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Patient Compliance in Diwania Hospital Treatment of Type 2 Diabetes Mellitus

Graduation Research

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By:

Ahmed Kareem Darney & Ahmed Nadhum Abd

Supervised By:

Dr.Bassim I. Mohammad

Professor of Clinical Pharmacology

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بسم الله الرحمن الرحيم

{يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ وَ الَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ}

صدق الله العلي العظيم

(سورة المجادلة- اية11)

Supervisor Certificate

I Certify that this Project

(Patient Compliance In Diwania Hospital , Treatment Of Type 2 Diabetes Mellitus)

was prepared under our supervision at the College Of Pharmacy, University of Al-Qadisiyah as Graduation research

Signature

Professor

Dr.Bassim I. Mohammad

DEDICATION

To my lovely father,

My great mother,

my family and professors,

and to all who quench homeland with their blood

to make us live peacefully

Ahmed Kareem **Ahmed Nadhum Abd**

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Owing to the blessing of GOD , this work has come to light

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Summery

Diabetes is a chronic condition caused by an absolute lack of insulin or relative lack of insulin as a result of impaired insulin secretion and action. In the long term these metabolic abnormalities contribute to the development of complications such as cardiovascular disease (CVD), retinopathy nephropathy, and neuropathy and a higher risk of cancer. The importance of regular follow-up of diabetic patients with the health care provider is of great significance in averting any long term complications. Treatment adherence in diabetes is an area of interest and concern to health professionals and clinical researchers even though a great deal of prior research has been done in the area.

This study included a total of 100 type 2 diabetic patients (50 men and 50 women) with a mean age of 45 years (18 to 65). The majority of patients with diabetes can significantly reduce the chances of developing long-term complications by improving self-care activities. Despite this fact, compliance or adherence to these activities has been found to be low, especially when looking at long-term changes. One hundred percent were treated with insulin only, percent of patients who adhere with treatment is 15%. Decreasing the patient's glycosylated hemoglobin level may be the ultimate goal of diabetes self-management but it cannot be the only objective in the care of a patient. Percent of patients who are measuring HbA1c is 20% and percent of patients who are measuring RBS, FBS is 10%. The lack of adherence to diabetes treatment is a widely recognized problem with magnitude in national and international scenes. However, there are gaps in the Educate diabetic patient. The act of taking daily

medication and several times a day tells the patient that he suffers from a chronic disease, which can trigger a framework of denial and drug treatment. Extended therapies, common in chronic diseases, such as diabetes, where does not exist a definite improvement of the results, reveal a lower motivation to adhere to a treatment plan.

Chapter One

Introduction

1.Introduction :

Diabetes is a chronic condition caused by an absolute lack of insulin or relative lack of insulin as a result of impaired insulin secretion and action. Its hallmark clinical characteristics are symptomatic glucose intolerance resulting in hyperglycemia an alterations in lipid and protein metabolism. In the long term these metabolic abnormalities contribute to the development of complications such as cardiovascular disease (CVD), retinopathy nephropathy, and neuropathy and a higher risk of cancer.[1, 2]

Genetically, etiologically, and clinically, diabetes is a heterogeneous group of disorders. Nevertheless, most cases of diabetes mellitus can be assigned to type 1 or type 2 diabetes The term gestational diabetes mellitus (GDM) is used to describe glucose intolerance that has its onset during pregnancy.

Glucose intolerance that cannot be ascribed to causes consistent with these three classifications include specific genetic defects in β cell function or insulin action (usually genetically defective insulin receptors); diseases of the exocrine pancreas; endocrinopathies; drug- or chemical-induced; infections; and other genetic syndromes.[3]

Early glucose intolerance or prediabetes is identified as impaired fasting glucose (IFG) or impaired glucose tolerance (IGT). The pathophysiology of IFG and IGT are somewhat different, and there is not 100% concordance between them.[4] IFG results predominantly from the failure to suppress hepatic gluconeogenesis caused by insulin resistance, whereas IGT results from inadequate insulin secretion and actions in the postprandial state.

Diabetes is a serious condition that places people at risk for greater morbidity and mortality relative to the nondiabetic population Diabetes is the seventh leading cause of death in the United States, although deaths attributed to diabetes and its complication are likely to be underreported. Compared with the general population, the mortality rate for people with diabetes is about twice that for people without diabetes.

Type 2 diabetes is much more common than type 1, accounting for 90% of people with diabetes. It usually occurs in those over the age of 40 years. Type 2 diabetes is associated with a variety of disorders, including dyslipidemia, hypertension, and premature atherosclerosis. Currently termed the metabolic syndrome .

The importance of regular follow-up of diabetic patients with the health care provider is of great significance in averting any long term complications. strict metabolic control can delay or prevent the progression of complications associated with diabetes .[5,6]

Treatment adherence in diabetes is an area of interest and concern to health professionals and clinical researchers even though a great deal of prior research has been done in the area. In diabetes, patients are expected to follow a complex set of behavioral actions to care for their diabetes on a daily basis. These actions involve engaging in positive lifestyle behaviors, including following a meal plan and engaging in appropriate physical activity; taking medications insulin or an oral hypoglycemic agent) when indicated; monitoring blood glucose levels; responding to and self treating diabetes- related symptoms; following foot-care guidelines; and seeking individually appropriate medical care for diabetes or other health-related problems.[7]

Chapter Two

Literatures Review

2. Literature review :

2.1. Diabetes Mellitus :

Diabetes mellitus is a clinical syndrome characterized by an increase in plasma blood glucose (hyperglycaemia), caused by an absolute or relative insulin deficiency.

It has been defined by the World Health Organization (WHO), on the basis of laboratory findings, as a fasting venous plasma glucose concentration of 7.0 mmol/L or more (on more than one occasion or once in the presence of diabetes symptoms(or a random venous plasma glucose concentration of 11.1 mmol/L or more. Sometimes an oral glucose tolerance test (OGTT) may be required to establish the diagnosis in equivocal cases.[8]

The incidence of diabetes is rising. Globally, it is estimated that 366 million people had diabetes in 2011 approximately 8.3% of the world population, or 3 new cases every 10 seconds), and this figure is expected to reach 552 million by 2030. This global pandemic principally involves type 2 diabetes, the prevalence of which varies considerably around the world , being associated with differences in genetic as well as environmental factors such as greater longevity, obesity, unsatisfactory diet, sedentary lifestyle, increasing urbanization and economic development.[9]

Diabetes has many causes (see Table 2-1) but is most commonly due to type 1 or type 2 diabetes .

Table 2-1 : Aetiological classification of diabetes mellitus

Type 1 diabetes
- Immune-mediated
- Idiopathic

Type 2 diabetes

Other specific types
- Genetic defects of β-cell function
- Genetic defects of insulin action (e.g. leprechaunism, lipodystrophies)
- Pancreatic disease (e.g. pancreatitis, pancreatectomy, neoplastic disease, cystic fibrosis, haemochromatosis, fibrocalculous pancreatopathy)
- Excess endogenous production of hormonal antagonists to insulin, e.g.
  Growth hormone – acromegaly
  Glucocorticoids – Cushing's syndrome
  Glucagon – glucagonoma
  Catecholamines – phaeochromocytoma
  Thyroid hormones – thyrotoxicosis
- Drug-induced (e.g. corticosteroids, thiazide diuretics, phenytoin)
- Uncommon forms of immune-mediated diabetes (e.g. IPEX (immunodysregulation polyendocrinopathy X) syndrome)
- Associated with genetic syndromes (e.g. Down's syndrome; Klinefelter's syndrome; Turner's syndrome; DIDMOAD (Wolfram's syndrome) – diabetes insipidus, diabetes mellitus, optic atrophy, nerve deafness; Friedreich's ataxia; myotonic dystrophy)

Gestational diabetes

2.2. Functional Physiology :

An understanding of the signs and symptoms associated with diabetes is based on a knowledge of glucose metabolism and the metabolic effects of insulin in nondiabetic and diabetic subjects during the fed (postprandial) and fasting (postabsorptive) states.

Blood glucose is tightly regulated and maintained within a narrow range. This is essential for ensuring a continuous supply of glucose to the central nervous system. The brain has little capacity to store energy in the form of glycogen or triglyceride and the blood–brain barrier is largely impermeable to fatty acids, so the brain depends on the liver for a constant supply of glucose for oxidation and hence generation of adenosine triphosphate(ATP).

Glucose homeostasis is achieved through the coordinated actions of multiple organs, but mainly reflects a balance between the entry of glucose into the circulation from the liver, supplemented by intestinal absorption of glucose after meals, and the uptake of glucose by peripheral tissues, particularly skeletal muscle and brain.[10]

After ingestion of a meal containing carbohydrate, normal blood glucose levels are maintained by:

• suppression of hepatic glucose production .

• stimulation of hepatic glucose uptake .

• stimulation of glucose uptake by peripheral tissues .

Insulin is synthesised in the pancreatic B-cells, initially as a polypeptide precursor, preproinsulin. The latter is rapidly converted in the pancreas to proinsulin. This forms equal amounts of insulin and C-peptide through removal of four amino acid residues. Insulin consists of 51 amino acids in two chains (the A chain contains 21 amino acids and B chain contains 30), connected by two disulphide bridges. In the islets, insulin and C-peptide (and some proinsulin) are packaged into granules. Insulin associates spontaneously into a hexamer containing two zinc ions and one calcium ion.[11] Glucose is the major stimulant to insulin release. The response is triggered both by the intake of nutrients and the release of gastro-intestinal peptide hormones.

2.2.1. Postprandial Glucose Metabolism In The Nondiabetic Individual :

After food is ingested, blood glucose concentrations rise and stimulate insulin release. Insulin is the key to efficient glucose utilization. It promotes the uptake of glucose, fatty acids, and amino acids and their conversion to storage forms in most tissues. Insulin also inhibits hepatic glucose production by suppressing glucagon and its effects.[12] In muscle, insulin promotes the uptake of glucose and its storage as glycogen. It also stimulates the uptake of amino acids and their conversion to protein. In adipose tissue, glucose is converted to free fatty acids and stored as triglycerides. Insulin also prevents a breakdown of these triglycerides to free fatty acids, a form that may be transported to other tissues for utilization. The liver does not require insulin for glucose transport, but insulin facilitates the conversion of glucose to glycogen and free fatty acids.[13]

2.2.2. Fasting Glucose Metabolism In The Nondiabetic Individual :

As blood glucose concentrations drop toward normal during the fasting state, insulin release is inhibited. Simultaneously, a number of counter-regulatory hormones that oppose the effect of insulin and promote an increase in blood sugar are released (e.g., glucagon, epinephrine, growth hormone, cortisol). As a result, several processes maintain a minimum BG concentration for the central nervous system. Glycogen in the liver is broken down into glucose (glycogenolysis). Amino acids are transported from muscle to liver ,where they are converted to glucose through gluconeogenesis. Uptake of glucose by insulin-dependent tissues is diminished to conserve glucose for the brain. Finally, triglycerides are broken down into free fatty acids, which are used as alternative fuel sources.[9]

2.3. Pathogenesis Of Diabetes :

2.3.1. Type 1 Diabetes :

2.3.1.1. Immune Mediated :

This form, previously called “insulin dependent diabetes” accounts for 5–10% of diabetes and is due to cellular-mediated autoimmune destruction of the pancreatic b-cells. Autoimmune markers include islet cell autoantibodies, autoantibodies to insulin, autoantibodies to GAD (GAD65), autoantibodies to the tyrosine phosphatases IA-2 and

IA-2b, and autoantibodies to zinc transporter 8 (ZnT8).[14] Type 1 diabetes is defined by the presence of one or more of these autoimmune markers.

The rate of b-cell destruction is quite variable, being rapid in some individuals(mainly infants and children) and slow in others (mainly adults). Immune-mediated diabetes commonly occurs in childhood and adolescence, but it can occur at any age . These patients are also prone to other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, Addison's disease, vitiligo, celiac disease, autoimmune hepatitis, myasthenia gravis, and pernicious anemia.[15]

2.3.1.2. Idiopathic Diabetes :

Some forms of type 1 diabetes have no known etiologies .This form of diabetes is not autoimmune mediated but is strongly inherited and more common in black and Asian people. The insulin requirement of affected people can fluctuate widely and the cause is unknown.[15]

2.3.2. Type 2 Diabetes :

This form, previously referred to as "noninsulin- dependent diabetes" accounts for ;90–95% of all diabetes. Type 2 diabetes encompasses individuals who have insulin resistance and usually relative (rather than absolute) insulin deficiency. At least initially, and often throughout their lifetime, these individuals may not need insulin treatment to survive.[16]

There are various causes of type 2 diabetes. Although the specific etiologies are not known, autoimmune destruction of b-cells does not occur, and patients do not have any of the other known[17] causes of diabetes. Most, but not all, patients with type 2 diabetes are obese. Obesity itself causes some degree of insulin resistance. Patients who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal region.

The risk of developing type 2 diabetes increases with age, obesity, and lack of physical activity. It occurs more frequently in women with prior GDM, in those with hypertension or dyslipidemia, and in certain racial/ethnic subgroups (African American, American Indian, Hispanic/ Latino, and Asian American). It is often associated with a strong genetic predisposition, more so than type 1 diabetes. However, the genetics of type 2 diabetes is poorly understood.[17]

2.4. Clinical Presentation :

2.4.1. Type 1 Diabetes :

As insulin secretion becomes compromised, progressive fasting hyperglycemia occurs. Glucosuria, which occurs when BG levels exceed the renal threshold, results in an osmotic diuresis, producing the classic symptoms of polyuria with compensatory polydipsia. If symptoms are untreated, weight loss occurs as glucose calories are lost in the urine and body fat and protein stores are broken down owing to increased rates of lipolysis and proteolysis. Muscle begins to metabolize its own glycogen stores and fatty acids for fuel, and the

liver begins to metabolize free fatty acids that are released in response to epinephrine and low insulin concentrations.[10]

An absolute lack of insulin may cause excessive mobilization of free fatty acids to the liver, where they are metabolized to ketones. This can result in ketonemia, ketonuria, and, ultimately, ketoacidosis. Patients present with complaints of fatigue, significant weight loss, polyuria, and polydipsia. A significant elevation in glycosylated hemoglobin (A1C) confirms weeks or months of preceding hyperglycemia.[18]

Because glucose provides an excellent medium for microorganisms, patients may present also with recurrent respiratory, vaginal, and other infections. Patients also may experience blurred vision secondary to osmotically induced changes in the lens of the eye. Treatment with insulin is essential to prevent severe dehydration, ketoacidosis, and death.[19]

2.4.2. Type 2 Diabetes :

Type 2 diabetes is typically diagnosed incidentally during a routine physical examination or when the patient seeks attention for another complaint. Because symptoms are mild in their onset, patients will rarely complain of fatigue, polyuria, and polydipsia but may admit to them during clinical examination. Because these patients have sufficient insulin concentrations to prevent lipolysis, there is usually no history of ketosis except in situations of unusual stress (e.g., infections, trauma).[18]

Weight loss is therefore uncommon because relatively high endogenous insulin levels promote lipogenesis. Macrovascular disease

is also often evident at diagnosis. Microvascular complications at diagnosis suggest the presence of undiagnosed or subclinical diabetes for 7 to 10 years. Because type 2 diabetes patients retain some pancreatic reserve at the time of diagnosis, they generally can be treated with medical nutrition therapy (MNT), physical activity, and noninsulin antidiabetic medications for several years. Nevertheless, many eventually require insulin for control of their symptoms.[20]

2.5. Diagnostic Tests For Diabetes :

Diabetes may be diagnosed based on A1C criteria or plasma glucose criteria, either the fasting plasma glucose (FPG) or the 2-h plasma glucose (2-h PG) value after a 75-g oral glucose tolerance test (OGTT) .[21]

Table 2-2 : Criteria for the diagnosis of diabetes [3]

	FPG	A1C	OGTT
Normal	<100 (5.6)	≤5.6%	<140 (7.8)
Prediabetes (i.e., impaired fasting glucose, impaired glucose tolerance)	100–125 (5.6–6.9)	≥5.7–6.4%	140–199 (7.8–11.0)
Diabetes (nonpregnant adult)	≥126 (7.0)	≥6.5%	≥200 (11.1)

The same tests are used to both screen for and diagnose diabetes. Diabetes may be identified anywhere along the spectrum of clinical

scenarios: in seemingly lowrisk individuals who happen to have glucose testing, in symptomatic patients, and in higher-risk individuals whom the provider tests because of a suspicion of diabetes. The same tests will also detect individuals with prediabetes.

2.6. Long-Term Complications :

Although acute hyperglycemic crises can occur in patients with diabetes, the long-term sequelae of diabetes account for most of the morbidity and mortality in the diabetic population.

Complications are typically designated as microvascular or macrovascular in nature. Glucose toxicity contributes most to the development and progression of microvascular complications (retinopathy, nephropathy, and neuropathy) owing to the particular susceptibility of these cell systems to elevated glucose.

Diabetes is the leading cause of new cases of adult blindness and kidney failure in the United States.[22]

About 60% to 70% of people with diabetes also have some manifestation of peripheral or autonomic neuropathy. Severe peripheral neuropathy coupled with abnormalities in immune function likely contribute to the high rate of lower extremity amputations among patients with diabetes.[22] Finally, poor glucose control promotes development of dental and oral complications and increases the risk of complications during pregnancy for both mother and fetus.

Macrovascular complications are multifactorial in their etiology and less dependent on hyperglycemia. Diabetes mellitus itself is a well-known risk factor for macrovascular disease (peripheral vascular

disease, CVD, stroke). Patients with diabetes have a threefold to fourfold elevated risk for MI and cardiovascular death compared with nondiabetic subjects.[23] Insulin resistance and the resultant hyperinsulinemia in type 2 diabetes mellitus contribute to the development of hypertension, dyslipidemia, and platelet hypersensitivity, all of which then contribute to the increased CVD risk in patients with diabetes. Thus, although tight glycemic control (A1C<7.0%) will dramatically reduce the risk for microvascular disease, its relationship to macrovascular disease is still under intense debate.[3,4]

2.7. Management Of Diabetes :

In new cases of diabetes, adequate glycaemic control can be obtained by diet and lifestyle advice alone in approximately 50%, 20–30% will need oral anti-diabetic medication, and 20–30% will require insulin. Regardless of aetiology, the choice of treatment is determined by the adequacy of residual β-cell function. However, this cannot be determined easily by measurement of plasma insulin concentration because a level which is adequate in one patient may be inadequate in another, depending upon sensitivity to insulin.[24] Consideration of the features in (Table 2-3) , and in particular the age and weight of the patient at diagnosis, usually indicate the type of treatment required. However, in each individual, the regimen adopted is effectively a therapeutic trial and should be reviewed regularly.

Table 2-3 : Classical features of type 1 and type 2 diabetes .

	Type 1	Type 2
Typical age at onset	< 40 yrs	> 50 yrs
Duration of symptoms	Weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Autoantibodies	Positive in 80–90%	Negative
Diabetic complications at diagnosis	No	25%
Family history of diabetes	Uncommon	Common
Other autoimmune disease	Common	Uncommon

2.7.1. Medical Nutrition Therapy :

For many individuals with diabetes, the most challenging part of the treatment plan is determining what to eat. It is the position of the ADA that there is not a one-size-fits-all eating pattern for individuals with diabetes. The ADA also recognizes the integral role of nutrition therapy in overall diabetes management and recommends that each person with diabetes be actively engaged in self-management, education, and treatment planning with his or her health care provider, which includes the collaborative development of an individualized eating plan .[24] Therefore, it is important that all members of the health care team be knowledgeable about diabetes nutrition therapy and support its implementation.

Table 2-4 : Dietary management of diabetes :

Aims of dietary management
• Achieve good glycaemic control • Reduce hyperglycaemia and avoid hypoglycaemia • Assist with weight management: Weight maintenance for type 1 diabetes and non-obese type 2 diabetes Weight loss for overweight and obese type 2 diabetes • Reduce the risk of micro- and macrovascular complications • Ensure adequate nutritional intake • Avoid 'atherogenic' diets or those that aggravate complications, e.g. high protein intake in nephropathy
Dietary constituents and recommended % of energy intake
• Carbohydrate: 45–60% Sucrose: up to 10% • Fat (total): < 35% n-6 Polyunsaturated: < 10% n-3 Polyunsaturated: eat 1 portion (140 g) oily fish once or twice weekly Monounsaturated: 10–20% Saturated: < 10% • Protein: 10–15% (do not exceed 1 g/kg body weight/day) • Fruit/vegetables: 5 portions daily

2.7.1.1. Carbohydrate Management :

Individuals with type 1 diabetes should be offered intensive insulin therapy education using the carbohydrate counting meal planning approach, [26] which has been shown to improve glycemic control.

Consistent carbohydrate intake with respect to time and amount can result in improved glycemic control for individuals using fixed daily insulin doses. A simple diabetes meal planning approach, such as portion control or healthful food choices, may be better suited for individuals with health literacy and numeracy concerns . [27]

2.7.1.2. Weight Loss :

Intensive lifestyle programs with frequent follow-up are required to achieve significant reductions in excess body weight and improve clinical indicators Weight loss of 2–8 kg may provide clinical benefits in those with type 2 diabetes, especially early in the disease process .[14] Although several studies resulted in improvements in A1C at 1 year not all weight-loss interventions led to 1-year A1C improvements The most consistently identified changes in cardiovascular risk factors were an increase in HDL cholesterol decrease in triglycerides and decrease in blood pressure .[28]

Weight-loss studies have used a variety of energy-restricted eating patterns, with no clear evidence that one eating pattern or optimal macronutrient distribution was ideal, suggesting that macronutrient proportions should be individualized .

Studies show that people with diabetes eat on average about 45% of their calories from carbohydrates , 36-40% of calories from fat, and ;16–18% from protein .[29]

- Macronutrients :

Carbohydrates :

Carbohydrates include sugar (sucrose), starch, and fiber and are liberally incorporated into the diet of a person with diabetes. In fact, the amount of dietary carbohydrate is the main determinant of insulin demand and is commonly used to determine the premeal insulin dose.[19] Furthermore, patients using fixed doses of insulin or

antihyperglycemic medications (e.g., sulfonylureas) must eat meals containing consistent amounts of carbohydrate to avoid hypoglycemia. Because isocaloric amounts of sucrose and starch produce the same degree of glycemia, sucrose can be substituted for a portion of the total carbohydrate intake and should be incorporated into an otherwise healthful diet.[30]

Whole grains, fruits, and vegetables high in fiber are recommended for people with diabetes, as they are for the general population. There is no evidence that larger amounts produce a differential metabolic benefit with regard to plasma glucose and lipid levels.

Protein :

For people with diabetes and no evidence of diabetic kidney disease, the evidence is inconclusive about recommending an ideal amount of protein for optimizing glycemic control or for improving one or more CVD risk measures . Therefore, these goals should be individualized. For people with diabetes and diabetic kidney disease (with albuminuria), reducing the amount of dietary protein below usual intake is not recommended because it does not alter glycemic measures, cardiovascular risk measures, or the course of glomerular filtration rate decline .[22] In individuals with type 2 diabetes, ingested protein appears to increase insulin response without increasing plasma glucose concentrations .[23]Therefore, carbohydrate sources high in protein should not be used to treat or prevent hypoglycemia. Protein's effect on blood glucose levels in type 1 diabetes is less clear.[30]

Sodium :

The ADA recommends a reduced sodium intake of less than 2,300 mg/day in normotensive and hypertensive individuals. For patients with diabetes and symptomatic heart failure, sodium should be further restricted to less than 2,000 mg/day to help reduce symptoms.[24] For all other patients, the ADA has no particular restrictions on sodium intake, but recommends individualizing amounts based on the patient's sensitivity to salt and concurrent conditions such as hypertension or nephropathy.[31]

Alcohol :

The ADA's recommendation for alcohol is consistent with general recommendations of no more than two alcoholic drinks per day for men or one drink per day for women. A drink is equivalent to 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits (each contains about 15 g of carbohydrate). Nevertheless, its caloric contribution must be considered (1 alcoholic beverage = 2 fat exchanges), and it should always be taken with food to minimize its hypoglycemic effect. In people with diabetes, light to moderate alcohol intake (one to two drinks per day) is associated with a decreased risk of CVD. A note of caution: Evening consumption of alcohol may increase the risk of nocturnal and fasting hypoglycemia, particularly in people with type 1 diabetes.[32]

Physical Activity :

Exercise is an important part of the diabetes management plan. Regular exercise has been shown to improve blood glucose control, reduce cardiovascular risk factors, contribute to weight loss, and improve well-being. Furthermore, regular exercise may prevent type 2 diabetes in high-risk individuals .[33] Physical activity is a key factor in the treatment of diabetes, particularly in type 2 diabetes, because obesity and inactivity contribute to the development of glucose intolerance in genetically predisposed individuals.

Regular exercise reduces cholesterol levels, raises HDL-C, lowers BP, augments weight-reduction diets, reduces the dose requirements or need for insulin or antihyperglycemic agents, enhances insulin sensitivity, and improves psychological well-being by reducing stress. Exercise increases glucose utilization, which is provided initially from the breakdown of muscle glycogen and, subsequently, from hepatic glycogenolysis and gluconeogenesis. These effects are mediated through norepinephrine, epinephrine, growth hormone, cortisol, and glucagon, along with the suppression of insulin secretion.[33]

In patients using insulin, hyperglycemia, normoglycemia, or hypoglycemia can occur secondary to exercise depending on the degree of control, recent administration of rapid-acting insulin, and food intake. Exercise in patients taking insulin must be tempered by increased food intake, potential delay in insulin administration, decreased doses of insulin, or a combination of these actions to minimize hypoglycemia.[28]

In patients with type 2 diabetes, plasma glucose concentrations usually decrease in response to exercise, but symptomatic

hypoglycemia is uncommon. The vascular benefits of exercise are particularly helpful in patients with diabetes given their predisposition to CVD. In general, exercise that produces moderate exertion (increase in heart rate of 20%–40% from resting baseline) is recommended with a starting goal of 150 minutes per week. The eventual goal is for patients to be able to achieve 50% to 70% of their age-adjusted maximal heart rate.[4]

2.7.2. Pharmacological Management :

Insulin, along with diet, is crucial to the survival of individuals with type 1 diabetes and plays a major role in the therapy of people with type 2 diabetes when their symptoms cannot be controlled with diet or noninsulin antidiabetic agents. Insulin also is used for people with type 2 diabetes during periods of intercurrent illness or stress (e.g., surgery, pregnancy). [8,9] The use of antidiabetic agents is reserved for the treatment of patients with type 2 diabetes whose symptoms cannot be controlled with diet and exercise alone.

2.7.2.1. Insulin Therapy :

Insulin is a hormone secreted from the pancreatic β cell in response to glucose and other stimulants (e.g., amino acids, free fatty acids, gastric hormones, parasympathetic stimulation, β-adrenergic stimulation).

The hormone is made up of two polypeptide chains (a 21-amino acid A chain and a 30-amino acid B chain),which are connected by two disulfide bonds. Proinsulin, the precursor of insulin, is a single-

chain, 86-amino acid polypeptide that is processed in the Golgi apparatus of β cells and then packaged into granules. In the storage granule, the connecting or C-peptide is cleaved from proinsulin to produce equimolar amounts of insulin and C-peptide.[34] Insulin and C-peptide are cosecreted, thus, measurable C-peptide levels indicate the presence of endogenously produced insulin and functioning β cells.

Insulin is crucial to the survival of individuals with type 1 diabetes, whose β cells have been destroyed. It also plays a major role in the therapy of individuals with type 2 diabetes when their symptoms cannot be controlled with diet and exercise alone or a combination of antidiabetic agents. Insulin also is used in patients with type 2 diabetes during pregnancy or periods of intercurrent illness or stress (e.g., surgery).[8]

Regular insulin, a solution, is the only insulin that can be administered by any parenteral route: IV, intramuscularly (IM), or subcutaneously (SC). All other insulins are only to be used SC.

Clinically, the most important differences among insulin products relate to their onset, peak, and duration of action (not the actual insulin levels, which is pharmacokinetics). Current insulin products can be categorized as rapid-acting, short-acting, intermediate-acting, and long-acting insulin.[5]

Table 2-5 : Duration of action (in hours) of insulin preparation

Insulin	Onset	Peak	Duration
Rapid-acting (insulin analogues: lispro, aspart, glulisine)	< 0.5	0.5–2.5	3–4.5
Short-acting (soluble (regular))	0.5–1	1–4	4–8
Intermediate-acting (isophane (NPH), lente)	1–3	3–8	7–14
Long-acting (bovine ultralente)	2–4	6–12	12–30
Long-acting (insulin analogues: glargine, detemir)	1–2	None	18–24

Insulin dosing regimens :

The choice of regimen depends on the desired degree of glycaemic control, the severity of underlying insulin deficiency, the patient's lifestyle, and his or her ability to adjust the insulin dose. Most people with type 1 diabetes require two or more injections of insulin daily. In type 2 diabetes, insulin is usually initiated as a once-daily long acting insulin, either alone or in combination with oral hypoglycaemic agents. However, in time, more frequent insulin injections are usually required.[35]

Twice-daily administration of a short-acting and intermediate-acting insulin (usually soluble and isophane insulins), given in combination before breakfast and the evening meal, is the simplest regimen and is still used commonly in many countries. Initially, two-thirds of the total daily requirement of insulin is given in the morning

in a ratio of short-acting to intermediate-acting of 1 : 2, and the remaining third is given in the evening.

Pre-mixed formulations are available containing different proportions of soluble and isophane insulins (e.g. 30 : 70 and 50 : 50). These are useful for patients who have difficulty mixing insulins, but are inflexible as the individual components cannot be adjusted independently. They need to be resuspended by shaking the vial several times before administration. Fixed-mixture insulins also have altered pharmacodynamic profiles, such that the peak insulin action and time to peak effect are significantly reduced compared with injecting the same insulins separately.[2]

Multiple injection regimens (intensive insulin therapy) are popular, with short-acting insulin being taken before each meal, and intermediate- or long-acting insulin being injected once or twice daily (basal-bolus regimen). This type of regimen allows greater freedom with regard to meal timing and more variable day-today physical activity.

2.7.2.2. Oral Antidiabetic Agents :

Table 2-6 : Effects of drugs used in the treatment of type 2 diabetes

	Insulin	Sulphonylureas and meglitinides	Metformin	Acarbose	Thiazolidinediones (glitazones)	DPP-4 inhibitors (gliptins)	GLP-1 receptor agonists	SGLT2 inhibitors
Fasting blood glucose	↓	↓	↓	↘	↓	↓	↓	↓
Post-prandial blood glucose	↓	↓	↓	↓	↓	↓	↓	↓
Plasma insulin	↑	↑	↓	↓	↓	↑	↑	↓
Body weight	↑	↑	→	→	↑	→	↓	↓
Risk of hypoglycaemia	++	+	-	-	-	-	-	-
Tolerability	Good	Good	Moderate	Moderate	Moderate	Good	Moderate	Limited experience

DPP-4 = dipeptidyl peptidase 4; GLP-1 = glucagon-like peptide 1; SGLT2 = sodium and glucose transporter 2)

Biguanides :

Metformin is the only biguanide now available. and it is now widely used as first-line therapy for type 2 diabetes, irrespective of body weight. Metformin is also given increasingly as an adjunct to insulin therapy in obese patients with type 1 diabetes. Approximately 25% of patients develop mild gastrointestinal side-effects with metformin, but only 5 % are unable to tolerate it even at low dose. The main side-effects are diarrhoea, abdominal cramps, bloating and nausea.

Mechanism of action :

The mechanism of action of metformin has not been precisely defined. Whilst classically considered an insulin sensitiser because it lowers insulin levels, its main effects are on fasting glucose and are insulinin dependent. Metformin reduces hepatic glucose production, may also increase insulin-mediated glucose uptake, and has effects on gut

glucose uptake and utilisation. At the molecular level, metformin acts as a weak inhibitor of mitochondrial respiration, which increases intracellular adenosine monophosphate (AMP) and reduces adenosine triphosphate (ATP). This has direct effects on the flux through gluconeogenesis, and activates the intracellular energy sensor AMP-activated protein kinase (AMPK), leading to multiple beneficial metabolic effects.[36]

Clinical use :

Metformin is a potent blood glucose-lowering treatment that is weight-neutral, does not cause hypoglycaemia and has established benefits in microvascular disease. It is employed as first-line therapy in all patients who tolerate it, and its use is maintained when additional agents are added as glycaemia deteriorates (see Fig. 21.9). Metformin is usually introduced at low dose (500 mg twice daily) to minimise the risk of gastrointestinal side effects. The usual maintenance dose is 1 g twice daily. There is a modified-release formulation of metformin which may be better tolerated by patients with gastrointestinal side-effects.

Metformin can increase susceptibility to lactic acidosis, although this is much less common than was previously thought. As metformin is cleared by the kidneys, it can accumulate in renal impairment, so the dose should be halved when estimated glomerular filtration rate (eGFR) is 30–45 mL/min, and it should not be used below an eGFR of 30 mL/min. Its use is also contraindicated in patients with impaired hepatic function and in those who drink alcohol in excess, in whom the risk of lactic acidosis is significantly increased. It should be discontinued, at least temporarily, if any other serious medical condition develops, especially one causing severe shock or hypoxia.

In such circumstances, treatment with insulin should be substituted if required.[23]

Sulphonylureas :

Sulphonylureas are 'insulin secretagogues', i.e. they promote pancreatic β-cell insulin secretion.

Mechanism of action :

Sulphonylureas act by closing the pancreatic β-cell ATP-sensitive potassium (KATP) channel, decreasing K+ efflux, which ultimately triggers insulin secretion. Meglitinides (e.g. repaglinide and nateglinide) also work in this way and, although short-acting, are essentially sulphonylurea- like drugs.

Clinical use :

Sulphonylureas are an effective therapy for lowering blood glucose and are often used as an add-on to metformin, if glycaemia is inadequately controlled on metformin alone. The main adverse effects of sulphonylureas are weight gain and hypoglycaemia. The weight gain is not ideal in patients with diabetes who are already overweight or obese, although sulphonylureas are effective treatments in this group.[36] Hypoglycaemia occurs because the closure of KATP channels brings about unregulated insulin secretion, even with normal or low blood glucose levels. There are a number of sulphonylureas. In the UK, gliclazide is the most commonly used; in contrast, in the USA, glibenclamide (also known as glyburide) is widely used. Glibenclamide, however, is long-acting and prone to induce

hypoglycaemia, so should be avoided in the elderly. Other sulphonylureas include glimepiride and glipizide. The dose–response of all sulphonylureas is steepest at low doses; little additional benefit is obtained when the dose is increased to maximal levels.[36]

Alpha-glucosidase inhibitors :

The α-glucosidase inhibitors delay carbohydrate absorption in the gut by inhibiting disaccharidases. Acarbose and miglitol are available and are taken with each meal. Both lower post-prandial blood glucose and modestly improve overall glycaemic control. They can be combined with a sulphonylurea. The main side-effects are flatulence, abdominal bloating and diarrhoea. They are used widely in the Far East but infrequently in the UK.

Thiazolidinediones :

Mechanism of action :

These drugs (also called TZDs, 'glitazones' or PPARγ agonists) bind and activate peroxisome proliferator activated receptor-γ, a nuclear receptor present mainly in adipose tissue that regulates the expression of several genes involved in metabolism. TZDs enhance the actions of endogenous insulin, in part directly (in the adipose cells) and in part indirectly (by altering release of 'adipokines,' such as adiponectin, which alter insulin sensitivity in the liver). Plasma insulin concentrations are not increased and hypoglycaemia does not occur.

TZDs increase pre-adipocyte differentiation, resulting in an increase in fat mass and in body weight.

Clinical use :

TZDs have been prescribed widely since the late 1990s, but recently a number of adverse effects have become apparent and their use has declined. One popular TZD, rosiglitazone, was reported to increase the risk of myocardial infarction and was withdrawn in 2010. The other TZD in common use, pioglitazone, does not appear to increase the risk of myocardial infarction but it does exacerbate cardiac failure by causing fluid retention, and recent data show that it increases the risk of bone fracture, and possibly bladder cancer. These observations have reduced the use of pioglitazone dramatically. Pioglitazone can be very effective at lowering blood glucose in some patients and appears more effective in insulin-resistant patients. In addition, it has a beneficial effect in reducing fatty liver. Pioglitazone is usually added to metformin with or without sulphonylurea therapy. It may be given with insulin therapy, when it can be very effective, but the combination of insulin and TZDs markedly increases fluid retention and risk of cardiac failure, so should be used with caution.[35]

Incretin-based therapies: DPP-4 inhibitors and GLP-1 analogues

The incretin effect is the augmentation of insulin secretion seen when a glucose stimulus is given orally rather than intravenously, and reflects the release of incretin peptides from the gut. The incretin hormones are primarily glucagon-like peptide 1 (GLP-1(and gastric inhibitory polypeptide (GIP). These are rapidly broken down by the peptidase DPP-4 (dipeptidyl peptidase 4). The incretin effect is

diminished in type 2diabetes, and this has stimulated the development of two incretin-based therapeutic approaches.

The ‘gliptins’, or DPP-4 inhibitors, prevent breakdown and therefore enhance concentrations of endogenous GLP-1 and GIP. The first DPP-4 inhibitor to market was sitagliptin; others now available include vildagliptin, saxagliptin and linagliptin. These drugs are very well tolerated and are weight-neutral .[2]

The GLP-1 receptor agonists have a similar structure to GLP-1 but have been modified to resist breakdown by DPP-4. These agents are not orally active and have to be given by subcutaneous injection. However, they have a key advantage over the DPP-4 inhibitors: because the GLP-1 activity achieved is supra-physiological, it delays gastric emptying and, at the level of the hypothalamus, decreases appetite. Thus, injectable GLP-1 analogues lower blood glucose and result in weight loss – an appealing therapy, as the majority of patients with type 2 diabetes are obese. Currently available GLP-1 receptor agonists include exenatide (twice daily), exenatide MR (once weekly) and liraglutide (once daily).[36]

Unlike sulphonylureas, both incretin-based therapies only promote insulin secretion when there is a glucose trigger for insulin secretion. Thus, when the blood glucose is normal, the insulin secretion is not augmented and so these agents do not cause hypoglycaemia.

SGLT2 inhibitors :

The sodium and glucose transporter 2 (SGLT2) inhibitor, dapagliflozin, was licensed for use in 2012. Glucose is filtered freely

in the renal glomeruli and reabsorbed in the proximal tubules. SGLT2 is involved in reabsorption of glucose. Inhibition results in approximately 25% of the filtered glucose not being reabsorbed, with consequent glycosuria.[8] Although this helps to lower blood glucose and results in calorie loss and subsequent weight loss, the glycosuria does result in increased urinary tract and genital fungal infections. With limited experience and evidence to date, the most appropriate position for SGLT2 inhibitors in the therapy of type 2 diabetes has yet to be established.[5]

2.8. Methods Of Monitoring :

Glycemic Control :

In addition to monitoring signs and symptoms associated with hyperglycemia, hypoglycemia, and the long-term complications of diabetes, an ongoing assessment of metabolic control is an integral component of diabetes management. Ideally, self monitoring of blood glucose (SMBG) results combined with laboratory measures of acute and chronic glycemia can be used to evaluate and adjust therapy. SMBG and A1C levels continue to be the two primary methods used to access glycemic control. Continuous glucose monitoring (CGM) of interstitial fluid is also available for people with diabetes.[4]

Blood Glucose :

FPG concentrations are commonly used to assess glycemic control in the fasting state because this is when glucose concentrations are most reproducible. FPG concentrations generally reflect glucose derived from hepatic glucose production because this is the primary source of

glucose in the postabsorptive state. The FPG is the most frequent test performed by patients when self-monitoring. Postprandial glucose concentrations (1–2 hours after the start of the meal) also are used to assess glycemic control when fasting glucose concentrations are within normal limits or when there is a need to assess the effects of food or drugs (e.g,. rapid-acting insulins, glinides) on meal-related glycemia. In nondiabetic individuals, glucose concentrations generally return to less than 140 mg/dL within 2 hours after a meal. One- to 2-hour postprandial concentrations primarily reflect the efficiency of insulin-mediated glucose uptake by peripheral tissue.[4]

Because glucose concentrations are affected by various factors (e.g., meals, medications, stress), single-time point measurements cannot be used to assess a patient's overall control. Most laboratories measure plasma glucose concentrations rather than whole blood because these values are not subject to changes in the hematocrit .The majority of glucose monitors report plasma referenced glucose concentrations. Whole BG concentrations are approximately 10% to 15% lower than plasma glucose concentrations because glucose is not distributed into red blood cells.

Self Monitoring Of Blood Glucose :

SMBG has made euglycemia, both preprandially and postprandially, an achievable goal (70–130 mg/dL). Patients and their health care providers can use SMBG to assess directly the effects of drug dose changes, meals, exercise, and illness on BG concentrations. With improved technology, decreasing costs, and increased coverage by health plans, SMBG is the day-to-day monitoring test of choice for all patients with diabetes. However, SMBG remains expensive for patients without health insurance, is invasive, and can be difficult for

some patients to perform depending on their technical ability.[3] Furthermore, to achieve maximal benefit from SMBG, both the clinician and patientmust be motivated and willing to spend the time required to interpret the data and modify therapy to improve glycemic control.

Patients with type 2 diabetes who are on therapy that can cause hypoglycemia: Individuals taking glinides, a sulfonylurea, or insulin therapy should know how to perform SMBG to detect hypoglycemia when experiencing symptoms consistent with hypoglycemia.

Patients with type 2 diabetes who are engaged in self-management of their diabetes: Even individuals using noninsulin therapies can benefit from SMBG to evaluate the impact of food, exercise, and antidiabetic medications on their BG.[33]

Blood ketones :

Monitoring of blood ketones may have a place in the home management of type 1 diabetes. A b-hydroxybutyrate below 0.60 mmol/L is normal, whereas values between 0.60 mmol/L and 1.0 mmol/L may necessitate more insulin, and concentrations greater than 1.0 mmol/L a warning to seek medical advice.

Glycated haemoglobin :

The glycosylated hemoglobin, or A1C, has become the gold standard for measuring chronic glycemia and is the clinical marker for predicting long-termcomplications, particularly microvascular complications. A1C is most commonly measured because it comprises the majority of glycosylated hemoglobin and is the least affected by

recent fluctuations in BG. A1C measures the percentage of hemoglobin A that has been irreversibly glycosylated at the N terminal amino group of the β-chain; the plasma glucose level and the life span of a red blood cell (RBC;∼120 days) determine its value.Thus,A1Cis an indicator of glycemic control during the preceding 2 to 3 months. In patients without diabetes, A1C comprises approximately4%to6%of the total hemoglobin. Values may be three times this level in patients with diabetes.[3,4]

The current A1C assay actually measures several different molecules of hemoglobin A (HgbA1c, HgbA1a, HgbA1b, HgbA0), not just A1C. Each laboratory establishes its own normal values for the A1C test (most are referenced to the normal range of 4%–6%).

A1C can be measured without any special patient preparation (e.g., fasting) and generally is not subject to acute changes in insulin dosing, exercise, or diet. A1C values can be used as an adjunct to assessing overall glycemic control in patients with diabetes or to diagnose diabetes and prediabetes.[14]

Chapter Three

Materials and Methods

3. Materials And Methods :

3.1. Formals and Study Design :

This descriptive cross sectional study was carried out on patients diagnosed with Type 2 DM for two years in the hospital to determine their compliance with diabetes .The study is formed by the patients in Diabetic Center of Diwanyia Hospital and it was carried out during December 2017 in the hospital with 100 patients who volunteered to participate in the research study.

The study about patient compliance in treatment of type 2 DM of two aspects :

1. Medication Adherence.
2. Self-monitoring of glycemic control.

The independent variables to explain the medication adherence included sociodemographic characteristics (age, gender, education level, marital status, professional activity, financial level), characteristics associated with disease and therapy (time since diabetes diagnosis, type of treatment, smoking state, microvascular or macrovascular complications…), and associated with medical care (decision making, follow-up by a specialist, acceptability of medical recommendations, ability for taking medicine alone, need for medical support or information on treatment…).

The research was approved by the scientific comity, college of pharmacy, university of Al- Qadisiyah, Iraq. verbal informed consent of the pharmacist was taken.

3.2. Statistical analyses :

DM Data were collected Through patient records at the Diabetes Center . Survey data obtained by this method were transferred to a computer program and analyzed with Excel sheet using a structured format .

Chapter Four

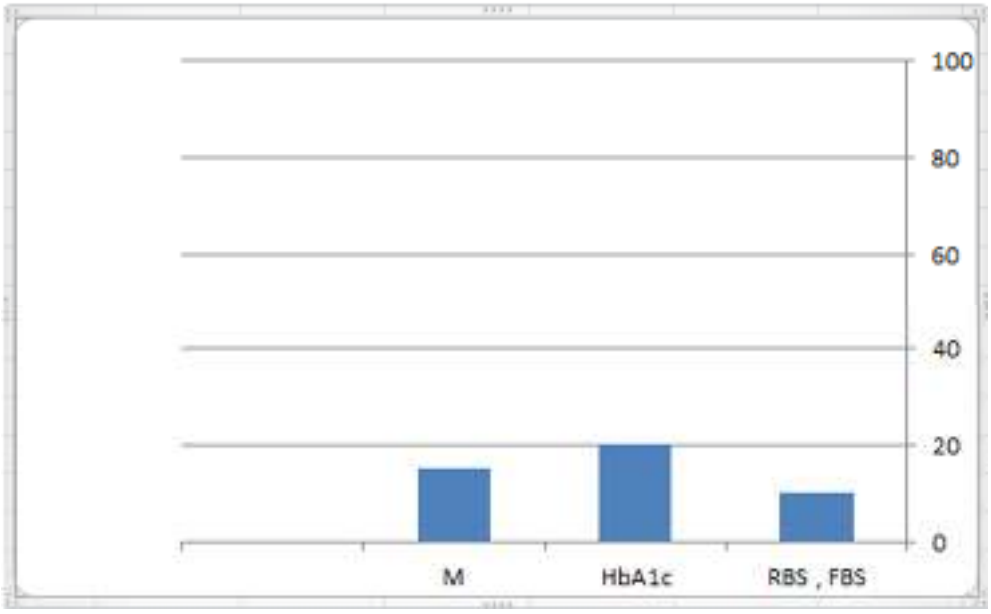
Results

4. Results:

This study included a total of 100 type 2 diabetic patients (50 men and 50 women) with a mean age of 45 years (18 to 65). The majority of patients with diabetes can significantly reduce the chances of developing long-term complications by improving self-care activities. Despite this fact, compliance or adherence to these activities has been found to be low, especially when looking at long-term changes. One hundred percent were treated with insulin only, percent of patients who adhere with treatment is 15%.

Self-monitoring of glycemic control is a cornerstone of diabetes care that can ensure patient participation in achieving and maintaining specific glycemic targets. The most important objective of monitoring is the assessment of overall glycemic control and initiation of appropriate steps in a timely manner to achieve optimum control. Self-monitoring provides information about current glycemic status, allowing for assessment of therapy and guiding adjustments in diet, exercise and medication in order to achieve optimal glycemic control. Self-care activities refer to behaviors such as following a diet plan, avoiding high fat foods, increased exercise, self-glucose monitoring, and foot care. Decreasing the patient's glycosylated hemoglobin level may be the ultimate goal of diabetes self-management but it cannot be the only objective in the care of a patient. Percent of patients who are measuring HbA1c is 20% and percent of patients who are measuring RBS , FBS is 10% .

Figure (4-1) : Percent of patient compliance



Chapter Five

Discussion

5. Discussion :

Adherence to treatment plan of diabetes is complex because it is not only restricted to taking medication. Other components are important such as self-monitoring, diet and physical activity. Improve adherence to treatment of diabetes mellitus is to improve glycemic control and therefore decrease morbidity and death associated to an uncontrolled diabetes and reduce the effective cost of the disease.

This particular adherence to treatment may be higher in elder patients, when compared to younger age groups, because among teenagers the lack of supervision, autonomy and social influences cause a decrease in it. Based on this idea and on the results of our studies we can infer that the greater the age of the diabetic patient, the greater the adherence to treatment.[37]

Socio-economic factors have been identified as important in adherence plan. The low level of education, illiteracy, unemployment, low income and geographic distance from health facilities can be significant barriers to adherence. It is expected that patients with higher duration of disease have a greater knowledge about the same, that can understand better the therapeutic regimen and consequently that can show a stronger adherence.

The act of taking daily medication and several times a day tells the patient that he suffers from a chronic disease, which can trigger a framework of denial and drug treatment. Extended therapies, common in chronic diseases, such as diabetes, where does not exist a definite improvement of the results, reveal a lower motivation to adhere to a treatment plan. According to the above statements and with the results of our study we can infer that the longer the duration of insulin administration, the lower the adherence to treatment. One of the

important factors in adherence is autonomy for carrying out self-care activities.[38] When the diabetic patient is not self-sufficient, it is essential that there is a good family support that contributes to the successful management and treatment compliance.

A factor that may influence adherence to therapy, perhaps the most common and most studied, is the knowledge that patients have about their disease and treatment regimen. In our research, more knowledge means better treatment adherence. These results demonstrated that patients, when facing disease, have to acquire knowledge and skills to manage their illness every day, its symptoms, its limitations, thus increasing adherence to treatment plan to achieve good metabolic control.

Daily treatment of DM requires a complex and demanding regimen, whose goal is to get closer of what is considered normal glycemic profile. Adherence to this regimen is difficult because it implies a multiplicity of daily self-care behaviors. For the study of adherence to treatment in diabetes, it is necessary to understand that there are several important self-care treatments, such as specific diet, practice of physical exercise, administration of medication, glucose monitoring, and feet care.[39] In our study, blood glucose monitoring and specific diet are the variables that influence treatment adherence, i.e. the higher the individual's involvement in blood glucose monitoring and specific diet the greater the adherence to treatment.

The relevance of adherence is unquestionable because the success of the treatment plan and the control of a chronic disease like diabetes depend on it. The lack of adherence to diabetes treatment is a widely recognized problem with magnitude in national and international scenes. However, there are gaps in the Educate diabetic patient.[40]

Chapter Six

Conclusions And Recommendation

6. Conclusions :

To prevent diabetes related morbidity and mortality, there is an immense need of dedicated self-care behaviors in multiple domains, including food choices, physical activity, proper medications intake and blood glucose monitoring from the patients.[41] Though multiple demographic, socio-economic and social support factors can be considered as positive contributors in facilitating self-care activities in diabetic patients, role of clinicians in promoting self-care is vital and has to be emphasized. Realizing the multi-faceted nature of the problem, a systematic, multipronged and an integrated approach is required for promoting self-care practices among diabetic patients to avert any long-term complications.[42]

Recommendations :

In summary, medication adherence is vital for effective diabetes management. Our findings point towards the interest of finetuning the primary care provider's approach to the individual patient by taking into account medication adherence. More evidence to support specific interventions that will be effective in overcoming adherence challenges for diabetes patients is needed. The patients should have a pivotal role in their diabetes management. Therefore, they need to acquire knowledge and skills, but also the ability for behavioural change, which often requires intensive patient-centred health education.[43] Health care providers should begin by taking time to evaluate their patients' perceptions and make realistic and specific recommendations for self-care activities. Some patients may experience difficulty in understanding and following the basics of

diabetes self-care activities. When adhering to self-care activities patients are sometimes expected to make what would in many cases be a medical decision and many patients are not comfortable or able to make such complex assessments. Furthermore, these requirements or modifications should be specific for each patient and should be altered depending on the patient's response.[44]

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داء السكري هو حالة مزمنة ناجمة عن نقص حاد في الأنسولين أو نقص نسبي للأنسولين نتيجة لإفراز الأنسولين والإجراء. على المدى الطويل تساهم هذه التشوهات الأيضية في تطوير مضاعفات مثل أمراض القلب والأوعية الدموية (CVD) واعتلال الشبكية اعتلال الكلية واعتلال الأعصاب وارتفاع خطر الإصابة بالسرطان. أهمية المتابعة المنتظمة لمرضى السكري مع مقدم الرعاية الصحية له أهمية كبيرة في تجنب أي مضاعفات على المدى الطويل.التعامل مع مرض السكري هو مجال الاهتمام والاهتمام للمهنيين الصحيين والباحثين السريرية على الرغم من قدر كبير من قبل وقد تم إجراء البحوث في المنطقة.

شملت هذه الدراسة ما مجموعه 100 من مرضى السكري من النوع 2 (50 رجلاً و 50 امرأة) بمتوسط عمر 45 سنة (18 إلى 65). يمكن أن تقلل غالبية المرضى المصابين بالسكري بشكل كبير من فرص تطوير مضاعفات على المدى الطويل من خلال تحسين أنشطة الرعاية الذاتية. وعلى الرغم من هذه الحقيقة ، وجد أن الامتثال أو الالتزام بهذه الأنشطة منخفض ، خاصة عند النظر في التغييرات طويلة الأجل. مائة في المئة تم علاجهم بالأنسولين فقط ، في المئة من المرضى الذين يلتزمون بالعلاج هو 15 ٪. قد يكون خفض مستوى الهيموغلوبين الغليكوزيلاتي المريض هو الهدف النهائي لإدارة مرضى السكري الذاتي ، ولكن لا يمكن أن يكون الهدف الوحيد في رعاية المريض. نسبة المرضى الذين يقيسون نسبة HbA1c هي 20٪ ونسبة المرضى الذين يقيسون RBS، FBS هي 10٪. إن عدم الالتزام بمعالجة مرض السكري هو مشكلة معترف بها على نطاق واسع من حيث الحجم في المشاهد الوطنية والدولية. ومع ذلك ، هناك فجوات في تثقيف المريض السكري. إن تناول الأدوية اليومية وعدة مرات في اليوم يخبر المريض أنه يعاني من مرض مزمن ، والذي يمكن أن يؤدي إلى إطار من الإنكار والعلاج من تعاطي المخدرات. إن العلاجات الممتدة ، الشائعة في الأمراض المزمنة ، مثل مرض السكري ، حيث لا يوجد تحسن واضح في النتائج ، تكشف عن وجود دافع أقل للالتزام بخطة العلاج.

وزارة التعليم العالي والبحث العلمي
جامعه القادسية / كليه الصيدلة

دراسة حول التزام المريض في مستشفى الديوانية علاج النوع الثاني من داء السكري

بحث تخرج مقدم إلى جامعه القادسية , كليه الصيدلة

تقدم به كلُ من :

الطالب أحمد كريم درني والطالب أحمد ناظم عبد

بأشراف

الأستاذ الدكتور

باسم أرحيم محمد الشيباني

كليه الصيدلة , جامعه القادسية

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