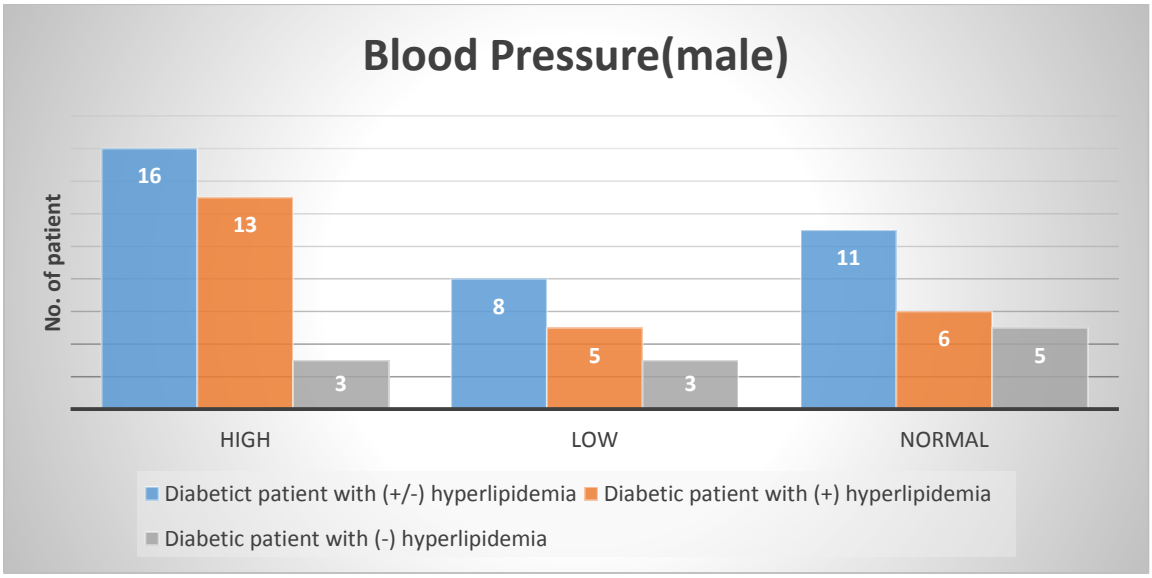
Lipid	Normal range	Male		Female	
		Study group	Mean	Study group	Mean
TC	<200	104-313	204.3 ± 56.0	115-348	207.0 ± 52.2
TG	<150	77-440	169.0 ± 72.9	76-1890	225.0 ± 189.1
HDL	45-65	26-53	42.0 ± 6.1	25-340	43.8 ± 31.1
LDL	<100	63-240	112.0 ± 40.1	38-382	111.3 ± 47.6

The mean of Total Cholesterol is 204.3 ± 56.0 of male patients and 207.0 ± 52.2 for female patients.The mean value for Triglyceride is 169.0 ± 72.9 and 225.0 ± 189.1 for male and female patients respectively. The mean value for HDL is 42.0 ± 6.1and 43.8 ± 31.1for male and female patients respectively. The mean of LDL is 112.0 ± 40.1of male patients and 111.3 ± 47.6for female patients

Table-9.Blood pressure of type-2 diabetic Male and Female patients in concern with Hyperlipidemia

Male patients	High	Low	Normal
Diabetic patient with (+/-) hyperlipidemia	**16**	**8**	**11**
Diabetic patient with (+) hyperlipidemia	13	5	6
Diabetic patient with (-) hyperlipidemia	3	3	5

Female patients	High	Low	Normal
Diabetic patient with (+/-) hyperlipidemia	**40**	**7**	**53**
Diabetic patient with (+) hyperlipidemia	32	6	42
Diabetic patient with (-) hyperlipidemia	8	1	11



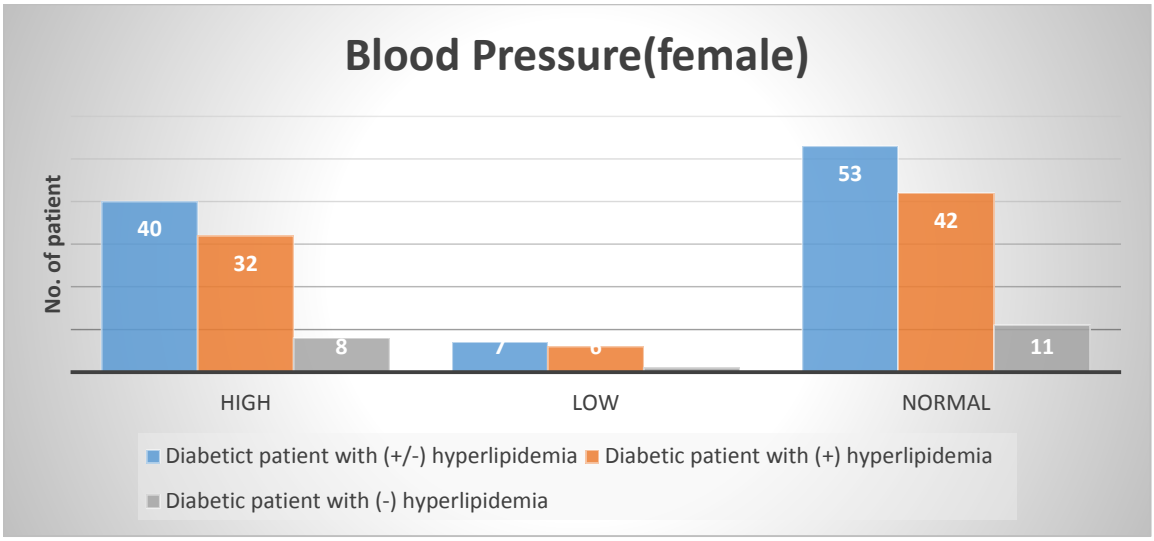


Figure 12: Lipid profile pattern of type-2 diabetic patient

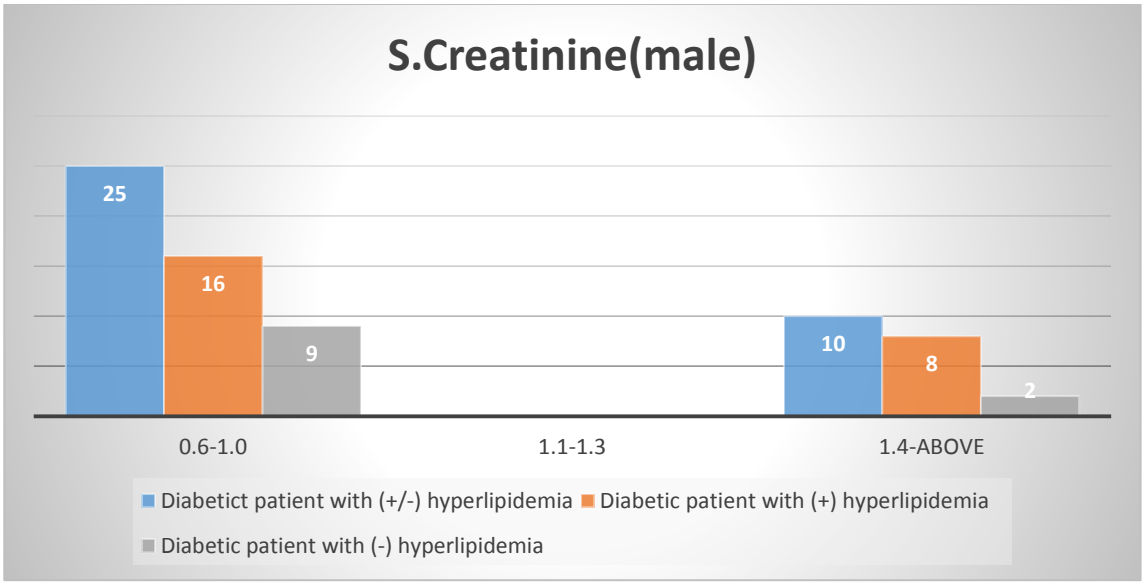
According to table-9 study reveals blood pressure of male patients gradually more increases rather than female patients relate with hyperlipidemia.

And in table-10 shows that creatinine level of both male and female diabetic patients with hyperlipidemia are ranging from(0.6-1)

Table 10: Creatinine level of type-2 diabetic Male and Female patients in concern with hyperlipidemia

Male Patient (S. Creatinine)	0.6-1.0	1.1-1.3	1.4-Above
Diabetic patient with (+/-) hyperlipidemia	**25**	**0**	**10**
Diabetic patient with (+) hyperlipidemia	**16**	**0**	**8**
Diabetic patient with (-) hyperlipidemia	**9**	**0**	**2**

Female Patient (S .Creatinine)	0.6-1.0	1.1-1.4	1.4-Above
Diabetic patient with (+/-) hyperlipidemia	**78**	**9**	**13**
Diabetic patient with (+) hyperlipidemia	**60**	**7**	**13**
Diabetic patient with (-) hyperlipidemia	**18**	**2**	**0**



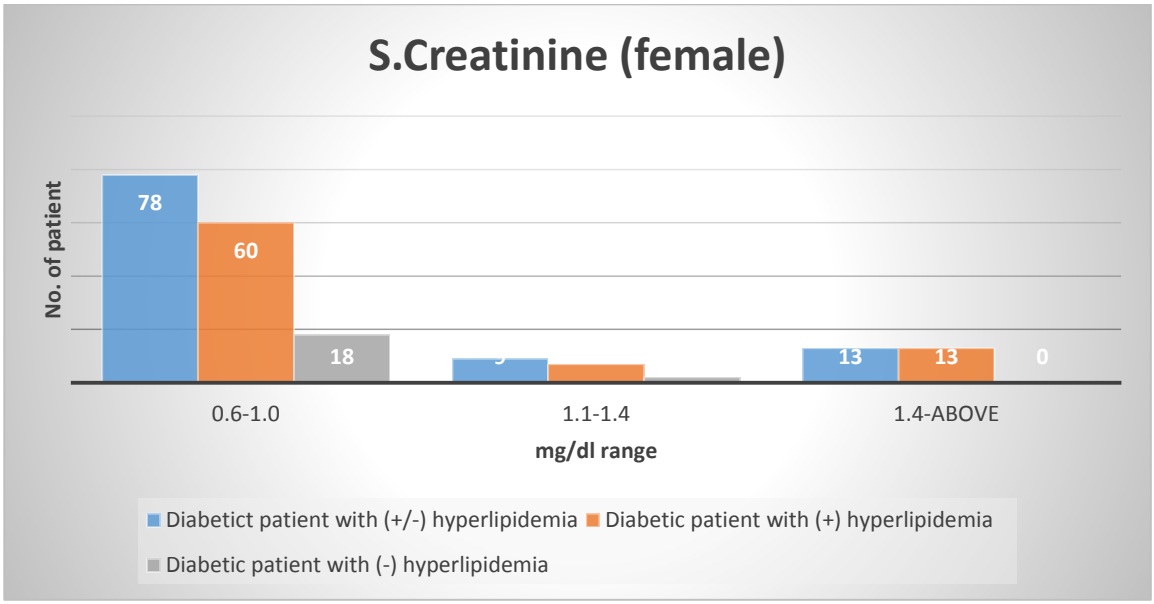


Figure 13: Creatinine level of type-2 diabetic Male and Female patients in concern with hyperlipidemi

Table-11: **Correlation studies between the blood glucose and serum lipid profile variables of diabetic patients**

	FBG	Creatinine	HbA1c	TC	TG	HDL	LDL
FBG	1						
Creatinine	-0.0006	1					
HbA1c	0.6676*	0.0308	1				
TC	0.0185	0.2797	0.1552	1			
TG	0.0184	0.289	0.1475	0.3448*	1		
HDL	-0.317	-0.0597	-0.3860*	-0.3704*	-0.3718*	1	
LDL	0.0466	0.1639	0.0523	0.6511*	0.3944*	-0.4223*	1

Note: (*) Correlation is significant at the 0.05 level.

As per study shows,

1. Fasting blood glucose shows a positive relationship with HbA1C with is significant.
2. TG shows a positive relationship with TC.
3. HDL shows a negative correlation with HbA1C and TG which is within significant level.
4. LDL showing a significant positive relation with TC & TG. And a negative relation with HDL.

6.Discussion

One hundred thirty five diabetic patients were subjected for the study made up of 100(74%) diabetics female and 35(26%) diabetic male. Of the diabetic patients male 25(72%) out of 35male patients and female 80(80%) out of 100 female patients are hyperlipidemic. Majority of the diabetic male hyperlipidemic patients (50%) were aged (51-60) and female hyperlipidemic patients (38%) aged (41-51). The age of diabetic male and female patients observed to be ≥40yrs confirmed earlier works that proves that age plays a significant role in the risk of developing hyperlipidemia in type 2 DM especially after 40yrs [26].According to study,40% male patients have diabetes in their family history and 52% female diabetes patients have family

fistory.In the present study, the results showed that the lipid profiles of the diabetics female were higher than that of the diabetics female. The absolute LDL concentration here is not altered significantly as it does not directly reflect the increased number of TG rich-small dense LDL particles which actually increase in number [29].

This study revealed that dyslipidemia was observed in the diabetic population, but that HDL and LDL were not significantly decreased and increased respectively. This study also showed that when the High TC, TG, HDL is comparatively higher in male patients whereas female patients show higher High TC, TG, LDL.

The high prevalence observed in this study could be attributed to urbanization in the population from the surrounding villages. Increasing urbanization has been observed to be associated with modernization of life style, which is largely characterized by physical inactivity, change in diet pattern and consequently development of obesity that is greatly considered as a risk factor for developing hyperlipidemia in type 2 DM. Our study revealed that (17%) type 2 male and (13%) type 2 female diabetics have high TC level. And (13%) male diabetic and (20%) female diabetic had high TG. High TC,TG, LDL was high in 25% male diabetic patients and High TC,TG, HDL was low in 25% female diabetics, which indicate that the diabetics are prone in future for developing cardiovascular, cerebrovascular complications.

The abnormal lipid profile observed in type 2 DM is said to be related to insulin resistance as reported in previous studies, which leads to increased release of free fatty acids from fatty tissue, impaired insulin dependent muscle uptake of free fatty acids and increase fatty acid release to the hepatic tissue [47] which has been closely associated with diabetic dyslipidemia, hypertension [48] and enormous risk to vascular diseases.

Chronic hyperglycemia causes glycation of apolipoproteins and interferes with the normal pathways of lipoprotein metabolism [49]. Free fatty acid levels especially from abdominal deposits with direct delivery to the liver, hyperinsulinemia, and hyperglycemia are all stimulators of VLDL-C production in the liver. Turnover of

plasma VLDL-C particles may be increased. The consequence may be elevation of plasma VLDL-C concentrations and reduction of plasma HDLC concentrations [50]. Furthermore, hepatic insulin resistance may result in increased lipoprotein secretion.

Management of high cholesterol in diabetes has improved in last few years and further hard work is required [54]. If awareness of complications of DM could be increased through media, then blood sugar levels can be tightly controlled resulting into good control of lipid levels which will result into less coronary artery disease and other complications.

7.Conclusion

In conclusion, hyperlipidemia is the commonest mellitus in patients of complication of diabetes mellitus and it predisposes to premature ahterosclerosis and macrovascular complications. Common lipid abnormalities in diabetic males are High TC,TG,low HDL and in female are High TC,TG and LDL.Therefore good glycaemic control can prevent development and progression of lipid profile.

Type 2 DM patients in this study had elevated levels of TG, TC with slightly elevated levels of LDL-C and reduced levels of HDL-C. This indicates the influence of type 2 DM on abnormal lipid profile of patients with its associated danger of elevated CVD risk. Serum TC, HDL-C and LDL-C were significantly higher in diabetic female in comparison to males, which is not so in controls, indicating gender influence on lipid in diabetics. TG was increased in most diabetic subjects,. Plasma glucose correlates positively with TG, while TC and LDL-C correlates positively with postprandial plasma glucose only.

Findings from this research will raise awareness on the need for routine lipid profile analysis and abnormal lipid analytes in clinic reviews and treatment of type 2 DM patients. Thus lipid profile analysis must be made an integral part of type 2 DM patients' clinical reviews. Type 2 DM and other diabetics must be educated on the risks they face as a result of their abnormal lipid levels and the necessary steps they need to manage it.

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A STUDY ON LIPID PROFILE PATTERN OF PATIENTS WITH *DIABETES MELLITUS AND RELATIONSHIP WITH BLOOD PRESSURE, SERUM CREATININE LEVEL AND HbA1c*

Questionnaire

A. Personal infomation

Name of the respondent _____

- Address _____
- Date

1. Sex

    1. Male
    2. Female

2. How old are you? _____ old

3. How tall are you? _____ years feet _____ inches

4. Weight? _____ pounds

5. What is your desired weight? What do you think would be a good, realistic weight for you? _____

6. What is your Religion?

    1 .Islam
    2. Hinduism
    3. Christianity
    4. Buddhism
    5. Others (Specify)

7. What is your Education level?

    1. Illiterate5
    2. Non-formal education
    3. Class 1-V
    4.Class VI-IX
    5.HSC or equivalent
    6.Graduate+

8. What is your Profession?

1. Farmer	3.Service	5.Unemployed
2. Businessman	4. Pension holder	6.Housewife

9. What is your marital status ?

1. Married and spouse lived together	4. Divorced
2. Spouse lived separately	5.Never married
3. Widow/Widower	

B. Diet Knowledge and Skills

Please answer all of the following questions about your eating. Place an "X" in the box that best describes you and your behavior.

During the past 3 months, how often did you:

1. Use the information about the number of calories in foods to make decisions about what to eat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
2. Use information about the of carbohydrates in foods to make decisions about what to eat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
3. Use information about the number of grams of fat in foods to make decisions about what to eat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
4. Deliberately skip a meal or snack to cut calories or fat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
5. Deliberately take small portion sizes to cut calories, sugar or fat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day

6. Use low-calorie, lite, reduced-fat, or fat-free products?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
7. Use sugar free or reduced sugar products?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
8. Resist the temptation to eat a food you want because it is too high in fat, sugar, or calories?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day
9. Use a written diet or meal plan to decide what foods to eat?	Never	1 time per month or less	2-3 times per month	1-2 times per week	4-6 times per week	1 or more times per day

C. Knowledge regarding risk factor

1. Smoking : Non-smoker Ex-smoker Smoker

2. Per day Smoking : 2-3 4-5 many

3. Any type of addiction : Tea Coffee Betel nuts Betel leaf Others

4. Overweight/Obesity present : Yes No

5. Are you physically active? Active Highly active inactive Highly inactive

6. Alcohol consumption ? Yes No

7. Do you have hypertension ? Yes No

8. Are you taking fruits and vegetables? A little A lot Moderate

9. Family history of Diabetes ……..

10. Family history of High lipid profile…….

9. Disease faced any……

1. Hypertension 2. Osteoporesis 3. CKD

4. Asthma 5. Dyslipidemia 6.Heart failure

7. Stroke

D. Disease information

1. Type of diabetes Type-1 Type-2 Gestational

2. How old were you when you were first diagnosed with diabetes? I was ……..Years old

3. Physical problem faced (symptoms)

1.

2.

3.

4. How long do you face diabetes…….

5. How long do you face dyslipidemia…….

6. Patients status….outpatients inpatients

7. Length of hospital stay…..

E. Medication use

1. What type of medicine are you taking ? Tablet Insulin Mixed Other

2. How often are you supposed to take these pills?

_____ I do not take pills for my diabetes

_____ Occasionally as needed

_____ Once per day

_____ Twice per day

_____ Three or more times per day

3. What is your drug name and regimen? ……

4. Has your doctor prescribed insulin shots for your diabetes?

_____ Yes

_____ No

5. How often are you supposed to take insulin?

_____ I don't take insulin

_____ Occasionally as needed

_____ Once a day

_____ Twice a day

_____ Three or more times a day

F. Blood glucose monitoring

1. How often do you actually test your blood glucose?

_____ I have not been told to test my blood glucose

_____ Occasionally as needed

_____ A couple times a month

_____ 1 or 2 times a week

_____ 3 to 6 times a week

_____ Once a day

_____ Twice a day

_____ 3 or 4 times a day

_____ 5 or more times a day

G. Physical Activity

1. Has your doctor advised you to get more exercise?

_____ Yes

_____ No

_____ Don't know

2. How active is your daily routine? How much physical activity do you get as a result of going to work, shopping, housework, yard work, and other daily activities?

_____ Very inactive

_____ Inactive

_____ A little activity

_____ A moderate amount of activity

_____ Active

_____ Very active

3. How often do you set aside time to exercise. How often do you do something physically active like walking, running, cycling, going to the gym or participating in sports?

_____ I never exercise

_____ A couple times a month

_____ 1 or 2 times a week

_____ 3 to 4 times a week

_____ 5 to 6 times a week

_____ Once a day

_____ More than once a day

H. Investigations

1. Height(cm)

2. Weight(kg)

3. Blood group

4. Systolic Blood Pressure……..

5. Diastolic Blood Pressure……

6. Glucose,fasting……..

7. Glucose,2 hrs ABF……...

8. Creatinine(Blood)…….

9. HbA1C……..

10. Serum Lipid Profile

10.1. Total Cholesterol…….

10.2. Triglyceride……

10.3. HDL-Cholesterol mg/dl…….

10.4. LDL-Cholesterol…..

11. Serum electrolyte……..

1. EL-Na+……..m.eq/l

2. EL-K+…….m.eq/l

3. EL-CL-……..

4. EL-CO2……..

12. ALT (SGPT)……..

13. AST (SGOT)……

14. BMI……