A PROSPECTIVE STUDY ON PRESCRIBING PATTERNS OF ANTI-DIABETIC DRUGS FOR PATIENTS WITH TYPE- II DIABETES MELLITUS AT A TERTIARY CARE HOSPITAL

A Dissertation submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI- 600 032

In partial fulfilment of the award of the degree of

MASTER OF PHARMACY

IN

Branch - VII – PHARMACY PRACTICE

Submitted by

Name: Mr. RETHINAM. A

REG.No. 261740208

Under the Guidance of

Dr. R. Kameswaran, M.Pharm., Ph.D.,

DEPARTMENT OF PHARMACY PRACTICE

J.K.K.NATTRAJA COLLEGE OF PHARMACY

KUMARAPALAYAM – 638183

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CERTIFICATES

EVALUATION CERTIFICATE

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Internal Examiner **External Examiner**

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Dr. R. Kameswaran, M.Pharm., Ph.D.,
Guide, Department of Pharmacy Practice

Dr. N. Venkateswaramurthy, M.Pharm., Ph.D.,
HOD, Department of Pharmacy Practice

Dr. R. Sambathkumar, M.Pharm., Ph.D.,
Principal

CERTIFICATE

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Place: Kumarapalayam

Date:

Dr. R. SambathKumar, M.Pharm., Ph.D.,
Principal,
J.K.K.Nattraja College of Pharmarcy,
Kumarapalayam – 638 183,
Tamil Nadu

CERTIFICATE

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Place: Kumarapalayam
Date:

Dr. N. Venkateswaramurthy, M.Pharm., Ph.D.,
Professor & Head,
Department of Pharmacy Practice,
J.K.K.Nattraja College of Pharmarcy,
Kumarapalayam – 638 183,
Tamil Nadu

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Place: Kumarapalayam
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Assistant Professor,
Department of Pharmacy practice,
J.K.K.Nattraja College of Pharmacy.
Kumarapalayam- 638 183.

DECLARATON

I do hereby declared that the dissertation **"A PROSPECTIVE STUDY ON PRESCRIBING PATTERNS OF ANTI-DIABETIC DRUGS FOR PATIENTS WITH TYPE- II DIABETES MELLITUS AT A TERTIARY CARE HOSPITAL"** submitted to "**The Tamil Nadu Dr. M.G.R Medical University - Chennai",** for the partial fulfilment of the degree of **Master of Pharmacy** in **Pharmacy practice,** is a bonafide research work has been carried out by me during the academic year 2018-2019, under the guidance and supervision of **Dr. R. Kameswaran, M. Pharm., Ph.D.,** Assistant Professor, Department of Pharmacy practice, J.K.K.Nattraja College of Pharmacy, Kumarapalayam.

I further declare that this work is original and this dissertation has not been submitted previously for the award of any other degree, diploma, associate ship and fellowship or any other similar title. The information furnished in this dissertation is genuine to the best of my knowledge.

Place: Kumarapalayam **Mr. RETHINAM. A**

Date: **Reg.no. 261740208**

Dedicated to Parents,

Teachers &

My Family

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Mr. RETHINAM. A

Reg.no.261740208

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LIST OF ABBREVIATIONS

DM	Diabetes Mellitus
TIDM	Type - I diabetes mellitus
T2DM	Type - II diabetes mellitus
BMI	Body mass index
NKT	Natural killer T cells
DC	Dendritic cells
IR	Insulin resistance
GLP-1	Glucagon like peptide one
DPP-4	Dipeptidyl peptidase-4
ACE	Angiotensin-converting enzyme
ARBs	Angiotensin II Receptor Blockers
AGEs	Advanced glycation end products
DR	Diabetic retinopathy
ADA	Americans with Disabilities Act
AACE	American Association of Clinical Endocrinologists
GI	Gastrointestinal
HGP	Hepatic glucose production;
MET	Metformin

QR	Quick release
SGLT2i	Sodium glucose cotransporter 2 inhibitor
TZD	Thiazolidinedione
AACE/ACE	American Association of Clinical Endocrinologists/ American College of Endocrinology
ADA	American Diabetes Association
GU	Genitourinary
HbA1c	Glycated hemoglobin
HF	Heart Failure
HYPO	Hypoglycemia
SU	Sulfonylurea

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1. INTRODUCTION

1.1. DIABETES MELLITUS

Diabetes Mellitus, defined as a chronic metabolic disorder characterized by, disturbance in carbohydrate, protein and lipid metabolism caused by an absolute or relative deficiency of insulin and insulin resistance.[1] Diabetes mellitus affects more than 194 million people worldwide, and there is a chance of increasing to 333 million by the year 2025. The highest probability of cases being seen in developing countries, diabetes is nowadays, addressed as a global problem. Furthermore, since India has the highest number of diabetic patients, it is considered as the "diabetic capital of the world".[2] It was predicted that by 2030 diabetes mellitus may affect about 79.4 million patients in India.[3]

1.2. ETIOLOGICAL CLASSIFICATION

1.2.1. Type - I Diabetes Mellitus:

Type - I diabetes mellitus (TIDM) comprises several diseases of pancreatic β cells which lead to an absolute insulin deficiency. This is usually the result of an autoimmune destruction of the pancreatic β cells (type I A). Some patients with TIDM with no evidence of β cell autoimmunity have underlying defects in insulin secretion often from inherited defects in pancreatic β cell glucose sensing.[3]

1.2.2. Type - II Diabetes Mellitus:

Type - II diabetes mellitus (T2DM) is by far the more common type of diabetes and is characterized by insulin resistance resulting from defects in the action of insulin on its target tissues (muscle, liver, and fat), but complicated by varying and usually progressive failure of β cells' insulin secretory capacity. Most patients with T2DM in the US and Europe are obese, however, in India and China, most T2DM patients have a lean body mass index (BMI), albeit

with increased visceral fat. Thus we have come to the view that obesity is often a result of insulin resistance rather than the cause.[4]

1.3 EPIDEMIOLOGY

Diabetes is a chronic metabolic disorder associated with significant morbidity and mortality affecting almost 6.2% of the world population. Type-2 diabetes is a disease marked by high levels of blood glucose due to the action of insulin and insufficient insulin production. Type-2 diabetes accounts for approximately 90% to 95% of all diagnosed cases of diabetes. Throughout the world, 9% of adults suffer from diabetes, 90% of whom are affected by type 2 diabetes mellitus (T2DM). Each year, diabetes is involved in approximately 1.5 million deaths.

The number of people affected with diabetes mellitus has increased from 108 million in 1980 to 422 million in the year 2014. The global prevalence of diabetes among adults over 18 years of age has increased from 4.7% in 1980 to 8.5% in 2014. The prevalence of diabetes in Tamil Nadu is 10.4%. The prevalence of diabetes mellitus has been rising more rapidly in middle and low-income countries. The chronic and uncontrolled diabetes mellitus is considered, a major cause of blindness, renal failure, heart attack, stroke, and lower limb amputation.[5]

1.4 ETIOLOGY

Type – 2 diabetes is characterized by insulin resistance and a progressive decline in pancreatic β cell insulin production. There is no autoimmune-mediated pancreatic β cell damage and most patients with type 2 diabetes do not need insulin during the initial stages of the disease.

Insulin resistance is a condition in which insulin is produced, but is not used properly: a given amount of insulin does not produce the expected result. In people who are obese it may be that the chronic inflammation associated with obesity affects the function of the insulin receptors on the cells in the liver, muscles, etc., decreases the number of insulin receptors, affects insulin signaling pathways, or inactivates insulin receptors.[6]

The progressive decline in pancreatic β cell function is due to decreased β cell mass caused by apoptosis[7]; this may be a consequence of aging, genetic susceptibility, and insulin resistance itself. The etiology of type 2 diabetes is complex and involved genetic and lifestyle factors

- Genetic factors: There are susceptibility genes that play a role in the development of type - 2 diabetes, but their contribution appears to be small. The effect of the known, common gene variants in creating a predisposition to type 2 diabetes is approximately 5%-10%, so unlike some inherited diseases, being homozygous for these susceptibility genes does not typically result in a case of type 2 diabetes unless certain environmental (in this case lifestyle) factors are present.
- Lifestyle factors/demographics: Obesity is a major risk factor for the development of type 2 diabetes,[8] and the greater the degree of obesity, the higher the risk.[9] Excess adipose tissue is typically in a state of chronic inflammation, and this inflammation is thought to cause insulin resistance in the adipose tissue and other organs. Other factors that increase the risk of developing type - 2 diabetes are the presence of the metabolic syndrome, age, and a sedentary lifestyle. Type - 2 diabetes is much more common in African-Americans than other ethnic groups. There may be a genetic explanation for this, but socio-economic factors are probably to blame.[10]

1.5. CLINICAL MANIFESTATION

1.5.1. COMMON SYMPTOMS OF DIABETES

Symptoms of diabetes don't usually present themselves clearly, which may lead to either misdiagnosis or getting diagnosed only when the disease is already in an advanced stage. If you are at risk of developing either type of diabetes, some of the symptoms that you should look out for include the following:

- **Increased urination or polyuria —** Because of the inefficient glucose uptake, the kidneys are forced to work overtime and try to absorb as much glucose as it can to filter out. This then leads it to leach fluids from other body systems, such as urine.
- **Fatigue and tiredness —** The imbalance in glucose levels can lead to incorrect energy allotment, which can trigger fatigue and make diabetes patients perpetually tired, even if they follow a healthy sleep pattern and get adequate food intake.
- **Muscle weakness —** Neuropathy brought on by diabetes may affect muscle strength and balance. About 60 to 70 percent of diabetes patients suffer from this nerve damage, which may manifest in different parts of the body.
- **Slow-healing sores —** The high glucose levels in the body slow down the healing process by obstructing proper blood circulation, which is essential for skin repair. This can also heighten the risk of wound infections.
- **Blurred vision —** If you notice that your eyesight is getting blurry, this does not necessarily mean that your eye health has deteriorated. The temporary blurred vision brought on by diabetes is commonly attributed to the high blood glucose levels that may cause the lens of the eyes to swell.

1.5.2. RARE AND OFTEN OVERLOOKED SYMPTOMS OF DIABETES

Aside from the common symptoms of diabetes mentioned above, some tell-tale symptoms are often overlooked by diabetics. These symptoms are commonly attributed to other possible health conditions instead of diabetes.

- **Skin changes —** Poor blood glucose levels can have an impact on skin health by triggering bacterial and fungal infections. Other skin conditions that diabetes may cause include skin tags, digital sclerosis, and blisters.

- **Excessive sweating —** Hyperhidrosis may be observed in people with diabetes because of possible neuropathy. Low blood glucose may also cause excessive sweating by triggering your fight-or-flight response.
- **Yellow nails —** The effect of diabetes on metabolic processes and nutrient breakdown can affect the production of collagen, which can cause yellowing in the nails.[11]

1.6. PATHOGENESIS OF TYPE - I DIABETES MELLITUS

The process of destruction of β cells is chronic in nature, often beginning during infancy and continuing over the many months or years that follow. At the time of clinical diagnosis of TIDM, about +80% of the β cells have been destroyed, the islets are infiltrated with chronic inflammatory mononuclear cells (insulitis), including CD8+ cytotoxic T cells. Once islet cell autoimmunity has begun, progression to islet cell destruction is quite variable, with some patients rapidly progressing to clinical diabetes, while others remain in a neuro progressive state.

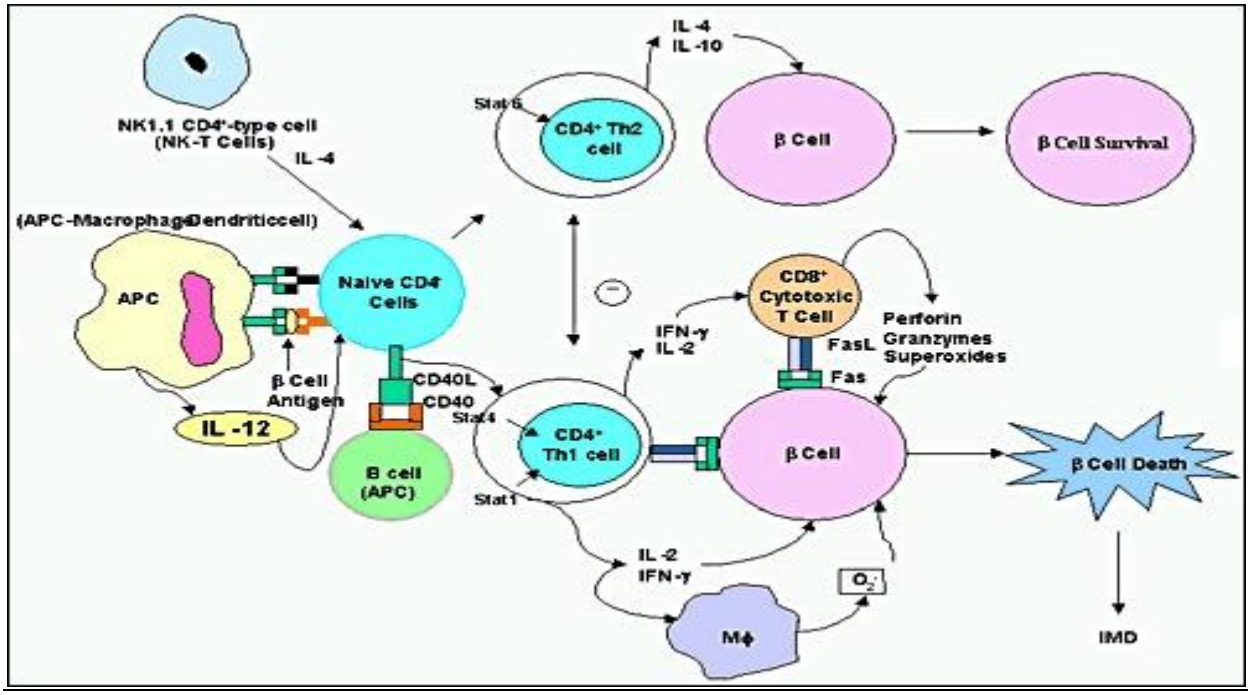


Figure 1: The pathogenesis of islet cell destruction

Islet cell proteins are presented by antigen-presenting cells (APCs) to naïve Th0 type CD4+ T cells in association with MHC class II molecules. Interleukin (IL)-12 is thus secreted by APCs

that promotes the differentiation of Th0 cells to Th1 type cells. Th1 cells secrete IL-2 and IFN-γ that further stimulate CD8+ cytotoxic T cells or macrophages to release free radicals (superoxide) or perforin/granzymes, leading to ß cell apoptosis or death. CD8+ cytotoxic T cells further mediate ß cell death by Fas-mediated mechanisms. Interleukin (IL)-4, on the other hand, secreted mainly by natural killer T (NKT) cells drives Th0 cell to Th2 pathway leading to benign insulitis.[12]

Diabetes risk and time to diabetes in relatives of patients directly correlates with the number of different autoantibodies present as already discussed. The pathogenesis of T1DM has been extensively studied, but the exact mechanism involved in the initiation and progression of β-cell destruction is still unclear. The presentation of beta cell-specific autoantigens by antigen-presenting cells (APC) [macrophages or dendritic cells (DC)] to CD4+ helper T cells in association with MHC class II molecules is considered to be the first step in the initiation of the disease process. Macrophages secrete interleukin (IL)-12, stimulating CD4 + T cells to secrete interferon (IFN)-γ and IL-2. IFN-γ stimulates other resting macrophages to release in turn, other cytokines such as IL-1β, tumor necrosis factor (TNF-α) and free radicals, which are toxic to pancreatic β-cells. During this process, cytokines induce the migration of β-cell autoantigen specific CD8+ cytotoxic T cells. On recognizing specific autoantigen on ß cells in association with class I molecules, these CD8+ cytotoxic T cells cause ß cell damage by releasing perforin and granzyme and by Fas-mediated apoptosis of the beta cells. Continued destruction of beta cells eventually results in the clinical onset of diabetes.[13]

1.7. PATHOGENESIS OF TYPE - II DIABETES MELLITUS

T2DM is characterized by insulin resistance in peripheral tissues (muscle, fat, and liver) with progressive β cell failure, ongoing loss of insulin secretory capacity with defective insulin secretion in response to a glucose stimulus, increased glucose production by liver, and no pancreatic autoimmunity.[14]

Table 1: Pathophysiologic Factors.	
Obesity/Insulin resistance (IR)	There is a significant increase in risk.
Intrauterine environment	There is a significant increase in risk.
Gestational diabetes	Studies in Pima Indian women showed a significant increased risk of developing T2DM in offspring of women with diabetes during pregnancy compared to non-diabetic mothers.
Ethnicity	There is a significant increased risk in certain ethnic/race groups.[15]
Gender and puberty	Puberty is a state of IR: There is a 30% decrease in insulin sensitivity and a compensatory increase in insulin secretion. The mean age at diagnosis of T2DM in children is 13.5 years, corresponding to the time of peak adolescent growth and development. Girls are 1.5-3 times more likely than boys to develop T2D as children or adolescents.[16]
Family History	Between 74-100% 74-100% of children with T2DM have a first or second degree relative with T2DM. The lifetime risk is 40% if one parent affected and 70% if both parents affected.[17]
Genetics	Genome-wide studies led to the discovery of single-nucleotide polymorphisms (SNPs) at several loci. To date, more than 30 diabetes-related SNPS (diabeto SNPs) have been identified. Several genes are associated with [18]

	1. Peroxisome proliferator-Activated receptor-γ2 (*PPAR*-γ2) Gene: An important regulator of lipid and glucose homeostasis. Missense mutation Pro12Ala in *PPAR*-γ2 is associated with decreased risk for T2DM.[19] 2. *Kir6.2 Gene (KCNJ11):* The missense mutationGlu23Lys in the *Kir6.2* gene has been associated with increased risk of T2DM.[20] 3. MODY genes (*HNF4*a *and HNF1β*) 4. Transcription Factor 7-like *(TCF7L2)* Gene: A product of HMG box-containing transcription factors that play a role in the glucose homeostasis. Specific polymorphysms in the *TCF7L2* gene increase the risk of progression from IGT to T2DM.[21] 5. Calpain-10 Gene: Calpains are Ca+2 dependent cysteine proteases and play a role in regulating insulin secretion and action.[22]

The natural history of progression to T2DM is that a person with IRS begins to decompensate, with a fall in the disposition index (the amount of insulin produced for the degree of insulin resistance).[23] Subsequently, fed levels of blood glucose rise, followed by elevations in fasting blood glucose levels rising later. At this early stage, diet, exercise, and insulin sensitizers are indicated. Later still, when the ability to secrete insulin becomes disabled, the addition of insulin secretagogues such as sulfonylureas, meglitinides and/or incretin mimetics need to be added to the therapeutic plan.[24] Whereas the glucagon-like

peptide one (GLP-1) analog exenatide (Byetta) given by subcutaneous injection twice daily before food will lower blood glucose levels and compliment metformin in provoking weight loss, it should be reserved for more severely diabetic adults and teenagers who have become unresponsive to diet and exercise programs.

It has not yet been recommended by the FDA for use in children. Sitagliptin (Januvia) blocks the dipeptidyl peptidase-4 (DPP-4) enzyme preventing it from inactivating GLP-1, thus prolonging the action of GLP-1 once induced by a meal.[25] Whereas the latter agent is in general weight neutral, it can be of adjunctive help in lowering hyperglycemic excursions and at least in rodents, may induce β cell replication over time. When these additional agents also fail to maintain near normoglycemia, then insulin should be given instead of the secretagogues. The typical dyslipidemia associated with IRS and T2DM should be treated by a reduced intake of animal fat and a fibrate such as gemfibrozil. However, those patients who have prominent elevations in LDL-cholesterol and should be treated by a statin. The mixed-use of a statin and a fibrate should be undertaken cautiously since the risks of muscle necrosis (rhabdomyolysis) with renal failure has been reported more with some combinations than with others. Hypertension, when present, should be aggressively treated, preferably with ACE and ARBs at least initially.[26]

1.8. COMPLICATIONS OF DIABETES MELLITUS

1.8.1. Epidemiology and Pathogenesis of Diabetic Complications

There is growing evidence that the underlying mechanisms in the pathogenesis of diabetic complications include certain genetic and epigenetic modifications, nutritional factors, and sedentary lifestyle.[27] It was found that DNA-methylation, in or near genes belonging to the DNA replication/DNA metabolism process group, might play a key role in this process. Conversely, smoking, hypertension, and duration of DM over 10 years proved to be predictive factors for microvascular complications.

1.8.2. Microvascular Complications

Diabetic nephropathy, neuropathy, and retinopathy are the main microvascular complications induced by chronic hyperglycemia via several mechanisms such as the production of advanced glycation end products (AGEs), the creation of a proinflammatory microenvironment, and the induction of oxidative stress.

It was concluded that oxidative stress leads to the production of chronic inflammation and the glomerular and tubular hypertrophy, which characterize the early stages of DN. The novel biomarkers indicate renal injury such as transferrin, ceruloplasmin, podocalyxin, and VEGF. These markers can detect renal injury even before the presence of microalbuminuria, which remains the most valid biomarker for DN in clinical practice. It may be hoped that these mechanisms can help towards defining new therapeutic approaches for this microvascular complication of DM.[28]

To estimate the severity of autonomic neuropathy, found that diabetic retinopathy is the most significant predictive factor for CAN. To identify risk factors for the development of diabetic retinopathy (DR) in patients with type 1 DM.

1.8.3. Macrovascular Complications

Atherosclerosis is more common in people with DM than in those without. For example, DM increases the risk for stroke in people aged 20 to 65 years more than 5 times.It was reported that patients with DM are more prone to have significant stenosis with calcified plaques and such findings are accompanied by higher hs-CRP levels. In this process, many intracellular signaling pathways contribute to increased oxidative stress, which in turn leads to deposition of hydroxyapatite minerals into the extracellular matrix and vascular calcification.[29]

1.8.4. Miscellaneous Complications

Diabetic cardiomyopathy is a specific complication that develops independently of coronary artery disease or hypertension and it is possible to lead to increased morbidity and mortality. Low dose ethanol consumption was associated with lower mean arterial pressure, lower heart rate, high hydroxyproline content, and collagen volume fraction in myocardial tissue, together with decreased expression of ALDH2 and downregulation of the JNK pathway.[30]

1.9. DIAGNOSIS

Recommendations for target glucose levels are shown in Table 2. Three ways to diagnosis diabetes are available and each must be confirmed on a subsequent day. Because of ease of use, acceptability to patients and lower cost, the fasting plasma glucose (FPG) is the preferred test.

Hyperglycemia not sufficient to meet the diagnostic criteria for diabetes is categorized as either impaired fasting glucose (IFG) or impaired glucose tolerance (IGT), depending on whether it is identified through a FPG or an oral glucose tolerance test (OGTT): IFG = FPG ≥110 mg/dl (6.1 mmol/l) and <and to 125 mg/dl (6.9 mmol/l) IGT = 2-h PG ≥140 mg/dl (7.8 mmol/l) Or FPG 110 mg/dl (6.1 mmol/l) 126 mg/dl (7.0 mmol/l) < 199 mg/dl (11.0 mmol/l) Or 2-h PG 140 mg/dl (7.8 mmol/l) to 200 mg/dl (11.1 mmol/l) Both categories, IFG and IGT, are risk factors for future diabetes and cardiovascular disease. Recent studies have shown that lifestyle interventions can reduce the rate of progression to type - II diabetes.[31]

Table 2: Recommendations for target glucose levels

ADA Recommendations[32]	
Non-pregnant Adults	
Pre-prandial blood glucose levels	80-130 mg/dL (4.4-7.2 mmol/L)
Peak postprandial levels	<180 mg/dL (<10.0 mmol/L)
Hemoglobin A_{1c}	<7% (53 mmol/mol) < 6.5% (48 mmol/mol)[a]
Pregnant women with pre-existing type 1 or 2 DM, gestational diabetes	
Fasting plasma glucose	<95 mg/dL (5.3 mmol/L)
1-hr postprandial	<140 mg/dL (7.8 mmol/L)
2-hr postprandial	<120 mg/dL (6.7 mmol/L)
Hemoglobin A_{1c}	6%-6.5% (42-48 mmol/mol) < 6% (42 mmol/mol)[a] < 7% (53 mmol/mol)[b]
AACE Recommendations[33]	
Non-pregnant Adults	
Fasting blood glucose	<110 mg/dL (6.1 mg/dL)
2-hr postprandial	<140 mg/dL (7.8 mmol/L)
Hemoglobin A_{1c}	<6.5% (48 mmol/mol)

1.10. Management plan

People with diabetes should receive medical care from a physician-coordinated team. Such teams include but are not limited to, physicians, nurses, dieticians, and mental health professionals with expertise and a special interest in diabetes. It is essential in this collaborative and integrated team approach that individuals with diabetes assume an active role in their care

The management plan should be formulated as an individualized therapeutic alliance among the patient and family, the physician, and other members of the health care team. Any plan should recognize diabetes self-management education as an integral component of care. In developing the plan, consideration should be given to the patient's age, school or work schedule, and conditions, physical activity, eating patterns, social situation and personality, cultural factors, and presence of complications of diabetes or other medical conditions. Treatment goals must be set together with the patient, family, and health care team. Patient self-management should be emphasized, and the plan should emphasize the involvement of the patient in problem-solving as much as possible. A variety of strategies and techniques should be employed to provide adequate education and development of problem-solving skills in the various aspects of diabetes management. Implementation of the management plan requires that each aspect be understood and agreed on by the patient and the care providers and that the goals and treatment plan are reasonable.

1.10.1. MANAGEMENT

The treatment of diabetes is crucial; if left unchecked, it may lead to complications such as stroke, glaucoma, Alzheimer's disease, and cancer. Anyone who has recently developed the symptoms needs to be tested for the disease immediately.

When it comes to treating diabetes, controlling blood sugar levels is the main goal. When blood sugar levels reach a high point, symptoms begin to manifest. To achieve this goal, three methods

must be practiced: a healthy diet, regular exercise and consistent monitoring of blood sugar levels.[34]

A Healthy Diet Is Key in Controlling Blood Sugar Levels

Your lifestyle choices, especially your diet, play a crucial role in the development of diabetes. Consuming foods high in sugar and Trans fat can elevate your glucose levels, which can lead to the development of symptoms.

The first step to healthy eating is drastically reducing your intake of carbohydrates. When digested, they break down into sugar and enter your bloodstream, causing your glucose levels to rise. If this keeps up for an extended period of time, insulin resistance develops.

Once you've eliminated sugars, carbohydrates, and processed foods from your diet, replace them with nutritious whole foods. Not only will they help you lose weight, but they will also help you maintain healthy blood sugar levels, which is crucial in reversing your condition. Here are some strongly recommended points you can follow:

- **Consume healthier types of fat** — Healthy fats from coconut, avocados and unpasteurized nuts are a healthier source of fuel compared to carbs because they last longer and burn more efficiently.
- **Increase omega-3 intake** — Omega-3 is an essential fatty acid that can be obtained by consuming fatty fish such as wild Alaskan salmon, sardines, and anchovies, and also through krill oil supplementation. Getting this healthy fat into your system regularly can help optimize your overall health further.
- **Add probiotics** — Research has shown that obese individuals have a different structure of gut bacteria compared to those who are lean. The more probiotics you have, the stronger your immune system will be in fending off disease.

- **Exercise can help you lose weight and control diabetes:** Exercise should be integrated into your diabetes treatment plan as it helps you lose weight quicker and aids in maintaining healthy blood sugar levels
- **Monitoring Your Blood Sugar:** Monitoring your blood sugar is a critical part of diabetes treatment, as it helps keep your nutrition and body symptoms in check. Testing is usually done several times a day.[35]

1.10.2. PHARMACOLOGICAL MANAGEMENT

Choices for the treatment of type - 2 diabetes mellitus (T2DM) have multiplied as our understanding of the underlying pathophysiologic defects has evolved. Treatment should target multiple defects in T2DM and follow a patient-centered approach that considers factors beyond glycemic control, including cardiovascular risk reduction. Eight core defects, collectively known as "the ominous octet," contribute to the pathophysiology of type 2 diabetes mellitus (T2DM). These include decreased insulin secretion, decreased incretin effect, increased lipolysis, increased glucose reabsorption, decreased glucose uptake, neurotransmitter dysfunction, increased hepatic glucose production, and increased glucagon secretion (Figure 2). Therapy choices should target these established pathophysiologic defects in T2DM, as well as follow a patient-centered approach that considers factors beyond glycemic control, including reduction of overall cardiovascular disease.[36]

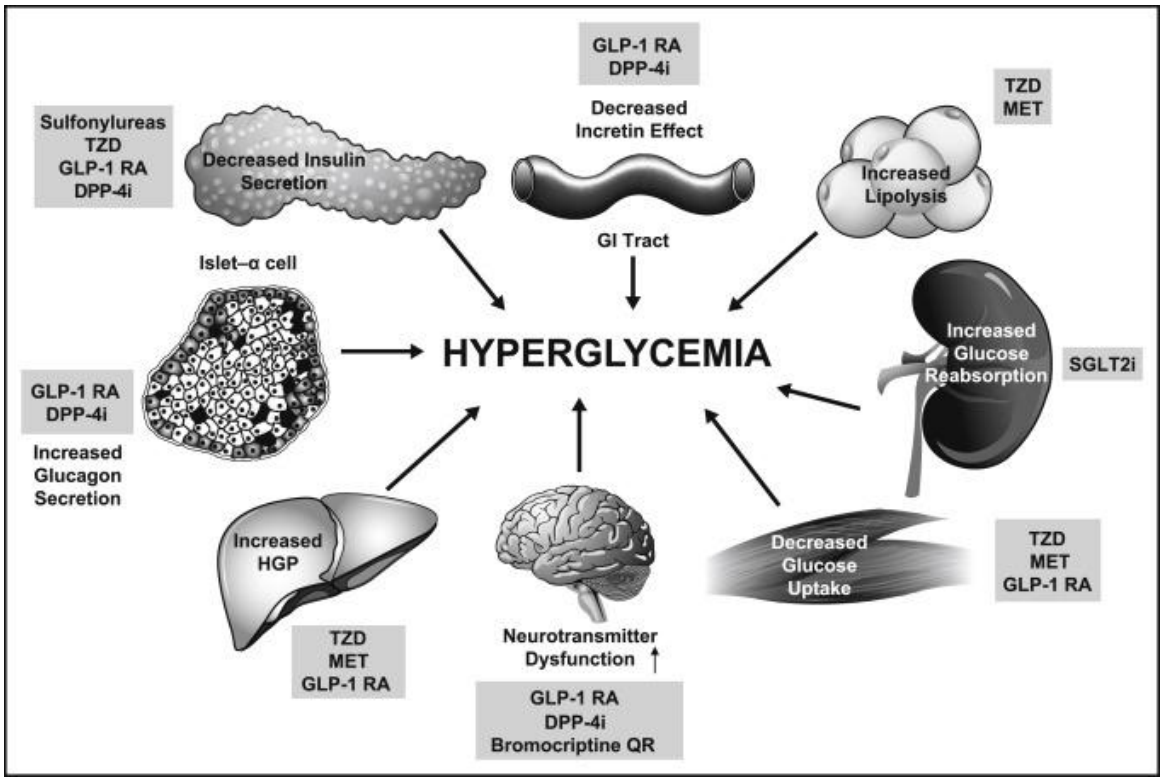


Figure 2. The ominous octet showing the mechanism and site of action of glucose-lowering medications based on pathophysiologic disturbances present in T2DM.1, 2, 3 DPP-4i = dipeptidyl peptidase-4 inhibitor; GI = gastrointestinal; GLP-1 RA = glucagon-like peptide-1 receptor agonist; HGP = hepatic glucose production; MET = metformin; QR = quick release; SGLT2i = sodium-glucose cotransporter 2 inhibitor; T2DM = type 2 diabetes mellitus; TZD = thiazolidinedione.[37]

The AACE/ACE algorithm suggests a preferred hierarchy of use for add-on therapy. In contrast, the ADA does not list a specific order for adding individual agents after metformin and lists 4 oral options (sulfonylurea, thiazolidinedione, dipeptidyl peptidase-4 [DPP-4] inhibitor, sodium-glucose cotransporter 2 [SGLT2] inhibitor) and 2 injectable agents (glucagon-like peptide-1 receptor agonist [GLP-1 RA] or basal insulin) as appropriate choices based on patient, disease, and drug characteristics (including cost), with the aim of decreasing blood glucose levels while minimizing adverse events, particularly hypoglycemia. The 6 classes of preferred noninsulin glucose-lowering agents common to the ADA and AACE/ACE are listed in Table 3 in order of

recommended use in the AACE/ACE hierarchy.[38] A brief discussion of therapeutic classes is presented in Table 3[39] in this order.

Table 3: Therapeutic classes of Type – II Diabetes Mellitus

Class	Agents (Route of Administration)	Cellular Mechanism(s)	Primary Physiologic Action(s)	Advantages	Disadvantages*	Cost
Biguanides	Metformin Metformin XR (oral)	Activates AMP-kinase (? Other)	•↓ Hepatic glucose production	• Extensive experience • Rare hypoglycemia •↓ CVD events (UKPDS)8, 9 • Relatively higher HbA1c efficacy	• GI side effects (diarrhea, abdominal cramping, nausea) • Vitamin B_{12} deficiency •Contraindications: eGFR <30 mL/min/1.73 m^2, acidosis, hypoxia, dehydration, etc. • Lactic acidosis (rare)	Low
GLP-1 RAs	Albiglutide Dulaglutide Exenatide Exenatide XR	Activates GLP-1 receptors	•↑ Insulin secretion (glucose-dependent)	• Rare hypoglycemia •↓ Weight •↓ Postprandial	• GI side effects (nausea, vomiting, diarrhea)	High

	Liraglutide Lixisenatide (SC injection)		•↓ Glucagon secretion (glucose-dependent) • Slows gastric emptying •↑ Satiety	glucose excursions •↓ Some CV risk factors • Associated with lower CVD event rate and mortality in patients with CVD (liraglutide, LEADER)[10]	•↑ Heart rate •? Acute pancreatitis • C-cell hyperplasia/medullary thyroid tumors in animals • Injectable • Training requirements	
SGLT 2 inhibitors	Canagliflozin Dapagliflozin† Empagliflozin (oral)	Inhibits SGLT2 in the proximal nephron	• Blocks glucose reabsorption in the kidney, increasing glucosuria	• Rare hypoglycemia •↓ Weight •↓ Blood pressure • Associated with lower CVD event rate and mortality in patients with	• Genitourinary infections • Polyuria • Volume depletion/hypotension/dizziness •↑ LDL-C •↑ Creatinine (transient) • DKA, urinary tract infections	High

				CVD (empagliflozin, EMPA-REG OUTCOME)[11]	leading to urosepsis, pyelonephritis	
DPP-4 inhibitors	Alogliptin Linagliptin Sitagliptin Saxagliptin (oral)	Inhibits DPP-4 activity, increasing postprandial incretin (GLP-1, GIP) concentration	•↑ Insulin secretion (glucose-dependent) •↓ Glucagon secretion (glucose-dependent)	• Rare hypoglycemia • Well tolerated	• Angioedema/urticaria and other immune-mediated dermatological effects •? Acute pancreatitis •↑ Heart failure hospitalizations (saxagliptin, ? alogliptin)	High
SUs	Second generation Glimepiride Glipizide Glyburide (oral)	Closes K_{ATP} channels on β-cell plasma membranes	•↑ Insulin secretion	• Extensive experience •↓ Microvascular risk (UKPDS)[12] • Relatively higher HbA1c	• Hypoglycemia •↑ Weight	Low

				efficacy		
TZDs	Pioglitazone† Rosiglitazone (oral)	Activates the nuclear transcription factor PPAR-γ	•↑ Insulin sensitivity	• Rare hypoglycemia • Relatively higher HbA1c efficacy • Durability •↓ Triglycerides (pioglitazone) •? ↓ CVD events (PROactive, pioglitazone)[13] • ↓ Risk of stroke and MI in patients without diabetes and with insulin resistance and history of recent stroke or TIA (IRIS	•↑ Weight • Edema/heart failure • Bone fractures •↑ LDL-C	

				study,[14] pioglitazone)		

The recommended initial T2DM management approach includes lifestyle changes and monotherapy (usually with metformin). If the HbA1c goal has not been met within approximately 3 months of starting initial therapy, treatment should be intensified by adding a second agent. Glycemic control should be reassessed again in approximately 3 months, and triple therapy should be considered if the HbA1c target is not achieved. If the HbA1c target is still not achieved, combination injectable therapy including basal insulin may be considered to obtain glycemic control. In patients with high baseline HbA1c levels, initial treatment with dual-combination therapy can be considered. The AACE/ACE suggests initial dual therapy (ie, metformin plus another agent in addition to lifestyle therapy) for patients with an entry HbA1c level ≥7.5%, whereas the ADA suggests considering initial dual therapy if the entry HbA1c level is ≥9%)[38]

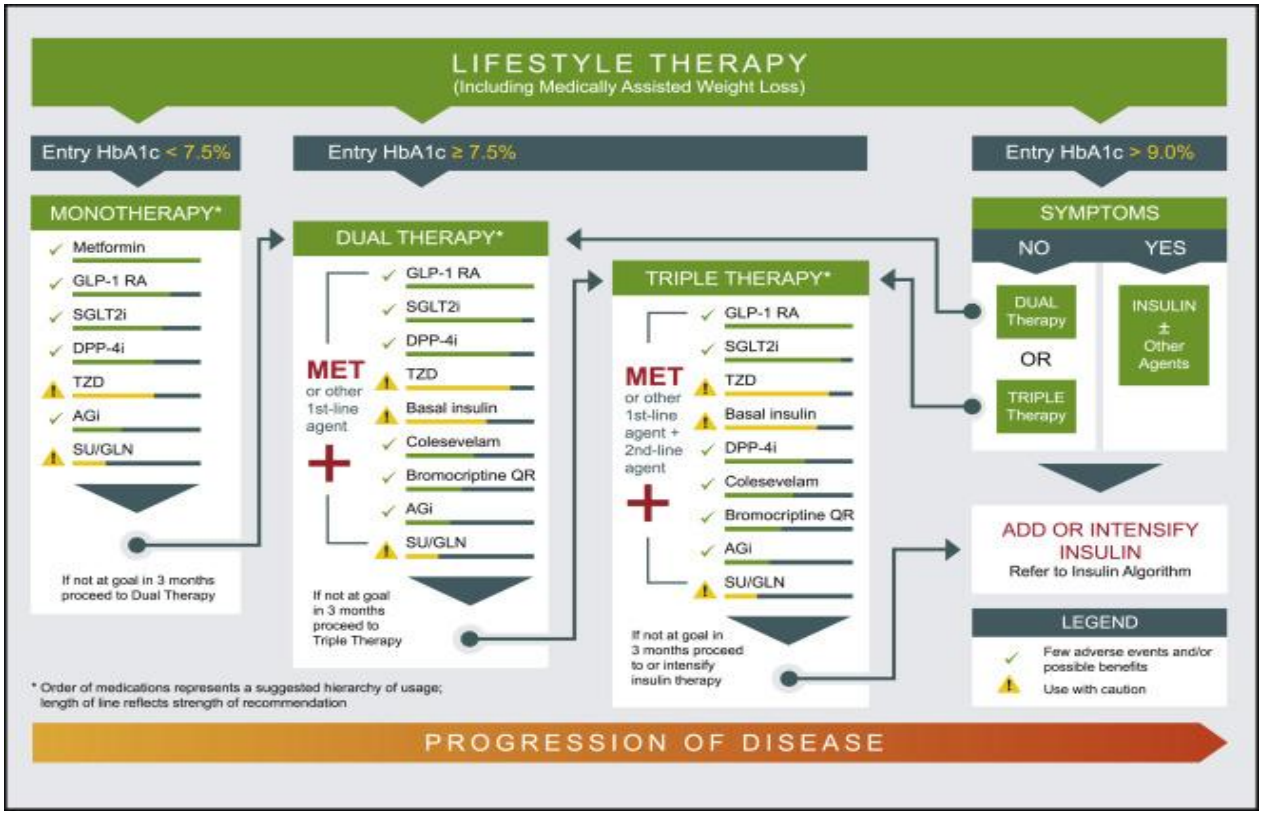


Figure 3: Glycemic control algorithm from the AACE/ACE. AACE/ACE = American Association of Clinical Endocrinologists/American College of Endocrinology; AGi = alpha-glucosidase inhibitor; DPP-4i = dipeptidyl peptidase-4 inhibitor; GLN = glinide; GLP-1 RA = glucagon-like peptide-1 receptor agonist; HbA1c = glycated hemoglobin; MET = metformin; QR = quick release; SGLT2i = sodium glucose cotransporter 2 inhibitor; SU = sulfonylurea; TZD = thiazolidinedione.[38]

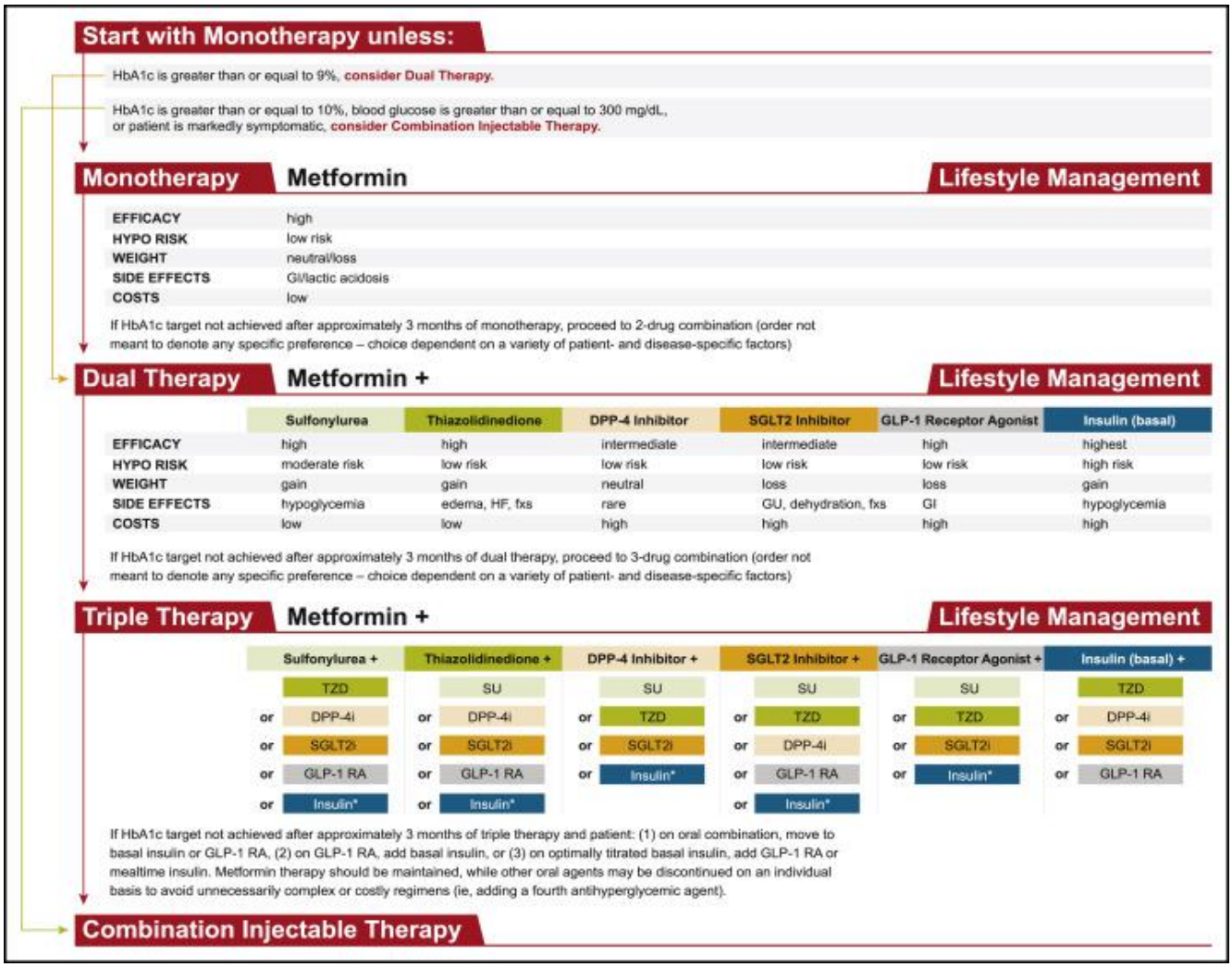


Figure 4: Glucose-lowering therapy in T2DM: general recommendations from the ADA.[39] The order in the chart was determined by historical availability and route of administration, with injectables to the *right*; it is not meant to denote any specific preference. Potential sequences of glucose-lowering therapy for patients with T2DM are displayed, with the usual transition moving vertically from top to bottom (although horizontal movement within therapy stages is possible, depending on the circumstances). *Usually a basal insulin (neutral protamine Hagedorn, glargine, detemir, degludec). ADA = American

Diabetes Association; DPP-4i = dipeptidyl peptidase-4 inhibitor; fxs = fractures; GI = gastrointestinal; GLP-1 RA = glucagon-like peptide-1 receptor agonist; GU = genitourinary; HbA1c = glycated hemoglobin; HF = heart failure; HYPO = hypoglycemia; SGLT2i = sodium-glucose cotransporter 2 inhibitor; SU = sulfonylurea; T2DM = type 2 diabetes mellitus; TZD = thiazolidinedione.)

Biguanide: Metformin

Metformin is the first choice for the treatment of T2DM unless contraindicated or not tolerated, based on its well-defined efficacy and safety profile, and low cost.[39] Metformin suppresses hepatic glucose production and improves insulin sensitivity

Metformin and/or metformin extended-release is available as a single-pill (ie, fixed-dose) combination with multiple other glucose-lowering agents, including sulfonylureas, thiazolidinediones, DPP-4 inhibitors, and SGLT2 inhibitors.[40] Although gastric tolerability with metformin can be a problem for some patients, this can be improved with appropriate dose up-titration over time or by changing to an extended-release formulation of metformin.

Glucagon-Like Peptide-1 Receptor Agonists

The term "incretin effect" is derived from the observation that insulin release from the pancreas is greater after oral than intravenous glucose administration.[41] GLP-1 and glucose-dependent insulinotropic polypeptide are both incretin hormones released from the gut after a meal. Stimulation of the GLP-1 receptor enhances insulin release and decreases glucagon secretion from the pancreas. Gastric emptying also may be delayed (particularly with shorter-acting pharmacologic compounds),[42] which may lead to appetite suppression

The efficacy and safety of GLP-1 RAs have been assessed in head-to-head trials; these include 2 short-acting agents (twice-daily exenatide and once-daily lixisenatide) and 4 long-acting agents (once-daily liraglutide and the once-weekly formulations of exenatide, albiglutide, and dulaglutide).

Sodium-Glucose Cotransporter 2 Inhibitors

SGLT2 inhibitors target the kidney to promote urinary glucose excretion and decrease hyperglycemia. Under normal conditions, the kidney reabsorbs nearly all of the filtered glucose, so that virtually no glucose is excreted into the urine. Renal glucose reabsorption occurs in the proximal tubule, primarily by the glucose transport protein SGLT2, and to a lesser extent by sodium-glucose cotransporter. Because of their noninsulin-dependent mode of action, SGLT2 inhibitors can be used in combination with any class of glucose-lowering agents and at any stage of disease, including in patients with long-standing T2DM who have minimal insulin secretion. Single-pill combinations of SGLT2 inhibitors and metformin are available, as are SGLT2 inhibitor/DPP-4 inhibitor single-pill combinations: empagliflozin/linagliptin and dapagliflozin/saxagliptin.[43]

Dipeptidyl Peptidase-4 Inhibitors

DPP-4 inhibitors reduce the enzymatic degradation of the incretin hormones, GLP-1, and glucose-dependent insulinotropic polypeptide by reducing the activity of serum DPP-4 by ≥80%. This leads to an increased availability of endogenous incretins, stimulating insulin secretion from pancreatic β-cells and inhibiting glucagon release from pancreatic α-cells in a glucose-dependent manner.[41] These agents may be used as monotherapy or combination therapy and are available as single-pill combinations with metformin or metformin extended-release. Besides, the DPP-4 inhibitor alogliptin is available in combination with a thiazolidinedione (alogliptin/pioglitazone).[44]

Other Oral Glucose-Lowering Therapies

The sulfonylureas and thiazolidinediones may be considered as an alternative to metformin for monotherapy or as an add-on option for dual- or triple-combination therapy. Although not preferred, these agents may be useful in select clinical settings. For example, thiazolidinediones may be useful for patients who require an insulin-sensitizing agent, but in whom metformin is

contraindicated. The thiazolidinediones also may be useful for patients with T2DM whose occupations preclude the use of insulin and in whom the risk of a hypoglycemic episode could have severe consequences; in such circumstances, a thiazolidinedione could be used as part of a triple-therapy regimen.

Thiazolidinediones stimulate peroxisome proliferator-activated receptors, nuclear receptors that alter the transcription of several genes involved in glucose and lipid metabolism, thereby promoting insulin sensitivity in adipocytes, muscle, and liver.[45] However, thiazolidinediones are associated with fluid retention, which can lead to weight gain, peripheral edema, and heart failure, and are thus contraindicated in patients with established New York Heart Association Class III or IV heart failure. Pioglitazone has been associated with decreased risk of stroke, whereas there has been controversy surrounding a potential increased risk of ischemic cardiovascular events with Rosiglitazone. The FDA restricted the use of Rosiglitazone based on initial concerns about a signal for increased cardiovascular risk in a published meta-analysis[46]; however, the restrictions were later eased after data were re-reviewed.[47] Thiazolidinediones decrease bone mineral density, which can lead to an increased risk of nonosteoporotic bone fractures, particularly in postmenopausal women and elderly men.

The sulfonylureas stimulate insulin release from the pancreas by binding to the sulfonylurea receptor on the adenosine triphosphate-sensitive potassium channel on the ß-cell membrane.[47] Other oral glucose-lowering agents, such as the α-glucosidase inhibitors, colesevelam (a bile acid sequestrant), and bromocriptine (a quick-release dopamine receptor agonist), may be considered in a combination therapy regimen for selected patients.[46] The α-glucosidase inhibitors slow intestinal absorption of carbohydrates and have modest HbA1c-lowering efficacy; the need for frequent dosing and gastrointestinal adverse events (flatulence and diarrhea) may limit use. Colesevelam provides modest reductions in HbA1c, rarely causes hypoglycemia, and decreases low-density lipoprotein cholesterol; however, it may increase triglycerides, cause constipation,

and affect the absorption of other medications. Bromocriptine rarely causes hypoglycemia, has slight HbA1c-lowering efficacy, and may cause nausea and orthostatic events.[47]

2. LITERATURE REVIEW

Jay Kumar Sharma *et al.*, (2018)[48] conducted a study on a cross sectional, observational study was conducted in type 2 diabetic outdoor patients with co-existing hypertension, for duration of one year. We used descriptive statistics to analyze data of 615 patients to determine prescribing pattern of drugs. 93.17% of patients were more than 40 years of age. Ratio of female to male patients was 1.30. Mean duration of diabetes was 5.81 years. Multi drug anti-diabetic regimes (54.47%) were common than monotherapy (43.90%). Commonly prescribed anti-diabetic drug groups were biguanides (89.27%), Sulfonylureas (43.90%) and insulin (15.28%). Metformin (89.27%), glibenclamide (29.11%) and insulin (15.28%) were commonly prescribed anti-diabetic drugs. Metformin was the most common monotherapy drug and biguanide + sulfonylurea was the most common two drug combination. Commonly prescribed drugs for co-morbid conditions were enalapril (83.41%), aspirin (30.41%), amlodipine (29.76%) atorvastatin (27.32%) and famotidine (26.34%). Average number of drugs prescribed in a prescription was 4.65. Prescriptions with injections were 15.93% and with brand names were 13.15%. Prescribing pattern of drugs was as per current practices and recommendations of guidelines. Still, there is room for improvement in choice of drug, prescribing drug with generic name and choosing drug from essential medicine list.

Misbahuddin *et al.*, (2018)[49] conducted a study on retrospective prescription information and medical records of patients who visited outpatient clinics during the last one year were used. The prescriptions were grouped into three: appropriate, partially appropriate and inappropriate. A total of 504 prescriptions were evaluated, while the male to female ratio was 3:1. The mean anti-diabetic drug per prescription was 2.08 ± 0.85. The most common prescriptions were metformin, sulfonylurea and insulin. More than two-thirds of the patients were on combination therapy. No prescriptions were found for thiazolidinediones, glucagon-like peptide-1 (GLP-1) analogues and α-glucosidase inhibitors. Metformin/sulfonylurea was

the most common combination. The patients that received insulin with an oral agent accounted for 8 % of the total prescriptions. While 62 % of the patients reached fasting blood glucose goal of ≤ 126 mg/dl, there was no correlation between normoglycemia and total number of drugs, gender or age group. Moreover, age, sex, initial glucose concentration, and total drugs had no effect on final glucose levels. Prescription patterns of anti-diabetic drugs are in accordance with international guidelines but some shortcomings were observed probably due to poor prescription writing.

Geetha *et al.*, (2017)[50] conducted a study on prospective study was carried out by evaluating 115 prescriptions of antidiabetic drugs over the period of 4 months to assess the prescribing pattern of antidiabetic drugs and also drugs used for other complications of Type 2 DM. Totally, 115 patients were evaluated, 58 were of male and 57 were of female. An average number of drugs per encounter were found to be 4.47. An average number of antidiabetic drugs were found to be 2.56. In this study, the most commonly prescribed oral hypoglycemic drug class as single-drug regimen was that of alpha-glucosidase inhibitors (16.326%), dipeptidyl peptidase-4 (DPP-4) inhibitors (14.62%), biquanides (12.9%), thiazolidine diones (9.8%), sulfonyl urea (7.82%) and meglitinides (2.38%), and in multi-drug regimen metformin + alpha-glucosidase inhibitors (11.56%) were commonly prescribed. Most commonly used drug was alpha-glucosidase inhibitors, followed by DPP-4 inhibitors and biguanides. All the patients received combination therapy to achieve the glycemic control.

Venkateswaramurthy *et al.*, (2016)[51] conducted a study on retrospective observational epidemiological study undertaken for a period of 8 months in outpatient unit of a tertiary care Hospital, Erode. The study enrolled 175 patient prescriptions. A total of 175 patients had co-morbid conditions along with diabetes. Commonly seen co-morbid condition in the study was hypertension. The study has shown metformin as the predominantly prescribed oral antidiabetic drug both as monotherapy as well as combination therapy. Overall, monotherapy

was found to be predominant over combination therapy. There was no significant increase in the prescribing of newer oral antidiabetic agents like GLP-1receptor inhibitors and DPP-4 inhibitors. Glimepride + Metformin combination was the most commonly prescribed combination. This study revealed that the pattern of antidiabetic prescription was largely comply with NICE guidelines.

Sekhar Mandal *et al.*, (2016)[52] conducted study on prospective observational study including 181 patients for a period of 6 months in Bankura Sammilani Medical College. Patients diagnosed as type 2 diabetes mellitus were included in the study. The demographic data, disease data and utilization of different classes of oral hypoglycaemic agents and insulin as well as other individual drugs were analysed using the World Health Organization (WHO) indicators for drug utilization studies. Results: The study population was predominantly male (61.33%) and nearly a third (30.9%) belonged to the age group of 50-59 years. Co-morbid conditions were found in 74% patients, among which hypertension (51.1%) was the most common co-morbid condition. The average number of drugs per prescription was 4.22 and the average number of antidiabetic drugs per prescription was 2.18. Metformin was the most commonly prescribed drug (79.6%), followed by sulfonylurea class of drugs (66.9%). Nearly 17.7% patients were on insulin preparations. Glimepiride and metformin was the most common combinations used (45.5%). Antibiotics were included in 15.5% prescriptions and proton pump inhibitors were prescribed in 32% cases. All the medicines were prescribed as generics and injections were prescribed in 17.7% cases. Conclusions: This study gives a picture of the pattern of drug use among diabetes patients in our set up. While metformin was the commonest drug used, glimepiride and metformin combination was the commonest combination therapy.

Tamoghna Maiti *et al.*, (2016)[53] conducted study on cross-sectional, institutional based, observational study done at diabetic OPD at B.S. Medical College for a duration of three

months. At the exit point, information was collected in pre-structured case report form including socio-demographic profile, histories of diabetes, co-morbidities if any, relevant investigations and details of medications after obtaining written consent. Result: Among 202 patients majority were males. Three forth of patients suffered from DM for less than 5 years of duration. Two third of them had one or more co-morbidities. A total of 726 drugs were prescribed of which nearly half were anti-diabetic drugs. Metformin was the leading used anti-diabetic drug given to 77.2% of patients. Newer OHAs were used only as part of combination therapy. Only 14 patients received Insulin. Statin and anti-hypertensive were used in good percentage. Average cost of prescription per day per patient was 26.33 INR. Conclusion: Number of drugs prescribed was less than contemporary drug utilization studies. It was a very short duration study incorporating only 202 patients. In future we plan to undertake this with larger number of patients in multiple centers.

Siddhartha Nuthakki *et al.*, (2016)[54] conducted study on prospective observational study, 415 prescriptions of type 2 diabetes mellitus (T2DM) patients were collected in Dr. Pinnamaneni Siddhartha Institute of Medical Sciences (PSIMS) hospital from January 2015 and June 2015. Medication adherence to AACE/ACE guidelines was assessed based on glycated haemoglobin (HbA1C) values. Results: A total of 201 (48.4%) male and 214 (51.6%) female patients were identified. The mean age was 53.57 ± 10.77 years (male) and 53.69 ± 10.71 years (females). Patients with HbA1C <7.5% (37.3%, male; 45.3%, female) were predominant followed by HbA1C 7.5% - 9% (32.3%, male; 35.3%, female) and HbA1C > 9.0% (30.4%, male; 19.2%, female). Hypertension (HTN) (39.8%, male; 39.7%, female) is the most predominant co-morbidity, followed by patients with both HTN and cardio vascular diseases (CVDs) (9.4%, male; 9.8%, female). Insulin was prescribed to control hyperglycaemia in most of the cases (40.0%) followed by dual therapy (26.9%) and triple therapy (17.8%). The overall adherence rate was 88.3% for patients with HbA1C <7.5% (P<

0.0001); 98.7% for patients with HbA1C 7.5%-9%(P<0.0001)and 100% for patients with HbA1C >9%(P<0.0001). Conclusion: Optimal medication adherence is the ultimate goal to control the hyperglycemia in DM. The present study results revealed that the anti-diabetic medication adherence to AACE/ACE 2015 guidelines were optimal by the prescribers.

Kayamkani Abedulla Khan *et al.*, (2016)[55] conducted study on prospective observational study was conducted to find out the drugs utilization and prescriptions pattern of antihyperglycemic agents in diabetic outpatients in urban Telangana. This study was conducted for a period of six months, known cases of Diabetic Mellitus who were receiving antihyperglycemic medicines as outpatients of either sex of all age groups were included and Patients with gestational diabetes were excluded from the study. In this study a total number of 250 prescriptions were collected which contained 1674 drugs. Among the prescriptions as an average each prescription contained more than 6.7drugs, this indicates polypharmacy. Among the antidiabetic drugs, metformin was found in a maximum number of prescription i.e. 109 (43.60%) and sitagliptin was found in a minimum number of prescriptions i.e.28 (11.20%) and most of the prescriptions contained antihypertensive agents, analgesics, vitamins and minerals preparations as supplements. This report alarm all the physicians and health care professionals about various drug interactions and adverse drug reactions which occur due to polypharmacy and also causing a more financial burden to chronic disease patients.

Ramesh Gorre *et al.*, (2015)[56] conducted study on prospective case study of the patients who are under anti-diabetics therapy associated with or without co-morbidities, which was approved by the Institutional Ethics Committee (IEC) of Narayana Hrudayalaya. Drug utilization of anti-Diabetics in various wards frequent use of sulfonylureas and biguanides in age group 50-70 was observed. The study described insulin and sulfonylureas were frequently prescribed drugs followed by biguanides.

Pratiksha Kasture *et al.*, (2015)[57] conducted study on pospective observational study was carried out at rural areas nearby to Islampur for a period of 3 months. 90 patients were screened having DM and a structured questionnaire was used to collect data and analysis

done. The prevalence was about 6.6% Type I DM and 93.33% of them about 50% patients were in above 50 years of age. Totally 76.66% of patients were on monotherapy and Metformin (Glycomate) was commonly prescribed. In combination therapy, Glibenclamide and Metformin (35.86%) were mostly. Type II DM was treated effectively with both Insulin and Oral hypoglycemic drugs. The average cost of Therapy during the Study was found to be Rs. 100-300/month in 70% of population under study. In the study carried out most of the prescriptions were rational, but further improvement is needed. The choice of drug should be based economic status, associated conditions. Rational prescribing should focus on dose and duration as well as interaction with other medications.

Alti Aparna *et al.*, (2015)[58] conducted study on prospective observational study carried out for a period of six months at RIMS kadapa, and two others diabetic centers. The diabetic patients who visited the medicine outpatient department were included. After obtaining approval from institutional ethical committee, a structured data collection form was used to collect demographic data, complete prescription details and other relevant information required for the study. The drug utilization pattern was determined. The drugs were categorized by Anatomical therapeutic classification (ATC) and DDD/1000 inhabitants/day was calculated by using WHO guidelines. Among all oral hypoglycemic agents the most effective drug/combination in this region was identified. 716 prescriptions were assessed out of which,401(56.0%) were females and 315(43.9%) were males, most of the patients were in the age group of 40-60 for males 175(55%) and females 205(51.1%). Hypertension was the most common co-morbid seen. The average number of drugs per prescription was 4.26 and antidiabetics per prescription were 1.79. DDD/1000 inhabitants/day for metformin (A10BA02) was 10.5, glimiperide (A10BB12) was 9.3, glibenclamide (A10BB01) was 7.91, pioglitazone (A10BG03) was 7.25. Out of 716 patients 311(45.25%) patients were on Monotherapy, and 405 (56.5%) were on Combination therapy. A total of 200 newly

diagnosed patients of diabetes mellitus were enrolled in the study out of which only 128 members were followed up successfully. The combinations of Metformin +Sulfonyl Ureas + Others showed a good control of fasting blood sugar when compared with only Metformin, only Sulfonyl Ureas or Metformin +Sulfonyl Ureas, Sulfonyl Ureas + Others. Metformin was the most utilized drug followed by glimiperide. Combination therapy was most frequent when compared to monotherapy in which metformin+glimiperide was commonly prescribed one. So by understanding the current prescribing patterns attempts can be made to improve rational prescribing. The combination of Metformin+Sulfonyl Ureas+Others is more effective combination.

Kannan *et al.*, (2011)[59] conducted study on prospective study was carried out in 202 out patients for a period of 9 months in a tertiary care hospital. Patients treated with oral hypoglycemic agents were used for the study. The demographic data, disease data and utilization of different classes of oral hypoglycemic agents as well as individual drugs were analyzed. The study found that 51.98% patients were males. The greatest number of patients(38.61%) were in the age group of 51-60 years. Sulfonylurea and biguanide combination (57.09%) was the most common class of drug used among the various oral antidiabetics prescribed. The average number of drugs per prescription was 4 and the average number of antidiabetic drugs per prescription was 1.4. Average cost of oral hypoglycemic agents per prescription for 1 month was found to be INR 275.35. Glimepiride and metformin combination drugs were the most commonly used drugs. Overall polypharmacy was high even though polypharmacy among antidiabetic drugs were low. The cost of drugs per prescription was found to be very high.

3. AIM AND OBJECTIVES

3.1 AIM

- To study on prescribing patterns of anti-diabetic drugs for patients with type- II diabetes mellitus.

3.2 OBJECTIVES

- To determine the demographic details of the diabetes mellitus patients
- To estimate the prevalence of diabetes mellitus.
- To assess the drug prescribing patterns of anti-diabetic drugs.
- To provide verbal patient counseling and educate to diabetes mellitus patients.

3.3 PLAN OF WORKS

The entire study was planned to be carried out for a period of 6 months.

The proposal is designed as given below:

- **Phase 1**
  - Identified the scope of work
  - Literature survey
  - Study designed including designing of questionnaire form and patient consent form
  - Obtained approval from the Institution's ethical committee.
- **Phase 2**
  - Obtained consent from hospital authorities and written consent from patients.
  - Collections of patient details from the patient's prescription and by conducting the direct patient interview.
- **Phase 3**
  - Data analysis
  - Submission of report

3.4 METHODOLOGY

Study design:

It is a prospective observational study.

Study site:

The research work was conducted at a tertiary care hospital, Erode, Erode district, Tamil Nadu.

Study period:

6 months

Inclusion criteria:

- Type-II diabetic outdoor patients.
- Patients with co-morbidities disease conditions.
- Aged more than 18 years, of both sexes.
- Who gave consent from the patient's side.

Exclusion criteria:

- Those patients, who had complications like retinopathy, neuropathy, diabetic foot, stroke, and myocardial infarction.
- Type-1 diabetic indoor and outdoor patients.
- Pediatric and pregnant women.
- Patients not willing to participate.

Source of data:

The data collected from the patient’s prescription and also through a direct patient interview.

Informed consent and ethical clearance

The study protocol had been approved by the Institutional Ethical Committee. The nature and purpose of the study was explained and their consent sought.

3.5 WORK METHODOLOGY

Based on the inclusion and exclusion criteria the study was conducted at a tertiary care hospital erode for six months. A complete of 426 prescriptions were enrolled and analyzed in diabetes patients at outpatient departments. Data of patients matching inclusion criteria were recorded. Before including within the study, patients were explained regarding the aspects of research work. Written consent was taken before including him or her into the study. Once the consultation by the doctor was over, patients were screened for study criteria. Interviewed such patients and reviewed their prescriptions. Details like age, sex, duration of illness, on-going treatment concurrent medicines, assess the prescription patterns, family history, coexistent diseases, socio-economic standing were recorded in the data entry form.

4. RESULTS

Table 1: Gender wise distribution of patients in treatment with anti-diabetes drugs

Gender	Number of patients (n=426)	Percentage (%)
Male	266	62.44
Female	160	37.55

Figure 1: Gender wise distribution of patients in treatment with anti-diabetes drugs

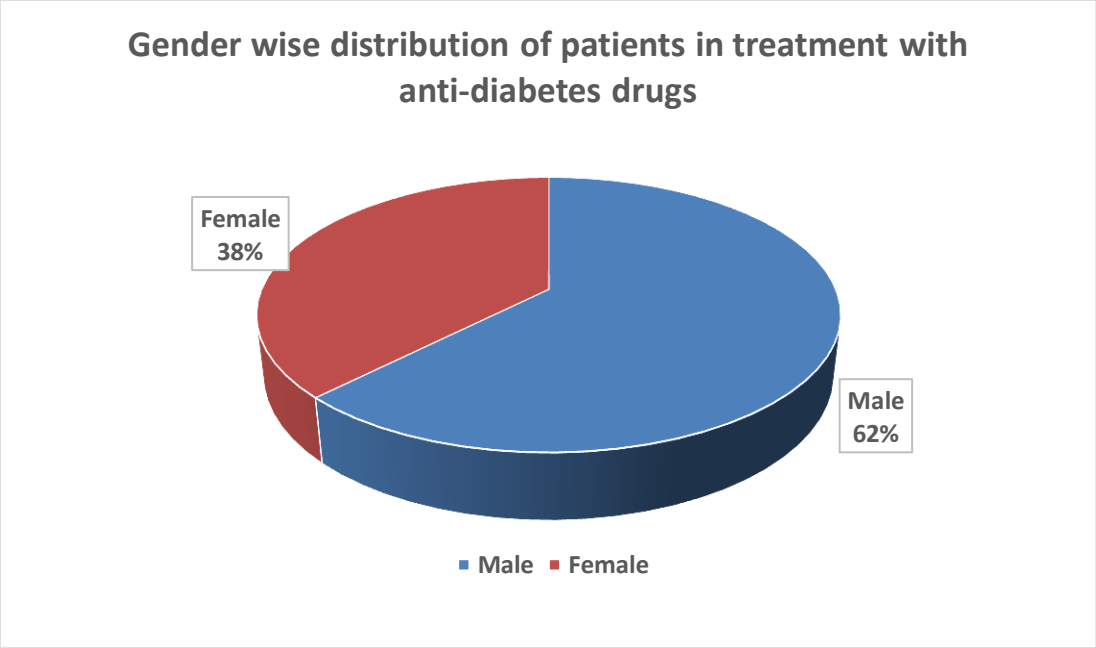


Table 2: Age wise distribution of patients in treatment with anti-diabetes drugs

Sl. No.	Age (in years)	Number of patients (n=426)	Percentage (%)
1.	<40	61	14.31
2.	41-60	216	50.70
3.	61-80	82	19.24
4.	>80	67	15.72

Figure 2: Age wise distribution of patients in treatment with anti-diabetes drugs

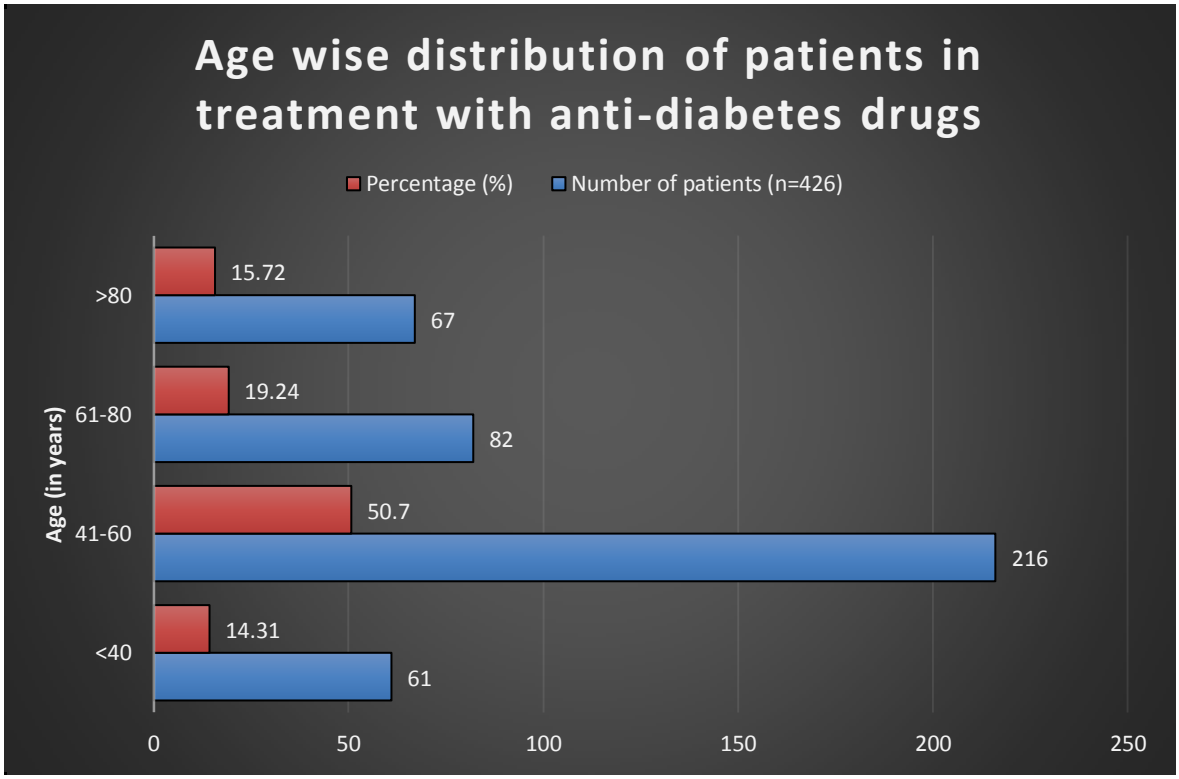


Table 3: Residential area-wise distribution of patients in treatment with anti-diabetes drugs

Residential area	Number of patients (n=426)	Percentage (%)
Rural	116	27.23
Urban	249	58.45
Mixed	61	14.31

Figure 3: Residential area-wise distribution of patients in treatment with anti-diabetes drugs

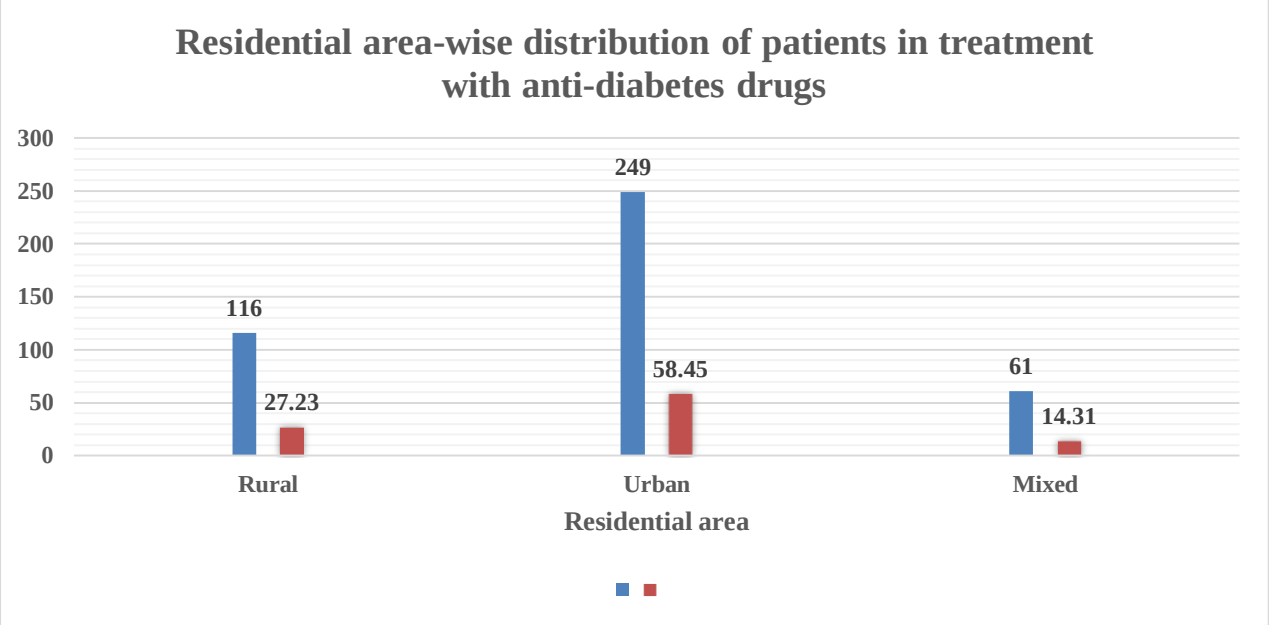


Table 4: Distribution of food consumption based on patients

Food consumption	Number of patients (n=426)	Percentage (%)
Vegetarian	91	21.36
Vegetarian & Non-vegetarian	335	78.63

Figure 4: Distribution of food consumption based on patients

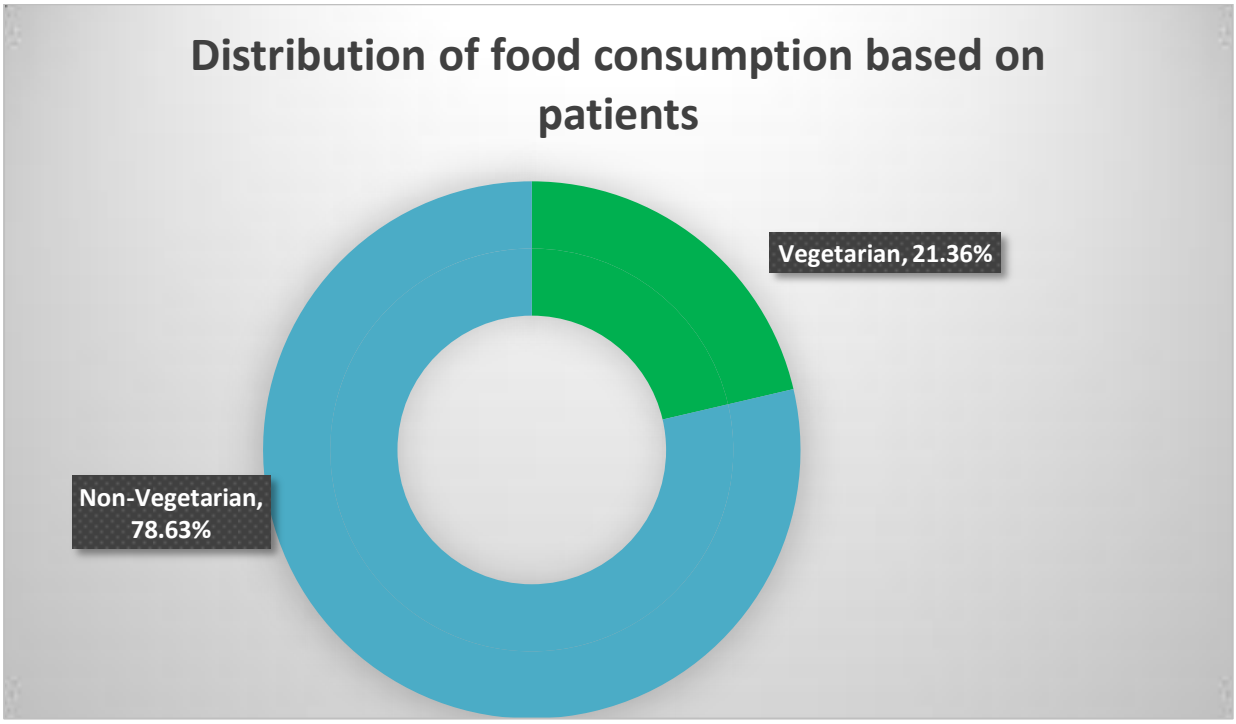


Table 5: Social distribution of diabetes patients

Social risk factors	Number of patients (n=426)	Percentage (%)
Smoking	94	22.06
Tobacco chewing	76	17.84
Alcohol	104	24.41
Smoking + tobacco + alcohol	99	23.23
None	53	12.44

Figure 5: Social distribution of diabetes patients

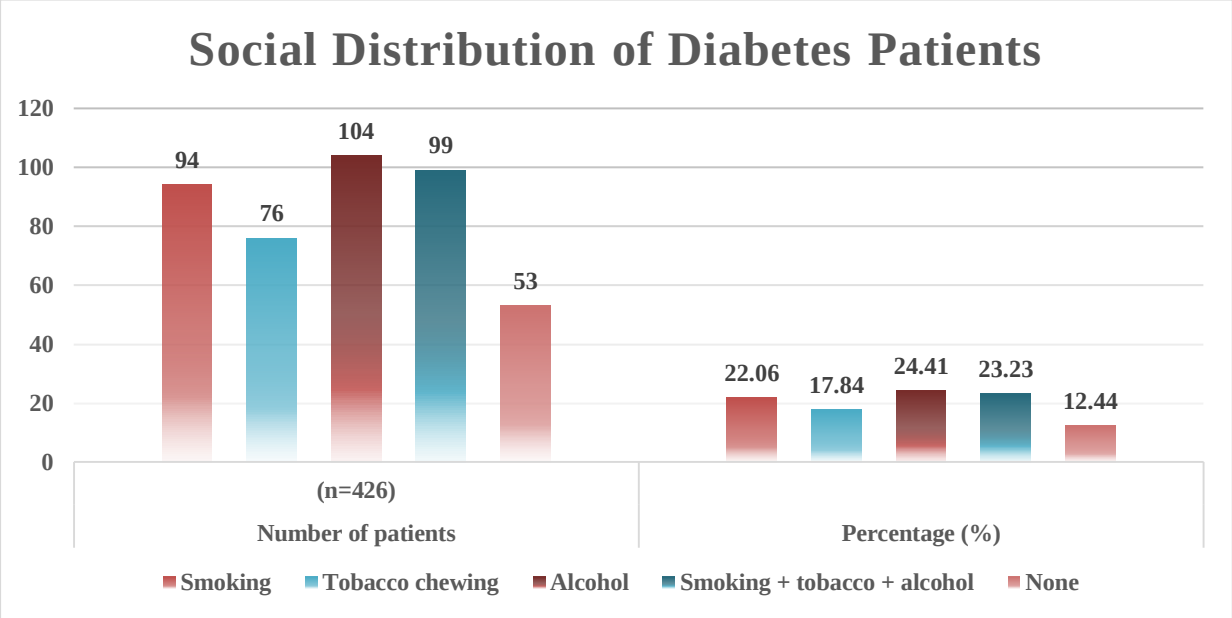


Table 6: Distribution of family history for diabetes patients

Family relationship	Number of patients (n=426)	Percentage (%)
Father	213	50.00
Mother	95	22.30
Both	45	10.56
Others	31	07.27
None	42	09.85

Figure 6: Distribution of family history for diabetes patients

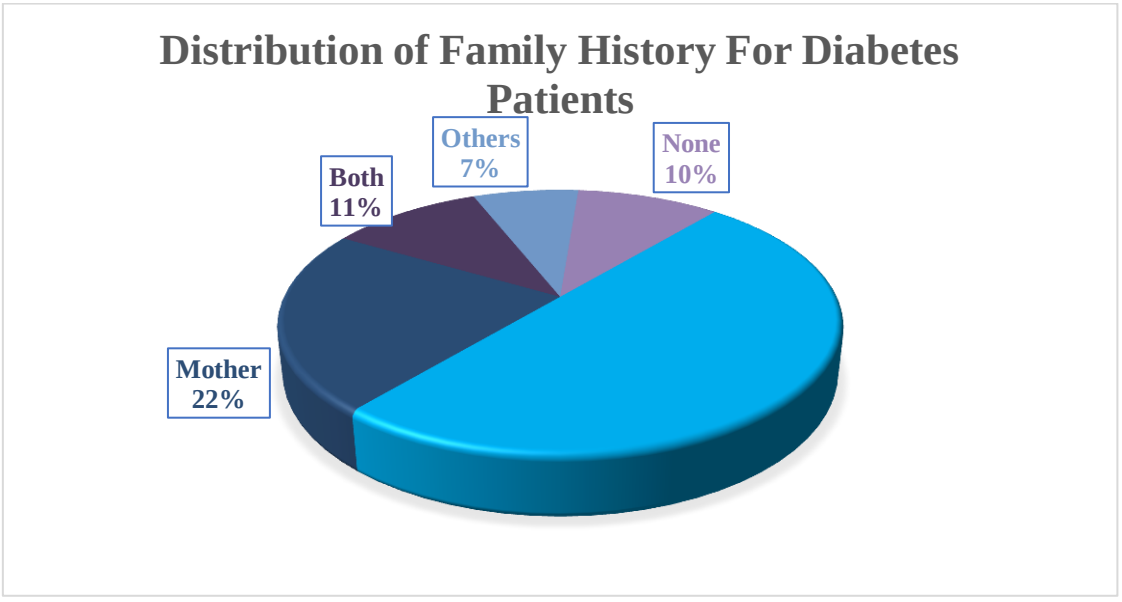


Table 7: Distribution of the diabetic patients according to duration of years

Duration of years	Number of patients (n=426)	Percentage (%)
Within Past 12 months	42	09.85
1 to 5 years	94	22.06
5 to 10 years	221	73.94
More than 10 years	69	16.19

Figure 7: Distribution of the diabetic patients according to duration of years

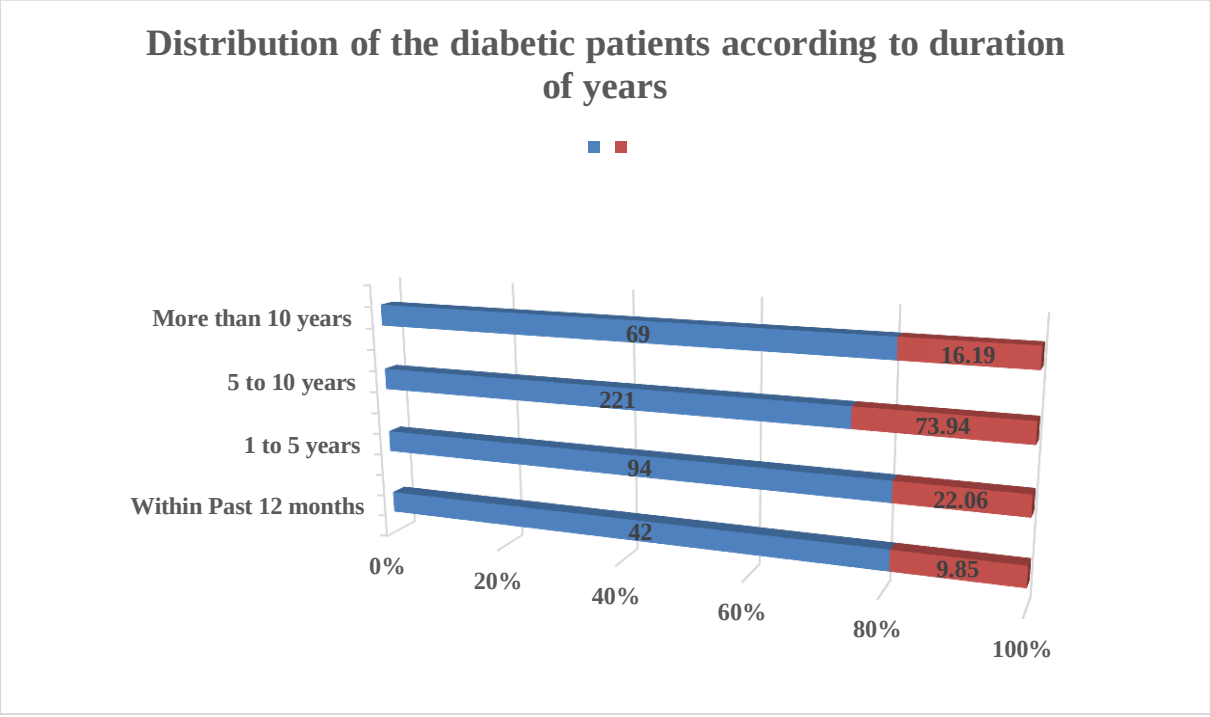


Table 8: Co-morbidities illness in patients with diabetes

Co-morbidities illness	Number of patients (n=426)	Percentage (%)
Diabetes mellitus alone	174	40.84
Diabetes + Hypertension	99	23.23
Diabetes + Cardiovascular diseases	49	11.50
Diabetes +Asthma	57	13.38
Diabetes + kidney disease	35	08.21
Diabetes + Hypertension + asthma	7	1.64
Diabetes + Hypertension + kidney disease	5	1.17

Figure 8: Co-morbidities illness in patients with diabetes

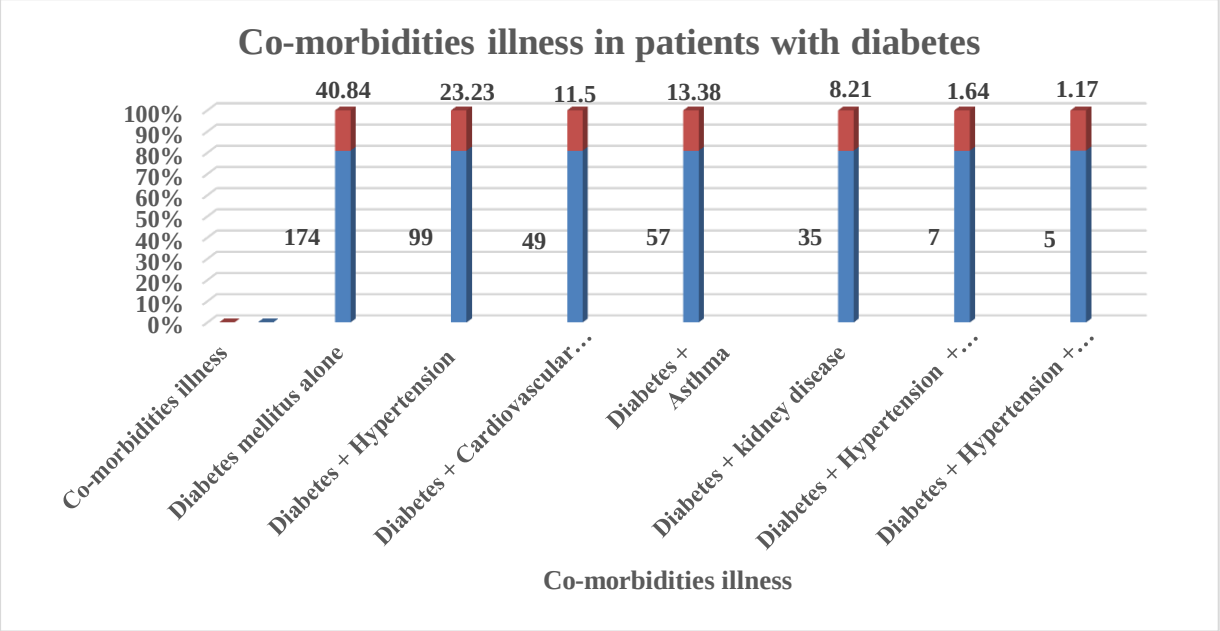


Table 9: Total drugs prescribed over the study period

SI.No	Drugs prescribed	Number of drugs prescribed (n=1565)	Percentage (%)
1.	Anti-diabetics	1074	68.62
2.	Anti-hypertensives	210	13.41
3.	NSAIDs	123	07.85
4.	Anti-asthmatic	12	07.66
5.	Statins	21	01.34
6.	Antianxiety/ antidepressants	56	03.57
7.	Miscellaneous (Supplement's)	69	04.40

Figure 9: Total drugs prescribed over the study period

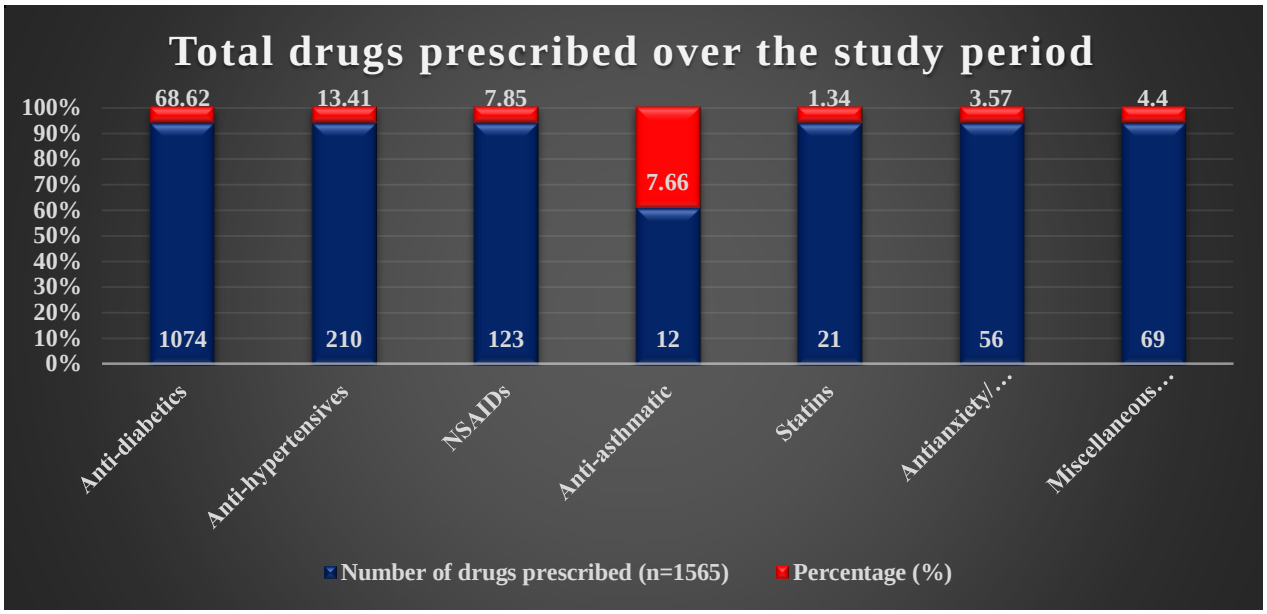


Table 10: Distribution of the diabetic patients according to prescribing pattern

Type of Therapy	Number of patients (n=426)	Percentage (%)
Monotherapy	212	49.76
Two drug therapy	156	36.61
Three drug therapy	36	08.45
Four drug therapy	22	05.16

Figure 10: Distribution of the diabetic patients according to prescribing pattern

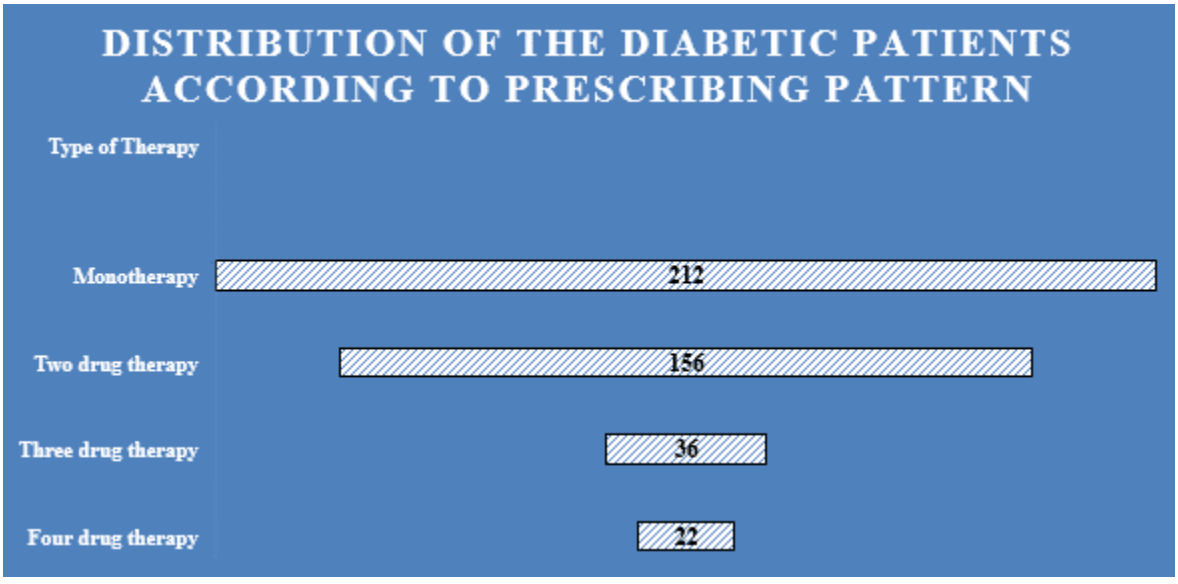


Table 11: Class of anti-diabetic therapy (MONOTHERAPY)

Class of drugs	Number of drugs prescribed (n=212)	Percentage (%)
Biguanides	86	40.56
Sulfonylureas	66	31.13
DPP-4 Inhibitors (Gliptins)	33	15.56
Thiazolidinediones	16	7.54
Alpha - glucosidase inhibitors	11	5.18

Figure 11: Class of anti-diabetic therapy (MONOTHERAPY)

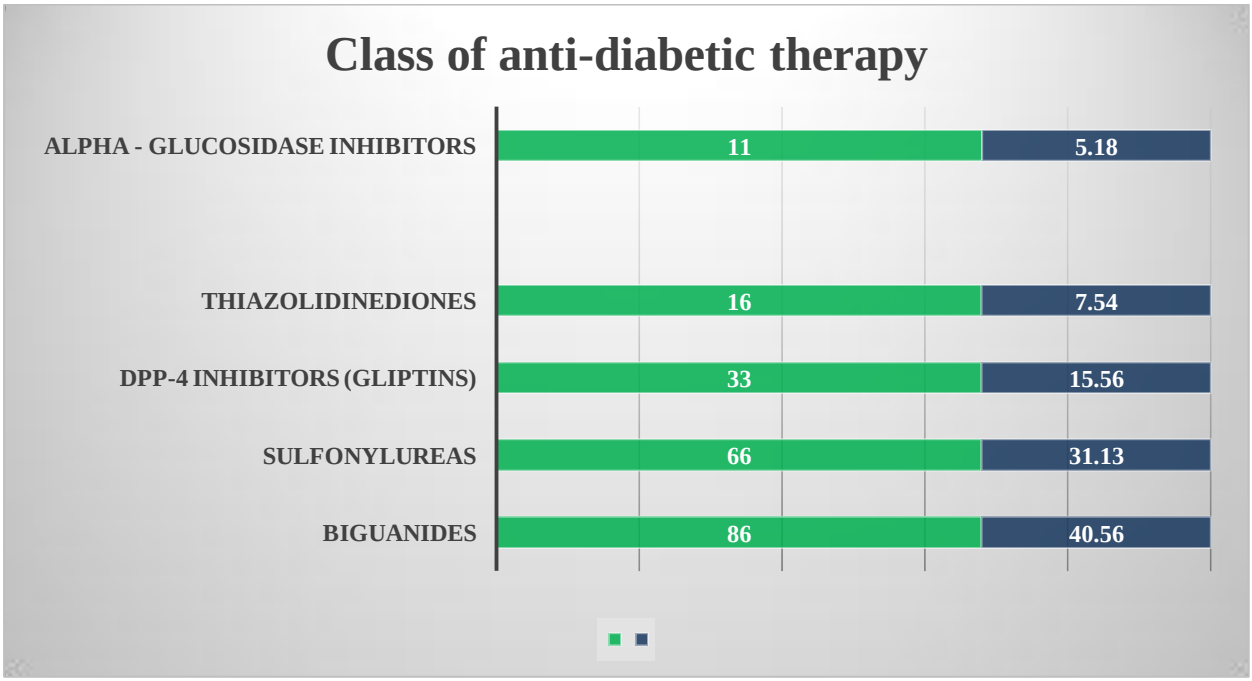


Table 12: Class of anti-diabetic therapy (TWO DRUG THERAPY)

Classes of drugs	Number of patients (n=156)	Percentage (%)
Biguanides +sulfonylureas	76	48.71
DPP 4 inhibitors +Biguanides	31	19.87
Biguanides +alpha glucosidase inhibitors	25	16.02
Biguanides +DPP 4 inhibitors	12	07.69
Sulfonyl ureas+Biguanides	7	04.48
Thiazolidinediones + Biguanides	5	03.20

Figure 12: Class of anti-diabetic therapy (TWO DRUG THERAPY)

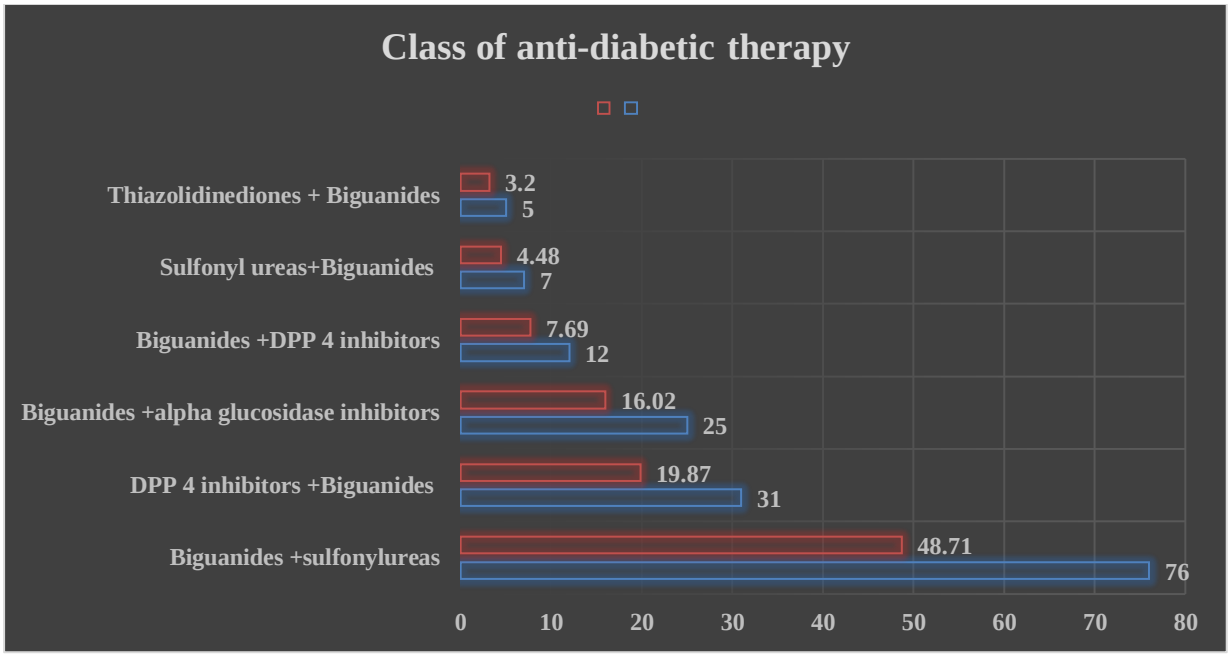


Table 13: Class of anti-diabetic therapy

(THREE DRUG THERAPY)

Class of drugs	Number of patients (n=36)	Percentage (%)
Biguanides +sulfonyl ureas+ thiazolidinedione	12	33.33
DPP 4 inhibitors + Biguanides + sulfonyl ureas	09	25.00
DPP 4 inhibitors+ Biguanides+ alpha glucosidase inhibitors	5	13.88
Biguanides+ DPP 4 inhibitors+ sulfonyl ureas	4	11.11
Biguanides+ sulfonyl ureas+ DPP 4 inhibitors	3	08.33
Sulfonyl ureas+ Biguanides+ thiazolidinedione	2	05.55
Alpha glucosidase inhibitors+ Biguanides+ thiazolidinedione	1	02.77

Figure 13: Class of anti-diabetic therapy
(THREE DRUG THERAPY)

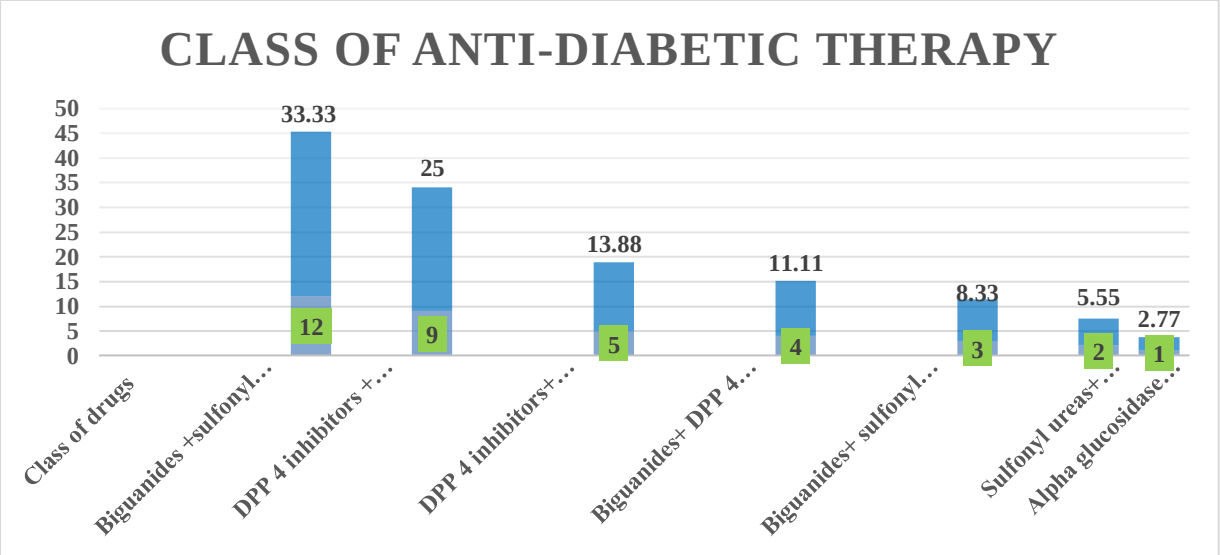


Table 14: Class of anti-diabetic therapy

(FOUR DRUG THERAPY)

Classes of drugs	Number of patients (n=22)	Percentage (%)
Biguanides + sulfonyl ureas + DPP 4 inhibitors + thiazolidinedione	10	45.45
DPP 4 inhibitors + Biguanides + sulfonyl ureas+ alpha glucosidase inhibitors	08	36.36
DPP 4 inhibitors + Biguanides + sulfonyl ureas + thiazolidinedione	04	18.18

Figure 14: Class of anti-diabetic therapy

(FOUR DRUG THERAPY)

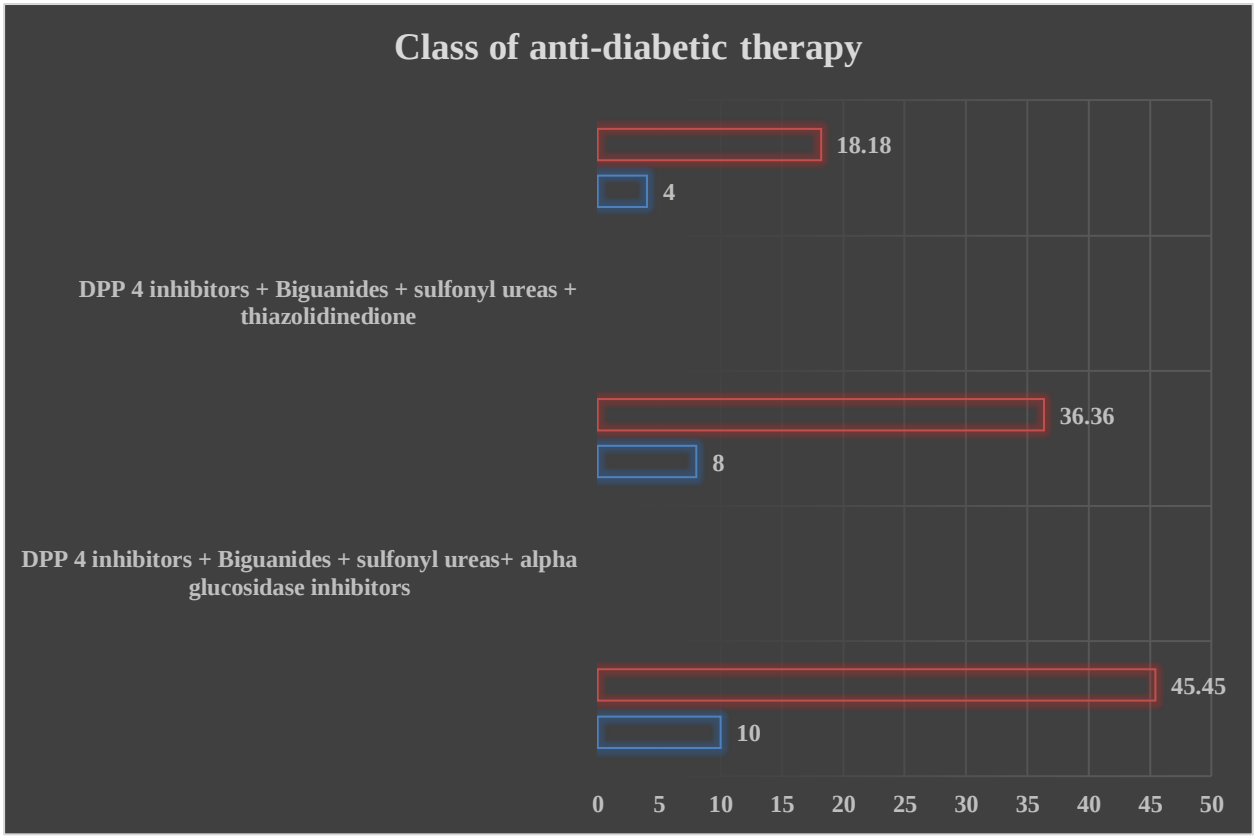


Table 15: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

MONOTHERAPY

Drugs prescribed	Number of patients (n=212)	Percentage (%)
Metformin	79	37.26
Glimepiride	51	23.94
Glibenclamide	31	14.62
Tenegliptin	22	10.37
Glipizide	08	03.77
Voglibose	7	03.30
Sitagliptin	6	02.83
Acarbose	4	01.88
Gliclazide	2	0.94
Vildagliptin	2	0.94

Figure 15: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

MONOTHERAPY

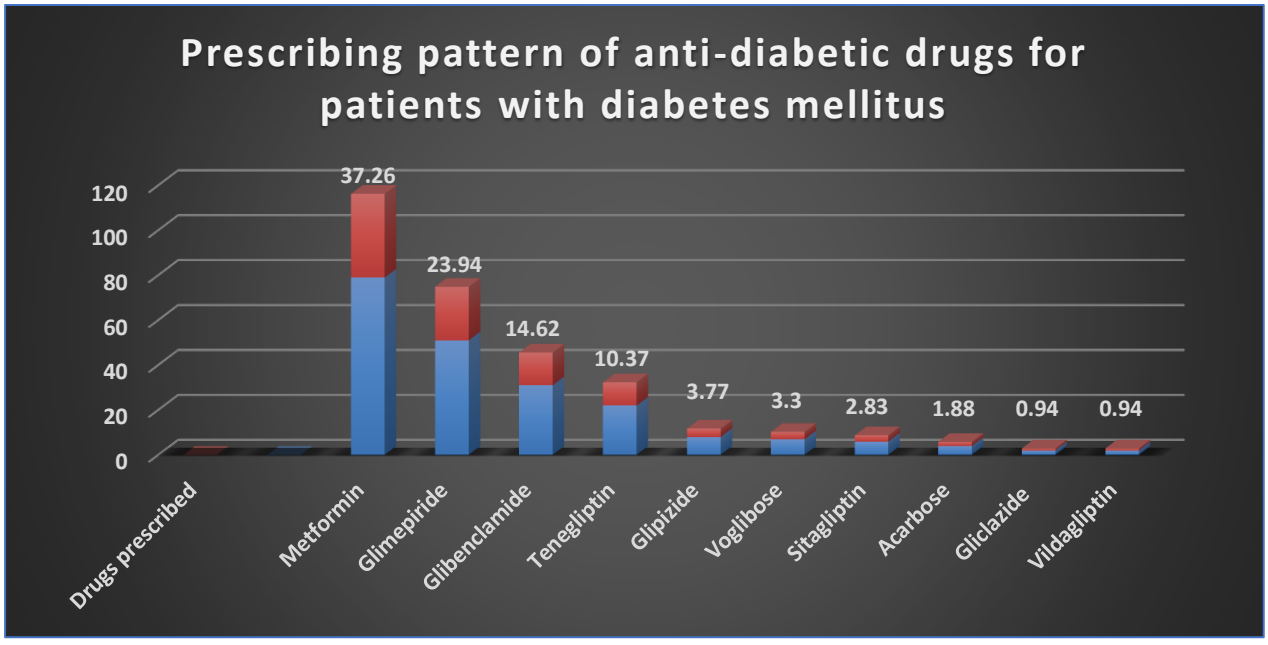


Table 16: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

TWO DRUG THERAPY

Drugs prescribed	Number of patients (n=156)	Percentage (%)
Metformin + Glimepiride	59	37.82
Vildagliptin + Metformin	41	26.28
Sitagliptin + Metformin	28	17.94
Gliclazide + Metformin	10	06.41
Teneleglitin + Metformin	9	05.76
Pioglitazone + Metformin	6	03.84
Glibenclamide + Metformin	3	01.92

Figure 16: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

TWO DRUG THERAPY

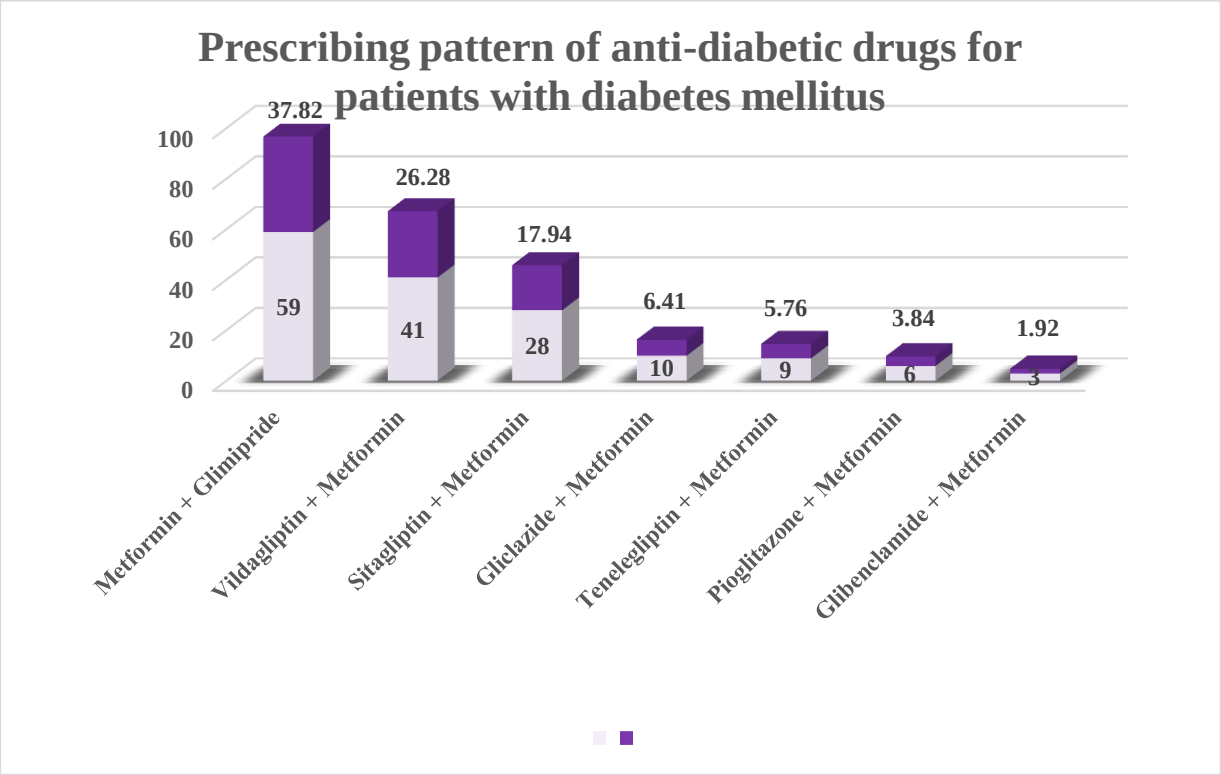


Table 17: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

THREE DRUG THERAPY

Drugs prescribed	Number of patients (n=36)	Percentage (%)
Metformin + Glimepiride + Pioglitazone	11	30.55
Vildagliptin + Metformin + Glimepiride	09	25.00
Sitagliptin + Metformin + Acarbose	7	19.44
Metformin + Sitagliptin + Glimepiride	4	11.11
Metformin + Glimepiride + Teneligliptin	2	05.55
Gliclazide + Metformin + Pioglitazpne	2	05.55
Voglibose + Metformin + Pioglitazone	1	02.77

Figure 17: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

THREE DRUG THERAPY

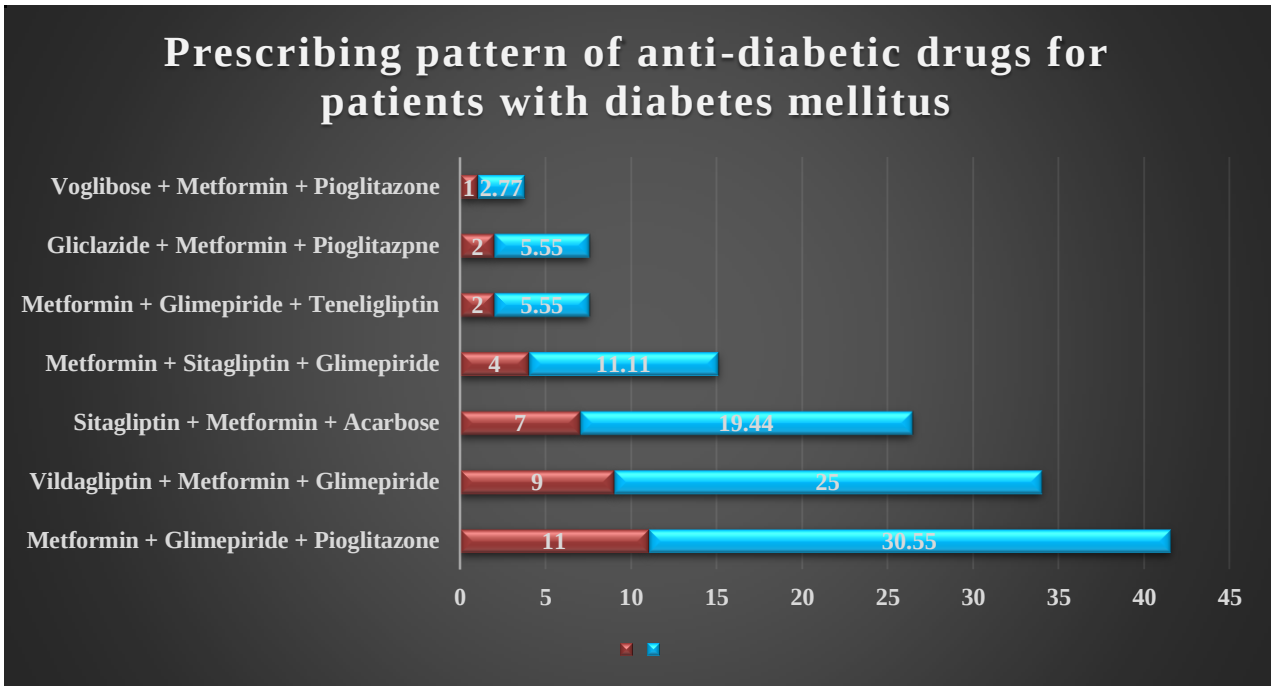


Table 18: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

FOUR DRUG THERAPY

Drugs Prescribed	Number Of Patients (n=22)	Percentage (%)
Metformin + Glimepiride + Sitagliptin + Pioglitazone	10	45.45
Vildagliptin + Metformin + Glimepiride + Acarbose	08	36.36
Sitagliptin + Metformin + Glicazide +Pioglitazone	04	18.18

Figure 18: Prescribing pattern of anti-diabetic drugs for patients with diabetes mellitus

FOUR DRUG THERAPY

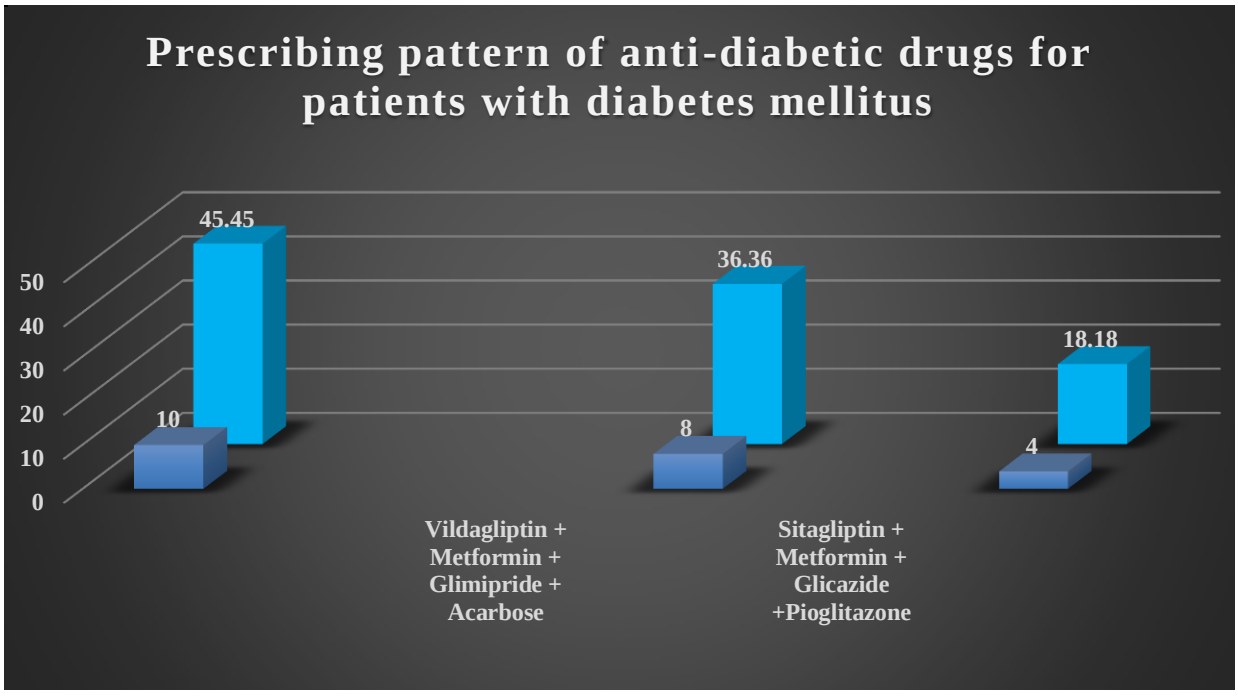
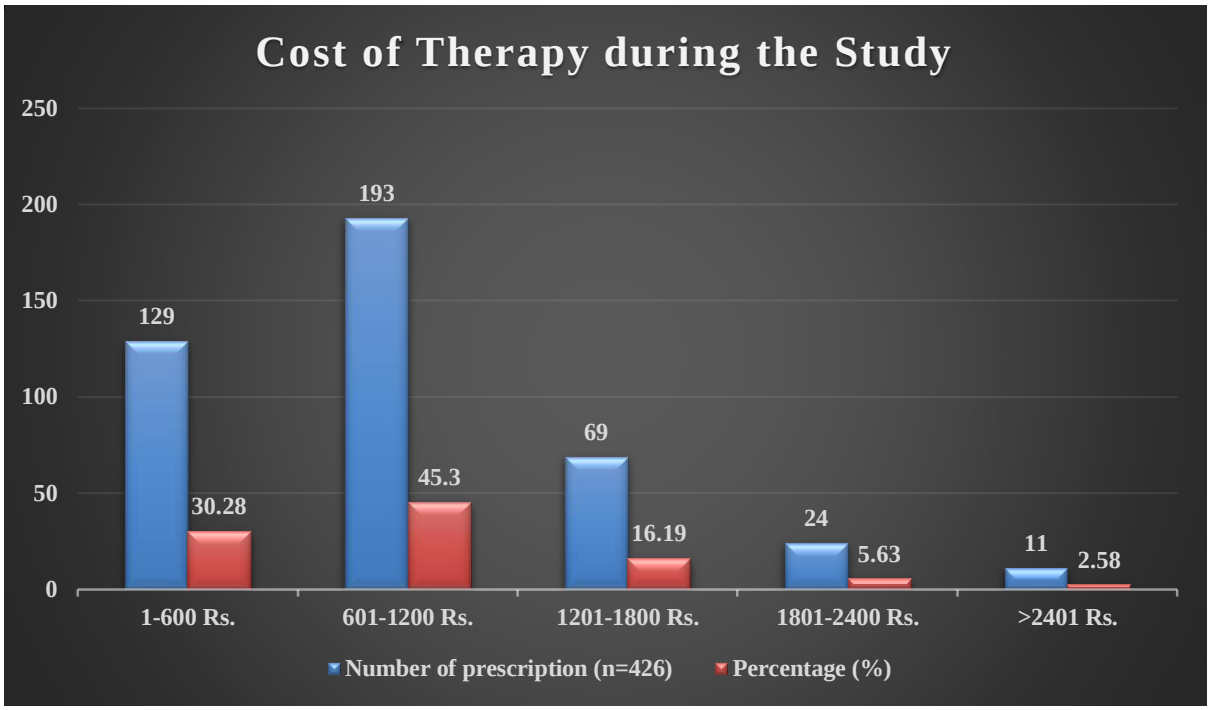


Table 19: Cost of Therapy during the Study

Cost in rupees (INR per month)	Number of prescription (n=426)	Percentage (%)
1-600 Rs.	129	30.28
601-1200 Rs.	193	45.30
1201-1800 Rs.	69	16.19
1801-2400 Rs.	24	05.63
>2401 Rs.	11	02.58

Figure 19: Cost of Therapy during the Study



5. DISCUSSION

A drug utilization study was considered to be one of the most effective methods to assess and to evaluate the prescribing attitude of a physician and helps to promote the rational use of drugs. Diabetes mellitus is a major public health problem worldwide. Its' prevalence was rising in many parts of the developing world and India, there is no exception to this. Individuals with Type 2 diabetes were considered on high priority as they are potential candidates for rapid evaluation to prevent and halt the progression of many complications.

Type 2 diabetes is a chronic disease requiring lifelong treatment. Although lifestyle modification plays an important role in managing diabetes, the usage of medication became unavoidable in many patients. A prescription based study was considered as one of the most effective methods to assess and to evaluate the prescribing pattern of medications. This study analyzed the prescribing pattern of type 2 diabetic patients who visited in the diabetic clinic, Erode.[51]

Out of 457 patients screened, 426patients were enrolled according to inclusion and exclusion criteria. Among them 62.44% were males and 37.55% were females (Table 1). The study found to be a higher incidence of diabetes among elderly patients, with a high incidence in the age group between 41-60 years (50.70%) and followed by 61‑80 years (19.24%) Table 2. Similar results were obtained in the studies done by Mandal S et al.[52] from their study it was found that the prevalence of type 2 diabetes was high in middle-aged persons, i.e. 40 to 60 years of age.

Among 426 diabetes patients living in urban areas 58.45%, followed by rural areas 27.23% in the study populations (Table 3). It was found to be that from 426 diabetes patients, 78.63% were more prone to consumption of non-vegetarian varieties of foods (Table 4). Based on the Social demographic risk factors study was observed that diabetes patients, 104 (24.41%) were more prone to alcohol consumption followed by 99 (23.23%) (Table 5).

Among the study population, family history for diabetes patients, majority of incidence in father alone 213 (50.00%) had more prone to diabetes in their family history followed by mother alone 95 (22.30%) (Table 6). This was a similar study conducted by Kannan et al.[59] Among the study population, 11 (5.44%) had their father alone suffering from diabetes, 17 (8.42%) had their mother alone suffering from diabetes, 103 (50.99%) had their other family members suffering from diabetes, 9 (4.45%) had both their father and mother suffering from diabetes and 62 (30.69%) patients had no family members with diabetes.

The study resolved that most of the patients were suffering from diabetes for 5 to 10 years, 221 (73.94%) of duration years followed by 1 to 5 years, 94 (22.06%) (Table 7). This was a similar study obtained in the studies done by Chakrabarty et al.[53] It is evident from the table that most of the patients were suffering from Type II DM for less than 5 years of durations. In this study found that there were a total of 426 co-existing illness from diabetes patients, among that 40.84% were diabetes alone, 23.23% were Diabetes + Hypertension, 11.50% Diabetes + Cardiovascular Diseases, and 13.38% were Diabetes + Asthma (Table 8). Similar study. Which was comparable with the study done by Kannan et al.[59] In this study there were a total of 306 co-existing illness in 202 patients. Hypertension was accounted for 33.33% of the total complications in diabetes patients, denoting the highest percentage of the complications. This study showed that hypertension was usually the most common co-existing illness seen with diabetes mellitus (DM).

A total of 1565 drugs were prescribed in the overall study period. 68.62% were diabetic drugs, 13.41% hypertensive drugs, 07.85% NSAIDs, 07.66% asthmatic drugs, 03.57% antidepressants, and 04.40% supplements of drugs (Table 9). Polypharmacy is associated with higher cost increase the risk of side effects, drug interactions, and non-compliance.[60] In general diabetic patients are at higher risk of developing depression. Studies suggest that diabetes doubles the risk of depression [61]. In this study, doctors prescribed antidepressants

almost 4% of the total drugs prescribed. These patients are more vulnerable to miss their medications, and the possibility of non-adherence is very high[62]. Doctors should be careful while prescribing Tricyclic antidepressants [TCAs] to these patients. According to one study, the use of a higher dose of Tricyclic antidepressants [TCAs] was associated with an increased risk of sudden cardiac death [63]. Overuse of vitamins were also observed [64]. In the present study, vitamins accounted only for 4.40% of the total drugs. This is low compared to other studies. This is a positive effect. Analgesics and anti-inflammatory drugs accounted for 07.85% of the total drugs. The prescriber should be aware of the interaction between OHAs and Non-steroidal anti-inflammatory drugs (NSAIDs). Concurrent use of NSAIDs and sulfonylurea may result in an increased risk of hypoglycemia[65].

In our study observed that the average number of drugs per prescription was 3.67. Which was similar results were obtained in the study done by Kannan et al.[59] The average number of drugs per prescription was 4. The high average number of drugs prescribed to patients with diabetes is not surprising. It is recognized that patients with diabetes mellitus are generally prescribed more drugs than other patients[66] In general, due to the multiple diseases, diabetes patients were at a greater risk of polypharmacy. In this study, 68.62% prescription contains two or more drugs. It shows that polypharmacy is high.

The study resolved that drugs were prescribed as monotherapy was 49.76%, two drug therapy were 36.61%, three-drug therapy were 08.45% and four-drug therapy was 05.16% (Table 10). Similar study done by Venkateswaramurthy et al.[51] Drugs were prescribed as monotherapy in 78.61% patients. Two drug combinations were prescribed to 17.92% patients and three-drug combinations were prescribed to 4.62% patients.

The study revealed that the most commonly prescribed anti-diabetic drugs in monotherapy were Biguanides 40.56%, followed by Sulfonylureas were 31.13%. Whereas in metformin 37.26% was mostly prescribed drugs in Biguanides, followed by Glimepiride 23.94% was

prescribed in Sulfonylureas (Table 11 and 15). Similar study conducted by Mandal S et al.[52] Amongst antidiabetic medications, metformin was the most commonly prescribed drug which was given in 144 (79.6%) patients followed by sulphonylureas in 121(66.9%) and pioglitazone in 37 (20.4%) patients. Most of the patients required two or more drugs to achieve glycemic control.[67,68] The most possible reason for this is that type 2 DM is a chronic disease with a progressive deterioration in glycemic control due to the continuing loss of β-cell function and henceforth. Monotherapy for type 2 diabetes may therefore not be sufficient to maintain glycemic control over time.[69]

In our study, the most commonly prescribed anti-diabetic drugs in two drug therapy was Biguanides +sulfonylureas 48.71%, followed by DPP 4 inhibitors +Biguanides 19.87%. Whereas in Metformin + Glimipride 37.82% was mostly prescribed in Biguanides + sulfonylureas, followed by Vildagliptin + Metformin 26.28% was mostly prescribed drugs in DPP 4 inhibitors +Biguanides (Table 12 and 16). Similar study conducted in two drug therapy done by Geetha et al.[50] The add-on therapy of sulfonylurea to metformin is the common procedure after the metformin fails to control glycemic levels, sulfonylureas have been associated with hypoglycemia, sometimes need of hospitalizations, particularly in elderly patients [70].

In three drugs therapy of anti-diabetic, mostly prescribed drugs was Biguanides +sulfonyl ureas+ thiazolidinedione 33.33%, followed by DPP 4 inhibitors + Biguanides + sulfonylureas 25%. In that Metformin + Glimepiride + Pioglitazone 30.55% was mostly prescribed in Biguanides +sulfonyl ureas+ thiazolidinedione, followed by Vildagliptin + Metformin + Glimepiride 25% was mostly prescribed in DPP 4 inhibitors + Biguanides + sulfonylureas (Table 13 and 17) the most common combination was observed by Venkateswaramurthy et al.[51] Under three-drug combinations, the combination of metformin, pioglitazone, and glimepiride (2.86%) was highly prescribed followed by metformin + glimepiride + sitagliptin

(1.71%). Pioglitazone comes under thiazolidinediones that help in increasing insulin sensitivity in target tissues. In combination with other hypoglycemic drugs, pioglitazone is an effective protocol in attaining glycemic control. Voglibose comes under alpha-glucosidase inhibitor that lowers the daily glycemic conversions and inhibits overwork of the pancreatic beta cells and shows a little effect on insulin sensitivity in patients with NIDDM. Several new drugs with certain advantages like high glucose-lowering efficacy are available, that include injectable glucagon-like peptide-1 agonists and DPP-4 inhibitors. These agents offer a low risk of hypoglycemia combined with sustained weight loss. Oral drug therapy for type 2 DM will achieve greater control on glycemic targets when it's used appropriately and safely with certain assistance patients can achieve glycemic targets within a short period. However, the progressive nature of type 2 DM usually requires a combination of two or more oral agents in the longer term, often as a prelude to insulin therapy.[71]

The study revealed that, four drug therapies the most commonly prescribed drug was Biguanides + sulfonylureas + DPP 4 inhibitors + thiazolidinedione 45.45%, followed by DPP 4 inhibitors + Biguanides + sulfonyl ureas+ alpha-glucosidase inhibitors 36.36%. Whereas in Metformin + Gimipride + Sitagliptin + Pioglitazone 45.455 was mostly prescribed drugs in Biguanides + sulfonylureas + DPP 4 inhibitors + thiazolidinedione, followed by Vildagliptin + Metformin + Glimipride + Acarbose 36.36% was mostly prescribed drugs in DPP 4 inhibitors + Biguanides + sulfonyl ureas+ alpha-glucosidase inhibitors (Table 14 and 18).

Cost of drug therapy is a cause for non-adherence. In this study resolved that higher cost in rupees in per month 601-1200 Rs. were 45.30% had consumed, followed by 1-600 Rs. were 30.28% had consumed (Table 19). Which is a similar study conducted by Kannan et al.[59] Cost of prescription is important in chronic diseases like diabetes. One of the better approaches to decrease the prescription cost is to prescribe cheaper brands. A study from Nepal reported a huge variation in cost among brands of a particular drug [72]. Also in India,

huge variations in the cost of antidiabetic medications have been documented [73]. A similar finding has been seen in other developing countries [74]. Thus there is a huge scope in reducing the prescription cost by prescribing cheaper alternatives. However, while choosing cheaper brands, one should keep in mind the quality of the brands.

6. CONCLUSION

The current study reported that type 2 diabetes was more prevalent in males than in females. A total of 426 patients had co-morbid conditions along with diabetes. Commonly seen co-morbid condition in the study was hypertension.

In this study, 426 anti-diabetic drugs prescribed, among that, the physician's most well-liked single-drug therapy more than multiple drug therapy and also the most often prescribed category was Biguanides category of anti-diabetic agents. Among Biguanides, Metformin was the foremost often utilized anti-diabetic drugs. The foremost prevalent combination of the drug was a two-drug therapy of Biguanides +sulfonylureas, among these combinations, Metformin + Glimipride was the foremost often utilized anti-diabetic drugs. Followed by 3 drug therapy were Biguanides +sulfonyl ureas+ thiazolidinedione and 4 drug therapies were Biguanides + sulfonylureas + DPP 4 inhibitors + thiazolidinedione.

Pharmacists can contribute drastically to promote the rational use of medicines, even in resource-limited settings. This, of course, requires strong collaboration between different institutions and commitments of the pharmacists to the cause. Pharmacist medication review, patient counseling and telephone follow-up can limit the Adverse Drug Reactions. Medication discrepancies before and after discharge were common targets of intervention.

These efforts need to encompass both studies to understand the high-quality strategies to support more successfully the splendid use of medications according to regular guidelines and progressive tools to support physician decision making and affected person compliance.

This study suggests that patient expertise about the drugs is low. In this find out about cost of tablets per prescription was found to be very high. The price of prescription can be decreased employing deciding on most monetary drugs without altering its quality.

7. REFERENCES

1. Rajalekshmi V, Amsaveni V. Antibiotic Susceptibility of Bacterial Pathogens Isolated From Diabetic Patients. *Int J Microbiol Res.* 2011;2(1):273-275.
2. Murugan S, Mani KR, Devi U. Prevalence of MSRA Among Diabetic Patients With Foot Ulcers And Their Antimicrobial Susceptibility Pattern. *J Clin Diagn Res* 2008;2(9):979-984.
3. Wild S, Roglic G, Green A, Sicree R, King H. Global Prevalence of Diabetes Estimates For The Year 2000 And Projections For 2030. *Diabetes Care.* 2004;27(7): 1047-1053.
4. Ergun-Longmire B, Maclaren NK. Etiology and Pathogenesis of Diabetes Mellitus in Children. *Diabetes Metab Res Rev.* 2007;85(3):489-491.
5. https://www.who.int/news-room/fact-sheets/detail/diabetes. Accessed 05 March 2019.
6. Allende-Vigo MZ. Pathophysiologic mechanisms linking adipose tissue and cardiometabolic risk. *Endocrine Practice*. 2010;16(4):692-698.
7. Butler AE, Janson J, Bonner-Weir S, Ritzel R, Rizza RA, Butler PC. Beta-call deficit and increased beta cell apoptosis in humans with type 2 diabetes. *Diabetes*. 2003;52(1):102-110.
8. Li S, Zhao JH, Luan J, *et al.* Genetic predisposition to obesity leads to increased risk of type 2 diabetes. *Diabetologia*. 2011;54(4):776-782.
9. Nguyen NT, Nguyen XM, Lane J, Wang P. Relationship between obesity and diabetes in a US adult population: findings from the National Health and Nutrition Examination Survey, 1999-2006. *Obesity Surgery*. 2011;21(3):351-355.
10. Link CL, McKinlay JB. Disparities in the prevalence of diabetes: is it race/ethnicity or socio-economic status? Results from the Boston Area Community Health (BACH) survey. *Ethnicity & Disease*. 2009;19(3):288-292.

11. Clark MD, Kathleen M, Susan G. Findings from the study to help improve early evaluation and management of risk factors leading to diabetes (shield). *Diabetes Care.* 2007;30(11): 2868-2873.
12. Maclaren NK, Alkinson MA. Insulin-dependent diabetes mellitus: the hypothesis of molecular mimicry between islet cell antigens and microorganisms. *Mol Med Today.* 1997;3(5):76-83.
13. Urakami T, Suzuki J, Mugishima H, Amemiya S *et al.* Screening and treatment of childhood type 1 and type 2 diabetes mellitus in Japan. *Pediatr Endocr Rev*. 2000;1(8):51-61.
14. Hales CN, Barker DJ. The thrifty phenotype hypothesis. *Br Med Bull.* 2001;60(5):5-20.
15. Pettitt DJ, Aleck KA, Baird HR, Carraher MJ, Bennett PH, Knowler WC. Congenital susceptibility to NIDDM. Role of intrauterine environment. *Diabetes.* 1987;37(8):622-628.
16. Samuel KL.Type 2 diabetes in children and adolescents. American Diabetes Association. *Diabetes Care*. 2007;23(1):381-389.
17. Scott LJ, Mohlke KL, Bonnycastle LL, Willer CJ. A genome-wide association study of type 2 diabetes in Finns detects multiple susceptibility variants. *Science*. 2007;316(4):1341-1345.
18. Reaven GM. Role of insulin resistance in human disease (syndrome X): an expanded definition. *Annu Rev Med.* 1993;44(2):121-131.
19. Kahn CR, Flier JS, Bar RS, Archer JA *et al*. The syndromes of insulin resistance and acanthosis nigricans. Insulin-receptor disorders in man. *New Engl J Med.* 1976;294(3):739-745.

20. Oral EA, Simha V, Ruiz E. Leptin-replacement therapy for lipodystrophy. *New Engl J Med.*. 2002;346(1):570-578.
21. Galloway PJ, Donaldson MD, Wallace AM. Sex hormone binding globulin concentration as a prepubertal marker for hyperinsulinaemia in obesity. *Arch Dis Child*. 2007;85(4):489-491.
22. Karnieli E, Cohen P, Barzilai N, Ish-Shalom Z, Armoni M, Rafaelov R. 1985 Insulin resistance in Cushing's syndrome. *Horm Metab Res.* 1985;17(6):518-521.
23. Rogers DL .Acanthosis nigricans. *Semin Dermatol.* 1991;10(2):160-163.
24. Tominaga K, Kurata JH, Chen YK, Fujimoto E, Miyagawa S. Prevalence of fatty liver in Japanese children and relationship to obesity. An epidemiological ultrasonographic survey. *Dig Dis Sci.* 1995;40(2):2002-2009.
25. Kautzky-Willer A., Harreiter J., Pacini G. Sex and gender differences in risk, pathophysiology and complications of type 2 diabetes mellitus. *Endocr Rev.* 2016;37(3):278–316.
26. Chilelli NC, Burlina S, Lapolla A. AGEs, rather than hyperglycemia, are responsible formicrovascular complications in diabetes: a 'glycoxidation-centric' point of view. *Nutr Metab Cardiovas.* 2013;23(4):913-919.
27. Nguyen DV, Shaw LC, Grant MB. Inflammation in the pathogenesis of microvascular complications in diabetes. *Front Endocrinol.* 2012;3(1):345-356.
28. Khoury JC, Kleindorfer D, Alwell K *et al.* Diabetes mellitus: a risk factor for ischemic stroke in a large biracial population. *Stroke*. 2013;44(6):1500-1504.
29. Jia G, DeMarco VG, Sowers JR. Insulin resistance and hyperinsulinaemia in diabetic cardiomyopathy. *Nat Rev Endocrinol.* 2016;12(3):144-153.
30. Boulton AJ. The Diabetic Foot: A Global View. *Diabetes Metab Res Rev* 2000;16(7):1520-7560.

31. Schwingshackl L, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet.* 2015;115(2):780-800.
32. Jenkins DJA, Wolever TMS, Taylor RH *et al*. 1981 glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr.* 1981;34(6):362-366.
33. Rodbard HW, Blonde L, Braithwaite SS *et al*. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the management of diabetes mellitus. *Endocr Pract.* 2007;13(1):1-68.
34. Salmeron J, Manson JE, Stampfer MJ *et al*. Dietary fiber, glycemic load, and risk of noninsulin-dependent diabetes mellitus in women. *JAMA.* 1997;277(4):472–477.
35. Mottalib A, Kasetty M, Mar JY *et a*l. Weight management in patients with type 1 diabetes and obesity. *Curr Diabetes Rep.* 2017;2(7):17:92.
36. Thrashe J. Pharmacologic Management of Type 2 Diabetes Mellitus: Available Therapies. *Am J Cardiol.* 2017;120(1):4-16.
37. http://care.diabetesjournals.org/content/36/Supplement_2/S127. Accessed 04 April 2019.
38. Garber AJ, Abrahamson MJ, Barzilay JI *et al.* Consensus Statement by the American Association of Clinical Endocrinologists and American College of Endocrinology on the Comprehensive Type 2 Diabetes Management Algorithm - 2017 Executive Summary. *Endocr Pract.* 2017;32(4):207-238.
39. American Diabetes Association**.** Pharmacologic approaches to glycemic treatment**.** *Diabetes Care*. 2017;40(1):64-67.
40. Zonszein P, Groop PH. Strategies for diabetes management: using newer oral combination therapies early in the disease**.** *Diabetes Ther*. 2016;7(1):621-639.

41. Drucker DJ, Nauck MA. The incretin system: glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase-4 inhibitors in type 2 diabetes. *Lancet*. 2006;368(1):1696-1705.
42. Madsbad S. Review of head-to-head comparisons of glucagon-like peptide-1 receptor agonists**.** *Diabetes Obes Metab.* 2016;18(1):317-332.
43. http://docs.boehringeringelheim.com/Prescribing%20Information/PIs/Glyxambi/Glyxambi.pdf. Accessed 22 March 2019.
44. http://www.accessdata.fda.gov/drugsatfda_docs/label/2016/022426s009lbl.pdf. Accessed 30 March 2019.
45. Monsalve FA, Pyarasani RD, Delgado-Lopez F, Moore-Carrasco R. Peroxisome proliferator-activated receptor targets for the treatment of metabolic diseases. *Mediators Inflamm.* 2013;44(1):549-627.
46. Nissen SE, Wolski K. Effect of rosiglitazone on the risk of myocardial infarction and death from cardiovascular causes**.** *N Engl J Med.* 2007;356(1):2457-2471.
47. Mitka M. FDA eases restrictions on the glucose-lowering drug rosiglitazone. *JAMA.* 2013;310(11):2604-2608.
48. Sharma JK, Parmar SP. Prescribing pattern of anti-diabetic drugs in patients suffering from type 2 diabetes mellitus with co-existing hypertension in a tertiary care teaching hospital. *Int J Basic Clin Pharmacol*. 2018;7:761-766.
49. MR Misbahuddin, AM Hussam, JG Zohair, MS Ziaullah. Anti-diabetic drug utilization patterns in a government hospital in Saudi Arabia. *Trop J Pharm Res.* 2018;17(6):1193-1200.
50. Geetha P, Shanmugasundharam P. Drug utilization evaluation of antidiabetic drugs among type 2 diabetes patients of tamil nadu. *Asian J Pharm Clin Res.* 2017;10(9):202-205.

51. Venkateswaramurthy N, Md. Shajeem S and Sambathkumar R: Prescribing pattern of antidiabetic drugs in type-2 diabetic patients. *Int J Pharm Sci Res*. 2016;7(11):4550-4555.
52. Mandal S, Maiti T, Das AK, Das A, Mandal A, Sarkar BS, et al. Drugutilization study in patients with type 2 diabetes mellitus diabetes clinic of a tertiary care hospital in rural Bengal. *Int J Basic Clin Pharmacol.* 2016;5:1647-54.
53. Tamoghna Maiti, Sourav Chakrabarty, Sonai Mandal, Amrita Panda, Tanmoy Gangopadhyay. A study on drug utilization pattern in patients of type II diabetes mellitus attending referral diabetic clinic at a tertiary care teaching hospital in rural bengal. *Eur J Pharm Med Res.* 2016;3(9):641-647.
54. Siddhartha Nuthakki, Sivaprasad Pendyala, Charan Vallabhu, Naveenbabu Kilaru, Siva R Challa. An Assessment of Anti-hyperglycemic Drug Utilization Patterns and Adherence to AACE/ACE 2015 Guidelines in South Indian Tertiary Care Teaching Hospital. *Ind J Pharm Prac.* 2016;9(4):226-230.
55. Kayamkani abedulla khan. Drug utilization study of antihyperglycemic agents in outpatients at opd clinics in south indian metropolitan city. *Int J Pharm Pharm Sci.* 2016;8(5):58-61.
56. Ramesh Gorre, Mahesh Ondanti, Jaya Jeevitha, Divya Jose, Sandeep Ankam. Drug utilization and evaluation of hypoglycemic agents in a tertiary care hospital. *Int J Innov Pharm.* 2015;3(8):1078-1085.
57. Pratiksha Kasture, Shivani Kasture, Nilesh Khade and Rahul Shivaji Adnaik. A prospective study on drug utilization pattern of anti-diabetic drugs in rural areas of Islampur, India. *Der Pharmacia Lettre.* 2015;7(5):33-37.

58. A Alti, SP Latha, GL Nagarjun, G Nagaraju, C Gopinath, PM Madhav. A Study on Drug Utilization Pattern and Effectiveness of Oral Hypoglycemic Agents in Diabetes Mellitus. *PharmaTutor.* 2015;3(7);31-37.
59. Kannan, Arshad, Senthil Kumar. A study on drug utilization of oral hypoglycemic agents in type-2 diabetic patients. *Asian J Pharm Clin Res.* 2011;4(4):60-64.
60. Ebbesen J, Buajorder I, Erickson J, et al. Drug-related deaths in a department of internal medicine. *Arch Intern Med.* 2001;161:2317-23.
61. Anderson RJ, Lustman PJ, Clouse RE, et al. Prevalence of depression in adults with diabetes: a systematic review. *Diabetes.* 2000;49(1):A64.
62. Taj R, Khan S. A study of reasons of noncompliance to psychiatric treatment. Department of Psychiatry, Pakistan Institute of Medical Sciences, Islamabad. Available from: PAST/17 -2/Rizwan%20Taj.htm.
63. Ray WA, Meredith S, Thapa PB, *et al.* Cyclic antidepressants and the risk of sudden cardiac death. *Din Pharmacol Ther.* 2004;75(3):234-41.
64. Lamichhane DC, Giri BR, Pathak OK, Panta OB, Shankar PR. Morbidity profile and prescribing patterns among outpatients in a teaching hospital in Western Nepal. *McGill J Med.* 2006;9(2):126-33.
65. Klasco RK, editor. DRUG-REAX® system. Greenwood Village, CO: Thomson Micromedex; (Edition expires [December 2006]).
66. Good CB. Polypharmacy in elderly patients with diabetes. *Diabet Spectr.* 2002;15:240-8.
67. Vengurlekar S, Shukla P, Patidar P, Bafna R, Jain S. Prescribing pattern of antidiabetic drugs in Indore city hospital. *Indian J Pharm Sci*. 2008; 70:637-40.
68. Dhanraja E, Raval AD, Yadav R, Bhansali A, Tiwari P. Prescribing pattern of antidiabetic drugs and achievement of glycemic control in T2DM patients in tertiary

care hospital in North India. International Journal of Diabetes in Developing Countries. *Indian J Pharm Sci*. 2013:33:140-6.

69. Barnett AH. Redefining the role of thiazolidinediones in the management of type 2 diabetes. *Vasc Health Risk Manag.* 2009;5(1):141-51.
70. Lipska KJ, Ross JS, Wang Y, Inzucchi SE, Minges K, Karter AJ, et al. National trents in US hospital admissions for hypoglycemia and hypoglycemia among Medicare beneficiaris, 1999 to 2011. *JAMA Intern Med.* 2014;174(7):1116-24.
71. Krentz AJ, Patel MB, Bailey CJ. New drugs for type 2 diabetes mellitus: what is their place in therapy? *Drugs.* 2008; 68(15):2131-62.
72. Humagain B, Bista B, Maharjan N, *et al.* variation of prices in medicine: a market survey. *Bull Nepal Pharm Assoc.* 2003;14:7-10.
73. Perappadan BS. Shocking variation in prices of same medicine. The Hindu Newspaper, 31st August 2006, New Delhi, India. Available from: URL:http://www.hindu.com (accessed on 21st)
74. Pear R, Freudenheim M. Web site shows variation in drug prices. New York Times, October 19, 2006, New York. Available from: URL:http://query.nytimes.com (accessed on 21 December 2006).

PATIENT INFORMATION FORM

Dear participant,

We are students of J.K.K. NATTRAJA COLLEGE OF PHARMACY, Kumarapalayam. Currently conducting a project entitled **"A prospective study on prescribing patterns of anti-diabetic drugs for patients with type- II diabetes mellitus at a tertiary care hospital"**. As a part of project, we need to collect data including past, social and family history, and history of drug and food allergy, lab reports and current medical data. However, no identifiable personal data will be disclosed.

Thank you very much for your kind participation.

CONSENT FORM

I, _____ have read and understand the above information. I have to allow my data to be eflicted for the project work.

_____ _____

Signature of participant Date

Translated by:

J. K.K.NATTRAJA COLLEGE OF PHARMACY, KUMARAPALAYAM-638183

DEPARTMENT OF PHARMACY PRACTICE

DATA ENTRY FORM

CASE NO:

PATIENT DETAILS:

Name:	**I P no:**
Age:	**dept:**
Sex:	**DOA:**
Wt:	**DOD:**
Ward:	

REASON FOR ADMISSION:

PAST MEDICAL HISTORY:

PAST MEDICATION HISTORY:

SOCIAL HISTORY:

PHYSICAL EXAMINATION & VITAL SIGNS:

LABORATORY INVESTIGATION:

DIAGNOSIS:

TREATMENT CHART

Sl. no	Generic name	Trade name	Dose	Frequency	Date							
1.												
2.												
3.												
4.												
5.												
6.												
7.												
8.												
9.												
10.												
11.												
12.												
13.												
14.												
15.												

PROGRESSION CHART:

OBSERVATION	**DAY 1**	**DAY 2**	**DAY 3**	**DAY 4**	**DAY 5**
	DAY 6	**DAY 7**	**DAY 8**	**DAY 9**	**DAY 10**

DISCHARGE SUMMARY:

NAME OF THE STUDENT:

CASE COLLECTED ON:

SIGNATURE OF STAFF: